# 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, non-reversible effects on cognitive development. This research evaluates the impact of reductions in fetal IDD on child schooling attainment in Tanzania that resulted from an intensive distribution of iodized oil capsules (IOC) in several districts of the country between 1986 and 1994. 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 13. Our findings suggest that reducing fetal IDD has significant benefits for child cognition: Children who receive iodine in utero attain an average of 0.36 years of education above siblings and older and younger children in their district. Furthermore, the effects appear to be substantially larger for girls, consistent with new evidence from laboratory studies in animals indicating greater cognitive sensitivity of the female fetus to in utero iodine deprivation, including sex-specific responses to maternal thyroid hormone restriction on genetic expression of neural thyroid hormone receptors. The results are consistent across household and district fixed effects models and patterns of variation in estimated effects are consistent with predictions regarding the relative vulnerability of specific subpopulations to fetal IDD. Cross-country regression estimates 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 IDD in explaining global patterns of gender differences in schooling.

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## 1 Introduction

There is an unresolved debate in the economic growth and development literature regarding the role of geographic variation in health environment on long-run economic outcomes. A number of recent cross-country studies provide evidence that ecological conditions related to health environment, such as malaria transmission rates, have a direct effect on economic growth (Sachs, 1997; Sachs and Gallup, 1998, 2002). One critical aspect of health environment that has received little attention in the literature is the concentration of trace elements in soil and rock, which differs widely across settings as a result of geographic variation in geological time (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 from laboratory studies in animals to have significant, irreversible effects on brain development (Cao et al., 1994; Hetzel and Mano, 1989; Pharoah and Connolly, 1987).[1]

This research looks for evidence of the influence of health environment on development by examining the effect on child schooling of reductions in iodine deficiency that resulted from an intensive and repeated distribution of supplements in several districts of Tanzania between 1986 and 1994. 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 exhibited higher rates of grade progression ten to fifteen years later. We exploit variation in the timing of coverage across districts using household and district fixed effects models to compare children in utero during the program to slightly older and slightly younger cohorts within the district. Of particular interest is the possible role of iodine deficiency in explaining gender differences in schooling outcomes in light of recent scientific evidence of biological differences between males and females in iodine sensitivity in utero.

If 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. More importantly, given that an estimated one billion people globally are at risk of brain damage from iodine deficiency disorder (IDD) worldwide, its influence on cognition may be a key determinant of cross-country differences in economic growth.[2] Although dietary patterns vary geographically with respect to a variety of micronutrients important for human development, iodine availability is potentially a key "missing link" in the growth puzzle for two reasons. First, iodine is unique among micronutrients in its effect on brain development and cognition, and small differences in average IQ at the group level could have large effects on social and economic outcomes. Second,

[1] Epidemiological criteria for assessing sufficient iodine intake is 100 μ/I or above.
[2] The World Health Organization has labeled IDD "the most common cause of preventable mental retardation (WHO, 1992)."

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 no direct influence on plant growth and therefore little correlation with local food availability in a population. Hence, while other human micronutrient deficiencies are likely to be resolved with economic growth by way of rising caloric intake, iodine deficiency is more likely to exert a persistent influence on economic outcomes.

In addition to providing evidence of a direct link between geography and development, our analysis contributes to the growing body of micro-level studies on the effects of malnutrition on schooling and labor outcomes.[3] Assessing the importance of physiological determinants of schooling informs a fundamental debate in the literature on barriers to schooling attainment in developing countries surrounding the importance of supply-driven explanations for low levels of human capital investment relative to differences across settings in returns to education. Schooling responses to reductions in IDD provide further evidence on the degree to which patterns of grade attainment are also influenced by biological differences in the cognitive costs of schooling. Unlike nutrition shortages during childhood or adulthood, fetal IDD is believed to permanently limit intellectual ability, so its impact is likely to be particularly acute and persistent. Furthermore, if girls are particularly susceptible to IDD in utero, geography may contribute directly to gender disparities in schooling outcomes by way of sex differences in rates of learning disability that result from widespread fetal IDD. This is a particularly compelling explanation for gender differences in schooling patterns in Tanzania, where lower female attainment is almost entirely accounted for by the abysmal 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 these changes are only now reaching school age in the most affected regions, 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

[3] The relationship between macro-nutrients (energy and protein intake) and education has been examined through subsidized school meal programs in Western Kenya and child nutritional supplements in Guatemala, which were associated with increased school participation and test scores (Vermeersch, 2003; Behrman et al, 2003). While little attention has been paid to the link between specific micronutrients and schooling, a number of studies have examined the benefits of micronutrient supplements on health and labor productivity. For instance, iron supplementation was found to increase labor force participation and work productivity in Indonesia (Thomas et al, 2003; Basta et al, 1979; Husain et al, 1981), and vitamin A deficiency has been associated with

will alter the global pattern of schooling attainment in the near future.

Although a number of countries undertook intensive iodine supplementation programs during the 1990s, there are two important advantages to studying the case of Tanzania. First, Tanzania was one of the largest and most intensive iodine supplementation programs, ultimately reaching approximately 25% of the population for an average of 4.2 years. As a result, an estimated 1.9 million babies born during and immediately after the program were fully 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 cognitive capacity as evidenced by its effect on schooling attainment: Children who are likely to be protected from iodine deficiency during their first trimester in utero attain an average of 0.36 years of education above siblings and older and younger children in their district. This result supports the common claim that the first three months of fetal growth are a critical period for cognitive development. Furthermore, the effects appear to be substantially larger for girls, indicating a potentially important role of micronutrient deficiencies in explaining gender differences in schooling attainment in many parts of the developing world. The finding is consistent with new evidence from laboratory studies in animals which find sex-specific responses to maternal thyroid hormone restriction indicating greater sensitivity of the female fetus to in utero iodine deprivation on cognition. The pattern of results is similar in household and district fixed effects models and consistent across datasets and points in time. Finally, observed variation in estimated effects matches predictions regarding the relative vulnerability of specific subpopulations to fetal IDD based on local diet.

On a global level, these findings suggest that schooling attainment may rise considerably over the next several decades in several countries that achieved reductions in IDD through USI. Results from cross-country regressions of school participation on baseline IDD and fraction of population consuming adequately iodized salt suggests that early salt iodization has already exerted a positive effect on female primary schooling levels. Based on our micro-level estimates from Tanzania, we calculate that the average increase in schooling attainment in Central and Southern Africa attributable to USI could be as large as 7.2% of baseline average schooling levels.

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increased mortality and morbidity from respiratory and gastrointestinal disease and blindness (Sommer et al., 1986, 1981; West et al., 1995; Glasizou et al, 1993; Beaton et al, 1992).

[4] Estimation is based on 1988-1994 population and (crude) birth rate data from the World Bank's World Development Indicators.

A remaining policy question is the value of devoting further resources necessary to fully eradicate IDD. Although USI is arguably the most successful micronutrient intervention in world history, legal regulations on salt production are rarely sufficient to guarantee dietary change among rural populations that consume mainly subsistence food products. In several African countries less than 30% of households 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 interventions.[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; hence, dietary iodine availability is determined primarily by soil composition and amount of seafood consumed. Since iodine deposits are concentrated in deep soil layers, well water is also an important source (Hetzel, 1989). 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. For this reason, pre-Cambrian rock, which covers a large ring of Central Africa including western Tanzania, is associated with particularly low concentrations of iodine in soil and ground water.

Iodine is needed for the biosynthesis of thyroid hormones, which are essential for physical and mental development. Perhaps most importantly, in utero development of the central nervous system required for intellectual functioning depends on an adequate supply of thyroid hormones, which influence the density of neural networks established in the developing brain (Lamberg, 1991).[6] Cretinism is the most severe manifestation of cognitive damage from insufficient maternal and fetal thyroid hormone. 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; however the existing evidence from human studies suggests that non-cognitive outcomes occur only under extreme deprivation and that damage from IDD is overwhelmingly cognitive (Zimmerman, 2005; Hetzel, 1983). Furthermore, animal studies indicate the brain is particularly sensitive to iodine deficiency during its formation in early

[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 begins at 50 mcg for infants under 12 months of age and rises to a level of 150 mcg for adults and 200 mcg for pregnant and lactating women (WHO, 1996).

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).

Although endemic goiter and cretinism associated with iodine deficiency have been depicted for centuries, only during the past decade has IDD been widely recognized as a leading cause of intellectual impairment worldwide (Merke, 1984; Delange, 2000; Haddow, 1999). Though cretinism is relatively rare, severely affected populations may have rates as high as 3 to 15 percent, imposing a major social and economic burden on the community (Boyages et al., 1988; Halpern et al., 1991; Pandav et al., 1982). More importantly, the effect of IDD on mental development is no longer believed to lead directly to severe mental retardation. Recent evidence from laboratory studies in animals indicates a continuous process by which brain development is sensitive to minor adjustments in thyroid hormone supply (Lavado-Autric R, 2003; Sundqvist et al, 1998; Dugbarty, 1998; Pop et al, 1999). Based on this evidence, 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 IDD in humans, there is suggestive evidence from community-based assessments of iodine intervention trials that supplementation can improve performance on cognitive tests (Bleichrodt et al., 1994). 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. The difference corresponded to a 10-15 point improvement in IQ relative to children in untreated communities (Shrethsa, 1994). To our knowledge, the long-term impact of increased iodine intake during pregnancy on children's human capital has not been measured in any setting.

**2.3 Gender Differences in Iodine Deficiency**

Evidence from multiple sources indicates gender differences in the importance of iodine supplementation for growth and development. In the study mentioned above, cognitive improvements were only found among girls, although the finding is merely suggestive given the limited number of subjects. Similarly, practitioners have noted that adolescent IDD is systematically higher among females relative to males (Allen et al, 2001). A fairly consistent global pattern is that both rates of goiter and average goiter severity among sufferers are higher for females (Simon, 1990). One central limitation of observational studies is their inability to attribute gender differences in IDD to physiological sex differences in iodine sensitivity as opposed to sex-specific dietary patterns.

More conclusive evidence of biologically-driven gender differences in iodine sensitivity comes from recent laboratory experiments of maternal thyroid deficiency in animals. 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 such studies lend strong support to the hypothesis of sex-specific sensitivity to iodine deficiency in utero. A 2000 study by Friedhoff et al. found that the effect of artificially restricting maternal thyroid hormone transferred in utero on fetal neurodevelopment and behavioral outcomes was significantly larger in female 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 is not fully understood, the finding is hypothesized to be related to elevated male androgen levels at the height of neural TR expression.

