University of Sussex

Three Essays on the Economics of Nutrition

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Declaration

I hereby declare that this thesis has not been and will not be submitted in whole or in part to another University for the award of any other degree.

I hereby also declare that chapter 1 is co-authored with Professor Peter Dolton. My contribution to this chapter was substantial. I have cleaned, managed and analysed the data. I have also taken the lead on writing the paper and helped in advancing the empirical strategy.

Chapters 2 and 3 are single-authored.

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Summary

This thesis investigates the relationship between the social and the spatial environment and nutrition of children in high and low income countries. Furthermore, this project investigates the implications of nutrition on human capital outcomes.

The first empirical chapter is concerned with whether exposure to fast food increases BMI of adolescents. This question is studied at a time when fast food restaurants started to open in the UK. We merge data on the location and timing of the openings of all fast food outlets in the UK from 1968 -1986, with data on objectively measured BMI from the British Cohort Survey. The relationship between adolescent BMI and the distance from the respondents' homes and time since opening, is studied. We find that fast food exposure had no effect on BMI. Numerous robustness checks do not change our findings.

The second empirical chapter is the first to provide evidence of a direct causal impact of iodine fortification in early life on cognitive skills in childhood. I apply a difference-in-differences strategy using exogenous variation from a nationwide iodine fortification policy in India, comparing test scores of school aged children in naturally iodine sufficient and deficient districts over time. I find that the policy increased the probability of attaining basic numeracy and literacy skills by 2.67 - 5.83%. Previous papers find a larger effect on longer term human capital for women. I do not find a gender differential for basic skills but I observe a positive effect on more difficult literacy tasks for girls but not for boys.

The third empirical chapter investigates the effect of iodised salt availability on children's heights using a large household survey from rural India. Medical evidence points to a mechanistic relationship between iodine deficiency and a decline in the production and functioning of biological factors affecting human growth. I use a two-stage-least-squares regression to circumvent concerns regarding the endogeneity of a household's availability of iodised salt and children's anthropometric status. I instrument for iodised salt consumption with the distance to the major salt producing state. Salt transported for longer distances is likely to be transported by rail rather than by road. Monitoring of iodised salt is only mandatory before and during rail transport. Therefore, distance serves as a proxy for the likelihood that the salt has been inspected for iodine, and thus iodised. I find that the availability of adequately iodised salt improves height-for-age by 0.664 Z-scores

for children up to 1 year.

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0.1 Introduction

Around 1 in 3 adults worldwide are either obese or overweight and 22.2% of all children are stunted due to long term undernutrition (Ng 2014, UNICEF 2018). Over - and under consumption of food does not only affect health and wellbeing but also has large consequences for the economy (Alderman et al. 2017, Tremmel et al. 2017). The global cost of obesity is around 3% of worldwide GDP and as obesity is expected to increase, so are future costs (Tremmel et al. 2017).

While obesity is rising in both high and low income countries, undernutrition continues to be the most pressing issue in the global south. India carries the largest burden of undernutrition in the world and approximately two thirds of Indian adults were stunted in childhood. The cost of stunting is estimated to comprise 10% of Indian GDP (Galasso & Shekar 2017). The societal burden of undernutrition is likely to be underestimated. Micronutrient deficiency relates to the inadequate intake of essential vitamins and minerals and is often referred to as "hidden hunger". Although such deficiencies are not always captured by anthropometric measures they can potentially have large implications for health and productivity (Horton & Ross 2003). The short term economic cost of micronutrient malnutrition has been estimated to be up to 2.5% of GDP in India (Tremmel et al. 2017).

Nutritional intake is a fundamental driver of individuals' overall health and thus essential for the accumulation of other forms of human capital (see; Behrman (1993), Currie & Almond (2011), Victora et al. (2008)). This has motivated micro economists to study the causes and consequences of inadequate nutritional intake. The extant literature shows that obesity predicts lower wages, a lower probability of employment and higher medical care costs in high income countries (Cawley 2015). Evidence from middle- and low income countries reveals that undernutrition reduces cognitive capacity, educational attainment, wages, productivity, income and expenditure (Victora et al. 2008, Behrman & Deolalikar 1988, Strauss & Thomas 1998)

The social, economic and physical environments are also important determinants of over- or under consumption of food. Economic research finds that the monetary price and time cost of food, such as the access to fast foods or convenience foods; peer effects; income; education and macroeconomic conditions are likely to cause obesity (Cawley 2015). Existing literature demonstrates that public policy, income, access to public services and health care, shocks, war and discrimination of women are important factors for malnutrition in low income countries (Victora et al. 2008).

A vast number of papers investigate the role of nutrition at an early age on later hu-

man capital attainment. Starting from conception, empirical applications of the foetal programming hypothesis observe that the intra-uterine nutritional environment has persistent effects on human capital throughout the life course (Almond & Currie 2011, Scholte et al. 2015, Maccini & Yang 2009). Many studies also show that the first couple of postnatal years matter for human capital attainment (Maluccio et al. 2009, Glewwe & King 2001). Nutritional availability at later stages prior to adulthood also has long-term impacts. Overweight and obese children and adolescents are likely to remain at an unhealthy weight in adulthood as the human body reaches a permanent number of fat cells in adolescence (Spalding et al. 2008). Additionally, food habits form at a young age and are likely to persist into adulthood. Therefore, it is more difficult to reduce body mass at an older age (Epstein et al. 1995)

Effective public policies targeting children and pregnant women are thus likely to yield high returns to investment. Furthermore, markets related to children's food choices are characterised by market failures which strongly justify government interventions. In addition to the negative externalities of health care costs, imperfect information regarding nutritional choices make children less likely to internalise the consequences of food choices compared to adults (Cawley 2010).

However, many areas of research studying the relationship between societal causes and consequences of nutrition yield mixed results. Empirical studies in this domain face many difficulties. Secondary data often lack information on past and present nutritional intake, energy expenditure and physiological characteristics related to nutritional status. Self-reported anthropometric information, prone to reporting bias, is more readily available than objectively measured anthropometric status.

Secondly, it is difficult to estimate any potential causal relationship between nutritional intake and the social or physical environment. As discussed previously, the extant literature has established that the direction of this particular relationship can go in both ways. Moreover, the decision of where to reside can be correlated with dietary preferences. Thus the risk for reverse causality is inherent in this area of research. Another form of endogeneity stems from omitted variable bias. Unobserved determinants of households' and individuals' nutritional choices, as for example time preference and availability and capacity to act upon information, also dictate human capital outcomes directly. Estimating the causal relationship between spatial and social drivers of inadequate nutritional intake and human capital outcomes is therefore prone to bias if endogeneity is not addressed. The application of methods exploiting the exogenous variation in the determinants of nutrition is

therefore key. However, public health policies with regards to nutrition have traditionally not been based on many large-scale studies using empirical methods permitting a causal inference.

This thesis seeks to explore how the social and physical environments causally relate to major global nutritional problems. Each chapter contributes to the existing literature by using quasi-experimental methods to investigate the impacts of different types of nutritional availability during a young age, on human capital. The first chapter explores the impact of the access to fast food on obesity using data from the UK. The subsequent chapters investigate the consequences of the other extreme of the nutrition spectrum – malnutrition, with regards to micronutrient deficiency. The second empirical chapter evaluates the impact of a salt iodisation policy in early life on on cognitive test scores in India. The third chapter investigates the implications of access to iodised salt for children's heights in India.

The access to fast food has often been blamed for the rise in obesity, particularly among children and adolescents (Swinburn et al. 2004). This has lead to the banning of fast food outlets and implementation of zoning laws (Sturm & Hattori 2015). It is difficult to study the impact of fast food access on obesity as fast food is abundant and ever present in high income countries today. Moreover, fast food companies have learnt where to strategically locate their outlets. Therefore, estimating the impact of access to fast food on obesity is likely to suffer from endogeneity issues originating from spatial sorting. More recent studies employ IV analysis or evaluate the impact of fast food bans or exogenous determinants in the relocation of individuals, the overall results are mixed (see; Dunn (2010), Anderson & Matsa (2011), Chen et al. (2013), Alviola et al. (2014), Sturm & Hattori (2015)).

In the first empirical chapter, jointly with Professor Peter Dolton, we offer a novel way of investigating the impact of access to fast food on BMI. We study the inception of fast food in the UK as this time period offers large temporal and spatial variation in the access to fast food. We merge data on the location and timing of the openings of all fast food outlets in the UK from 1968 -1986, with data on objectively measured BMI from the British Cohort Survey. We focus on BMI at age 16 as adolescents are more likely to visit fast food outlets more often and have more control over their food choices compared to younger children (Nielsen et al. 2002, Block et al. 2013). Previous studies often use aggregate area level information. By using individual level data, we are able to control for many predictors of BMI and to account for individual specific proximity to fast food.

We study the relationship between adolescent BMI and the distance from the respondents' homes and time since opening. Analysing variations in BMI when fast food restaurants started to penetrate a previously untapped market allows us to examine the total cumulative effect of fast food exposure on BMI. We find that fast food exposure, measured either in continuous or non-linear distance, duration of exposure and distance and duration jointly, had no effect on BMI. Additionally, we do not find any effects on BMI at age 10 or 26 nor on the probability of being overweight or obese.

We test the robustness of our results in three ways which do not change our findings. We estimate the effects of a sharp and unexpected supply shock of the largest incumbent fast food company in anticipation of the entry of a new fast food company. This supply shock left the incumbent company with little time to strategically locate their outlets and provides us with greater confidence in the near random exposure to fast food. Secondly, we conduct an IV-analysis, where we instrument the proximity to a fast food outlet with the proximity to a fast food distribution centre. Thirdly, we examine the possibility of reverse causality by testing whether fast food companies sited their outlets in more obesogenic areas. We do not find evidence of fast food density varying systematically with residential characteristics.

Our paper contributes to the literature by coming closer to identifying a causal effect of fast food exposure on BMI outside of the US. The external validity of our findings are thus of large significance for most European countries battling obesity.

The remaining empirical chapters focus on the effects of access to iodised salt on human capital outcomes in a low-income setting. Iodine deficiency is found in almost all countries across the world and over 2 billion people are estimated to be at risk for the deficiency. Due to the large burden of iodine deficiency, Universal Salt Iodisation (USI) is now implemented in nearly all countries worldwide under the initiative by the WHO (Andersson et al. 2010). Iodine deficiency in utero and postnatally has been linked to permanently reduced cognition. However, limited large-scale empirical evidence exists on the causal impact of iodine intake on cognitive skills. Additionally, no previous study has evaluated the effect of the wide-spread USI programmes.

The second empirical chapter is the first to directly assess the cognitive impact of USI. Existing studies such as; Adhvaryu et al. (2018), Politi (2010*b*,*a*), Field et al. (2009) have either looked at the effect of the historical availability of iodised salt in high income countries or studied the effect of targeted iodine supplementation programmes. The evidence from such studies cannot be readily extrapolated to the potential impacts of USI today.

Historical access to iodised salt in the US during the 1920's was not regulated and relied on consumer demand. Therefore, self selection into iodised salt consumption is likely to be a threat to identification. Moreover, such studies have low levels of external validity for the potential impact of USI in countries with the highest burden of iodine deficiency today - low income countries. Additionally, this chapter contributes to our understanding of the direct impact of iodised salt on cognition. Previous large scale studies analyse the impact of iodisation policies on educational attainment and not cognitive skills. These studies have therefore not been able to explain the immediate mechanism behind the relationship between iodine intake and long-run human capital outcomes.

I apply a difference-in-differences (DD) strategy to analyse the effect of being exposed to mandatory USI during early life on children's cognitive test scores in India, across naturally iodine deficient and iodine sufficient districts over time. I use data from the Annual Survey of Education report from rural India which tests both in- and out of school children at home. I estimate the effects on cognitive test scores in numeracy and literacy for school aged children.

The results suggest that being exposed to USI in early life increases the probability of mastering basic numeracy and literacy skills, measured by the ability of at least being able to recognise numbers and letters, by 1.9 – 4 percentage points. I also observe that girls improved their overall reading ability, which takes more difficult reading tasks into account, while no effects are found for boys. Children who reside in states which experienced larger (smaller) relative increases in iodised salt consumption also gained more (less) in terms of learning outcomes.

The positive effects on cognitive test scores cannot be attributed to changes in the composition of children attending school due to children being tested at home. Moreover, placebo checks show that the main results are not driven by compositional changes across the cohorts, or by coinciding improvements in health endowments and health investments in early life or changes to the food environment induced by increased trade. Furthermore, the treatment effects hold when using geographical predictors for the risk of natural iodine deficiency in an IV-analysis and using an alternative dataset for the risk of iodine deficiency. Additionally, I show that an earlier mandatory fortification policy which was in place only during two years, also had a positive, albeit smaller, impact on test scores. This reduces the potential worry that the main findings are driven by unobserved shocks affecting children in early life in areas at risk of iodine deficiency.

The third empirical chapter investigates the role of iodine on children's growth. Height

in childhood is positively related to educational attainment and wages in adulthood (Vogl 2014, Case 2008). Existing research shows that nutrition in early life is an important determinant of height in childhood and final adult height (Alderman et al. 2017, Hoddinott et al. 2013, Case 2008, Glewwe & Edward A 2007). However, less is known about what specific aspects of undernutrition causes short stature. Medical evidence has established a physiological relationship between iodine deficiency and a decline in the production and functioning of factors causing growth, such as growth hormones, insulin like growth factors and other determinants of skeletal development. Therefore, it is likely that access to iodised salt can affect height for populations at risk for iodine deficiency. The last empirical chapter investigates the impact of access to iodised salt on height-for-age Z-scores (HAZ).

The existing evidence concerning the impact of iodine supplementation on children's somatic growth is mixed and limited. Studies using large scale survey data find positive associations between access to iodised salt and children's anthropometric status. Due to concerns about omitted variable bias and measurement error with respect to previous consumption of iodised salt, these studies are not able to estimate a causal effect of iodised salt availability on children's heights.

This empirical chapter uses exogenous variation in the access to adequately iodised salt across Indian states to investigate the effect of iodised salt availability on children's HAZ. I use IV regression to circumvent concerns regarding the endogeneity of household availability of iodised salt and children's growth. I instrument for iodised salt consumption by using distance to the major salt producing state which manufactures around 80% of all salt in India. Salt transported for longer distances is likely to be transported by rail. Monitoring of the iodine content of salt is only mandatory before rail transport but not for road transport. Therefore, distance from the major salt exporting state serves as a proxy for the likelihood that the salt has been inspected for iodine, and thus iodised.

Using a rich household survey for rural India, which includes objectively measured reports of households' consumption of adequately iodised salt and children's anthropometric status, I find that the availability of iodised salt improves height-for-age by 0.664 standard deviations and height by 1.845 cm for children up to 1 year. No effects are found on weight which is in line with the medical literature not finding a mechanistic relationship between iodine deficiency and adipose tissue. The absence of an impact on weight also rules out that the estimates are driven by a concurrent increase in overall nutritional intake for those affected by the IV.

Splitting the sample by age categories reveals that children aged 0–6 months benefit the most from iodised salt consumption with respect to linear growth. Children residing in states with a higher natural predisposition to iodine deficiency experienced somewhat larger effects. The effects of the availability of adequately iodised salt on children's HAZ are larger than many other public policy programmes (see Hammer & Spears (2016), Jain (2015)). As approximately 40% of all children in India are stunted (Menon et al. 2018), the findings from this chapter suggests that access to adequately iodised salt can potentially have a large impact for the reduction of short stature.

In summary, this thesis offers rigorous empirical evidence concerning potential drivers, and a lack thereof, of nutritional status and its impact on human capital outcomes. It is structured as follows: Chapter 1 investigates whether proximity to fast food is a potential driver of childhood and adolescent obesity in the UK. Chapter 2 evaluates the impact of mandatory USI in India on cognitive test scores of school-aged children. In chapter 3, I study the impact of plausibly-exogenous access to adequately iodised salt on children's height. I summarise the findings and discuss the limitations and possible future research questions in the conclusion, in Section 4.

Chapter 1

Childhood obesity, is fast food exposure a factor?

The increasing prevalence of childhood and adolescent obesity is a major public health problem. Today, one in three British and American children are overweight or obese, and obesity in early life is likely to persist into adulthood (Reilly 2006, Ogden et al. 2014, Guo et al. 2002).

It has been suggested that there is a positive relationship between the proximity to fast food and childhood obesity (Chou et al. 2004, Currie et al. 2010, Davis & Carpenter 2009). Fast foods are typically energy dense, have a high glycemic index and are often served in large portion sizes with soft drinks. These are all factors that could contribute to childhood obesity (Ebbeling et al. 2002). Moreover, the consumption of "junk food" exhibits strong habit formation and risk for addiction (Chen et al. 2011, Corwin & Grigson 2009). Therefore, more accessible fast food might predispose one to becoming obese, especially for children and adolescents as they are less capable to fully internalise the future consequences of their actions.

The overall evidence of the role of fast food access on obesity is mixed. It is of economic relevance to study if such a market merits intervention in order to increase social welfare (Cawley 2004). Currie et al. (2010), propose that future research should study ".. fast food restaurant entry in a society where fast food is scarce."[1] The motivation stems from that it is difficult to study the effect of fast food when it is ever present. Intrinsically this means it is hard to retrieve a causal effect of fast food proximity on BMI. Rather, if we can study an era when fast food is being introduced and some locations do not have exposure to it - or get it later - then by near random draw on geographical location, we can observe

[1] Currie et al. (2010, p. 61)

the treatment effect of fast food proximity independent of the choice of residential location. Following this logic, our study exploits the inception of fast food entry in Great Britain. Global obesity rates started to rise in the 1980s led by the US and the UK (Chinn & Rona 2001). The increased supply of cheap, accessible, convenient and energy-dense foods, is often blamed for the rising obesity prevalence (Swinburn et al. 2004). The timing also coincides with the explosion in the number of fast food establishments in these countries (Chou et al. 2004).[2]

We estimate the effect of the proximity from home and the duration of exposure to all fast food outlets on BMI in 1986. We have collected data on all fast food outlets in the UK, from inception to 1986. We combine the data with the 16 year follow-up of the 1970 British Cohort Survey (BCS). We focus on the BMI of adolescents as they are both more likely to visit fast food restaurants frequently and to underestimate the caloric content of fast foods, compared to other age groups (Paeratakul et al. 2003, Nielsen et al. 2002, Block et al. 2013). This makes them more susceptible to weight gain if exposed to fast food.

Our departure from the previous literature, is that we measure fast food exposure not only in terms of distance, but also in terms of duration. Moreover, we generate a fast food intensity measure which is a function of both distances and durations of all outlets nearby. Our additional contributions stem from the use of rich cohort data and objectively measured weights and heights. Most research on the relationship between fast food availability and obesity use self reported weights and heights which suffer from non-classical measurement error (Cawley et al. 2015). Moreover, by using individual level data we are able to control for many predictors of BMI and our study is not at risk of ecological fallacy.

Our data also allows us to examine the total cumulative effect of fast food exposure on BMI. Thus, analysing variations in BMI when fast food restaurants started to penetrate a previously untapped market, provides our study with a dramatic variation in the proximity and duration of fast food access. We exploit this natural experiment in fast food exposure to assess the impact of fast food proximity on adolescent obesity. Our paper is the first

[2]The proportion of overweight children started to increase from the mid 1980s. Between 1984 and 1994, the prevalence of overweight English and Scottish boys increased with 3.6% and 4.1% respectively. The corresponding rates for both English and Scottish girls was 5.4% (Chinn & Rona 2001). Adult obesity rates also experienced an upward trend, the percentage of 16-64 year olds who are obese in England increased from 6% of men and 8% of women in 1980, to 13% and 15% obese men and women respectively in 1991 (Cutler et al. 2003).

to come closer to identify a causal effect of fast food exposure on BMI outside of the US. This is of importance as the spatial patterning of demographic characteristics and the built environment in many countries greatly differ from the US (Cummins & Macintyre 2002, Walker et al. 2010).

Moreover, the timing of our analysis enables us to add to the literature regarding the role of economic development and technological innovations in food processing and packaging, on the rise of obesity rates. We investigate whether the introduction of cheaper and faster consumption of mass produced and processed food [3] such as fast foods contributed to the rising obesity epidemic which started taking place during the same time.

We test the robustness of our results in three ways which do not change our conclusions. First, we examine the possibility of reverse causality by testing whether fast food companies sited their outlets in more obesogenic areas. Secondly, we exploit a sudden increase in the opening of outlets from one company. In the years of 1977-1978, the fast food company, Wimpy, the largest of 4 companies in our data, opened 438 outlets all over the UK, constituting almost half of all fast food restaurants established by 1986. This sharp supply shock provides us with greater confidence in the near random exposure to fast food. Thirdly, we conduct an IV-analysis where we instrument the proximity to a fast food outlet with the proximity to a fast food distribution centre.

This chapter is organised as follows. Section 1.2 presents the background of the fast food market in the UK until 1986. In Section 1.4 we present our empirical framework and the data. Summary statistics are discussed in Section 1.3. The results are provided in Section 1.5. The robustness of our main results is tested and discussed in Section 1.6 and concluding remarks are reported in Section 1.7.

1.1 Previous literature

One of the first studies on the topic of fast food proximity and obesity in economics is Currie et al. (2010). The authors observe that having access to a fast food outlet within 0.1 of a mile of a school increases obesity rates by 0.52% among 9^{th} graders. Similarly, Davis & Carpenter (2009) find that adolescents which have a fast food outlet within 0.5 mile of their schools are more likely to be overweight compared to adolescents in schools without nearby outlets. British studies, such as Pieroni & Salmasi (2014) show that areas with a higher density of restaurants and lower prices of takeaway have a higher proportion of obese inhabitants. Burgoine et al. (2014) find that those who are most exposed to

[3] See Cutler et al. (2003) and Lakdawalla & Philipson (2009).

outlets offering takeaway in the commuting, home and work environment are estimated to have a 1.21 higher BMI compared to individuals who do not have such outlets in any of these environments.

These observational studies do not address the endogeneity problem stemming from the potential correlation between the location of fast food restaurants and unobserved obesogenic characteristics of the individuals residing nearby. This has motivated the use of instrumental variable (IV) estimation in later studies. Using the distance to the nearest major highway as an IV, Dunn (2010) finds a positive relationship between fast food proximity and BMI in women and in minority populations within counties of medium population density. However, the results do not necessarily hold for other sub populations in their study. The findings are comparable to Grier & Davis (2013) who observe stronger associations for the proximity between school and fast food outlets and BMI of black and hispanic students in schools located in low-income and urban areas, compared to other demographic groups.

On the other hand, Anderson & Matsa (2011) study a predominantly white and rural American sample. They use the distance from an individual's town to an interstate highway as an IV for the distance to fast food. The authors do not find a positive relationship between restaurant consumption and obesity. They suggest that the extra calories consumed from fast food are being offset by eating less energy dense food at home. Chen et al. (2013), use the amount of zoned non-residential land as an IV for access to fast food. After accounting for spatial dependence and heteroscedasticity across observations, the authors find that a lower access reduces BMI by a statistically significant but economically small amount. Alviola et al. (2014), instrument the distance from a school to the closest fast food restaurant with the distance to closest major highway to estimate the effect on the proportion of obese students from kindergarten throughout 10^{th} grade. They observe that the addition of a fast food outlet within 1 mile increases obesity levels by 1.23 percentage points, which supports the overall findings in Currie et al. (2010) and Davis & Carpenter (2009).

More recent literature evaluates natural experiments but do not find any effects of fast food access on obesity. Zhao et al. (2014) estimate the effects of "Moving to Opportunity", a public policy programme which randomly allocated housing vouchers for people to move out of areas with high poverty rates. The authors do not find that the changes in the availability of fast food were significant in explaining BMI. However, household's decision of moving into areas with high or low fast food availability is unlikely to be completely

random. Sturm & Hattori (2015) evaluate the Los Angeles Fast Food Ban, which restricted the openings of new fast food outlets in south Los Angeles. The authors show that obesity rates have in fact increased faster in areas covered by the ban. A potential caveat to this study is that fast food outlets were still able to open up in malls after the implementation of the ban.

1.2 Fast food in Great Britain 1968 - 1986

We define fast food outlets as restaurants which are open at any time of the day specialising in easily prepared processed foods that are served quickly, often using counter service. Following Dunn (2010) and Alviola et al. (2014), we focus on the biggest franchised "limited service" restaurants which also offered takeaway.[4] Four fast food franchises consisting of 952 addresses in the UK met this criterion: Burger King (11), KFC (82), McDonald's (230) and Wimpy (646).

We treat fish and chip shops as a constant background factor as they have a long history in the UK and its consumption was essentially constant during the time period of our study, see Figure A.1 in Appendix A.1.1. Fish and chip shops were not considered due to the nature of their operations: "sales are concentrated at particular times of day, early lunchtime and evening, often very late evening and do not normally operate outside those times.." They also engage in batch production as opposed to continuous production and do not usually employ seating (Sault et al. 2002). Moreover, teenagers consume typical fast food meals, such as burgers, more frequently than fish and chips (see Table A.2 in Appendix A.1.3. Figure 1.1 illustrates the rates of expansion of our four companies throughout time.

[4][..]In full service restaurants, the customer pays after eating. In limited-service restaurants, the customer pays before eating (Anderson & Matsa 2011).

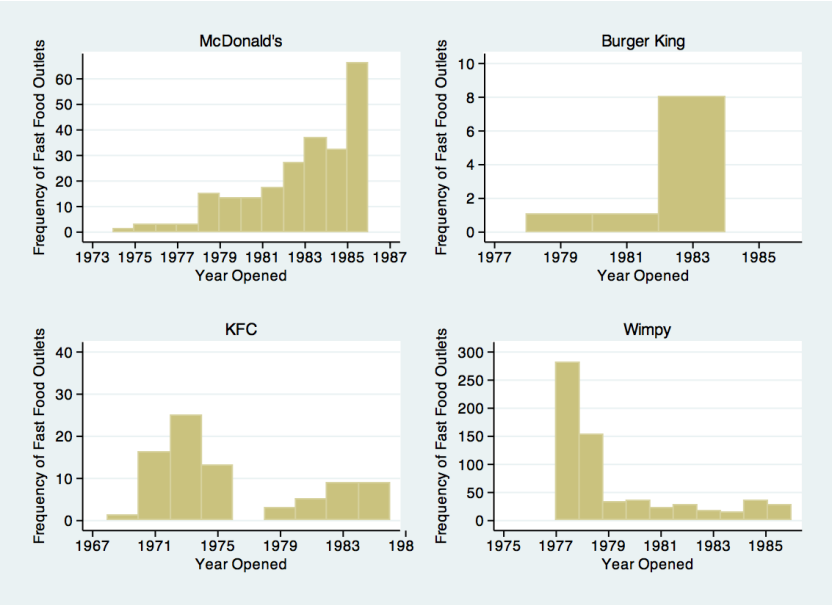


Figure 1.1: Opening of fast food outlets by company

As observed in Figure 1.1, KFC was the first fast food chain in the UK in 1968. The Figure also confirms the large market share of Wimpy and shows that Burger King was not a major contender during the time of our study. We observe a big jump in the openings of Wimpy outlets in 1977. The number of Wimpy restaurants decreased afterwards at the same time as McDonald's establishments started to increase.

Wimpy started off as a subsection in J. Lyon's restaurants in 1954. These restaurants were usually located in high end locations. From the 1960s and onwards, Wimpy became separate fast food restaurants. In 1977 Wimpy greatly increased its number of outlets and started to offer takeaway (Tassiopoulus 2008, pp. 92). The sudden supply shock of Wimpy was a result of the company's national site plan of opening up a restaurant in every British town with a population greater than 30,000 and in smaller towns with a high influx of tourists. Other determinants of fast food outlet location were the location of; pedestrian crossings, traffic lights, traffic counts, competitors and other stores and general spending power of the area (Voss et al. 1985, p. 255).

McDonald's concentrated their early expansion in Greater London as the firm's only distributor was based in outer London. Like other successful retail firms, opening decisions were made centrally and sequentially. McDonald's started from one location and expanded gradually to other large cities and towns. The franchise focused on already established shopping locations.[5] McDonald's became the dominant provider of fast food shortly after our time period of study, see Toivanen & Waterson (2011).

[5]During the time of the study there were no "drive-thrus.

We have not been able to collect information on possible closures of outlets. However, it does not appear that KFC, Burger King and McDonalds, closed any, or a substantial number of outlets during the time of our analysis. The start of Wimpy closures occurred mainly after the time period of our study. The fact that some Wimpy outlets might have closed at the time of the survey should not pose a significant threat to our analysis as we are assuming that fast food exposure has a cumulative effect on BMI. Please see Appendix A.1.1 for the methodology used to obtain the locations of the outlets along with an in depth description of the data.

According to Voss et al. (1985, p. 255), there was less abundant supply of cheap sites in European cities compared to American cities. Therefore, in conjunction with the discussed determinants of early fast food location, it is unlikely that any unobserved obesogenic household characteristics determine the location of fast food outlets up to 1986 to the same extent as they do today.

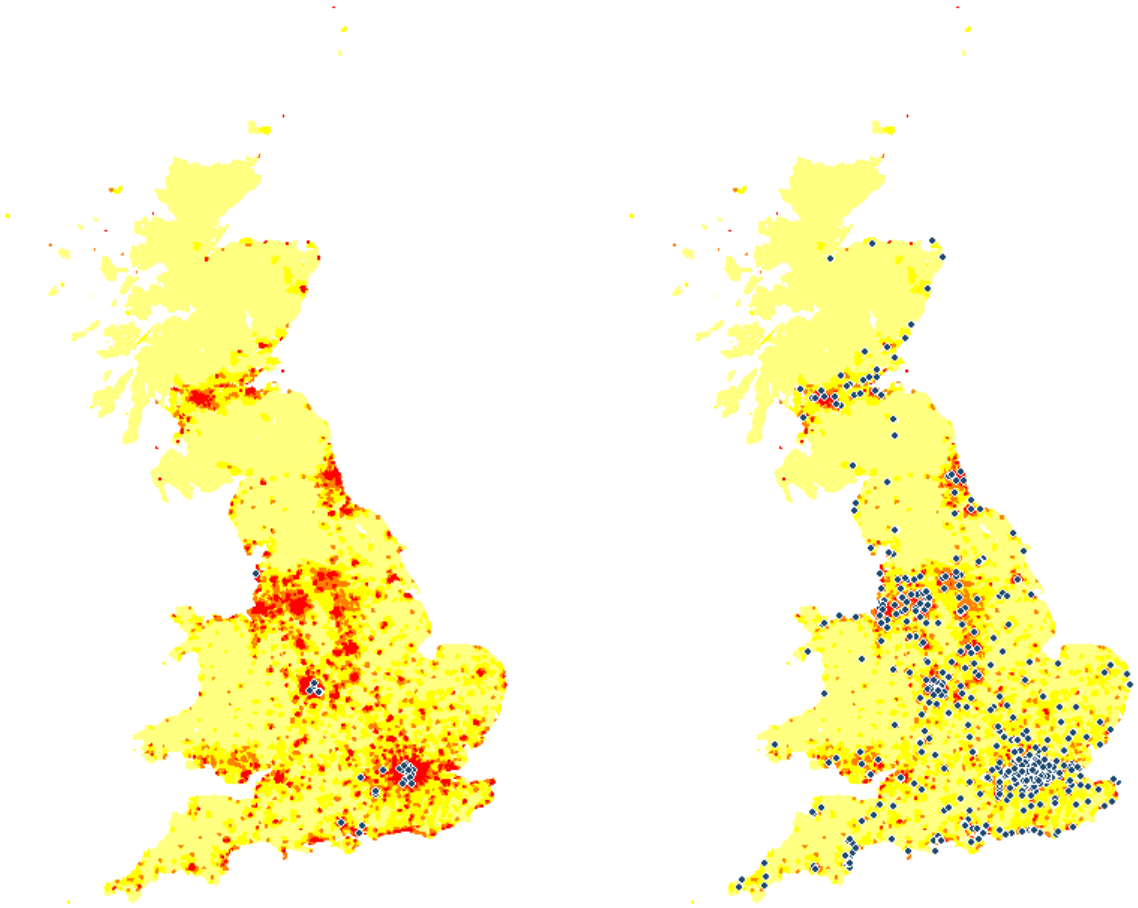
Another concern regarding the use of historical data to study the relationship between fast food and obesity, is the increase in fast food servings over time (Young & Nestle 2000). However, previous studies have found that the current global obesity epidemic can not be explained by growing portion sizes, and in particular, not by the larger fast food servings (Cutler et al. 2003).

Figure 1.2 illuminates the geographical and time dimensions of the entry process, from the first outlets established between 1968 and 1972, to 1986 - the year of the 16 year BCS follow-up survey. The locations of fast food outlets are depicted on heat maps. Yellow, orange and red areas indicate a low, medium and high population density, respectively. We observe that fast food outlets opened up in areas with a high population density.[6] One notices a dramatic increase in the access to fast food all over Great Britain. These key dates justify the emphasis on the time period prior to 1986 as it provides the study with considerable variation in the access to fast food, both spatially and temporally.

There is no evidence that the early influx of fast food restaurants in the UK was able to target households that are more obesity prone. The determinants of the location of establishment was central geography, location of competitors and other services, traffic and pedestrian flow and demographic and area characteristics such as purchasing power and proportion of young people, population size and population density (Toivanen & Waterson 2011). We return later to the potential question of whether fast food restaurants locate where those at risk for obesity reside, by explicitly examining the evidence of a potential

[6] A potential caveat to our analysis is that fast food density is correlated with population density. However, the literature on the effect on urban sprawl is mixed, see Zhao & Kaestner (2010).

reverse regression.



(a) 1972 (b) 1986

Figure 1.2: Fast food outlets in Great Britain 1972-1986.

These figures graph the location of fast food outlets through time. The blue dots illustrate the location of fast food outlets on heat maps representing the population density per ward according to the 1981 UK Census. Red areas denote the highest tertile of population per ward and yellow areas define the lowest population density per ward.

1.3 Data and descriptive statistics

We use individual panel data from the British Cohort Survey (BCS) to study the effects of fast food exposure on BMI. BMI is defined as an individual's weight in kilograms divided by one's height in metres squared (kg/m^2). The BCS surveyed all children born in England, Scotland and Wales in the week between the 5^{th} and 11^{th} of April 1970. The cohort has been followed up 6 times and we include outcomes from 1970, 1980 and 1986 in our regressions. The outcome variable, BMI, is derived from the respondents' weights

and heights measured by the school doctor at age 16. We also calculate BMI at age 10 and parental BMI from the survey wave of 1980.

The mean BMI is 21.275, which corresponds to a healthy body weight. Around 8.6% of our sample is considered to be obese and more than a fifth is overweight. More than one quarter of our sample is exposed to at least one fast food outlet within five miles radius of one's home. We also note that only 10.1% and 3.5% have access to an outlet within two miles and one mile, respectively. The average distance from one's home to the closest outlet is 4.445 miles. The average duration of one's closest fast food outlet in 1986 is 3.920 years. Even though most BCS respondents do not have a fast food outlet in their very near vicinity, they consume on average one takeaway meal per week.

Table 1.1: Descriptive statistics

Variable	Mean	SD	N
Body Mass Index at age 16	21.275	3.230	4,536
Proportion Obese	0.086	0.280	4,536
Proportion Overweight	0.217	0.412	4,536
Proportion with fast food within 5 miles	0.256	0.436	4,536
Proportion with fast food within 2 miles	0.101	0.301	4,536
Proportion with fast food within 1 mile	0.035	0.184	4,536
Distance to closest fast food outlet	4.445	6.417	4,536
Duration of closest fast food outlet in 1986	3.920	4.489	4,536
Intensity of fast food exposure	6.464	13.882	4,536
Takeaway per week	1.000	1.212	2,767
Household owns a Microwave	0.408	0.492	3,870
BMI at age 10	16.867	2.092	3,996
Mother's BMI	23.362	3.647	4,267
Father's BMI	24.453	2.973	4,083
Proportion of Smokers	0.110	0.313	4,536

1.4 Econometric specification

Shorter distances to fast food outlets can increase the demand for fast food due to lower travel costs (and opportunity cost) and due to supplier-induced demand (Jekanowski et al. 2001). Since fast foods can be addictive, it is likely that the introduction of fast food generates its own demand through taste formation. Taste formation means that past consumption has a reinforcing effect on the marginal utility of present and future consumption

(Stigler & Becker 1977). If preferences for fast foods induce taste formation, its introduction might cause weight gain over time. However, the relationship between the duration of fast food exposure and BMI is an empirical question. The temporal variation in fast food access in our data permits such an analysis. We write the basic relationship between fast food exposure and BMI for individual i with exposure to fast food restaurant j, as:

$$\mathrm{BMI}_{\mathrm{ij}} = \alpha_0 + \gamma Distance_{\mathrm{ij}} + \delta Duration_{\mathrm{ij}} + \theta(Distance_{\mathrm{ij}} * Duration_{\mathrm{ij}}) + \beta X_{\mathrm{ij}} + \mu_{\mathrm{ij}} (1)$$

The outcome variable, $\mathrm{BMI}_{\mathrm{ij}}$, is the BMI for individual i at age 16. Although there is substantial variation in individual body composition and other factors affecting comorbidities of obesity which are not captured by the given measure, BMI has been established as the best available proxy for an anthropometric estimate of adiposity (Hall & Cole 2006).

The explanatory parameters of interest are; *Distance*, or its reciprocal, measuring the distance (or alternative functional forms) from respondent i's house to the closest fast food restaurant in 1986, and *Duration*, which measures the effect the time since opening of the given fast food outlet. X is a vector of observable individual characteristics and μ is the error term clustered on Local Educational Areas (LEA).

As children spend more time at home than in school we follow the majority of studies by measuring the access to fast food in the home environment. During the time of our study a substantial proportion of school-aged children were entitled to free school meals, see Cobb et al. (2015) and von Hinke Kessler Scholder (2013).[7] Only 10.9% of the BCS respondents buy school lunches from outside, see Table A.2 in Appendix A.1.3. Additionally, we do not see that a lower average distance to fast food outlets per LEA increases the chance of buying lunch outside of school, see Table A.3 in Appendix A.1.3. It is therefore more likely that the food environment surrounding one's home, had a bigger impact on children's weights.

[7]The 1980's experienced a number of school reforms during the Thatcher government. In 1980 free school meals for all pupils were abolished. Fast foods started to be served in the school cafeteria as nutritional standards no longer had to be met. The reforms caused a drop in school meals consumed and an increase in the proportion of children bringing packed lunches from home. Despite the entry of unhealthy school lunches, previous research does not find that the reform affected children's weights. More importantly, as the reforms could have led to an increased demand for food outside the school cafeteria, such as fast foods, there is no evidence that the changes in the school food environment affected the weight status of children (von Hinke Kessler Scholder 2013).

θ is the coefficient on the interaction between distance and duration, denoted *Duration*(1/Distance*^{2}*)*. Allowing for the possibility of non-linear effects of fast food exposure on weight gain, we vary the functional form of both distance and duration.

We control for lagged BMI to account for differences in initial body weight. We add covariates for parental BMI, where the parent-offspring correlation, consisting of both genetic- and environmental traits, can be up to 80% Bray (2004). We also control for birth weight as many studies have established a positive relationship between birth weight and BMI later in life (Oken & Gillman 2003, Yu et al. 2011).

We control for gender due to biological differences in BMI and due to evidence of gender differences in the size of the geographic boundary used for leisure. Boys are more likely to cover larger distances in their free time compared to girls (Harrison et al. 2011).[8] Furthermore, we add ethnic group dummy variables as there is evidence of heterogeneity in BMI and in the share of body mass consisting of fat and lean tissue across different ethnic groups (Burkhauser & Cawley 2008).

Rennie & Jebb (2005), and descriptive evidence from all UK surveys [9] show that individuals from lower socio-economic classes are more likely to be obese. These findings are supported by a review carried out by Parsons et al. (1999) that finds that adolescents from lower socio-economic positions were more likely to be overweight in adulthood. Therefore we include controls for social class measured by parental occupation.

Moreover, we control for cigarette smoking as smokers tend to have a higher metabolic rate and are likely to consume fewer calories in comparison to non-smokers (Chou et al. 2004). As there are large differences in obesity rates across both UK countries and regions (Hawkins et al. 2007), we include covariates for residing in London, in an inner urban area and for land of residence (England, Scotland or Wales).

There was a rapid rate of technological progress in food production in 1970-80 resulting in reduced costs of energy-dense and processed foods. In considering the effect of fast food availability on BMI, a potential confounding variable may be the consumption of other high-caloric convenience foods. Previous literature suggests that the introduction of microwaves was associated with increasing obesity rates (Finkelstein & Strombotne 2010). Therefore, we include a dummy variable for household ownership of a microwave to control for the consumption of other processed food. See Appendix A.1.2 for a list and

[8] We can not estimate the regressions separately for girls and boys due to the small sample size.

[9] Such as: the Health Survey for England series (1993-2002), the Scottish Health Surveys (1995, 1998 and 2001), the Health in Wales Survey (1985-96), Welsh Health Survey (1995, 1998) and the National Diet and Nutrition Surveys (1992, 1994, 1997, 2000).

explanation of all included variables.

The 1986 BCS contains self reported information on food intake and on sports habits of the adolescents in 1986. In addition, the survey also contains the mothers' reports of the adolescents' eating habits. After extensive examination of this data, we have not included the self reported energy accounting variables in our analysis as we judge the data to contain measurement error and its inclusion heavily reduces our sample size.

Next we specify an econometric model which measures the impact of the intensity of fast food exposure. We construct *Intensity* by taking the sum of the durations since the time of the establishment of the closest 20 fast food outlets, divided by their respective distances to one's home.

$$Intensity = \sum_{\forall j:\, d \leq 5} \frac{Duration_j}{Distance_j}$$

We estimate Equation (2), as set out below, in similarity with Equation (1), but our parameter of interest is now the coefficient on *Intensity*.

$$\mathrm{BMI_{ij}} = \alpha_0 + \beta X_{\mathrm{ij}} + \theta Intensity_{\mathrm{ij}} + \mu_{\mathrm{ij}} \tag{2}$$

1.5 Results

We begin by estimating the determinants of BMI at age 16 in 1986. The regression results are presented in Table 1.2. Throughout all specifications, we find that BMI at age 10 explains around 80% of BMI at age 16. We, fail to find support of an inverse relationship between BMI and social class. Even though the sample size decreases heavily when we include all control variables in column 6, we note that lagged BMI, gender, parental BMI and household ownership of microwave are statistically significant in predicting current BMI.

In order to confirm that the determinants of BMI also predict the probability of reaching an unhealthy weight, we estimate the same specifications with the outcomes being the probability of being overweight and obese. Children and adolescents with BMI at, or above the 85^{th} percentile, are considered overweight, and those with BMI above the 95^{th} percentile are defined as obese (Barlow & Dietz 1998). See Appendix A.1.6 for an explanation of how the BMI cutoffs were measured.

When controlling for all covariates, we find that lagged BMI, parental BMI and mi-

crowave ownership are statistically significant predictors the probability of being overweight. Additionally, the risk for overweight increases with a reduced socio-economic status and decreases with smoking status, see Table A.13 in Appendix A.1.6. When estimating the effects on the probability of being obese, we note that Asian ethnicity has a negative effect, see Table A.14 in Appendix A.1.6. However, we do not detect a socio-economic gradient in obesity. This is in line with Parsons et al. (1999) who do not find a consistent relationship between socio-economic status and childhood fatness also using the 1970 BCS.

Table 1.2: Determinants of BMI

	(1)	(2)	(3)	(4)	(5)	(6)
	BMI	BMI	BMI	BMI	BMI	BMI
BMI at age 10	0.844***	0.839***	0.796***	0.795***	0.795***	0.791***
	(0.026)	(0.026)	(0.027)	(0.029)	(0.029)	(0.032)
Girl		0.523***	0.511***	0.519***	0.520***	0.473***
		(0.077)	(0.081)	(0.085)	(0.085)	(0.093)
Father's BMI			0.094***	0.089***	0.089***	0.093***
			(0.017)	(0.017)	(0.017)	(0.019)
Mother's BMI			0.076***	0.074***	0.074***	0.069***
			(0.015)	(0.015)	(0.015)	(0.016)
Asian				-0.008	0.021	-0.081
				(0.359)	(0.372)	(0.269)
West Indian				-0.281	-0.259	-0.323
				(0.319)	(0.324)	(0.232)
Other Ethnicity				-0.933	-0.908	-0.655
				(0.699)	(0.702)	(0.772)
Social Class II				0.012	0.015	0.035
				(0.184)	(0.186)	(0.196)
Social Class III				0.156	0.161	0.193
				(0.165)	(0.166)	(0.180)
Social Class IV				0.047	0.052	0.099
				(0.188)	(0.190)	(0.213)
Social Class V				0.231	0.235	0.326
				(0.214)	(0.214)	(0.246)
Other Social Class				0.084	0.091	0.044
				(0.340)	(0.341)	(0.390)
Urban					-0.055	-0.057
					(0.118)	(0.118)
London					0.018	0.002
					(0.310)	(0.307)
Scotland					-0.022	-0.063
					(0.170)	(0.163)
Wales					0.047	0.080
					(0.130)	(0.125)
HH owns Microwave						0.425***
						(0.103)
Smoker						-0.015
						(0.159)
Birth Weight						0.005
						(0.099)
Constant	7.007***	6.824***	3.504***	3.555***	3.573***	3.452***
	(0.433)	(0.429)	(0.554)	(0.603)	(0.601)	(0.613)
Observations	3996	3996	3683	3531	3531	3093
R^2	0.318	0.325	0.344	0.345	0.345	0.341

Standard errors clustered on LEA. Dependent variable is BMI at age 16 for all who remained in their LEA.

* $p < .10$, ** $p < .05$, *** $p < .01$

1.5.1 Distance analysis

We estimate the effect of distance to one's closest fast food outlet in 1986 on BMI at age 16. The regression results from various measures of continuous distances and distance bins of 0.5, 1, 2 and 5 miles are reported in Table 1.3 and in Table 1.4, respectively.[10]

Table 1.3: Effect of fast food proximity on BMI: Continuous distance

	BMI							
Distance to nearest fast food outlet	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Distance	0.005	-0.004	0.034**	-0.000				
	(0.010)	(0.006)	(0.016)	(0.013)				
Distance^2			-0.005**	-0.001				
			(0.002)	(0.158)				
$(1/Distance)/10^8$					-0.015	-0.080**	-0.060	-0.117
					(0.045)	(0.033)	(0.075)	(0.073)
1/ Distance ^{2}							0.000	0.000
							(0.000)	(0.000)
Observations	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.002	0.341	0.000	0.342	0.000	0.342

Notes: Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4, 6 and 8: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

[10] We cannot use closer distance bins as a very low fraction of the BCS respondents had a fast food outlet within their immediate vicinity in 1986. Applying the distance bins used in (Currie et al. 2010) we note that only 0.06% had a fast food outlet within 0.1 miles.

Table 1.4: Effect of fast food proximity on BMI: Distance bins

	BMI									
Distance to nearest fast food outlet	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Fast food outlet $\leq$ 0.5 mile	0.099	-0.352							0.090	-0.259
	(0.564)	(0.345)							(0.651)	(0.424)
Fast food outlet $\leq$ 1 mile			0.038	-0.156					-0.103	-0.218
			(0.271)	(0.256)					(0.350)	(0.324)
Fast food $\leq$ 2 miles					0.095	0.056			0.173	0.070
					(0.170)	(0.173)			(0.217)	(0.235)
Fast food $\leq$ 5 miles							-0.006	0.094	-0.064	0.099
							(0.114)	(0.125)	(0.125)	(0.149)
Observations	4536	3093	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341	0.000	0.341	0.000	0.341

Notes: Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4, 6, 8 and 10: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

From column (1) and (2) in Table 1.3 we observe small and insignificant coefficients on the distance to one's closes fast food outlet on BMI. Neither, does the inclusion of $Distance^2$ in column (3) and (4) suggest that proximity to fast food increases BMI. A potential concern is that our small sample size may not allow us to detect the true effect of fast food exposure on BMI given the small effect size found in previous papers.

However, it should be noted that the effect found in some previous papers, such as Currie et al. (2010), might be underestimated due to the short time of exposure to the given fast food outlets used in their analysis. Therefore, it is difficult to estimate what the true effect of fast food proximity would be, taking the duration of fast food exposure into account. As an attempt to circumvent the issue of power, we measure distance as 1/Distance and 1/Distance^2. We divide 1/Distance by 10^8 due to the very small effect size. Irrespective of how we define distance, we do not observe that living closer to a fast food outlet is associated with a higher BMI.

From Table 1.4, we note rather fluctuating and statistically insignificant estimates on the effect of being exposed to fast food outlets within different distance bins. To allow for a calculation of the cumulative effect of having a fast food restaurant within 0.5 miles relative to the case where there is no fast food restaurant within at least 2 miles, we include all distance dummies in the same regression in columns (7) and (8). Again, the estimates are not statistically significant.

Neither do we find that weight gain between 1980 and 1986 is associated with the introduction of a fast food outlet within 1, 3 and 5 miles radius of one's home in comparison

to those who never gained nearby access. The regression results are presented in Table A.6 in Appendix A.1.4.

We do find some support for the proximity to fast food increasing the consumption of takeaway. The regression results from estimating the impact of distance to one's closest fast food outlet on take-away consumption, are presented in Table 1.5. A reduction of 1 mile to one's closest fast food outlet is associated with an increase in the frequency of takeaway meals by 0.013 per week, see Panel A in Table 1.5. However, we do not find any positive effects of residing within closer distance bins compared to further away, see Panel B in Table 1.5. Viner & Cole (2006) find that eating takeaway meals twice or more per week and consuming two or more carbonated drinks per day was associated with an increase in BMI Z-score between 16 and 30 years of the BCS respondents. The lack of an impact on BMI in this study can potentially be explained by the very small increase, if any, in takeaway consumption.

