IODINE DEFICIENCY AND SCHOOLING ATTAINMENT IN TANZANIA†

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Abstract: An estimated one billion people globally are at risk of iodine deficiency disorder (IDD), the only micronutrient deficiency known to have significant, irreversible effects on cognitive development. Reductions in human capital due to cognitive damage from IDD have potentially important implications for economic growth in afflicted settings. 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 between 1986 and 1997. We look for evidence of improvements in cognitive ability attributable to the intervention by assessing whether children who benefited from iodine supplements 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 weak evidence that the program also reduced child but not fetal or infant mortality, which may bias downward the estimated effect on education. There is no indication that IOC improved rates of illness or school absence due to illness among children 10 to 14, suggesting that IOC improves schooling through its effect on cognition rather than its effect on health. Cross-country regression estimates also indicate a strong negative influence of total goiter rate and strong positive influence of salt iodization on female school participation. These findings provide micro-level evidence of the direct influence of ecological conditions on economic development, in addition to suggesting 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, 2003; Sachs and Gallup, 2001). 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).[1]

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 disorder (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.[2] 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 human 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. Furthermore, since fetal IDD permanently limits intellectual functioning, its impact is likely to be particularly acute and persistent.

This research looks for evidence of the influence of iodine deficiency on human capital 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

[1] Epidemiological criteria for assessing sufficient iodine intake is 100 μg or above.
[2] Shields are large areas of exposed Precambrian rock found in regions that have been little affected by tectonic processes, which occur near plate boundaries. As a result, surface rock on shields is over 570 million years old.

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 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 malnutrition and human capital.[3] Assessing the importance of physiological determinants of schooling informs a fundamental debate in the literature on barriers to education in developing countries surrounding the importance of supply-driven explanations for low levels of human capital investment relative to differences 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.

Of particular interest is the possible role of iodine deficiency in explaining gender differences in schooling outcomes in light of new evidence from laboratory studies in animals which find greater sensitivity of the female fetus to maternal thyroid deprivation on cognitive development. If girls are more susceptible to cognitive damage from IDD in utero as the laboratory evidence suggests, 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

[3] The influence of macro-nutrients (energy and protein intake) has been examined through subsidized school meal programs and nutrition supplements, 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 find benefits of micronutrients 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).

largest and most intensive programs, ultimately reaching approximately 25% of the population. As a result, an estimated 1.9 million babies born during and immediately after the program were protected from fetal IDD.[4] 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 program's initial effect on children born during the intervention 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 cognition 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 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 subpopulations to fetal IDD based on amount of iodine-depleting foods in the local diet. Our results also 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 and no apparent differences in health status of survivors according to program participation, this influence is unlikely to account for the schooling results and could bias downward the estimated effects on education.

To examine the implications of our findings at a macro level, we also run cross-country regressions of school participation on baseline IDD and fraction of population consuming adequately iodized salt. The results reveal a negative correlation between baseline iodine deficiency and female secondary schooling, and indicate that early salt iodization has already exerted a positive effect on female primary schooling attainment. Based on our micro-level estimates from Tanzania and cross-country data on baseline IDD and recent reductions in TGR, 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

[4] Estimation based on 1988-1994 population and (crude) birth rate.

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 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.[5]

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. 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 areas where bedrock is rich in iodine (Hetzel, 1989).

Humans require iodine for the biosynthesis of thyroid hormone. In utero development of the central nervous system required for intellectual functioning depends on an adequate supply of thyroid hormone, which influences the density of neural networks established in the developing brain (Lamberg, 1991).[6] Cretinism, a relatively rare form of mental retardation that occurs under extreme deprivation, is the most severe manifestation of cognitive damage from insufficient maternal and fetal thyroid hormone.[7] In utero 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 in childhood and adolescence, although the evidence is mixed.[8] In general, existing evidence from human studies suggests that non-cognitive outcomes occur only under extreme deprivation and that in utero damage from IDD is overwhelmingly cognitive (Zimmerman, 2005; Hetzel, 1983). Furthermore, animal and human studies indicate that cognition is sensitive to

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[5] Hetzel (2000) estimates that less than half the population in 83 developing countries consumed adequately iodized salt in the mid-1990s.

[6] The recommended daily iodine intake is 50 mcg for infants under 12 months, 150 mcg for adults and 200 mcg for pregnant and lactating women (WHO, 1996).

[7] 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)

[8] See Allen L and Gillespie S (2001) for a review of the evidence.

iodine deficiency during early fetal life, prior to mid-gestation, whereas physical growth and psychomotor development are believed to be most severely affected by iodine deficiency in childhood (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 has IDD been widely recognized as a leading cause of intellectual impairment (Merke, 1984; Delange, 2000; Haddow, 1999).[9] 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 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 that iodine prophylaxis given before or during pregnancy resulted in improved cognitive functioning in 50 offspring examined two to three years later (Shrethsa, 1994). The difference corresponded to a 10-15 point improvement in IQ relative to children in untreated communities. To our knowledge, the long-term impact of increased iodine intake during pregnancy 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, lack of statistical significance and questionable validity of the comparison groups (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.

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

More conclusive evidence of biologically-driven gender differences in iodine sensitivity comes from recent laboratory experiments of maternal thyroid deficiency in animals. Despite the fact that thyroid conditions of all types are consistently higher among women, 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 biochemicals of maternal origin 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, a 2000 study by Friedhoff et al. found that the effect of artificially restricting maternal thyroid hormone in utero on fetal neurodevelopment and behavioral outcomes was significantly larger in female relative to male rat progeny.

Although the mechanism underlying sex-selective effects of maternal nutrient deprivation on brain development could not be directly addressed by their experiment, a recent study of gene expression in nutrient deprived fetal guinea pigs by Chan et al (2005) provides insight into the cellular pathways. In particular, 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 (van der Haar et al, 1998). In endemic regions, 13% of children under five and 52% of pregnant and lactating women showed manifestations of iodine deficiency prior to the intervention.

3.2 Schooling in Tanzania

The Tanzanian formal education system involves seven years of primary education, four years of junior secondary, and two years of senior secondary. 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 while distance to primary schools was an issue only for a minority of rural households, 8% of which reported the nearest primary school to be more than 6 kilometers away (THBS, 2001). Throughout the country, gross enrollment ratios are higher than net enrollment ratios because many over-age children are present in primary schools due to beginning schooling late and progressing slowly.

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. However, 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. Admission to secondary schools is screened by performance on the mandatory Primary School Leaving Exam (PSLE), which students take one or two years after grade 6, and gender differences in secondary enrollment are almost entirely accounted for by differences in PSLE pass rates. Although in 2001 roughly the same numbers of boys and girls completed primary school and sat for the national exam required for secondary school, boys were 69% more likely to pass. As there is cost and no benefit other than admission to taking the test, this fact alone suggests that parents' preferences for male 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 relatively 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).[10] As show 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.

[10] Two districts, Bukoba Rural, Kagera, and Mbinga, Ruvuma, were added late and are excluded from the study.