## 3 Setting

### 3.1 Iodine deficiency in Tanzania

Our study examines the long-run impact of an intensive iodized oil supplementation program that took place in Tanzania between 1986 and 1994. 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 have iodine deficiency disorders, including 3% with severe IDD and 22% with moderate symptoms. In severely affected regions, 13% of children under five and 52% of pregnant and lactating women showed manifestations of iodine deficiency prior to the intervention (van der Haar et al, 1998).

### 3.2 Schooling in Tanzania

The Tanzanian formal education system involves seven years of primary education, four years of junior secondary (ordinary level), and two years of senior secondary (advanced level). 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. In 2001, gross enrollment in primary school was 85% but only 7% at the secondary school level, largely due to an insufficient supply of

secondary schools. According to data from 2001, one quarter of rural households reported being over 20 kilometers from a secondary school (THBS, 2001). Meanwhile, distance to primary schools is now an issue only for a minority of rural households, 8 percent of which reported the nearest primary school to be more than 6 kilometers away. Throughout the country, gross enrolment ratios are higher than net enrolment ratios because many over-age children are present in primary schools due to beginning schooling late. In rural areas, slightly over 50 percent of children are studying by age nine.

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. Girls are less likely than boys to be in school beginning at age thirteen, and the difference increases steadily thereafter. Gender differences in secondary enrollment are almost entirely accounted for by differences in PSLE pass rates for boys and girls. Alarmingly, 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 substantial cost and no benefit other than high school admission to taking the test, this fact alone suggests that traditional social roles and explicit parental preferences for male over female secondary schooling are not fully responsible for gender differences in education outcomes.

### 3.3 Iodized Oil Capsule (IOC) Distribution in Tanzania

Unlike many afflicted countries, Tanzania was targeted for iodine supplementation relatively early. In 1986, a massive supplementation intervention was scheduled to begin in 25 of 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 interventions for combating IDD in areas with high rates of TGR and low coverage of iodized salt on account of the immediacy of health improvements and duration of coverage (Delange, 1998). In selected districts, all women of child-bearing age were targeted to receive 200 mg and 380 mg capsules once every two years, the expected duration of protection.[7] From 1986–1994, approximately five million women and children received at least one dose of iodine through the IOC program.

Districts were chosen based on 1984 field measurements of TGR, or the number of children

[7] 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).

with visible goiters. The minimum TGR for participation in the IOC program was 10%, which resulted in 25 treatment districts encompassing 25% of the country's population (Peterson, 2000).[8] As shown on the map 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.

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 districts. 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%.[9] 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 too frequently (Peterson, 2000).

Although the long-term impacts of the program have 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 between 51-56 cents), and a handful of initial studies indicating that visible and total goiter rates (VGR, TGR) had decreased among individuals who received supplements. A 1991 evaluation in three of the intervention districts found that VGR had decreased by over 50% and TGR by over 25% (Peterson, 2000). Likewise, 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 who were protected from IDD during pregnancy is likely to be even higher.

## 3 Empirical Analysis

### 3.1 Data

We examine the program effect on children born to mothers who were targeted for IOC during pregnancy using data from the 2000 Tanzanian Household Budget Survey (THBS) and the 2004 Tanzanian Demographic and Health Survey (TDHS). To these data, we append the district-level information from Table 1 on timing of IOC distribution across intervention districts. The THBS is a

[8] Two districts – Bukoba Rural, Kagera, and Mbinga, Ruvuma – were added to the program later, and are excluded from this analysis.

[9] The average coverage rate among districts and years included in our analysis sample was 68%.

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 Tanzanian Demographic and Health Survey (TDHS) covers a total of a total of 4,987 households, 1,034 of which reside in intervention areas. Both surveys collect individual information on school enrollment, grade attainment, and health status 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, while the TDHS focuses heavily on fertility and infant health.

For the empirical analysis, we restrict both analysis samples to all children or grandchildren of the household head who are between ages 10 and 13 in 2000 or between 10 and 14 in 2004. 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, school enrollment for both boys and girls peaks at age 10 and falls monotonically thereafter. Since no data are available from either survey on age of school entrance, restricting the sample to ages by which most children can be assumed to have entered school 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 (the 1986 program first affected those born in 1987). In 2004, the upper age limit is driven by the fact that children in the sample select out of households strongly beginning at age 15 the reasons for which are likely to be different for boys and girls.[10] Because schooling data 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 2276 children in 1696 households living in the 25 intervention districts that began IOC by 1992. Within-household estimates reduce this sample to 1080 kids in the 499 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 336 boys in 159 households and 307 girls in 148 households. Among children in our sample, 89 percent are enrolled in Standards I to VII (primary school) and 11 per cent are not studying (with the exception of three 13-year-olds who report enrollment in secondary school). The DHS analysis sample contains 3675 children ages 10-15 in 2523 households across the country, 515 of which reside in intervention districts. Within-household estimates reduce this sample to 1699 kids in 799 households with more than one family member aged 10 to 14, and sex-specific estimates reduce the sample to 643 boys in 307 households and 535 girls in

[10] Comparing birth history data to the household roster for children of the respondent, approximately 19% of 15-year-olds are no longer part of the household roster and therefore have no education data, whereas the fraction of children missing from the roster below age 15 is ~6%.

259 households. In the 2004 sample, 85.4% are enrolled in Standards I to VII (primary school), 4.3% are enrolled in Standards VIII to X (secondary school) and 9.76% are not studying.

Although the DHS sample is considerably smaller than the THBS, it has four principal advantages. First, month of birth is available for approximately 80% of children in the sample, allowing us to construct a more precise indicator of IOC treatment, described in the proceeding section.[11] 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. In addition, by 2004 program children have had slightly more opportunity to transition from primary to secondary school, enabling us to better gauge whether the benefits of in utero IOC extend to secondary school. However, since it is necessary to restrict the sample to children under 15, the ability to observe secondary school transition remains limited: The modal age for entering secondary school is 16; among children in the DHS sample enrolled in the first year of secondary school, 9% are 12 or 13, 23% are 14, 26% are 15, 28% are 16, and 14% are 17. Finally, information from the TDHS on birth histories allow us to explore the possibility that IOC influenced fertility outcomes and thereby selection into the sub-samples of treated and untreated kids, which could confound interpretation of our results.

### 3.2 Definition of Program Participation

To analyze the impact of IOC distribution, we defined an indicator of program participation equal to one if an individual was born one to three years after an intervention year *t*. The period of treatment was based on the likelihood that the mother of a child was protected from IDD at some point during her first trimester of pregnancy, given the IOC dosage of 380 mg. According to previous studies, this level of iodine, which is stored in the adipose tissue, should have a prophylactic effect for a minimum of 24 months.[12] First trimester was chosen based on laboratory studies indicating that maternal hypothyroxinemia increases the risk of neuro-developmental deficits of the fetus only before mid-gestation, a period during which the mother is the only source of thyroid hormone (Cao et al.,

[11] Month of birth is missing for 1,313 (~20%) children of the head between ages 9 and 17 either because their mother did not live in the household (34 cases) or because their mothers were not included in the individual survey (740 cases) that collected birth histories.

[12] In a longitudinal study carried out for two years in the Darfur region, western Sudan, researchers concluded that a single 400 mg oral dose of iodized oil is effective in the correction of iodine deficiency, reducing the goiter size and preventing the recurrence of goiter for at least two years (Eltom et al., 1985).

1994a; Hetzel & Mano, 1989; Pharoah & Connolly, 1987).[13] Since brain development of the fetus takes place during the first month of pregnancy, it is believed that most of the consequences become permanent by the second trimester. This view is consistent with a wider body of scientific thought regarding the importance of micronutrients during the "critical period" of the first three months of pregnancy (Barker et al., 1989; Painter et al., 2005).

The likelihood that the mother of a child born *t* months after a program year *p* was protected from IDD at any point during the first trimester of pregnancy is equal to the probability that the mother received IOC on or before *t-7* (in time to protect the child prior to end of the first trimester given 9-month gestation) multiplied by the probability that sufficient stores of maternal iodine were remaining at *t-9* to protect the child during this critical period. The likelihood that mothers of kids born 0 to 1 year after *p* received IOC by the end of their first trimester depends on the first probability, while the probability that kids born 2 to 4 years after the program were covered depends on the second. Since no data are available on month of IOC distribution, the first probability calculation requires an assumption regarding the distribution of iodine supplementation over the year which we assume to be uniform. Hence, children born *t* months after the start of the program are protected with probability equal to *(9-t)/12* when *t* is greater than 8 and less than or equal to 24.

The second probability calculation, which pertains to kids born 2-4 years after the program (for whom the first probability is always equal to 1), requires an assumption regarding the rate at which iodine stores from 380 mg supplements are depleted from the body. Unfortunately there is little information on which to base this assumption given large variance across populations and individuals in the speed of iodine depletion and few studies that follow subjects for more than a year.[14] In general, iodine stored in fatty tissue appears to be depleted hyperbolically with the vast majority of urinary extraction occurring in the first week and then tapering off gradually.[15] Since only trace amounts are needed for adequate production of thyroid hormone, the rate of depletion is only relevant after the point at which iodine stores begin to reach ineffective levels in the treated population. Since baseline iodine deficiency varies across treated individuals, the fraction of treated who are adequately covered will presumably decline after this unobservable point at an unknown but presumably gradual and

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

[14] 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).

[15] According to Wei Jun and Li Jianqun (1985) there is generally an 85% loss of iodine within 72 hours of iodized oil taken orally, 30% in two weeks for intramuscular injection and 96% in 72 hours for iodized salt.

decreasing rate.[16] Four studies of the approximate half-lives of urinary iodine excretion after oral oil administration to iodine-deficient populations reveal half lives at time 0 of between 3 and 7.5 months (Wolff, 2001). The correct specification is important for the analysis: Assuming a simple hyperbolic function and a half-life of three months implies that no trace amounts of iodine will remain in the majority of people soon after 24 months, whereas a six-month half-life implies that adequate iodine will remain in the body for almost three years.

Given these limitations, we assume that adequate iodine remains in the body for all treated individuals until 24 months, which implies a half-life of 4.5 months, after which point iodine levels continue to drop hyperbolically for an additional 12 months until they reach ineffective levels to protect anyone in the population (>45mg). The exact probabilities for each birth month are given in Appendix A. 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 interacting month of birth with an indicator of being born 3-4 years after supplementation, the period over which depletion assumptions apply.