Table 1.5: Effect of fast food proximity on takeaway consumption

The effect of fast food proximity on takeaway consumption								
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Continuous Distance								
Distance	-0.006	-0.013**	-0.002	0.005				
	(0.004)	(0.005)	(0.007)	(0.017)				
$Distance^2$			-0.001	-0.009				
			(0.001)	(0.008)				
$1/Distance$					-0.042**	-0.020	-0.025	-0.007
					(0.021)	(0.025)	(0.036)	(0.051)
$1/Distance^2$							-0.000	-0.000
							(0.000)	(0.000)
Observations	2767	1975	2767	1975	2767	1975	2767	1975
McFadden's Pseudo R^2	0.000	0.024	0.000	0.025	0.001	0.023	0.001	0.023
Panel B: Distance Bins								
Fast food outlet $\leq$ 0.5 mile	-0.427**	-0.616*						
	(0.200)	(0.316)						
Fast food outlet $\leq$ 1 mile			-0.193	-0.131				
			(0.138)	(0.187)				
Fast food $\leq$ 2 miles					-0.077	-0.040		
					(0.075)	(0.089)		
Fast food $\leq$ 5 miles							0.007	0.007
							(0.058)	(0.069)
Observations	2767	1975	2767	1975	2767	1975	2767	1975
McFadden's Pseudo R^2	0.000	0.024	0.000	0.023	0.000	0.023	0.000	0.023

Dependent variable is frequency of takeaway per week. All models are estimated using a negative binomial regression. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4, 6 and 8: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

1.5.2 Duration of exposure

We investigate the effect of the duration of fast food exposure by regressing BMI on the number of years since the opening of the first fast food outlet within: 1, 3 and 5 miles radius of the respondent's home. The regression results are displayed in Table 1.6. The estimates on duration are very close to zero and do not obtain statistical significance in any given boundary.

Table 1.6: Effect of duration of fast food exposure on BMI

	BMI					
Duration of first fast food outlet within:	(1)	(2)	(3)	(4)	(5)	(6)
$\leq$ 1 mile	0.005	-0.002				
	(0.017)	(0.020)				
$\leq$ 3 miles			0.006	0.007		
			(0.012)	(0.012)		
$\leq$ 5 miles					0.003	0.006
					(0.013)	(0.012)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341

Notes: Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

We have assumed that the longer a fast food outlet has been near an individual's home, the higher the likelihood of weight gain. However, there is no consensus in the literature regarding the habit formation of foods away from home (Dynan 2000).[11] Habit formation predicts that past consumption has a satiating effect which reduces the marginal utility of current consumption, see Carroll et al. (2000). Consumers might experience temporal satiation where one is induced to seek variety and substitute to other food alternatives over time.

There is also the possibility of novelty seeking behaviour where there is a high demand during the introduction of a new good followed by lower demand with time (Hirschman 1980). Therefore, the cumulative effect on weight gain as a function of the duration of fast food exposure is not necessarily positive. Moreover, there might be non-linear demand for fast food as a function of time since the establishment due to waves of heavy marketing, whereas previous research has shown that fast food advertisement increases the demand for fast food (Andreyeva et al. 2011). Other trends in the demand for fast food could originate from increased health awareness stemming from public health campaigns (Becker et al. 1990).

Assuming that the introduction of fast food induced habit formation, fast food consumption would be higher for those who were newly introduced to fast food and then fade

[11] Early evidence from the US concluded that, at best, the evidence for habit formation in food consumption is weak.

away over time. We test whether newer fast food outlets have a positive effect on body weight. We regress BMI on a dummy variable taking value 1 if the respondent received his or her closest fast food outlet within a radius of 1, 3 and 5 miles, and 0 otherwise, in the last 1, 2 or 3 years, respectively. We do not find that newer outlets have a positive effect on BMI, see Table A.5 in Appendix A.1.4.

1.5.3 Interaction analysis

We estimate the impact of fast food exposure on BMI as specified in Equation 1. From Table 1.7 we do not observe that the interaction of $1/Distance^2$ and duration of fast food point to a positive and statistically significant association with adolescent BMI. As the respondent's closest fast food outlet in 1986 was not necessary always one's closest outlet, we also estimate the specification for the individual's first fast food outlet within five miles of home. Similarly, we do not find a positive effect of fast food exposure on adolescent BMI, see Table A.7 below.

Table 1.7: Effect of the interaction of distance and duration of fast food access on BMI

	BMI					
Exposure to nearest fast food outlet	(1)	(2)	(3)	(4)	(5)	(6)
Duration of nearest fast food outlet	0.006	0.007	0.006	0.008	0.006	0.008
	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)	(0.010)
$1/(Distance^2/10^8)$			0.000	-0.004**	0.000	-0.005*
			(0.000)	(0.002)	(0.000)	(0.003)
$Duration * 1/(Distance^2/10^8)$					-0.001	0.002
					(0.004)	(0.004)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.342	0.000	0.342

Notes: Dependent variable is BMI. Analysis carried out for the sample of adolescents who remained in their LEA from age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

1.5.4 Intensity results

Next, we present the regression results from Equation (2) where we estimate the aggregate effect of the proximity and duration of up to 20 closest fast food outlets on BMI. As the importance of such characteristics for demand might vary with the order of proximity

to one's home, we also create a weighted *Intensity* measure by allocating more weight to closer fast food outlets. Additionally, we take the natural logarithm of the intensity measure. However, no matter how we modify the intensity of fast food "treatment", we fail to find support for a positive effect of fast food exposure on BMI, see Table 1.8.

Table 1.8: Effect of fast food intensity on BMI

	BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
Intensity of fast food	0.002	-0.003				
	(0.004)	(0.004)				
Weighted Intensity of fast food			0.001	-0.002		
			(0.002)	(0.002)		
Ln(Intensity) of fast food					-0.017	-0.032
					(0.062)	(0.067)
Observations	4536	3093	4536	3093	2407	1617
R^2	0.000	0.341	0.000	0.341	0.000	0.347

Notes: Standard errors clustered on LEA in parentheses. Analysis carried out for the pooled sample of adolescents who remained in their LEA from age 10. The following set of control variables is included in specifications 2, 4 and 6 gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. * $p < .10$, ** $p < .05$, *** $p < .01$.

1.6 Threats to identification: Robustness analysis

Our main analysis uses information on the distance to all fast food outlets. One might argue that very far away restaurants are less likely to have an impact on the demand for fast food. Restricting the analytical sample to consist of outlets within five miles only does not change our conclusions, see Appendix A.1.4.

We have also re-estimated our main specifications changing the outcome variables to the probability of being overweight or obese. The results on the effect of fast food proximity on the probability of being overweight and obese are qualitatively similar, see Appendix A.1.6.

Additionally, we investigate whether fast food exposure in one's school area affects BMI. As we do not have information on the location of schools in the BCS, we regress BMI on the average distance to one's closest fast food outlet per LEA. A lower average distance to fast food per LEA does not increase BMI, see Table A.4 in Appendix A.1.4. Moreover, there is no evidence of fast food exposure affecting the BCS respondents' BMI at age 10 or their parents' BMI in 1980, see Table A.20 in Appendix A.1.7.[12]

1.6.1 Predictors of fast food density

Some previous papers which use current data on fast food establishments, such as Swinburn et al. (2011) have found a relationship between constituents of an obesogenic environment and fast food location. An obesogenic environment is described as "..the sum of influences that the surroundings, opportunities or conditions of life have on promoting obesity in individuals or populations" (Swinburn et al. 2011)

Conceivably, fast food companies might locate themselves near households with strong preferences for fast food and who are less concerned with dietary health. Moreover, a high density of fast food outlets might be more prevalent in a built environment providing little opportunity to exercise (Papas et al. 2007). Reverse causality could constitute another problem if parents of overweight children choose to reside where fast food restaurants proliferate (Anderson et al. 2003). On the other hand, restaurants might target consumers with a high opportunity cost of domestic food preparation which therefore might bias our estimates of fast food proximity on BMI downwards (Anderson et al. 2003).

Therefore, we check the robustness further. We first consider whether fast food companies were deliberately targeting more obesity prone households. We regress exposure to a fast food outlet within 1, 3 and 5 miles after 1980 on the BMI of the BCS respondents

[12] Parental information on weight is only found in the 1980 wave of the BCS.

and their parents in 1980. Our results do not show that fast food outlets were targeting households with a lower concern of maintaining a healthy body weight, see Table 1.9 below.

Table 1.9: Reverse causality: The effect of child and adult BMI in 1980 on fast food proximity in 1986.

	1 mile		3 miles		5 miles	
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Child BMI						
BMI at age 10	0.001	0.001	-0.000	0.002	-0.000	0.002
	(0.002)	(0.003)	(0.003)	(0.004)	(0.003)	(0.005)
Control Variables		✓		✓		✓
Observations	5144	1889	5144	1889	5144	1889
R^2	0.000	0.158	0.000	0.256	0.000	0.282
Panel B: Parental BMI						
Sum of parental BMI	0.000	0.002	0.000	0.003	0.000	0.003
	(0.001)	(0.002)	(0.001)	(0.002)	(0.001)	(0.002)
Control Variables		✓		✓		✓
Observations	5285	1976	5285	1976	5285	1976
R^2	0.000	0.151	0.000	0.287	0.000	0.287

Dependent variable is obtaining a fast food outlet after 1980 within 1 mile (column 1 and 2), within 3 miles (column 3 and 4) and within 5 miles (column 5 and 6). The following control variables are included in columns 2, 4 and 6: ethnicity, social class and location. Standard errors clusters on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Neither do we observe a relationship between less or more healthy Local Authorities (LA) measured by smoking rates, frequency of sport participation, socio-economic status and satisfaction with sporting facilities and the density of fast food outlets in 1974 using data from the National Child Development Study (see Table A.21 in Appendix A.1.7). We do observe a negative relationship between the proportion of fathers of low social class and early school leaving fathers per LA and fast food density in 1974 (see Table A.22 in Appendix A.1.7).

Most studies from high-income countries find a negative social gradient in obesity prevalence (McLaren 2007). Several papers also find that more deprived areas have a higher density of fast food outlets compared to less deprived areas (Conrad & Capewell 2012, Morland et al. 2002). We analyse the relationship between area level deprivation

and the density of fast food outlets in 1981. Deprivation is measured by the Townsend index which is a composite score of standardised deprivation proxies per geographical area (Townsend et al. 1987). The following four area characteristics have been computed, standardised and aggregated per ward as of the 1981 UK Census; percentage of unemployed individuals over the age of 16, percentage of households that are overcrowded[13], percentage of households that do not own their home, and the percentage of households without access to a car.

We estimate a negative binomial regression due to most wards not having any fast food outlets. The outcome variable is the count of fast food outlets per ward with an offset of the log of population/10,000 per ward.[14] Wards in the 4^{th} and 5^{th} quintiles of deprivation are not more likely to have more fast food outlets than the least deprived wards after controlling for the proportion of youth, immigrants and retirees, see Table 1.10 below.

[13]Overcrowding is defined by greater than or equal to 1.5 persons per room.

[14]We follow Anderson & Matsa (2011) where fast food density is defined as the count of outlets per 1000 inhabitants. We measure fast food outlets per 10,000 inhabitants per ward as we observe fewer outlets per capita in our study.

Table 1.10: Predictors of fast food density

Dependent Variable: Fast Food Outlets/10 000 individuals/Ward		
	(1)	(2)
Quintile of Deprivation II	0.659***	0.431**
	(0.194)	(0.198)
Quintile of Deprivation III	0.667***	0.388**
	(0.174)	(0.176)
Quintile of Deprivation IV	0.579***	0.186
	(0.175)	(0.178)
Quintile of Deprivation V	0.631***	0.142
	(0.194)	(0.190)
Area of ward in km^2	-0.055***	-0.047***
	(0.010)	(0.009)
Proportion of Youth	8.920***	17.346***
	(2.543)	(2.205)
Proportion of Immigrants	5.089***	4.455***
	(0.639)	(0.660)
Proportion of Retirees		17.125***
		(1.326)
Constant	1.089	0.853
	(0.126)	(0.134)
Observations	8578	8578
Pseudo R^2	0.0797	0.1068

Notes: Data Source: 1981 UK Census. Dependent variable is the count of fast food outlets with log of population/10 000/wards as offset. Standard errors are clustered on wards and shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Moreover, we do not find that areas with a higher unemployment claimant rate have more fast food outlets (see Table A.23 in Appendix A.1.7. Additionally, we regress the average intensity of fast food exposure per LA on several characteristics found in all the Youth Cohort Surveys 1-7 over the period of 1983-1992. From Table A.24 in Appendix A.1.7, we observe that fast food restaurants located in LAs with a higher educational score, truancy, more one-parent families and a higher proportion of non-white residents.

In light of the area level analysis of the determinants of fast food density up to 1986, we do not find robust and systematic evidence of fast food companies targeting certain social groups or areas with less or more healthy inhabitants.

1.6.2 Wimpy analysis

As observed in Section 1.2, the biggest fast food company in terms of market share prior to 1986, Wimpy, followed a different expansion path than the other companies. Figure 1.1 shows that there was a dramatic increase of Wimpy restaurants in 1977 and 1978. The vast majority of the Wimpy restaurants opening up in these years were new restaurants as the company was overtaken by United Biscuits in 1977. Plotting the location of Wimpy outlets across the UK in 1978, shown in Figure 1.3, we see a greater geographic dispersion in the supply of Wimpy outlets during these two years compared to other fast food brands.

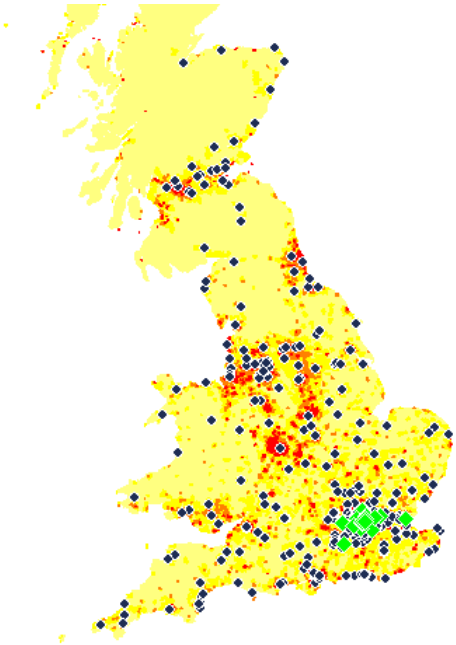


Figure 1.3: Fast food outlets established in 1977 and 1978

The map is a heatmap of Great Britain with population densities per wards in 1981. The red areas are the most densely populated areas while yellow areas are the least densely populated areas. The blue circles denote Wimpy outlets and the green diamonds are the locations of McDonald's outlets.

The fast expansion over the country reflected the company management's aim to compete with the arrival of McDonald's. The quick increase in Wimpy outlets did not allow for a strategic targeting of more obesity prone households. Therefore, we estimate the impact of distance to Wimpy outlets as the scope for non-random treatment in the exposure to this particular fast food company is minimized. We do not observe that living closer to a Wimpy restaurant has a positive effect on BMI at age 16 using various continuous distance measures, see Table A.25, or distance bins, see Table A.27 in Appendix A.1.7.

1.6.3 IV analysis.

Lastly, we follow the literature on the industrial organization of Wal-Mart (see Holmes (2011), Courtemanche & Carden (2011), Neumark et al. (2008)) and instrument for the location of a fast food outlet with the distance to its distribution centre. The location of a major distributor is likely to influence the location of a fast food outlet as it determines the distribution costs in terms of a driver's time, cost of transportation, inventory feedback and variable cost (Holmes 2011).

Previous research on the fast food market in the UK prior to the 1990's supports the validity of our proposed instrument. Toivanen & Waterson (2011) conclude that the spread of fast food outlets in the early era in the UK was restricted to the proximity of their distributors. The authors explain the higher density of McDonald's outlets in London by the fact that the company's only distributor at this time was located in North West London. McDonald's, as other successful retail firms, start from one location and expand organically, whereas the expansion takes time and opening decisions are made centrally. Toivanen & Waterson (2011) write "They do not open immediately even in markets that must have seemed profitable to enter right away. In the case we study, it took McD, a very large firm with lots of experience in opening outlets by the time it established itself in the UK in 1974, 7 years to reach the 2nd largest city in the UK, only a little over 100 miles from its first store. Service firms seem also to expand round their existing outlets."

Wimpy, on the other hand, had multiple distribution centres which have been supplying to J Lyon's restaurants for many years.[15] See Appendix A.1.1 for more information on the fast food distribution centre data. A map of the location of distribution centres and fast food outlets in 1986 is shown in Figure A.2 in Appendix A.1.7.

There were only three McDonald's distribution centres prior to 1986 and all opened in 1977-1982 and were located in North Outer London. Therefore it is unlikely that the location of both Wimpy's and McDonald's distribution centres were correlated with unobserved tastes of residents or obesogeneic traits of certain areas at a later time period.

The following two step model is estimated. We regress the distance from the BCS respondent's home to its closest fast food distributor in the first stage as specified in Equation 4. Subsequently, we estimate BMI using the instrumented distance to one's closest fast food outlet in the second stage, see Equation 5 below.

$$DistFF_{\mathrm{ijt}} = \alpha_0 + \alpha_1 DistDistr_{\mathrm{ij}} + \alpha_2 X_{\mathrm{ij}} + \mu_{\mathrm{ij}} \tag{4}$$

[15] No information could be accessed regarding the distributors for KFC and Burger King during the relevant time period.

$$BMI_{ij} = \beta_0 + \beta_1 DistFF_{ij} + \beta_2 X_{ij} + \mu_{ij} \quad (5)$$

We define *DistDistr* as a continuous distance variable analogous to Courtemanche & Carden (2011) and Neumark et al. (2008), we also define the distance from one's house to a distributor using distance bins. We specify the distance from an individual's home to a fast food distribution centre in three ways. First as $1/Distance$ and then as two binary variables which take value 1 if the respondent has at least one fast food distribution centre within 50 or 15 miles of one's home, respectively, and 0 otherwise. The first stage point to that all the distance measures are relevant with F-statistics exceeding the rule-of-thumb value of 10, see Table 1.11.

Table 1.11: First stage: The effect of distance to a fast food distribution centre on the distance to one's closest fast food outlet

	Distance to fast food					
	(1)	(2)	(3)	(4)	(5)	(6)
$1/Distance$ to closest distribution centre	-21.357***	-14.667***				
	(5.279)	(3.681)				
Distance $\leq$ 50 miles to closest distribution centre			-2.996***	-1.980***		
			(0.411)	(0.358)		
Distance $\leq$ 15 miles to closest distribution centre					-3.013***	-1.696***
					(0.371)	(0.333)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.018	0.074	0.037	0.080	0.010	0.070
F-statistic	16.36	15.88	53.12	30.54	66.01	25.90
Prob > F	0.0001	0.0001	0.0000	0.0000	0.0000	0.0000

Notes: Dependent variable is distance to one's closest fast food outlet. The sample consists of 16 year olds who did not change LEA since age 10 and who have non-missing information on weight and height. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

The regression results from the second stage are presented in Table 1.12. The coefficients on the instrumented fast food exposure on BMI are never statistically significantly different from zero.

Table 1.12: IV results: The effect of fast food proximity on BMI

	BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
Distance to closest fast food outlet	-0.046	-0.039	0.069	0.049	-0.125	-0.166
	(0.070)	(0.044)	(0.049)	(0.052)	(0.078)	(0.129)
Observations	4536	3093	4536	3093	4536	3093
R^2		0.336	.	0.330	.	0.237

Notes: Dependent variable is BMI. Distance to closest fast food outlet is instrumented with $1/Distance$ to closest distribution centre in column (1), having a distribution centre $\leq$ 50 miles in column (2) and having a distribution centre $\leq$ 15 miles in column (3). The sample consists of 16 year olds who did not change LEA since age 10 and who have non-missing information on weight and height. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$. R^2 for columns (1), (3) and (5) is negative, meaning the model sum of squares is negative. STATA's ivregress command suppresses the printing of a negative R^2 which is why the values are not reported in the table.

1.7 Conclusion

This paper have studied the relationship between the exposure to fast food and adolescent BMI using historical data relating to the inception of fast food in Great Britain. The data on the timing of establishment and location of all fast food outlets prior to 1986 allowed us to investigate whether distance affects BMI. This paper has filled a gap in the existing literature which has mostly focused on the distance to ever-present fast food restaurants using cross-sectional data from the US. We do not find any evidence of a positive association between numerous measures of fast food exposure and adolescent BMI and our conclusions hold after conducting a battery of robustness checks. The lack of a relationship is supported by previous research, such as; Anderson & Matsa (2011), Fraser et al. (2012), Crawford et al. (2008), Lee (2012).

To assess whether fast food treatment was indeed "as if" randomly assigned, we have investigated the determinants of fast food location throughout the time period of our study using several other datasets. We do not find consistent area level determinants of fast food density during the period of study. One fast food company, Wimpy, suddenly increased its supply of fast food outlets, not allowing for a strategic location in obesogenic areas of residence. Restricting the proximity analysis to estimating the effect of distance to Wimpy outlets on BMI, confirms the zero results from our previous analysis. Moreover,

our results are robust to an IV analysis where the distance to the closest fast food outlet is instrumented with the distance to a fast food distribution centre.

There are several potential explanations why we do not find an effect of fast food exposure on BMI.

Firstly, the effect of fast food proximity on obesity rates may be highly context-, or country specific, see Dunn (2010), Dunn et al. (2012), Grier & Davis (2013), Anderson & Matsa (2011). Most of the studies in this literature study current obesity rates in the US where fast food is eaten several times a week. The majority of British children and adolescents consumed (and are still consuming) fast food between once per week to once a month (Fraser et al. 2012). Another explanation might be that the overall obesogenic built environment is more correlated with the location of obesity prone sub-populations in the US compared to the UK (Walker et al. 2010).

Positive effects have been found for suburban females and non-whites, see Dunn (2010), Dunn et al. (2012), urban youth with a high proportion of ethnic minorities, see Currie et al. (2010), Grier & Davis (2013) and youth living in the poorest and one of the least healthy states, see Alviola et al. (2014) but not for rural whites in Anderson & Matsa (2011), Dunn et al. (2012). Our sample of adolescents in the UK resembles the less diverse subsamples in rural or suburban areas in the US analysed in previous papers which do not find an effect of fast food access on obesity rates. The population studied in this paper might not have a propensity to gain weight if exposed to fast food.

Another reason for why we do not find a positive relationship between fast food access and BMI is that adolescents may have less control over food choices in their home environment compared to in their school environment. Alternatively, the time period of our study may not translate to large effects on obesity as only a small proportion of our sample was exposed to fast food very near their home.

Additionally, fast foods are relatively cheaper than other foods today (Wiggins et al. 2015). Some papers, such as Lakdawalla & Philipson (2009) argue that the current obesity epidemic is due to low current relative fast food prices. Undoubtedly, fast food prices were higher in the early era of fast food inception compared to today. In 1974 a Big Mac was 45.8% more expensive than a Big Mac today. However, the price of Big Mac in 1986 was only 6.6% more expensive then today.[16]

Despite the caveats of our analysis, and irrespective of how we define fast food access,

[16]The price of a Big Mac in the UK in 2016 was £2.69 (*The Big Mac Index* 2015). A Big Mac in 1974 was 45 pence which in real 2016 prices is equivalent to £4.19 , and the price of a Big Mac in 1986 was was £1.10 which is equivalent to £2.88 (Lynch 2015, *The Big Mac Index* 2015, Officer & Williamson 2016).

there seems to have been very little scope for the introduction of fast food to nudge a sufficient behavioural change resulting in weight gain amongst adolescents in the UK in the 1980s. Evidence from Griffith et al. (2016) showing that there has been a decrease in total calories purchased since the 1980's support our overall findings. Thus, we suggest that it is unlikely that the prevalence and proximity of fast food outlets caused the obesity pandemic.

Evaluating bans on the expansion of fast food companies show that it is practically very difficult to implement such laws that will result in curbing childhood obesity. For example, the "South LA fast food ban" did not result in lower obesity levels for the affected area (Sturm & Hattori 2015). Moreover, recent evidence investigating the effects of the adoption of the "Commonsense Consumption Acts" where individuals cannot hold fast food companies accountable for their weight gain, and thus incentivize individuals to take more personal responsibility of their fast food consumption, did not result in lower prevalence of obesity (Carpenter & Tello-Trillo 2015).

The policy implications of our paper is that public health programmes at the family level should be given a higher priority than laws decreasing the availability to fast foods in order to curtail the child and adult obesity epidemic. Due to the persistence of BMI it is important to prevent obesity at an early stage. In addition, due to the strong intra-generational transmission of BMI, it is of great importance to involve the parents as the most successful obesity prevention programmes has been at the family level (Bray 2004, pp. 83). Proposed policies might be programmes consisting of education; nutritional information on food labels, public advertisement of consequences of obesity and nutrition or exercise education. Although the home environment is the most important factor for childhood and adolescent obesity, very little is known about what those specific home influences are (Bray 2004, pp. 110). Therefore, more such research should be conducted to disentangle the indirect and direct predictors of obesity in conjunction with genetic data.

Chapter 2

The effect of mandatory salt iodisation on cognitive test scores in rural India

2.1 Introduction

Mass fortification of food with micronutrients constitutes a very cost-effective way of improving overall health, reducing mortality and increasing productivity (Black et al. 2008, 2013). The WHO has established that iodine deficiency in early life is the most common predictor of permanent and irreversible brain damage in the world (Aburto et al. 2014). More than 140 countries have implemented Universal Salt Iodisation (USI) programmes since the 1990s where the goal is to reach at least 90% of households with adequately iodised salt (UNICEF 2015). Salt containing 15-40 μg iodine/g salt is defined as adequately iodised. USI programmes are regarded as having been largely successful as almost 70% of the global population now consume iodised salt (Zimmermann & Andersson 2012).

This study is the first to evaluate the effects of a USI policy on human capital. Existing literature such as Adhvaryu et al. (2018), Politi (2010*b*,*a*) observe that access to iodised salt in the US and Switzerland during the 1920s improved schooling attainment, labour market outcomes and income. However, historical salt iodisation differs from current government led USI programmes initiated by the WHO. For instance, the spread of iodised salt in the US occurred in the private sector, without any regulation, standardisation or controls. It relied on marketing from private producers and the demand from health conscious consumers which might have led to selection in the take-up. It is further difficult to assess whether the historical experiences of salt iodisation are comparable to the goals and effects

of current USI programmes. Previous papers do not observe nationwide consumption of iodised salt throughout time, nor the iodine content of the salt at the level of consumption.

Secondly, this paper directly assesses the effect of USI in early life on cognitive skills. I exploit the introduction of USI across naturally iodine deficient and sufficient areas as a natural experiment. Despite an abundance of observational epidemiological studies in this area, causal evidence of iodine intake in early life on human cognition is limited. Feyrer et al. (2017) demonstrate that the availability of iodised salt in the US increased the probability of men from previously deficient areas to be selected into cognitively more demanding military sections during World War II. However, this paper uses data on a select sample of young men and is prone to the aforementioned caveats regarding the evaluation of historical salt iodisation. Furthermore, cognition in adulthood is likely to be affected by dynamic complementarities, where capabilities produced at one stage in life raise the productivity of investment at subsequent stages (Cunha & Heckman 2007). I estimate the impact on cognitive skills in childhood for a representative sample of young children. Therefore, this chapter yields more robust evidence of the effect of iodine availability in early life on cognitive endowment.

Previous literature such as Adhvaryu et al. (2018), Politi (2010*b*,*a*), Field et al. (2009) conclude that the positive effects on educational attainment and labour market outcomes are solely due to improvements in cognition. Additionally, all of the previous papers find that the treatment effects are either larger for women, or entirely driven by women. However, these studies do not observe cognitive attainment and no previous study have established the causal population-wide effects of large scale iodine supplementation on cognitive skills. By analysing the effects on cognition for both genders, this empirical chapter helps in explaining the mechanism by which previous papers have found improvements in long term human capital outcomes, particularly for women. Thus, it reduces the potential threat that the effects found in the related literature are driven by improved health, increased school attendance or unobserved differential trends in factors affecting education and labour market opportunities for women compared to men.

Thirdly, by evaluating a current USI programme in India, this study contributes to understanding the gains from USI in middle- and low income countries where iodine deficiency is more prevalent (Hetzel 2002). The existing research in economics on the effect of iodine on human capital in developing countries is inconclusive and limited (see Field et al. (2009) and Bengtsson et al. (2017)). Furthermore, it focuses on the evaluation of targeted iodine supplementation programmes which are less common than USI and viewed

as short term policies (Bougma et al. 2013, UNICEF 2015).

The experience and efficacy of USI in India has greater external validity for currently developing nations, compared to the historical provision of iodised salt in high income countries. For instance, the treatment effects are likely to vary due to large differences in the nutrition and disease environments in currently developing countries in the global south compared to high income countries during the 1920's. Moreover, institutional capacity and the supply and demand for iodised salt in low income countries is likely to differ from the contexts previously studied. For example, mandating salt iodisation in India did not result in reaching USI immediately. This is due to lacking enforcement of the policy, the general population being unaware of the benefits of iodine and because salt iodisation is costly for the many small and medium scale producers (Kumar et al. 2013, Vir 2003).[1]

I apply a difference-in-differences (DD) strategy to analyse the effect of being exposed to a ban on non-iodised salt during early life on children's cognitive test scores, across naturally iodine deficient and sufficient districts over time. The nationwide mandate was implemented in 2006 and available data shows that it increased the consumption of adequately iodised salt by at least 20 percentage points over 2002-2004 to 2005-2006. The most recent data from 2015-2016 reveals that more than 90% of Indian households consumed salt with some iodine. As the risk of iodine deficiency is mainly determined by geography (Hetzel 2002), I use historical information on the endemicity of iodine deficiency to identify districts that are likely to benefit the most from mandatory salt iodisation.[2]

The results suggest that being exposed to mandatory USI in utero until at least age 2, increases the probability of recognising at minimum simple numbers or letters by 1.9 - 4 percentage points among primary school aged children in rural India. Somewhat larger estimates are found on basic skills for girls, but the gender differences are not statistically significant across most specifications. I also observe that girls improved their overall reading ability, which takes more difficult reading tasks into account, while no effects are found for boys. Children who reside in states which experienced larger (smaller) relative increases in iodised salt consumption also gained more (less) in terms of learning outcomes. The main results are not driven by compositional changes across the cohorts in naturally iodine deficient and sufficient areas, nor by coinciding improvements in health

[1] The spread of iodised salt in the US relied on a high degree of health consciousness among the consumers and on profit motives of relatively few and large salt manufacturers to distribute a product endorsed by medical experts (Adhvaryu et al. 2018, Bishai & Nalubola 2002).

[2] Unlike other micronutrients, iodine does not occur naturally in specific foods. Rather, it is present in the soil and is ingested through foods grown on either iodine rich or poor soils. Soils from mountain ranges, areas with high rainfall and frequent flooding are particularly likely to be deficient (Hetzel 2002).

endowments or health investments in early life. The analysis in this paper further benefits from data on test scores of both in- and out of school children. Thus, the results are not driven by changes in the composition of children attending school. Furthermore, the treatment effects hold when using geographical predictors as Instrumental Variables (IV) for naturally occurring iodine deficiency and using an alternative dataset for pre-existing iodine deficiency. Additionally, I show that an earlier mandatory fortification policy which was in place only during two years, also had a positive, albeit smaller, impact on test scores.

This paper adds to the extant knowledge by showing that USI improves basic cognitive skills for both genders. At the same time, the larger treatment effects for girls' overall literacy ability lend support to the findings that women experienced larger improvements in schooling attainment and labour market outcomes following a higher iodine availability in utero, see Adhvaryu et al. (2018), Politi (2010*b*,*a*), Field et al. (2009). The female treatment effects do not vary with differences in area-level gender preferences which strengthens our confidence that the observed effects are driven by biology. On the other hand, lower son-preference decreases the treatment effect of USI on basic numeracy skills for boys. This might be suggestive of gender differences in parental reinforcement of observable cognitive endowments.

Investigating the determinants of learning outcomes in developing countries is by itself of large importance for policy. It is cognitive skills rather than schooling attainment which drive individual earnings and economic growth (Hanushek & Woessmann 2008). Although school enrolment and attainment have risen in a large part of the developing world, learning outcomes have remained poor in many countries. The findings from this study highlight the relevance of intersectoral action to improve academic skills.

The remainder of this paper is organised as follows. In Section 2.2, I discuss the biological role played by iodine in the human body and review the previous literature on its impact on human capital. I subsequently describe the iodine fortification policy I evaluate in Section 2.3, followed by the data in Section 2.4. The empirical strategy and the results are presented in Section 2.5. The robustness of the main results is tested and discussed in Section 2.6 and concluding remarks are reported in Section 2.7.

2.2 Iodine deficiency and its effects on human capital

Iodine is needed to regulate thyroid hormone availability. The thyroid gland secretes 80 μg of iodine per day in the form of thyroid hormones. Thyroid hormones are released into the blood stream to control the metabolism (conversion of oxygen and calories to energy)

of all cells in the human body. The WHO recommends the following daily iodine intake: 90 μg for children of 0-59 months, 120 μg for ages 6-12 and 150 μg for older children and adults. Two teaspoons of adequately iodised salt provides 150 μg of iodine. Pregnant and lactating women have a greater need for iodine and require 250 μg iodine per day (Andersson et al. 2010).[3] When the thyroid does not receive sufficient amounts of iodine, it becomes enlarged such that it can produce more thyroid hormones for a given level of iodine (Zimmermann 2009). This condition is called goitre and has by itself no ill effects on health.[4]

More important, normal concentrations of thyroid hormones are required for the development of the central nervous system during early life. The most critical time for overall brain development is during the foetal stage. Thyroid hormones influence the myelination, neuronal migration (the process by which neurons migrate from their place of origin to their final location in the brain), glial differentiation and density of neural networks established in the developing brain. Extreme foetal iodine deficiency can also lead to physical defects such as cretinism, deaf-mutism, abortions, stillbirths, congenital anomalies and increased perinatal and infant mortality (Zimmerman 2012).

There is abundant evidence of the association between iodine deficiency and cognition (Zimmermann 2012). However, most medical and epidemiological literature involving humans is correlational. A recent systematic review of 89 studies on the effects of iodised salt provision recorded a reduction in the risk of low intelligence (defined as IQ $\leq$70) of 72-76% and an increase of 8.2-10.5 IQ points (Aburto et al. 2014). Another systematic review of high quality randomised controlled trials show that iodine supplementation in utero increased IQ with an average of 7.4 points (Bougma et al. 2013). There is also convincing clinical and epidemiological evidence that mild iodine deficiency in early life reduces cognitive skills, see Lavado-Autric et al. (2003) and Zimmermann (2012).[5]

The evidence of particularly critical time periods in utero is mixed.[6] A review conduc-

[3]The human body cannot store iodine as it is excreted in the urine. However, we can store thyroid hormones which can meet the body's requirements for up to 3 months (Ahad & Ganie 2010).

[4]Some vegetables such as; cassava, some species of millet and crucieferous vegetables can lead to goitre by blocking thyroidal uptake of iodine. However, they are not of clinical importance unless they are consumed in large amounts and there is coexisting iodine deficiency (Zimmermann 2009).

[5]Some studies also show that mild iodine deficiency in childhood have concurrent effects on cognitive functioning. For example, results from a randomised trial in New Zeeland showed that iodine supplementation of young children improves perceptual reasoning in mildly iodine deficient children (Gordon et al. 2009).

[6]Older medical evidence point to that cognition is sensitive to iodine deficiency exclusively prior to mid gestation (Cao et al. 1994). Later clinical research finds that if pregnant women were previously deficient in

ted by Zoeller & Rovet (2004) conclude that thyroid hormones affect the developmental process in all areas of the brain which makes it difficult to identify specific critical time periods. Because different areas of the brain develop at different times, critical periods of iodine intake are temporally shifted. In addition, postnatal thyroid hormone insufficiency is associated with poorer language, auditory processing, attention, memory and fine motor skills.

Field et al. (2009) is the first paper in economics to study the effects of congenital iodine deficiency on human capital attainment. The authors use the roll-out of a maternal supplementation programme of iodine capsules in Tanzania, to estimate the causal effect of iodine supplementation during the first trimester in utero on schooling outcomes. They find that iodine supplementation increased educational attainment by 0.35 years on average, and that the effect was larger for girls. Bengtsson et al. (2017) argue that the treatment effects in Field et al. (2009), which stem exclusively from supplementation during the first trimester, are large given the effect sizes found in medical research. Bengtsson et al. (2017) replicate the previous study and improve the model with a more precise calculation of treatment probability with up to date medical knowledge and more detailed institutional information and increase the sample size with additional data. Their estimates are close to zero and statistically insignificant.

More recent papers such as; Feyrer et al. (2017), Adhvaryu et al. (2018), Politi (2010*b*,*a*) study the effects of iodine fortification of table salt on human capital outcomes in the US and Switzerland during the 1920's.[7] These studies identify the effect of iodine deficiency on human capital using the introduction of iodised salt, in conjunction with geographic variation in pre-existing levels of naturally occurring iodine deficiency. The idea is that if added iodine in early life improves cognition, one should see a relative increase in human capital among populations in previously deficient areas, compared to populations living in always iodine sufficient areas, after the introduction of iodised salt.

Feyrer et al. (2017) estimate the impact of iodine fortification of salt in the US on the probability of being accepted in to the cognitively more demanding Air Forces. They identify treatment status by the interaction of pre-existing goitre prevalence per military section, with year of birth dummies indicating whether one experienced fortification in early life or not. The authors find that men from previously high goitre areas are 3.8-10

iodine during early gestation but became sufficient later in pregnancy, infant development was not affected (Pop et al. 2003).

[7]The empirical methodology from this strand of literature originates from papers studying the effect of the eradication of other diseases, see Bleakley (2007), Cutler et al. (2010), Bleakley (2010), Lucas (2010).

percentage points more likely to enter the Air Forces compared to earlier cohorts. This corresponds to an increase of approximately 15 IQ points. The effect is larger than what is found in most epidemiological research using observational data. However, we are unable to compare the estimates to the medical literature as we do not know the proportion of households consuming iodised salt nor the iodine content of the fortified salt. Moreover, we do not know what the population-wide effect might be as their data comprises of a select sample of young men.

Adhvaryu et al. (2018) employ the same natural experiment as Feyrer et al. (2017), but analyse the effects on labour market outcomes using US census data. The census respondents are assigned the goitre rate in their state of birth. The authors find that cohorts who benefited from access to iodised salt from states with a higher goitre rate experienced an increase in; income, labour force participation and the probability of being in full-time employment. These impacts were driven by females who also experienced a small increase in educational attainment of about 2 weeks of additional schooling.

This study only finds a statistically significant increase in income of 1% for men conditional on working. No other effects are found for most labour market outcomes for men. This is rather surprising as Feyrer et al. (2017) document a very large increase in cognitive skills for men evaluating the same natural experiment. Adhvaryu et al. (2018) reconcile these effects by women and men facing different labour market conditions.

Politi (2010*b*,*a*) applies a similar identification strategy to evaluate the effects of salt iodisation in Switzerland. The author shows that salt iodisation increased secondary and tertiary schooling as well as the probability of entering top-tier occupations with higher cognitive demands and wages. The effects are larger for women, in similarity with Field et al. (2009), Adhvaryu et al. (2018).

The differential effect for men and women first observed by Field et al. (2009) has previously been explained by biological gender differences. It is important to note that the majority of medical studies involving humans do not show gender differences in iodine sensitivity.[8] Due to the lack of robust conclusive medical evidence of such biological sex differences, Adhvaryu et al. (2018) suggest that the heterogeneity in treatment is more

[8] A few lab and epidemiological studies find foetal gender differences in iodine sensitivity. Friedhoff et al. (2000) study the effect of severe prenatal iodine deficiency on rats and find that female rats appeared to be more vulnerable to the effects on learning than male rats. Nonetheless, it is questionable how well these results translate to humans with mild iodine deficiency. Murcia et al. (2011) study the correlation between a diet low in iodine, proxied by self-reported fish consumption and mineral supplement intake, among pregnant mothers and infant neurodevelopment. The authors report gender differences in cognitive outcomes but the is not able to account for omitted variable bias.

likely to be explained by there being a much larger scope for growth in female employment than male employment.

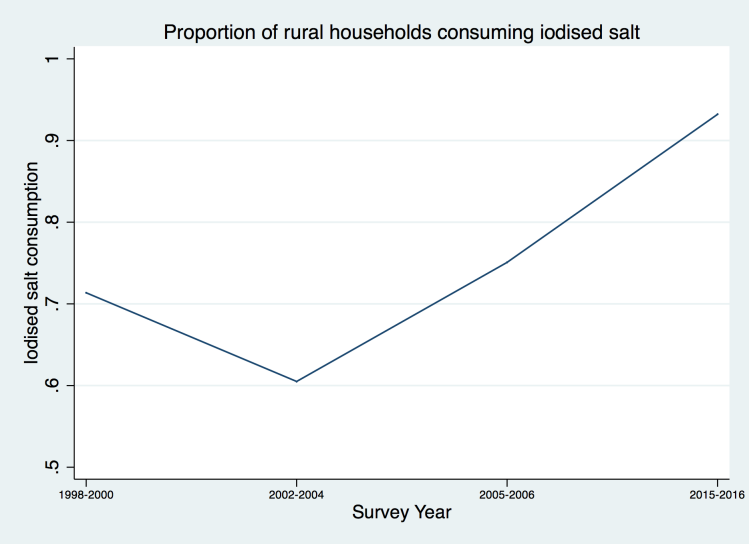
2.3 Mandatory universal salt iodisation in India

On 27 May 2005, the Government of India notified a national ban on the sale and storage of non-iodised salt for direct human consumption under the 1954 Prevention of Food Adulteration Act. The ban came into effect on 17 May 2006 and stipulated the minimum iodine content of salt at the production and consumption levels at 30 and 15 μg/g salt, respectively. Food inspectors in each state are responsible for monitoring the implementation of the ban, which includes testing of salt samples from producers and traders. If the samples are not found to be adequately iodised at the retail level, all responsible persons will be fined and subject to non-bail warrants or imprisonment (Vir 2011). Shopkeepers who stock non-iodised salt will also be penalised (Kapil et al. 2005).

This policy increased the production of iodised salt from 1.69 million tonnes prior to the federal mandate to 5.1 million tonnes in 2007. The consumption of fortified salt has continued to rise with time (Vir 2003).[9] The national rural coverage of adequately iodised salt at the household level increased from approximately 30% in 2002-2004 to 51% around the notification and implementation of the ban in 2005-2006 and reached 71% in 2009 (UNICEF 2011). The National Family Health Survey (NFHS)-4 indicates that 92% of the rural population consumed iodised salt (salt with any iodine) in 2015-2016. See Figure 2.1 for the trends in iodised salt consumption across national surveys from 1998-2000 to 2015-2016.

[9]Moreover, potassium iodate, which is used for iodine fortification of salt, has been supplied for free to selected iodisation units by some donors since 2005 (Pandav 2013).

Figure 2.1: Nationwide consumption of iodised salt over time



The figure depicts the trends in the proportion of rural households consuming iodised salt across India. Data from the NFHS II is used for the years of 1998-2000 and the DLHS II is used for the years of 2002-2004. Statistics from the NFHS III and the NFHS IV is used for the survey years of 2005-2006 and 2015-2016, respectively.

From Figure 2.1 we note that there was a decreasing national trend in the consumption of iodised salt prior to mandatory USI in 2006. This is due to the removal of a similar but short lived federal ban on non-iodised salt in 2000 (Pandav 2013).[10] This mandate was implemented in 1998 but did not manage to increase iodised salt production nor consumption due to it being introduced at the same time as other factors affecting the supply and regulation of salt. The salt producing areas of Gujarat, a state which produces around 80% of all salt in India, were hit by a cyclone in 1997 and later by an earthquake. Moreover, the de-licensing of the salt industry in 1996 made it more difficult for the Salt Department to regulate production (Pandav et al. 2003, Salt Commissioner's Organisation, Department of Industrial Policy and Promotion 2004).

The Government of India withdrew this policy with the motivation that "..matters of public health should be left to informed choice and not enforced." (Rah et al. 2015). All states, besides Gujarat and Arunachal Pradesh, kept their state level bans during the absence of nationwide mandatory USI.[11] Gujarat, which is by far the dominant salt exporting state within the country, has a salt market which comprises of many medium and small producers and traders. Salt iodisation is costly for such producers as they operate within narrow profit margins. State level iodisation policies suffer from weak

[10] Please see Appendix B.1 for a summary of the history of iodine fortification in India.

[11] The state of Orissa lifted the ban initially but reimposed it after six months.

enforcement.[12] Therefore, any USI legislation which does not enforce iodisation in Gujarat will not be effective (Vir 2003).

The spread of non-iodised salt prior to the ban of 2006 was enhanced by the difficulty for both intermediate suppliers and consumers to distinguish iodised from non-iodised salt. Salt is procured by wholesalers, who often purchase the salt in bulk, and subsequently repackage it. Salt procured in bulk is often non-iodised, but the wholesalers and retailers are not able to recognise it. Non-, or inadequately iodised salt is sold in packages with similar design, brands and logos to those of iodised salt, but at a lower price. They are often falsely labelled as adequately iodised and the consumer has no ability to distinguish the non-iodised salt from iodised salt (Vir 2003).

Additionally, a nationwide study by Kumar et al. (2013) finds that even though the majority of the Indian population are aware of iodised salt, only a quarter of individuals have knowledge about it. Few know about other ill-effects of iodine deficiency than goitre. 17.1% know about mental retardation as an outcome of iodine deficiency and the percentage is likely to be lower among rural households (Kumar et al. 2013). The logo of the "Smiling Sun" used to mark that the salt is adequately iodised, is known to only 4% of respondents and the printing of the iodine content on packets is known to 15% (Kumar et al. 2013). Vir (2011, pp. 596) evaluates state programmes aiming to increase iodised salt consumption and conclude that "..even if the public is made aware of the significance of iodized salt and convinced to consume only adequately iodized salt, the consumers are not in a position to distinguish adequately iodized salt from non-iodized or inadequately iodized salt due to the misleading practice for incorrect labelling regarding iodine content". The high level of information asymmetries in the distribution and consumption of iodised salt also suggests that selection in the uptake of iodised salt does not pose a significant threat to the evaluation of the effects of mandatory salt iodisation in India.

These factors explain why there was a reduction in iodised salt consumption following the removal of the first attempt at mandating USI in 1998 despite this policy not appearing to have an effect due to coincidental negative shocks to the supply of salt at the time of its implementation. The drop in iodised salt coverage spurred advocacy among public health authorities and NGOs for the re-introduction of mandatory USI. Academic institutes, civil

[12] For example, despite Bihar having implemented a ban on non-iodises salt prior to 2006, enforcement was practically non-existent. An iodine deficiency disorder control task force was set up in 1988 but has not been functioning. Other public health policies such as Polio eradication and vitamin A campaigns gained more political support and higher prioritisation and crowded out performance with respect to other programmes (Sankar et al. 2006).

society, international agencies and ministries lobbied for a re-implementation of mandatory USI which resulted in the current policy being implemented in 2006.

I evaluate the impact of being exposed to this ban during early life compared to older children who were in early life during the absence of federal mandatory USI in 2001-2005. I will not focus on the earlier USI policy of 1998-2000, due to its short time span. Children who were in utero during the first ban, were still in a critical postnatal time period for brain development when it was abolished.

2.4 Data

2.4.1 District level goitre endemicity

Most iodine in soils is derived from the atmosphere, where in turn, it has been derived from the oceans which contain the highest concentration of iodide (Fuge 2007). Therefore, coastal soils are likely to be richer in iodine compared to inland soils. In many parts of India, deficiency of iodine in the soil-water ecosystem is due to heavy rainfall, steep gradient and poor vegetation cover resulting in quick run-off and little time for transfer of iodine. For instance, the soils in the Himalayan foothills are low in iodine due to glaciation during the last ice age, which stripped the soil of iodine. As it takes thousands of years for rain water to replenish the soil with iodine, the iodine content of the soil and water of mountainous regions remains low (Fuge 2007).

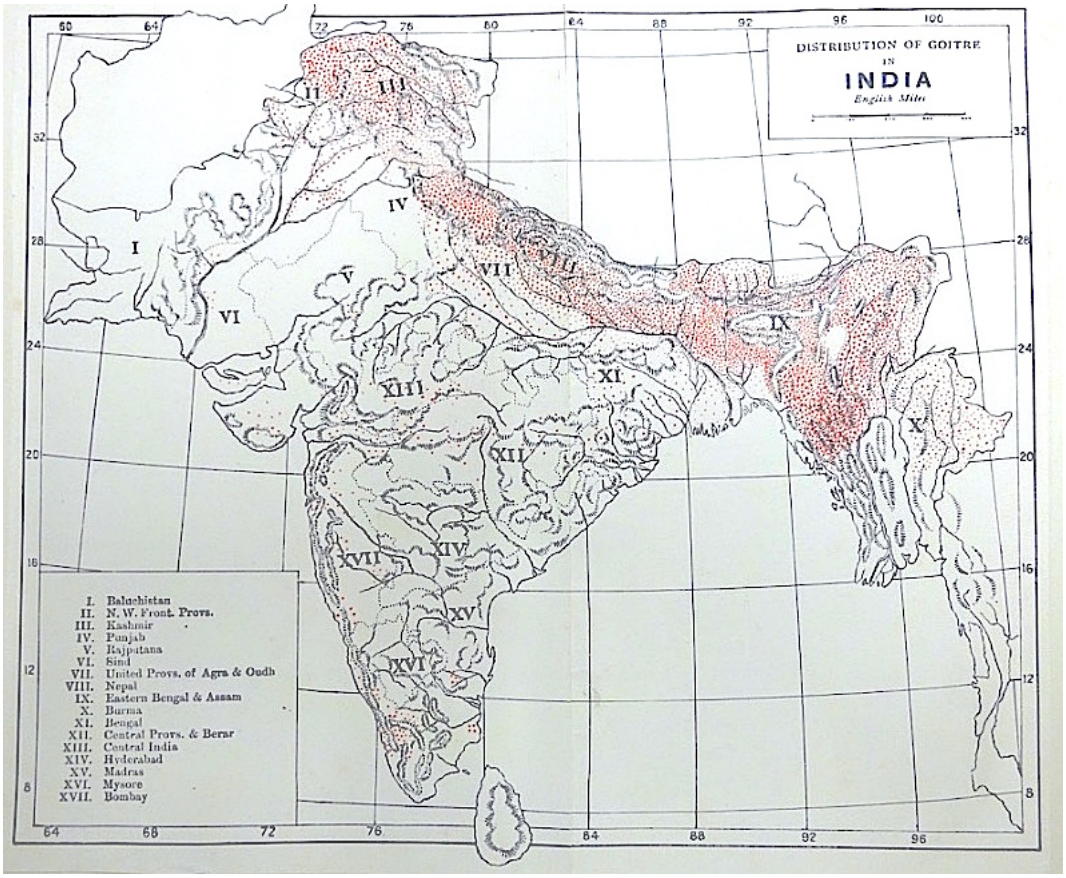
Due to heterogeneity in iodine availability in the soil accessible to humans and because of the lack of nationally representative data on iodine content in soil and groundwater, the best measure of inadequate local iodine availability is the prevalence of pre-fortification goitre.[13] The "Himalayan goitre belt" is the world's largest and most intense goitre endemic area, spanning over 2,400 km. It runs along the southern slopes, foothills and adjacent plains of the Himalayas and the level of iodide in the drinking water is extremely low (Pandav 1982). Other areas, such as pockets of the Indian west coast also have a high prevalence of iodine deficiency due to heavy rainfalls, alluvial soils and less saline ground waters (Smedley 2004).