In program districts, women of child-bearing age were targeted to receive 380 mg capsules once every two years, the expected duration of protection from this dose.[11] 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. Furthermore, penetration rates were lower than planned, ranging from 60 to 90 percent of the target population with average coverage across all districts and all years of 64%.[12] Coverage declined inversely to distribution round, at least in part due to rumors that IOC was a family planning aide (Magombo, 1990). Finally, districts were reached less frequently than once every two years due to administrative problems and caution over administering supplements frequently (Peterson, 2000).

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 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 program impact on children of women protected from IDD during pregnancy is likely to be even 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

[11] 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).

[12] 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.

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.

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 that are residing in the household and that 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.[13,14] Excluding non-resident children is necessary due to the fact that schooling outcomes are only available for household members. Excluding observations that cannot be linked to mothers is necessary to minimize the number of children born outside the district and hence measurement error in program participation 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. In intervention districts as well as rural districts outside of the intervention areas, enrollment rates for both boys and girls peak at 10 and fall monotonically thereafter. Across the entire rural sample, only slightly more than 50% of nine-year-old children are enrolled. Since no data are available from either survey on age of school entrance, which is determined largely by local school policies and norms, restricting the sample to ages by which most children can be assumed to have entered minimizes variation in grade attainment that is independent of children's cognitive or physical health. 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

[13] 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.

[14] 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.

[15] Comparing birth history data to the household roster, 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

were collected only for children living in the 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 full 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 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% are enrolled in Standards I to VII, 4.3% are enrolled in Standards VIII to X and 9.76% are not studying.

Although the TDHS sample is considerably smaller than the THBS, it has two principal advantages. First, information on birth month 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 age cut-offs and 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 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 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). Furthermore, since brain development of the fetus takes place during the first month of pregnancy, it is believed that most

are 25% more likely and at 17 they are 64% more likely to have left. Common reasons for leaving before age 18 are likely to be marriage for girls and high school attendance for boys.

[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 distribution scheme designed to administer as many pills as possible in a short amount of time (Peterson et al., 1998).

of the consequences become permanent by the second trimester.[18] 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 et al., 1989; Painter et al., 2005).

Under these assumptions, 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 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 rounds, which we assume to be three months based on project reports, and the timing of distribution rounds over the year, which we assume to be uniform (Peterson et al, 1998). 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 $t > 9$.

The second probability calculation, which pertains to kids born 2-4 years after the program, requires an assumption regarding the depletion rate of iodine from 380mg supplements, which is stored in the adipose tissue and excreted gradually from the body. Unfortunately there is little information on which to base the depletion assumption given large variance across populations and individuals in the speed of iodine depletion and few scientific studies that follow subjects for more than a year.[19] Based on existing evidence, we make the following assumptions: First, we assume that 85% of iodine is extracted in urine immediately, implying an initial loss of 323 mg of iodine in the first month, after which point it is depleted hyperbolically.[20] In general, iodine stored in fatty tissue appears to be depleted hyperbolically with the majority of urinary extraction occurring in the first week and then tapering off gradually (Wolff, 2001). Second, based on results from three separate human studies of 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). These two assumptions allow us to calculate the rate of depletion after the point of full protection. In particular, given that baseline iodine deficiency varies across treated individuals, the

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[18] One experimental study on the timing of iodine supplements for preventing cretinism confirms this: iodine treatment during the first trimester protected the fetal brain from cognitive damage, while treatment later in pregnancy and after delivery had no effect on neurologic status (Cao et al, 1994).

[19] Patterns of iodine extraction are 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).

[20] 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.

fraction of treated who are adequately covered will presumably decline after the point of full protection (24 months) at an unknown but gradual and decreasing rate.[21] If 6.5 mg is the minimum amount necessary to fully protect a pregnant woman for one complete month, our first two assumptions imply a half-life (at 1) of 3 months, which means that iodine levels will continue to fall for an additional 14 months after t=24 until they reach ineffective levels to protect anyone in the population (>4.2 mg) at t=38.[22]

Figure 2 illustrates the pattern of iodine depletion implied by our assumptions, and exact probabilities for each birth month are provided in Appendix A and presented in Figure 3. Since these assumptions are impossible to verify, we include in the regression analysis a correction factor for potential measurement error in the estimated treatment probability by including 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.

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. 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

As robustness check, 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.

4.3 Estimation Strategy

We estimate the effect of IDD 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

[21] We assume based on the recommended daily allowance for pregnant women (2 mg) that 6.5 mg of iodine stores are sufficient to ensure at least 1 full month of coverage.

[22] The half-life implied by our assumptions falls within the range identified from a meta-analysis of four studies of the approximate half-lives of urinary iodine excretion after oral oil administration to iodine-deficient populations, which revealed half lives of 3 to 7.5 months (Wolff, 2001).

is high variance in the rate at which children progress through school. Meanwhile, since few children drop out of school in the age range to which our analysis is restricted (primary school) progression is presumably a considerably more sensitive indicator of future schooling attainment than enrollment.

Table 2 presents summary statistics from the full THBS sample divided according to the timing of IOC implementation. 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 greater distance between households and secondary schools, and have lower consumption of fish, a rich source of iodine. 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 pattern suggests that districts in which IOC began early were better off than late districts, consistent with the most common source of delay being poorly organized distribution networks. 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:

$$grade_{\mathrm{if}} = \alpha + \beta_1\,(\mathrm{T_{if}}) + \beta_2\,(\mathrm{A_{if}}) + \beta_3\,(\mathrm{X_{if}}) + \mu_{\mathrm{f}} + \varepsilon_{\mathrm{if}}$$

Here T_i is the likelihood child i in family f was protected from IDD during the first trimester, 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 analysis samples is the use of birth month data in the TDHS estimates, which is used to refine the definition of treatment and also added to the controls to account for the independent effect of small differences in age on school entrance or progression. The household fixed effects model minimizes the potential confounding role of unobservable cohort effects that might vary systematically by 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. 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.

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 the outcome 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.

Importantly, there are few potential confounding factors that would not be absorbed in the fixed effects. First, since treatment occurred at the district level, potential confounders must be district-wide trends that coincide with multiple rounds of IOC distribution in timing and duration. Furthermore, unless such trends systematically lagged IOC by several years, they would have to impact children in utero but not early childhood in order to have had a lasting effect on their outcomes relative to slightly older peers. This reduces the set of potential confounders to changes in fetal health environment other than iodine that coincided with IOC distribution. For instance, if the timing of distribution rounds were driven by intermittent declines in the quality of district prenatal health services, 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 a critical stage of development.

Information from program reports also provides evidence that variation in treatment was independent of other fetal or infant health shocks. A post-intervention study by Peterson (2000) provides a detailed account of sources of delay gleaned from IOC program reports and administrative records, interviews with past and present program managers, and supervision visits to selected districts. In all cases, start date was ultimately determined by an external rather than an internal force. According to the study, lags in program start date were due to administrative delays resulting from the logistical challenges of district-wide IOC distribution. 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.[23] Given the central role of external resource

[23] 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

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.

In addition, we assess the degree to which possible health shocks pose a threat to our estimation strategy by testing whether variation in IOC is related to observable health status of children and reported number of 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 health status between treated and untreated children is evidence that the treatment effect is driven by IOC. Testing for a program effect on childhood health status is also useful for assessing the possibility that IOC operates through reducing childhood illness rather than improving cognition.