For the analysis sample without month of birth data, we calculate the birth year-specific likelihoods of receiving adequate coverage for children born *x* years after IOC by averaging the monthly probabilities. Under two different assumptions regarding the fraction of women with sufficient iodine stores after month 24, these likelihoods are the following:[17]

| *Birth year - program year (x):* | -1 | 0 | 1 | 2 | 3 | 4 |
|---|---|---|---|---|---|---|
| Likelihood of IDD protection in trimester 1, assuming gradual depletion months 25-36: | 0 | 0.10 | 0.85 | 0.99 | 0.62 | 0.05 |
| Likelihood of IDD protection in trimester 1, assuming gradual depletion months 25-34: | 0 | 0.10 | 0.85 | 0.99 | 0.507 | 0.014 |

Based on these probabilities, we treat only those individuals born one to three years after IOC distribution as protected from IDD in utero since the likelihood of IOC for a child born up to three years after the program is greater than 50%. In contrast, we classify children born in the same year as the program as untreated since during year 0 only 10% of children are expected to receive *any* coverage, and they are more likely to receive it in month 3 than in month 1 of pregnancy. As

[16] Hyperbolic depletion implies a fast initial rate of depletion that slows quickly. We assume based on the recommended daily allowance for pregnant women (2 mg) that 60 mg of iodine stores are sufficient to ensure at least 1 full month of coverage in the population.

[17] Likelihoods were calculated using the following formula: The second probability assumed sufficient stores of maternal iodine for a full 24 months, and a gradually decreasing probability of sufficient stores during months 25-36.

robustness checks, we run all regressions using the continuous measure of program participation rather than the binary indicator, and adjusting the above probabilities to account for seasonality in births observed in non-intervention districts in the TDHS, which has little effect on the probabilities.

Because iodine dosage administered to pregnant women varied according to whether they were above age 22 (as a precaution against supplement-induced fetal hyperthyroidism), we also adjust the binary indicator of treatment according to mother's age at birth. Since children born to mothers who were younger than 23 during pregnancy were given half the level of iodine, we assume that these children experienced half the protective effect as children with older mothers. This adjustment is complicated by the fact that household members can not be perfectly linked across generations. Based on age and relationship to household head, approximately 80% of children in our sample can be matched to mothers with relative certainty.[18] In the results presented here, children that cannot be matched are assumed to have mothers that were older than 23 at birth, however the results are robust to making the opposite assumption and to excluding unmatched observations from the analysis. Given high reported rates of orphanhood in Tanzania, some fraction of mother-child pairs are 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.

### 3.3 Estimation Strategy

Using the program indicator described above we identify the effect of IDD on child schooling. The primary outcome of interest is grade attainment, or years of completed schooling. 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 fewer households with private sources of drinking water and solid floors, have greater distance between households and secondary schools, and have lower

[18] We matched mothers and kids 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 observations (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.

consumption of fish, a rich source of iodine. Such differences make comparisons between participating and non-participating districts difficult.

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 occurrence of 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 durable goods is significantly higher in late districts, while the average number of meals consumed per day and fish consumed per week are relatively constant across program start dates. Nonetheless, the general patterns suggest that the districts in which IOC began early were better off than late districts, consistent with the most common source of delay being poorly organized distribution networks. Hence, program effect estimates based on comparisons across participating districts only are also likely to be biased.

On account of these patterns, our regression estimates are restricted to within-district comparisons. In particular, we compare children born one to three years after the intervention to those born in the same district immediately prior to or during the intervention with the following fixed-effects regression:

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

Here $T_i$ is the liklihood child $i$ in district $f$ was protected from IDD during his or her first trimester, $A$ is a vector of birth year dummies, and $X$ includes the following set of binary controls: gender, birth order, sex-specific birth order, and whether mother was under age 23 when child was born. Since the fetal effects of IDD have been hypothesized to be stronger for females, we also run the above regression separately by gender. Note that the gender-specific regressions are necessarily run on the subset of families with at least two children of the same gender, which gives rise to potential selection issues relevant for comparison across estimates. We also estimate the effect of IOC by comparing children born to the same mother before and after the program among the subset of households with more than one child in the relevant age range. 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. We estimate the above model of grade attainment using both the samples. The only difference in regression specification across the two samples is the use of birth month information in the TDHS estimates, which is used to refine the definition of treatment and also added to the set of controls to account for the independent effect of small differences in age on school entrance or progression.

Since within districts and households treatment is determined entirely by age, in the above

equation $\beta_1$ 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 ($\mu_f$). If cohort differences in treatment are positively correlated with other trends that effect 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 are correlated with IOC distribution. Furthermore, unless such trends systematically lagged IOC by several years, they would have had to impact IOC children while in utero or early childhood *and* have had a lasting effect on their outcomes relative to children in the district who are slightly older or slightly younger. This reduces the set of potential confounders to other improvements in fetal or infant health environment that are correlated with IOC distribution. In other words, the principal concern in the fixed effect estimates is that delays or gaps in program coverage coincided with reductions in fetal health inputs other than iodine that had a lasting effect on schooling. For instance, if the timing of program cycles were driven by intermittent declines in the quality of district health services, children in utero during program gaps may have experienced other deficiencies in health inputs relative to those born immediately before or after, which could lead to permanently poorer health – and possibly schooling – among children who did not benefit from IOC that is independent of reductions in IDD. Similarly, if program timing was driven by district-level income shocks, children in utero during the program may have received better nutrition at critical stages of development.

In Section 4.3 we assess the extent of this problem 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. Although we cannot completely rule out the possibility that non-IOC-related health shocks might have influenced schooling through their effects on cognition rather than physical health, as long as health insults other than IOC that are large enough to impact cognition are also associated with differences in physical health status (unlike IOC), the absence of an observable difference in health status between treated and untreated children is evidence that the treatment effect is driven by IOC.

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 distribution delay gleaned from IOC program reports and administrative records, interviews with past and present program managers, and supervision visits to

selected districts. 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, which was distributed from the government to district health centers between 1986 and 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.[19] In all cases, the start date was ultimately determined by an external rather than an internal force. Hence, although early and late districts are not safely comparable, given the central role of external resource provision in determining distribution timing there is little reason to suspect that variation in program timing was related to income shocks or changes in the quality of health care services within districts.

### 3.5 Heterogeneous Program Effects

To refine our estimate of program impact, our analysis makes use of anticipated variation in the impact of iodine supplementation based on district variation in iodine deficiency. Given that the level of iodine provided through IOC supplements was uniform across districts and rates of program coverage were not explicitly based on level of need, the relationship between baseline IDD rates and program impact is likely to be non-linear. In other words, we anticipate a threshold level of IDD below which rates are too low to observe a significant treatment effect, and a second threshold above which 380 mg of iodine will be insufficient to protect against maternal iodine deficiency due to factors which raise daily requirements for iodine intake. Since all participating districts had high pre-program TGR, the program impact among districts in the lower tercile of the treated population is likely to be larger than it is in districts with the highest baseline rates of IDD.

These predictions are tested by studying variation in program effect by level of consumption of goitrogenous foods. Goitrogens – including cabbage, legumes, chaya leaves, and cassava – are foods that contain cyanogenic glycosides, which impede absorption of iodine by the thyroid gland (Bourdoux et al, 1978). Frequent consumption of such foods is one of the leading causes of IDD, and diets high in natural goitrogens can induce IDD even if the diet is rich in iodine (Gaitan 1990; Thilly 1992). Consistent with this, laboratory evidence suggests that goitrogens play a significant role in

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

influencing biochemical events that are unique to the developing brain (Rao and Lakshmy, 1995).

Cassava is a staple in much of Africa and a large part of the diet in rural Tanzania. According to the 1991 THBS data, which contain detailed information on household food items consumed, cassava (either flour, dried or fresh) was the second most important food product after maize in terms of calories per day, and in 2000 was ranked third after maize and sorghum.[20] As one of the most goitrogenic food products, cassava has the potential to significantly decrease iodine absorption if not properly fermented.[21] 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).[22] Although the need for iodine increases with consumption of goitrogenous foods, so does the rate at which iodine – including that provided by the supplement – is depleted from the body by regular intake of goitrogens. Hence, in addition to serving as a central explanatory factor for baseline rates of IDD in the study population, the cyogenic effect of high cassava consumption may have impaired the effect of IOC in certain districts.

The district rate of cassava production is used to proxy for variation in dietary intake 10 to 15 years prior. To construct this measure, a household was defined as a cassava producer if they reported consuming during the past month any fresh or dried cassava or cassava flour that was produced at home, and districts were classified according to the fraction of households in the district that reported consuming home-produced cassava. Given that households also eat cassava that is not produced at home this clearly underestimates the actual intake of goitrogens. However, it is intended as an indicator of geographic variation in produce availability and therefore a reasonable predictor of dietary differences a decade earlier. Given that diets of non-agricultural households may have changed over the past ten years, this is arguably a preferable proxy of past diet than is current consumption.

In the initial regressions, districts were divided into terciles of cassava production. In high production regions, between 41 and 60% of households consume home-grown cassava; in medium production regions between 11 and 40% of households do so; and in low productions districts fewer than 10% of households consume home-grown cassava. Although sample sizes prevent us from undertaking a detailed study of the amount of goitrogens consumed, the relationship between program impact and diet is studied more closely by testing for non-linearities in program impact across quintiles of cassava production.

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[20] 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.

[21] As explained by Hetzel (2000), "Although a number of other staple foods contain potential goitrogens (e.g. maize, lima beans, sweet potatoes), in contrast to cassava the goitrogens are in the inedible portions of the plants and do not contribute importantly to IDD."

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

## 4 Results

### 4.1 Grade Attainment

Regressions of grade attainment on program participation yield large and significant estimates of the impact of IOC on progression through school. In household fixed-effects regressions from 2000 (Table 3), children who participate in the program are an average of 0.36 years ahead in school relative to their siblings. Over this interval, the gap widens continuously as children age, as illustrated in Figure 2. Since enrollment is virtually unchanged over this interval, the results indicate that fetal IDD influences the rate at which students progress through school. The 2004 estimates of primary school attainment detailed in Table 4 are strikingly consistent with the 2000 estimates. Although the sample members were born in different years and in distinct enumeration areas to the THBS sample, the estimated program effect is nearly identical in magnitude to the Table 3 estimates. In addition, when the correction factor for rate of iodine depletion is added to the regression, it is significant and positive, indicating that coverage falls at a slower rate than assumed.