Following Feyrer et al. (2017), Adhvaryu et al. (2018), Politi (2010*b*,*a*), I define naturally iodine deficient areas by the spatial prevalence of goitre endemicity prior to any availability of iodine supplementation. Individuals who reside in previously endemic areas

[13] Various characteristics of a soil can lead to different iodine-fixation points where the iodine from the soil is fixed in the soil and not taken up by the roots of plants and thus not transferred to humans (Johnson 2003).

are thus more likely to benefit from iodine fortification in comparison to individuals who live in areas which have always been sufficient in iodine. I use information on the location of goitre endemicity in 1915 compiled by the British physician Sir Robert McCarrisson. This is the only available nationwide data on goitre prior to any form of iodine supplementation. Sir McCarrisson was provided data on goitre incidence by administrative medical officers and civil surgeons in British India (McCarrison 1915).[14] The original map of goitre endemicity in McCarrison (1915) is shown in Figure 2.2.

Figure 2.2: Location of goitre endemic areas by McCarrisson (1915)



This figure shows the location of historical goitre endemicity from McCarrison (1915). The dots represent areas which were found to be goitre endemic prior to 1915.

McCarrison (1915) writes;

In the accompanying map I have indicated by means of red dots those localities where goitre has been reported to prevail. It is of course impossible, in a map of these dimensions to indicate every area with the accuracy of detail that is desirable. The map, therefore

[14] McCarrison (1915) writes: "Through the kindness of Administrative Medical Officers, and with the generous assistance of Civil Surgeons, I have been enabled to collect detailed information regarding the prevalence and distribution of goitre in almost every part of British India."

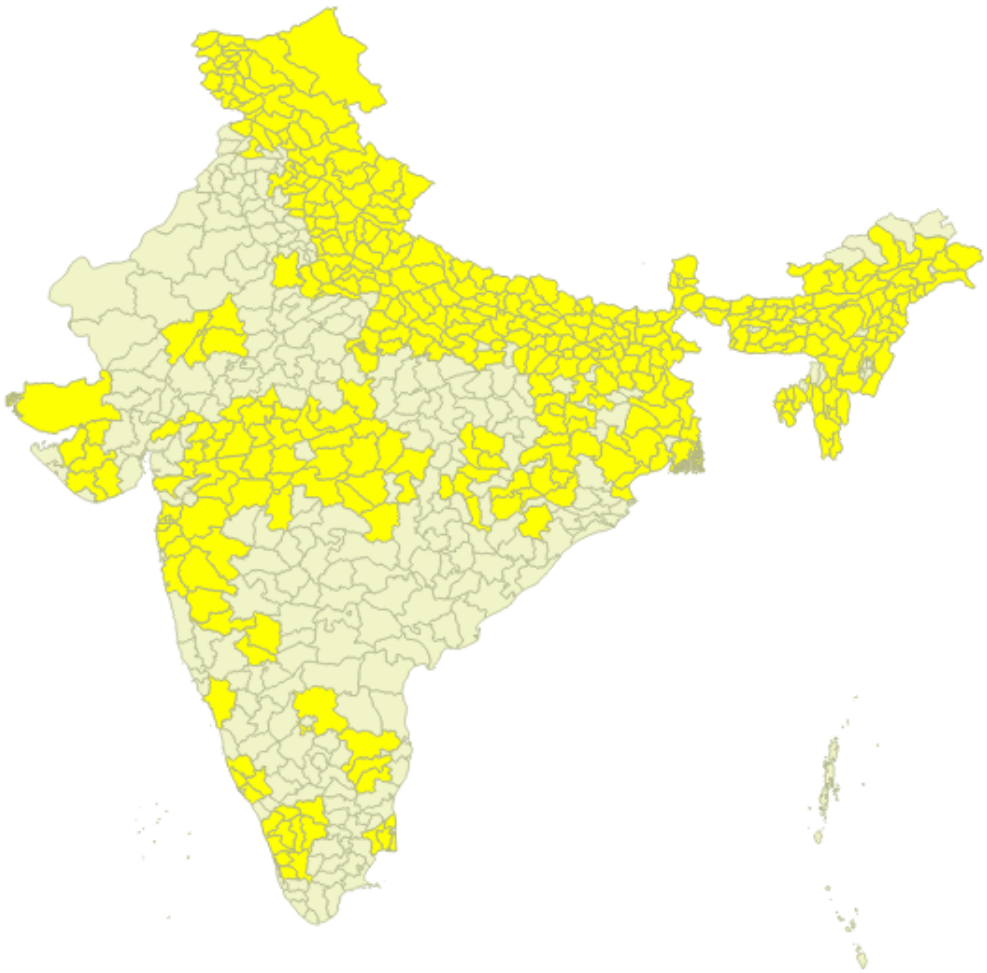
is to be regarded only as affording an approximately accurate indication of the general distribution of the disease over India.

For the analysis, I define a district according to the 2001 census to be goitre endemic if it contains at least one dot indicating goitre endemicity in 1915.[15] Districts containing no dots are defined as non-endemic.[16] The generated map of endemic districts in India prior to 1915 is shown in Figure 2.3. The dark yellow districts represent goitre endemic districts and the light yellow districts represent non-endemic districts.

[15]I use the geographic information system (GIS) software QGIS, to digitise the location of the dots as of Figure 2.2 into a GIS file. I then merge the information on the location of the dots using a boundary GIS file of the districts according to the 2001 Indian Census.

[16]There is no information about the size of the area each dot represents, nor the intensity of goitre per dot.

Figure 2.3: Historically goitre endemic districts



This figure shows the location of historical goitre endemicity of Indian districts as of 2001. The bright yellow areas represent districts that contained at least one area which was goitre endemic according to McCarrison (1915). These districts are deemed to be pre-fortification goitre endemic and the light yellow districts are defined as non-endemic.

While other micronutrient deficiencies are likely to decrease with rising caloric intake, the risk of iodine deficiency is locally persistent due to its geographical determinants. Therefore, the population residing in the goitre endemic localities shown in McCarrison (1915) should have a higher risk of current iodine deficiency without supplementation of iodine. The validity of the spatial goitre prevalence in McCarrison (1915) is confirmed both by state level thyroid prevalence in 2005-2006 and previous studies. I use the 2005-2006 National Family Health Survey (NFHS) III to check the relationship between historical pre-fortification goitre endemicity as of McCarrison (1915) per state and more recent thyroid related illness prevalence. I regress the proportion of adults, 35 years and older,

who report having thyroid related illnesses on the number of historically goitrous areas per state population. The results show a positive and statistical significant association between current thyroid problems and historical goitre endemicity, see Table B.16 in the Appendix.

Furthermore, there is no evidence of the consumption of iodine rich foods having risen more in historically endemic areas compared to iodine sufficient areas over time. Fish has the highest iodine content of all foods but fish consumption is low in India as the majority of the population is vegetarian. Individuals living in coastal areas consume relatively more fish compared to their inland counterpart. Coastal areas are therefore less likely to have been goitre endemic compared to inland areas. The share of food expenditure on fish does not appear to have increased disproportionally in inland states compared to maritime states.[17]

Sub-national surveys on goitre from the 1940's and onward, such as Pandav (1982), corroborate the location of areas prone to iodine deficiency. Despite later public health efforts to supplement deficient populations with iodine, the spatial occurrence of iodine deficiency in McCarrison (1915) appears to have understated the historical goitre rate, as more districts have been found to be goitre endemic (Pandav 2013). Therefore, using the information provided in McCarrison (1915) to identify districts that are likely to benefit from iodised salt will at most underestimate the true effect of iodine fortification on human capital in India.

District level total goitre rate surveys

In order to confirm the validity of the spatial information of goitre endemicity in McCarrison (1915), I use additional data on the total goitre rate. The IDD and Nutrition Cell, Directorate of Health Services, Ministry of Health and Family Welfare India report district level averages of the goitre rate among primary school aged children measured in 1940-2010.[18] The data is not representative at state or country level and does not include all districts. Areas with previously known goitre prevalence are likely to have been included in the survey and surveyed earlier. Furthermore, the data consists of surveys collected

[17]Data from the National Sample Study Organization, show that the percentage of food expenditure on fish increased from 2.03% in 1983-1984, to 2.42% in 1999-2000 for all of India. The corresponding change over time for maritime states have been 2.77% to 3.51% and the increase in fish consumption has been lower for non-maritime states have been 1.43% to 1.55% (Mruthyunjaya 2004).

[18]The report has been shared with me by Dr. Kapil Yadav, at the All Indian Institute of Medical Sciences (AIIMS).

over a long period of time making it prone to measurement error.[19]

To obtain a measure of the underlying intensity of naturally occurring iodine deficiency, I restrict the analysis of the goitre rate data to the 263 districts as of the 2001 Indian Census that were surveyed prior to the implementation of any district, state or national iodine fortification policies.[20] The sample of surveyed districts have an average total goitre rate (TGR) of 25.94 with a SD of 15.74, the proportion of children with goitre ranges from 0.01 - 85.35%.

Even though this data is not nationally representative, we note that the historical goitre rate for India far exceeds the maximum prevalence of the historical goitre rate in previous papers, such as Feyrer et al. (2017). The estimates reported here are more in line with historical data from other currently low or middle income countries.[21] More important, this district level data validates the use of the endemicity indicator variable derived from McCarrison (1915). I estimate a linear probability model with the outcome being the probability that a district contains at least one goitre endemic area (as in McCarrison (1915)), on the proportion of children with goitre and different cut-offs of goitre prevalence. From Table B.17 in the Appendix one can observe a positive association between the spatial occurrence of goitre across both datasets.

2.4.2 Data on cognitive test scores

I use the Annual Status of Education Report (ASER) to measure the effect of USI on cognitive test scores. ASER is a cross-sectional survey which tests around 500,000 children aged 5-16 in rural India each year in reading and mathematics.[22] Publicly available household surveys began in 2007, and have been conducted yearly between September-November. The survey is representative at the rural district level. ASER is unique in that it includes both in- and out of school children. I use all available surveys, for years 2007-2014. See a further explanation of the ASER data in Appendix B.2.1.

I construct a basic numeracy score which is a binary variable taking value 1 if the

[19] See further descriptions and discussion of the data in Appendix B.2.2

[20] The number of districts in India has increased over time. I match districts surveyed prior to 2001, to districts as of 2001 that were contained within the boundaries of the older districts. The matching of districts was made based on the reported divisions of districts throughout census years 1971-2001 by (Kumar & Somanathan 2009). I also match districts of 2001 to the older districts given that the old district constitute at least 90% of the area of the new district.

[21] In the mid 1950s many endemic regions in for instance Nicaragua, Colombia, Sierra Leone, Sudan, Malaysia and Indonesia had a goitre rate above 40% (Kelly & Snedden 1960).

[22] They also test children in English. Due to regional differences in English proficiency, I do not study the effects on English.

child can recognise single-digit numbers or more (double-digit number recognition, two-digit subtraction with carry over, and three digit by one digit division), and 0 if the child cannot recognise single digit numbers. Similarly, I generate a basic literacy score which takes value 1 if the child recognises letters and above (words, a short paragraph - a grade 1 level text, and a short story -a grade 2 level text) and 0 if the child cannot recognise letters. Focusing on basic academic proficiency can help to reveal heterogeneous impacts of iodine fortification, with possibly greater effects on the children who are more likely to have low test scores to begin with. Moreover, I estimate the effect on overall age standardised numeracy and literacy skills. The raw test score ranges 0-4, where the maximum score corresponds to the highest level of proficiency in the ASER tests.

The ASER data also includes other household and village level information. For instance, the household reports the material of their house which can be used as a proxy for household wealth. "Pucca" denotes a house made of durable materials such as brick, stones or cement, "Kutcha" denotes a house made of less durable materials such as mud, reeds, or bamboo, and "Semi-Pucca" denotes something in between. Hence, Pucca is a proxy for relatively high economic status. The survey also contains information on the existence of a government primary school, Anganwadi centre and a ration shop in the village and whether the village is connected to a pucca road. An Anganwadi centre offers basic health care and services related to nutrition and schooling of young children. A ration shop provides food from the public distribution system.

2.4.3 Descriptive statistics

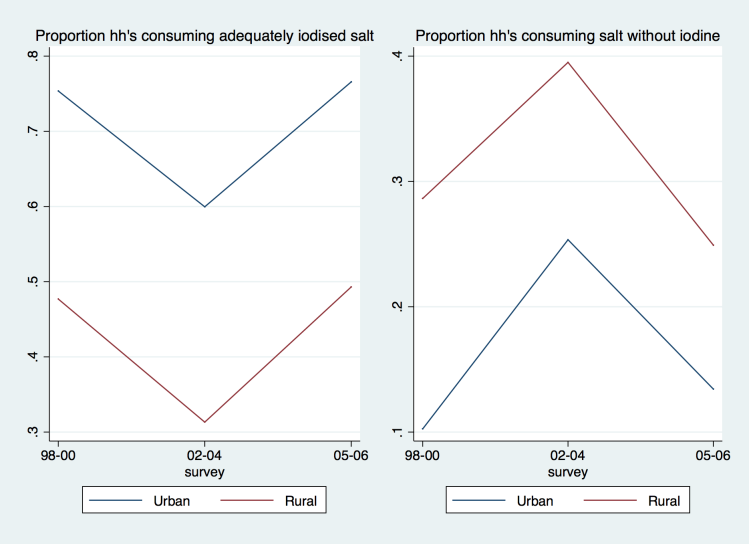
I begin by showing the effects of the mandatory iodisation policy on the consumption of adequately iodised salt. Data from the Indian Demographic and Health Surveys - the National Family Health Survey (NFHS), II and III surveyed in 1998-2000 and 2005-2006, respectively, are used with the District Level Health Survey (DLHS) II from 2002-2004. These surveys include information on objectively measured iodine levels of salt at the household level. Survey enumerators measure the level of iodine in table salt using a rapid-test kit (IIPS. 2007).[23] The salt is judged to be adequately iodised if it contains at least 15 μg iodine/g salt, in line with government requirements. WHO has established

[23]The test kit consists of a solution which will change colour, from light blue through dark violet, depending on the level of iodine in the salt. The interviewer then matches the colour of the salt as closely as possible to a colour chart provided and records the iodine levels. The surveys report a categorical measure of the iodine content in salt; no iodine, some iodine and whether the salt has an adequate amount of iodine (IIPS. 2007).

that urinary iodine excretion is the best measure of iodine deficiency. Consistent data on urinary iodine excretion across time does not exist for India. However, research shows that there is a strong correlation between iodised salt consumption and urinary iodine excretion. Horton & Miloff (2010) find that a 1% increase in iodised salt consumption is associated with a 0.73% increase in urinary iodine excretion for developing countries.

As the main analysis will be carried out for rural children only, I plot the proportion of households consuming adequately iodised salt across time for rural and urban households separately in Figure 2.4.[24]

Figure 2.4: Nationwide consumption of adequately iodised salt and non-iodised salt over time



The figure depicts the trends in the proportion of urban and rural households who consume iodised and non-iodised salt. Survey 98-00 denotes the NFHS II which covers the years of 1998-2000. Survey 02-04 denotes the DLHS II of 2002-2004 and Survey 05-06 represents the NFHS III for the years of 2005-2006.

The proportion of rural households consuming adequately iodised salt is depicted by the red line and the trend for urban households is given by the blue line. One needs to keep in mind that the data collection for the 2005-2006 NFHS III mostly took place before the implementation of the 2006 mandate and thus understates the effect of the legislation on iodised salt consumption.

From Figure 2.4, we observe a sharp increase from around 32% of rural households consuming adequately iodised salt in the 2002-2004 DLHS II, during the absence of a ban, to around 49% in the 2005-2006 NFHS III which captures some households' iodised salt

[24]Descriptive statistics of the surveys are given in Appendix B.2.3

consumption directly after the notification of mandatory USI. We can also see that the level of adequately iodised salt consumption during the 2005-2006 NFHS III is similar to the 1998-2000 NFHS II when the previous nationwide ban was implemented. Plotting the proportion of households consuming salt without any iodine reveals a similar trend.

What is further important for the identification strategy is that the increase in iodised salt availability following the ban of 2006 reduced iodine deficiency in areas identified as naturally prone to iodine deficiency by McCarrison (1915). I use the 2005-2006 NHFS III and the NFHS IV surveys to plot trends for the proportion of individuals with self reported thyroid problems, including goitre. I have merged the data with the number of goitre endemic areas from McCarrison (1915), by state as of the 2001 Indian Census. We observe that states at or above the 75^{th} percentile, in comparison to those at, or below the 25^{th} percentile of the distribution of the number of endemic goitre areas per state, experienced a larger decrease in thyroid related illnesses given an increase in iodised salt consumption, see Figure B.2.[25]

For the main analysis, I merge the ASER data with historical district level prevalence of goitre endemicity. In order to show descriptive statistics of the sample prior to treatment, I present summary statistics for the earlier control cohorts who did not benefit from iodine fortification in early life across goitre endemic and non-endemic districts. The means of learning outcomes, child-, household- and village level characteristics are shown for 5-10 year olds, born in 2002-2004 in goitre endemic and non-endemic districts in Table 2.1. In addition, differences in means and accompanying t-statistics are provided.

[25]I cannot conduct a district level analysis as district identifiers are not provided in the 2005-2006 NFHS III. An increased iodine intake above the recommended intake can be detrimental for health as it can cause hyperthyroidism. Some medical evidence indicate that iodine-induced hyperthyroidism is more common among those in areas with chronic long-standing iodine deficiency. Therefore, more access to iodine might have increased thyroid issues for those with no previous iodine deficiency and also increased such problems for those with previous iodine deficiency (Zimmermann et al. 2008). These mechanisms might explain the overall upward trend in thyroid related problems in rural India during 2005/2006 - 2015/2016.

Table 2.1: Descriptive statistics from ASER: Children in early life during the absence of mandatory USI across goitre endemic and non-endemic districts.

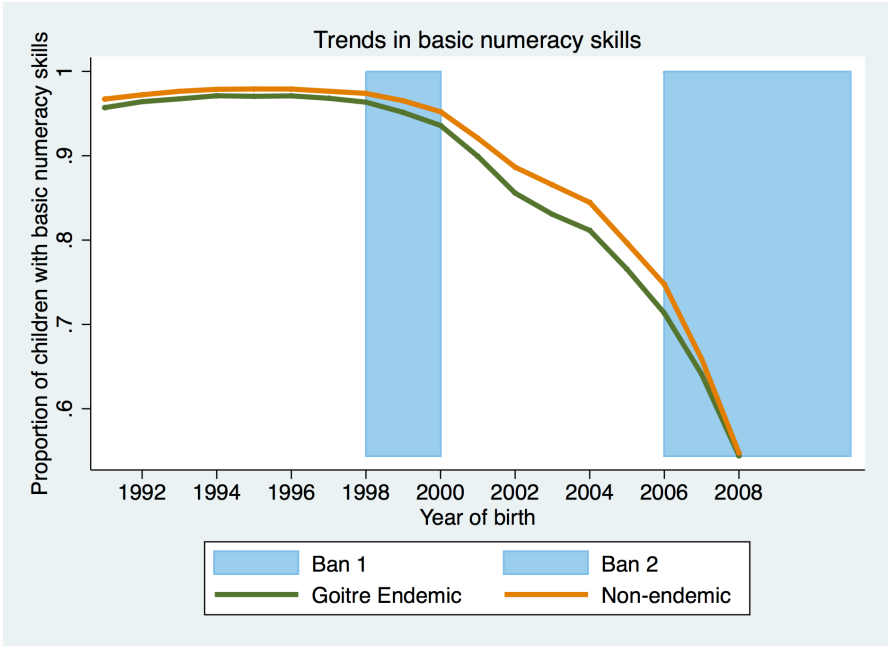
	Endemic		Non-Endemic		Difference	
	Mean	SD	Mean	SD	Difference	t-statistic
Enrolled	0.98	0.13	0.99	0.11	0.01***	(21.07)
Dropped out	0.01	0.07	0.00	0.07	-0.00***	(-5.52)
Recognises numbers 1 to 9 or better	0.82	0.38	0.86	0.35	0.04***	(41.53)
Overall numeracy score	1.67	1.19	1.75	1.16	0.09***	(31.72)
Age standardised overall numeracy score	-0.02	1.05	0.07	0.94	0.09***	(38.98)
Reads letters or better	0.81	0.39	0.85	0.36	0.04***	(47.31)
Overall literacy score	1.81	1.36	1.94	1.33	0.13***	(41.43)
Age standardised literacy score	-0.02	1.08	0.09	0.95	0.11***	(45.98)
Grade	2.66	1.48	2.79	1.48	0.13***	(36.86)
In private school	0.28	0.45	0.28	0.45	0.01***	(8.19)
Age	7.51	1.71	7.53	1.70	0.02***	(4.90)
Girl	0.46	0.50	0.46	0.50	-0.00	(-1.53)
Years of maternal education	3.76	4.41	4.09	4.48	0.33***	(32.83)
Kutcha House	0.41	0.49	0.33	0.47	-0.08***	(-75.02)
Pucca House	0.28	0.45	0.37	0.48	0.10***	(89.19)
Household Size	6.84	3.31	6.30	3.01	-0.53***	(-76.95)
Village has a government school	0.92	0.27	0.93	0.25	0.01***	(22.12)
Anganwadi in village	0.90	0.29	0.95	0.21	0.05***	(78.04)
Access to pucca road in village	0.70	0.46	0.82	0.39	0.12***	(123.62)
Ration shop in village	0.68	0.47	0.73	0.44	0.05***	(46.03)
Observations	534048		337490		871538	

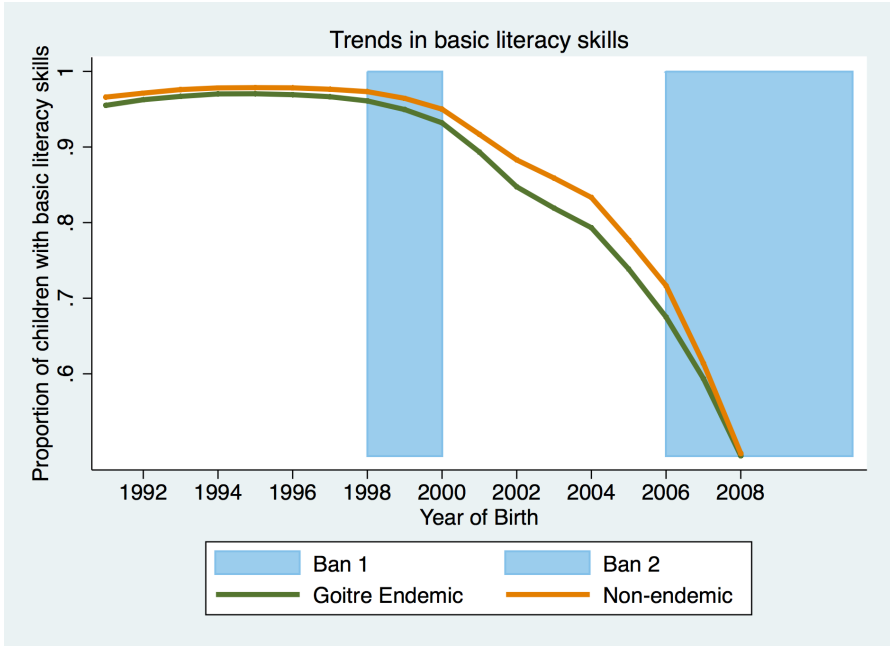
Notes: This table reports the means and standard deviations for children who were in early life during no nationwide iodisation policy, born in 2002-2004, in historically goitre endemic and non-endemic districts. The last two columns report the differences in the means for the endemic and non-endemic groups and the corresponding t-statistics.

We note that a higher proportion of children know some math and can do some reading in non-endemic districts compared to endemic districts. The same goes for the overall numeracy and literacy score, ranging from 0-4. Households in districts predisposed to iodine deficiency seem to be worse off with respect to many characteristics. As Adhvaryu et al. (2018), Politi (2010*b*,*a*) show that iodised salt improves human capital and income for previously iodine deficient populations one cannot rule out the explanation that these districts are worse off due to long lasting iodine deficiency. Assuming that the endemic and non-endemic groups have experienced similar trends in household and village characteristics, these baseline differences should not be a problem in a DD analysis. I investigate whether compositional differences or differential trends in other predictors of test scores constitute threats to identification in Section 2.6.

The trends in basic test scores are plotted for children aged 5-16 using pooled ASER surveys in goitre endemic and non-endemic districts in Figure 2.5. I use data on all children to plot the raw trends in basic cognitive scores, as restricting it to only primary school children as in the analytical sample, will omit older cohorts and thus not show any trends in test scores of children in early life before any attempt at mandating USI. The trends for children aged 5-10 are shown in Figure B.4 in the Appendix.

Figure 2.5: Proportion of children with basic skills by birth year and goitre endemicity using the ASER data





The figure depicts the proportion of children aged 5-16 in the ASER data who have mastered basic numeracy and literacy scores by birth year. The trends are plotted for children residing in goitre endemic and non-endemic districts separately. The blue areas represent the years of establishment of a ban on non-iodised salt by birth year.

Figure 2.5 shows that children from goitre endemic districts have lower basic academic skills across birth cohorts compared to children from non-endemic districts. In line with the hypothesis, we observe converging trends in the proportion of children mastering basic literacy and numeracy for cohorts born after mandatory USI in 2006. The absence of the policy also coincides with a divergence in test scores between children in endemic and non-endemic districts born between 2000 and 2006.

Given that the repeated annual cross-sectional ASER surveys test children aged 5-16 from 2007 to 2014, pooling the surveys results in earlier (later) cohorts comprising predominantly of children tested at older (younger) ages. The changing age-composition across birth years can explain the declining trend in basic test scores over time. The nationwide increase in school enrolment and a change in the composition of children who attend school also contribute to the overall drop in test scores. India passed the Right of Children to Free and Compulsory Education Act in 2009 (Kumar & Rustagi 2016). Under this act, every child up to the age of 14 is guaranteed free and compulsory education, and no child can be held back or be expelled until grade 10. The increase in enrolment has not been met with a corresponding increase in other inputs, such as classrooms, teachers and learning materials. Moreover, teachers in India have not been able to change teaching according to the changed composition in school enrolment as teachers must cover the entire year's formal curriculum according to law. Repeated surveys have found that Indian students perform significantly below grade-level standards in both math and reading, with little or no improvements over the past years (ASER Centre 2014).[26]

What is important for the DD identification strategy is to observe parallel trends in test scores for children in endemic and non-endemic districts prior to any effective mandatory USI policy. In fact, the trends appear to be parallel until birth year 1999, which is one year after the first brief attempt to mandate USI. As previously mentioned, factors decreasing the production and distribution of iodised salt occurred at the same time as the implementation of the ban of 1998 which reduced the efficacy of this ban.[27] The changing age-composition across cohorts is an important caveat to the interpretation of the trends in test scores for older cohorts as being parallel in Figure 2.5. As a large share of children from older cohorts are tested at older ages this might reduce their variation in basic skills. As the raw data does not permit a graphic investigation of the parallel trends assumption, the question is revisited in Section 2.5.1 where the changing age-composition

[26] Large surveys in other developing countries also find poor learning outcomes, see (Banerjee et al. 2016).

[27] Although, as discussed in Section2.3, the subsequent removal of this policy had substantial effects on the production and consumption of adequately iodised salt.

is controlled for among other factors.

Additionally, I inspect the long-run trends in literacy and schooling for endemic and non-endemic districts prior to the time period shown in Figure 2.5. I use information on the respondents' mothers' reading ability and on both parents' highest grade attained in school from the 2009 ASER.[28] I graph the changes in reading and schooling attainment for cohorts born in 1955 to 1982 as national iodine fortification of salt started in 1983, for parents living in historically endemic and non-endemic districts. From Figures B.7, B.8 and B.9 we cannot distinguish differing trends in literacy or schooling attainment across previously endemic and non-endemic districts.

2.5 Empirical analysis

I apply a DD strategy to investigate the impact of mandatory iodine fortification in early life, on cognitive test scores using the ASER data merged with the information on pre-fortification goitre endemicity. Cohorts who were in early life at the time of iodine fortification in historically goitre endemic districts, are likely to have experienced an improvement in cognition in comparison to cohorts in the same districts during no policy, relative to cohorts in districts with no prior goitre endemicity. I begin by presenting a flexible empirical specification where I do not constrain cohorts to be in a treatment or control group depending on year of birth, but rather let the data tell the story. Next, I define treatment status by the presence of the mandatory iodine fortification of 2006 in early life.

2.5.1 Preliminary analysis: Flexible treatment specification

I regress test scores on individual year of birth dummies interacted with the endemicity indicator variable. The model is specified in Equation 2.1;

$$\begin{aligned}\text{Test Scores}_{idt} = \alpha_0 + \sum_{t \neq 2000} \delta_t [yob \times Endemic] + \beta X_{idt} \\ + \phi_d + \phi_{yob} + \phi_{survey} + \phi_{survey*yob} + \mu_{idt}\end{aligned} \tag{2.1}$$

The outcome variable Test Scores, is a binary variable denoting basic literacy and numeracy skills for child i, in district d and born in year t. Linear probability models are estimated for basic numeracy and literacy skills separately. yob denotes year of birth dummies where the omitted reference year is 2000, consisting of children who were in utero

[28] Mother's literacy is only tested in the ASER survey of 2009.

during the first policy.[29] I choose birth year 2000 to be the reference group in order to show parallel trends prior to the bans on non-iodised salt. Even though a ban was implemented in 1998, it did not change the consumption of iodised salt compared to previous years. Therefore, in comparison to birth year of 2000, one would not expect any diverging trends in test scores for children born prior to 2000 in endemic and non-endemic districts.

Endemic is a binary variable which takes value 1 if child i resides in a pre-fortification goitre endemic district, and 0 if the child resides in a non-endemic district. The coefficient of interest is δ which captures the interaction effect of year of birth and goitre endemicity, in comparison to those born during the first mandatory salt iodisation policy in 2000. I include district fixed effects, ϕ_{d}, and year of birth fixed effects ϕ_{yob}. Interactions between birth years and survey years $\phi_{\text{survey*yob}}$, are added to control for changes in education for different years, and this also controls for the age of the child.

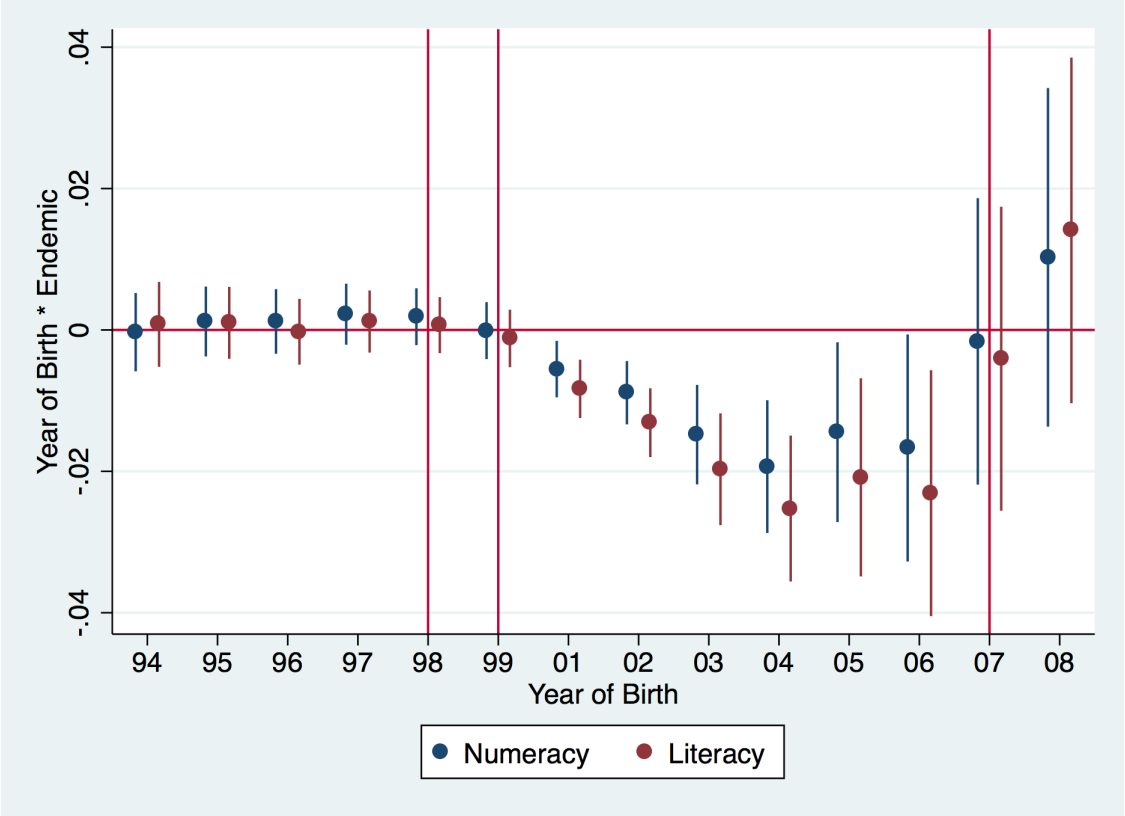
I add the following household level covariates; housing type (semi-pucca and pucca compared to the omitted category kutcha), years of maternal education and household size. I include the following dummy variables on whether the respondent's village of residence has: a government primary school, an Anganwadi centre and a rationshop. Moreover, I control for whether the village is connected to a pucca road. μ_{idt} is the error term. The standard errors are clustered at the district level to control for within-district serial correlation. I include children of all ages, 5-16 years old, as it allows for a better understanding of the trends prior to mandatory USI. One should keep in mind that the inclusion of all children in the data results in later cohorts comprising of a larger proportion of young children compared to older cohorts.

The graph in Figure 2.6 plots the coefficients on δ from Equation 2.1 for the pooled sample.[30] The probability of mastering any skill is given on the y-axis and year of birth is displayed on the x-axis. The dots represent the coefficients on basic numeracy and literacy for a child born in a given year, with reference birth year 2000, in an endemic district compared to a non-endemic district.

[29] The ASER data does not provide exact date of birth, only age at the time of the survey. I generate year of birth = survey year - current age; but this measure of iodine fortification policy at each age will be somewhat noisy.

[30] The graphs in Figure B.5 show the coefficients for boys and girls separately.

Figure 2.6: Leads and lags of birth year * endemicity



This graph uses data from ASER to plot the coefficients on birth year interacted with endemicity status of one's district of residence as specified in Equation 2.1. Controls for district level trends are omitted and the reference category birth year is 2000. The y-axis shows the magnitude of the coefficients, the x-axis represents the birth year and the lines through the plotted coefficients are confidence intervals.

We observe no statistically significant differences in test scores for children born prior to 2000 in endemic, compared to non-endemic districts. Also, we do not notice any direct or lagged positive effects from the implementation of the first ban in 1998 in comparison to older cohorts. This is in line with there not being a spike in iodised salt consumption following its implementation. As previously stated, this is likely to have been due to natural disasters striking the salt producing areas and changes to regulations of the salt industry occurring just before or at the time of its implementation. What is more important, is that one clearly sees that children in endemic areas performed significantly worse on basic numeracy and literacy scores if they were born during the absence of a central mandatory salt iodisation in 2001-2006, thus conceived in 2000-2005.

Learning outcomes improved for cohorts in endemic districts as they benefited from increased iodised salt availability after 2005. The estimates are less precise after 2002

which is likely to be caused by a reduced sample size for later cohorts and that later cohorts comprise of a larger share of children tested at younger ages.

We also observe an increasing positive trend in test scores after 2006 for children in previously endemic districts. Cohorts born in 2007-2008, thus being in utero at, or after, the time of implementation of the second ban in 2006, experienced an increase in cognitive outcomes, compared to children born during the absence of a ban. However, we cannot reject that the pre-2000 cohorts are statistically significantly different from the cohorts born in 2006 and 2007. This observation can possibly be driven by an increasing coverage of iodised salt over time following the policy. Alternatively, it might be due to the fact that an extended duration of mandatory iodine fortification before birth allows the mother to replenish previously depleted iodine stores. The findings are in line with Qian et al. (2005) who show that a positive impact of iodine supplementation on IQ is mainly observed in children born 3.5 years after such a programme was introduced.

2.5.2 Main analysis: Effects of the 2006 ban on non-iodised salt

In the following analysis, I will focus on the impact of the central prohibition of non-iodised salt notified in 2005 and implemented in 2006, compared to the absence of the ban, 2000-2005. The ban of 2006 led to a large increase in the coverage of iodised salt which increased with time and the ban is still in place today. I present the DD model in Equation 2.2:

$$\begin{aligned}\text{Human Capital}_{idt} = \alpha_0 + \delta\text{Iodised}_t + \gamma\text{Endemic}_d + \theta(\text{Iodised}_t * \text{Endemic}_d) \\ + \beta X_{idt} + \phi_d + \phi_{yob} + \phi_{district*yob} + \phi_{survey} + \phi_{survey*yob} + \mu_{idt}\end{aligned} \tag{2.2}$$

The outcome variables are jointly denoted as Human Capital. I estimate the probability of mastering basic numeracy and literacy and the effects on overall learning scores, for child i, in district d, born in year t. The regressions are estimated for 5-10 year old children to reflect the Indian primary school age.

Iodised is a binary treatment variable taking value 1 if the respondent was born in 2007-2008 and thus benefited from the fortification policy in utero and throughout his/her life. Iodised takes value 0 if the child was in early life during no federal policy, thus born in 2002-2004. The choice of control cohorts allows for a one year lag after the change in policy in 2000, and thus constitutes of children who were in utero 2001-2004. As the first 1000 days (foetal life up to age 2) are critical for overall brain development and for iodine intake (see Stinca et al. (2017)), the control cohorts must not have been exposed to the

policy from one year prior to birth up to age 2.[31]

Endemic is a binary variable denoting whether child i resides in a pre-fortification goitre endemic district. The coefficient of interest is the interaction term θ capturing the DD effect of being in early life during the nationwide mandatory iodine fortification policy implemented in 2006, compared to no policy, in naturally iodine deficient districts compared to iodine sufficient districts, on cognitive test scores over time. The regressions are estimated for the pooled sample and for girls and boys separately.

As in Equation 2.1, the following fixed effects are included; district fixed effects, ϕ_{d}, year of birth fixed effects ϕ_{yob}, interactions between birth year fixed effects and survey years $\phi_{\mathrm{survey*yob}}$. I account for district level specific trends, $\phi_{\mathrm{d*yob}}$ so that θ is estimated from the variation around linear district time trends. Partialling out district trend variation is required due the large size and population of Indian districts. Moreover, districts are the key administrative units administering all major programmes in the Indian education system (Department of Education 1993). When the regressions are estimated for the pooled sample of girls and boys, I include gender specific district linear time trends.

I further control for the type of house the child lives in, years of maternal education, household size and village characteristics. μ_{idt} is the error term and the standard errors are clustered at the district level. The regression estimates are presented with and without village level controls as the ASER surveys from 2007 and 2008 do not contain information on village characteristics.

The regression results for basic numeracy and literacy, i.e. the likelihood of recognising simple numbers and letters or better, are shown in Table 2.2. From column (2) we observe that children who benefited from the prohibition of non-iodised salt in early life experienced an increased probability of recognising single digit numbers or more, by 2.6 percentage points, after the inclusion of all covariates. Splitting the sample by gender reveals that girls benefited somewhat more. From column (6) we see that girls who were in early life after the implementation of the mandate were more likely to have obtained basic numeracy skills by 3.4 percentage points. The corresponding DD coefficient for boys is nearly half the effect size and indicates that boys improved their probability of mastering basic numeracy skills by 1.9 percentage points, see column (10). The gender differences are not statistically significant. The proportion of girls aged 5-7 with some numeracy skills is 70.07% and the corresponding proportion is 71.11% for boys. Exposure to a higher availability of iodised

[31] The econometric specification possibly underestimates the true effect of mandatory USI on cognition. The cohorts in the control group were in early life between two iodine fortification policies and therefore their control status might be confounded due to the storage and depletion of iodine.

salt in early life improved basic numeracy skills by 4.81% for girls and with 2.67% for boys.

Table 2.2: Effect on basic skills

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.014	0.026***	0.019**	0.030***	0.023**	0.034***	0.031***	0.040***	0.006	0.019*	0.009	0.022**
	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.011)	(0.010)	(0.011)	(0.010)	(0.011)	(0.010)
Mother's education	0.010***	0.010***	0.011***	0.011***	0.010***	0.010***	0.012***	0.011***	0.010***	0.009***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.036***	0.034***	0.037***	0.034***	0.035***	0.033***	0.036***	0.033***	0.036***	0.035***	0.038***	0.035***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.064***	0.062***	0.068***	0.064***	0.065***	0.063***	0.069***	0.065***	0.063***	0.061***	0.067***	0.063***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000*	-0.000*	-0.001***	-0.001***	-0.000	-0.000	-0.001**	-0.001**	-0.000	-0.000*	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.814***	2.786***	-0.316	-0.828								
	(0.991)	(1.055)	(0.985)	(1.038)								
Gvt primary school in vlg		0.012***		0.018***		0.011***		0.017***		0.013***		0.020***
		(0.003)		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg has anganwadi		0.002		0.001		0.003		0.001		0.001		0.002
		(0.003)		(0.003)		(0.004)		(0.004)		(0.003)		(0.004)
Vlg is connected to a pucca road		0.011***		0.009***		0.011***		0.009***		0.010***		0.009***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.010***		0.011***		0.010***		0.010***		0.011***		0.011***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Observations	824511	692890	828556	696175	384636	324064	386531	325576	439875	368826	442025	370599
R^2	0.843	0.855	0.832	0.842	0.841	0.852	0.831	0.841	0.846	0.857	0.833	0.843

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

The effect of iodine fortification in early life is slightly larger for the probability of being able to at least recognise letters, compared to the previously discussed effects on basic numeracy skills. The fortification policy increased the probability of having some literacy skills with 3 percentage points for the pooled sample after the inclusion of all covariates, see column (4) in Table 2.2. The DD coefficients are 0.04 for girls (see column (8)) and 0.022 for boys (see column (12)). The gender differences are not statistically significant. The estimates correspond to an increase of 5.83% in the likelihood of having basic literacy skills for girls (mean 68.66%) and 3.21% for boys (mean 69.24%).

I investigate the effects of iodine fortification on the overall numeracy and literacy score, ranging from 0-4. 0 corresponds to failing to recognise any letters or numbers. A score of 4 is given to children who can read a paragraph or do division and corresponds to what is required from a second or third grader in Indian primary education. Due to the relatively young sample of children who benefited from the policy (5-7 year olds), one might not expect a large effect on the total learning score. I estimate the effect on age standardised numeracy and literacy scores. While no effects are found for boys, a positive and statistically significant improvement of 6.6% of a standard deviation increase in girls' overall age standardised literacy score, see column (8) in Table 2.3.[32] The larger effects of an increased access to iodine on literacy compared to numeracy, corroborate the findings in Huda et al. (1999). The authors argue that reading skills are more likely to reflect a long-term cumulative process of the children's learning rather than current functioning.

[32]The effects on the raw score ranging from 0-4 is consistent with the impact on the age standardised scores, see Table B.5.

Table 2.3: Effect on age standardised overall test scores

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
standard deviation of the overall score in	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.009	0.013	0.041*	0.037*	0.026	0.019	0.078***	0.066**	-0.005	0.007	0.009	0.011
	(0.021)	(0.019)	(0.021)	(0.020)	(0.025)	(0.024)	(0.027)	(0.027)	(0.025)	(0.025)	(0.025)	(0.025)
Mother's education	0.043***	0.043***	0.043***	0.043***	0.044***	0.044***	0.044***	0.045***	0.042***	0.042***	0.041***	0.042***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.109***	0.107***	0.122***	0.120***	0.103***	0.101***	0.115***	0.114***	0.114***	0.113***	0.127***	0.124***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.005)	(0.006)	(0.006)	(0.006)
Pucca house	0.242***	0.242***	0.245***	0.242***	0.238***	0.238***	0.248***	0.247***	0.245***	0.245***	0.242***	0.238***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)	(0.006)	(0.006)	(0.006)	(0.007)
Household size	-0.001	-0.001*	-0.001	-0.001	-0.001*	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	-5.667***	-8.500***	-4.038*	-4.601*								
	(2.105)	(2.332)	(2.217)	(2.470)								
Gvt primary school in vlg		0.045***		0.050***		0.050***		0.049***		0.041***		0.051***
		(0.010)		(0.011)		(0.012)		(0.012)		(0.012)		(0.012)
Vlg has anganwadi		0.004		0.006		0.006		0.006		0.003		0.007
		(0.009)		(0.009)		(0.009)		(0.010)		(0.010)		(0.010)
Vlg is connected to a pucca road		0.033***		0.032***		0.034***		0.031***		0.032***		0.034***
		(0.006)		(0.006)		(0.006)		(0.007)		(0.006)		(0.007)
Vlg has ration shop		0.041***		0.037***		0.039***		0.036***		0.043***		0.038***
		(0.005)		(0.005)		(0.006)		(0.006)		(0.006)		(0.006)
Observations	824511	692890	828556	696175	384636	324064	386531	325576	439875	368826	442025	370599
R^2	0.175	0.186	0.145	0.154	0.198	0.211	0.161	0.171	0.157	0.166	0.133	0.141

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are age-standardised overall numeracy and literacy scores for children aged 5-10 from all states but Kerala. The raw score is in the range of 0-4, where 0 denotes no numeracy or literacy ability and 4 denotes that the child can master reading a paragraph or do division, respectively. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

The results also largely hold when using the standardised number of historically goitrous areas as defined in (McCarrison 1915) per 2001 districts, in place of the binary measure. A one standard deviation increase in pre-fortification goitre endemic locations per district improves basic numeracy and literacy by 1.6 and 2.2 percentage points, respectively, for girls. The corresponding increase for boys' basic literacy score is 0.9 percentage points while no effects are observed on basic numeracy, see Table B.14 in the Appendix. Consistent with the main results, girls also improved their overall age standardised test scores, see Table B.15 in the Appendix.

The observed improvements in cognitive test scores are not driven by increased school enrolment (see Table B.6 in the Appendix). This is not surprising as primary school enrolment has become near universal in India in recent years. We note that iodine fortification is associated with a reduction in the likelihood of dropping out of school of 0.2 percentage points for the pooled sample, see column (4) in Table B.6 in the Appendix. However, no significant effects are found on dropping out for either gender separately.[33]

Children in private schools have, on average, better test scores (Muralidharan & Kremer 2009). Differential trends in private school enrolment in areas more or less prone to iodine deficiency might pose a threat to the identification strategy. I investigate this by regressing the probability of being enrolled in a private school compared to a government or Madrasa (islamic) school, on the right hand side variables in Equation 2.2. I do not find that the increased basic cognitive test scores for children in endemic areas are caused by coincidental increases in private school enrolment, see Table B.7 in the Appendix. Neither do I find any effect on the probability of taking private tuition (tutoring outside of school), see Table B.8 in the Appendix. These findings further strengthen our confidence that the improved numeracy and literacy skills are driven by increased cognitive skills.

Heterogeneous treatment effects: State level differences in the monitoring of iodised salt

I analyse whether the differences in basic cognitive test scores vary with changes in the availability of iodised salt across policy regimes. Gujarat was the only major salt producing state which revoked its state ban in 2000 following the removal of the first national

[33]There is some evidence of a positive impact on grade progression for both genders. However the effect size decreases significantly and the estimates are no longer statistically significant when village level covariates are included, see Table B.4.

mandate.[34] The mode of salt transport from Gujarat is determined by distance as it is more cost effective to use road transportation for shorter distances and rail transportation for longer distances (Vir 2011, p.586).

Salt transported by rail is subject to monitoring and registration of the producer. Moreover, controls of iodine content of salt are only obligatory in transport by rail but not by road by federal policy. These rules applied before and after the implementation of the federal bans. This created less incentive for the salt producers and distributors to adequately iodise salt which was going to be transported by road (Vir 2011, p.586). The north eastern states; Sikkim, Mizoram, Meghalaya, Nagaland, Tripura, Arunachal Pradesh, Manipur, Assam and West Bengal import their salt by rail due to their far off location. These states also use a nominee system which consists of appointed traders who procure salt for the states. This system is biased in favour of large and registered salt producers who are more likely to produce adequately iodised salt and have their salt undergo inspections (Vir 2011, p.586).

Therefore, during the absence of central mandatory salt fortification, states within 500 km of Gujarat imported salt which was less likely to have been checked compared to salt transported by rail. States with rail transportation always had a higher proportion of households consuming iodised salt and were less affected by changes in the federal ban (Vir 2011).[35]

I plot the trends in iodised salt consumption for rural households in the north eastern states of India which import their salt by rail. I also plot separate trends for Gujarat and states within 500 km of Gujarat; Rajasthan, Uttar Pradesh, Madhya Pradesh and Maharahstra, which use predominantly road transportation by salt, see Figure B.6 in the Appendix. The graphs confirm the differential coverage of iodised salt over time. "Rail states" experienced an increase of around 11% of iodised salt consumption following the implementation of the ban in 2006. States relying on road transport from Gujarat experienced an improvement of over 25% following the legislation.[36]

[34] Besides meeting its own requirement, Gujarat caters to the North Eastern States, West Bengal, Bihar, Uttar Pradesh, Madhya Pradesh, Maharashtra, Goa, Rajasthan, Delhi, Jammu and Kashmir and Orissa.

[35] Other policies might additionally have affected the changes in the availability of iodised salt. For instance, in April 2001, the freight for transporting salt by rail experienced a price hike which led to an increase in the transportation by road. Most states offer subsidised salt through the public distribution system (PDS) but there is no support of PDS having affected the supply and consumption of iodised salt. As 3.9% of households in 2012 reported purchasing salt from PDS shops, the consumption of salt from PDS have remained low after the re-introduction of the ban. Moreover, quality assurance of adequate iodine levels in salt sold through PDS have been heavily criticised (Pandav 2012).

[36] We also note a larger discrepancy between "rail states" and "road states" is found in the proportion

I run separate regressions for children in states depending on their transportation mode of salt. Children in, and near Gujarat experienced stronger improvements in cognition if they were in early life during the ban on non-iodised salt. Mandatory fortification of salt during early life is associated with an increase in the probability of knowing some numeracy with 7.5 percentage points for girls and 4.6 percentage points for boys, see Table 2.4 below. The ban also improved basic literacy scores. Girls and boys are 6.8 and 3.6 percentage points more likely to know some literacy, respectively. I do not find any positive effects on test scores for children residing in "rail states", see Table B.13 in the Appendix.

of households consuming salt without any iodine.