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). Since enrollment is virtually unchanged over this interval, the results indicate that fetal IDD influences the rate at which students progress through school. When the regressions are run separately by gender, the estimated effect is twice as large and statistically significant among girls but not boys. 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), which suggests that orphaned children are likely to live in the district 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 similarity between household and district fixed effect results when both samples are restricted to households with more than one sample member (columns 10 and 11).

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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.

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 in almost every regression, 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.

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.[24] 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 by practitioners and program administrators. Even if they were not, coverage is likely to be higher than average among pregnant women since they are more likely than men or children over age 1 to visit health centers where IOC were frequently distributed in regions with low coverage. 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

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

years of schooling 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 right-censored, although the direction of bias is ambiguous. In general, gaps in grade attainment widen over time as a disproportionate number of slow achievers drop out of school. Furthermore, it is possible that schooling outcomes are more sensitive to learning disabilities at the secondary relative to the primary level. 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. 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, and censored data models are unlikely to be appropriate for estimating the total effect of the program on schooling attainment given the substantial barriers to secondary school enrollment which are likely to generate sharp discontinuities in grade attainment around age 14.

In 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. The gender difference is particularly stark – and statistically significant – when the program effect is allowed to vary by age. This indicates catch-up among 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, suggesting drop out at completion of primary school.

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. 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), baseline gender differences in age of entry are relatively small. To account for the full gender difference in IOC, a 0.2 year difference in age of entry would have to correspond to twice the effect of IOC on attainment, which could only happen if the influence of ability on pass rates were highly non-linear with age.

A more compelling reason for differences across girls and boys in the impact of IOC 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 opportunity, financial or social cost of enrolling girls in school were higher than that of boys due to girls' higher productivity at home or greater opportunities for marrying young. If so, the same cognitive benefit of IOC could translate into greater schooling improvements for girls. Unfortunately, without data on cognitive capacity, there is no simple way to distinguish this explanation from a disproportionate improvement in cognitive capacity among girls. However, we can investigate the possibility by assessing whether girls' rate of school progression is more sensitive to ability in a regression of educational attainment on ability measures available in an outside sample of Tanzanian school children. To do so, we use data from the 1993 Tanzanian *Living Standards Measurement Survey*, one of the few nationally representative household surveys to collect cognitive test scores from a subset of survey respondents. In particular, 404 children between the ages of 7 and 14 who were present in the home at the time of the interview were randomly chosen to take a streamlined 16-question version of the Sabot's test of cognitive ability, a test that measures basic math and reading skills which has been used in evaluations of the Tanzanian school system since the 1970s (Knight and Sabot, 1990). With this sample, we regress grade attainment on gender, test score, and their interaction, controlling for year of birth. The results are presented in Appendix B. If our previous results reflect parents' gender-specific responses to ability, test score should be a more powerful predictor of female than of male schooling attainment. Instead, the estimates indicate that male and female school progression and enrollment are equally sensitive to ability, which supports the interpretation that gender differences in schooling responses to IOC reflect gender differences in the effect of IOC on ability.

5.2 Program Effects According to Local Diet

The final regression exercise divides districts in the sample according to level of cassava consumption in order to examine variation in program effect according to intake 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 that are unique to the developing brain (Rao and Lakshmy, 1995).

Cassava, one of the most goitrogenous food products, 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.[25] Cassava has the potential to significantly decrease iodine absorption if not properly fermented.[26] 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 (Bilabina et al, 1995; Delange et al, 1994).[27]

We predict a highly non-linear relationship between cassava consumption and program impact. 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, if consumed in high enough quantities, the cyogenic effect of high cassava is likely to impair the impact of IOC. As a result, we anticipate a threshold level of cassava consumption below which rates of IDD are too low to observe a significant average treatment effect, and a second threshold above which 380 mg of iodine will be insufficient to protect against IDD due to heightened daily requirements for iodine intake.

To test these predictions, the district rate of cassava production in 2000 is used to proxy for variation in dietary intake 10 to 15 years prior. Specifically, 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

[25] 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.
[26] 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."
[27] In one study in Tanzania, insufficient cassava processing was correlated with TGR (Peterson, 1994).

terciles of cassava production. In high production regions, between 41 and 60% of households consume home-grown cassava, compared with between 11 and 40% in medium production regions and fewer than 10% of households in low productions districts. Given that households also eat cassava produced outside, this clearly underestimates actual intake of goitrogens. However, geographic variation in produce availability is likely to constitute a reasonable predictor of dietary differences a decade earlier, which is arguably a preferable proxy of past diet than is current diet given recent changes in household diet.

As predicted, the regression estimates reveal an inverted u-shaped relationship between cassava consumption and impact of IOC on fetal brain development (Table 5). In areas with highly goitrogenous diets the program appears to have had little effect, suggesting that maternal iodine from IOC was depleted by intake of goitrogens. Meanwhile, in areas with relatively little cassava consumption, the program effect is also small, which presumably reflects the fact that these were the districts with relatively low baseline TGR. Figure 5 splits the sample 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 areas with the highest TGR benefit the least from even a program as intensive as IOC.

5.3 Robustness Checks

One possible concern is that, because we are making inferences about program effect based on cohort differences, our results may simply reflect time trends in schooling attainment 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 2-year schedule, there are multiple instances of an older but not a younger sibling receiving IOC, allowing 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. In total, among sibling pairs in the 2000 sample in which only one individual was treated, the older sibling was treated in 23% of cases.[28]

Figure 6 shows the average difference in grade attainment across all sibling pairs in the 2000 sample, 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

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

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. Such a pattern could only be explained by a very complicated non-monotonic and district-specific time trend in schooling. Furthermore, there is no measurable difference in average schooling attainment when both siblings are treated versus when neither is treated. Regression estimates of grade differences on sibling age gaps and indicators of which sibling received IOC reveal statistically significant (at the 10% level) program effects of the expected sign in both comparisons (Table 6).

To verify that estimated program effects are not driven by time invariant district-specific patterns of schooling attainment by age, we estimate a placebo regression of grade attainment among children in the sample districts that were 10 to 13 in 1988 on a pseudo-indicator of IOC that pertains to kids of the same age and district in 2000. Since children who were between 10 and 13 in 1988 were too old to benefit from IOC in utero, if our results truly reflect an effect of IOC we should observe no relationship between the treatment variable and schooling outcomes in this sample. These results are presented in Table 7. For consistency with the previous estimates, we run the regressions separately by gender using both household and district fixed effects. 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 sample, 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.

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, the channel of influence is impossible to verify. Since 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 attend and progress through school.[29] Iodine deficiency has been demonstrated in laboratory studies to influence fetal brain development much more readily than physical development, however 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.

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

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 member's 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 fetal (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. 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. In addition, we approximate the program effect on conception and early fetal mortality by looking at 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 the 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, 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. This is strikingly high given baseline child mortality of 8.9% in treatment districts.

This result not only provides new evidence of the health benefits of IOC, but also has relevance for the findings on grade attainment. The magnitude of the estimated effect and the fact that previous studies have only found reductions in child mortality in combination with similar improvements in neo- and peri-natal mortality, this finding should be interpreted with caution. Nonetheless, there are three implications worth noting. First, the child mortality result 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 give rise to higher schooling attainment at ages 10-13 ceteris paribus given that child mortality presumably selects out the most vulnerable. As a result, 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.