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 THBS estimates, girls appear to benefit twice as much as boys from IOC in utero, although the difference is only significant at the 10% level in the district-level fixed effects regressions. In the 2004 estimates (Table 4), we also present the regression including the interaction between program year and age. The gender difference is particularly stark – and statistically significant – when the program effect is allowed to vary by age, possibly reflecting "catch-up" on the part of boys as girls begin to drop out of school.

One possible concern is that, because we are making inferences about program effect based on cohort differences, our results may simply be picking up 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 with the intended 2-year gap, 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 which only one individual was treated, the older sibling was treated in 23% of cases.[23]

Figure 3 shows the average difference in grade attainment across all sibling pairs in the sample, for three categories of sibling pairs: (1) those in which both either did or did not benefit from

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

IOC; (2) those in which the older but not the younger sibling benefited from IOC; and (3) those in which the younger but not the older sibling benefited from IOC. Comparison across these groups reveals that the program effect is symmetric across the latter two cases: When an older but not a younger sibling is protected from IDD in utero, the difference in schooling attainment widens, and when the younger but not the 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 5).

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, although it is unclear whether coverage was higher among specific target groups.[24] If the rate applies equally to pregnant women, total coverage implies an average program effect of 0.53 years. However, for two 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 might 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).

The estimates of grade attainment also generally underestimate the effect of the program since schooling outcomes are right-censored. Furthermore, given that primary school enrollment levels reached 94% by 2000, it is possible that schooling outcomes are far 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 could disappear over time. Unfortunately, the THBS data are too early to enable an examination of secondary school outcomes. 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. The 2004 TDHS estimates provide a limited glimpse of the possible effect of IOC on secondary schooling. In Appendix Table B, the

likelihood of transitioning to secondary school is positively correlated with IOC for both genders, and the point estimate is suggestively though not significantly larger for girls. However, given that so few sample members have entered secondary school, these estimates should be interpreted with caution. This issue is explored further in section 5.1 with district data on PSLE pass rates.

Estimates in Table 6 of differences in the program effect across levels of cassava consumption are consistent with the predictions. Dividing districts in the sample according to cassava production reveals an inverted u-shaped relationship between amount of goitrogens consumed and impact of IOC on fetal development: In areas with highly goitrogenous diets the program appears to have had little effect, suggesting that maternal iodine levels were depleted by intake of cassava. Meanwhile, in areas with relatively little cassava in the diet, the program effect is also small, which presumably reflects the fact that these were the districts with the lowest levels of TGR at the start of the program. Figure 4 splits the sample even further into five categories of cassava production. Results from regressions on the separate sub-samples indicate that only the extreme outliers were unaffected by the intervention. Most striking is the indication that the areas with the highest levels of TGR in fact benefit the least from even a program as intensified as IOC. Similarly, the program effect varies in an anticipated fashion when dividing the sample according to level of maternal iodine received. Among children of mothers who were targeted to receive 200 mg of iodine, the estimated program effect is close to zero and insignificant while the estimated program effect on children whose mothers were targeted to receive 380 mg is 0.43 years and highly significant.

**4.2 Control Experiment**

To verify that estimated program effects are not driven by time invariant district-specific patterns of schooling attainment by age, we regress grade attainment of children in the sample districts that were 10 to 13 in 1988. For this sample, we construct a pseudo-indicator of IOC program participation that is equivalent to the indicator assigned to kids of the same age and district in the 2000 data. Since children who were between 10 and 13 in 1988 were indisputably too old to benefit from IOC in utero, if our results truly reflect an effect of IOC we should observe no relationship between the program variable and education outcomes in within-sibling regressions on this sample. These results are presented in Table 7. For consistency with the previous estimates, we run the regressions separately by gender and also divide the sample according to 2000 levels of cassava production. All regressions yield insignificant estimates of the effect of our age- and district-specific indicator of program participation on 1988 schooling.

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

### 4.3 Health Outcomes

Our principal identifying assumption is that variation in program activity (including both program gaps and delays in program start dates) is uncorrelated with other in utero influences on schooling attainment. As discussed in Section 3.3, the only potential violation of this assumption is the possibility that gaps in program coverage were associated with within-district changes in other fetal health inputs, which in turn affected schooling through persistent differences in health status. We test whether our observed program effects could be driven by differences in fetal health inputs other than iodine by looking for differences across siblings in childhood health outcomes according to program timing.

Table 8 presents results from regressions of child health 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, as well as the 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 the alternative hypothesis since school days missed due to illness is the mechanism through which in utero health damage would most likely account for some or all of the observed program effect on schooling. The estimates indicate no relationship between IOC coverage and school days missed due to illness, nor is there any evidence that children covered by the program report fewer episodes of ill health. Although the long run health 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 any observable aspect of child health at ages 10-13. Since the effects of fetal iodine deficiency are thought to be overwhelmingly cognitive, whereas deficiencies in other health inputs are more likely to show up in physical health outcomes, this is consistent with the interpretation that the estimated program effect operates through IOC, although it is impossible to rule out in utero cognitive damage resulting from the absence of health inputs other than IOC.

### 4.5 Discussion

The patterns of results observed in Tables 4-8 are consistent with a change in the cognitive cost of schooling resulting from lower incidence of fetal IDD, which is widely believed to lead to permanent reductions in cognitive capacity. Although IQ is unobservable, it is difficult to imagine other possible channels through which program participation could have influenced those children of mothers born during the program relative to their siblings or peers born a few years earlier and later. One such possibility is that iodine levels in utero influenced overall physical health of offspring which

in turn increased their ability to attend and progress in school. Two arguments suggest that this is not the primary channel: First, iodine deficiency has been demonstrated in laboratory studies to influence brain development much more readily than physical development in utero. Second, data on incidence of illness indicate that children born during the program are no more likely to become sick (Table 5).

Another possible channel of influence related to the effects of iodine on physical development is a selection story in which iodine in utero protects children against fetal or childhood mortality. Although we cannot test this directly without information on children in the household that have died, age patterns do not indicate fertility or cohort size effects of the program. Secondly, it is unlikely that increases in survival rates would give rise to higher rates of schooling attainment at ages 10 to 13.

The results on gender differences are more ambiguous. While observed gender differences in the impact of IOC may reflect physiological differences in the importance of iodine for fetal brain development, there are two other possible interpretations for this finding. First, gender differences may reflect the fact that girls in Tanzania systematically enter school at an earlier age than boys, a pattern that is observed in the 1988 Census data as well as the 2000 THBS and 2004 TDHS data. Hence, by a given age they have already reached a higher grade than their male peers. If the importance of cognitive ability on school pass rates increases with grade, as is likely to be the case, girls between the ages of 10 and 13 will benefit more from the intervention simply because they are more likely to be on the margin of influence. Although the two trends are impossible to separate without information on age of entry (unavailable from these sources), it is relevant to note that baseline gender differences in grade for age are relatively small. To account for the full gender difference in IOC, an 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.

The second possible reason we might observe differences across girls and boys in the impact of IOC in utero is that parents' decision to invest in girls' schooling is 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 opportunities for marrying young. If this is true, boys and girls might experience the same cognitive benefits of IOC at a biological level, but that translates into greater schooling benefits for girls.

Unfortunately, without data on cognitive capacity, there is no simple way to distinguish the last story from a disproportionate improvement in cognitive capacity among girls. The following section attempts to take a step in this direction by utilizing information on the rates at which students pass the Primary School Leaving Examination (PSLE).

## 5 PSLE Test Scores

In order to better connect our results on schooling attainment to improvements in cognitive ability, we make use of district-level aggregate data on 2004 Primary School Leaving Examination (PSLE) pass rates by gender, available from the Ministry of Education for 83 of the 106 districts in the country.[25] PSLE data are available on the number of boys and girls in each district who take the test in 2004, and the number of boys and girls who receive each of five categories of test grade, three of which constitute passing grades. Test-takers in 2004 are likely to fall between the ages of 14 and 18, so correspond almost perfectly to the cohort of children most affected by IOC distribution, or kids aged 10-14 in the 2000 THBS data.

At the end of primary school (standard 7), 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 percent. In 1997, only 20.1 percent of pupils who sat for the PSLE passed the examination, which has since increased to 22.0 percent in 2000 and 28.6 percent 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 percent of girls who sat for the PSLE passed the examination compared to 36.2 percent 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 not well 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 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.[26] We examine the fraction of students who take the PSLE, and the fraction of test-takers who pass and therefore transition to secondary school with the following set of regressions, run separately by gender:

$$\ln(testta\ker s04_d) = \alpha + \beta_1 (T_d) + \beta_2 (\ln(pop04_d)) + \beta_3 (hsrate88_d) + \beta_4 (X_d) + \varepsilon_d \quad (2)$$

$$\ln(testtpasser s04_d) = \alpha + \beta_1 (T_d) + \beta_2 (\ln(testta\ker s04_d)) + \beta_3 (hsrate88_d) + \beta_4 (X_d) + \varepsilon_d \quad (3)$$

[25] 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.

[26] 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 close cohorts.

In both estimates, an observation is a district. The first outcome, *testtakers04,* is the number of male or female individuals in district *d* who take the PSLE in 2004, *T* is whether there was IOC distribution in district *d* between 1986 and 1992, *pop04* is the number of males or females in district *d* between the ages of 10 and 14 in the 2002 Census (therefore the population of high-school age in 2004), and *hsrate88* is the fraction of females or males age 21-25 in district *d* who were ever enrolled in form 1 or above according to the 1988 Census. In the second regression, the outcome is the number of girls or boys who *pass* the 2004 PSLE (achieve a grade of C or above) controlling for the number of test-takers. In both regressions, $X_d$ contains the following set of district-level controls: 2000/2001 Gini coefficient of income inequality and percent of population below poverty line.[27] Unfortunately, 2002 census data on school enrollment are currently unavailable, but 2004 PSLE pass rates are likely to be a reasonable proxy for the secondary school transition rate in the cohort of test-takers. As such, one caveat is that conclusions about secondary school enrollment drawn from this analysis depend on a low rate at which children who pass the test fail to enroll in secondary school, which is likely to be the case given the extreme competition for slots in Tanzanian secondary schools and low PSLE pass rates.

Results from these regressions are presented in Table 9. 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. We do, however, observe that the number of individuals passing the PSLE is higher in IOC districts and particularly so for females. The point estimates of 0.24 and 0.15 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 particularly evident in a comparison of test score distributions across gender and program participation (Figure 5), 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. Regression estimates that control for district characteristics 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. The decrease is statistically significant for both genders in regression estimates but is larger in magnitude for females.

## 6 Impact of universal salt iodization on cross-country comparisons

The magnitude of the estimated effect of IOC on schooling in Tanzania implies that

[27] 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).