Table 2.4: Effect on basic skills for children in Gujarat and in nearby states

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.032**	0.060***	0.026*	0.051***	0.046***	0.075***	0.045***	0.068***	0.020	0.046***	0.010	0.036**
	(0.014)	(0.014)	(0.014)	(0.014)	(0.016)	(0.017)	(0.016)	(0.016)	(0.016)	(0.017)	(0.016)	(0.017)
Mother's education	0.012***	0.011***	0.013***	0.012***	0.012***	0.012***	0.013***	0.013***	0.011***	0.011***	0.012***	0.012***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.044***	0.042***	0.044***	0.043***	0.043***	0.041***	0.044***	0.043***	0.045***	0.042***	0.045***	0.042***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca house	0.077***	0.076***	0.079***	0.078***	0.078***	0.079***	0.080***	0.081***	0.075***	0.074***	0.078***	0.075***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)
Household size	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	-0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	5.397***	5.129***	1.640	0.956								
	(1.668)	(1.847)	(1.631)	(1.753)								
Gvt primary school in vlg		0.019***		0.024***		0.023***		0.026***		0.016**		0.023***
		(0.006)		(0.006)		(0.007)		(0.007)		(0.007)		(0.007)
Vlg has anganwadi		0.003		0.008		0.004		0.009		0.001		0.008
		(0.005)		(0.005)		(0.006)		(0.006)		(0.005)		(0.006)
Vlg is connected to pucca road		0.006*		0.004		0.007		0.001		0.006		0.006
		(0.004)		(0.004)		(0.004)		(0.005)		(0.004)		(0.004)
Vlg has ration shop		0.010***		0.010***		0.012***		0.011***		0.008**		0.010***
		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)
Observations	342979	289137	344167	290087	156614	132592	157155	133009	186365	156545	187012	157078
R^2	0.811	0.821	0.803	0.811	0.805	0.814	0.798	0.806	0.816	0.827	0.807	0.816

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 in Gujarat, Rajasthan, Uttar Pradesh, Madhya Pradesh and Maharahstra. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Heterogeneous treatment effects: Son preference

The coefficients in this study are marginally larger on girls' basic skills, but the gender differences are not statistically significant across the baseline estimations. However, we observe that girls experienced a positive effect on the overall literacy score while no effects were found for the subsample of boys. Previous studies in economics which evaluate the impact of iodine in early life on human capital have found larger effects on female human capital outcomes in childhood and adulthood. Field et al. (2009) explain this finding by female foetal brain development being more sensitive to iodine compared to male brain development. However, these papers do not observe cognition directly and there is no conclusive support for the suggested gender differential in iodine sensitivity in the medical literature.

It should also be mentioned that other papers which study the impact of various shocks in early life on schooling outcomes find larger effects for girls compared to boys (see; Bobonis et al. (2006), Maccini & Yang (2009), Maluccio et al. (2009), Hoynes et al. (2016), Bleakley (2007)). Moreover, it should be noted that it is difficult to disentangle the effect of nature versus nurture as we often do not observe parental behaviour, such as reinforcement or compensation with regards to observed cognitive endowments and gender.

I investigate whether the treatment effects vary with the preferences for sons in order to shed light on whether the cognitive gains from USI are affected by social gender institutions. Son preference is a well known fact in many parts of India. This has led to unbalanced sex ratios and adverse human capital outcomes for girls and women. This phenomenon is driven either by prenatal sex selection or lower investments in early life of girls leading to higher female mortality rates in infancy and childhood.[37]

I interact the DD variables with district level standardised sex ratios of the number of girls to 1000 boys aged 0-6 years from the 2001 Census. A larger sex ratio indicates that a district is more balanced with regards to gender and suffers less from observed son bias. The regression results are presented in Table 2.5 below. No effects of the ban with respect to son preference is found on test scores for girls. We can therefore rule out that the somewhat larger cognitive effects from USI for girls are driven by social gender institutions.

[37] On the other hand, there is evidence that sex selection could lead to a reduction in the prevalence of malnutrition among girls, see Hu & Schlosser (2015).

Table 2.5: Effect on basic skills by standardised son preference

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Sex Ratio* Iodised * Endemic	-0.005	-0.012*	0.002	-0.006	-0.003	-0.008	0.004	0.001	-0.006	-0.015**	-0.000	-0.011
	(0.007)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
Iodised * Endemic	0.016*	0.027***	0.019**	0.030***	0.023**	0.033***	0.030***	0.038***	0.009	0.021**	0.009	0.022**
	(0.009)	(0.009)	(0.009)	(0.009)	(0.010)	(0.010)	(0.011)	(0.010)	(0.011)	(0.010)	(0.011)	(0.010)
Mother's education	0.010***	0.010***	0.011***	0.011***	0.010***	0.010***	0.012***	0.011***	0.010***	0.009***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.036***	0.034***	0.037***	0.034***	0.036***	0.033***	0.036***	0.033***	0.036***	0.034***	0.038***	0.035***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.064***	0.062***	0.068***	0.064***	0.065***	0.062***	0.069***	0.065***	0.063***	0.061***	0.067***	0.063***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000*	-0.000*	-0.001***	-0.001***	-0.000	-0.000	-0.001**	-0.001**	-0.000	-0.000*	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.908***	2.820***	-0.190	-0.745								
	(0.997)	(1.063)	(0.995)	(1.049)								
Gvt primary school in vlg		0.012***		0.018***		0.011***		0.017***		0.012***		0.019***
		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg has anganwadi		0.003		0.002		0.004		0.002		0.002		0.002
		(0.003)		(0.003)		(0.004)		(0.004)		(0.003)		(0.004)
Vlg is connected to a pucca road		0.011***		0.009***		0.011***		0.009***		0.010***		0.009***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.010***		0.010***		0.010***		0.010***		0.010***		0.011***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Observations	809296	680194	813246	683395	377336	317906	379174	319366	431960	362288	434072	364029
R^2	0.842	0.854	0.831	0.841	0.840	0.851	0.830	0.840	0.845	0.857	0.832	0.843

Notes: This table reports the coefficients from a similar specification as in Equation 2.2, but the treatment variable is now interacted with son preference per district. I use the ASER data merged with historical information on district level goitre endemicity and standardised sex-ratios per district. The sex ratio data stems from the Indian census of 2001 and represents the number of girls to 1000 boys aged 0-6 years. The independent variable of interest is the interaction term between benefiting from iodine fortification and one's sex ratio per district. The outcome variables are; the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters), for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

In column (10) we note that a more balanced sex ratio have a negative impact of mandatory USI on boys' cognitive test scores. If boys are less sensitive to iodine in early life, this differential effect can potentially be explained by differences in parental reinforcement of observed cognitive endowments depending on son-preference. Adhvaryu & Nyshadham (2014) build upon the study by Field et al. (2009) and observe that postnatal investments such as vaccinations and breastfeeding responded to the positive shock to cognition and thus reinforced the positive effect on human capital. Additionally, previous research finds that parents in India do not notice higher abilities of their daughters while they do so for their sons, see (Chari & Maertens 2014). Given this evidence, parents in districts with a higher son preference might be more likely to reinforce (smaller) observed cognitive abilities of their sons, and not their daughters.

2.6 Robustness

2.6.1 Validity tests

The DD estimates are only valid if the variation in iodised salt policies in early life did not coincide with other policies affecting malnutrition, cognition and future schooling. Therefore, I conduct several robustness checks to test for other potential drivers of the main results.

I begin by testing whether the treatment is systematically correlated with changes at the household or village level. A correlation between the treatment and the changes in these observable characteristics would make us worried that the same could apply to unobservable variables. I stepwise regress the change in one household or village level covariate which was previously used as a control variable in Equation 2.2 while all the other control variables remain unchanged. I then estimate similar regressions but without including the other covariates; see Tables B.34 and B.35 for the regression results in the Appendix.

There are no statistically significant differences in years of maternal education, the likelihood that a child's village of residence has a government primary school, ration shop or is connected to a pucca road. I do find a negative effect on the probability of living in a pucca house and the village having an Anganwadi centre. However, these small and negative effects rule out the possibility that any improvement in either household wealth or accessibility to an Anganwadi are causing the improvement in cognitive skills.

Additionally, I investigate whether the DD estimates are driven by differential health

care investments, varying disease or sanitation environments of children during early life. A potential threat to internal validity is the roll out of the National Rural Health Mission. The programme was implemented in 2005 and has decentralised and improved the quality of the health delivery system in deprived rural areas. It has for instance improved immunisation coverage and quality. I estimate placebo regressions using the econometric strategy set out as in Equation 2.2. Data on health investments for the last and second to last born child to surveyed woman in the DLHS II and III is used for the analysis. The outcome variables are the probability of being vaccinated against BCG and Measles. In addition, I analyse whether newborn children born after the implementation of a ban on non-iodised salt were more likely to receive another micronutrient supplement - Vitamin A. Lastly, I estimate the effects on the incidence of diarrhoea two weeks prior to the survey. Diarrhoea incidence is an overall proxy for the health and sanitation environment. The regression results are presented in Table B.36. All estimates but one are statistically insignificant. We notice that the probability of receiving vaccination against BCG is 5.7 percentage points lower for birth cohorts who were in early life during the 2006 ban on non-iodied salt in historically goitre endemic districts. These findings reduce potential worry about improved health care services and health in early life causing the observed improvements in cognitive test scores.

2.6.2 Effects of the 1998-2000 ban

In order to prove that the DD effects are not driven by coincidental improvements in cognition of children born after 2006, I show that children who were in early life during the first ban of 1998 also experienced increases in learning outcomes. Given the previously discussed caveats regarding the short duration of the first ban and the importance of iodine for postnatal brain development, one would not expect the treatment effects to be as large as from the later ban. I estimate the same regression specification as in Equation 2.2 with the exception that the treatment cohorts now capture children who were in early life during the 1998-2000 ban on non-iodised salt. Treatment is defined as; Iodised (1^{st} ban) = 1 if a child was born in 1999-2000, corresponding to children who were in utero in 1998-2000, and 0 if the child was born in 2002-2004. In addition, I investigate the treatment effect of both bans together by pooling the cohorts from the first and second ban together. Iodised (Both Bans) = 1 if the child is born in 1999, 2000, 2007 or 2008 and equals 0 if the child is born in 2002-2004.

The first ban has a positive and statistically significant effect on the probability of

knowing basic literacy for both boys and girls, see Table B.9 in the Appendix. Only boys appear to have experienced improved basic numeracy skills following the ban of 1998. The effect sizes are smaller compared to the later ban. We observe rather similar findings on age-standardised overall test scores. No effects are found on girls' test scores but boys experienced an improved age standardised overall literacy and numeracy scores across all specifications; see Table B.11 in the Appendix. Combining cohorts affected by both bans result in positive and significant effects on basic skills and for age standardised overall test scores for all sub samples; see Table B.10 and B.12 in the Appendix.

2.6.3 Results using district level total goitre rate

In this subsection, I present regression results using data on the pre-iodisation total goitre rate of school aged children per district. I estimate Equation 2.2 but I substitute the former goitre endemicity dummy variable with a dummy variable taking value 1 if a district has an above median goitre rate, and 0 otherwise. When interpreting these DD estimates we need to keep in mind that they are showing the difference for children residing in districts with a high risk for naturally occurring iodine deficiency compared to those at risk for mild to moderate iodine deficiency.

The results are presented in Table B.19 in the Appendix. The findings largely support the main estimates. Children residing in districts with a previously high goitre rate who were in early life during the ban on non-iodised salt of 2006 are 1.9-4.5 percentage points more likely to know basic numeracy or literacy. However, the effects are only significant at a conventional level of statistical significance when village level covariates are excluded. The control group in this dataset consists of districts with some pre-fortification goitre prevalence. The lack of statistical significance can potentially be explained by the absence of a definite control group in this selected sample. Another possible explanation is the reduction in sample size. The corresponding effects on basic numeracy are improvements by 1.7 and 1.9 percentage points respectively. Using this measure of pre-fortification iodine deficiency results in positive effects on overall age standardised numeracy and literacy for both boys and girls. However, the estimates do not reach statistical significance when I include village level controls, see Table B.20 in the Appendix.

2.6.4 Instrumental variable analysis

The data on goitre endemicity per district might potentially suffer from measurement error. As previously discussed, there is not sufficient information about the sampling

methods, nor the collection of data in McCarrison (1915) and in the district level TGR surveys. In order to address measurement error, I instrument for pre-fortification goitre endemicity using topological and hydro-geological determinants of the iodine content in the soil and groundwater.[38] This is a valid strategy provided that the measurement error in the geographical data is uncorrelated with the measurement error in either goitre dataset. This is a reasonable assumption given the different contexts and methods of data collection.

The iodine content in soils is determined mainly by soil type and locality. Most iodine in soils is derived from the atmosphere where, in turn, it has been derived from the oceans. Iodine deficiency in the soil-water ecosystem is due to heavy rainfall, steep gradient and poor vegetation cover resulting in quick run-off and little time for transfer of iodine (Fuge 2007). Soil erosion and leaching leads to iodine deficient soils and hilly topography encourages natural erosion of the surface layers (Brady 1996, pp.48-49). Iodine deficient soils are therefore common in mountainous areas (Zimmermann 2009). Drinking water accounts for 10-20% of total iodine intake (Rasmussen et al. 2002). High concentrations of iodine in ground waters can be found in saline waters (Smedley 2004). The majority of the iodine in the groundwater stems from organic matter decomposition in the marine strata with sea water influence (Wen et al. 2013).

I instrument district level goitre endemicity by maximum elevation and ground water salinity. The elevation data comes from the Shuttle Radar Topography Mission from FAO Harmonized World Soil Database v 1.2. I have geo-traced the location of the degree of saline ground water from a map on groundwater quality in shallow aquifers from the Central Ground Water Board in India (Central Ground Water Board 2010) using QGIS. I estimate the following first stage regressions:

$$\text{Goitre Endemicity}_d = \alpha_0 + \delta \text{ Elevation}_d + \gamma(1/\text{Groundwater Salinity})_d + \phi_d + \mu_d \tag{2.3}$$

$$\text{TGR}_d = \alpha_0 + \delta \text{ Elevation}_d + \gamma(1/\text{Groundwater Salinity})_d + \phi_d + \phi TGRsurveyyear + \mu_d \tag{2.4}$$

The first stage regression results are presented in Table B.21 in the Appendix. The inverse of ground water salinity per district and the maximum elevation per district are relevant predictors of district level goitre endemicity based on McCarrison (1915), prior to the introduction of iodised salt. The first stage F-statistic is 61.31. The variables are

[38] This is similar to Cutler et al. (2010) who instrument for malaria endemicity.

also relevant predictors of above median TGR where the F-statistic is 18.19. I control for the timing of the various TGR surveys due to the possibility that more goitrous districts were sampled first. Controlling for the survey year reduces the predictive power of the instruments, but the F-statistic on the instruments are just barely under the rule of thumb cut off - 9.33.

The IV results from Equation 2.4 are presented in Table B.22. I test the overidentifying restrictions using the Hansen J Statistic when estimating the main specification. The χ^2 p-value for the overidentification test is reported at the bottom of the table. I fail to reject the hypothesis that the elevation and groundwater salinity variables are jointly valid instruments conditional on all covariates. Instrumenting for the goitre endemicity variable, the DD coefficients show that the fortification policy implemented in 2006 increased the probability of having basic numeracy skills with 7.6 percentage points for the whole sample. The TSLS coefficients show that girls experienced an increase in their probability of knowing basic numeracy by 10.5 percentage points. The corresponding increase for boys is half the effect size as for girls, 5.2 percentage points. The coefficients for boys and girls are statistically significantly different from each other. Girls and boys experienced an increase of 15% and 7.3%, respectively, in the probability of knowing any math at their respective group means.[39]

Girls who benefited from prohibition of non-iodised salt during early life are 14.4 percentage points more likely to have attained basic literacy skills. The corresponding increase is 9.3 percentage points for boys. The gender differences in the effects on literacy are also statistically significant. The coefficients correspond to an improvement of 20.8% for girls and 13.4% for boys.

[39]No effects are found on the probability of having enrolled in school.

Table 2.6: IV Results: Effect on basic skills

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is basic :	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.080***	0.076***	0.122***	0.116***	0.102***	0.105***	0.139***	0.144***	0.061*	0.052*	0.107***	0.093***
	(0.029)	(0.027)	(0.031)	(0.030)	(0.033)	(0.032)	(0.035)	(0.035)	(0.033)	(0.031)	(0.035)	(0.033)
Mother's Education	0.010***	0.010***	0.011***	0.011***	0.011***	0.010***	0.012***	0.011***	0.010***	0.010***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Kutcha House	0.036***	0.034***	0.037***	0.035***	0.036***	0.033***	0.036***	0.034***	0.037***	0.035***	0.038***	0.035***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca House	0.064***	0.062***	0.068***	0.064***	0.065***	0.062***	0.068***	0.065***	0.063***	0.061***	0.067***	0.063***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000*	-0.000*	-0.001***	-0.001**	-0.000	-0.000	-0.001**	-0.000*	-0.000	-0.000*	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.825***	2.785***	-0.308	-0.815								
	(1.002)	(1.065)	(1.003)	(1.056)								
Gvt Primary School in Vlg		0.012***		0.018***		0.011**		0.016***		0.013***		0.020***
		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg has Anganwadi		0.003		0.001		0.004		0.002		0.002		0.001
		(0.003)		(0.003)		(0.004)		(0.004)		(0.003)		(0.004)
Vlg has pucca road		0.011***		0.009***		0.011***		0.009***		0.010***		0.008***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.011***		0.010***		0.011***		0.010***		0.011***		0.010***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Overidentification test	11.605	0.010	5.003	0.402	4.841	0.790	2.985	1.020	15.074	1.137	5.179	0.029
χ^2 p-value	0.0007	0.9207	0.0253	0.5258	0.0278	0.3741	0.0841	0.3126	0.0001	0.2862	0.0229	0.8652
Observations	805829	677364	809727	680542	376108	316986	377937	318455	429721	360378	431790	362087
R^2	0.021	0.022	0.024	0.024	0.022	0.023	0.025	0.026	0.021	0.021	0.023	0.024

This table reports the regression results from the TSLS estimation specified in Equation 2.4. Historical goitre endemicity is instrumented with inverse of a salinity score ranging 1-3 per district and the maximum elevation per district. The outcome variables are the respective probabilities of knowing basic numeracy and literacy for children aged 5-10 using the ASER data. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. The robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

IV analysis also increases the coefficients on overall age-standardised test scores for girls. Girls improved their standardised score in literacy by 26.7% of a standard deviation and by 17.4% of a standard deviation for numeracy, see Table B.23 in the Appendix. Applying IV estimation also results in positive and significant effects on grade progression, especially for girls; see Table B.24 in the Appendix.

Similarly, the coefficients increase greatly when an IV analysis is applied to the TGR data. Being in early life during a fortification policy increases the probability of knowing any numeracy with 12.8-17.00 percentage points for the pooled sample after the full subset of controls is included. The corresponding increase is 13.2-19.4 percentage points for basic literacy. In addition, the DD estimates are now statistically significant for all sub samples and we observe larger estimates for girls in comparison to boys after using TSLS.

These results suggest that the measurement error in the goitre datasets have led to an underestimation of the true effect of mandatory iodine fortification on test scores using OLS. Moreover, larger gender differences are found using TSLS. A potential explanation for this difference might be that McCarrison (1915) oversampled areas that were part of British India which also had lower levels of gender bias. Roy & Tam (2016) show that states which were a part of British India benefited from better legislation against female discrimination. The authors find that these historical institutions have persisted until today where girls fare better in areas that were part of British India, compared to areas that were independent princely states.

As an additional check using geographical predictors, I use variation in natural iodine availability stemming from the fact that coastal areas are less likely to be deficient as most iodine is derived from the oceans. Therefore, the few districts bordering the sea which have been deemed goitre endemic by McCarrison (1915) are likely to have a lower rate of goitre, compared to endemic districts in more inland regions. Following this logic, children who live in sea bordering goitre endemic districts should have experienced a smaller increase in cognition following iodine fortification in early life compared to more inland endemic districts. Consistent with this hypothesis, insignificant and smaller estimates are found on test scores when restricting the analysis to districts bordering the sea, see Tables B.27 and B.28 in the Appendix.

2.6.5 Inter-district trade in agricultural products

A final threat to internal validity stems from the fact that district boundaries might not be representative of the area of food markets. Therefore, the district level might not

be the correct spatial area to define the district population's current underlying risk of iodine deficiency. India is still dominated by smaller rural agricultural primary markets meeting local demand (FAO 2005). Despite trade liberalisation, internal trade remains low. Interstate tariffs, extensive trade regulations, and high transport costs constitute large barriers and affect rural households in particular (Atkin 2013).[40] In order to rule out that the main results are confounded by differences in road connectivity in early life and thus differential market access, I conduct a falsification test. I regress the probability of one's village of residence being connected to an all weather road applying the DD model specified in Equation 2.2 using data from the DLHS II and III. I do not find that road connectivity is associated with the treatment of interest (see Table B.29 in the Appendix).

Atkin (2013) proves that Indian agricultural markets consist of small segmented markets within states. He defines these markets using the regions from the National Sample Survey (NSS). The NSS regions are drawn along agro-climatic boundaries within states. As to allow for inter-district trade in agricultural produce, I now define goitre endemicity per NSS region. I compute and standardise the number of goitre points in McCarrison (1915) per NSS region. I control for NSS region specific time trends and cluster the standard errors on NSS regions. Otherwise, I estimate an identical DD model as specified in Equation 2.2.

After, controlling for all covariates, the DD estimates point to an increase in basic skills of 1.4 - 2.3 percentage points for the samples of boys and girls, see Table B.30 in the Appendix. As when using district goitre variation, we note that girls experienced an increase in their overall numeracy and literacy, see Table B.31. Moreover, these positive and significant effects remain when I change the level of analysis to standardised goitre areas per state, see Tables B.32 and B.33.

2.7 Conclusion

This study estimates the causal impact of mandatory iodine fortification of salt on cognitive test scores in rural India. I use a difference-in-differences strategy comparing cohorts who were in early life after the implementation of the policy to earlier cohorts, across districts with and without a geographical predisposition to iodine deficiency. As iodine deficiency is largely determined by the geography, I use historical information on the location of deficient areas to identify districts which are likely to benefit from the policy.

[40] External imports in Agriculture are low. In 2007/2008 agricultural imports were 4.32% of Indian GDP, see (Chand et al. 2010)

This information is merged with annual cross-sectional data on both in and out of school children's test scores for 2007-2014 from the Annual Status of Education Report.

Exposure to mandatory salt iodisation in early life increases the likelihood of children recognising simple numbers and letters or better, by 1.9 - 4 percentage points at ages 5-7. Girls also improved their overall literacy score which includes more difficult levels of mastery by 6.6% of a standard deviation. In comparison with studies on other inputs in early life using the same data on test scores, mandatory salt iodisation raises cognitive skills at least as much as avoiding drought in utero and more than being exposed to a sanitation campaign in early life, see Shah & Steinberg (2017) and Spears & Lamba (2016).

The results pass several robustness tests such as using an event study model, ruling out that other health improvements in early life or compositional changes are driving the findings and showing that a previous, although shorter, fortification policy also improved test scores. Taking account of trade in agricultural products across districts does not change the findings. Furthermore, I address the potential measurement error in the historical data on the spatial risk of iodine deficiency. I apply an IV analysis using geographical predictors of iodine content in soil and groundwater as IVs for the risk of the deficiency. The results from the IV analysis point to larger effects of salt iodisation on cognitive test scores. The main results also hold when using an alternative dataset on the rate of pre-existing iodine deficiency.

This is the first paper to use non-historical data on human capital to evaluate the cognitive returns from salt iodisation. As more than 140 countries have implemented USI legislation, these findings have global implications. Additionally, this study shows the mechanism by which historical salt iodisation has improved schooling attainment and labour market outcomes found in Adhvaryu et al. (2018), Politi (2010*b*,*a*) by directly investigating the effects on cognition in childhood. Moreover, this study provides important evidence of the efficacy of USI in a developing country context given the mixed conclusions in the current literature on iodine supplementation from the developing world, see Field et al. (2009), Bengtsson et al. (2017).

Previous papers find large gender differences in the treatment effects, and in some instances only positive effects for women. This study adds to the literature by revealing heterogenous effects by gender depending on the difficulty of the academic tests. Additionally, I investigate whether the effects vary with district level son preference. I do not find that the coefficients on female skills vary with area level son preference which suggests that the policy has the potential to close gender gaps in learning outcomes in

settings where women face discrimination. On the other hand, I find that the effect of salt iodisation on boys' numeracy skills are reduced in areas with more balanced gender ratios. This suggests that parents are more likely to reinforce observable higher cognitive attainment for boys in areas with higher son preference. Further research is needed to understand how parental inputs vary with exogenous shocks to cognitive endowments and whether such inputs vary with the gender of the child.

India was deemed iodine sufficient in 2016 but many low income countries in Africa and Asia are still iodine deficient and have low coverage of iodised salt consumption (Iodine Global Network. 2017). Thus, there are still gains to be made for many countries by ensuring commitment to USI, as even moderate and short term variation in iodised salt consumption will have persistent effects on cognition. Large effects of reaching USI can be expected for countries which have a very low proportion of households consuming adequately iodised salt. A back of the envelope calculation using the lower bound effects found in this study, suggests that increasing the national coverage of iodised salt from 10% to 90% could increase the proportion of children attaining basic academic skills by at least 10%.

Chapter 3

The impact of iodised salt consumption on children's height in rural India

3.1 Introduction

Height is often used as a proxy for individuals' accumulated health stock, particularly in studies analysing data from developing countries (Lundborg et al. 2009, Alderman et al. 2006, Spears 2012*a*, Hoddinott et al. 2013, Case 2008, Glewwe & Edward A 2007, Vogl 2014). Existing research has established the importance of adequate nutritional intake in early life (Hoddinott et al. 2013). However, less is known about what specific aspect of undernutrition constitutes a potential driver of height, and thus health capital. This chapter seeks to contribute to this knowledge gap by investigating the role of iodine intake on health capital.

Medical research suggests that iodine intake affects physiological processes involved in human growth (Zimmermann et al. 2007). It is therefore possible that the availability of iodised salt is a predictor of height among populations at risk for iodine deficiency. Mason et al. (2002) and Krämer et al. (2016) report positive associations between iodised salt availability and child growth among iodine deficient populations in low and middle income countries. Household consumption of iodised salt is likely to be correlated with omitted determinants of children's heights and contain measurement error. Simultaneity bias is also a potential threat to identification. The previous studies do not address these issuses and are therefore not able to estimate a causal impact of iodised salt consumption on growth. This chapter adds to the literature by exploiting exogenous variation in the

availability of iodised salt to estimate its effect on children's height-for-age Z-scores (HAZ), using a large household survey for rural India.

Despite a rapid economic growth, India has experienced slow improvements in child anthropometric outcomes (Deaton 2009). 38% of all Indian children under five years old are stunted, meaning that their HAZ is below two standard deviations compared to a healthy reference population. One in three of all stunted children worldwide live in India (Menon et al. 2018). It is therefore of large policy relevance to understand the causes of short stature in India. Previous studies have found that open defecation (Hammer & Spears 2016), inequality in human capital investment depending on birth order and gender (Jayachandran & Pande 2017) and female nutritional deprivation reducing nutritional intake in utero (Osmani & Sen 2003) are determinants of children's heights specific to the Indian context. Iodine intake is likely to be an additional predictor of height in India due to the high risk of iodine deficiency caused by the geography and diet (Pandav et al. 2003).

This empirical chapter uses exogenous variation in the regulation of iodised salt by state, prior to the nationwide mandatory USI. Around 80% of all salt in India is produced in Gujarat. Salt is then transported to other states either by rail or road. Previous studies such as Vir (2011), report a strong relationship between the proportion of salt transported by rail to a given state and the consumption of adequately iodised salt ($\geq$ 15 μg iodine g/salt) per state. This can be explained by differences in regulation across transport modes. National law mandates the control of iodised salt prior to rail transport while monitoring is not mandatory for salt undergoing road transportation. Distance from the salt producing state dictates the cost-effectiveness of a given transportation mode. States far away from Gujarat are more likely to have their salt transported by rail and nearby states are more likely to import their salt by road. Therefore, following the implications from differences in monitoring across forms of transportation, states far from Gujarat are more likely to have a higher access to iodised salt compared to households residing in Gujarat or in nearby states.[1]

Therefore, I use distance to Gujarat per state as an instrumental variable (IV) for the access to adequately iodised salt in a two-stage-least-squares (TSLS) estimation. The IV results indicate that the consumption of adequately iodised salt at the household level improves children's height-for-age by 0.664 standard deviations and height by 1.845 cm,

[1] Currently, the relationship between the mode of salt transport and the availability of adequately iodised salt is likely to be weaker due to nationwide mandatory USI. The latest nationwide survey, the 2015-2016 National Family Health Survey (NFHS) IV, points to a high consumption of salt containing some iodine across all states. However, it does not report the consumption of adequately iodised salt.

on average. The estimates are robust to the inclusion of covariates capturing infant and maternal health and nutrition status. I do not find any effects on weight-for-age Z-scores (WAZ). This is in line with the lack of a physiological relationship between thyroid hormones and adipose tissue among children. Additionally, the absence of an impact on weight also rules out the possibility that the main results are driven by an overall improvement in concurrent caloric availability. Specification checks show that the instrument is not systematically correlated with other determinants of child growth. The effect of the availability of adequately iodised salt on young children's height appears to be larger than the effects from other public policies. The results highlight the importance of access to adequate intake of micronutrients, such as iodine, for height and the overall accumulation of health capital.

This chapter is structured as follows. I begin with reviewing the evidence on the relationship between iodine and human growth in Section 3.2. Subsequently, I describe the production and transport of salt in India in Section 3.3. The data and the IV strategy is described in Section 3.4. The econometric strategy is presented in Section 3.5. Descriptive statistics are provided in Section 3.6 and the results are shown and discussed in Section 3.7. The validity of the results is tested in Section 3.8 and concluding remarks are reported in Section 3.9.

3.2 Iodine and height

Iodine deficiency from conception and onwards increases the risk of reduced thyroid hormone production. It has long been observed that foetuses and newborns to mothers with diseases characterised by thyroid problems have stunted growth, see Shields (2011).[2] Hypothyroidism, an illness where the thyroid gland does not produce enough thyroid hormone, is a well-recognised cause of short stature among children (Zimmermann et al. 2007). Delayed bone maturation is also observed in children with hypothyroidism (Robson et al. 2002). On the other hand, accelerated growth has been observed in children with hyperthyroidism. This is a condition where the thyroid gland produces excessive levels of thyroid hormones (Tarim 2011).

Thyroid hormones are involved in many physiological processes which determine growth both in utero and postnatally (Zimmermann et al. 2007). Normal thyroid hormone levels are required for growth and development of the skeleton and peripheral tissues. Addition-

[2]Examples of such illnesses are Graves' disease, hypothyroidism, or thyroid hormone resistance (Shields 2011).

ally, they are essential for the production and functioning of both growth hormone and insulin-like growth factors (Zimmermann et al. 2007, Robson et al. 2002). The relationship between thyroid hormones, such as thyroxin, and growth hormones and insulin-like growth factors has been observed across in-vivo and in-vitro animal studies (Ezzat et al. 1991, Samuels et al. 1989). Iodine supplementation of iodine deficient populations has been shown to increase insulin-like growth factors (Zimmermann et al. 2007).

In particular there seems to be a strong link between thyroid hormones and skeletal development (Robson et al. 2002). Thyroid hormones are needed for the expression of target genes to regulate skeletal development. The relationship is supported by animal studies and in-vitro studies among humans (Abu et al. 1997). There is no clear physiological relationship between thyroid hormones and soft tissue (Shields 2011).

Despite the established biological pathways, there is limited evidence on the effect of iodine on prenatal and postnatal growth and the overall findings are mixed (Zimmermann et al. 2007, Farebrother et al. 2018). A review on the impact of iodine supplementation of women during preconception and pregnancy by Harding & DeRegil (2017) finds no impact of the two studies which measure the impact on birth weight. Harding & DeRegil (2017) conclude that their meta-analysis should be interpreted with caution due to the limited number of studies comprising of low-quality trials from populations with mild- to moderate iodine deficiency. The observations might therefore not be applicable to areas with moderate-to severe iodine deficiency which are common in many middle and low income countries. For instance, Zimmermann et al. (2007) find that iodine repletion improves thyroid hormone levels and somatic growth of children who are severely iodine deficient but not for those who are moderately iodine deficient.

Farebrother et al. (2018) conduct a systematic review of the effects of any form of iodine supplementation, including iodised salt, of pregnant women and children on prenatal and postnatal growth outcomes. The review consists of 18 studies and concludes that while postnatal iodine repletion may improve growth factors, they are uncertain whether it affects somatic growth. The authors also note that the quality of the overall evidence is low. Additionally, most of these studies do not capture a long time period of exposure to iodine supplementation. Additionally, the possible relative effects on growth in utero and postnatally have not been established (Farebrother et al. 2018).

Studies using cross-sectional data find positive associations between iodised salt availability and child anthropometric status. Mason et al. (2002) observe significant associations between iodised salt use and child growth in Bangladesh, India (Andhra Pradesh),

Nepal, and Sri Lanka. In particular, they find positive associations for WAZ and mid-upper-arm circumference whereas the association with HAZ is weaker. However, the study only controls for a limited set of covariates and the effects are only reported for selected locations within the respective countries. Krämer et al. (2016) analyse the association between household unavailability of iodised salt and child growth across 46 low and middle income countries. They use 89 nationally representative, repeated, cross sectional demographic and health surveys (DHS) conducted between 1994 and 2012. The paper finds that the unavailability of iodised salt is associated with 3% higher odds of being stunted, 5% higher odds of being underweight and 9% higher odds of low birth weight. What is further interesting is that the sample for India is driving the associations for all outcomes except birth weight. When data from India is omitted from the analysis, only the association for low birth weight remains significant. It should also be noted that the main effects in Krämer et al. (2016) are not robust to the inclusion of covariates related to food intake.

The limited available cross-sectional evidence using survey data is likely to suffer from endogeneity issues such as omitted variable bias, measurement error regarding the duration of iodised salt use and retrospectively self-reported accounts of children's birth weights, and potential simultaneity bias. This study builds upon the extant literature by estimating a causal impact of adequately iodised salt on very young children's HAZ using a large nationwide survey for rural India. The endogeneity issues are circumvented by the application of IV regression.

3.3 The production and transport of salt in India

I use the distance to the major salt producing state of Gujarat as an instrument for the access to iodised salt. A lower consumption of iodised salt has been observed in Gujarat and states near Gujarat compared to states further away (Vir 2011, Kaur et al. 2017, Sundaresan 2009). The association between iodised salt availability and the proximity to salt producers is due to differential monitoring policies of salt transported by rail compared to road. Salt is more likely to be transported by rail if the salt producer is further away. Monitoring of iodised salt is only mandatory prior to rail, but not, road transport. In this section, I provide an overview of the Indian salt market with a focus on the production and export of salt from Gujarat. I emphasize the time period of 2005 and 2006 as the household data used for the analysis was collected during these years.

The most common type of salt in India stems from sea water evaporation.[3] The western coastal state of Gujarat has always been the largest producer and exporter of salt to the rest of India due to its long coast line and favourable climatic conditions for sea water brine evaporation. Salt production units in Gujarat are found in thirteen districts across the coastline and periphery of the salt marsh in the Little Rann of Kutch (Saline Area Vitalization Enterprise Limited 2005). During 2005-2006, Gujarat produced 77.5% of all salt consumed in India. Other salt producing states are Tamil Nadu, Rajasthan and Andhra Pradesh which produced 11.5%, 7.2% and 2.3% respectively of all salt in India in 2005-2006 (*Salt Commissioner India: Transport of salt by rail* 2006).

The majority of all salt produced in India and Gujarat comes from the private sector. Data for 2003 indicates that 62% of salt manufacturers are large scale producers (plots of over 100 acres). As per Indian federal policy, all large salt manufacturers have to obtain licenses and register with the Salt Department. Manufacturers of salt for local use are exempted from this. The next largest category (27%) comprises of small producers who hold up to 10 acres of land. They do not require licenses and are not registered with the Salt Department. Thus, there is a substantial proportion of small scale and unregulated salt producers and traders in India (Saline Area Vitalization Enterprise Limited 2005). The close proximity of salt producers and merchants in Gujarat has also helped them to form a strong lobby which influences the national supply and price of salt (Saline Area Vitalization Enterprise Limited 2005).

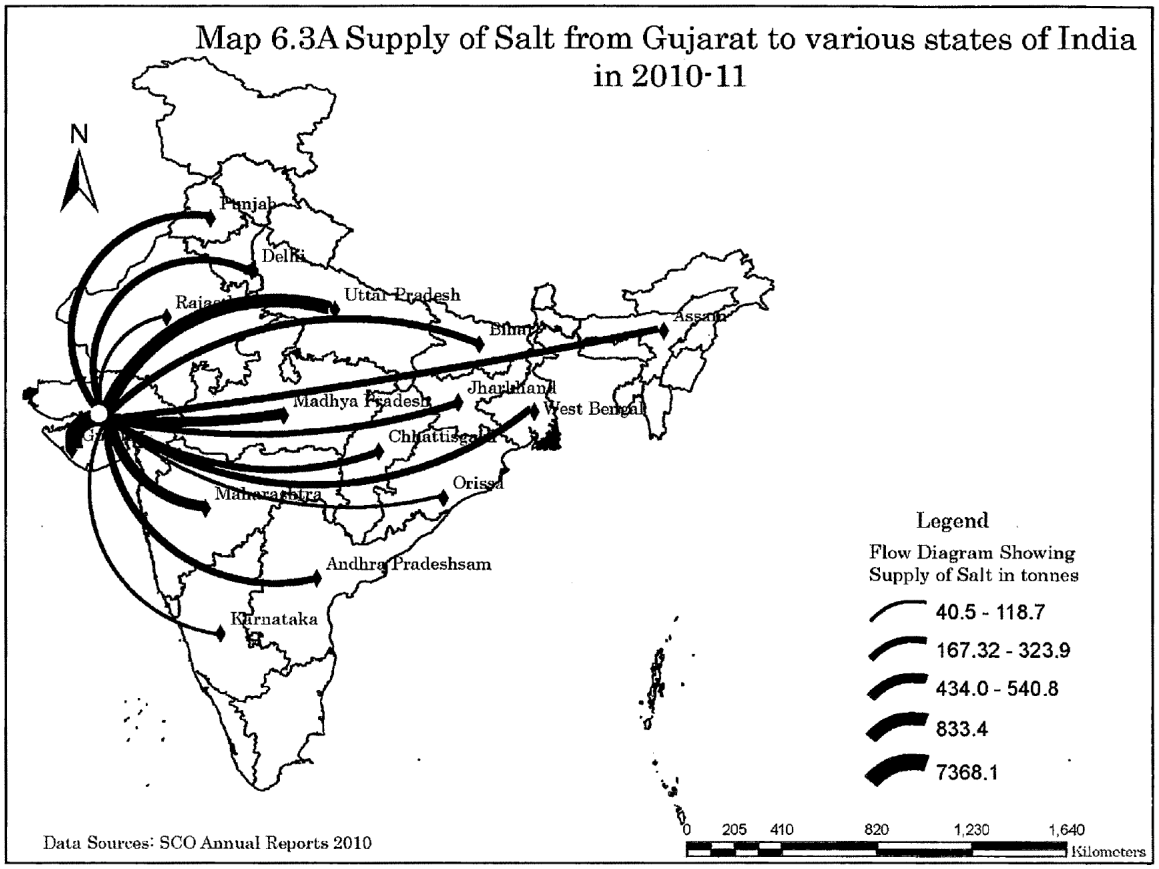
3.3.1 Monitoring and movement of salt

Salt production in Gujarat caters mainly to the eight north eastern states (Arunachal Pradesh, Assam, Manipur, Meghalaya, Mizoram, Nagaland, Sikkim and Tripura), West Bengal, Bihar, Uttar Pradesh, Madhya Pradesh, Maharashtra, Goa, Rajasthan, Delhi, Jammu and Kashmir and Orissa (Vir 2003). See Figure 3.1 for a visualisation of the size and destination of salt exported from Gujarat in 2010-2011 from the Salt Commissioner's Organisation, Department of Industrial Policy and Promotion (2011).[4] As seen from the figure, most Indian states import salt from Gujarat. The southern states import little or no salt from Gujarat. This is due to their close proximity to the next largest salt producing state - Tamil Nadu.

[3] Other types of salt production are inland salt from sub-soil ground water evaporation, lake salt from saline lakes and rock salt mining.

[4] I was not able to access comparable information for the time period of this study. Therefore, Figure 3.1 serves as an approximation of the export of salt from Gujarat during 2005-2006.

Figure 3.1: Export of salt from Gujarat



This figure depicts the inter-state salt export flows from Gujarat across India in 2010-2011 from the Salt Commissioner's Organisation, Department of Industrial Policy and Promotion (2011).

In 2006, about 57% of salt for human consumption moved by rail and the rest by road (*Salt Commissioners Organization* 2016).[5] The mode of transport is determined by distance. It is more cost effective to use road transportation for shorter distances and rail transportation for longer distances (Vir 2011, p.586). Sankar et al. (2006) observe that road transportation up to 1000 km appears to be more economical. As discussed in Chapter 2, a ban on the sale of non-iodised salt was implemented on the 17$^{\text{th}}$ of May 2006. The analysis in this chapter uses data just prior to its implementation. At this time, all states besides Gujarat and the small state of Arunachal Pradesh had a ban in place. These state level policies required that iodised salt should contain at least 15 μg iodine g/salt at the level of consumption. Therefore, the salt produced for export in Gujarat should be adequately iodised. On the other hand, salt produced both for domestic use

[5] A marginal proportion of all salt is transported by sea.

and inter-state export in Tamil Nadu and Rajasthan should be adequately iodised.

Monitoring of iodised salt occurs at the level of production. Officials from the Salt Department test the iodine content of salt at iodisation plants (Pandav et al. 2003). However, control of iodised salt is restricted to major salt producers only and it is not being carried out in a systematic manner. Therefore, the many small producers who are not registered with the Salt Department are not monitored (Sundaresan 2009). Thus, the control of iodised salt production is not sufficient to ensure adequate iodised salt supply, in particular from small producers.

Therefore, additional levels of monitoring play a key role. Salt rakes transported by rail undergo inspections from the Salt Department before it is loaded on the train. Only adequately iodised salt is given permission for transport. The control of salt transported by rail was implemented along the national "Iodine Deficiency Disorder Control programme" in 1973 (Kaur et al. 2017). Transportation of salt by rail also requires the registration of the producer which favours large salt producers for whom iodisation is less costly compared to smaller manufacturers (Kaur et al. 2017, Vir 2011, p.586). For example, the north eastern states and West Bengal import their salt by rail and use a nominee system which consists of appointed traders who procure salt for the states. This system is biased in favour of large and registered salt producers who are more likely to produce adequately iodised salt (Vir 2011, p.586).

On the other hand, there is no monitoring of salt transported by road. Therefore, small scale producers, who are less likely to comply with salt iodisation standards, often choose to transport their salt by road. Transportation by road also involves less capital compared to moving salt by rail which is further favourable for smaller producers (Kaur et al. 2017, Sundaresan 2009).

In summary, a non-salt producing state is more likely to import salt by rail compared to road if it is further away from the salt producer. Due to mandatory monitoring of iodised salt transported by rail, a greater distance to Gujarat increases the likelihood of access to adequately iodised salt.

3.4 Data

The main data source for this study is India's version of the Demographic Health Survey, the 2005-2006 National Family and Health Survey (NFHS) III. This survey is representative for households with at least one eligible woman aged 15-49 at the state level

(IIPS. 2007).[6] The survey consists of a rich variety of information, including household background characteristics, maternal and child health care utilisation and anthropometric status.

Surveyors measure the lengths and heights of all children aged 0-59 months. Children under 24 months are measured lying down and older children are measured standing up. For consistency, I refer to the length of children measured lying down as height. Height-for-Age (HAZ) is defined as the difference between an individual's height and the mean height of a same-aged healthy reference population defined by the WHO in 2006, divided by the standard deviation of the reference population (de Onis 2006). The calculated HAZ is provided in the NFHS. HAZ is frequently used as a measure for growth in developing countries including India, see for instance; Jayachandran & Pande (2017), Hammer & Spears (2016), Spears (2012*a*), Jain (2015). In particular it is thought to capture long term nutritional status and illness. The use of HAZ in the Indian context has further been discussed and validated by Tarozzi (2008). Additionally, I estimate the impact on height measured in cm and on the risk of being stunted. Stunting is defined as $\leq$ -2 HAZ and is thought to be a measure of severe long term nutritional deprivation.

The data contains information on objectively measured availability of adequately iodised salt at the household level. The NFHS surveyor measures the iodine content of the household's salt using a rapid-salt-testing kit. The survey reports the level of iodine available in the household in three categories; adequately iodised salt ($\geq$ 15 μg iodine g/salt), salt with some iodine or salt with no iodine. In the analysis, I omit the category referring to salt with an unknown level of iodine but below what is deemed adequate. This is due to the uncertainty regarding the dose-response relationship for the production and functioning of thyroid hormones.[7]

The analytical sample consists of rural households who were interviewed prior to May 2006 to reduce the risk that the access to adequately iodised salt is confounded by the implementation of the nationwide ban on non-iodised salt on the 17$^{\text{th}}$ of May 2006.[8]

[6]The 2005-2006 NFHS III does not contain district level identifiers due to anonymity concerns in the collection of HIV information.

[7]This also motivates the use of the 2005-2006 NFHS III for the analysis compared to the more recent 2015-2016 NFHS IV. The 2015-2016 NFHS IV only reports whether a household consumes iodised salt or not.

[8]I do not to use the exogenoues variation in iodised salt availability stemming from the implementation of the nationwide ban in May 2006. The 2005-2006 NFHS III does not show a discontinuity around the month of implementation which can be explained by the lack of instantenous enforcement of the ban discussed further in Chapter 2.

I further restrict the sample to households with children who are up to one year old at the time of the survey. This is because medical research indicates to that prenatal and early postnatal time periods are particularly sensitive for overall iodine intake (Zoeller & Rovet 2004). However, it should be noted that there is no consensus regarding the role of thyroid hormones for growth in utero compared to postnatally or for various early postnatal time periods, see Farebrother et al. (2018). As we cannot assume that a household which was found to consume iodised salt during the interview also did so previously, restricting the analysis to very young children is more likely to capture the effect of iodised salt during a larger proportion of their lives compared to selecting older children for the analysis.[9] The analytical sample consists of children up to 12 months with non-missing information on HAZ in households who consume either adequately iodised salt or salt with no iodine. As the source of exogenous variation is determined by the state of residence, I further restrict the sample to households where the mother reports to have been living in the same area for at least one year.[10]

Lastly I merge in information on the state's distance to Gujarat. I use the GIS software QGIS to first locate the centroid per state as of the 2001 Indian Census. I subsequently calculate the distance from each state's centroid to the centroid of Gujarat.

3.4.1 Definition of the instrumental variable

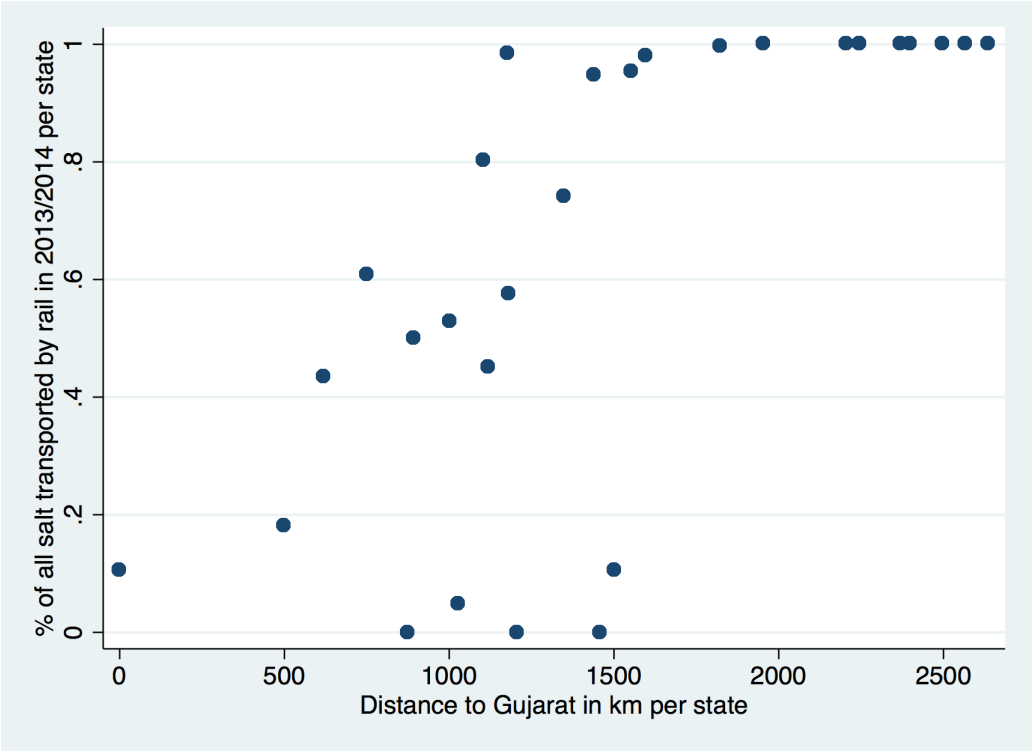
I start by investigating the relationship between the distance to Gujarat and the mode of transport per state. The earliest available information on the state-wise proportion of salt transported by rail compared to road is found in the 2013-2014 Salt Commissioner Annual Report, see Salt Commissioner's Organisation, Department of Industrial Policy and Promotion (2014). In Figure 3.2, I plot the relationship between the distance to Gujarat and the proportion of all salt transported by road compared to rail per state in 2013-2014. This figure serves as an approximation of the relationship during the 2005-2006 NFHS III. The report by Salt Commissioner's Organisation, Department of Industrial Policy and Promotion (2014) records that 26.5% of all salt in India in 2013-2014 moves by rail and 72.4% moves by road. This indicates an increase in the proportion of salt transported by road compared to the numbers reported for 2005-2006 by *Salt Commissioners Organization* (2016). Therefore, the scatter plot in Figure 3.2 should be interpreted with caution. Nonetheless, the anticipated relationship between distance from Gujarat and rail trans-

[9] Estimating the effect of current iodised salt use on previous (and self-reported) anthropometric outcomes such as birth weight, would also introduce much uncertainty.