While selection on unobservables from differences in rates of child mortality associated with IOC is not likely to bias our estimates towards finding an effect or account for the gender results, it is possible that lower mortality among treated children corresponds to higher average health status among survivors. Not only does this imply that the program effect may operate through differences in health status rather than cognition, but it could also account for the gender difference if girls' schooling is more sensitive to illness. To study this more closely, Table 9 presents results from regressions of survivors' health status at ages 10-13 on program participation analogous to the Table 3 estimates. 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, or an accidental injury 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. The latter outcome is a particularly useful test of whether the program effect operates through illness since school days missed due to illness is the mechanism through which in utero health damage would most likely account for the observed program effect on schooling.

Regression results indicate that children in utero during the program are no more likely to experience illness at ages 10-13, suggesting no significant program effect on the average health status of survivors. The estimates also indicate no relationship between IOC and school days missed due to illness (column 1), nor is there any evidence that children covered by the program report fewer episodes of ill health (column 2). Although the long run effects of in utero health shocks might show up in a number of different childhood health outcomes given that fetal micronutrient deficiencies are thought to weaken an individual's overall immune system, the estimates in columns 2 through 6 indicate no program effect on a wide range of observable measures of child health at ages 10-13. 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.

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. At the end of primary school, children sit for the PSLE, which is required for admittance to any lower secondary school in the country. The pass rate for this examination has traditionally been abysmal 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. Although roughly equal numbers of boys and girls take the test, gender differences in PSLE pass rates are striking: In 2001, only 21.4% of girls who sat for the PSLE passed the examination compared to 36.2% of boys. By 2004, although the number and gender ratio of test-takers had changed little, 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, reducing the gender difference considerably in terms of pass rates while maintaining roughly the same percentage point gender gap.

Our empirical analysis tests whether secondary school transition rates in 2004 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.[30] 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. The following PSLE data are available from the Ministry of Education for 93 of the 106 districts in the country: number of boys and girls who take the test in 2004, and number of boys and girls who receive each of five categories of test grade, three of which constitute passing grades.[31] With this information, we examine the fraction of students who take the PSLE, and the fraction of test-takers who pass and therefore transition to secondary school by running the following regressions separately by gender:

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

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

In both estimates, an observation is a district. The first outcome, $testtakers04_d$, 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, $pop04_d$ is the number of males or females in district *d* between the ages of 10 and 14 in the 2002 Census (therefore the high-school-age population in 2004), and $hsrate88_d$ 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 number of girls or boys who pass the 2004 PSLE (achieve a grade of C or above), and the regression conditions on 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.[32] Unfortunately, 2002 census data on school enrollment are currently unavailable, but 2004 PSLE pass

[30] 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.
[31] 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.
[32] 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).

rates are likely to be a reasonable proxy for the secondary school transition rate in the cohort of test-takers given the extreme competition for slots in 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 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 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 on 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[33]; 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.[34] To approximate the level of iodine deficiency prior to salt iodization, TGR from a year prior to 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.[35] We then test whether reductions in IDD over this period are associated with improvements

[33] 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.

[34] 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 the goiter rate vs. a single number, etc).

[35] Control variables collected primarily from the World Bank Group's World Development Indicators. All regressions include the following set of controls: Malaria prevalence, HIV prevalence, urbanicity (urban

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 10.

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%.[36] 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 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.

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population as % of total population), population density (per square kilometer), log GDP per capita, log GDP per capita squared, and terms of trade (Export value Index / Import value index).

[36] 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.

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.[37] 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, 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. Our estimates indicate that variation in iodine availability likely plays an important role in economic growth as 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.

[37] 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.

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.

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 attainment in much of Africa and other parts 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 in order 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 future returns well outweigh the costs.

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Figure 1: Intervention districts