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.

### 6.1 Global trends in IDD and Salt Iodization

The International Council for the Control of Iodine Deficiency Disorders (ICCIDD) came into existence 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 6 shows the current prevalence of IDD, and Figure 7 shows 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).

### 6.2 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[28]; 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

[28] 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.

Status (CIDDS) database maintained by the ICCIDD.[29] 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 8 plots IDD and degree of salt iodization for countries in Africa. As can be seen in the scatter plot, within Africa alone there is a great deal of variation in both baseline IDD (as measured by 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.[30] We then test whether reductions in IDD over this period are associated with improvements in schooling attainment by adding to the regression the fraction of households consuming iodized salt in 2000. All cross-country regression results are presented in Table 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%.[31] 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

[29] 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).

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

[31] 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.

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 the estimated effect 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-data 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.

**6.3 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.[32] 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 5% to 45%, with the largest gains in Africa. Based on our estimates, XXX should experience an XX% improvement in schooling attainment, and XXX a XX% gain. 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%.

**7 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 its influence on cognition. Second, our findings support the laboratory evidence that female fetuses are more sensitive to in utero iodine exposure, such that endemic iodine deficiency may give rise to gender differences in cognitive ability and related outcomes. The possibility that physiological gender differences exert a significant influence on schooling has important implications for how we interpret gender differences in schooling attainment across the globe and over time. An important caveat in interpreting the differential impact of reductions in iodine deficiency by gender is that we cannot fully rule out the possibility that gender differences are driven by sex-specific household responses to improvements in cognition rather than disproportionate increases in female cognitive capacity. However, the corresponding evidence of gender differences in fetal sensitivity to maternal iodine levels from controlled laboratory studies in animals should not be discounted.

Finally, reduced levels of IDD due to wide-scale salt iodization in the 1990s are likely to have a visible impact on schooling attainment in previously afflicted areas over the next two decades, and these changes are likely to disproportionately benefit girls. Our estimates indicate that at least 41 million children have been affected by these reforms, which could increase average schooling attainment in many countries by over 10%. In areas with baseline IDD comparable to districts in the middle range of our sample, universal salt iodization could go far towards achieving gender parity in schooling attainment. The possible role of 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

[32] 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.

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 distribution or changing methods of cassava production are necessary to achieve current Millennium Development Goals regarding micronutrient deficiencies. Although such approaches are significantly more costly than salt iodization, future returns to gains in schooling attainment are likely to outweigh the costs.

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Figure 1: Intervention districts

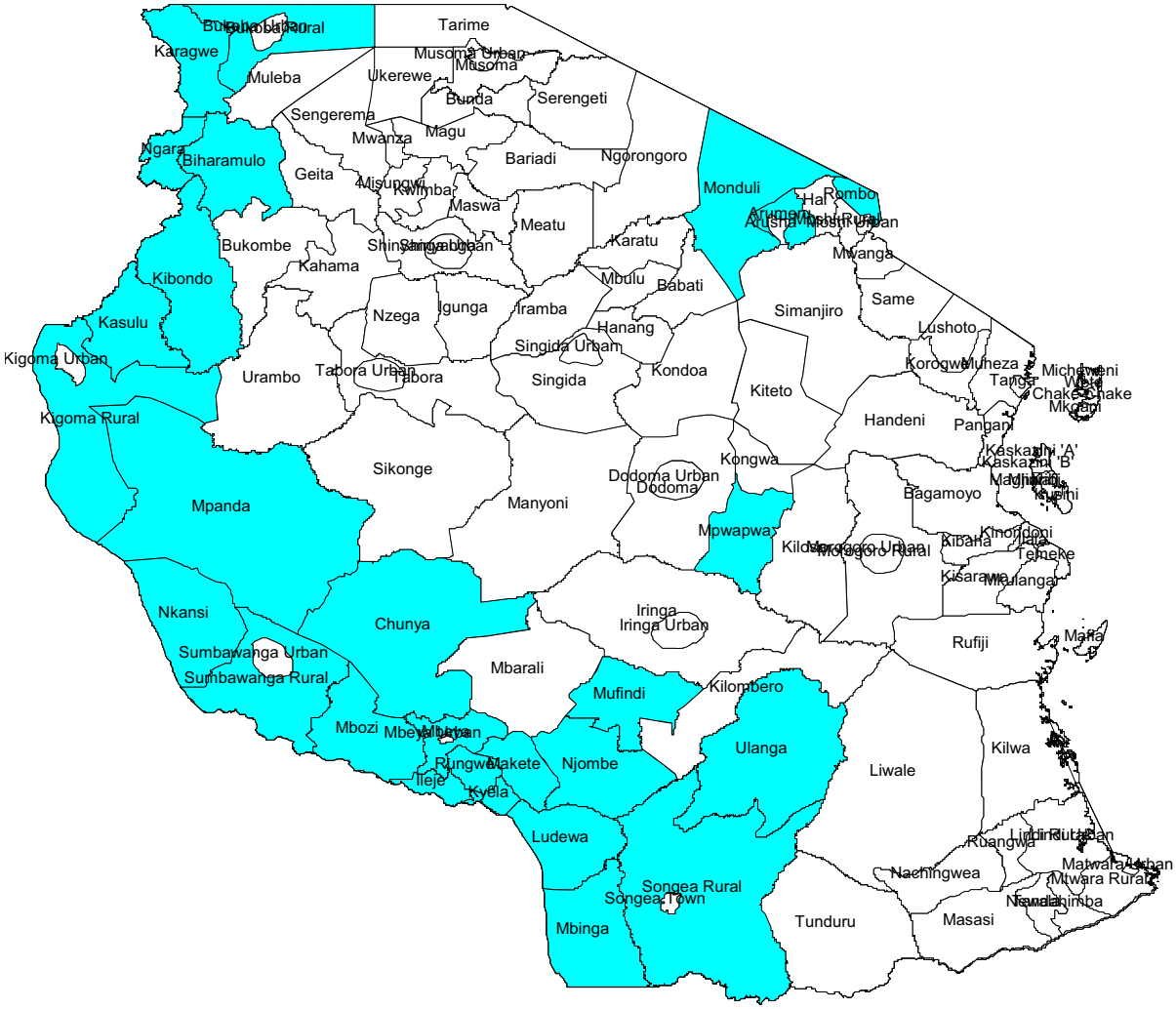
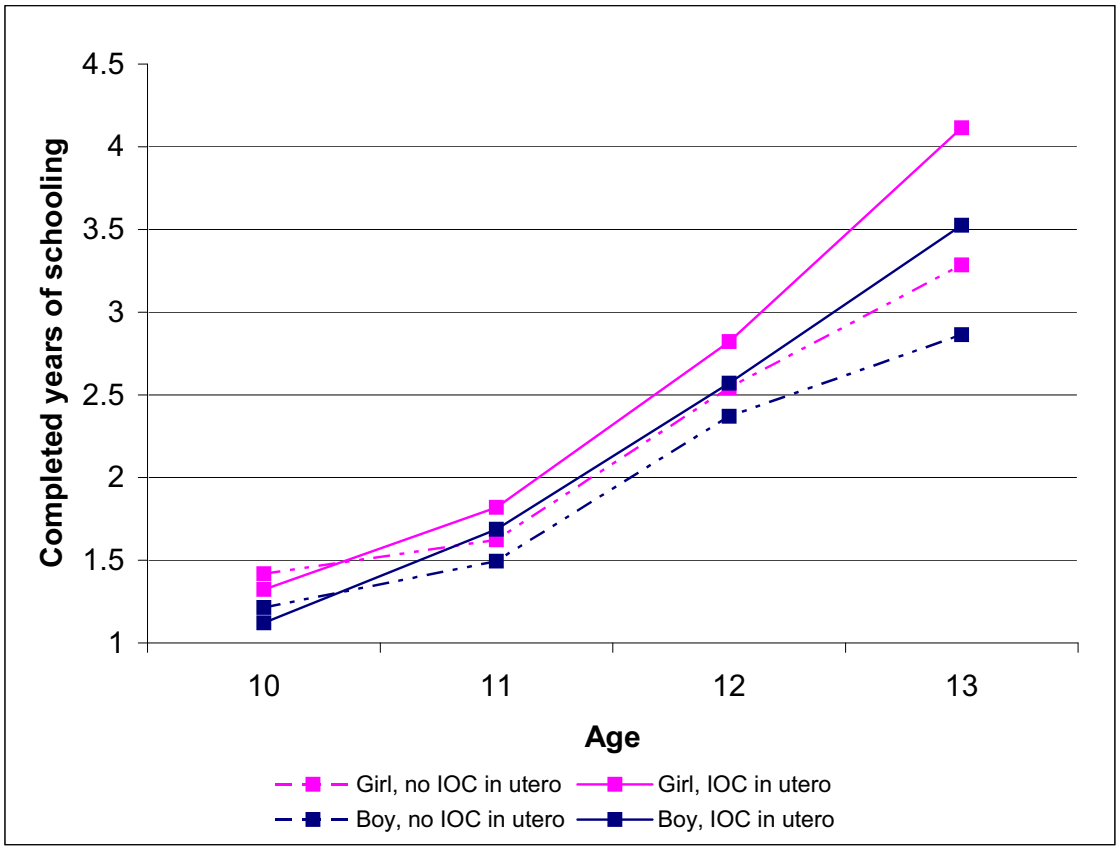
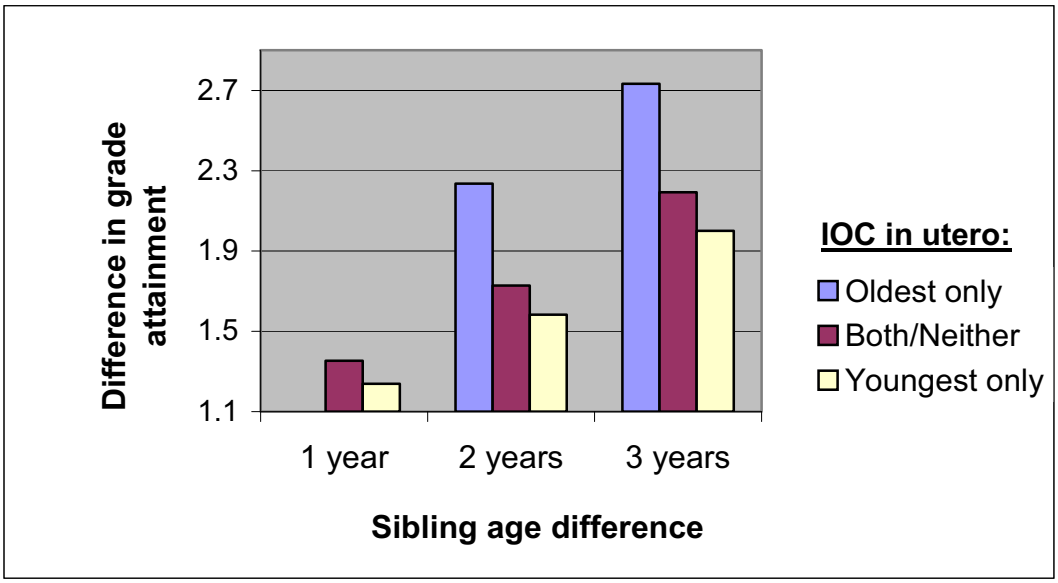