[10] Inter-state migration in India is very small, see Topalova (2005).

port remains. From Figure 3.2 we note that there is a strong and non-linear positive association between distance to Gujarat and the proportion of all salt transported by rail compared to road. A clear threshold appears around 1500 km. Almost all states located further away than 1500 km from Gujarat import their salt by rail.

Figure 3.2: Distance to Gujarat and the proportion of salt transported by rail per state

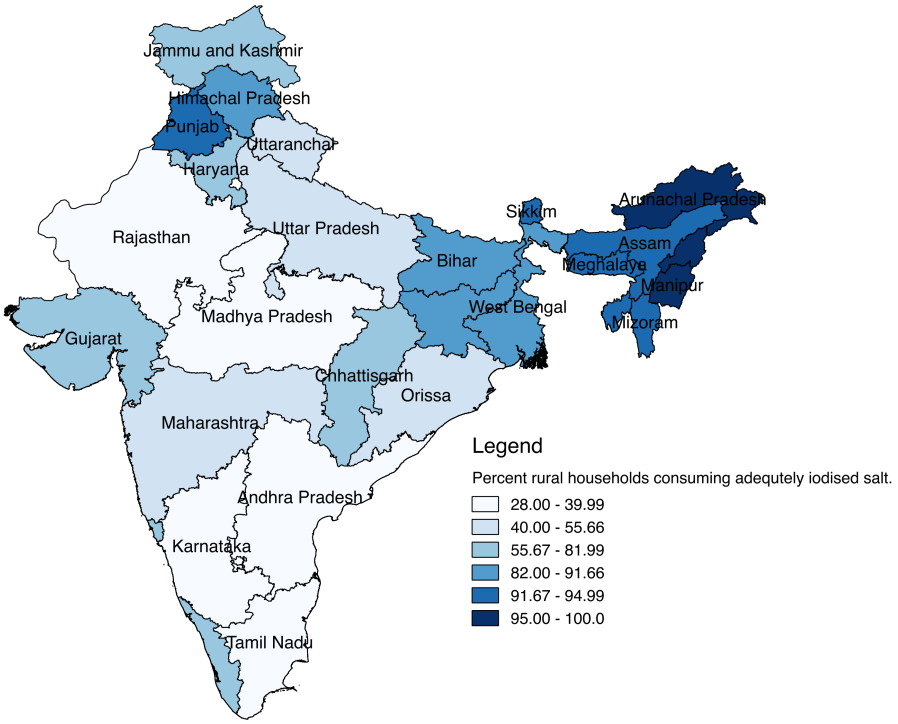


This scatter plot shows the relationship between the distance to Gujarat from each state centroid and the proportion of all salt being transported by rail per state in 2013-2014. Information on the proportion of all salt transported by rail versus road is given by Salt Commissioner's Organisation, Department of Industrial Policy and Promotion (2014).

To inspect the spatial relationship between the proximity to Gujarat and access to iodised salt further, I have mapped the proportion of households consuming adequately iodised salt using the 2005-2006 NFHS III per state in Figure 3.3 3.3. From Figure 3.3 we observe a relationship between the coverage of adequately iodised salt per state and its distance from Gujarat. This map also highlights the relative efficacy of monitoring policies related to the transport of salt compared to state level policies. All states but Gujarat and Arunachal Pradesh had a ban on the sale and consumption of non-iodised salt during the years of the survey. We note that the coverage of adequately iodised salt was almost universal in Arunachal Pradesh where salt is likely to have been transported

by rail despite the absence of a state level mandate. On the other hand, we observe that iodised salt use was low in states near Gujarat despite their state level bans on non-iodised salt.

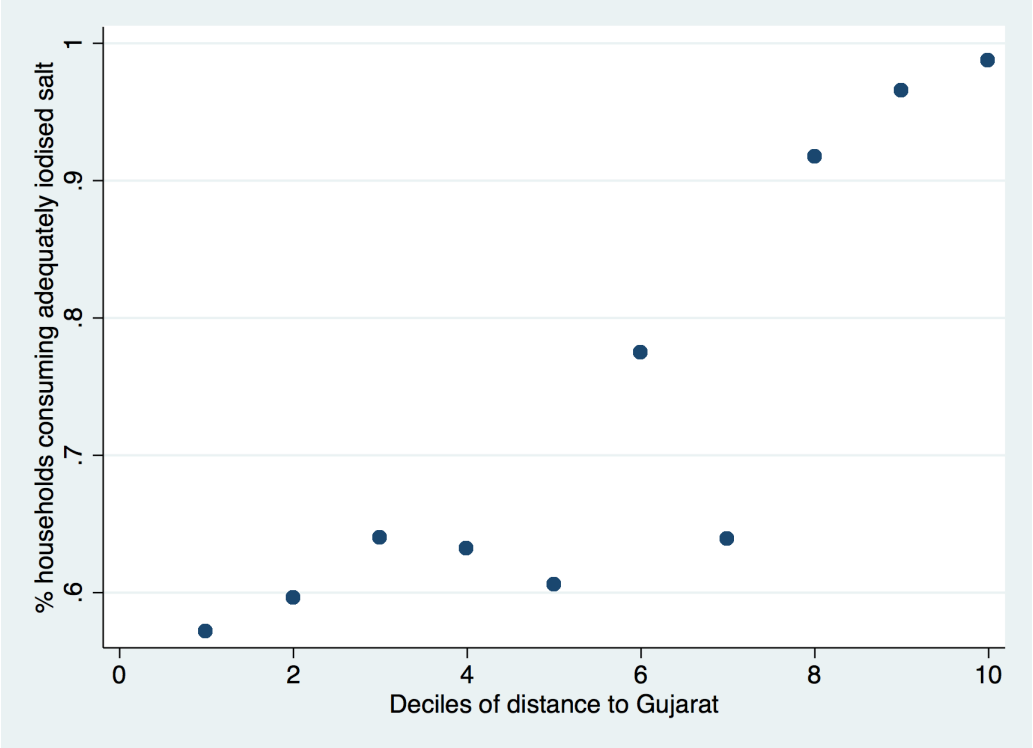
Figure 3.3: Iodised salt consumption per state in 2005-2006



This figure shows the percentage of rural households consuming adequately iodised salt per state based on data from the 2005-2006 NFHS III.

I use the distance to Gujarat per state to construct an instrumental variable for the availability of adequately iodised salt. Previous evidence from Vir (2011), Sankar et al. (2006) along with data presented in Figure 3.2 suggest a non-linear relationship between distance and the likelihood of rail transport. Therefore, I present the relationship between each decile of the distance distribution to Gujarat and the percentage of households consuming adequately iodised salt per state in the 2005-2006 NHFS III in Figure 3.4.

Figure 3.4: Iodised salt consumption per deciles of distance to Gujarat



This figure shows the percentage of rural households consuming adequately iodised salt per state from the 2005-2006 NFHS III in relation to the deciles of the distance distribution per state to Gujarat.

From Figure 3.4 we observe a large jump in the proportion of housholds consuming adequately iodised salt in the upper three deciles. I use this threshold to construct a binary instrumental variable. The instrumental variable takes value 1 if the state is in the upper three deciles of the distance distribution ($\geq$ 1553.558 km) from each state to Gujarat. The instrumental variable takes value 0 if the state is in the 7 lower deciles. The cut-off also corresponds well to the observed threshold of the relationship between distance and transport mode in Figure 3.2.

Moreover, I exclude states consuming a substantial proportion of salt not produced in Gujarat. I omit the two other salt exporting states from the analysis, Tamil Nadu and Rajasthan. As shown from Figure 3.1 we note that the southern states are less likely to consume salt from Gujarat due their close proximity to Tamil Nadu. Therefore, I exclude neighbouring states to Tamil Nadu from the main analysis.

3.5 Econometric specification

To start with, I estimate an OLS regression, specified in Equation 3.1.

$$\text{HAZ}_{is} = \alpha_0 + \beta\text{Iodised Salt}_{is} + \beta X_{is} + \mu_{is} \tag{3.1}$$

The outcome variable is HAZ for child i in state s. The independent variable of interest, *Iodised salt*, takes value 1 if the household was found to have adequately iodised salt and 0 if the salt had no iodine. X is a vector of covariates described below.

I account for any differences across regions by including regional dummies.[11] This is important due to large variation in both height and state governance across regions, see Deaton (2009).[12] Additionally, I control for any region-specific temporal variation, such as seasonal variation in the nutrition and disease environment, by adding year specific month of birth dummies interacted with region of residence. I further control for month of interview to remove any nationwide variation stemming from a potential increase in overall iodised salt coverage due to the notification of the federal ban in 2005 and subsequent anticipation of the implementation of the ban in May 2006.

The following state level variables are controlled for; GDP in 100 000 rupees per capita in 2004-2005 compiled by *National Institution for Transforming India, Government of India* (2006), health expenditure per capita in rupees in 2005-2006 found in Berman (2017), population density measured in 1000 inhabitants per km^2 calculated from the 2001 Indian Census. I also include a binary indicator variable for whether the state is an "Empowered Action Group" (EAG) state. This denotes that the state is socioeconomically backwards and has a higher priority of federal public health programmes such as the National Rural Health Mission (Kumar & Singh 2016).[13] I partial out variation stemming from the institutional capacity, quality and efficiency in the delivery of public services by controlling for the proportion of institutional deliveries per state from the 2005-2006 NFHS III.[14]

I control for the number of goitre endemic areas in McCarrison (1915) per 100,000

[11] As the analytical sample omits states which import a large share of their salt from Tamil Nadu, the southern region is not included in the final sample. Therefore, five out of six regional dummies are included.

[12] Due to the high degree of multicollinearity between the instrumental variable and state, I am not able to include state level fixed effects.

[13] The following states are defined as EAG: Bihar, Chhattisgarh, Jharkhand, Madhya Pradesh, Odisha, Rajasthan, Uttarakhand and Uttar Pradesh.

[14] I do not control for whether the specific child was delivered at a facility as this would decrease the sample size substantially.

population per state.[15] Accounting for known and longstanding goitre endemicity is likely to partial out any differences in state commitments to the eradication of iodine deficiency.

The following household covariates are included: the proportion of children aged 5 or younger, caste, religion, a dummy for whether the household's native tongue is Hindi, wealth quintile,[16] and whether the household uses water from an unprotected water source. I control for the mother's educational attainment and mother's age as these variables often are shown to be strongly associated with children's human capital development (Borooah 2005). Previous work by Spears (2012*b*) and Hammer & Spears (2016) emphasize the impact of sanitation on height. Therefore, I add an indicator variable denoting whether the mother practices open defecation.

The demand for iodised salt is likely to be positively related to other nutritional investments and availability. I reduce the risk for bias by accounting for the nutritional intake of the mother due to its impact on the nutritional availability for the child in utero and postnatally through breastfeeding. I control for whether the mother eats fish at least weekly. Fish is the most iodine rich food and thus the control variable captures dietary iodine availability. I further control for the incidence of other micronutrient deficiencies by accounting for the mother's anaemia status based on the haemoglobin test conducted by the DHS. The following categories are reported; no anaemia, mildly anaemic, moderately anaemic and severely anaemic.[17]

Moreover, health information and the ability to process such information is likely to affect the demand for iodised salt and other unobserved preventive health care investments. Therefore, I add controls for the mother's health knowledge. I generate a categorical variable for the mother's health knowledge based on her knowledge about tuberculosis (TBC), oral rehydration salt (ORS) and AIDS. The variable takes value 0 if the mother does not know about either category, 1 if she knows about one of the categories, 2 if she knows about two of the categories and 3 if she has knowledge about TBC, ORS and AIDS.

Finally, further child-specific covariates are incorporated in the specification. A dummy variable for whether the child is from a singleton or multiple birth is included as children

[15] Please see chapter 2 for a description and discussion of the constructed data set based on the map of goitre endemic areas in India in 1915 by McCarrison (1915).

[16] DHS uses information on ownership of household items, dwelling characteristics, home construction materials and access to a bank or post office account to construct a composite wealth index based on principle component analysis. This score is then divided into quintiles ranking from 1 (poorest) to 5 (richest).

[17] I do not control for mother's height or weight as these outcomes might be directly affected by the availability of iodised salt.

from multiple births often are shorter than those from singleton births. I also control for birth dummies for birth order 1-5 and 6 and above as Borooah (2005) shows that child order matter in explaining variations of child height in India. Additionally, I control for gender and the interaction of gender and birth order as Jayachandran & Pande (2017) find that gender specific birth order is an important predictor of children's heights in India. I also control for the current health status of the child by controlling for whether the child had diarrhoea, fever or cough within two weeks prior to the survey. Child health investment and the availability of health care services is accounted for by adding covariates for whether the child accessed any Integrated Child Development Services (ICDS). The ICDS provide health care, supplementary nutrition and pre-school services to children aged 0-6 years old (Jain 2015).

I cluster the standard errors on the state level due to the federal nature of India and the IV being defined by state.

Even though I control for a large set of covariates, the OLS estimate of the impact of adequately iodised salt use on children's HAZ is at risk for bias. Iodised salt consumption is likely to be positively associated with unobserved characteristics related to health knowledge and preferences for investing in children's health. Omitted variable bias can therefore cause an upward bias in the coefficient of interest using OLS.

HAZ is a function of accumulated levels of health and nutrition, including the intake of iodine. The 2005-2006 NFHS III reports the current levels of iodine in the household's salt and there is a risk that the current use of iodised salt does not represent previous consumption. Therefore, the OLS specification might suffer from measurement error. Assuming that the measurement error is classical, i.e. that the measurement error has mean zero and is uncorrelated with the true dependent and independent variables and with the equation error, it is likely to bias the OLS estimate downwards. Lastly, there is a possibility of simultaneity bias as the consumption of iodised salt might depend on a child's characteristics. Therefore, I estimate a TSLS regression. The first stage is specified in Equation 3.2.

$$\text{Adequately Iodised Salt}_{is} = \alpha_0 + \gamma \text{Far}_s + \beta X_{is} + \mu_{is} \tag{3.2}$$

The instrumental variable *Far* is a binary variable which takes value 1 if the state's centroid is located far from Gujarat. Meaning that it is in the upper three deciles of the distance distribution from Gujarat. The variable takes value 0 if the household's state

of residence is Gujarat or if the state is located in the lower 7 deciles of the distance distribution to Gujarat. X denotes the same set of covariates as in the OLS specification. The predicted estimates for *Adequately Iodised* salt are used in the second stage specified in Equation 3.3 below. The same set of covariates are included and standard errors are clustered at the state level.

$$\text{HAZ}_{is} = \alpha_0 + \delta \text{Adequately Iodised}_{is} + \beta X_{is} + \mu_{is} \tag{3.3}$$

3.6 Summary statistics

The means and standard deviations of various characteristics are shown for households who consume adequately iodised salt and for households who consume salt with nil iodine, respectively, in Table 3.1. The difference in means across the samples and accompanying t-statistics are provided in the last two columns of the table. We observe that children in households with adequately iodised salt have better anthropometric outcomes in terms of HAZ, height and WAZ, and a lower risk for stunting. We also note that the subsamples do not differ with reference to the health status of children or by mothers' anaemia status. In fact, children from households with non-iodised salt are more likely to use ICDS compared to children with access to adequately iodised salt. Households who consume adequately iodised salt are more likely to reside in states with higher rates of historical goitre endemicity. This can potentially be explained by larger state government commitments to eradicating iodine deficiency given that this is a prevalent and long standing problem.

The rationale for the use of IV regression is strengthened by observing that mothers in households with access to adequately iodised salt are more likely to have a better health knowledge and to consume fish more often. Furthermore, EAG state status and two important predictors of child nutritional status; maternal education and open defecation, also differ substantially across the samples. This systematic relationship suggests that the consumption of iodised salt could be potentially correlated with additional unobserved determinants of children's growth.

Table 3.1: Descriptive statistics by household consumption of adequately iodised salt

	Iodised Salt		Non-iodised salt		Difference	
	Mean	SD	Mean	SD	Difference	t-statistic
HAZ	-0.75	1.78	-1.22	1.70	-0.47***	(-6.57)
Stunted	0.22	0.42	0.31	0.46	0.09***	(4.83)
Height (cm)	64.74	6.69	63.75	6.61	-0.99***	(-3.60)
WAZ	-1.03	1.28	-1.50	1.23	-0.48***	(-9.15)
Age in months	6.45	3.40	6.59	3.39	0.15	(1.04)
Girl	0.48	0.50	0.54	0.50	0.06**	(2.67)
Singleton Birth	0.99	0.10	0.99	0.12	-0.00	(-0.77)
Birth Order	2.61	1.60	2.93	1.67	0.32***	(4.64)
Child used ICDS	0.32	0.46	0.38	0.49	0.06**	(3.13)
Child had diarrhoea recently	0.15	0.36	0.17	0.38	0.02	(1.30)
Child had fever recently	0.18	0.39	0.17	0.38	-0.01	(-0.53)
Child had cough recently	0.23	0.42	0.24	0.43	0.01	(0.47)
Mother's Age	25.28	5.46	24.98	5.43	-0.29	(-1.30)
Mother heard of AIDS	0.61	0.49	0.38	0.49	-0.23***	(-11.64)
Mother heard of ORS	0.74	0.44	0.67	0.47	-0.07***	(-3.55)
Mother heard of TBC	0.87	0.34	0.77	0.42	-0.10***	(-5.89)
Mother is anemic	0.63	0.48	0.64	0.48	0.02	(0.86)
Mother eats fish at least weekly	0.32	0.47	0.15	0.36	-0.17***	(-10.12)
Proportion of children under 5 in household	0.31	0.13	0.32	0.12	0.01	(1.12)
Unprotected water source	0.25	0.43	0.25	0.43	0.00	(0.19)
Mother practices open defecation	0.49	0.50	0.82	0.38	0.33***	(19.14)
Hindi	0.38	0.48	0.64	0.48	0.27***	(13.38)
Scheduled Caste	0.15	0.36	0.19	0.39	0.03*	(2.12)
Scheduled Tribe	0.25	0.43	0.22	0.41	-0.03	(-1.72)
Other Backward Caste	0.25	0.43	0.33	0.47	0.09***	(4.49)
Poor Household	0.43	0.50	0.60	0.49	0.17***	(8.43)
Mother has some education	0.63	0.48	0.41	0.49	-0.22***	(-10.86)
Hindu	0.60	0.49	0.82	0.38	0.22***	(12.84)
Muslim	0.14	0.35	0.12	0.33	-0.02	(-1.49)
Empowered action group state	0.39	0.49	0.66	0.47	0.27***	(13.68)
Central	0.07	0.26	0.25	0.43	0.18***	(10.95)
East	0.21	0.41	0.13	0.34	-0.08***	(-5.22)
North	0.27	0.44	0.42	0.49	0.14***	(7.25)
North East	0.37	0.48	0.04	0.20	-0.33***	(-26.16)
West	0.07	0.26	0.16	0.37	0.09***	(6.45)
Proportion institutional deliveries/state	0.84	0.09	0.84	0.10	-0.00	(-0.82)
State health expenditure * 1000 rupees/capita	0.03	0.02	0.02	0.02	-0.01***	(-14.19)
Historical goitre endemic locations/100 000 population per state	0.35	0.56	0.07	0.23	-0.28***	(-18.62)
State population (per 1000) density per km^2	0.33	0.28	0.37	0.23	0.04***	(4.30)
State GDP/capita (per 10000 rupees)	89.05	108.75	159.44	121.86	70.39***	(14.42)
Observations	2024		826		2850	

Next, I present the descriptive statistics for households in states near and far from Gujarat according to the definition of the IV indicator in Table 3.2. First we note that 93% of all households in states in the upper three deciles of the distance distribution were found to consume adequately iodised salt. The respective proportion is only 55% in Gujarat or nearby states. Children living far away from Gujarat have better anthropometric outcomes. The samples are balanced in terms of general child characteristics such as; gender, age, singleton birth status and birth order. Additionally, no statistically significant differences are found for whether the child had fever or cough recently, the proportion of children under 5 in the household, the proportion of muslims and the percentage of births delivered at a facility.

A clear difference in terms of health and nutrition between households in states near and far from Gujarat does not emerge. While mothers in states far away from Gujarat are more likely to have heard of AIDS and TBC, they are less likely to have heard of ORS. Households in states with closer proximity to the main salt exporting state are more likely to report a higher incidence of diarrhoea. They also report a higher utilisation of ICDS. It is not certain whether the higher use of ICDS reflects need or an improved access to health care services. Whilst nutritional intake, measured by the frequency of fish consumption, is better in states far from Gujarat, mothers in states near Gujarat are less likely to be anaemic. Other determinants of children's heights, such as the hygiene and sanitation environments, do not appear to vary consistently by IV assignment. Open defecation is higher in states nearer Gujarat while the proportion of households who access drinking water from an unprotected water source is lower. There does not seem to be a clear systematic relationship between predictors of height and IV status.

Table 3.2: Descriptive statistics by instrumental variable

	Near Gujarat		Far from Gujarat		Difference	
	Mean	SD	Mean	SD	Difference	t-statistic
Adequately Iodised Salt	0.55	0.50	0.93	0.26	-0.38***	(-26.27)
HAZ	-1.02	1.72	-0.71	1.82	-0.30***	(-4.49)
Stunted	0.27	0.44	0.22	0.41	0.05**	(2.99)
Height (cm)	64.15	6.62	64.87	6.74	-0.72**	(-2.83)
WAZ	-1.27	1.20	-1.01	1.38	-0.27***	(-5.30)
Age in months	6.46	3.37	6.53	3.43	-0.07	(-0.56)
Girl	0.49	0.50	0.51	0.50	-0.02	(-1.20)
Singleton Birth	0.99	0.10	0.98	0.12	0.01	(1.25)
Birth Order	2.67	1.57	2.76	1.70	-0.09	(-1.46)
Child used ICDS	0.39	0.49	0.25	0.43	0.15***	(8.42)
Child had diarrhoea recently	0.18	0.38	0.13	0.34	0.05***	(3.34)
Child had fever recently	0.17	0.38	0.19	0.39	-0.02	(-1.24)
Child had cough recently	0.22	0.42	0.25	0.43	-0.02	(-1.52)
Mother's Age	24.93	5.04	25.56	5.95	-0.63**	(-2.97)
Mother heard of AIDS	0.51	0.50	0.59	0.49	-0.09***	(-4.70)
Mother heard of ORS	0.74	0.44	0.69	0.46	0.05**	(3.03)
Mother heard of TBC	0.81	0.39	0.88	0.32	-0.07***	(-5.22)
Mother is anemic	0.60	0.49	0.69	0.46	-0.09***	(-4.77)
Mother eats fish at least weekly	0.14	0.35	0.46	0.50	-0.32***	(-18.99)
Proportion of children under 5 in household	0.31	0.13	0.32	0.13	-0.01	(-1.35)
Unprotected water source	0.19	0.39	0.33	0.47	-0.14***	(-7.91)
Mother practices open defecation	0.73	0.44	0.39	0.49	0.35***	(19.41)
Hindi	0.63	0.48	0.22	0.41	0.41***	(24.51)
Scheduled Caste	0.21	0.40	0.10	0.31	0.10***	(7.67)
Scheduled Tribe	0.15	0.36	0.37	0.48	-0.21***	(-12.84)
Other Backward Caste	0.32	0.47	0.21	0.41	0.11***	(6.84)
Poor Household	0.45	0.50	0.52	0.50	-0.07***	(-3.78)
Mother has some education	0.55	0.50	0.59	0.49	-0.04*	(-2.07)
Hindu	0.82	0.38	0.44	0.50	0.38***	(22.31)
Muslim	0.11	0.31	0.18	0.39	-0.07***	(-5.46)
Empowered action group state	0.57	0.50	0.34	0.47	0.23***	(12.48)
Central	0.22	0.41	0.00	0.00	0.22***	(21.30)
East	0.08	0.27	0.34	0.47	-0.27***	(-17.54)
North	0.54	0.50	0.00	0.00	0.54***	(44.09)
North East	0.00	0.00	0.66	0.47	-0.66***	(-47.93)
West	0.17	0.37	0.00	0.00	0.17***	(18.20)
Proportion institutional deliveries/state	0.83	0.10	0.86	0.06	-0.03***	(-8.42)
State health expenditure * 1000 rupees/capita	0.03	0.02	0.04	0.02	-0.01***	(-13.00)
Historical goitre endemic locations/100 000 population per state	0.11	0.22	0.49	0.67	-0.38***	(-18.84)
State population (per 1000) density per km^2	0.34	0.21	0.35	0.33	-0.02	(-1.71)
State GDP/capita (per 10000 rupees)	152.05	126.54	50.80	68.03	101.24***	(27.50)
Observations	1651		1199		2850	

3.7 Results

The regression results from the OLS specification (columns 1-2), first stage (columns 3-4), reduced form (5-6) and TSLS (columns 7-8) specifications are presented in Table 3.3. The OLS estimates show that the availability of adequately iodised salt is associated with an increase by 0.156 height-for-age Z-scores after controlling for the full set of controls. Turning to the first stage in columns 3-4 we note that residing far from Gujarat increases the probability that a household consumes adequately iodised salt by 37.6 percentage points when no covariates are included and 55.2 percentage points after including the full set of controls. The corresponding Kleibergen-Paap F-statistic, shown at the bottom of Table 3.3 is 29.457 for the fully specified model and indicates that the instrument is not weak. The reduced form also shows that the assignment of the IV is positively related to children's HAZ although it becomes statistically insignificant when the full set of covariates is included in column 6.

The TSLS result indicate that access to adequately iodised salt improves height-for-age by 0.664 standard deviations. The IV estimate is larger than the corresponding OLS coefficient, but we also note that the standard errors increase substantially from the OLS specification. The Durbin-Wu-Hausman test reveals that we fail to reject the null hypothesis that the consumption of adequately iodised salt is exogenous at the 1% level of statistical significance.

One would assume that any potential omitted characteristics which are positively related to the consumption of iodised salt, also have a positive effect on children's growth. Given this assumption, the risk for omitted variable bias could potentially cause OLS to be upward biased. As the IV results indicate that the OLS estimates were downward biased, OVB is not likely to have biased the OLS regressions significantly. This can potentially be explained by the inclusion of a rich set of covariates, particularly related to the nutritional environment and the mother's health practices and health knowledge.

Due to the increase in the coefficient of interest from the OLS to the IV estimations, the largest source of bias in the OLS specification is likely to be attenuation bias caused by measurement error. The risk for measurement error in the availability of adequately iodised salt is likely to stem from differences in current consumption of adequately iodised salt compared to previous consumption.[18] As HAZ is a function of current and past health and nutrition investments, one would ideally like to include information on past

[18]The bias is less likely to be caused by measurement error in the iodine content of the household's salt consumption at the time of the survey as the iodine content has been measured objectively.

consumption of iodised salt in the analysis. As the IV is correlated with the consumption of adequately iodised salt but uncorrelated with the error term, IV regression identifies the true effect of adequately iodised salt consumption on HAZ.

Table 3.3: Effect on HAZ - OLS, first stage, reduced form and TSLS regressions.

	OLS		First Stage		Reduced Form		TSLS	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	HAZ	HAZ	Adequately Iodised Salt	Adequately Iodised Salt	HAZ	HAZ	HAZ	HAZ
Adequately Iodised Salt	0.467***	0.156*					0.805**	0.664*
	(0.105)	(0.082)					(0.389)	(0.364)
Far from Gujarat			0.376***	0.552***	0.303*	0.367		
			(0.054)	(0.102)	(0.164)	(0.239)		
Girl		0.184		-0.054		0.175		0.211*
		(0.124)		(0.032)		(0.124)		(0.115)
Child used ICDS		-0.048		0.039**		-0.035		-0.061
		(0.092)		(0.014)		(0.091)		(0.090)
Child had diarrhoea recently		-0.041		-0.004		-0.044		-0.041
		(0.069)		(0.020)		(0.069)		(0.066)
Child had fever recently		-0.057		0.006		-0.059		-0.063
		(0.082)		(0.022)		(0.081)		(0.076)
Child had cough recently		0.104		-0.026		0.100		0.118
		(0.102)		(0.029)		(0.100)		(0.098)
Mother mildly anaemic		-0.104		-0.015		-0.108		-0.099
		(0.087)		(0.023)		(0.087)		(0.081)
Mother moderately anaemic		-0.217***		0.002		-0.218***		-0.219***
		(0.074)		(0.026)		(0.074)		(0.072)
Mother severely anaemic		-0.348		0.046		-0.338		-0.369*
		(0.224)		(0.050)		(0.225)		(0.212)
Mother: Primary education		-0.164**		0.049		-0.157*		-0.189***
		(0.073)		(0.032)		(0.076)		(0.071)
Mother: Secondary education		0.113		0.046*		0.118		0.087
		(0.092)		(0.027)		(0.090)		(0.099)
Mother: Higher education		0.395*		0.058		0.391*		0.353*
		(0.200)		(0.051)		(0.199)		(0.205)
Mother eats fish at least weekly		-0.094		0.005		-0.084		-0.087
		(0.107)		(0.031)		(0.110)		(0.104)
Mother heard of one of ORS or TBC or AIDS		0.212		0.103***		0.227		0.159
		(0.139)		(0.030)		(0.138)		(0.145)
Mother heard of two out of ORS, TBC and AIDS		0.217		0.120***		0.238*		0.159
		(0.127)		(0.032)		(0.126)		(0.135)
Mother heard of ORS and TBC and AIDS		0.338**		0.126***		0.368**		0.284*
		(0.150)		(0.033)		(0.148)		(0.154)
Unprotected water source		-0.046		-0.049***		-0.063		-0.031
		(0.067)		(0.017)		(0.065)		(0.067)
Mother practices open defecation		-0.050		-0.034		-0.053		-0.030
		(0.100)		(0.025)		(0.100)		(0.096)
Poorer		-0.268**		0.042*		-0.262**		-0.289***
		(0.118)		(0.023)		(0.117)		(0.111)
Middle		0.080		0.028		0.085		0.067
		(0.119)		(0.029)		(0.120)		(0.107)
Richer		0.199		0.120***		0.223		0.144
		(0.133)		(0.040)		(0.133)		(0.115)
Richest		0.523**		0.192***		0.556***		0.429**
		(0.189)		(0.053)		(0.186)		(0.168)
Constant	-1.222***	-3.560***	0.552***	0.901***	-1.018***	-3.439***	-1.462***	-4.038***
	(0.088)	(0.857)	(0.051)	(0.276)	(0.087)	(0.900)	(0.267)	(0.856)
Kleibergen-Paap F-statistic					-		48.058	29.457
Observations	2850	2476	2850	2476	2850	2476	2850	2476
R^2	0.014	0.158	0.168	0.312	0.007	0.158	0.007	0.145

Notes: The outcome variable is HAZ. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions in columns 7 and 8. The covariates included in columns 2, 4, 6 and 8 are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

The increase in HAZ is supported by an observed increase in raw height measured in cm. I estimate the effect on height while controlling for age in months. The regression results are presented in Table 3.4. From column 6 we note that the IV results show that the access to adequately iodised salt increases height by 1.845 cm for children up to 12 months.

Even though access to iodised salt has a negative effect on the risk of being stunted, both the OLS and IV estimates are not significant at a conventional level of statistical significance after including all controls, see Table C.1 in the Appendix. This possibly implies that access to iodised salt does not greatly improve height for children who have experienced severe long term nutritional deprivation.

Iodine is hypothesized to have a positive effect on somatic growth as thyroid hormones are needed to regulate skeletal development (Abu et al. 1997). According to Shields (2011), very little research has looked into the impact of thyroid hormone on foetal or infant adiposity as the biological relationship is less clear. A clinical trial of 5-15 year old children in Tibet with Kashin-Beck disease who received intramuscular iodised oil finds that iodine supplementation increased HAZ-scores, whereas WAZ-scores decreased, see Moreno-Reyes et al. (2003).[19] Therefore, one would not readily expect a positive effect of iodised salt on children's weights.

I analyse the impact of adequately iodised salt access on children's WAZ by estimating similar OLS and IV equations specified previously. I restrict the analysis to the sample of children used in the main regressions. From Table C.2 in Appendix C we do not observe a positive effect of iodised salt on WAZ from the OLS results nor the IV results after controlling for all covariates. The absence of an effect on children's weights indicates that an overall increase in current nutritional intake, nor associated omitted characteristics, are unlikely to be driving the overall results on HAZ.

[19]The children were part of a selenium supplementation trial where a randomized group received intramuscular iodised oil before being further randomly assigned to receive selenium or placebo.

Table 3.4: Effect on height - OLS, reduced form and TSLS regressions.

	OLS		Reduced Form		TSLS	
	(1)	(2)	(3)	(4)	(5)	(6)
Adequately Iodised Salt	0.986***	0.478**			1.910	1.845***
	(0.314)	(0.185)			(1.288)	(0.709)
Far from Gujarat			0.719	1.035*		
			(0.517)	(0.508)		
Girl		-1.350***		-1.379***		-1.280***
		(0.268)		(0.272)		(0.234)
Child used ICDS		0.078		0.115		0.043
		(0.188)		(0.187)		(0.184)
Child had diarrhoea recently		0.003		-0.006		0.002
		(0.159)		(0.158)		(0.156)
Child had fever recently		-0.042		-0.048		-0.058
		(0.184)		(0.184)		(0.171)
Child had cough recently		0.309		0.298		0.346
		(0.240)		(0.238)		(0.234)
Mother mildly anaemic		-0.311		-0.325		-0.298*
		(0.192)		(0.193)		(0.179)
Mother moderately anaemic		-0.575***		-0.578***		-0.581***
		(0.154)		(0.154)		(0.156)
Mother severely anaemic		-1.219**		-1.191**		-1.277***
		(0.495)		(0.494)		(0.477)
Mother: Primary education		-0.340*		-0.316*		-0.406**
		(0.170)		(0.179)		(0.166)
Mother: Secondary education		0.330		0.345		0.259
		(0.236)		(0.230)		(0.242)
Mother: Higher education		1.041**		1.030**		0.923*
		(0.477)		(0.473)		(0.473)
Mother eats fish at least weekly		-0.297		-0.269		-0.278
		(0.236)		(0.239)		(0.234)
Mother heard of one of ORS or TBC or AIDS		0.234		0.279		0.088
		(0.393)		(0.390)		(0.402)
Mother heard of two out of ORS, TBC and AIDS		0.324		0.385		0.163
		(0.336)		(0.337)		(0.339)
Mother heard of ORS and TBC and AIDS		0.529		0.616		0.381
		(0.369)		(0.368)		(0.366)
Unprotected water source		-0.208		-0.258*		-0.166
		(0.137)		(0.137)		(0.130)
Mother practices open defecation		-0.216		-0.227		-0.165
		(0.251)		(0.251)		(0.245)
Poorer		-0.566**		-0.548**		-0.625**
		(0.257)		(0.252)		(0.249)
Middle		0.235		0.249		0.198
		(0.245)		(0.247)		(0.227)
Richer		0.400		0.471		0.249
		(0.314)		(0.312)		(0.296)
Richest		1.299***		1.398***		1.042**
		(0.438)		(0.426)		(0.412)
Constant	63.752***	44.363***	64.150***	43.968***	63.097***	41.863***
	(0.329)	(11.685)	(0.384)	(11.546)	(1.023)	(12.421)
Observations	2850	2477	2850	2477	2850	2477
R^2	0.004	0.691	0.003	0.690	0.001	0.684

Notes: The outcome variable is height measured in cm. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions in columns 5 and 6. The covariates included in columns 2, 4, and 6 are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

3.7.1 Heterogeneous effects

Next, I estimate the effect for separate age groups of children aged 0-6 months, 7-12 months, 13-18 months and 19-24 months. The results are presented in Table 3.5. We note that the effect of adequately iodised salt availability on HAZ is biggest for children up to 6 months. The IV estimate for children between 6 and 12 months is positive but not statistically significant. The effects for the older age categories are not statistically significant across both OLS and IV specifications. The results can possibly be interpreted as the impact of iodised salt availability being largest for very young children. However, medical research has not established the relative importance of access to iodine for linear growth of different age groups (Farebrother et al. 2018).

In the main regressions, I have controlled for the number of historical goitre endemic areas per population per state. This is to account for the naturally occurring risk for iodine deficiency per state and for any unobserved state level differences in public policy aiming to reduce known and long standing iodine deficiency. Intuitively the effect of iodised salt on HAZ should be larger for children at higher risk for iodine deficiency. To investigate whether the effect differs according to the risk for iodine deficiency, I estimate the main regressions for two sub samples separately; children residing in states in the upper three quartiles of the state-goitre distribution, and children from states in the three lower quartiles of the state-goitre distribution.[20] In line with the expectations, we observe a larger effect on HAZ in states with a higher risk for iodine deficiency. Children from states in the second and higher quartile of the historical state-goitre endemicity distribution experienced an improvement of height-for-age by 1.45 standard deviations.

The medical literature concerning the importance of thyroid hormones, and thus iodine for populations at risk for deficiency, does not observe consistent gender differences. Due to the differential impact of iodised salt on cognitive test scores shown in Chapter 2, I also estimate the effect of access to iodised salt on HAZ for boys and girls separately. The regression results are provided in Table C.3 in Appendix C. Even though the IV coefficient appears to be somewhat larger for girls, see column (2) in Table C.3, the treatment effects for boys and girls are not statistically significantly different from each other.

[20] I am not able to investigate the effect for mutually exclusive groups in the state-goitre distribution. States with high historical goitre endemicity are likely to be further away from Gujarat which leaves insufficient variation in the IV assignment indicator.

Table 3.5: Effect on HAZ - OLS and TSLS regressions for separate age groups

	0-6 Months		7-12 Months		13-18 Months		19-24 Months	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	OLS	TSLS	OLS	TSLS	OLS	TSLS	OLS	TSLS
Adequately Iodised Salt	0.265	1.232**	0.070	0.251	0.101	-0.199	0.054	-0.044
	(0.191)	(0.568)	(0.077)	(0.261)	(0.103)	(0.462)	(0.134)	(0.489)
Girl	0.076	0.111	0.239**	0.238**	0.267	0.284*	0.113	0.115
	(0.228)	(0.192)	(0.111)	(0.106)	(0.161)	(0.163)	(0.154)	(0.144)
Child used ICDS	-0.067	-0.104	0.094	0.096	0.211**	0.203**	-0.084	-0.085
	(0.125)	(0.116)	(0.081)	(0.078)	(0.088)	(0.088)	(0.114)	(0.107)
Child had diarrhoea recently	-0.289**	-0.327***	-0.031	-0.029	-0.259***	-0.258***	-0.213	-0.215
	(0.123)	(0.118)	(0.080)	(0.074)	(0.084)	(0.081)	(0.152)	(0.140)
Child had fever recently	0.069	0.054	-0.031	-0.026	0.193	0.174	0.117	0.120
	(0.137)	(0.127)	(0.074)	(0.072)	(0.119)	(0.124)	(0.163)	(0.149)
Child had cough recently	0.197	0.289*	0.008	0.005	-0.103	-0.094	-0.047	-0.052
	(0.163)	(0.159)	(0.067)	(0.063)	(0.102)	(0.102)	(0.140)	(0.124)
Mother mildly anaemic	-0.139	-0.156	-0.026	-0.022	-0.013	-0.017	0.139	0.137
	(0.111)	(0.105)	(0.064)	(0.060)	(0.096)	(0.091)	(0.116)	(0.106)
Mother moderately anaemic	-0.112	-0.115	-0.211**	-0.210**	-0.083	-0.087	0.088	0.089
	(0.147)	(0.134)	(0.095)	(0.090)	(0.107)	(0.105)	(0.139)	(0.129)
Mother severely anaemic	-0.213	-0.271	-0.207	-0.210	0.018	0.006	-0.383	-0.375
	(0.410)	(0.399)	(0.162)	(0.153)	(0.273)	(0.265)	(0.356)	(0.325)
Mother: Primary education	-0.047	-0.074	-0.088	-0.089	-0.107	-0.119	0.003	0.005
	(0.121)	(0.113)	(0.066)	(0.063)	(0.126)	(0.114)	(0.131)	(0.121)
Mother: Secondary education	0.095	0.041	0.034	0.027	-0.169	-0.165	0.175	0.177
	(0.155)	(0.157)	(0.120)	(0.115)	(0.199)	(0.185)	(0.159)	(0.150)
Mother: Higher education	0.171	0.016	0.407*	0.398**	0.109	0.139	0.928***	0.938***
	(0.288)	(0.289)	(0.198)	(0.184)	(0.224)	(0.202)	(0.210)	(0.214)
Mother eats fish at least weekly	-0.101	-0.091	-0.055	-0.056	-0.179	-0.179	-0.023	-0.027
	(0.148)	(0.154)	(0.109)	(0.102)	(0.151)	(0.143)	(0.126)	(0.122)
Mother heard of one of ORS or TBC or AIDS	0.372*	0.298	0.178	0.167	0.188	0.186	0.023	0.038
	(0.203)	(0.191)	(0.107)	(0.103)	(0.171)	(0.154)	(0.143)	(0.149)
Mother heard of two out of ORS, TBC and AIDS	0.317*	0.214	0.196*	0.185*	0.221	0.222	-0.101	-0.088
	(0.154)	(0.148)	(0.105)	(0.101)	(0.160)	(0.142)	(0.170)	(0.166)
Mother heard of ORS and TBC and AIDS	0.450**	0.385**	0.361***	0.343***	0.476**	0.499***	0.067	0.084
	(0.208)	(0.191)	(0.112)	(0.111)	(0.217)	(0.190)	(0.170)	(0.153)
Unprotected water source	-0.109	-0.080	0.046	0.054	0.133	0.114	0.249	0.245*
	(0.108)	(0.106)	(0.090)	(0.085)	(0.116)	(0.120)	(0.156)	(0.141)
Mother practices open defecation	0.263	0.316**	-0.186*	-0.177*	-0.096	-0.117	-0.077	-0.081
	(0.163)	(0.153)	(0.096)	(0.091)	(0.137)	(0.125)	(0.122)	(0.116)
Poorer	-0.303	-0.330*	0.027	0.020	0.253*	0.271**	0.264**	0.271**
	(0.185)	(0.183)	(0.106)	(0.101)	(0.142)	(0.138)	(0.118)	(0.121)
Middle	0.111	0.067	0.193**	0.185**	0.284**	0.316**	0.318**	0.323**
	(0.173)	(0.178)	(0.090)	(0.086)	(0.134)	(0.135)	(0.140)	(0.139)
Richer	0.255	0.172	0.260*	0.240*	0.351*	0.396**	0.579***	0.590***
	(0.234)	(0.223)	(0.149)	(0.141)	(0.180)	(0.173)	(0.198)	(0.184)
Richest	0.539	0.397	0.633***	0.597***	0.668**	0.741***	0.626***	0.643***
	(0.317)	(0.300)	(0.201)	(0.201)	(0.252)	(0.274)	(0.204)	(0.195)
Constant	-2.890***	-3.448***	-4.456***	-4.638***	-4.638***	-4.219***	-5.589***	-5.535***
	(0.991)	(0.798)	(0.736)	(0.715)	(1.139)	(1.144)	(1.335)	(1.392)
Observations	1241	1241	2804	2804	1351	1351	1150	1150
R^2	0.162	0.119	0.210	0.208	0.215	0.210	0.213	0.212

Notes: The outcome variable is HAZ. The regressions are estimated separately for children in different age categories. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions shown in columns 2, 4, 6 and 8. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, ***

Table 3.6: Effect on HAZ - OLS and TSLS by historical goitre endemicity

	Higher historical goitre endemicity		Low historical goitre endemicity	
	(1)	(2)	(3)	(4)
	OLS	TSLS	OLS	TSLS
Adequately Iodised Salt	0.222**	1.126**	0.143	0.832***
	(0.081)	(0.448)	(0.087)	(0.265)
Girl	0.338***	0.351***	0.126	0.169
	(0.079)	(0.082)	(0.150)	(0.112)
Child used ICDS	-0.106	-0.138	-0.002	-0.027
	(0.109)	(0.112)	(0.114)	(0.109)
Child had diarrhoea recently	-0.016	-0.027	-0.024	-0.044
	(0.085)	(0.083)	(0.070)	(0.062)
Child had fever recently	-0.078	-0.084	-0.030	-0.021
	(0.090)	(0.085)	(0.105)	(0.093)
Child had cough recently	0.143	0.175	0.118	0.133
	(0.118)	(0.114)	(0.119)	(0.120)
Mother mildly anaemic	-0.036	-0.028	-0.050	-0.036
	(0.091)	(0.084)	(0.107)	(0.098)
Mother moderately anaemic	-0.223**	-0.244***	-0.202***	-0.221***
	(0.083)	(0.088)	(0.061)	(0.059)
Mother severely anaemic	-0.219	-0.245	-0.403	-0.416
	(0.256)	(0.238)	(0.292)	(0.271)
Mother: Primary education	-0.155*	-0.209**	-0.024	-0.075
	(0.084)	(0.083)	(0.079)	(0.082)
Mother: Secondary education	0.071	0.011	0.153	0.130
	(0.114)	(0.114)	(0.116)	(0.121)
Mother: Higher education	0.502*	0.446*	0.594*	0.559*
	(0.240)	(0.231)	(0.310)	(0.320)
Mother eats fish at least weekly	-0.094	-0.099	-0.201	-0.227
	(0.128)	(0.122)	(0.146)	(0.139)
Mother heard of one of ORS or TBC or AIDS	0.175	0.098	0.153	0.090
	(0.148)	(0.167)	(0.160)	(0.154)
Mother heard of two out of ORS, TBC and AIDS	0.190	0.110	0.156	0.084
	(0.132)	(0.143)	(0.142)	(0.126)
Mother heard of ORS and TBC and AIDS	0.280	0.199	0.207	0.150
	(0.172)	(0.181)	(0.156)	(0.135)
Unprotected water source	-0.025	0.007	-0.051	-0.016
	(0.083)	(0.077)	(0.088)	(0.075)
Mother practices open defecation	-0.057	-0.003	0.017	0.036
	(0.128)	(0.126)	(0.128)	(0.127)
Poorer	-0.316**	-0.347***	-0.203*	-0.231**
	(0.118)	(0.110)	(0.106)	(0.095)
Middle	0.171	0.159	0.011	-0.002
	(0.148)	(0.141)	(0.133)	(0.117)
Richer	0.336*	0.251	0.060	-0.046
	(0.174)	(0.161)	(0.131)	(0.103)
Richest	0.572**	0.439**	0.683***	0.506***
	(0.234)	(0.204)	(0.117)	(0.114)
Constant	-2.848***	-3.585***	-4.576***	-5.343***
	(0.969)	(0.789)	(0.676)	(0.674)
Kleibergen-Paap F-statistic		80.735		62.945
Observations	2005	2005	1675	1675
R^2	0.178	0.143	0.163	0.132

The outcome variable is HAZ. The regressions are estimated separately for children in states in the upper three quartiles of the state-goitre distribution (columns 1-2), and children from states in the three lower quartiles of the state-goitre distribution (columns 3-4). The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions shown in columns 2, 4 and 6. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, ***

3.7.2 Characteristics of compliers

The Local Average Treatment Effect (LATE) theorem says that IV regression estimates the average causal effect of treatment on the sub-population of compliers. The external validity from the IV results is stronger if the compliant subpopulation is similar to other populations of interest (Angrist & Pischke 2009, pp.150). Next, I investigate whether the LATE is driven by a specific subpopulation being induced to comply with the assignment who would have not otherwise been treated. I investigate the likelihood of compliers consisting of households with certain characteristics. Following Angrist & Pischke (2009), I provide the relative likelihood of a complier of a certain characteristic as given by the ratio of the first stage for a given group to the overall first stage.

The overall first stage is 0.376 and provided in column 3 in Table 3.3. The relative likelihood of compliance is provided in column 3 in Table 3.7. While positive relative likelihoods are observed for most characteristics related to worse socio-economic status and overall access to adequate nutrition, we note that poor households, households using unprotected water sources and households where the mother has little health knowledge, are particularly more likely to comply with the IV. Households where mothers have some education are less likely to be compliers. This suggests that the complier sub-population consist of worse off households who could be more likely to benefit from added iodine due to a lower overall nutritional intake. In addition to the threat of measurement error in the OLS, the larger estimates from the IV regression compared to OLS can be explained by potentially higher gains for the groups who are more likely to comply relative to the general population.[21]

Table 3.7: Complier characteristics

Characteristic	Overall proportion with characteristic	First stage for characteristic	Relative first stage
	(1)	(2)	(3)
Poor (<third wealth quintile)	0.586	0.489	1.300
Unprotected water source	0.247	0.459	1.220
Open defecation	0.586	0.386	1.025
Mother has anaemia	0.631	0.388	1.030
Mother eats fish weekly or more often	0.272	0.342	0.909
Mother has not heard of TBC, ORS or AIDS	0.075	0.540	1.434
Mother has some education	0.566	0.310	0.824

[21] In the absence of omitted variables and measurement error biases, OLS estimates approximate average effects for everyone.

3.7.3 Selection effects

Previous studies suggest that iodine supplementation of iodine deficient populations can potentially improve fertility and reduce infant mortality (Zimmermann 2012). If access to adequately iodised salt improves fertility and child survival, the effects on HAZ are possibly underestimated following the survival of marginal children.

Therefore, I investigate the effect of adequately iodised salt consumption on the probability of a child dying within the first year of birth, and on the number of children the mother has given birth to during the past 3 years. I estimate similar regressions as specified in Equations 3.1 and 3.3, but I exclude child specific covariates observed after birth.

The effect on infant mortality is analysed for the sample of all births from 2001 up to one year prior to the year and month of interview. I am not able to restrict the sample to births occurring within 12 months prior to the survey as this would potentially lead to right-censoring, meaning that the survival status of the child might not be known at the time of the analysis. Therefore, I include earlier births who were conceived during the absence of the nationwide ban on non-iodised salt.[22] The IV results point to a small and negative but statistically insignificant effect on infant mortality, see Table C.5. Additionally, a reduced form regression of the IV on infant mortality does not find an effect, see column 5 in Table C.8.

3.8 Validity checks

In the main regressions I have focused on the differential effect of access to adequately iodised salt compared to salt with no iodine, on height. As we do not know the iodine content of salt categorised as containing some iodine but below what is deemed adequate, we are unsure about its implications for children's growth. However, given that it contains less iodine than adequately iodised salt, the consumption of inadequately iodised salt should have a smaller impact on HAZ, if any.