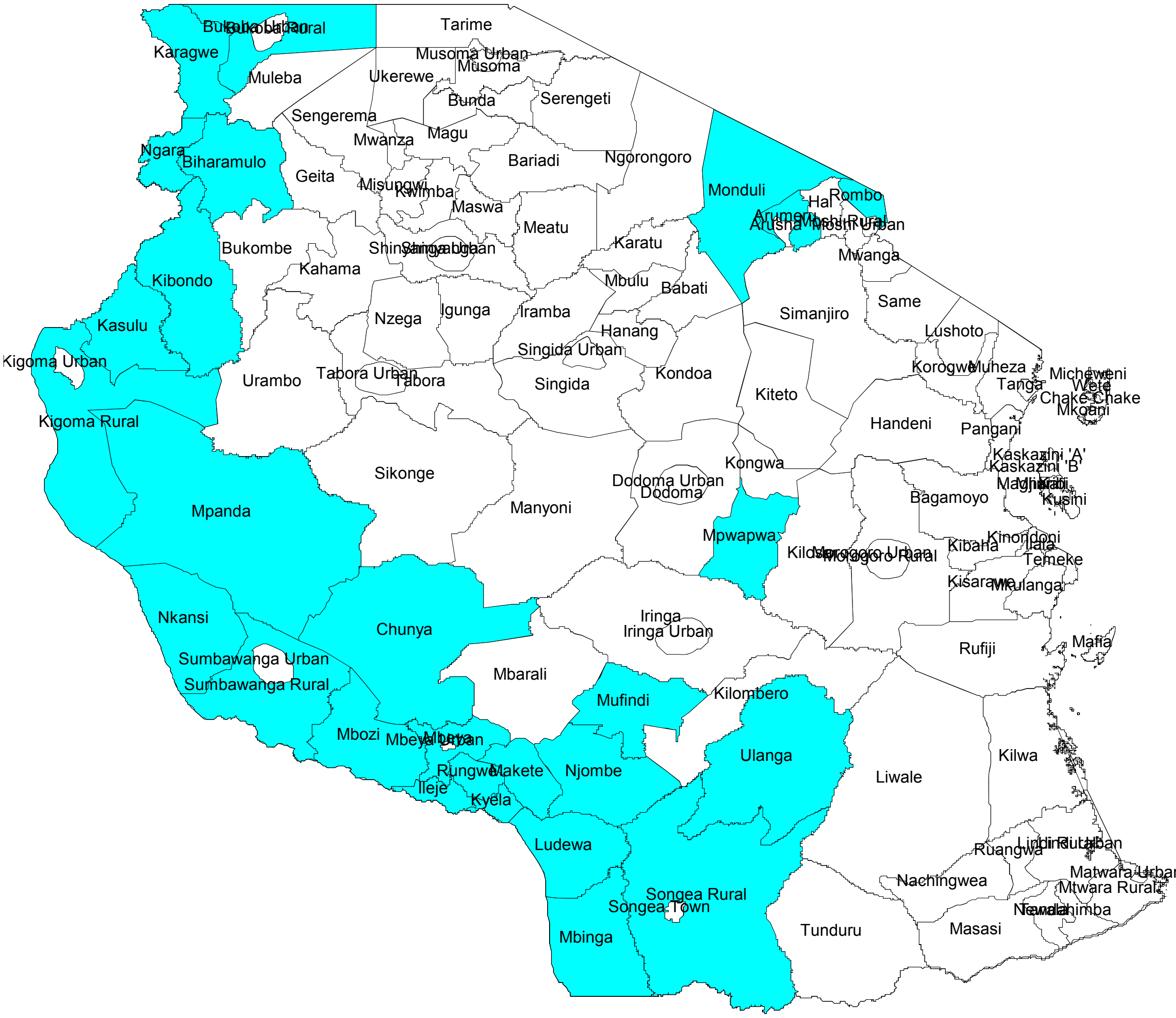


Figure 2: Iodine remaining in body after administration of 380mg iodized oil

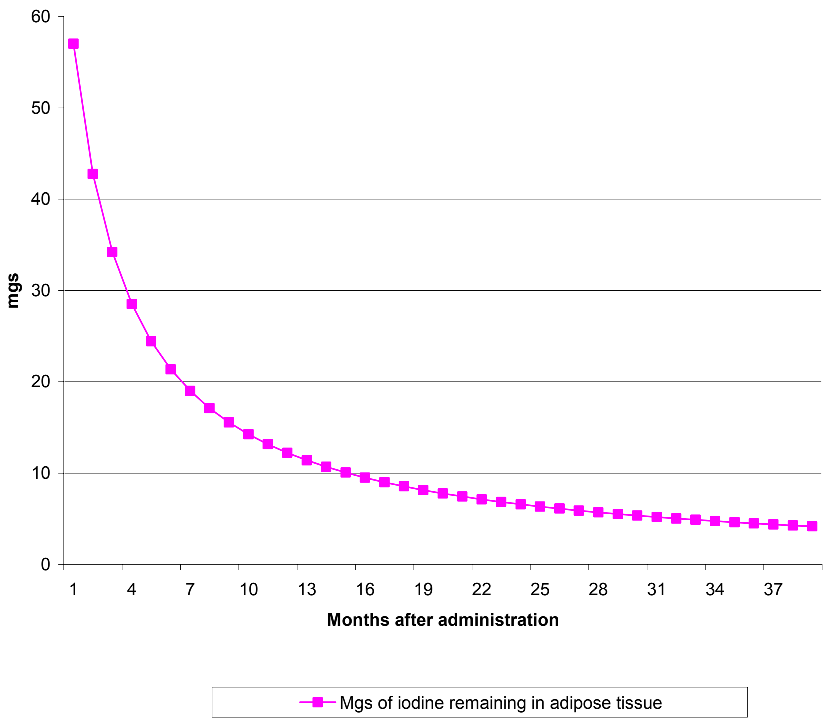


Figure 3: Child's likelihood of protection from IDD during first tri-mester

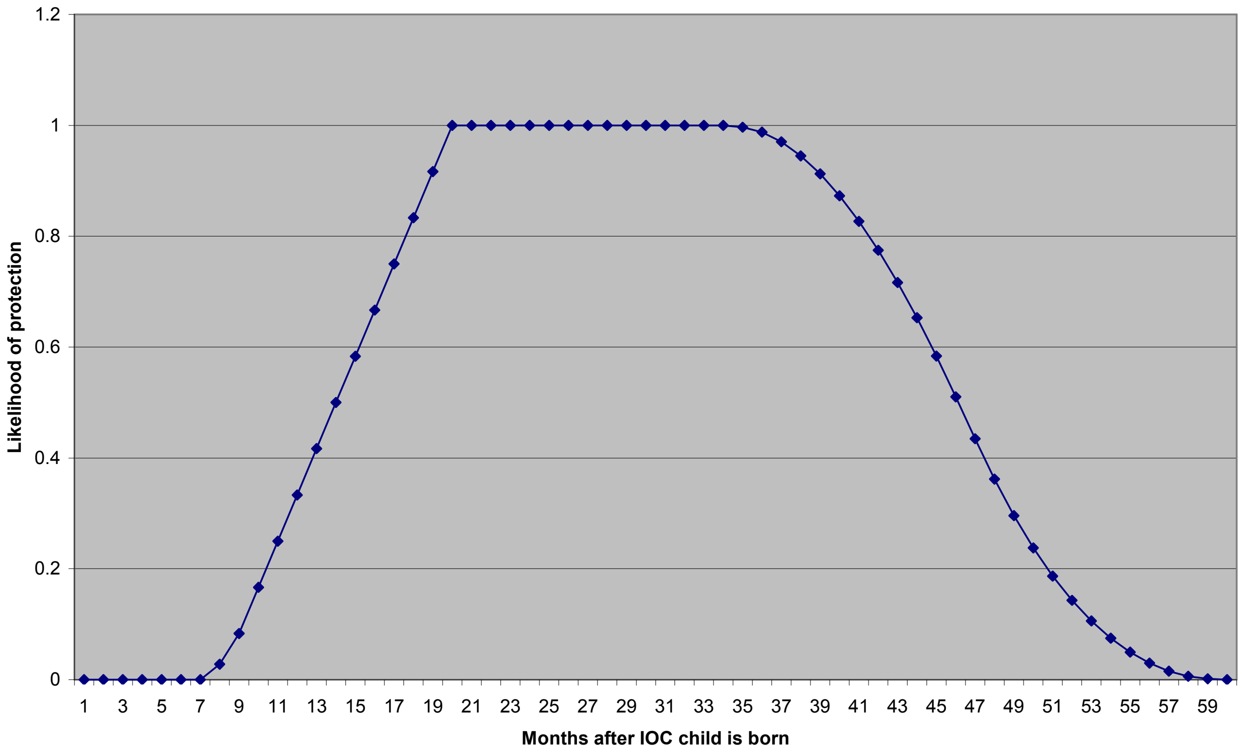
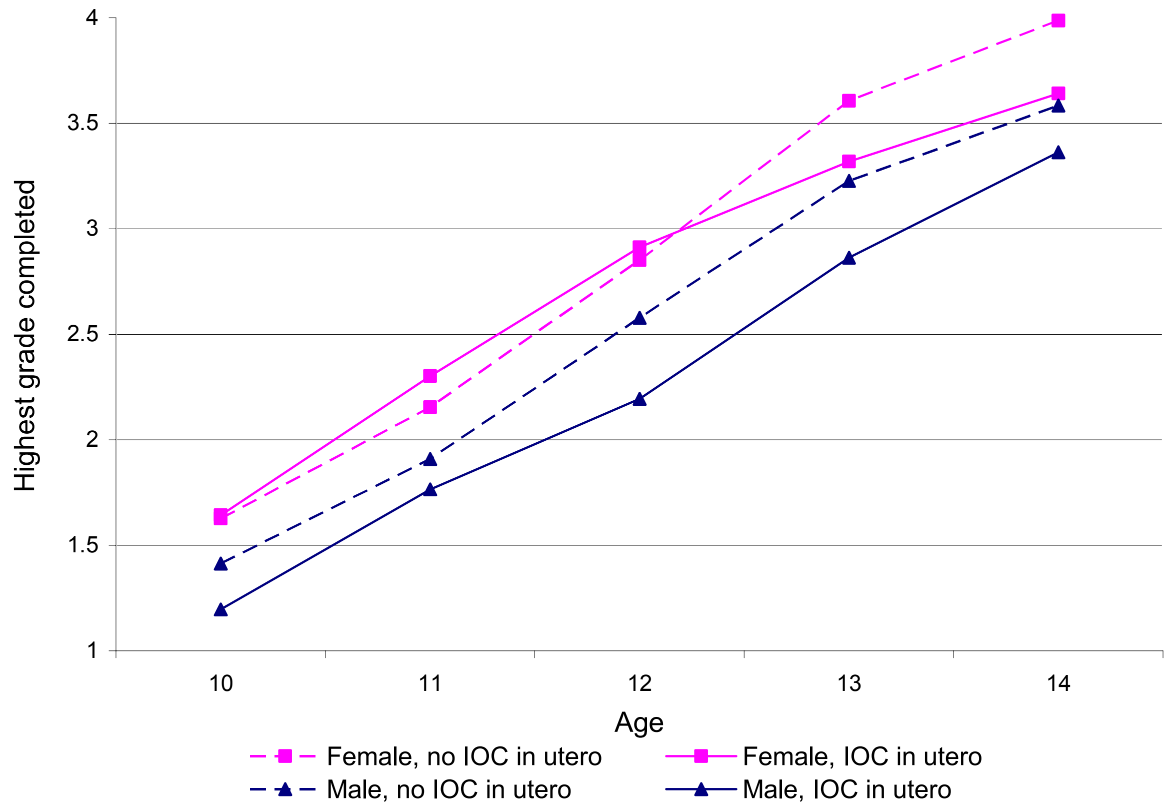
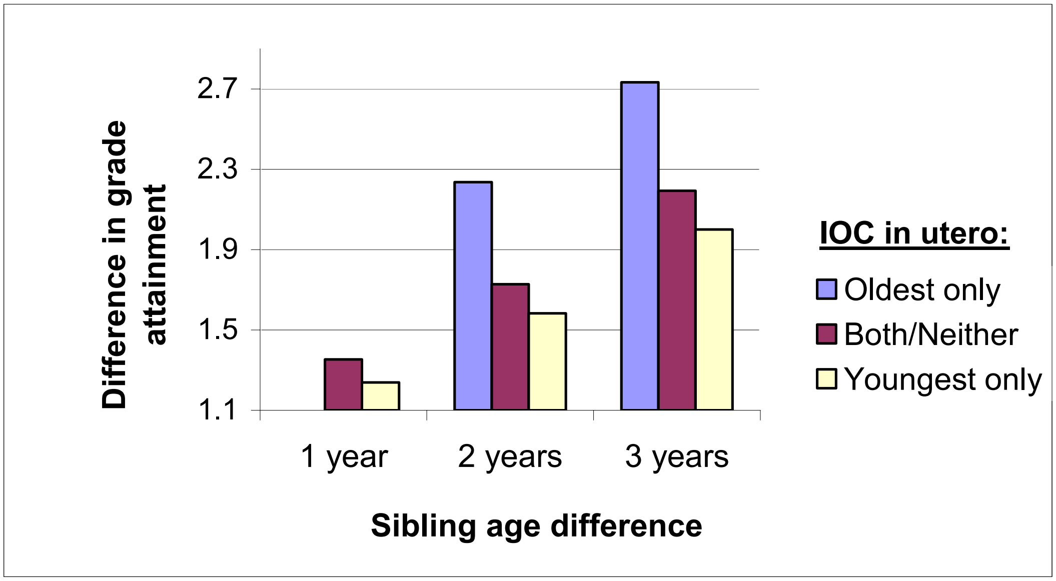