Figure 2: Grade progression by gender and IOC



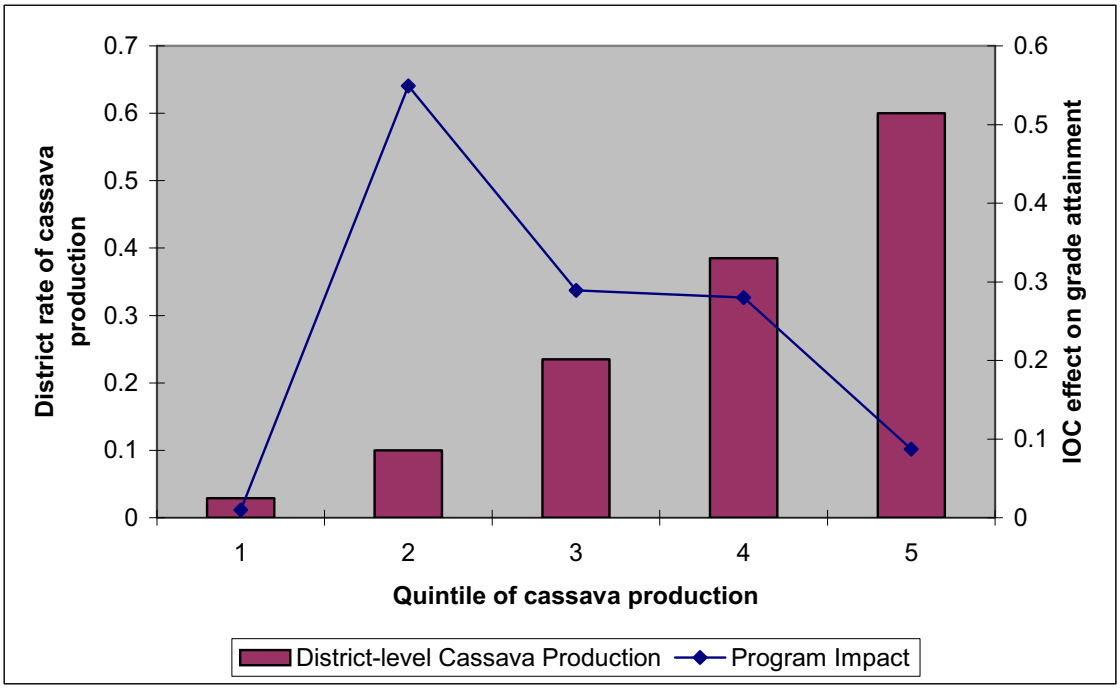

Notes: Data from the 2000 Tanzania Household Budget Survey. 2277 observations are all children in project districts between the ages of 10 and 13 that are children or grandchildren of the household head or spouse. X-axis is child age and y-axis is completed years of schooling. IOC in utero refers to whether iodized oil capsules distributed in district of residence 1 or 2 years prior to child's year of birth. Since IOC prevents maternal iodine deficiency for an estimated 24 months, IOC distributed 1-2 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 3: Sibling differences in schooling by age difference and IOC



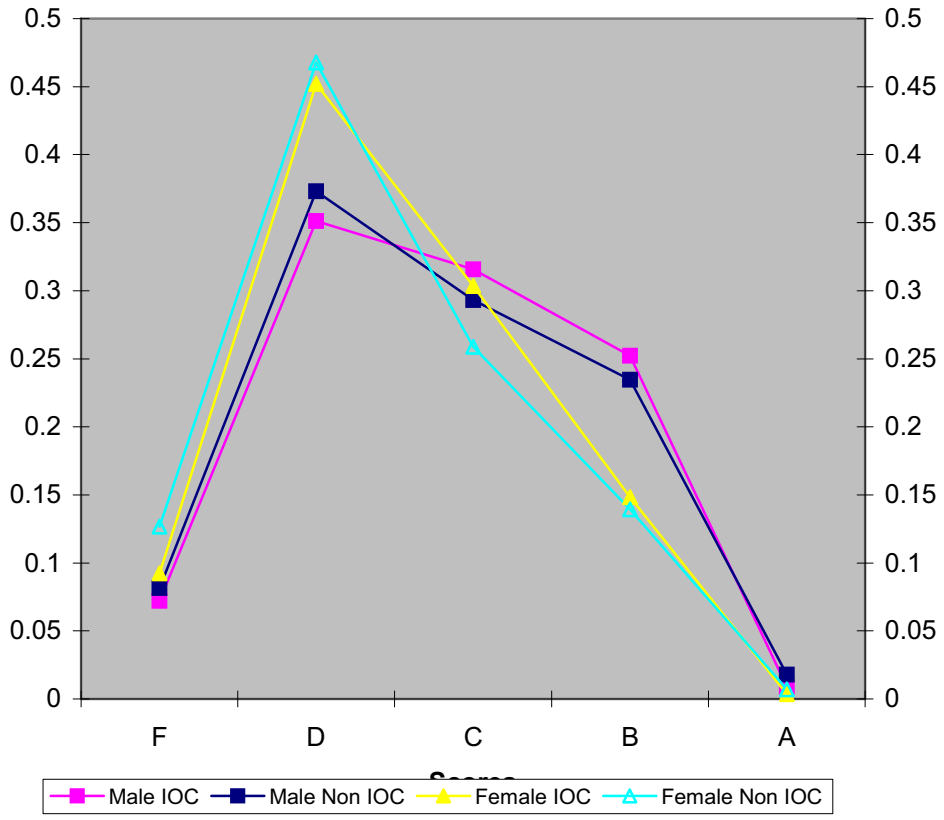

Notes: Data from the 2000 Tanzania Household Budget Survey. 576 observations comprise all sibling pairs in 25 pre-1994 project districts in which both children are between the ages of 10 and 13 and are children or grandchildren of the household head or spouse. Mother-child linkages are not perfectly 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 4: Schooling Effect of IOC by District Level of Cassava Production




Notes: Data from the 2000 Tanzania Household Budget Survey. 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 5: PSLE Score Distribution by Gender and IOC participiation




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 6: IDD Prevalence across African countries, 2000-2005