As a robustness check I estimate the effect of inadequately iodised salt compared to non-iodised salt, on HAZ, otherwise following Equations 3.1 - 3.3. The regression results are presented in Table C.4 in Appendix C. The IV is associated with a decreased likelihood of consuming salt with some iodine, see columns 3 and 4. The Kleibergen-Paap F-statistic is 47.186 which indicates that the instrument is not weak. This is in line with only

[22] I do not include children born during the initial central ban on non-iodised salt in 1998-2000. This is due to the fact that Gujarat was also covered by this ban which makes the instrument less likely to be relevant prior to 2000.

adequately iodised salt being given permission for rail transport. Moreover, no significant relationship between inadequately iodised salt and HAZ is observed across both OLS and IV.

Third, I test whether the instrument is systematically correlated with changes in other determinants of height. I run reduced form placebo regressions where I stepwise estimate the effect of the instrument on the variables previously used as covariates. I restrict the analysis to the household-, mother- and child specific covariates that were not balanced at baseline. A correlation between the instrument and the changes in other factors affecting growth positively would indicate a potential violation of the exclusion restriction. The reduced-form estimations do not just underline the validity of the instrument. They also shed light on other potential mechanisms by which the instrument might work through.

From Table C.6 in Appendix C we note that the assignment status of the IV is associated with a higher risk of using water from an unprotected water source, lower maternal age, reduced health knowledge, less frequent consumption of fish and lower use of ICDS. Turning to more direct predictors of children's nutritional and health status, such as; whether the child received vitamin A supplementation, iron supplementation, deworming drugs, anaemia status and retrospectively reported birth weight, does not reveal positive associations with residing far from Gujarat, see Table C.7 in Appendix C. All coefficients are statistically insignificant besides the effect on deworming which points to a negative relationship with the IV.

Additionally, the IV is not positively correlated with pregnancy related variables. Reduced form placebo regressions show that mothers in states far from Gujarat are less likely to give birth at a health facility and complete fewer ANC visits, see Table C.8 in Appendix C. Moreover, no statistically significant effects are found on months of breastfeeding nor taking iron supplementation during pregnancy.

The IV is not systematically correlated with observed factors improving children's heights. This reduces potential worries about the IV not satisfying the exclusion restriction. However, the reduced form placebo regressions show that those with positive IV assignment status are marginally worse off in terms of some determinants of nutrition and health. The main results might therefore be picking up a treatment effect specific to those with a higher capacity to benefit from iodised salt due to otherwise lower health investments after controlling for all covariates. This was also highlighted in the discussion of the LATE.

3.9 Conclusion

This paper shows that the access to adequately iodised salt has a large effect on height-for-age Z-scores (HAZ) for children up to 1 year in rural India. I use exogenous variation in the availability of adequately iodised salt stemming from differences in the feasibility of monitoring of iodised salt depending on the distance from the major salt producing state in India.

The IV estimates point to an improved height-for-age by 0.664 Z-scores and increased height by 1.845 cm. The findings contribute to the mixed and limited scope of evidence from trials on iodine supplementation and somatic growth. Moreover, it improves upon the empirical strategy used in cross-sectional studies assessing the impact of iodised salt on children's growth, which do not address endogeneity concerns.

The effects on HAZ are largest for younger children, aged 0-6 months. This contributes further to an understanding of possibly particularly sensitive time periods of iodine intake for health. No effect is found on weight which is consistent with the lack of an established physiological relationship between iodine supplementation and soft tissue in children.

The effects from this study are bigger than the effects from other public health programmes on HAZ in India. Hammer & Spears (2016) find that a village sanitation intervention improves height-for-age by 0.3 standard deviations and Jain (2015) finds that supplementary daily feeding of girls up to 2 years improves height-for-age by 0.4 standard deviations. The large treatments effects from this study can potentially be explained by the sub-population of compliers. The compliers are more likely to be households who are socio-economically worse off. Children from such households may have a higher propensity to benefit form added iodine. Nonetheless, I show that access to adequately iodised salt plays a large role in improving health capital by reducing the prevalence of short stature in India.

Chapter 4

Conclusions

This thesis analysed the effects of plausibly exogenous variations in the access to nutrition, on children's anthropometric status and in turn, on cognitive attainment. This project has explored data from two very different contexts, the UK and India, which has permitted the investigation of the potential drivers and societal implications of both under- and over consumption of food. Due to the inherent difficulty in estimating a causal relationship between the immediate food environment or food choices, and human capital outcomes, the analyses have relied on quasi-experimental methods. Therefore, the overall evidence offers an important basis for policy making related to nutrition.

In the first empirical chapter, jointly with Professor Peter Dolton, we show that the influx of fast food in the UK is unlikely to have caused an increase in BMI among children. We studied the relationship between the exposure to fast food and adolescent BMI in 1986 using data on the timing of establishment and location of all fast food outlets in Great Britain. This time period is characterised by large variation in the spatial and temporal access to fast food. Which also allowed us to investigate whether distance and duration of fast food exposure affected BMI.

We do not find any evidence of a positive association between numerous measures of fast food exposure and adolescent BMI, or the probability of being overweight or obese. The extant literature in this field is mixed and often unable to address empirical challenges such as; spatial sorting, potential reverse causality and fast food being ever-present. We do not find systematic area level determinants of fast food density for the time period of our study. This suggests that fast food exposure in our research is near to being "as if" randomly assigned compared to using current data. Moreover, the results hold when exploiting other sources of exogenous variation. Firstly, we study the effects on BMI from a sharp and unanticipated supply shock from this time period's largest fast food company.

Secondly, we estimate an IV regression by instrumenting for the proximity to fast food with the distance to a fast food distribution centre. Our study makes an important contribution by being the first study which comes closer to estimating a causal effect using data outside of the US.

A possible caveat to our analysis is that children might be more affected by fast food outlets in their immediate school environment rather than the home environment. Data limitations hindered us from investigating this relationship directly. We have attempted to account for the access to fast food in the school environment by estimating the effect of fast food density by school catchment areas.

Another concern might be that our sample size is not large enough to detect the very small effect sizes demonstrated in the studies which do find an effect of fast food proximity on body weight. We have tried to address this issue by using different functional forms of distance to fast food. The impact of fast food proximity on takeaway consumption supports our overall conclusions. Given the upper bound on the effect on takeaway purchases, it is not likely that the spatial access to fast food had a substantial impact on weight gain. This is particularly likely to happen if people offset takeaway consumption with eating less calories at home as shown in Anderson & Matsa (2011). The overall conclusions are also in line with Griffith et al. (2016) who show that while there has been an increase in calories from food eaten out of home from the 1980's and onwards, there has been a decrease in total calories purchased. Thus, we conclude that it is unlikely that the introduction of fast food outlets caused the obesity pandemic.

While this particular time period allows for a better identification of fast food exposure on BMI, one might question whether our findings can be extrapolated to today. Currently, fast food density is higher and the relative price of fast food compared to other foods have changed as have other determinants of energy intake and expenditure. Therefore, more evidence is needed. Another potential explanation of why we do not find an effect is that the impact of fast food proximity on obesity may be context-, or country specific, see Dunn (2010), Dunn et al. (2012), Grier & Davis (2013), Anderson & Matsa (2011). As fast food outlets have started to proliferate in middle- and low income countries, future studies should analyse data from such contexts as well.

The second empirical chapter reveals important implications of the intake of a particular micronutrient, on human capital. Iodine deficiency is found in most countries in the world. More than 140 nations have implemented Universal Salt Iodisation (USI) legislation on the initiative by the WHO (Andersson et al. 2010). Abundant epidemiological

research show associations between iodine deficiency and cognition (Zimmermann 2012). Yet, causal evidence among humans is limited and no study has evaluated the potential impact of widespread USI legislation on cognitive skills.

This thesis contributes to the literature by analysing whether the exposure to mandatory USI during early life has impacted cognitive test scores of children in India. I use a difference-in-differences strategy, comparing cohorts who were in early life after the implementation of the policy to earlier cohorts, across districts with and without a geographical predisposition to iodine deficiency. I analyse the effects on test scores using large annual cross–sectional data from the Annual Status of Education Report (ASER). The results demonstrate that exposure to USI in-utero and up to age 2 increased the likelihood of mastering basic numeracy and literacy by 1.9 - 4 percentage points at ages 5-7. Girls also improved their overall literacy score which includes more difficult reading tasks.

The effects on cognition are helpful in explaining the mechanism of the positive impact of historical access to iodised salt or targeted iodine supplementation programmes, observed in previous studies such as; Adhvaryu et al. (2018), Politi (2010*b*,*a*), Field et al. (2009), on schooling attainment and labour market outcomes. As this chapter finds evidence for stronger cognitive effects for girls, this can also explain the relatively larger effects found for women in the aforementioned papers.

This study highlights the importance of sustainable and enforced USI policies, particularly in countries with a high overall prevalence of undernutrition. In the Indian context, USI has raised cognitive skills at least as much as avoiding a drought in utero (Shah & Steinberg 2017) and more than being exposed to a sanitation campaign in early life (Spears & Lamba 2016). While iodised salt coverage has increased greatly in India, many other developing countries still have low levels of consumption. For nations with an average consumption of iodised salt at, or below 10%, increasing the coverage to 90% would increase children's basic academic skills by at least 10%.

The advantage of the dataset used for the analysis in this chapter is that it tests both in- and out of school children at home. Therefore, the results are not driven by selection in schooling enrolment or attendance. The downside with the ASER survey is that it does not provide information on nutritional intake. The analysis therefore relies on an intention-to-treat design as I do not observe the consumption of iodised salt during early life for the children studied in this chapter.

The obstacle of missing data on households' iodine content of their salt has been overcome in the next chapter which examines the implications of iodised salt use for children's

heights. Medical research also suggests that iodine intake is needed for various physiological processes affecting somatic growth. Many studies in economics, across high and low income countries, document robust associations between height in childhood and other human capital outcomes, such as educational attainment and labour market outcomes. Undernutrition in early life has been established to reduce height in childhood and in adulthood. Less is known about what particular aspects of undernutrition are causing short stature. Therefore, the last empirical chapter is concerned with the role of iodised salt availability in determining children's heights.

I analyse the impact of the access to adequately iodised salt on Height-for- Age Z-scores (HAZ) for children up to 12 months in rural India. Data from the Indian 2005-2006 Demographic and Household Survey (DHS) is used where HAZ and the households' consumption of iodised salt is measured and reported objectively. I estimate an IV regression and instrument for the availability of iodised salt with the distance to the main salt producing state. The exogenous variation stems from differences in monitoring of the iodine content of salt depending on the mode of transport. The choice of transport is subsequently determined by the distance from the salt producer to a household's state of residence. The empirical strategy in this study improves upon the methodology used in the few existing non-trial studies assessing the associations between iodised salt and children's growth.

The IV estimates find that iodised salt improves height-for-age by 0.664 standard deviations and increases height by 1.845 cm, on average. Furthermore, the effects are larger for the younger children up to 6 months old and for children residing in states with a higher natural propensity for iodine deficiency. The effect sizes in this study are larger than the effects from public policies related to sanitation and supplementary feeding found in Hammer & Spears (2016), Jain (2015).

A potential methodological limitation is that I am unable to control for unobserved differences across small geographical areas. The survey does not contain district level identifiers and due to strong multi-collinearity in the instrument and state of residence, I am not able to account for state level fixed effects. However, I control for a vast set of child, household and state specific covariates in addition to fixed effects at the regional level and regional-specific-time variation. Furthermore, no effect is found on children's weights which is consistent with the lack of an established physiological relationship between iodine and adipose tissue. This also suggests that the main results are not driven by an overall unobserved increase in nutritional availability. Additionally, reduced form placebo regressions point to a lack of a systematic relationship between the IV and other possible

determinants of children's heights.

Even though this thesis is not concerned with identifying the effect of how height is related to other human capital outcomes, it has been suggested by Case (2008) that the link between height and various human capital outcomes is primarily driven by the strong association between height and cognition. This thesis demonstrate evidence in favour of this mechanism by showing that iodine is an underlying determinant of both cognition (Chapter 2) and height (Chapter 3). Spears (2012*a*) shows that the height-cognition gradient is up to 25 times larger in India compared to the US. Therefore, this calls for further policy efforts to increase iodised salt consumption particularly in low income countries.

Moreover, the third empirical chapter documents that households experience differential access to iodised salt depending on the feasibility for monitoring. This highlights the importance of taking various structures related to the market for salt and other food vehicles into account when designing and enforcing fortification policies.

This thesis has shown what matters, and what is less likely to matter, for nutritional intake and its corresponding effects on other human capital outcomes of children in high and low income countries. Moreover, this project has highlighted the importance of studying the impact of adequate nutritional intake, beyond energy accounting and macro nutrients, on human capital development. A caveat throughout all chapters is the lack of biological information related to the accumulation of the nutritional components of the specific foods studied in this thesis. In the first empirical chapter, body fat percentage, blood lipids or blood glucose levels would have been preferred indicators for potentially harmful nutritional outcomes of fast food consumption. Chapters 2 and 3 would greatly benefit from observing individual levels of iodine in the body.

The outlook for future studies relating to nutrition is very optimistic as more and improved data on various biomarkers related to health and nutrition is becoming available. For example, an increasing number of recent data sets from high income countries include biomarker information on various measures of adiposity and genetics. Therefore, future studies can, for example, investigate potential heterogeneous effects of the spatial environment on various measures of inadequate nutritional intake with respect to genetic predisposition for obesity. The wide spread DHS programme has also started collecting additional biomarkers, including urinary iodine excretion. Future studies can make use of this information to analyse the dose-response relationship between societal factors and nutritional status. Prospective research based on such improved data will be able to ad-

vance our knowledge on the interaction between society and human biology further and improve policy making.

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Appendix A

1

A.1 Data appendix

A.1.1 Fast food outlet data

McDonald's opening dates and locations were obtained direct from the company's UK head office and included information on store location (including postcode and telephone number), store type (franchised, company owned, drive through facility or not), exact opening date and store number. Out of the 1260 McDonald stores opened between 1974 and 2006, 230 locations opened in or prior to 1986.

KFC opening dates and locations were also obtained from the company's UK head office. This list included the store name, opening date, address with full postcode, and longitude and latitude. Out of the 736 restaurants opened between 1968 and 2008, 82 (11.14 %) of those stores were opened during the relevant research period. None of those stores closed prior to 1986 or had missing postcode entries.

Burger King and Wimpy head offices did not supply a list the opening and closing date of their stores. Therefore, old copies of the White Pages from the British Telecom archive in London were consulted to obtain first dates of listing. Since entry listings in the White Pages are free and all outlets had a telephone, we judge the listings to be representative of all the companies' outlets. A comprehensive search of all published directories throughout the UK from 1977 through 1986 for Wimpy and Burger King restaurants was conducted. The start dates were determined based on Burger King and Wimpy history, BCS 1970 data availability, and research period. This yielded a total of 676 Wimpy store locations and 11 Burger King locations between 1977 and 1986.

Information on area, street number and street address, was collected. Since the postcode was not provided in many cases, the "Postcode Anywhere" address management web

service was used to match the company, building, street, and town to postcodes. Because many old Wimpy outlets have closed we matched the locality of the street number, street address, and town rather than company. Out of the 676 Wimpy locations, 592 were exact matches (87.6%) percent, 54 (8%) close matches, and 30 (4.4%) no matches. If a specific building or street number was not located, the street number was adjusted by one unit until a match was found. We went as far as 10 street numbers in either direction until the nearest retail unit to the original address was located. These locations comprise the close matches found. All Burger King locations were perfectly matched. Out of the 213 locations that McDonald's had opened between 1974 and 1986, 62 (29%) had no original postcode. All omissions were perfectly matched. KFC's locations, between 1968 and 1986, had no missing postcodes.

No closures were reported for KFC. Information on closing dates was not given by McDonald's UK head office. We know that 6 out of the 230 McDonald's outlets were closed prior to 2006 but we do not know when. As information on Wimpy and Burger King restaurants was retrieved from old copies of the White Pages we were not able to identify if some of the outlets which opened prior to 1987 closed in, or prior to, 1986. From Figure 1.1 we note that most McDonalds' outlets opened up in 1986. In that year, McDonald's introduced the first franchise-run outlet, "drive-thru and the Happy Meal. Wimpy outlets started to close down starting from 1986 due to the fierce competition from McDonald's (Stead 2017). The largest decline of Wimpy outlets occurred in the early 1990s. In 1989 there were 381 Wimpy outlets left and Grand Metropolitan purchased its owner United Biscuits and turned many old Wimpy outlets into Burger King (Bold 2017, The Herald 1989).

There are no national records of the locations of fish and chip shops during the time period of the study. Moreover, fish and chips differ from other fast food as fish and chip sales were (and are) concentrated very much at particular established times of day, early lunchtime and evening, often very late evening, and do not normally operate outside those times. Nor, for historical reasons, did they operate on Sundays. By contrast, burger outlets commonly open from around 9 am, seven days a week and supply continually until mid or late evening (Sault et al. 2002). As seen in FigureA.1 below, fish and chips consumption remained rather stable until 1986.Therefore fish and chips can be understood as a constant background factor. This is in contrast to the trends in the consumption of burgers, buns and other meat products which often are sold at fast food restaurants.

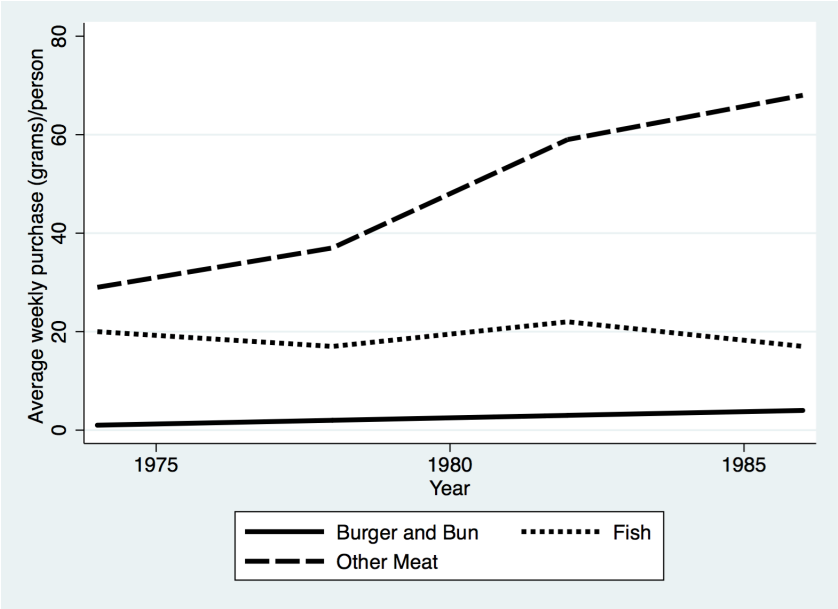


Figure A.1: Takeaway purchases by type, 1974-1984

Data source: Department for Environment Food & Rural Affairs: National Food Survey 1974-1984

Following Kwate et al. (2009), we do not consider ethnic takeaway as fast food in our analysis as it tends to have a greater variety of options that are healthier than fast food.The only research to date on the effects of ethnic "fast food" on weight gain is Giuntella (2018) who shows that living in proximity to a Mexican restaurant is associated with a lower likelihood of excessive weight gain compared to living close to conventional fast food chains. We do not include pizza in our primary definition of fast food as the largest franchise of pizza eateries during the time period of our study - "Pizzaland", was a sit-down, full-service restaurant.Moreover, the first franchised pizza delivery outlet in the UK (Pizza Hut) opened in 1988 which is after the time period of our study (Piz 2009).

Fast food distribution data

The distance from a BCS respondent's home to one's closest distribution centre was used as an IV for the distance to one's closest fast food outlet in a TSLS regression. The following distribution centres were identified : McDonald's McKey Food Service (Milton Keynes), Golden West Foods (Watford) and Golden West Foods (Hernel Hempstead). McKey food Service supplies hamburger patties, bacon and pork products and Golden West Foods supply Buns, ketchup, milk shake syrups, multi-temperature distribution. McKey Food Service was established in 1978 to exclusively supply McDonald's with hamburger patties. The main facility in Milton Keynes was built in 1980. Golden West Foods was established

in Watford in 1977 and relocated to a in Hemel Hempstead in 1982. The distribution centres for Wimpy are the following, Henry Telfer Ltd (Cadby Hall, Stratford and Moulton Park Estate, Northampton) producing meat products. Bolton Abattoir Ltd (Bolton), J.W. French Ltd (Fyfield Wick, Abingdon), Chessington Bakery (Chessington), Crawley Bakery (Crawley).

A.1.2 British Cohort Survey, summary statistics and baseline regressions

British Cohort Survey

From the BCS 1970 data, children were selected for study if they met the following requirements: their postcode was recorded at time of interview and available via the Centre for Longitudinal Studies, and the medical examination form, consisting of height and weight, was completed. From 16,135 children, only those in the 1986 sweep (11,622 children) also had available information on postcodes.

Due to the confidentiality agreement between the BCS70 cohort and the CLS which conceals the identity of the individuals, the CLS agreed to provide only the distances between all fast food outlets and each child of the 1986 sweep provided the fast food outlets' postcodes without revealing further, identifying information. The following procedure was used to collect distances: after obtaining the postcodes for each outlet, the outlet and cohort member postcodes were converted to XY coordinates using postcode directories from UKBORDERS. These directories contain complete versions of current and historical postcode directories as well as a Grid Reference for each postcode. 96% (914) of all outlets and 98% (11,422) of the entire 1986 sample size was coded. Next, Pythagoras's theorem was used to obtain distances from each individual to all outlets- this provided straight line point A to B distances. The nearest outlet was then identified and distance recorded. The duration of time the children were exposed to the nearest outlet was calculated by taking the difference between the year of the survey (1986 or 1980), and the year the outlet opened.

Although postcodes were not available for the 10-year-old children, locations were assigned to those individuals that remained in their LEA between age 10 and 16. From the 14,940 children of the 1980 sweep, 66% (9,831) remained in their respective LEAs. Since information on weights at age five was not recorded in the 1975 survey, this wave was excluded from the analysis.

From the 11,622 teenagers, 53% (6,143) had complete medical examination forms. The sample of 16 year olds fell to 5,498 after removing the people that had incomplete height or weight responses and did match the individuals with coded postcodes from the pool of 10-year-olds. Of the 9,831 10-year-old children, 81% (8,011) had complete height/weight responses and corresponded with the individuals that had coded postcodes.

Explanatory Variables

The following variables were used from BCS 1980:

Father's Social Class I use a generated measure of father's social class in 1980. It is classed into the 5 principal classes (I, II, III (non manual and manual), IV, V (`BCS3FCL`). The first group is used as the reference category. These are the following categories.

1 Professional occupations.
2 Managerial and technical occupations.
3.1 Skilled occupations (non manual).
3.2 Skilled occupations (manual).
4 Partly skilled occupations.
5 Unskilled occupations.
6 Unclassifiable occupations or occupations with insufficient info/armed forces/carer/unemployed/sick/
7.Missing information.

Mother's BMI: Constructed from variable `e1_2` - mother's weight in kgs and variable `e1_1` - mother's height in centimetres.

Father's BMI: Constructed from variable `e2_2 - PI` - father's weight in kgs and variable `e2_1` - father's height in centimetres.

Child's BMI at age 10: Constructed from variable `meb19_1` -child's weight in grams and `meb17` - child's height in millimetres.

The following variables were used from the BCS 1986:

Ethnic group: Variable `C6_14` 1=European, 2= West Indian, 3=Asian, 4=Other.

Sex: Describes child as being male or female, constructed from variable `sex86` where male=1 and female=0.

Smoker: variable `gh11` describes whether teen is a smoker (=1) or non-smoker (=0). Survey asked if teenager smokes at all. We recoded the variable so that yes, cigarettes, yes, cigars, pipes, etc means the teen does smoke, and never smoked and no, but ex smoker to

mean the teen does not smoke. Those questions that had responses not sure were coded as missing values.

Household Ownership of Microwave: Variable `PG1_22` - Has your household a microwave oven? Yes=1 and No=0.

Land: Variable `OA2_1` Teenager's Country of Birth. 0 if the respondent lives in England, 1 if the respondent lives in Scotland and 2 if the respondent lives in Wales.

London: 1 if the respondent lives in London, 0 if respondent does not live in London.

Urban: Variable `M307`: Inner Urban Area: Yes=1 and No=0.

Table A.1: Descriptive statistics for the whole sample

Variable	Mean	SD	N
Body Mass Index at age 16	21.255	3.209	4,999
Proportion Obese	0.086	0.2730	4,999
Proportion Overweight	0.217	0.412	4,999
Proportion having a fast food outlet within 5 miles	0.187	0.391	11,621
Proportion having a fast food outlet within 2 miles	0.077	0.266	11,621
Proportion having a fast food outlet within 1 mile	0.026	0.164	11,621
Distance to closest fast food outlet	5.302	11.593	11,621
Duration of closest fast food outlet in 1986	2.543	4.077	11,621
Intensity of fast food exposure	4.513	12.431	11,621
Takeaway per week	1.016	1.255	5,400
Household owns a microwave	0.428	0.495	6,969
BMI at age 10	16.868	2.097	8,520
Mother's BMI	23.425	3.846	9,250
Father's BMI	24.491	3.025	8,785
Proportion of smokers	0.106	0.308	10,016

A.1.3 Summary statistics and determinants of BMI

Table A.2: Descriptive statistics - takeaway and school meal consumption

	Mean	SD	N
Eats Burgers	0.382	0.486	11555
Eats Fish and Chips	0.295	0.456	11555
Eats Pizza	0.330	0.470	11555
Eats Indian Takeaway	0.0273	0.163	11555
Eats Chinese Takeaway	0.160	0.367	11555
Lunch Bought Outside	0.109	0.312	11555
Free School Meal	0.0550	0.228	11555
Brought Lunch	0.167	0.373	11555
Went Home For Lunch	0.180	0.385	11555

Table A.3: Density of fast food outlets per LEA and its effect on buying lunch outside of school

	Lunch Bought Outside	Lunch Bought Outside
Average distance to fast food outlet per LEA	0.002**	0.002**
	(0.001)	(0.001)
Control Variables		✓
Observations	9100	4965
R^2	0.001	0.020

Dependent variable is the probability of buying lunch outside of school. The following control variables are included in specification 2: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

A.1.4 Specification checks of fast food exposure

Table A.4: Average distance to fast food per LEA

BMI		
	BMI	BMI
Average distance to closest fast food outlet per LEA	-0.002	0.001
	(0.010)	(0.008)
Observations	4567	3102
R^2	0.000	0.340

Dependent variable is BMI, overweight and obese. The estimates are for the sample of respondents who remained in their LEA between age 10 and 16. The following set of control variables are included in specifications 2 - 8: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses.

Table A.5: Effect of first fast food outlet in 1, 2 and 3 years

	BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A; First Fast Food outlet established in the past year						
First fast food outlet within 1 mile (> 1984)	0.050	0.115				
	(0.150)	(0.157)				
First fast food outlet within 3 miles (> 1984)			0.001	-0.032		
			(0.109)	(0.102)		
First fast food outlet within 5 miles (> 1984)					0.064	-0.006
					(0.108)	(0.099)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341
Panel B: First Fast Food Outlet established in the past 2 years						
First fast food outlet within 1 mile (> 1983)	0.054	0.104				
	(0.155)	(0.160)				
First fast food outlet within 3 miles (> 1983)			0.009	-0.049		
			(0.104)	(0.092)		
First fast food outlet within 5 miles (> 1983)					0.040	-0.050
					(0.106)	(0.091)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341
Panel C: First Fast Food outlet established in the past 3 years						
First fast food outlet within 1 mile (¿1982)	-0.021	0.009				
	(0.164)	(0.163)				
First fast food outlet within 3 miles (> 1982)			-0.017	-0.034		
			(0.111)	(0.108)		
First fast food outlet within 5 miles (> 1982)					-0.005	-0.042
					(0.110)	(0.105)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341

Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4, 6 and 8: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.6: Differences analysis

BMI						
First outlet opening 1980-1986, within:	(1)	(2)	(3)	(4)	(5)	(6)
$\leq$ 1 mile	-0.278*	-0.222				
	(0.146)	(0.180)				
$\leq$ 3 miles			-0.084	0.006		
			(0.130)	(0.139)		
$\leq$ 5 miles					-0.102	-0.048
					(0.119)	(0.122)
Observations	2136	1710	2136	1710	2136	1710
R^2	0.339	0.343	0.338	0.342	0.338	0.342

Notes: Dependent variable is BMI. Analysis carried out for the pooled sample of adolescents who remained in their LEA from age 10. The following control variables are included in specifications 2, 4 and 6 gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.7: The effect of distance and duration to the first fast food outlet within 5 miles.

BMI						
Exposure to the first fast food outlet $\leq$ 5 miles	(1)	(2)	(3)	(4)	(5)	(6)
Duration	0.003	0.006	0.002	0.007	0.002	0.006
	(0.013)	(0.012)	(0.013)	(0.012)	(0.013)	(0.012)
$1/(Distance^2)$			0.000	-0.004**	-0.001	-0.006**
			(0.003)	(0.002)	(0.005)	(0.003)
Duration * $1/(Distance^2)$					0.000	0.000
					(0.001)	(0.000)
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.342	0.000	0.342

Notes: Dependent variable is BMI. Analysis carried out for the pooled sample of adolescents who remained in their LEA from age 10. The following control variables are included in specifications 2, 4 and 6 gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.8: Intensity regressions

	BMI			
	(1)	(2)	(3)	(4)
Intensity, $1/(Distance^2)$	0.000	-0.001*		
	(0.001)	(0.000)		
Intensity, $1/(Duration^2)$			0.000	-0.000
			(0.000)	(0.000)
Observations	4536	3093	4536	3093
R^2	0.000	0.341	0.000	0.341

Notes: Dependent variable is BMI. Analysis is carried out for the pooled sample of adolescents who remained in their LEA from age 10. The following control variables are included in specifications 2, 4 and 6 gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

A.1.5 Results for outlets within 5 miles

Table A.9: Effect of fast food proximity on BMI: Continuous Distance

	BMI							
Exposure to nearest fast food outlet within 5 miles	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Distance	0.007	0.042	0.071	0.235				
	(0.040)	(0.045)	(0.018)	(0.013)				
Distance^2			-0.016	-0.039				
			(0.036)	(0.039)				
$1/Distance$					0.009	-0.095	-0.000	-0.156**
					(0.045)	(0.035)	(0.074)	(0.073)
1/ Distance^2							0.000	0.000
							(0.000)	(0.000)
Observations	3249	2195	3249	2195	3249	2195	3249	2195
R^2	0.000	0.344	0.000	0.341	0.000	0.345	0.000	0.342

Notes: Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4, 6 and 8: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.10: Effect of fast food proximity on BMI: Distance bins

	BMI							
Distance to nearest fast food outlet within 5 miles	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Fast food outlet $\leq$ 0.5 mile	0.155	- 0.382					0.090	-0.255
	(0.565)	(0.431)					(0.651)	(0.424)
Fast food outlet $\leq$ 1 mile			0.096	-0.197			-0.103	-0.248
			(0.350)	(0.258)			(0.350)	(0.324)
Fast food $\leq$ 2 miles					0.162	0.056	0.188	0.132
					(0.179)	(0.173)	(0.200)	(0.202)
Observations	3249	2195	3249	2195	3249	2195	4536	3093
R^2	0.000	0.341	0.000	0.341	0.000	0.341	0.000	0.341

Notes: Dependent variable is BMI. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.11: Effect of fast food proximity and duration on BMI

	BMI					
Exposure to fast food outlets within 5 miles	(1)	(2)	(3)	(4)	(5)	(6)
Duration	0.019*	0.002	0.019	0.003	0.019*	0.002
	(0.011)	(0.010)	(0.011)	(0.010)	(0.011)	(0.011)
$1/(Distance^2/10^8)$			0.004	0.041*	0.025	-0.067**
			(0.000)	(0.002)	(0.037)	(0.028)
$Duration * 1/(Distance^2/10^8)$					0.003	0.003
					(0.006)	(0.004)
Observations	3249	2,195	3249	2,195	3249	2,195
R^2	0.000	0.344	0.000	0.345	0.000	0.345

Notes: Dependent variable is BMI. Analysis carried out for the sample of adolescents who remained in their LEA from age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.12: Effect of fast food intensity on BMI

	BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
Intensity of fast food	0.004	-0.004				
	(0.004)	(0.004)				
Weighted Intensity of fast food			0.002	-0.002		
			(0.002)	(0.002)		
Ln(intensity) of fast food					-0.000	-0.029
					(0.062)	(0.067)
Observations	3249	2195	3249	3093	2331	1571
R^2	0.000	0.344	0.000	0.344	0.000	0.355

Notes: Standard errors clustered on LEA are shown in parentheses. Analysis carried out for the pooled sample of adolescents who remained in their LEA from age 10. The following set of control variables is included in specifications 2, 4 and 6 gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. * $p < .10$, ** $p < .05$, *** $p < .01$.

A.1.6 Results: Probability of being overweight and obese

We cannot use the same BMI cut-offs for children or adolescents as for adults as we need to account for growth spurts. The BMI of the children in our sample was compared with UK reference data for BMI by means of software or a Microsoft Excel add-in to access growth references based on the LMS method created by University College London Institute of Child Health (Cole et al. 2007). The Child Growth Foundation's obesity charts, used by health practitioners, utilize this 1990 reference data. The 1990 reference data encompasses 11 UK surveys carried out between 1978 and 1990 from a nationally representative sample of English children. According the software, the following cut-offs were used to identify obese and overweight children, where; a BMI of 24.90 kg/m^2 and 22.75 kg/m^2 are the 95$^{\text{th}}$ and 85$^{\text{th}}$ percentiles respectively for boys aged 16 and a BMI of 25.85 kg/m^2 and 23.54 kg/m^2 are the 95$^{\text{th}}$ and 85$^{\text{th}}$ percentiles respectively for girls aged 16. For 10 year olds the cutoffs are; a BMI of 20.27 kg/m^2 and 18.59 kg/m^2 are the 95$^{\text{th}}$ and 85$^{\text{th}}$ percentiles respectively for boys aged 10. A BMI of 21.35 kg/m^2 and 19.44 kg/m^2 are the 95$^{\text{th}}$ and 85$^{\text{th}}$ percentiles respectively for girls aged 10 (Cole et al. 2007).

Table A.13: Determinants of the probability of being overweight

	(1)	(2)	(3)	(4)	(5)	(6)
BMI at age 10	0.091***	0.091***	0.086***	0.085***	0.085***	0.085***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)
Girl		0.016	0.014	0.011	0.011	0.007
		(0.010)	(0.011)	(0.011)	(0.011)	(0.012)
Father's BMI			0.012***	0.012***	0.012***	0.012***
			(0.002)	(0.002)	(0.002)	(0.002)
Mother's BMI			0.009***	0.008***	0.008***	0.008***
			(0.002)	(0.002)	(0.002)	(0.002)
Asian				0.044	0.042	-0.070
				(0.074)	(0.072)	(0.054)
West Indian				-0.014	-0.016	-0.076***
				(0.042)	(0.042)	(0.027)
Other Ethnicity				-0.099	-0.098	-0.057
				(0.065)	(0.066)	(0.064)
Social Class II				0.028	0.028	0.040*
				(0.023)	(0.023)	(0.024)
Social Class III				0.046**	0.046**	0.054**
				(0.021)	(0.021)	(0.022)
Social Class IV				0.034	0.034	0.050*
				(0.027)	(0.027)	(0.029)
Social Class V				0.072**	0.072**	0.073*
				(0.036)	(0.036)	(0.040)
Social Class VI				0.009	0.010	-0.016
				(0.061)	(0.061)	(0.061)
Urban					-0.000	-0.001
					(0.017)	(0.016)
London					-0.003	-0.039
					(0.031)	(0.035)
Scotland					-0.029	-0.033
					(0.027)	(0.028)
Wales					-0.005	-0.000
					(0.016)	(0.017)
Household has a microwave						0.040***
						(0.014)
Smoker						-0.035*
						(0.020)
Birth Weight						-0.011
						(0.015)
Constant	-1.322***	-1.328***	-1.749***	-1.753***	-1.750***	-1.725***
	(0.049)	(0.049)	(0.070)	(0.076)	(0.076)	(0.095)
Observations	3996	3996	3683	3531	3531	3093
R^2	0.216	0.217	0.237	0.235	0.236	0.238

Standard errors clustered on LEA are shown in parentheses

Dependent variable is the probability of being overweight at age 16 for all who remained in their LEA.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table A.14: Determinants of the probability of being obese

	(1)	(2)	(3)	(4)	(5)	(6)
BMI at age 10	0.051***	0.051***	0.048***	0.047***	0.047***	0.046***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Girl		0.004	0.004	0.002	0.002	0.002
		(0.007)	(0.008)	(0.008)	(0.008)	(0.009)
Father's BMI			0.006***	0.006***	0.006***	0.006***
			(0.002)	(0.002)	(0.002)	(0.002)
Mother's BMI			0.006***	0.006***	0.006***	0.005***
			(0.001)	(0.001)	(0.001)	(0.002)
Asian				-0.061	-0.055	-0.076**
				(0.048)	(0.049)	(0.037)
West Indian				0.017	0.021	-0.024
				(0.039)	(0.040)	(0.027)
Other Ethnicity				0.016	0.023	0.040
				(0.066)	(0.065)	(0.069)
Social Class II				-0.014	-0.015	-0.013
				(0.015)	(0.015)	(0.016)
Social Class III				0.015	0.015	0.018
				(0.015)	(0.015)	(0.016)
Social Class IV				0.007	0.007	0.011
				(0.019)	(0.019)	(0.019)
Social Class V				0.024	0.024	0.032
				(0.023)	(0.023)	(0.025)
Social Class VI				-0.007	-0.009	-0.011
				(0.036)	(0.036)	(0.036)
Urban					-0.004	0.002
					(0.010)	(0.011)
London					-0.012	-0.025
					(0.020)	(0.019)
Scotland					0.001	0.003
					(0.017)	(0.017)
Wales					-0.003	-0.003
					(0.011)	(0.012)
Household has a microwave						0.023**
						(0.009)
Smoker						-0.003
						(0.013)
Birth Weight						-0.005
						(0.008)
Constant	-0.772***	-0.774***	-1.015***	-1.004***	-1.001***	-0.977***
	(0.049)	(0.048)	(0.070)	(0.073)	(0.073)	(0.073)
Observations	3996	3996	3683	3531	3531	3093
R^2	0.150	0.150	0.166	0.165	0.165	0.163

Standard errors are clustered on LEA.

Dependent variable is the probability of being obese at age 16 for all who remained in their LEA.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table A.15: Distance regressions: Probability of being overweight or obese

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A: Probability of being overweight								
Distance to nearest fast food outlet								
Distance	0.000	-0.000	0.002	-0.001				
	(0.001)	(0.001)	(0.002)	(0.002)				
$Distance^2/10^4$			-0.306	0.164				
			(0.251)	(0.229)				
$1/Distance$					-0.001	-0.008	0.000	0.000
					(0.005)	(0.005)	(0.008)	(0.010)
$1/Distance^2/10^4$							-0.000	-0.000
							(0.000)	(0.000)
Control Variables		✓		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.238	0.000	0.238	0.000	0.238	0.000	0.239
Panel B: Probability of being obese								
Distance to nearest fast food outlet								
Distance	-0.000	-0.001	0.001	-0.001				
	(0.001)	(0.000)	(0.001)	(0.001)				
$Distance^2/10^4$			-0.239*	0.044				
			(0.140)	(0.115)				
1/Distance					0.001	-0.005*	-0.003	-0.001
					(0.004)	(0.003)	(0.006)	(0.005)
$1/Distance^2/10^4$							0.000	-0.000
							(0.000)	(0.000)
Control Variables		✓		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.164	0.001	0.164	0.000	0.164	0.000	0.164

Analysis on sample of respondents who remained in the same LEA as at age 10. Dependent variable is probability of being overweight or obese at age 16. The following control variables is included in specifications 2, 4 and 6: lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.16: Effect of fast food proximity on the probability of being overweight and obese: Distance bins

	BMI									
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Panel A: Probability of being overweight										
Distance to nearest fast food outlet										
Fast food outlet $\leq$ 0.5 mile	-0.021	-0.058							-0.026	-0.050
	(0.065)	(0.065)							(0.082)	(0.077)
Fast food outlet $\leq$ 1 mile			-0.003	-0.020					-0.005	-0.016
			(0.031)	(0.034)					(0.046)	(0.041)
Fast food $\leq$ 2 miles					0.007	0.000			0.018	-0.004
					(0.022)	(0.022)			(0.032)	(0.030)
Fast food $\leq$ 5 miles							-0.003	0.013	-0.008	0.017
							(0.014)	(0.015)	(0.016)	(0.017)
Observations	4536	3093	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.238	0.000	0.238	0.000	0.238	0.000	0.238	0.000	0.238
Panel B: Probability of being obese										
Distance to nearest fast food outlet										
Fast food outlet $\leq$ 0.5 mile	-0.008	-0.044							-0.023	-0.066
	(0.045)	(0.045)							(0.055)	(0.056)
Fast food outlet $\leq$ 1 mile			0.009	0.010					0.015	0.004
			(0.024)	(0.031)					(0.034)	(0.043)
Fast food $\leq$ 2 miles					0.004	0.021			0.004	0.020
					(0.015)	(0.015)			(0.020)	(0.021)
Fast food $\leq$ 5 miles							-0.001	0.012	-0.004	0.006
							(0.009)	(0.012)	(0.011)	(0.014)
Observations	4536	3093	4536	3093	4536	3093	4536	3093	4536	3093
R^2	0.000	0.164	0.000	0.163	0.000	0.164	0.000	0.164	0.000	0.164

Notes: Dependent variable is the probability of being overweight and obese, respectively. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p <$ .10, ** $p <$ .05, *** $p <$ .01.

Table A.17: Effect of duration of fast food exposure on the probability of being overweight and obese

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Probability of being overweight						
Duration of nearest fast food outlet within:						
≤ 1 mile	-0.000	-0.001				
	(0.002)	(0.002)				
≤ 3 miles			0.001	0.001		
			(0.001)	(0.002)		
≤ 5 miles					0.000	0.001
					(0.002)	(0.002)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.238	0.000	0.238	0.000	0.238
Panel B: Probability of being obese						
Duration of nearest fast food outlet within:						
≤ 1 mile	-0.000	-0.001				
	(0.002)	(0.002)				
≤ 3 miles			0.001	0.001		
			(0.001)	(0.002)		
≤ 5 miles					0.000	0.001
					(0.002)	(0.002)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.238	0.000	0.238	0.000	0.238

Notes: Dependent variable is the probability of being overweight and obese, respectively. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.18: Effect of distance and duration of fast food exposure on the probability of being overweight or obese

	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Probability of being overweight						
Exposure to nearest fast food outlet						
Duration	-0.000	0.000	-0.000	0.000	-0.000	0.000
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
$1/(Distance^2)/10^8$			-0.000	-0.006**	-0.002	-0.013***
			(0.004)	(0.003)	(0.006)	(0.004)
Duration * $1/(Distance^2)/10^8$					0.000	0.000
					(0.000)	(0.000)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.238	0.000	0.239	0.000	0.239
Panel B: Probability of being obese						
Exposure to nearest fast food outlet						
Duration	0.000	0.001	0.000	0.001	0.000	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
$1/(Distance^2)/10^8$			0.001	-0.004**	-0.003	-0.006***
			(0.003)	(0.002)	(0.004)	(0.002)
Duration * $1/(Distance^2)/10^8$					0.000	0.000
					(0.000)	(0.000)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	4536	3093
R^2	0.000	0.164	0.000	0.164	0.000	0.164

Notes: Dependent variable is the probability of being overweight and obese, respectively. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.19: The effect of fast food intensity on the probability of being overweight or obese

	(1)	(2)	(3)	(4)	(5)	(6)
Panel B: Probability of being overweight						
Intensity of fast food	0.000	0.000				
	(0.000)	(0.001)				
Weighted Intensity of fast food			0.000	0.000		
			(0.000)	(0.000)		
Ln(intensity) of fast food					-0.001	-0.002
					(0.008)	(0.008)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	2407	1617
R^2	0.000	0.238	0.000	0.238	0.000	0.257
Panel B: Probability of being obese						
Intensity of fast food	0.000	-0.000				
	(0.000)	(0.000)				
Weighted Intensity of fast food			0.000	-0.000		
			(0.000)	(0.000)		
Ln(intensity) of fast food					-0.002	-0.001
					(0.005)	(0.005)
Control Variables		✓		✓		✓
Observations	4536	3093	4536	3093	2407	1617
R^2	0.000	0.163	0.000	0.163	0.000	0.177

Notes: Dependent variable is the probability of being overweight and obese, respectively. The sample consists of 16 year olds who did not change LEA since age 10. The following set of control variables is included in specifications 2, 4 and 6: gender, lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

A.1.7 Robustness checks

Table A.20: Effect of access to fast food on child and parental BMI in 1980

	BMI					
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Children's BMI at age 10 in 1980						
Distance to nearest fast food outlet:						
FF $\leq$ 1 mile	-0.108	-0.081				
	(0.131)	(0.164)				
FF $\leq$ 3 miles			-0.049	0.043		
			(0.082)	(0.092)		
FF $\leq$ 5 miles					-0.013	0.019
					(0.085)	(0.087)
Control Variables		✓		✓		✓
Observations	2936	2370	2936	2370	2936	2370
R^2	0.000	0.081	0.000	0.081	0.000	0.081
Panel B: Mother's BMI in 1980						
Distance to nearest fast food outlet:						
FF $\leq$ 1 mile	0.139	-0.096				
	(0.198)	(0.208)				
FF $\leq$ 3 miles			0.036	-0.154		
			(0.144)	(0.159)		
FF $\leq$ 5 miles					0.023	-0.182
					(0.129)	(0.129)
Control Variables		✓		✓		✓
Observations	3149	2370	3149	2370	3149	2370
R^2	0.000	0.020	0.000	0.020	0.000	0.020
Panel C: Father's BMI in 1980						
Distance to nearest fast food outlet:						
FF $\leq$ 1 mile	-0.056	-0.121				
	(0.152)	(0.145)				
FF $\leq$ 3 miles			-0.189	-0.053		
			(0.128)	(0.141)		
FF $\leq$ 5 miles					-0.112	0.033
					(0.114)	(0.123)
Control Variables		✓		✓		✓
Observations	3032	2370	3032	2370	3032	2370
R^2	0.000	0.026	0.001	0.026	0.000	0.026

Notes: Dependent variable is BMI for children and their parents in 1980. The following set of control variables is included in specifications 2, 4 and 6: gender, social class, location, ethnicity, ownership of microwave and smoking status. Additionally, parental BMI and birth weight is controlled for in panel A. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.21: Area level determinants of fast food density in 1974

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mean BMI	-0.000						
	(0.000)						
Prop. Overweight Children		-2.371					
		(15.994)					
Teacher Judges Child to be overweight			6.318				
			(13.888)				
Prop Mother Smoking				1.351			
				(5.486)			
Prop. Father Smoking					-3.339		
					(3.234)		
Prop. do not practice sport- outside						-0.507	
						(7.819)	
Prop. do not practice sport- inside							10.174
							(7.074)
Constant	-15.670	-15.152	-16.093	-16.148	-13.390	-15.338	-20.006
	(0.774)	(2.002)	(1.687)	(2.682)	(1.882)	(2.069)	(3.500)
Constant	2.466	2.541	2.473	2.513	2.511	2.532	2.376
	(0.957)	(0.888)	(0.914)	(0.925)	(0.895)	(0.896)	(0.936)
Observations	63	63	63	63	63	63	63
Pseudo R^2	0.008	0.001	0.003	0.001	0.006	0.000	0.027

Standard errors clustered on county are shown in parentheses. Dependent variable is the count of fast food outlets per county in 1974 with log of population/1000/wards as offset.

Table A.22: Area level determinants of fast food density in 1974

	(1)	(2)	(3)	(4)
Prop. Fathers in Soc. Class III-V	-16.101*			
	(8.503)			
Prop. Fathers left school before age 15		-14.570***		
		(5.532)		
Prop. Mothers left school before age 15			1.495	
			(6.269)	
Prop. Overcrowded Households				-21.130
				(15.194)
Constant	-5.924	-7.419	-16.171	-10.438
	(4.772)	(2.992)	(2.831)	(3.637)
lnalpha				
Constant	2.004	2.000	2.532	2.202
	(0.976)	(0.898)	(0.906)	(1.175)
Observations	63	63	63	63
Pseudo R^2	0.073	0.076	0.001	0.042

Standard errors clustered on LA are shown in parentheses. Dependent variable is the count of fast food outlets per county in 1974 with log of population/1000/wards as offset.