Figure 4: Grade Progression by Gender and IOC



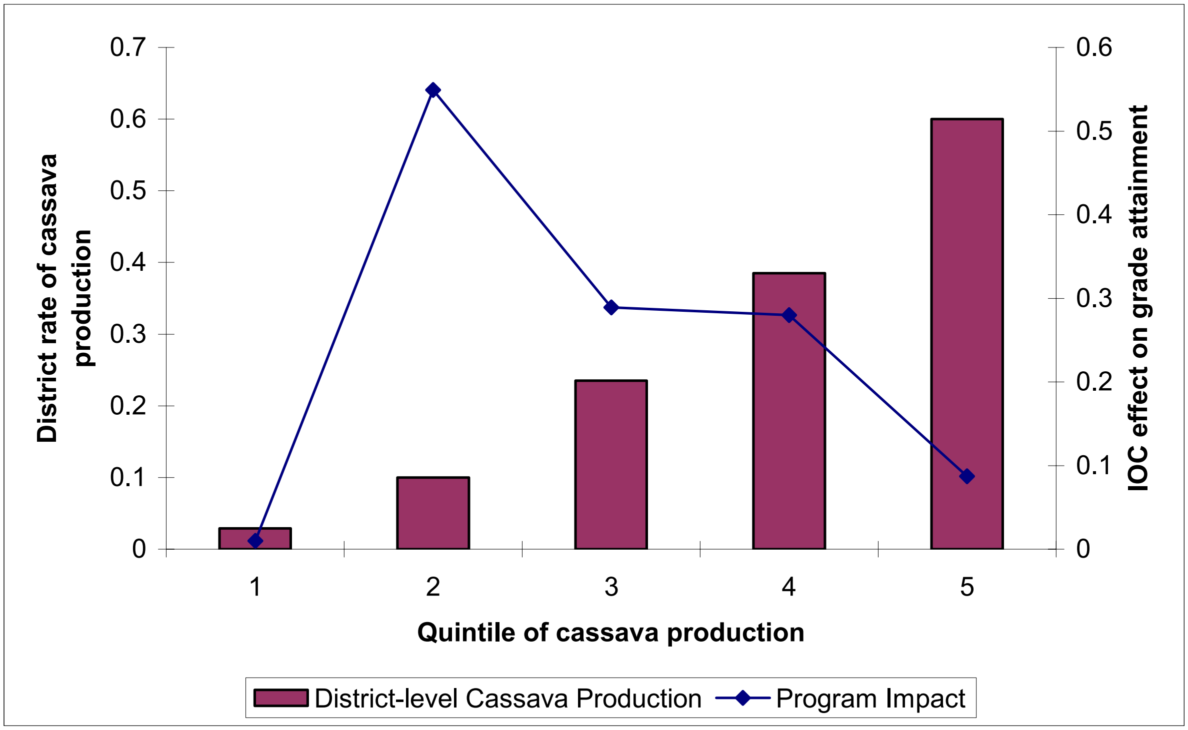
Notes: Data from the 2004 Tanzania Demographic Health Survey. 3675 observations, including all children in project districts between the ages of 10 and 14 that are children of respondents to the birth history module and who reside in the household. X-axis is child age and y-axis is completed years of 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: Sibling differences in schooling by age difference and IOC