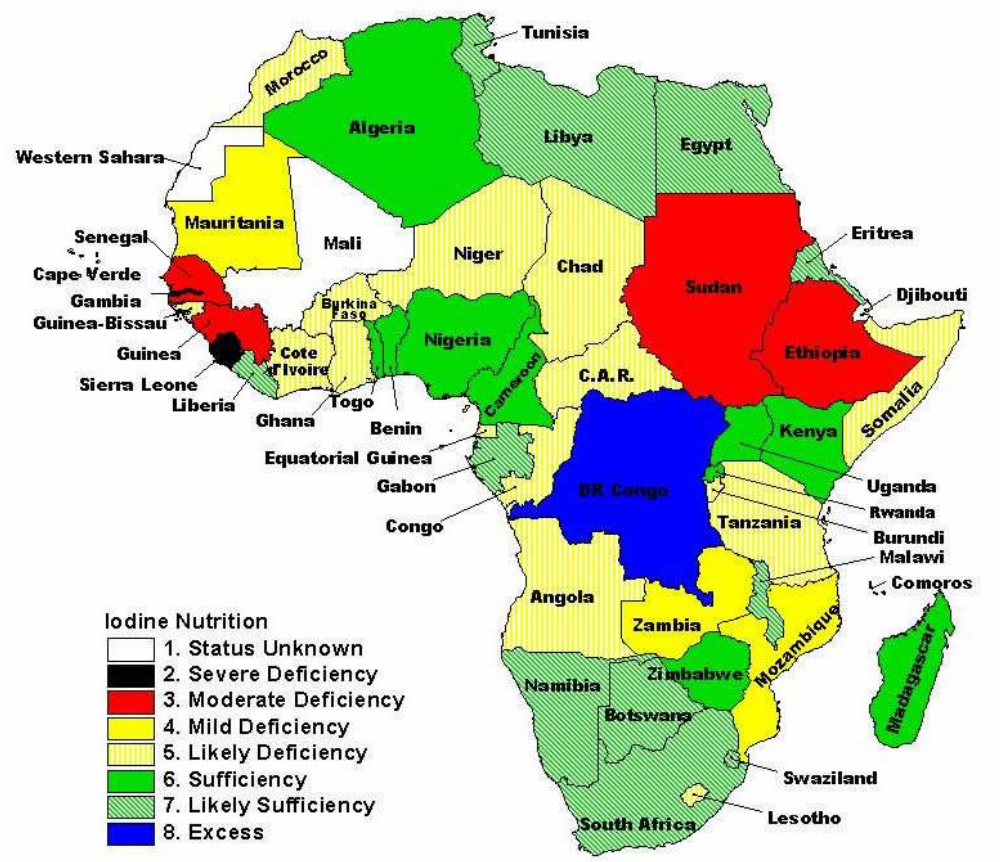


Figure 7: Fraction of households consuming iodized salt, 2000-2005

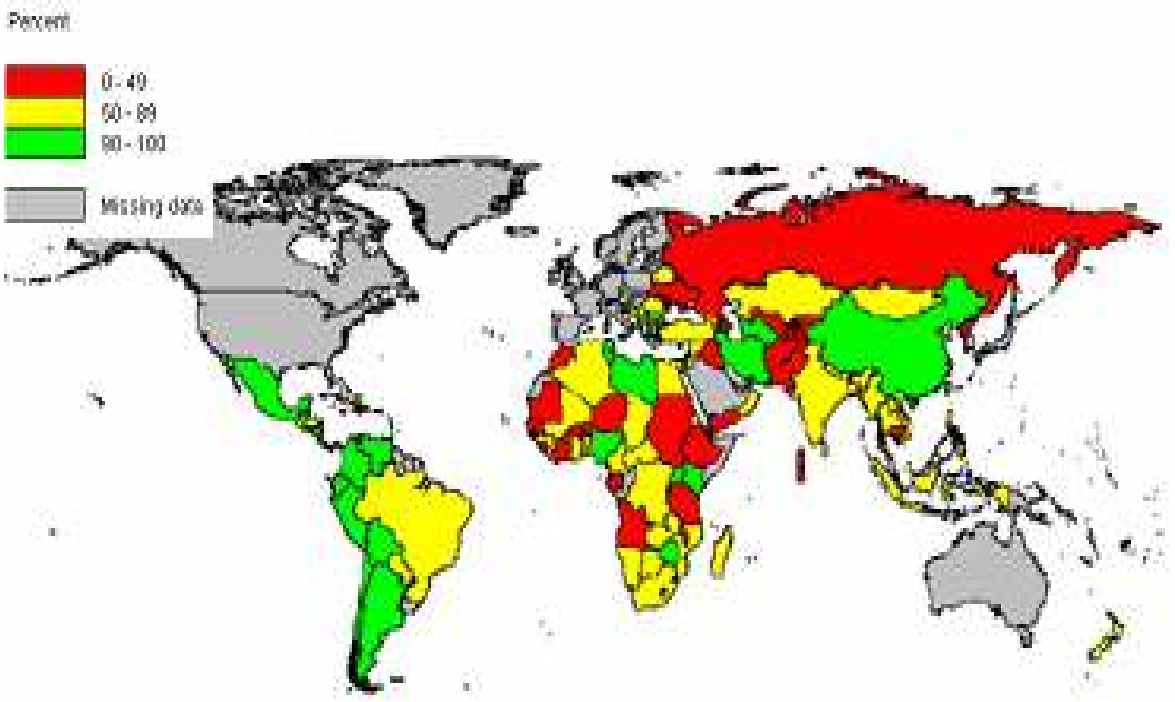


Figure 8: TGR pre-1995 and % of Households Consuming Iodized Salt, 2000-2005, by African Country

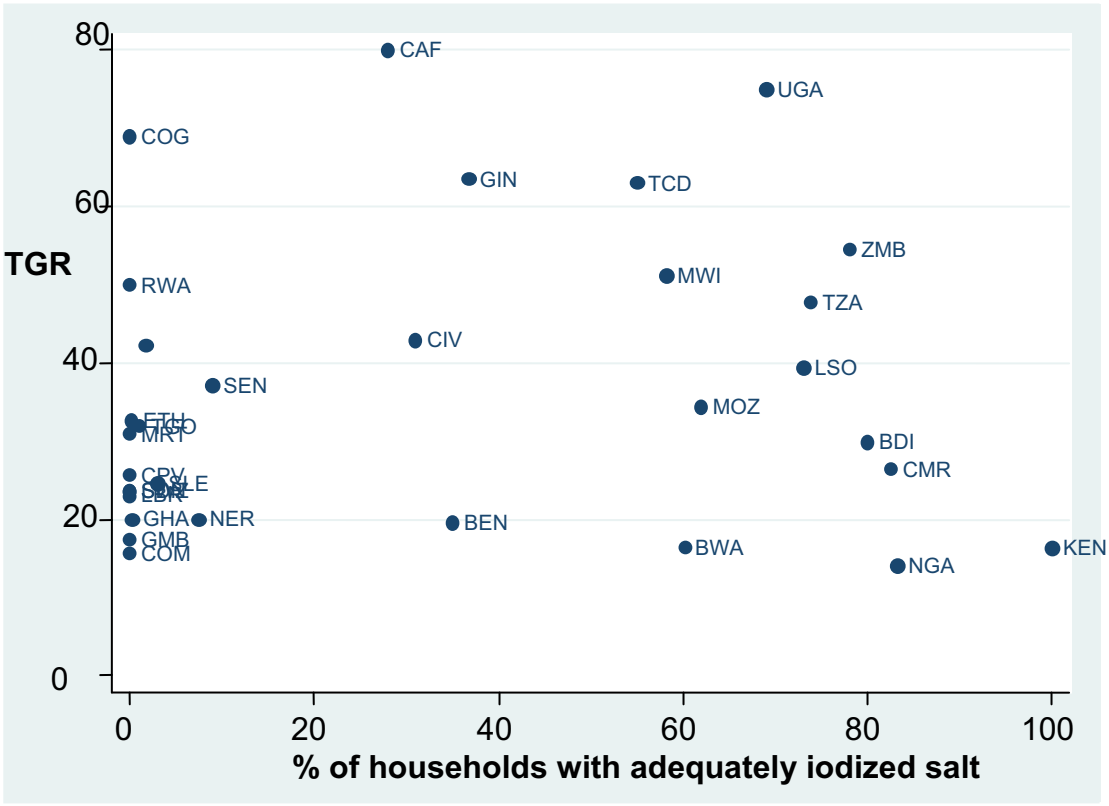


Table 1: Summary of Timing and Coverage of Intervention Across Districts

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

Notes: Dates and coverage rates collected from various Tanzanian Food and Nutrition Centre (TFNC) Zafari Reports stored in the archives of TFNC library. Coverage was calculated using 1988 Tanzanian Census data and adjusted for proportion of population in target age group.

Table 2: Summary Statistics by Timing of Intervention Across Districts

| | IOC Program Timing | | | | $t_{\Delta}$ |
|---|---|---|---|---|---|
| | 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

| | Boys and girls | Boys | Girls | Boys and girls | Boys | Girls | Boys | Girls |
|---|---|---|---|---|---|---|---|---|
| IOC in utero | 0.357 | 0.315 | 0.77 | 0.157 | -0.027 | 0.383 | | |
| *(IOC in utero =born 1-3 years after program)* | [0.142]* | [0.267] | [0.298]* | [0.112] | [0.154] | [0.166]* | | |
| IOC in utero | | | | | | | -0.023 | 0.408 |
| *(IOC in utero =birth year Pr(receiving IOC))* | | | | | | | [0.177] | [0.187]* |
| Age 11 | 0.558 | 0.571 | 0.743 | 0.401 | 0.45 | 0.296 | 0.451 | 0.251 |
| | [0.140]** | [0.272]* | [0.256]** | [0.091]** | [0.125]** | [0.135]* | [0.126]** | [0.135] |
| Age 12 | 1.293 | 1.237 | 1.531 | 1.18 | 1.206 | 1.148 | 1.209 | 1.08 |
| | [0.118]** | [0.216]** | [0.234]** | [0.086]** | [0.120]** | [0.124]** | [0.121]** | [0.125]** |
| Age 13 | 2.049 | 1.952 | 2.657 | 1.866 | 1.714 | 2.015 | 1.719 | 1.941 |
| | [0.148]** | [0.278]** | [0.293]** | [0.096]** | [0.132]** | [0.141]** | [0.127]** | [0.134]** |
| Female | 0.247 | | | 0.213 | | | | |
| | [0.090]** | | | [0.063]** | | | | |
| Mother < age 23 at birth | -0.195 | 0.128 | -0.31 | 0.071 | 0.022 | 0.11 | 0.024 | 0.093 |
| | [0.202] | [0.356] | [0.501] | [0.070] | [0.096] | [0.103] | [0.095] | [0.101] |
| Number same sex siblings | | | | 0.208 | 0.187 | 0.25 | 0.187 | 0.258 |
| | | | | [0.076]** | [0.104] | [0.113]* | [0.104] | [0.113]* |
| Fixed effects | *House-hold* | *House-hold* | *House-hold* | *District* | *District* | *District* | *District* | *District* |
| *Observations* | *2251* | *1154* | *1097* | *2251* | *1154* | *1097* | *1154* | *1097* |

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 in 27 districts targetted for iodized oil capsule (IOC) distribution between 1986 and 1995. In columns 1-6, IOC in utero is equal to 1 or 0.5 (depending on mother's age at birth) if a child was born 1-3 years after IOC was distributed in the district; in columns 7-8, IOC in utero is equal to 1 or 0.5 (depending on mother's age at birth) if a child was born 1-2 years after IOC was distributed in the district. All regressions control for birth order and sex-specific birth order. * 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) | 0.313 | 0.442 | 0.235 | 0.457 | 0.718 | 0.397 | 0.65 | 0.933 | 0.319 | 0.799 | 1.033 | 0.607 |
| | [0.165]+ | [0.245]+ | [0.232] | [0.190]* | [0.468] | [0.362] | [0.219]** | [0.322]** | [0.306] | [0.250]** | [0.601]+ | [0.437] |
| (IOC, Years 3 & 4)* | 0.023 | 0.053 | -0.01 | 0.031 | 0.081 | 0.027 | 0.021 | 0.049 | -0.011 | 0.027 | 0.074 | 0.025 |
| Birth month | [0.018] | [0.027]* | [0.025] | [0.022] | [0.056] | [0.039] | [0.018] | [0.027]+ | [0.025] | [0.022] | [0.057] | [0.039] |
| Pr(IOC in utero)*Age | | | | | | | -0.132 | -0.194 | -0.033 | -0.131 | -0.116 | -0.086 |
| | | | | | | | [0.056]* | [0.083]* | [0.078] | [0.062]* | [0.139] | [0.101] |
| Age 11 | 0.509 | 0.504 | 0.534 | 0.334 | 0.306 | 0.441 | 0.52 | 0.529 | 0.535 | 0.342 | 0.32 | 0.433 |
| | [0.069]** | [0.102]** | [0.095]** | [0.108]** | [0.242] | [0.193]* | [0.069]** | [0.103]** | [0.095]** | [0.108]** | [0.243] | [0.194]* |
| Age 12 | 1.076 | 1.175 | 0.968 | 0.96 | 0.858 | 1.169 | 1.109 | 1.236 | 0.975 | 0.98 | 0.87 | 1.178 |
| | [0.069]** | [0.103]** | [0.095]** | [0.124]** | [0.281]** | [0.211]** | [0.070]** | [0.106]** | [0.096]** | [0.124]** | [0.281]** | [0.212]** |
| Age 13 | 1.674 | 1.839 | 1.492 | 1.39 | 1.662 | 1.329 | 1.722 | 1.917 | 1.503 | 1.422 | 1.689 | 1.343 |
| | [0.071]** | [0.107]** | [0.098]** | [0.166]** | [0.384]** | [0.282]** | [0.074]** | [0.112]** | [0.102]** | [0.166]** | [0.386]** | [0.283]** |
| Age 14 | 2.229 | 2.401 | 2.088 | 2.096 | 2.153 | 2.262 | 2.283 | 2.49 | 2.101 | 2.129 | 2.174 | 2.283 |
| | [0.074]** | [0.113]** | [0.099]** | [0.201]** | [0.453]** | [0.342]** | [0.077]** | [0.119]** | [0.103]** | [0.201]** | [0.453]** | [0.343]** |
| Month of birth | -0.036 | -0.048 | -0.029 | -0.029 | -0.055 | -0.025 | -0.035 | -0.048 | -0.029 | -0.028 | -0.052 | -0.023 |
| | [0.007]** | [0.010]** | [0.009]** | [0.010]** | [0.023]* | [0.017] | [0.007]** | [0.010]** | [0.009]** | [0.010]** | [0.023]* | [0.017] |
| Female | 0.315 | | | 0.348 | | | 0.315 | | | 0.351 | | |
| | [0.044]** | | | [0.062]** | | | [0.044]** | | | [0.062]** | | |
| District FE | no | no | no | yes | yes | yes | no | no | no | yes | yes | yes |
| Household FE | yes | yes | yes | no | no | no | yes | yes | yes | no | no | no |
| *Observations* | *3672* | *1797* | *1875* | *3672* | *1797* | *1875* | *3672* | *1797* | *1875* | *3672* | *1797* | *1875* |