Table A.23: The effect of the proportion of unemployment claimants per ward and fast food density per ward

Fast food density/ward				
	1	2	3	4
% Claimants in 1983	-0.871***			
	(0.344)			
% Claimants in 1984		-0.851***		
		(0.390)		
% Claimants in 1985			-0.735***	
			(0.324)	
% Claimants in 1986				-0.579***
				(0.325)
Constant	-10.889	-10.822	-10.757	-10.721
	(0.088)	(0.097)	(0.087)	(0.087)
Observations	9261	9261	9261	9261
R^2	0.003	0.003	0.003	0.002

Dependent variable is the count of fast food outlets in 1983, 1984, 1985 and 1986, respectively, with log of population/10 000 as offset. Standard errors clustered at LEA in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table A.24: Youth Cohort Surveys 1-7: Predictors of fast food density

	(1)	(2)
Variables	Intensity	Intensity
Mean Overweight	16.667	21.596
	(22.490)	(16.853)
% Non Manual Fathers	-52.185	-21.448
	(36.267)	(27.412)
% Owner Occupiers	23.958	-16.182
	(25.44)	(19.655)
Average Education Score	2.553***	1.859***
	(0.955)	(0.720)
%Youth Unemployment	-565.945***	-282.110***
	(82.830)	(70.809)
% Truancy	161.684***	40.614
	(51.942)	(41.538)
% Dads in Work	-100.999***	-16.345
	(36.099)	(28.890)
% One Parent Families	112.402*	55.262 ***
	(59.109)	(44.799)
% Non-White	-	96.066***
	-	(11.556)
Constant	65.339	16.240
	(39.660)	(30.284)
Observations	96	96
R^2	0.55	0.75

Standard errors are shown in parentheses

* $p < .10$, ** $p < .05$, *** $p < .01$

Table A.25: Wimpy distance degressions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Distance to closest Wimpy	0.001	-0.003	0.033*	0.005				
	(0.010)	(0.007)	(0.017)	(0.014)				
Distance$^2/10^4$ to closest Wimpy outlet			-4.768**	-1.189				
			(0.000)	(0.000)				
$1/Distance$ to closest Wimpy outlet					-0.011	-0.076**	-0.047	-0.127*
					(0.050)	(0.031)	(0.085)	(0.073)
$1/Distance$ to closest Wimpy outlet$/10^4$							0.000	0.000
							(0.000)	(0.000)
Control Variables		✓		✓		✓		✓
Observations	3036	2088	3036	2088	3036	2088	3036	2088
R^2	0.000	0.348	0.002	0.348	0.000	0.349	0.000	0.349

Dependent variable is BMI for the pooled sample of BCS respondents at age 16 in 1986 who did not change LEA since 1980. The following set of control variables is included in specifications 2, 4 and 6: lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table A.26: Wimpy distance regressions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Wimpy $\leq$ 0.5 miles	-0.038	-0.430*							0.028	-0.342
	(0.282)	(0.257)							(0.322)	(0.286)
Wimpy $\leq$ 1 mile			-0.064	-0.224					-0.053	-0.212
			(0.175)	(0.142)					(0.217)	(0.176)
Wimpy $\leq$ 2 miles					-0.048	0.005			0.086	0.150
					(0.130)	(0.115)			(0.168)	(0.152)
Wimpy $\leq$ 5 miles							-0.199	-0.018	-0.234	-0.022
							(0.144)	(0.132)	(0.166)	(0.155)
Control Variables		✓		✓		✓		✓		✓
Observations	3036	2088	3036	2088	3036	2088	3036	2088	3036	2088
R^2	0.000	0.349	0.000	0.349	0.000	0.348	0.001	0.348	0.001	0.349

Dependent variable is BMI for the pooled sample of BCS respondents at age 16 in 1986 who did not change LEA since 1980. The following set of control variables is included in specifications 2, 4 and 6: lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table A.27: Wimpy distance regressions

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Wimpy $\leq$ 0.5 miles	-0.038	-0.430*							0.028	-0.342
	(0.282)	(0.257)							(0.322)	(0.286)
Wimpy $\leq$ 1 mile			-0.064	-0.224					-0.053	-0.212
			(0.175)	(0.142)					(0.217)	(0.176)
Wimpy $\leq$ 2 miles					-0.048	0.005			0.086	0.150
					(0.130)	(0.115)			(0.168)	(0.152)
Wimpy $\leq$ 5 miles							-0.022	0.035	-0.234	-0.022
							(0.110)	(0.094)	(0.166)	(0.155)
Observations	3036	2088	3036	2088	3036	2088	4536	3093	3036	2088
R^2	0.000	0.349	0.000	0.349	0.000	0.348	0.000	0.341	0.001	0.349

Dependent variable is BMI for the pooled sample of BCS respondents at age 16 in 1986 who did not change LEA since 1980. The following set of control variables is included in specifications 2, 4 and 6: lagged BMI, parental BMI, social class, location, ethnicity, ownership of microwave, smoking status and birth weight. Standard errors clustered on LEA are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

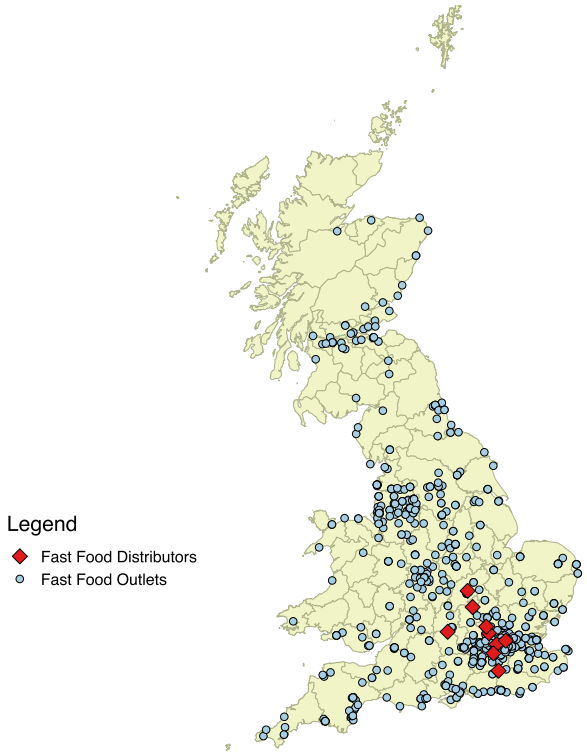


Figure A.2: Fast food outlets established in 1977 and 1978

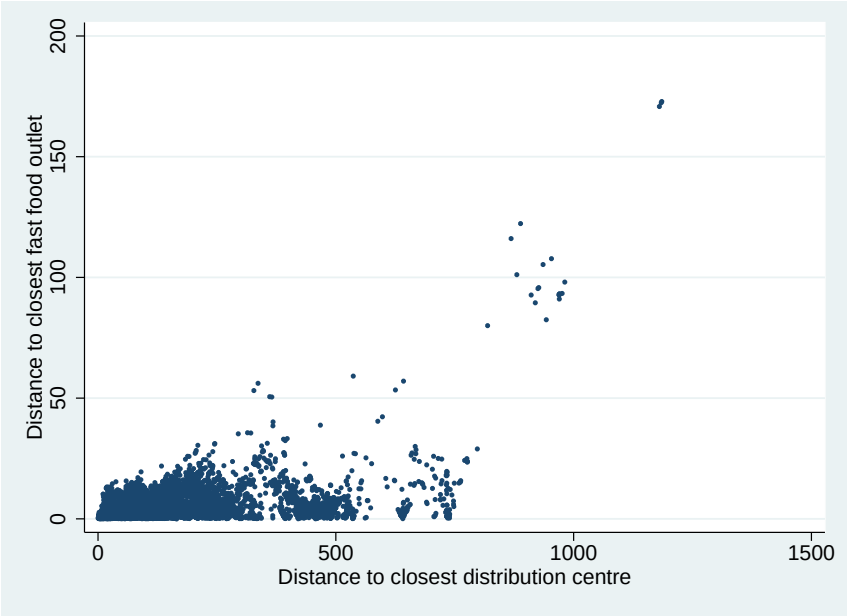


Figure A.3: Scatter plot of the distance to the closest fast food outlet and to the closest fast food distributor

Appendix B

2

B.1 History of iodisation policies in India

Iodine fortification of salt has a long history in India. In 1962, the National Goitre Control Programme was launched with the attempt to provide iodised salt to districts with a high prevalence of goitre. The programme was considered a low priority due to the perception of goitre being a cosmetic concern. Research providing evidence of adverse health effects of iodine deficiency led to a higher priority to its eradication (Pandav et al. 2003). Nationwide iodisation of salt started in a phased manner in 1986 as surveys showed that all states were prone to the deficiency (Pandav et al. 2003, Pandav 2013). The proportion of households consuming iodised salt has been increasing since the 1980s causing a decline in goitre prevalence in previously endemic areas (Toteja et al. 2004). Prior to the implementation of this ban on 27 May 1998, all states except Kerala, Andhra Pradesh and Maharashtra had state-level bans on the sale of non-iodised salt.

Salt policies constitute a sensitive political issue in India. Appearing to force the population to pay higher prices for salt, the legislation of 1998 also resembled the unfair colonial taxes and monopolies on salt. Before, 1923, the salt tax was contributing 1/3 of total revenue earned by the British Government in India (Saline Area Vitalization Enterprise Limited 2005). Such grievances have remained in Indian politics and many dissenting voices, especially with roots in the independence movement were raised which caused the removal of the ban on 13 September 2000 (Pandav 2005).

B.2 Data appendix

B.2.1 ASER data

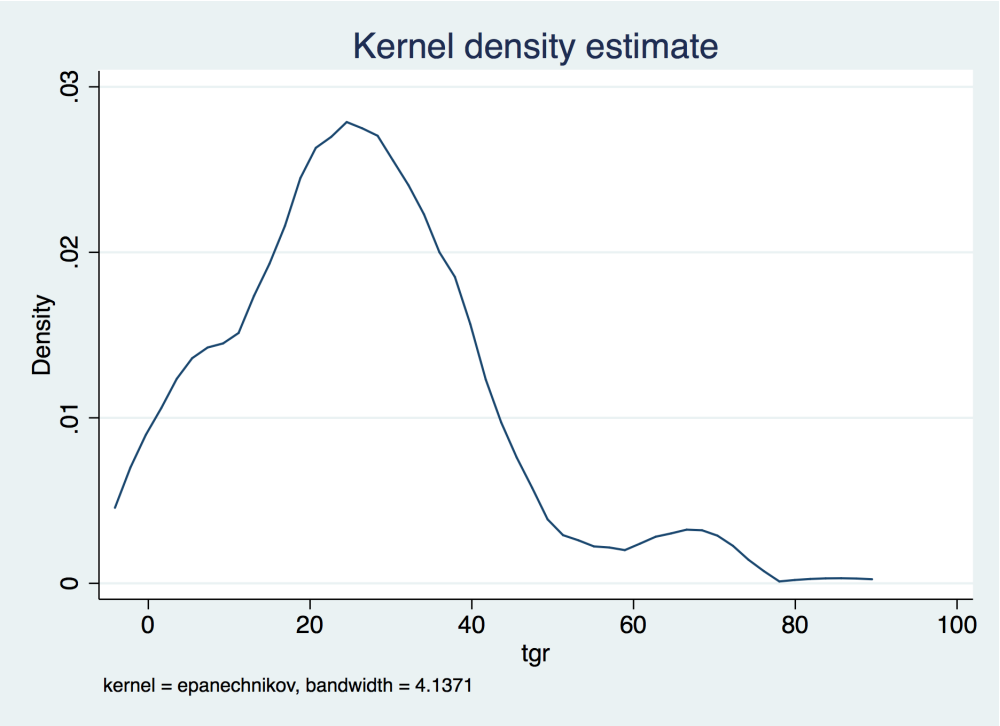
The enumerators conduct the survey on Sundays, when people generally do not work and children are not in school. The enumerators must return to households where children are not present at the time of the survey. The dataset consists of children who are currently enrolled in school, children who have dropped out and children who have never enrolled.

The reading assessment consists of 4 levels of mastery: letter recognition, word recognition, reading comprehension of a short paragraph (a class 1 level text), and a short story (a class 2 level text). Similarly, the math assessment consists of four levels: single-digit number recognition, double-digit number recognition, two-digit subtraction with carry over, and three digit by one digit division. For both tests separately, the child is marked at the highest level he or she can do with scores ranging from 0 to 4: a score of 0 means that the child can not do even the most basic level, a score of 4 means that he or she can do level 4 in the respective subject.

B.2.2 District level total goitre rate data

Simple regressions of the probability of a district being surveyed, on McCarrisson's binary goitre endemicity indicator point to that severely goitrous districts were more likely to be sampled (see Table B.18 in the Appendix). Moreover, a negative and statistically significant relationship appears between the year of survey and the binary goitre. A similar association is found for survey year and TGR as well, see Table B.18. This indicates that more goitrous areas districts likely to be surveyed first and can potentially lead to an overestimation of goitre endemicity in severely endemic areas. 86.31% were goitre endemic according to the earlier definition of endemicity by the WHO used in the report of a cut-off of 10% (Aburto et al. 2014). The WHO revised the cut-offs for goitre endemicity in 1994. Mild endemicity corresponds to a prevalence of 5-19.9%, moderate to 20-29.90% and severe goitre endemicity to 30% or more (Aburto et al. 2014). Using the revised definitions, we observe that 24.33% of the surveyed school children in India prior to any policies on salt iodisation, had mild iodine deficiency, 27% were moderately iodine deficient and 36.50 % have severe iodine deficiency.

Figure B.1: Kernel density graph of goitre prevalence of school aged children per district prior to any bans on non-iodised salt.



A kernel density graph of TGR per district is shown in Figure B.1. One notes the high density of TGR in the range of 20-40%, indicating a high TGR in the sampled districts.

B.2.3 Descriptive statistics on iodised salt consumption

Summary statistics of household and village characteristics of non-iodised salt, salt with some iodine and adequately iodised salt are presented in Tables B.1, B.2 and B.3. We can observe a wealth gradient across all policy states in the consumption of adequately iodised salt. However some studies point to that wealth appear to influence the choice for a certain salt type in India rather than a conscious decision to buy iodised salt per (Wheeler & van der Haar 2004).Unfortunately, only NFHS II includes information on the consumption of refined salt and not later surveys. However, we note that an important determinant of having adequately iodised salt at home in during the first ban is the purchasing of refined salt in comparison to coarse un-refined salt. What is further interesting is that household who consume salt with some iodine, are households who are worse off in terms of wealth, knowledge about aids (proxy for overall health knowledge), purifying water, haemoglobin levels and of bigger household size, during the bans. The unexpected effect of distance to nearest town or distance to nearest railway station might be due to the fact that there is more competition in salt suppliers in larger urban markets.

B.3 Descriptive statistics

Table B.1: Descriptive statistics during the first ban using the 1998-2000 NFHS II

	Non-iodised	Inadequately Iodised	Adequately Iodised
Refined Salt	0.15	0.24	0.68
Quintiles of Wealth Index	2.44	2.36	3.06
Max. years of education in household	6.79	6.62	8.16
Number of children aged 5 and under in household	0.94	0.98	0.90
Has ever heard of AIDS	0.29	0.25	0.38
Female household head	0.11	0.09	0.10
Purify Water	0.26	0.22	0.35
Household Size	6.40	6.53	6.45
Kutcha	0.39	0.44	0.36
Semi-pucca house	0.42	0.40	0.40
Pucca	0.19	0.16	0.24
Owns Tv	0.17	0.17	0.33
Owns Radio	0.31	0.32	0.41
Owns Telephone	0.02	0.01	0.05
Currently Pregnant	0.08	0.08	0.08
Haemoglobin Level (g/dl - 1 decimal)	115.76	114.96	117.29
Eats meat/fish	0.65	0.74	0.66
Hindu	0.85	0.80	0.75
Muslim	0.08	0.12	0.10
Christian	0.04	0.06	0.07
Scheduled Caste	0.29	0.30	0.27
Scheduled Tribe	0.20	0.22	0.28
Other Backward Caste	0.51	0.48	0.44
Distance to nearest town (km)	15.04	15.82	15.03
Distance to nearest railway station (km)	28.11	31.81	34.47
Distance to transport service (km)	14.92	13.67	13.66
Observations	20951	17277	34576

Notes: This table uses the 1998-2000 NFHS II data to report the means of household characteristics by differences in iodised salt consumption; non-iodised salt, inadequately iodised salt ($\leq$15 μg iodine/g salt) and adequately iodised salt ($\geq$15 μg iodine/g salt).

Table B.2: Descriptive statistics during the absence of a ban using the 2002-2004 DLHS II

	Non-iodised	Inadequately Iodised	Adequately Iodised
Tertiles of Wealth Index	1.38	1.42	1.79
Max. years of education in household	8.40	8.68	9.85
Has ever heard of AIDS	0.35	0.37	0.59
Female household head	0.09	0.08	0.08
Household Size	6.17	6.24	6.05
Kutcha	0.46	0.49	0.38
Semi-pucca house	0.39	0.36	0.36
Pucca	0.25	0.24	0.41
Owns TV	0.22	0.25	0.46
Owns Radio	0.25	0.32	0.41
Owns Telephone	0.05	0.07	0.18
Hindu	0.87	0.77	0.70
Muslim	0.08	0.12	0.09
Christian	0.03	0.07	0.10
Scheduled Caste	0.21	0.19	0.15
Scheduled Tribe	0.16	0.19	0.22
Other Backward Caste	0.43	0.38	0.31
Distance to nearest town (km)	15.34	16.90	18.42
Distance to nearest railway station (km)	41.70	65.26	71.26
Observations	174869	128229	136708

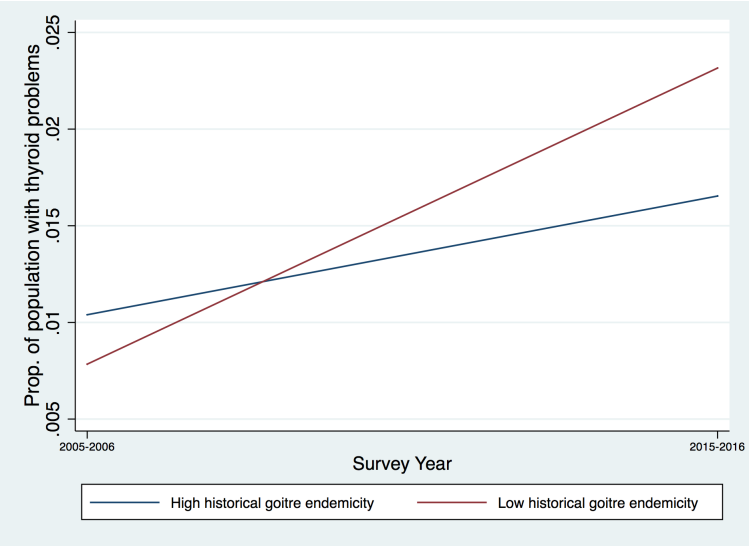
Notes: This table uses the 2002-2004 DLHS II data to report the means of household characteristics by differences in iodised salt consumption; non-iodised salt, inadequately iodised salt ($\leq$15 μg iodine/g salt) and adequately iodised salt ($\geq$15 μg iodine/g salt).

Table B.3: Descriptive statistics during the absence of a ban using the 2005-2006 NFHS III

	Non-iodised	Inadequately Iodised	Adequately Iodised
Quintiles of Wealth index	2.41	2.39	3.18
Max. years of education in household	7.26	7.26	9.06
Number of children aged 5 and under in household	0.69	0.77	0.65
Has ever heard of AIDS	0.48	0.47	0.68
Female household head	0.13	0.14	0.15
Purify Water	0.30	0.27	0.45
Household Size	5.70	5.94	5.70
Kutcha	0.18	0.21	0.13
Semi-pucca house	0.54	0.55	0.49
Pucca	0.28	0.24	0.38
Owns TV	0.31	0.29	0.49
Owns Radio	0.26	0.28	0.39
Owns Telephone	0.07	0.06	0.18
Wealth Index	2.41	2.39	3.18
Currently Pregnant	0.06	0.06	0.05
Hemoglobin Level (g/dl - 1 decimal)	115.00	114.64	116.85
Eats meat/fish	0.54	0.64	0.65
Hindu	0.86	0.78	0.67
Muslim	0.08	0.11	0.12
Christian	0.03	0.07	0.14
Scheduled Caste	0.20	0.20	0.15
Scheduled Tribe	0.15	0.17	0.21
Other Backward Caste	0.42	0.39	0.30
Observations	19006	19837	38823

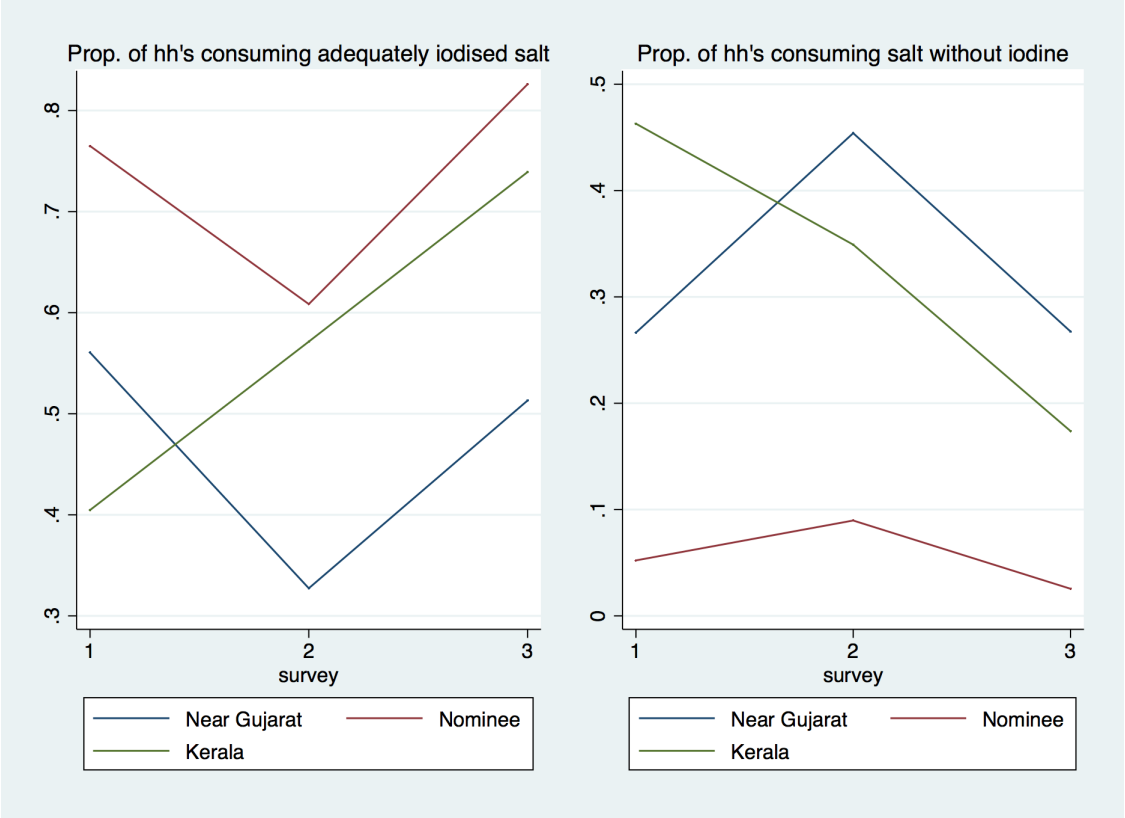
Notes: This table uses the 2005-2006 NFHS III data to report the means of household characteristics by differences in iodised salt consumption; non-iodised salt, inadequately iodised salt ($\leq$15 μg iodine/g salt) and adequately iodised salt ($\geq$15 μg iodine/g salt).

Figure B.2: Trends in thyroid related illnesses for states with high and low historical goitre endemicity



The figure depicts the trends in the proportion of rural household who have thyroid related health problems in states the number of areas with goitre endemicity being at or above the 75th percentile, compared to those at or below the 25th percentile. Survey year 2005-2006 denotes the NFHS III and Survey 2015-2016 denotes the NFHS IV.

Figure B.3: Consumption of iodised salt over time

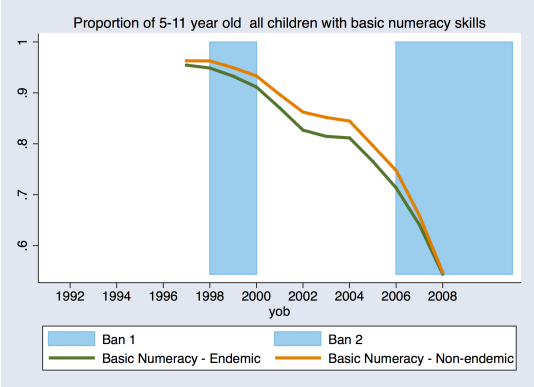


The figure depicts the trends in the proportion of rural household who consume iodised and non-iodised salt in; states near Gujarat, states with a nominee system and predominantly rail transportation of salt and in Kerala. Survey 1 denotes the NFHS II which covers the years of 1998-2000. Survey 2 denotes the DLHS II of 2002-2004 and Survey 3 represents the NFHS III for years 2005-2006.

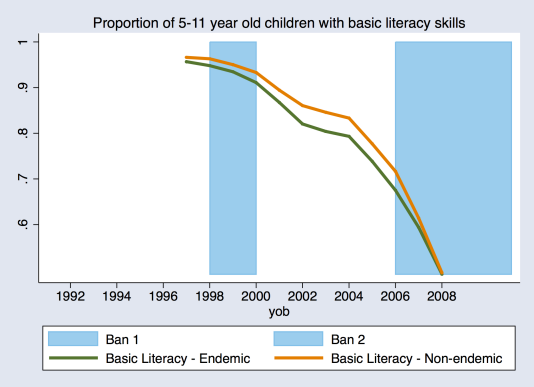
B.3.1 Regression appendix

Figure B.4: Test scores for pooled sample of 5-10 year olds.

(a) Prop of children with basic numeracy skills

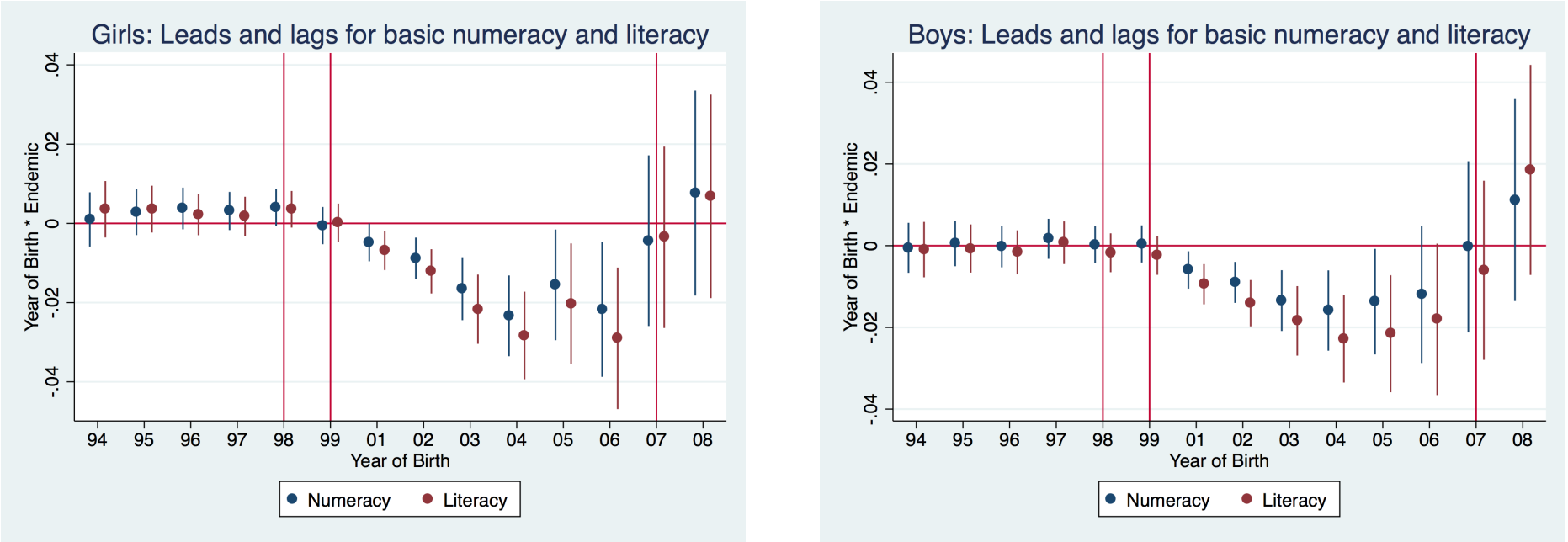


(b) Prop of children with basic literacy skills



The figure depicts the proportion of children aged 5-10 in the ASER data who have mastered basic numeracy and literacy scores by birth year. The trends are given for children residing in pre-fortification goitre endemic and non-endemic districts. The blue areas represent the duration of a ban on non-iodised salt by birth year.

Figure B.5: Leads and lags of birth year * endemicity



These graphs plot the coefficients on birth year interacted with endemicity status of one's district of birth from Equation 2.1 omitting the inclusion of trends, using data from ASER for boys and girls aged 5-16. The reference category birth year is 2000. The y-axis shows the magnitude of the coefficients, the x-axis represents the birth year and the lines through the plotted coefficients are confidence intervals.

Table B.4: Effect on grade progression in primary school

	Pooled		Girls		Boys	
	(1)	(2)	(3)	(4)	(5)	(6)
Iodised * Endemic	0.042**	0.008	0.041*	0.010	0.042**	0.006
	(0.016)	(0.017)	(0.021)	(0.022)	(0.020)	(0.021)
Mother's education	0.005***	0.006***	0.006***	0.007***	0.004***	0.005***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.043***	0.044***	0.042***	0.043***	0.043***	0.045***
	(0.005)	(0.005)	(0.006)	(0.006)	(0.005)	(0.006)
Pucca house	0.043***	0.040***	0.048***	0.048***	0.037***	0.033***
	(0.005)	(0.006)	(0.006)	(0.007)	(0.006)	(0.006)
Household size	-0.003***	-0.003***	-0.002***	-0.002***	-0.004***	-0.004***
	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	8.011***	11.720***				
	(2.034)	(2.222)				
Gvt primary school in vlg		0.042***		0.046***		0.038***
		(0.011)		(0.012)		(0.012)
Vlg has anganwadi		-0.010		-0.010		-0.010
		(0.009)		(0.010)		(0.010)
Vlg is connected to a pucca road		0.006		0.010*		0.002
		(0.005)		(0.006)		(0.006)
Vlg has ration shop		0.008*		0.011*		0.005
		(0.005)		(0.006)		(0.005)
Observations	881444	762618	412454	357786	468990	404832
R^2	0.934	0.935	0.937	0.938	0.931	0.932

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variable is the primary school class attended by a child aged 5-10 from all states except Kerala, using the ASER data. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.5: Effect on overall test scores

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
overall score in:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.007	0.007	0.050*	0.038	0.028	0.017	0.094***	0.075**	-0.010	-0.003	0.010	0.006
	(0.022)	(0.021)	(0.026)	(0.025)	(0.027)	(0.026)	(0.033)	(0.032)	(0.027)	(0.026)	(0.029)	(0.030)
Mother's education	0.045***	0.046***	0.050***	0.051***	0.046***	0.047***	0.052***	0.053***	0.044***	0.044***	0.048***	0.049***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.114***	0.114***	0.142***	0.142***	0.109***	0.108***	0.135***	0.136***	0.118***	0.119***	0.148***	0.148***
	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.008)	(0.006)	(0.006)	(0.006)	(0.007)
Housing material: Pucca	0.253***	0.256***	0.284***	0.285***	0.251***	0.253***	0.289***	0.291***	0.256***	0.258***	0.280***	0.279***
	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.008)	(0.008)	(0.009)	(0.006)	(0.007)	(0.007)	(0.008)
Household size	-0.001*	-0.001*	-0.001*	-0.001	-0.001*	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	-8.687***	-12.275***	-5.671**	-6.373**								
	(2.112)	(2.396)	(2.467)	(2.812)								
Gvt primary school in vlg		0.048***		0.057***		0.053***		0.056***		0.044***		0.058***
		(0.011)		(0.013)		(0.012)		(0.015)		(0.012)		(0.014)
Vlg has anganwadi		0.006		0.009		0.007		0.008		0.005		0.010
		(0.009)		(0.010)		(0.010)		(0.012)		(0.010)		(0.011)
Vlg is connected to a pucca road		0.034***		0.037***		0.035***		0.035***		0.033***		0.039***
		(0.006)		(0.007)		(0.007)		(0.008)		(0.007)		(0.008)
Vlg has ration shop		0.042***		0.043***		0.040***		0.042***		0.045***		0.044***
		(0.005)		(0.006)		(0.006)		(0.007)		(0.006)		(0.007)
Observations	824511	692890	828556	696175	384636	324064	386531	325576	439875	368826	442025	370599
R^2	0.773	0.782	0.755	0.763	0.771	0.781	0.756	0.764	0.774	0.784	0.754	0.762

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the raw overall numeracy and literacy scores for children aged 5-10 from all states but Kerala. The score is in the range of 0-4, where 0 denotes no numeracy or literacy ability and 4 denotes that the child can master reading a paragraph or do division, respectively. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.6: Effect on enrolment and dropout

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
	Enrolled	Enrolled	Dropped out	Dropped out	Enrolled	Enrolled	Dropped out	Dropped out	Enrolled	Enrolled	Dropped out	Dropped out
Iodised* Endemic	-0.001	0.002	-0.001	-0.002*	-0.003	0.000	-0.002	-0.002	0.000	0.003	-0.001	-0.001
	(0.002)	(0.002)	(0.001)	(0.001)	(0.002)	(0.002)	(0.001)	(0.002)	(0.002)	(0.002)	(0.001)	(0.001)
Mother's education	0.001***	0.001***	-0.000***	-0.000***	0.001***	0.001***	-0.000***	-0.000***	0.001***	0.001***	-0.000***	-0.000***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.006***	0.006***	-0.002***	-0.002***	0.007***	0.006***	-0.002***	-0.002***	0.005***	0.005***	-0.002***	-0.002***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)
Pucca house	0.008***	0.008***	-0.003***	-0.003***	0.009***	0.008***	-0.003***	-0.003***	0.007***	0.007***	-0.003***	-0.003***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.000)	(0.000)
Household size	-0.000***	-0.000***	-0.000	-0.000	-0.000*	-0.000**	-0.000	-0.000*	-0.000**	-0.000***	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	-1.088***	-1.252***	0.162	0.272**								
	(0.213)	(0.226)	(0.110)	(0.125)								
Gvt primary school in vlg		0.005***		-0.001*		0.005***		-0.000		0.005***		-0.001**
		(0.001)		(0.000)		(0.001)		(0.001)		(0.001)		(0.001)
Vlg has anganwadi		0.004***		-0.001*		0.005***		-0.002**		0.003***		-0.000
		(0.001)		(0.001)		(0.001)		(0.001)		(0.001)		(0.001)
Vlg is connected to a pucca road		0.001***		-0.000		0.001**		-0.000		0.001**		-0.000
		(0.000)		(0.000)		(0.001)		(0.000)		(0.001)		(0.000)
Vlg has ration shop		0.000		-0.001***		0.000		-0.001***		-0.000		-0.000*
		(0.000)		(0.000)		(0.001)		(0.000)		(0.001)		(0.000)
Observations	1045467	852979	1045467	852979	486657	397679	486657	397679	558810	455300	558810	455300
R^2	0.988	0.989	0.014	0.015	0.987	0.987	0.015	0.017	0.989	0.990	0.013	0.014

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of ever having enrolled in primary school and ever having dropped out of school, for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses.
* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.7: Effect on private school enrolment

	Pooled		Girls		Boys	
	(1)	(2)	(3)	(4)	(5)	(6)
Iodised * Endemic	-0.010	-0.005	-0.008	-0.002	-0.012	-0.007
	(0.007)	(0.007)	(0.009)	(0.009)	(0.009)	(0.009)
Mother's education	0.024***	0.024***	0.024***	0.024***	0.024***	0.023***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.048***	0.044***	0.042***	0.038***	0.053***	0.050***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca house	0.160***	0.158***	0.151***	0.148***	0.167***	0.166***
	(0.004)	(0.004)	(0.005)	(0.005)	(0.004)	(0.004)
Household size	0.003***	0.003***	0.002***	0.002***	0.004***	0.004***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	3.178***	2.194*				
	(1.074)	(1.138)				
Gvt primary school in vlg		-0.069***		-0.069***		-0.068***
		(0.007)		(0.007)		(0.007)
Vlg has anganwadi		0.007*		0.006		0.008*
		(0.004)		(0.004)		(0.005)
Vlg is connected to a pucca road		0.033***		0.033***		0.032***
		(0.003)		(0.003)		(0.003)
Vlg has ration shop		0.038***		0.034***		0.042***
		(0.003)		(0.003)		(0.003)
Observations	829887	707469	385724	329611	444163	377858
R^2	0.468	0.476	0.440	0.446	0.489	0.498

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variable is the probability of being enrolled in a private school, compared to a public school or a madrasa (islamic school), for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.8: Effect on taking paid tuition

	Pooled		Girls		Boys	
	(1)	(2)	(3)	(4)	(5)	(6)
Iodised * Endemic	0.002	0.001	-0.005	-0.003	0.008	0.005
	(0.005)	(0.006)	(0.007)	(0.007)	(0.008)	(0.008)
Mother's education	0.011***	0.010***	0.011***	0.010***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.035***	0.033***	0.032***	0.030***	0.037***	0.036***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.079***	0.076***	0.074***	0.071***	0.083***	0.080***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Household size	-0.000	-0.000	-0.000	-0.000	0.000	0.000
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	-1.246	-0.945				
	(0.894)	(0.920)				
Gvt primary school in vlg		0.001		0.001		0.001
		(0.004)		(0.004)		(0.004)
Vlg has anganwadi		0.006*		0.008*		0.005
		(0.004)		(0.004)		(0.004)
Vlg is connected to a pucca road		0.022***		0.021***		0.022***
		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.020***		0.018***		0.022***
		(0.002)		(0.002)		(0.002)
Observations	750195	694202	348375	322578	401820	371624
R^2	0.349	0.349	0.341	0.341	0.356	0.356

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variable is the probability of taking paid tuition (tutoring outside of school), for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.9: Effect of being in early life during the ban of 1998 on basic numeracy skills.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised (First Ban) * Endemic	0.010*	0.004	0.012**	0.007	0.004	-0.002	0.004	0.016**	0.016**	0.010*	0.019***	0.012**
	(0.006)	(0.005)	(0.006)	(0.005)	(0.007)	(0.006)	(0.007)	(0.007)	(0.007)	(0.006)	(0.007)	(0.006)
Mother's education	0.007***	0.006***	0.008***	0.007***	0.007***	0.007***	0.008***	0.007***	0.007***	0.006***	0.007***	0.007***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.032***	0.030***	0.033***	0.031***	0.032***	0.030***	0.032***	0.032***	0.032***	0.030***	0.033***	0.031***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.049***	0.047***	0.052***	0.050***	0.050***	0.049***	0.054***	0.047***	0.047***	0.046***	0.051***	0.048***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.793***	4.182***	1.904***	2.566***								
	(0.706)	(0.826)	(0.725)	(0.847)								
Gvt primary school in vlg		0.010***		0.013***		0.009***				0.010***		0.013***
		(0.003)		(0.003)		(0.004)				(0.003)		(0.004)
Vlg has anganwadi		0.005*		0.005*		0.006*				0.005		0.005
		(0.003)		(0.003)		(0.003)				(0.003)		(0.004)
Vlg is connected to a pucca road		0.008***		0.006***		0.009***				0.008***		0.006***
		(0.002)		(0.002)		(0.002)				(0.002)		(0.002)
Vlg has rationshop		0.009***		0.009***		0.010***				0.009***		0.009***
		(0.002)		(0.002)		(0.002)				(0.002)		(0.002)
Observations	921536	684955	926392	688812	426196	317515	428460	495340	495340	367440	497932	369521
R^2	0.895	0.905	0.888	0.897	0.892	0.902	0.886	0.898	0.898	0.908	0.890	0.899

This table reports the coefficients from Equation 2.2 but now the treatment of interest is based on children who were exposed to the initial ban of 1998-2000. Iodised thus captures cohorts who were born in 1999 - 2000. The data used for the analysis is the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.10: Effect of being in early life during either ban on basic numeracy skills.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised (Both Bans) * Endemic	0.016***	0.017***	0.019***	0.021***	0.017***	0.018***	0.020***	0.022***	0.015***	0.017***	0.019***	0.021***
	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.004)	(0.004)	(0.004)	(0.004)
Mother's education	0.009***	0.009***	0.010***	0.010***	0.009***	0.009***	0.010***	0.010***	0.009***	0.008***	0.009***	0.009***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.032***	0.031***	0.032***	0.031***	0.031***	0.030***	0.031***	0.030***	0.033***	0.032***	0.033***	0.032***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.056***	0.055***	0.059***	0.057***	0.056***	0.056***	0.059***	0.058***	0.055***	0.055***	0.058***	0.057***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000**	-0.000*	-0.001***	-0.001***	-0.000*	-0.000	-0.001***	-0.001**	-0.000**	-0.000*	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.715***	3.342***	0.676	0.518								
	(0.585)	(0.685)	(0.575)	(0.680)								
Gvt primary school in vlg		0.010***		0.014***		0.010***		0.014***		0.010***		0.015***
		(0.003)		(0.003)		(0.003)		(0.004)		(0.003)		(0.003)
Vlg has anganwadi		0.003		0.002		0.003		0.002		0.003		0.002
		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)
Vlg has pucca road		0.009***		0.008***		0.010***		0.007***		0.009***		0.008***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.009***		0.010***		0.009***		0.010***		0.009***		0.010***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Observations	1080282	834744	1085367	838797	500852	388019	503224	389896	579430	446725	582143	448901
R^2	0.871	0.875	0.863	0.865	0.868	0.872	0.861	0.863	0.874	0.878	0.865	0.867

This table reports the coefficients from Equation 2.2 but now the treatment of interest is based on children who were exposed to the initial ban of 1998-2000 and the ban implemented in 2006. Iodised thus captures cohorts who were born in 1999, 2000, 2007 and 2008. The data used for the analysis is the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.11: The effect of being in early life during the ban of 1998 on overall age standardised numeracy and literacy scores.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
standard deviation of the overall score in	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised (First Ban) * Endemic	0.012	0.016	0.022	0.031**	-0.009	-0.006	-0.003	0.013	0.031*	0.036*	0.044**	0.048**
	(0.014)	(0.015)	(0.015)	(0.015)	(0.019)	(0.020)	(0.019)	(0.020)	(0.017)	(0.019)	(0.018)	(0.019)
Mother's education	0.022***	0.021***	0.024***	0.023***	0.023***	0.022***	0.025***	0.025***	0.020***	0.019***	0.023***	0.022***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.102***	0.101***	0.101***	0.100***	0.103***	0.101***	0.101***	0.100***	0.101***	0.101***	0.102***	0.100***
	(0.005)	(0.006)	(0.005)	(0.006)	(0.006)	(0.007)	(0.006)	(0.007)	(0.006)	(0.006)	(0.006)	(0.006)
Pucca house	0.156***	0.156***	0.161***	0.159***	0.162***	0.163***	0.167***	0.167***	0.150***	0.149***	0.155***	0.152***
	(0.006)	(0.007)	(0.006)	(0.007)	(0.007)	(0.008)	(0.007)	(0.008)	(0.006)	(0.007)	(0.006)	(0.007)
Household size	-0.002***	-0.002***	-0.003***	-0.003***	-0.002***	-0.002***	-0.003***	-0.003***	-0.002***	-0.002***	-0.003***	-0.002***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	5.527**	9.661***	3.546	4.996*								
	(2.472)	(3.021)	(2.357)	(2.933)								
Gvt primary school in vlg		0.033***		0.041***		0.034***		0.043***		0.033***		0.039***
		(0.009)		(0.010)		(0.011)		(0.012)		(0.010)		(0.010)
Vlg has anganwadi		0.023**		0.022**		0.025**		0.024**		0.022**		0.021*
		(0.010)		(0.010)		(0.011)		(0.011)		(0.011)		(0.011)
Vlg has pucca road		0.024***		0.016***		0.024***		0.013*		0.024***		0.018***
		(0.006)		(0.006)		(0.007)		(0.007)		(0.007)		(0.007)
Vlg has ration shop		0.027***		0.026***		0.029***		0.029***		0.025***		0.024***
		(0.006)		(0.006)		(0.007)		(0.007)		(0.006)		(0.006)
Observations	921536	684955	926392	688812	426196	317515	428460	319291	495340	367440	497932	369521
R^2	0.070	0.069	0.083	0.082	0.078	0.077	0.092	0.093	0.064	0.062	0.075	0.074

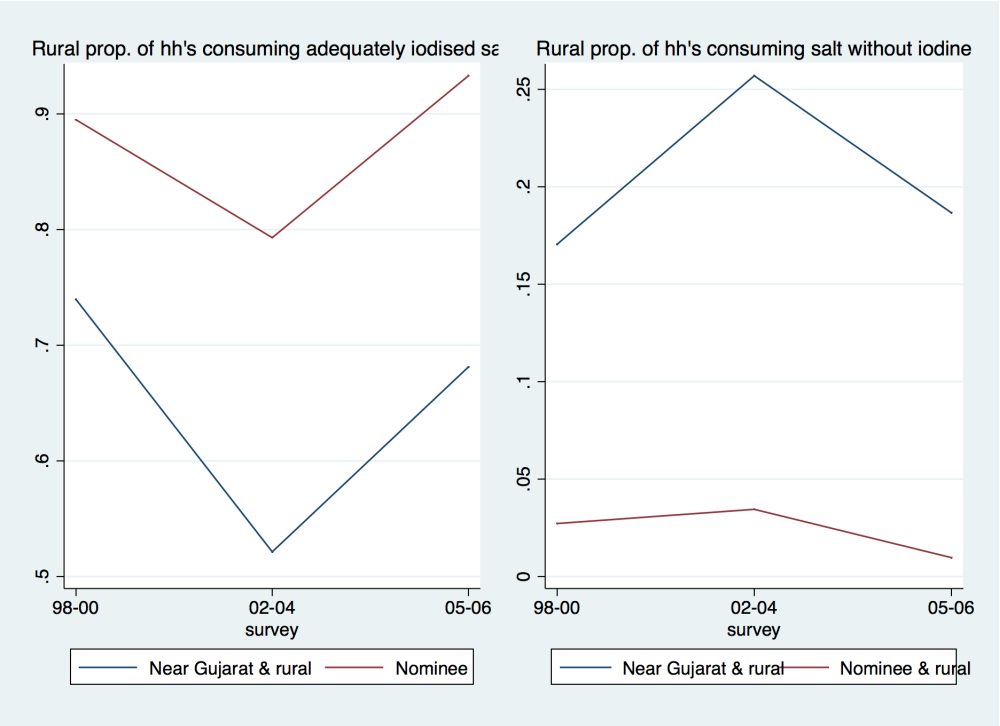
This table reports the coefficients from Equation 2.2 but now the treatment of interest is based on children who were exposed to the initial ban of 1998-2000. Iodised thus captures cohorts who were born in 1999-2000. The data used for the analysis is the ASER data merged with historical information on district level goitre endemicity. The outcome variables are age-standardised overall numeracy and literacy scores for children aged 5-10 from all states but Kerala. The raw score is in the range of 0-4, where 0 denotes no numeracy or literacy ability and 4 denotes that the child can master reading a paragraph or do division, respectively. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.12: Effect of being in early life during either ban on age standardised overall test scores.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
standard deviation of the overall score in	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised (Both Bans) * Endemic	0.032***	0.041***	0.047***	0.058***	0.035***	0.043***	0.050***	0.063***	0.029***	0.039***	0.043***	0.054***
	(0.008)	(0.009)	(0.009)	(0.010)	(0.010)	(0.011)	(0.010)	(0.012)	(0.009)	(0.010)	(0.009)	(0.010)
Mother's education	0.025***	0.025***	0.027***	0.027***	0.026***	0.026***	0.028***	0.029***	0.024***	0.024***	0.026***	0.026***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.099***	0.098***	0.098***	0.096***	0.099***	0.097***	0.096***	0.094***	0.100***	0.099***	0.100***	0.097***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)	(0.005)	(0.006)	(0.005)	(0.006)
Pucca house	0.165***	0.166***	0.169***	0.168***	0.169***	0.171***	0.173***	0.173***	0.161***	0.162***	0.165***	0.164***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.008)	(0.007)	(0.008)	(0.006)	(0.007)	(0.006)	(0.007)
Household size	-0.002***	-0.001**	-0.002***	-0.002***	-0.002**	-0.001*	-0.002***	-0.002***	-0.002**	-0.002**	-0.002***	-0.002**
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	2.647	3.639*	-1.386	-2.572								
	(1.607)	(1.881)	(1.490)	(1.795)								
Gvt primary school in vlg		0.033***		0.042***		0.033***		0.042***		0.033***		0.042***
		(0.009)		(0.009)		(0.010)		(0.011)		(0.010)		(0.010)
Vlg has anganwadi		0.016*		0.013		0.017*		0.013		0.016		0.014
		(0.008)		(0.008)		(0.010)		(0.010)		(0.009)		(0.010)
Vlg has pucca road		0.026***		0.018***		0.025***		0.015**		0.026***		0.020***
		(0.006)		(0.006)		(0.006)		(0.006)		(0.006)		(0.006)
Vlg has ration shop		0.026***		0.026***		0.027***		0.028***		0.025***		0.025***
		(0.005)		(0.005)		(0.006)		(0.006)		(0.005)		(0.005)
Observations	1080282	834744	1085367	838797	500852	388019	503224	389896	579430	446725	582143	448901
R^2	0.081	0.082	0.096	0.099	0.090	0.091	0.106	0.109	0.074	0.075	0.089	0.091

This table reports the coefficients from Equation 2.2 but now the treatment of interest is based on children who were exposed to the initial ban of 1998-2000 and the ban implemented in 2006. Iodised thus captures cohorts who were born in 1999, 2000, 2007 and 2008. The data used for the analysis is the ASER data merged with historical information on district level goitre endemicity. The outcome variables are age-standardised overall numeracy and literacy scores for children aged 5-10 from all states but Kerala. The raw score is in the range of 0-4, where 0 denotes no numeracy or literacy ability and 4 denotes that the child can master reading a paragraph or do division, respectively. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Figure B.6: Nationwide consumption of adequately iodised salt and non-iodised salt over time: Heterogeneous effects



The figures depict the trends in the proportion of rural household who consume iodised and non-iodised salt in; Gujarat and states near Gujarat with predominantly road transportation of salt and in north eastern states with predominantly rail transportation of salt. Survey 98-00 denotes the NFHS II which covers the years of 1998-2000. Survey 02-04 denotes the DLHS II of 2002-2004 and Survey 05-06 represents the NFHS III for years 2005-2006.