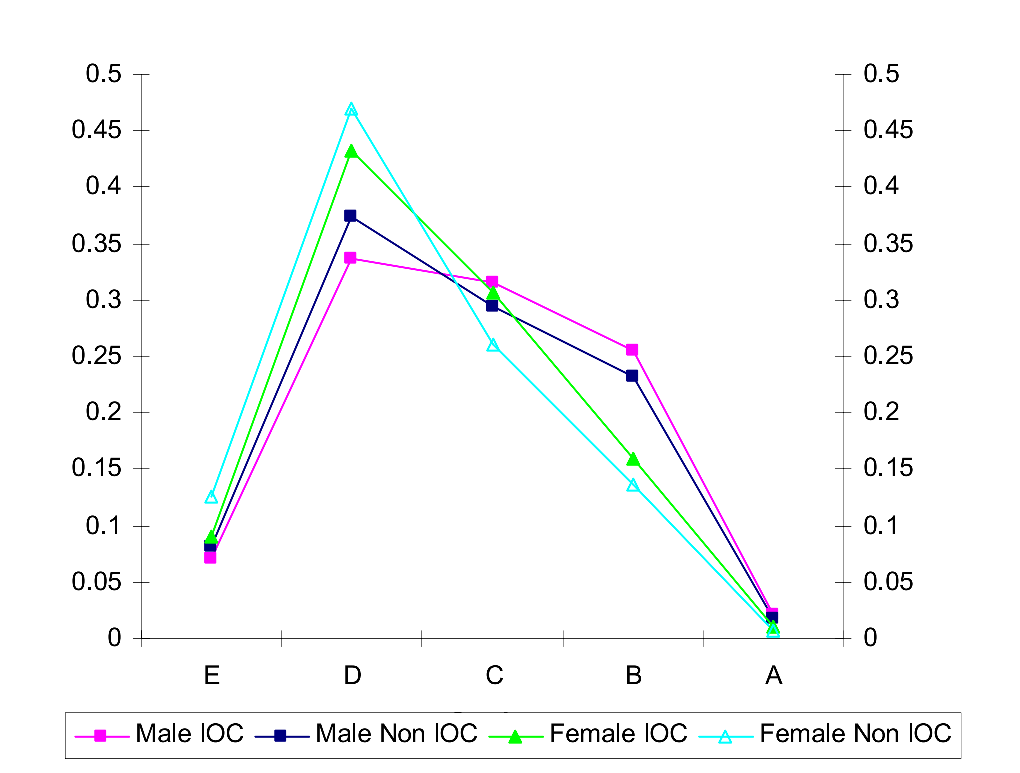
Notes: Data from the 2000 Tanzania Household Budget Survey. 576 observations comprise all sibling pairs in 25 pre-1994 project districts in which both children are between the ages of 10 and 13 and are children or grandchildren of the household head or spouse. Mother-child linkages are not perfectly recorded, so children may not be true siblings. Because month of birth is unobservable, there is no observable variation in likelihood of IOC in utero for siblings of 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 of residence 1 or 2 years prior to the birth year of each child. Since IOC prevents 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.

Figure 6: 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 seperately 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 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

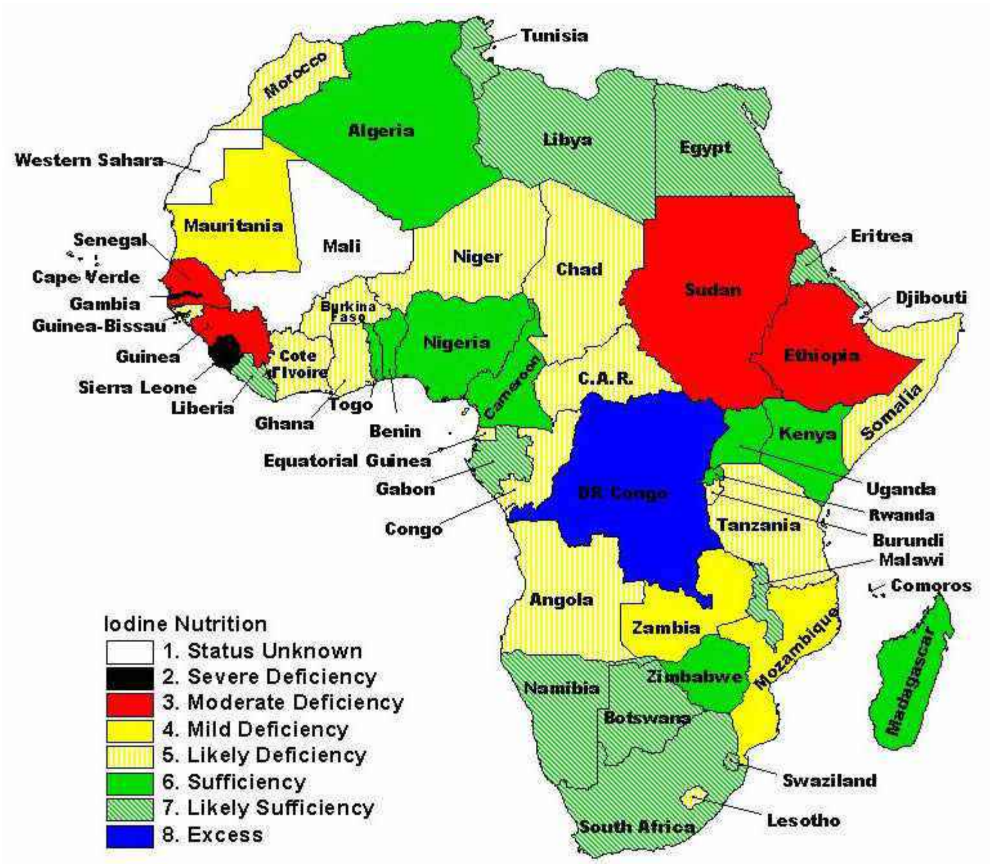


Figure 9: Fraction of households consuming iodized salt, 2000-2005

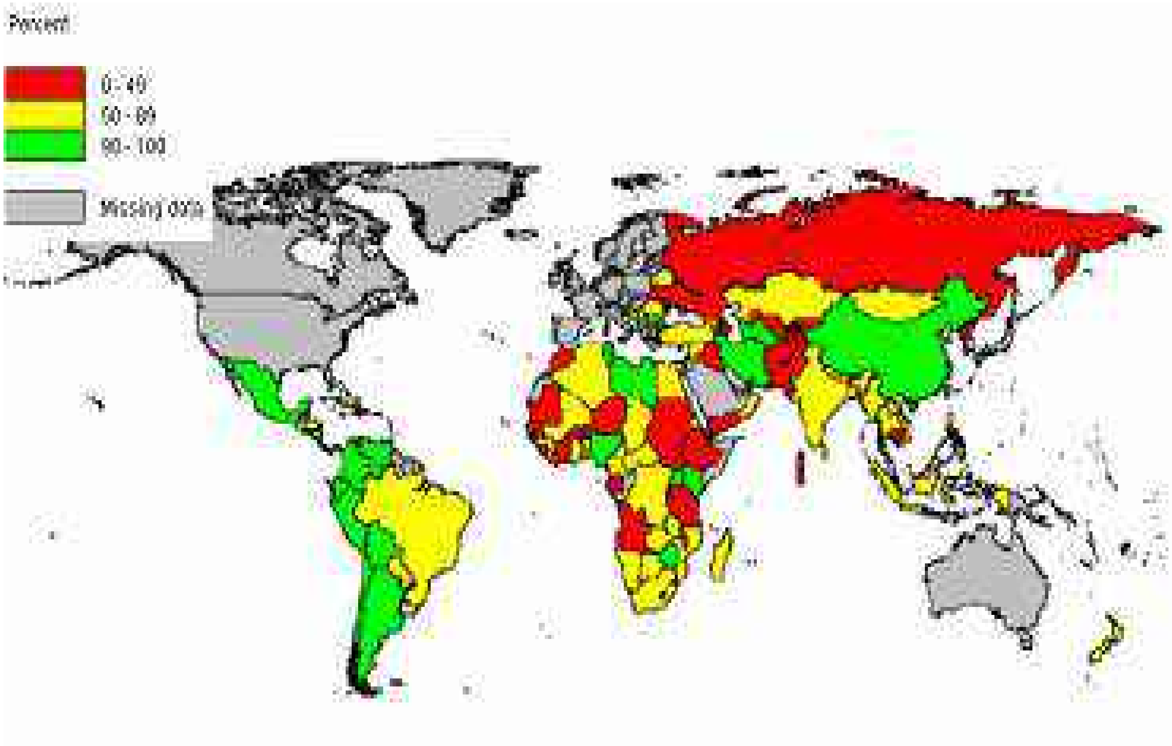


Figure 10: TGR pre-1995 and % of Households Consuming Iodized Salt, 2000-2005, by African Country