Notes: Data from the 2004 Tanzanian Demographic and Health Survey, sample restricted to children ages 10-14. Pr(IOC in utero) is the probability that IOC was distributed in the district before or during the first tri-mester of pregnancy, times the likelihood that sufficient store of iodine remain in the mother's body to protect the child during month 1 of pregnancy. These values are presented in Appendix A of the paper. All regressions control for dummy indicators of sex-specific birth order. + significant at 10%; * significant at 5%; ** significant at 1%.

Table 4: Difference in Grade Attainment and IOC Supplementation By Birth Order

| | | |
|---|---|---|
| IOC in utero, eldest only | 0.383<br>(0.201)* | 0.383<br>(0.212)* |
| IOC in utero, youngest only | -0.225<br>(0.129)* | -0.225<br>(0.134)* |
| IOC in utero, both | | -0.001<br>(0.127) |
| Age difference = 1 year | 0.616<br>(0.176)** | 0.616<br>(0.176)** |
| Age difference = 2 years | 0.99<br>(0.160)** | 0.99<br>(0.159)** |
| Age difference = 3 years | 1.333<br>(0.197)** | 1.333<br>(0.198)** |
| Age oldest | 0.157<br>(0.057)** | 0.157<br>(0.088)** |
| Both female | -0.041<br>(0.123) | -0.041<br>(0.124) |
| Both male | -0.115<br>(0.117) | -0.115<br>(0.117) |
| Birth order | -0.008<br>(0.030) | -0.008<br>(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* | | | *Amount of IOC* | |
|---|---|---|---|---|---|
| | High (0.41-0.62) | Medium (0.10-0.40) | Low (< 0.10) | Mother>22 at birth (380 mg) | Mother<23 at birth (200 mg) |
| IOC in utero | 0.046 | 0.508 | -0.02 | 0.431 | 0.066 |
| *(IOC in utero =born 1-3 years after program)* | (0.391) | (0.165)** | (0.252) | (0.198)* | (0.199) |
| Female | 0.417 | 0.172 | 0.154 | 0.252 | 0.304 |
| | (0.188)* | (0.146) | (0.148) | (0.146) | (0.156) |
| Age 11 | 0.68 | 0.46 | 0.451 | 0.783 | 0.401 |
| | (0.281)* | (0.222)* | (0.245) | (0.225)** | (0.242) |
| Age 12 | 1.64 | 1.224 | 0.967 | 1.524 | 1.232 |
| | (0.241)** | (0.179)** | (0.205)** | (0.195)** | (0.210)** |
| Age 13 | 2.086 | 2.051 | 1.724 | 2.27 | 1.684 |
| | (0.298)** | (0.232)** | (0.295)** | (0.253)** | (0.263)** |
| Household fixed effects | yes | yes | yes | yes | yes |
| *Observations* | *669* | *804* | *778* | *983* | *799* |

Notes: Data from the 2000 Tanzanian Household Budget Survey, sample restricted to children ages 10-13 in 27 districts targetted for iodized oil capsule (IOC) distribution between 1986 and 1995. Children and women below age 23 were given IOC containing 200mg of iodine and women over 22 were given IOC containing 380 mg of iodine. In all regressions, IOC in utero is equal to one if a child was born 1-3 years after IOC was distributed in the district. Regressions also control for birth order and sex-specific birth order. Rate of cassava consumption defined as fraction of THBS households in district that report growing cassava.

Table 7: Control Experiment, IOC Distribution and Grade Attainment of Older Cohort

| | | | | *Rate of Cassava Consumption in District* | | |
|---|---|---|---|---|---|---|
| | Boys and girls | Boys | Girls | *High* | *Medium* | *Low* |
| IOC in utero | -0.021 | 0.058 | -0.035 | -0.002 | 0.070 | -0.021 |
| *(IOC in utero =born 1-3 years after program)* | (0.020) | (0.037) | (0.036) | (0.058) | (0.057) | (0.039) |
| Age 11 | 0.647 | 0.672 | 0.563 | 0.691 | 0.67 | 0.55 |
| | (0.023)** | (0.045)** | (0.043)** | (0.043)** | (0.040)** | (0.040)** |
| Age 12 | 1.505 | 1.51 | 1.351 | 1.458 | 1.573 | 1.416 |
| | (0.027)** | (0.055)** | (0.053)** | (0.050)** | (0.046)** | (0.047)** |
| Age 13 | 2.38 | 2.372 | 2.225 | 2.377 | 2.424 | 2.248 |
| | (0.035)** | (0.072)** | (0.069)** | (0.062)** | (0.059)** | (0.063)** |
| Female | | | | 0.251 | 0.288 | 0.267 |
| | | | | (0.027)** | (0.025)** | (0.024)** |
| Household fixed effects | *yes* | *yes* | *yes* | *yes* | *yes* | *yes* |
| *Observations* | *113932* | *57613* | *56319* | *37144* | *36900* | *39888* |

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 27 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, IOC in utero is equal to one if a child was born 9-11 years before IOC was distributed in the district. Regressions also control for birth order and sex-specific birth order.

Table 8: Effect of IOC Distribution on Reported Health Status

| | *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* |
|---|---|---|---|---|---|---|---|---|---|---|
| IOC in utero | 0.013 | 0.039 | -0.017 | -0.011 | 0.007 | 0.008 | -0.009 | 0.008 | -0.008 | -0.062 |
| *(IOC in utero =born 1-3 years after program)* | [0.038] | [0.033] | [0.013] | [0.014] | [0.008] | [0.011] | [0.008] | [0.006] | [0.019] | [0.153] |
| Age 11 | -0.026 | 0.043 | -0.015 | -0.002 | -0.011 | 0 | -0.012 | -0.003 | -0.016 | -0.01 |
| | [0.044] | [0.038] | [0.015] | [0.016] | [0.010] | [0.013] | [0.010] | [0.007] | [0.022] | [0.182] |
| Age 12 | -0.002 | -0.018 | -0.018 | -0.013 | -0.001 | 0.012 | 0.005 | 0.005 | 0.01 | 0.135 |
| | [0.037] | [0.031] | [0.013] | [0.013] | [0.008] | [0.010] | [0.008] | [0.006] | [0.018] | [0.202] |
| Age 13 | 0.002 | 0.036 | -0.023 | 0 | -0.015 | 0.003 | -0.005 | 0.003 | 0.018 | 0.161 |
| | [0.047] | [0.040] | [0.016] | [0.017] | [0.010] | [0.013] | [0.010] | [0.008] | [0.023] | [0.222] |
| Household fixed effects | yes | yes | yes | yes | yes | yes | yes | yes | yes | yes |
| *Observations* | 2277 | 2277 | 2277 | 2277 | 2277 | 2277 | 2277 | 2277 | 2277 | 2277 |

Notes: Outcome is whether child reported by respondent to have experienced any of above health problems during last four weeks; last column is amount of absence due to sickness during 4 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 targetted for iodized oil capsule (IOC) distribution between 1986 and 1992. In all regressions, IOC in utero is equal to one if born 1-2 years before IOC distributed in district. Regressions also control for sex, birth order and sex-specific birth order.

Table 9: Male and Female PSLE Performance by IOC Intervention

| *Dependent Variable:* | ln (number individuals taking PSLE)[1] | | ln(number individuals with passing grade on PSLE)[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) | (0.08) | (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) | | (0.12) | | (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) | | (0.18) | | (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 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 PSLE data were unavailable for the island regions of North and South Pemba and Zanzibar. The 2002 Census from the National Bureau of Statistics reports a total of 117 districts in mainland Tanzania, 18 of which were newly constructed from the division of existing districts in the 1988 Census. In order to construct a panel with the 1988 Census, divided districts were re-aggregated in the 2004 PSLE and 2002 Census samples, such that the complete panel contains 99 districts. In addition, a total of 6 districts were excluded from the analysis due to lack of information on PSLE scores from the Iringa region.

* 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 10: 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 the following information about the sample that was used to calculate TGR: minimum age, maximum age, gender (both, male, or female), sample level (national or regional), and year of study.

5 Many countries were excluded from the above analysis due 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 WHO's Micronutrient Deficiency Information System and supplmented by the Current Iodine Deficiency Status (CIDDS) database maintained by the International Council for the Control of Iodine Deficiency Disorders.

Table 11: 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 | Percentage increase in grade attainment [4] [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**

| | Jan | Feb | March | April | May | June | July | Aug | Sept | Oct | Nov | Dec | Birth year average |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *Program year t* | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.08 | 0.17 | 0.25 | 0.33 | 0.42 | 0.10 |
| *t + 1* | 0.50 | 0.58 | 0.67 | 0.75 | 0.83 | 0.92 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 0.85 |
| *t + 2* | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 1.00 | 0.99 | 0.98 | 0.96 | 0.99 |
| *t + 3* | 0.93 | 0.90 | 0.85 | 0.81 | 0.75 | 0.69 | 0.62 | 0.54 | 0.46 | 0.38 | 0.31 | 0.25 | 0.62 |
| *t+ 4* | 0.19 | 0.15 | 0.10 | 0.07 | 0.04 | 0.02 | 0.01 | 0.00 | 0.00 | 0.00 | 0.00 | 0.00 | 0.05 |