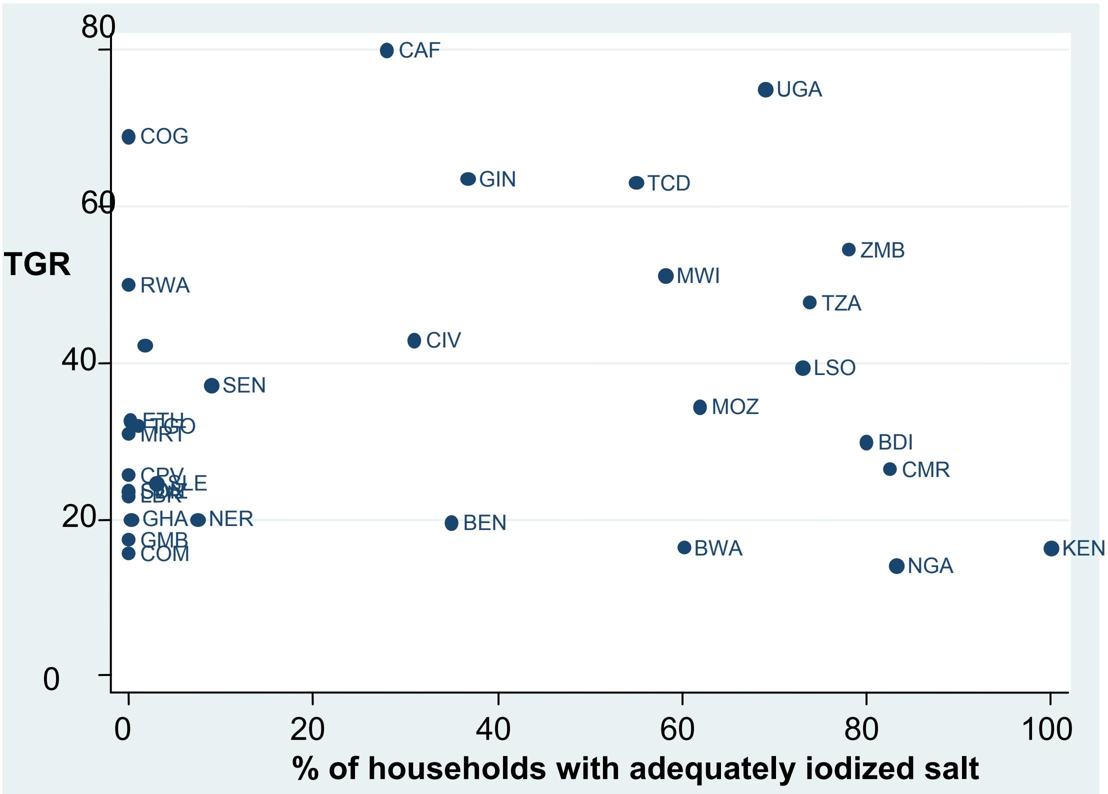


Table 1: Summary of Timing and Coverage of Intervention Across Districts

	Region	District	Year of Intervention (Coverage - %)* 1	2	3	4	5	Average Frequency (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.

Table 2: Summary Statistics by Timing of Intervention Across Districts

	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
Mudd 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 in itiated between 1985 and 1995 in 27 districts of the country.

Table 3: Grade Attainment and IOC Supplementation in Utero

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
				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)$_{3\le t<5}$	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)$_{3\le t<5}$ * 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 targetted 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 hosuehold. 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 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≤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 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 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)* $_{3\leq t<4}$ 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 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 also control for binary indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%

Table 5: 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	0.99	0.99
	(0.160)**	(0.159)**
Age difference = 3 years	1.333	1.333
	(0.197)**	(0.198)**
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 targetted 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 sample 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 yolder not treated was selected second, otherwise two siblings chosen at random.

Table 6: 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 targetted 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 ni 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 7: 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	*59,473*	*17,889*	*17,943*	*113,932*	*57,613*	*56,319*

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 targetted 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 control for sex-specific birth order and household or district fixed effects.

Table 8: Mortality and IOC Supplementation in Utero

	Fertility (Birth in month m)			*Stillbirth* (Died within 30 days of birth)			*Infant Mortality* (Died within 1 year of birth)			*Child Mortality* (Died between ages 1 and 15)		
	(10)	(11)	(12)	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	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)$_{3\leq t<5}$ * 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)$_{3\leq t<5}$ * 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)* $_{3\leq t<4}$ 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 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 ro 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 ni 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: Schooling attainment and cognitive ability

	(1)	(2)	(3)	(4)	(5)	(6)
	Years of completed education			*Currently enrolled in school*		
Male	-0.092	-0.297	-0.041	0.005	0.002	-0.003
	[0.331]	[0.280]	[0.311]	[0.058]	[0.049]	[0.053]
Sabot's score	2.371			-0.071		
	[0.529]**			[0.092]		
Male* Sabot's score	-0.602			-0.019		
	[0.771]			[0.135]		
Sabot's math score		1.754			-0.001	
		[0.455]**			[0.078]	
Male* Sabot's math score		-0.125			-0.009	
		[0.686]			[0.119]	
Sabot's reading score			1.458			-0.122
			[0.428]**			[0.074]
Male* Sabot's reading score			-0.635			-0.002
			[0.607]			[0.104]
Observations	*398*	*398*	*398*	*404*	*404*	*404*

Notes: Data from 1993 Tanzanian Living Standards Measurement Survey, in which 404 household members between the ages of 7 and 14 were randomly chosen to take the Sabot's test of cognitive ability in a one-time survey supplement. Test is composed of 10 math and 6 reading multiple-choice questions. "Sabot's math score" and "Sabot's reading score" are fraction of questions on each section answered correctly; "Sabot's score" is fraction of questions on all sections answered correctly. Columns present coefficient estimates from seperate OLS regressions with year of birth fixed effects. Outcome in columns 1-3 is years of completed schooling, outcome in columns 4-6 is binary indicator of whether respondent is enrolled in school at time of survey. Schooling attainment data is missing for 6 observations. Standard errors in brackets account for sample strata and clusters. * denotes significance at 5%; ** denotes significance at 1%.