Table B.13: Effect on basic skills for children in the north eastern states and West Bengal

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	-0.034	-0.022	-0.038**	-0.025	-0.033	-0.020	-0.049**	-0.038*	-0.036	-0.025	-0.029	-0.013
	(0.027)	(0.033)	(0.018)	(0.017)	(0.034)	(0.041)	(0.020)	(0.020)	(0.028)	(0.033)	(0.023)	(0.024)
Mother's education	0.009***	0.008***	0.010***	0.009***	0.009***	0.008***	0.010***	0.010***	0.009***	0.008***	0.010***	0.009***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.022***	0.020***	0.024***	0.022***	0.019***	0.016***	0.021***	0.018***	0.025***	0.023***	0.027***	0.025***
	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.004)	(0.003)	(0.004)	(0.004)
Pucca house	0.044***	0.040***	0.050***	0.045***	0.036***	0.032***	0.045***	0.041***	0.053***	0.048***	0.054***	0.049***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)	(0.005)	(0.005)	(0.005)	(0.005)
Household size	-0.001**	-0.001**	-0.002***	-0.002***	-0.001*	-0.001*	-0.001**	-0.001**	-0.001**	-0.001	-0.002***	-0.002***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	-1.881	-1.855	-0.394	-1.046								
	(2.170)	(2.193)	(2.149)	(2.192)								
Gvt primary school in vlg		0.004		0.012		0.003		0.005		0.006		0.018*
		(0.008)		(0.009)		(0.009)		(0.010)		(0.009)		(0.010)
Vlg has anganwadi		0.004		-0.000		0.004		0.002		0.003		-0.002
		(0.006)		(0.006)		(0.007)		(0.007)		(0.006)		(0.006)
Vlg is connected to a pucca road		0.013***		0.013***		0.015***		0.014***		0.011**		0.011**
		(0.004)		(0.005)		(0.005)		(0.005)		(0.004)		(0.005)
Vlg has ration shop		0.015***		0.015***		0.014***		0.016***		0.015***		0.014***
		(0.004)		(0.004)		(0.005)		(0.005)		(0.005)		(0.005)
Observations	121033	105045	121919	105786	57866	50158	58282	50516	63167	54887	63637	55270
R^2	0.900	0.906	0.888	0.895	0.899	0.906	0.889	0.895	0.900	0.907	0.888	0.894

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on district level goitre endemicity. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 in Sikkim, Mizoram, Meghalaya, Nagaland, Tripura, Arunachal Pradesh, Manipur, Assam and West Bengal. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.14: Effect on basic skills using the SD of goitre points per district.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * SD goitre points per district	0.011***	0.011***	0.014***	0.015***	0.015***	0.016***	0.021***	0.022***	0.007	0.007	0.008*	0.009**
	(0.004)	(0.004)	(0.004)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.004)	(0.005)	(0.005)
Mother's education	0.010***	0.010***	0.011***	0.011***	0.011***	0.010***	0.012***	0.011***	0.010***	0.010***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.036***	0.034***	0.037***	0.035***	0.036***	0.033***	0.036***	0.034***	0.037***	0.035***	0.038***	0.036***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca house	0.064***	0.062***	0.068***	0.064***	0.065***	0.062***	0.068***	0.065***	0.063***	0.061***	0.067***	0.063***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000*	-0.000*	-0.001***	-0.001**	-0.000	-0.000	-0.001**	-0.000*	-0.000	-0.000	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.916***	2.936***	-0.262	-0.716								
	(1.000)	(1.066)	(1.000)	(1.053)								
Gvt primary school in vlg		0.012***		0.018***		0.010**		0.016***		0.013***		0.020***
		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg has anganwadi		0.002		0.001		0.003		0.001		0.001		0.001
		(0.003)		(0.003)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg is connected to a pucca road		0.010***		0.008***		0.011***		0.008***		0.010***		0.008***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.011***		0.011***		0.011***		0.010***		0.011***		0.011***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Observations	809189	680318	813090	683500	377677	318356	379504	319824	431512	361962	433586	363676
R^2	0.842	0.854	0.831	0.841	0.840	0.851	0.830	0.840	0.845	0.857	0.832	0.842

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on the standard deviation of pre-fortification goitre endemic areas per district. The outcome variables are the probability of knowing some numeracy (at least being able to recognise simple numbers) and literacy (at least being able to recognise letters) for children aged 5-10 for all states but Kerala. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.15: Effect on age standardised overall test scores using SD of goitre points per district.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
age standardised overall test scores in:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * SD goitre points per district	0.018*	0.017*	0.030***	0.029***	0.033***	0.033***	0.050***	0.049***	0.005	0.002	0.011	0.011
	(0.009)	(0.009)	(0.010)	(0.010)	(0.012)	(0.012)	(0.013)	(0.013)	(0.011)	(0.011)	(0.010)	(0.010)
Mother's education	0.027***	0.027***	0.030***	0.030***	0.028***	0.028***	0.031***	0.031***	0.026***	0.026***	0.029***	0.029***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.104***	0.103***	0.105***	0.103***	0.106***	0.104***	0.105***	0.104***	0.103***	0.102***	0.106***	0.103***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.007)	(0.006)	(0.007)	(0.005)	(0.006)	(0.006)	(0.006)
Pucca house	0.178***	0.177***	0.184***	0.180***	0.185***	0.183***	0.189***	0.186***	0.173***	0.172***	0.180***	0.175***
	(0.006)	(0.007)	(0.006)	(0.007)	(0.007)	(0.008)	(0.008)	(0.008)	(0.006)	(0.007)	(0.006)	(0.007)
Household size	-0.001**	-0.001**	-0.002***	-0.002***	-0.001*	-0.001	-0.002**	-0.002**	-0.001*	-0.001*	-0.002***	-0.002**
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	2.004	0.673	-4.782**	-7.004***								
	(2.406)	(2.668)	(2.385)	(2.623)								
Primary gvt school in vlg		0.036***		0.049***		0.032***		0.045***		0.039***		0.054***
		(0.009)		(0.010)		(0.011)		(0.012)		(0.011)		(0.011)
Vlg has anganwadi		0.012		0.009		0.016		0.009		0.008		0.008
		(0.008)		(0.009)		(0.010)		(0.010)		(0.010)		(0.010)
Vlg is connected to a pucca road		0.028***		0.020***		0.029***		0.018**		0.028***		0.021***
		(0.006)		(0.006)		(0.007)		(0.007)		(0.007)		(0.007)
Vlg has rationshop		0.027***		0.026***		0.028***		0.027***		0.026***		0.025***
		(0.005)		(0.005)		(0.006)		(0.007)		(0.006)		(0.006)
Observations	809189	680318	813090	683500	377677	318356	379504	319824	431512	361962	433586	363676
R^2	0.090	0.089	0.107	0.108	0.099	0.099	0.117	0.119	0.083	0.082	0.099	0.099

Notes: This table reports the coefficients from Equation 2.2 using the ASER data merged with historical information on the standard deviation of pre-fortification goitre endemic areas per district. The outcome variables are age-standardised overall numeracy and literacy scores for children aged 5-10 from all states but Kerala. The raw score is in the range of 0-4, where 0 denotes no numeracy or literacy ability and 4 denotes that the child can master reading a paragraph or do division, respectively. The subsample of analysis and the outcome variable is reported at the top of the table. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample. Robust standard errors clustered on district are presented in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.16: Relationship between historical goitre per state on current thyroid prevalence.

	(1) Current prevalence of thyroid problems	(2) Logarithm of current prevalence of thyroid problems	(3) Current prevalence of thyroid problems
Historical goitrous areas	0.021**	1.424***	
/10 000 population/state	(0.009)	(0.479)	
Logarithm of historical goitrous areas			0.003***
/10 000 population/state			(0.001)
Constant	0.012***	-4.805***	0.024***
	(0.003)	(0.175)	(0.005)
Observations	28	28	27
R^2	0.123	0.148	0.168

Notes: This table reports the coefficients from three separate OLS models estimating the prevalence of individuals with thyroid related problems per state on the number of historical goitre areas per state and population. Data from the 2015-2015 NFHS IV is used on state level averages individuals 35 years and older reporting having thyroid related problems, such as goitre. The data is merged with the number of goitre endemic areas per states as of 2011 is geocoded from the map by McCarrison (1915) and with the population per state as of census 2011. Robust standard errors are clustered on state. Standard errors are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.17: Relationship between district level goitre rate and historical goitre endemicity

	(1)	(2)	(3)
	Historical Endemicity	Historical Endemicity	Historical Endemicity
Goitre rate	0.006***		
	(0.002)		
Goitre rate $\geq$ 10%		0.251***	
		(0.085)	
Goitre rate $\geq$ 20%			0.062
			(0.057)
Constant	0.476***	0.333***	0.516***
	(0.057)	(0.082)	(0.052)
Observations	262	582	582
R^2	0.037	0.015	0.002

Notes: This table reports the coefficients from three separate linear probability models estimating the likelihood that a district has been identified as historically goitre (containing at least one goitre endemic area from the map by McCarrison (1915)) on later district level goitre rate among children. This data stems from district level averages of the goitre rate among primary school aged children measured during 1940-2010 by the IDD and Nutrition Cell, Directorate of Health Services, Ministry of Health and Family Welfare India. Robust standard errors are clustered on district. Rural district as of the 2001 Census and which are included in the ASER survey are included. Standard errors are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.18: Relationship between goitre prevalence per district and timing of goitre survey

	(1)	(2)	(3)
	Goitre survey year	Goitre survey year	District included in goitre survey
Historical Endemicity	-13.087***		0.103***
	(1.261)		(0.038)
Goitre rate per district		-0.141***	
		(0.045)	
Constant	1986.809***	1982.068***	0.342***
	(1.011)	(1.361)	(0.029)
Observations	263	263	666
R^2	0.292	0.037	0.011

Notes: This table reports the coefficients from three separate linear probability models estimating the effect of various measures of goitre per district on the year of goitre rate survey and the probability of a district having been included in the goitre survey. The historical endemicity measure is a binary measure for whether a district as of census 2001 contains at least one goitre endemic area from the map by McCarrison (1915). Goitre rate per district stems from district level data on the goitre rate among primary school aged children measured during 1940-2010 by the IDD and Nutrition Cell, Directorate of Health Services, Ministry of Health and Family Welfare India. Robust standard errors are clustered on district. Rural district as of the 2001 Census and which are included in the ASER survey are included. Goitre survey year denotes the year the survey was carried out per district. District included in goitre survey denotes whether the district has been sampled to measure the rate of goitre among children. Standard errors are shown in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.19: Effect on basic skills using district level total goitre rate

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Median TGR	0.041***	0.019	0.040***	0.022	0.037**	0.019	0.039**	0.025	0.045***	0.020	0.042**	0.019
	(0.013)	(0.013)	(0.015)	(0.015)	(0.015)	(0.015)	(0.017)	(0.017)	(0.016)	(0.016)	(0.017)	(0.017)
Mother's Education	0.010***	0.009***	0.010***	0.010***	0.010***	0.009***	0.011***	0.011***	0.009***	0.009***	0.010***	0.010***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.033***	0.031***	0.032***	0.030***	0.034***	0.033***	0.032***	0.032***	0.032***	0.029***	0.031***	0.029***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca	0.056***	0.053***	0.059***	0.056***	0.058***	0.055***	0.060***	0.057***	0.054***	0.052***	0.058***	0.054***
	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)
HH size	-0.001***	-0.001***	-0.002***	-0.002***	-0.001***	-0.001***	-0.002***	-0.001***	-0.001***	-0.001***	-0.002***	-0.002***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.558	3.657**	-1.605	-1.203								
	(1.612)	(1.741)	(1.571)	(1.712)								
Gvt Primary School in Vlg		0.012**		0.020***		0.008		0.013**		0.017***		0.026***
		(0.005)		(0.005)		(0.006)		(0.007)		(0.006)		(0.006)
Vlg has Anganwadi		0.002		-0.001		0.001		-0.002		0.003		0.000
		(0.004)		(0.004)		(0.005)		(0.005)		(0.005)		(0.005)
Vlg is connected to a pucca road		0.010***		0.008**		0.011***		0.009***		0.008**		0.007*
		(0.003)		(0.003)		(0.003)		(0.003)		(0.004)		(0.004)
Vlg has ration shop		0.010***		0.009***		0.009***		0.007**		0.012***		0.011***
		(0.003)		(0.003)		(0.003)		(0.004)		(0.003)		(0.003)
Observations	349291	292773	350971	294168	164796	138409	165592	139068	184495	154364	185379	155100
R^2	0.860	0.870	0.849	0.858	0.859	0.869	0.849	0.858	0.860	0.871	0.848	0.858

The outcome variables are the probability of knowing some numeracy and literacy for children aged 5-10 using the ASER data. Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.20: Effect on age standardised overall numeracy and literacy scores using district level total goitre rate

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the overall score in:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Median TGR	0.095***	0.052	0.085**	0.052	0.091**	0.057	0.087**	0.065	0.101***	0.050	0.084**	0.041
	(0.032)	(0.032)	(0.035)	(0.036)	(0.038)	(0.040)	(0.043)	(0.044)	(0.037)	(0.039)	(0.039)	(0.040)
Mother's Educ	0.025***	0.024***	0.028***	0.027***	0.026***	0.025***	0.029***	0.029***	0.024***	0.024***	0.027***	0.026***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.093***	0.091***	0.089***	0.088***	0.100***	0.099***	0.092***	0.094***	0.088***	0.085***	0.086***	0.083***
	(0.007)	(0.008)	(0.007)	(0.008)	(0.009)	(0.010)	(0.009)	(0.010)	(0.008)	(0.008)	(0.008)	(0.009)
Pucca house	0.153***	0.151***	0.158***	0.154***	0.160***	0.158***	0.164***	0.161***	0.146***	0.145***	0.153***	0.148***
	(0.010)	(0.010)	(0.010)	(0.010)	(0.012)	(0.013)	(0.012)	(0.013)	(0.010)	(0.011)	(0.010)	(0.010)
HH size	-0.003***	-0.004***	-0.005***	-0.004***	-0.004***	-0.004***	-0.005***	-0.004***	-0.003***	-0.003***	-0.004***	-0.004***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	3.469	5.179	-6.140	-6.024								
	(3.894)	(4.357)	(3.728)	(4.210)								
Gvt Primary School in Vlg		0.030**		0.043***		0.017		0.026		0.043***		0.059***
		(0.013)		(0.014)		(0.016)		(0.016)		(0.015)		(0.016)
Vlg has Anganwadi		0.013		0.005		0.011		0.003		0.015		0.007
		(0.012)		(0.012)		(0.013)		(0.013)		(0.015)		(0.015)
Vlg is connected to a pucca road		0.025***		0.017*		0.027***		0.015		0.023**		0.017
		(0.009)		(0.009)		(0.009)		(0.010)		(0.010)		(0.011)
Vlg has ration shop		0.025***		0.022**		0.024**		0.020*		0.026***		0.023***
		(0.008)		(0.008)		(0.009)		(0.011)		(0.008)		(0.008)
Observations	349291	292773	350971	294168	164796	138409	165592	139068	184495	154364	185379	155100
R^2	0.092	0.091	0.108	0.108	0.099	0.099	0.117	0.118	0.086	0.085	0.101	0.101

The outcome variables are the effect on overall numeracy and literacy scores for children aged 5-10 using the ASER data. Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.21: Ecological determinants of pre-fortification goitre endemicity

	(1)	(2)	(3)	(4)
	Summary Statistics	Goitre Endemic District (McCarrisson)	Above Median TGR	Above Median TGR
Maximum Elevation (km)	0.811	0.047***	0.083***	0.074***
	(1.155)	(0.011)	(0.023)	(0.024)
1/Groundwater Salinity	0.752	0.555***	0.396***	0.263*
	(0.071)	(0.075)	(0.122)	(0.133)
Year TGR surveyed				-0.008***
				(0.003)
Constant		0.116**	0.120	16.207***
		(0.058)	(0.096)	(6.168)
Observations	585	579	253	253
R^2		0.119	0.105	0.134
F-stat		61.31	18.19	9.33
Prob $\geq$ F		0.000	0.000	0.000

The first column shows the summary statistics for the variables denoted on the left. The respective means are displayed with corresponding standard errors in parenthesis below. Columns 2-4 show the first stage results from Equation 2.3. The respective pre-fortification goitre indicator variables are regressed on current district level maximum elevation and 1/groundwater salinity. Robust standard errors clustered on district are in parentheses. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.22: IV Results: Effect on basic skills

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is basic :	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.080***	0.076***	0.122***	0.116***	0.102***	0.105***	0.139***	0.144***	0.061*	0.052*	0.107***	0.093***
	(0.029)	(0.027)	(0.031)	(0.030)	(0.033)	(0.032)	(0.035)	(0.035)	(0.033)	(0.031)	(0.035)	(0.033)
Mother's Education	0.010***	0.010***	0.011***	0.011***	0.011***	0.010***	0.012***	0.011***	0.010***	0.010***	0.011***	0.011***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Kutcha House	0.036***	0.034***	0.037***	0.035***	0.036***	0.033***	0.036***	0.034***	0.037***	0.035***	0.038***	0.035***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Pucca House	0.064***	0.062***	0.068***	0.064***	0.065***	0.062***	0.068***	0.065***	0.063***	0.061***	0.067***	0.063***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.002)
Household size	-0.000*	-0.000*	-0.001***	-0.001**	-0.000	-0.000	-0.001**	-0.000*	-0.000	-0.000*	-0.001***	-0.001**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.825***	2.785***	-0.308	-0.815								
	(1.002)	(1.065)	(1.003)	(1.056)								
Gvt Primary School in Vlg		0.012***		0.018***		0.011**		0.016***		0.013***		0.020***
		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)		(0.004)
Vlg has Anganwadi		0.003		0.001		0.004		0.002		0.002		0.001
		(0.003)		(0.003)		(0.004)		(0.004)		(0.003)		(0.004)
Vlg has pucca road		0.011***		0.009***		0.011***		0.009***		0.010***		0.008***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Vlg has ration shop		0.011***		0.010***		0.011***		0.010***		0.011***		0.010***
		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)		(0.002)
Overidentification test	11.605	0.010	5.003	0.402	4.841	0.790	2.985	1.020	15.074	1.137	5.179	0.029
χ^2 p-value	0.0007	0.9207	0.0253	0.5258	0.0278	0.3741	0.0841	0.3126	0.0001	0.2862	0.0229	0.8652
Observations	805829	677364	809727	680542	376108	316986	377937	318455	429721	360378	431790	362087
R^2	0.021	0.022	0.024	0.024	0.022	0.023	0.025	0.026	0.021	0.021	0.023	0.024

This table reports the regression results from the TSLS estimation specified in Equation 4. Historical goitre endemicity is instrumented with inverse of a salinity score ranging 1-3 per district and the maximum elevation per district. The outcome variables are the respective probabilities of knowing basic numeracy and literacy for children aged 5-10 using the ASER data. The Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.23: IV Results: Effect on age standardised overall numeracy and literacy scores
using district level total goitre rate

	Pooled				Girls				Boys			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Dependent variable is the overall score in:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic	0.136**	0.068	0.168**	0.127**	0.198**	0.174**	0.257***	0.267***	0.086	-0.022	0.093	0.008
	(0.068)	(0.061)	(0.068)	(0.063)	(0.079)	(0.076)	(0.086)	(0.086)	(0.081)	(0.076)	(0.077)	(0.075)
Mother's Education	0.043***	0.044***	0.043***	0.044***	0.044***	0.045***	0.045***	0.045***	0.042***	0.043***	0.041***	0.042***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.110***	0.109***	0.122***	0.120***	0.105***	0.103***	0.116***	0.114***	0.115***	0.114***	0.128***	0.125***
	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.007)	(0.005)	(0.006)	(0.006)	(0.006)
Pucca house	0.242***	0.242***	0.245***	0.242***	0.238***	0.239***	0.248***	0.247***	0.245***	0.244***	0.243***	0.238***
	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)	(0.007)	(0.007)	(0.006)	(0.007)	(0.006)	(0.007)
HH size	-0.001	-0.001*	-0.001	-0.001	-0.001	-0.001	-0.001	-0.000	-0.001	-0.001	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	-5.455**	-8.485***	-4.129*	-4.658*								
	(2.134)	(2.366)	(2.255)	(2.511)								
Gvt Primary School in Vlg		0.045***		0.051***		0.050***		0.050***		0.041***		0.052***
		(0.011)		(0.011)		(0.012)		(0.012)		(0.012)		(0.012)
Vlg has Anganwadi		0.006		0.006		0.007		0.005		0.005		0.008
		(0.009)		(0.009)		(0.009)		(0.010)		(0.010)		(0.010)
Vlg is connected to a pucca road		0.031***		0.032***		0.033***		0.031***		0.029***		0.033***
		(0.006)		(0.006)		(0.006)		(0.007)		(0.006)		(0.007)
Vlg has ration shop		0.040***		0.036***		0.038***		0.035***		0.043***		0.037***
		(0.005)		(0.005)		(0.006)		(0.006)		(0.006)		(0.006)
Observations	805829	677364	809727	680542	376108	316986	377937	318455	429721	360378	431790	362087
R^2	0.175	0.186	0.146	0.155	0.197	0.210	0.161	0.172	0.157	0.165	0.133	0.141

This table reports the regression results from the TSLS estimation specified in Equation 4. Historical goitre endemicity is instrumented with inverse of a salinity score ranging 1-3 per district and the maximum elevation per district. The outcome variables are age standardised overall scores in numeracy and literacy for children aged 5-10 using the ASER data. The Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.24: IV results: Effect on grade progression.

	Pooled		Girls		Boys	
	(1)	(2)	(3)	(4)	(5)	(6)
	Grade	Grade	Grade	Grade	Grade	Grade
Iodised * Endemic	0.203***	0.116*	0.283***	0.225***	0.137*	0.023
	(0.070)	(0.064)	(0.085)	(0.083)	(0.076)	(0.075)
Mother's Educ	0.003***	0.003***	0.004***	0.004***	0.002***	0.003***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Kutcha	0.040***	0.041***	0.040***	0.042***	0.040***	0.040***
	(0.005)	(0.005)	(0.006)	(0.006)	(0.005)	(0.006)
Pucca	0.037***	0.034***	0.041***	0.041***	0.033***	0.028***
	(0.005)	(0.005)	(0.006)	(0.007)	(0.006)	(0.006)
HH size	-0.002***	-0.002***	-0.001**	-0.002***	-0.003***	-0.003***
	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	1.936	5.484**				
	(1.933)	(2.147)				
Gvt Primary School in Vlg		0.044***		0.048***		0.041***
		(0.011)		(0.012)		(0.011)
Vlg has Anganwadi		-0.010		-0.009		-0.011
		(0.009)		(0.010)		(0.010)
Vlg is connected to a pucca road		0.001		0.005		-0.002
		(0.005)		(0.006)		(0.006)
Vlg has ration shop		0.005		0.009		0.002
		(0.005)		(0.006)		(0.005)
Observations	752552	642486	351386	300833	401166	341653
R^2	0.919	0.919	0.921	0.922	0.916	0.917

This table reports the regression results from the TSLS estimation specified in Equation 4. Historical goitre endemicity is instrumented with inverse of a salinity score ranging 1-3 per district and the maximum elevation per district. The outcome variable is current grade in primary school for children aged 5-10 using the ASER data. Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.25: TSLS results: Effect of iodine fortification on basic numeracy using TGR data

	Pooled						Girls						Boys					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
Iodised (2nd Ban) * Median TGR	0.222***	0.163***					0.232***	0.179***					0.214***	0.148***				
	(0.049)	(0.045)					(0.055)	(0.053)					(0.052)	(0.049)				
Iodised (1st Ban) * Median TGR			-0.049*	0.043*					-0.041	0.063*					-0.056*	0.026		
			(0.025)	(0.024)					(0.030)	(0.032)					(0.029)	(0.029)		
[1em] Iodised (Both Bans) * Median TGR					-0.049*	0.043*					-0.041	0.063*					-0.056*	0.026
					(0.025)	(0.024)					(0.030)	(0.032)					(0.029)	(0.029)
Mother's Education	0.010***	0.009***	0.006***	0.005***	0.006***	0.005***	0.010***	0.009***	0.006***	0.006***	0.006***	0.006***	0.009***	0.009***	0.006***	0.005***	0.006***	0.005***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.033***	0.031***	0.029***	0.029***	0.029***	0.029***	0.034***	0.033***	0.029***	0.029***	0.029***	0.029***	0.032***	0.029***	0.030***	0.029***	0.030***	0.029***
	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca	0.056***	0.053***	0.041***	0.041***	0.041***	0.041***	0.058***	0.055***	0.043***	0.042***	0.043***	0.042***	0.054***	0.051***	0.040***	0.039***	0.040***	0.039***
	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.004)	(0.003)	(0.004)
HH size	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***	-0.001***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.601	3.685**	1.140	1.342	1.140	1.342												
	(1.615)	(1.744)	(0.998)	(1.209)	(0.998)	(1.209)												
[1em] Gvt Primary School in Vlg		0.013**		0.008*		0.008*		0.008		0.004		0.004		0.017***		0.011**		0.011**
		(0.005)		(0.004)		(0.004)		(0.006)		(0.005)		(0.005)		(0.006)		(0.005)		(0.005)
Vlg has Anganwadi		0.002		0.005		0.005		0.002		0.005		0.005		0.003		0.006		0.006
		(0.004)		(0.005)		(0.005)		(0.005)		(0.005)		(0.005)		(0.005)		(0.005)		(0.005)
Vlg is connected to a pucca road		0.009***		0.007**		0.007**		0.012***		0.008**		0.008**		0.008**		0.006**		0.006**
		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.004)		(0.003)		(0.003)
Vlg has ration shop		0.010***		0.007***		0.007***		0.009***		0.008***		0.008***		0.012***		0.006**		0.006**
		(0.003)		(0.002)		(0.002)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)
Observations	349291	292773	388393	287226	388393	287226	164796	138409	181970	134714	181970	134714	184495	154364	206423	152512	206423	152512
R^2	0.859	0.870	0.909	0.917	0.909	0.917	0.859	0.869	0.907	0.916	0.907	0.916	0.860	0.871	0.910	0.919	0.910	0.919

This table reports the regression results from the TSLS estimation specified in Equation 4. The district level TGR is instrumented with inverse of a salinity score ranging 1-3 per district and the maximum elevation per district. The outcome is the probability of knowing basic numeracy for children aged 5-10 using the ASER data from a TSLS regression. Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.26: IV results: Effect on basic literacy using TGR data

	Pooled						Girls						Boys					
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)
Iodised (Second Ban) * Median TGR	0.040***	0.022					0.039**	0.025					0.042**	0.019				
	(0.015)	(0.015)					(0.017)	(0.017)					(0.017)	(0.017)				
Iodised (First Ban) * Median TGR			-0.013	0.011					-0.020*	0.003					-0.008	0.018*		
			(0.009)	(0.008)					(0.010)	(0.009)					(0.010)	(0.009)		
Iodised (Both Bans) * Median TGR					0.011*	0.018***					0.010	0.017**					0.012*	0.019***
					(0.007)	(0.007)					(0.007)	(0.007)					(0.007)	(0.007)
Mother's Education	0.010***	0.010***	0.007***	0.006***	0.009***	0.009***	0.011***	0.011***	0.007***	0.007***	0.009***	0.009***	0.010***	0.010***	0.007***	0.006***	0.009***	0.009***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Semi-pucca house	0.032***	0.030***	0.028***	0.028***	0.028***	0.028***	0.032***	0.032***	0.027***	0.029***	0.027***	0.029***	0.031***	0.029***	0.029***	0.028***	0.028***	0.027***
	(0.003)	(0.003)	(0.002)	(0.003)	(0.002)	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca	0.059***	0.056***	0.044***	0.043***	0.050***	0.049***	0.060***	0.057***	0.045***	0.045***	0.050***	0.050***	0.058***	0.054***	0.044***	0.041***	0.050***	0.048***
	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.004)	(0.003)	(0.003)
HH size	-0.002***	-0.002***	-0.002***	-0.001***	-0.002***	-0.001***	-0.002***	-0.001***	-0.002***	-0.002***	-0.002***	-0.001***	-0.002***	-0.002***	-0.001***	-0.001***	-0.001***	-0.002***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	-1.605	-1.203	0.905	0.598	-0.359	-0.696												
	(1.571)	(1.712)	(1.018)	(1.223)	(0.863)	(1.084)												
Gvt Primary School in Vlg		0.020***		0.013***		0.013***		0.013**		0.010*		0.009		0.026***		0.017***		0.016***
		(0.005)		(0.005)		(0.005)		(0.007)		(0.006)		(0.006)		(0.006)		(0.005)		(0.005)
Vlg has Anganwadi		-0.001		0.003		-0.001		-0.002		0.003		-0.001		0.000		0.003		0.000
		(0.004)		(0.004)		(0.004)		(0.005)		(0.005)		(0.004)		(0.005)		(0.005)		(0.005)
Vlg is connected to a pucca road		0.008**		0.005*		0.007**		0.009***		0.004		0.008**		0.007*		0.005*		0.007**
		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.004)		(0.003)		(0.003)
Vlg has ration shop		0.009***		0.007***		0.008***		0.007**		0.007**		0.007**		0.011***		0.007***		0.010***
		(0.003)		(0.003)		(0.003)		(0.004)		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)
Observations	350971	294168	390409	288888	460401	354291	165592	139068	182907	135492	216207	166649	185379	155100	207502	153396	244194	187642
R^2	0.849	0.858	0.902	0.910	0.877	0.878	0.849	0.858	0.902	0.909	0.877	0.878	0.848	0.858	0.903	0.911	0.877	0.879

The outcome is literacy score ranging from 0-4 for children aged 5-10 using the ASER data from a TSLS regression. The district level TGR has been instrumented with inverse of a salinity score ranging 1-3 and the maximum elevation per district. Robust standard errors clustered on district are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear district trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.27: For children in sea bordering districts: Effect on basic numeracy

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Iodised * Endemic	0.008	0.008			0.030	0.009			-0.011	0.008		
	(0.028)	(0.032)			(0.032)	(0.039)			(0.034)	(0.036)		
Iodised (Both bans) * Endemic			0.006	0.011			0.004	0.010			0.008	0.012
			(0.015)	(0.014)			(0.015)	(0.014)			(0.017)	(0.016)
Mother's Education	0.007***	0.007***	0.006***	0.007***	0.007***	0.007***	0.006***	0.006***	0.008***	0.007***	0.006***	0.007***
	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)
Semi-pucca house	0.024***	0.025***	0.021***	0.021***	0.022***	0.025***	0.019***	0.020***	0.026***	0.025***	0.023***	0.021***
	(0.005)	(0.005)	(0.004)	(0.004)	(0.006)	(0.006)	(0.005)	(0.005)	(0.006)	(0.006)	(0.005)	(0.005)
Pucca	0.042***	0.034***	0.034***	0.028***	0.041***	0.033***	0.033***	0.027***	0.042***	0.033***	0.036***	0.029***
	(0.005)	(0.006)	(0.005)	(0.005)	(0.006)	(0.007)	(0.005)	(0.006)	(0.007)	(0.007)	(0.005)	(0.006)
HH size	-0.001*	-0.001**	-0.001	-0.001	0.000	-0.000	-0.000	0.000	-0.002**	-0.002**	-0.001**	-0.001*
	(0.001)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	0.002	0.002	0.001	0.002								
	(0.003)	(0.003)	(0.002)	(0.002)								
Gvt Primary School in Vlg		0.023**		0.017**		0.031***		0.022***		0.015		0.013
		(0.009)		(0.007)		(0.009)		(0.007)		(0.011)		(0.009)
Vlg has Anganwadi		-0.018		-0.014		-0.019		-0.012		-0.017		-0.016
		(0.011)		(0.010)		(0.012)		(0.011)		(0.015)		(0.012)
Vlg is connected to a pucca road		0.012**		0.010**		0.016**		0.014**		0.009		0.006
		(0.005)		(0.004)		(0.006)		(0.005)		(0.006)		(0.005)
Vlg has ration shop		0.015***		0.014***		0.017**		0.015**		0.012**		0.013**
		(0.005)		(0.004)		(0.007)		(0.006)		(0.006)		(0.005)
Observations	61873	50938	83070	62316	29666	24469	39647	29809	32207	26469	43423	32507
R^2	0.886	0.900	0.910	0.915	0.888	0.902	0.911	0.917	0.884	0.899	0.909	0.914

Standard errors in parentheses

Robust Standard Errors Clustered on District. Year of Birth, Survey year, survey year*year of birth linear district trends.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.28: For children in sea bordering districts: Effect on basic literacy

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
Iodised * Endemic	-0.022	-0.018			0.009	0.006			-0.052*	-0.042		
	(0.024)	(0.027)			(0.030)	(0.035)			(0.028)	(0.033)		
Iodised (both bans) * Endemic			0.011	0.013			0.006	0.013			0.015	0.013
			(0.015)	(0.014)			(0.014)	(0.013)			(0.017)	(0.016)
Mother's Education	0.007***	0.007***	0.006***	0.006***	0.007***	0.007***	0.006***	0.006***	0.007***	0.007***	0.006***	0.006***
	(0.001)	(0.001)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.001)
Semi-pucca house	0.031***	0.028***	0.026***	0.023***	0.028***	0.024***	0.024***	0.019***	0.034***	0.030***	0.028***	0.025***
	(0.007)	(0.006)	(0.005)	(0.005)	(0.008)	(0.008)	(0.007)	(0.007)	(0.007)	(0.008)	(0.006)	(0.006)
Pucca	0.045***	0.033***	0.036***	0.028***	0.044***	0.031***	0.035***	0.025***	0.045***	0.035***	0.037***	0.030***
	(0.007)	(0.007)	(0.005)	(0.006)	(0.008)	(0.008)	(0.006)	(0.006)	(0.008)	(0.008)	(0.006)	(0.007)
HH size	-0.001*	-0.001**	-0.001*	-0.001**	-0.000	-0.001	-0.000	-0.000	-0.002**	-0.002**	-0.001*	-0.002*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Girl	0.010***	0.011***	0.008***	0.010***								
	(0.003)	(0.003)	(0.002)	(0.003)								
Gvt Primary School in Vlg		0.014*		0.011		0.020***		0.015**		0.007		0.007
		(0.008)		(0.007)		(0.008)		(0.006)		(0.011)		(0.009)
Vlg has Anganwadi		-0.015		-0.012		-0.020*		-0.012		-0.011		-0.012
		(0.011)		(0.009)		(0.012)		(0.011)		(0.014)		(0.012)
Vlg is connected to a pucca road		0.012**		0.010**		0.017**		0.015**		0.007		0.006
		(0.006)		(0.005)		(0.007)		(0.006)		(0.007)		(0.005)
Vlg has ration shop		0.015***		0.014***		0.021***		0.019***		0.009		0.010*
		(0.005)		(0.005)		(0.007)		(0.007)		(0.006)		(0.006)
Observations	62173	51160	83411	62567	29791	24561	39797	29921	32382	26599	43614	32646
R^2	0.877	0.890	0.903	0.907	0.881	0.895	0.907	0.912	0.873	0.886	0.900	0.904

Standard errors in parentheses

Robust Standard Errors clustered on District. Controls for year of Birth, survey year, survey year*year of birth linear district trends are included.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.29: Placebo regression: Village connected to an all weather road.

	(1)	(2)
Iodised * Endemic	0.006	0.012
	(0.024)	(0.025)
Girl	2.552	2.812
	(2.763)	(2.761)
Mother's Education	0.004***	0.004***
	(0.001)	(0.001)
House: Semi-Pucca	0.029***	0.028***
	(0.005)	(0.005)
House: Pucca	0.045***	0.044***
	(0.006)	(0.006)
Household Size	-0.001**	-0.001**
	(0.001)	(0.001)
Gvt Primary School in Vlg.		0.028**
		(0.013)
Vlg has Anganwadi		0.080***
		(0.011)
Observations	63031	63022
R^2	0.863	0.864

Standard errors clustered on district in parentheses

Controls for year of birth, survey year, survey year*year of birth and linear district trends are included.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.30: Effect on basic skills using NSS regions.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Standard Deviation Goitrous areas per NSS region	0.017***	0.016***	0.019***	0.019***	0.019***	0.018***	0.022***	0.023***	0.015**	0.014**	0.016***	0.015**
	(0.005)	(0.005)	(0.004)	(0.006)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.007)	(0.005)	(0.006)
Mother's Education	0.011***	0.011***	0.012***	0.012***	0.012***	0.011***	0.013***	0.013***	0.011***	0.010***	0.012***	0.011***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Semi-pucca house	0.036***	0.034***	0.037***	0.034***	0.036***	0.033***	0.036***	0.033***	0.037***	0.034***	0.038***	0.034***
	(0.003)	(0.002)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Pucca house	0.065***	0.062***	0.068***	0.064***	0.066***	0.063***	0.069***	0.066***	0.064***	0.061***	0.067***	0.063***
	(0.004)	(0.005)	(0.004)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.004)	(0.004)	(0.004)	(0.005)
Household Size	-0.000	-0.000	-0.001	-0.001	-0.000	-0.000	-0.001	-0.000	-0.000	-0.000	-0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.001)	(0.001)
Girl	2.654**	2.684**	-0.498	-0.993								
	(1.095)	(1.133)	(0.895)	(0.939)								
Gvt Primary School in Vlg		0.010**		0.017***		0.009*		0.015**		0.012*		0.018***
		(0.005)		(0.006)		(0.005)		(0.006)		(0.006)		(0.006)
Vlg has Anganwadi		0.005		0.004		0.005		0.004		0.004		0.004
		(0.003)		(0.004)		(0.004)		(0.004)		(0.003)		(0.004)
Vlg is connected to a pucca road		0.014***		0.012***		0.014***		0.012***		0.014***		0.012***
		(0.003)		(0.003)		(0.003)		(0.003)		(0.003)		(0.004)
Vlg has ration shop		0.013***		0.014***		0.013***		0.014***		0.012***		0.014***
		(0.002)		(0.002)		(0.003)		(0.003)		(0.002)		(0.002)
Observations	827547	695403	831614	698703	386104	325264	388003	326780	441443	370139	443611	371923
R^2	0.840	0.852	0.828	0.838	0.838	0.849	0.827	0.837	0.843	0.855	0.829	0.840

The outcome variable is the probability of mastering basic numeracy and literacy for children aged 5-10 using the ASER data. Robust standard errors clustered on National Sample Survey regions and are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear national sample survey trends. Gender specific linear trends are included in the estimates for the pooled sample. * $p < .10$, ** $p < .05$, *** $p < .01$

Table B.31: Effects on age standardised overall test scores using NSS regions.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
age standardised overall score in:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic (Std. goitre areas per NSS region)	0.023**	0.021*	0.029**	0.029**	0.029***	0.025**	0.038**	0.040**	0.019	0.016	0.019*	0.019
	(0.010)	(0.012)	(0.011)	(0.013)	(0.009)	(0.011)	(0.015)	(0.017)	(0.014)	(0.015)	(0.011)	(0.013)
Mother's Education	0.030***	0.029***	0.033***	0.033***	0.031***	0.031***	0.034***	0.035***	0.029***	0.028***	0.031***	0.031***
	(0.002)	(0.002)	(0.003)	(0.003)	(0.002)	(0.002)	(0.003)	(0.003)	(0.002)	(0.002)	(0.002)	(0.003)
House Material: Semi-Pucca	0.104***	0.100***	0.104***	0.100***	0.105***	0.102***	0.104***	0.101***	0.104***	0.099***	0.105***	0.099***
	(0.007)	(0.007)	(0.008)	(0.008)	(0.009)	(0.009)	(0.009)	(0.009)	(0.008)	(0.008)	(0.008)	(0.009)
House Material: Pucca	0.180***	0.176***	0.185***	0.179***	0.186***	0.183***	0.191***	0.187***	0.174***	0.170***	0.180***	0.173***
	(0.013)	(0.014)	(0.013)	(0.015)	(0.015)	(0.016)	(0.015)	(0.016)	(0.012)	(0.013)	(0.012)	(0.014)
Household Size	-0.001	-0.001	-0.002	-0.002	-0.001	-0.001	-0.002	-0.002	-0.001	-0.001	-0.002	-0.002
	(0.001)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)	(0.001)	(0.002)	(0.002)
Girl	1.585	0.352	-5.115**	-7.296***								
	(2.582)	(2.880)	(2.296)	(2.641)								
Gvt Primary School in Vlg		0.031**		0.045***		0.026*		0.039**		0.035**		0.050***
		(0.013)		(0.015)		(0.013)		(0.015)		(0.016)		(0.016)
Vlg has Anganwadi		0.020**		0.019*		0.023**		0.020*		0.017*		0.018*
		(0.009)		(0.010)		(0.011)		(0.012)		(0.009)		(0.010)
Vlg is connected to pucca road		0.037***		0.028***		0.037***		0.026***		0.038***		0.030***
		(0.008)		(0.009)		(0.008)		(0.009)		(0.009)		(0.010)
Vlg has rationshop		0.033***		0.035***		0.035***		0.038***		0.031***		0.033***
		(0.006)		(0.006)		(0.007)		(0.008)		(0.006)		(0.006)
Observations	827547	695403	831614	698703	386104	325264	388003	326780	441443	370139	443611	371923
R^2	0.075	0.074	0.090	0.090	0.082	0.081	0.098	0.099	0.068	0.067	0.083	0.082

The outcome variables are age standardised overall numeracy and literacy scores for children aged 5-10 using the ASER data. Robust standard errors clustered on National Sample Survey regions and are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear national sample survey trends. Gender specific linear trends are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.32: Effect on basic skills using the standardised number of endemic goitre areas per state.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic (Std. goitre points per state)	0.022***	0.016***	0.022***	0.018***	0.022***	0.017***	0.024***	0.021***	0.021***	0.014**	0.021***	0.016***
	(0.005)	(0.005)	(0.004)	(0.006)	(0.005)	(0.005)	(0.005)	(0.006)	(0.006)	(0.006)	(0.005)	(0.006)
Mother's Education	0.011***	0.011***	0.012***	0.012***	0.012***	0.011***	0.013***	0.013***	0.011***	0.010***	0.012***	0.012***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.002)	(0.001)	(0.001)	(0.001)	(0.001)
Housing material: Semi-pucca	0.036***	0.033***	0.037***	0.034***	0.036***	0.033***	0.036***	0.033***	0.037***	0.034***	0.038***	0.034***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
Housing material: Pucca	0.066***	0.062***	0.068***	0.064***	0.066***	0.063***	0.070***	0.066***	0.064***	0.061***	0.067***	0.063***
	(0.005)	(0.006)	(0.006)	(0.006)	(0.006)	(0.007)	(0.006)	(0.007)	(0.005)	(0.006)	(0.005)	(0.005)
Household Size	-0.000	-0.000	-0.001	-0.001	-0.000	-0.000	-0.001	-0.000	-0.000	-0.000	-0.001	-0.001
	(0.000)	(0.000)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	2.761**	2.693**	-0.495	-0.956								
	(1.183)	(1.133)	(0.919)	(0.788)								
Government Primary School in Vlg		0.010*		0.017***		0.009		0.015**		0.012*		0.019***
		(0.006)		(0.005)		(0.006)		(0.006)		(0.006)		(0.006)
Vlg has Anganwadi		0.004		0.004		0.005		0.004		0.003		0.004
		(0.004)		(0.005)		(0.004)		(0.006)		(0.004)		(0.005)
Vlg is connected to pucca road		0.014***		0.012***		0.014***		0.011***		0.014***		0.012***
		(0.003)		(0.003)		(0.002)		(0.003)		(0.003)		(0.004)
Vlg has rationshop		0.013***		0.014***		0.013***		0.014***		0.012***		0.014***
		(0.002)		(0.002)		(0.003)		(0.002)		(0.002)		(0.003)
Observations	827547	695403	831614	698703	386104	325264	388003	326780	441443	370139	443611	371923
R^2	0.840	0.852	0.828	0.838	0.838	0.849	0.827	0.836	0.843	0.855	0.829	0.840

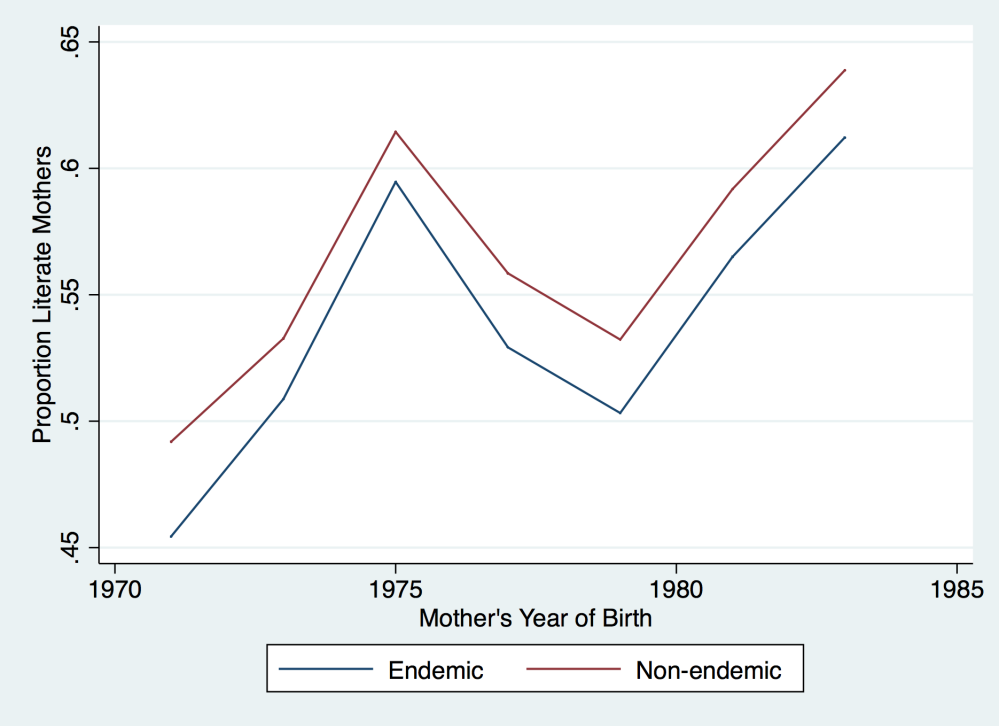
This tables shows the regression results from Equation 2.2 with the exception that I measure endemicity by the standardised number of goitre areas per state. The outcome variable is the probability of mastering basic numeracy and literacy for children aged 5-10 using the ASER data. Robust standard errors clustered on states and are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear state trends. Gender specific linear trends on state level are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.33: Effect on overall age standardised numeracy and literacy scores using standardised goitre areas/states.

	Pooled				Girls				Boys			
Dependent variable is the	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)
probability of knowing basic:	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy	Numeracy	Numeracy	Literacy	Literacy
Iodised * Endemic (Std. goitre points per state)	0.003	-0.002	0.007	0.003	0.004	-0.000	0.015*	0.012**	0.002	-0.004	-0.001	-0.005
	(0.007)	(0.005)	(0.006)	(0.005)	(0.008)	(0.005)	(0.008)	(0.005)	(0.007)	(0.007)	(0.006)	(0.006)
Mother's Education	0.047***	0.046***	0.046***	0.047***	0.047***	0.048***	0.048***	0.049***	0.045***	0.045***	0.044***	0.045***
	(0.003)	(0.003)	(0.004)	(0.004)	(0.003)	(0.003)	(0.004)	(0.004)	(0.003)	(0.003)	(0.003)	(0.003)
Housing material: Semi-pucca	0.117***	0.109***	0.123***	0.118***	0.106***	0.103***	0.118***	0.113***	0.117***	0.114***	0.128***	0.122***
	(0.009)	(0.010)	(0.008)	(0.008)	(0.009)	(0.010)	(0.008)	(0.008)	(0.010)	(0.010)	(0.009)	(0.009)
Housing material: Pucca	0.264***	0.247***	0.250***	0.245***	0.245***	0.244***	0.255***	0.253***	0.251***	0.249***	0.246***	0.239***
	(0.013)	(0.014)	(0.013)	(0.014)	(0.014)	(0.014)	(0.014)	(0.015)	(0.013)	(0.014)	(0.013)	(0.013)
Household Size	-0.000	-0.001	-0.001	-0.000	-0.001	-0.001	-0.001	-0.000	-0.001	-0.001	-0.001	-0.001
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.001)	(0.001)	(0.002)	(0.002)
Girl	-5.363*	-8.335*	-3.713	-3.927								
	(3.102)	(4.360)	(3.747)	(4.642)								
Gvt Primary School in Vlg		0.040**		0.042**		0.042**		0.039*		0.038**		0.045***
		(0.016)		(0.017)		(0.018)		(0.020)		(0.016)		(0.016)
Vlg has Anganwadi		0.007		0.010		0.010		0.010		0.005		0.010
		(0.010)		(0.011)		(0.007)		(0.009)		(0.013)		(0.014)
Vlg is connected to a pucca road		0.044***		0.044***		0.044***		0.041***		0.044***		0.045***
		(0.010)		(0.012)		(0.010)		(0.012)		(0.012)		(0.013)
Vlg has ration shop		0.048***		0.046***		0.047***		0.046***		0.049***		0.046***
		(0.005)		(0.006)		(0.005)		(0.006)		(0.005)		(0.007)
Observations	827547	695403	831614	698703	386104	325264	388003	326780	441443	370139	443611	371923
R^2	0.149	0.163	0.123	0.131	0.175	0.187	0.137	0.146	0.136	0.143	0.111	0.117

This tables shows the regression results from Equation 2.2 with the exception that I measure endemicity by the standardised number of goitre areas per state. The outcome variable are age standardised overall test scores in numeracy and literacy for children aged 5-10 using the ASER data. Robust standard errors clustered on states and are presented in parentheses. The following fixed effects are included; year of birth, survey year, survey year*year of birth and linear state trends. Gender specific linear trends on state level are included in the estimates for the pooled sample.* $p < .10$, ** $p < .05$, *** $p < .01$

Figure B.7: Pre-trends in literacy

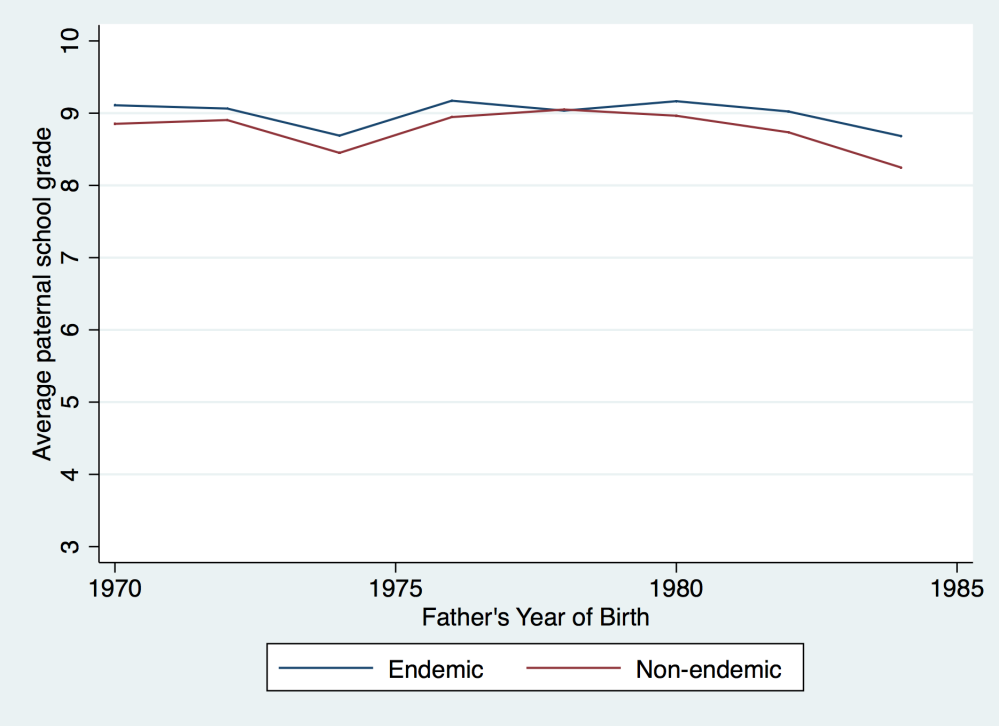


This graph plots the trends in the proportion of mothers to the children surveyed in ASER who are literate, by their year of birth. Trends are shown by historical goitre endemicity status of the district of residence and prior to the access to iodised salt.

Figure B.8: Pre-trends in schooling attainment - mothers

This graph plots the trends in the average grade completed of mothers to the children surveyed in ASERm by their year of birth. Trends are shown by historical goitre endemicity status of the district of residence and prior to the access to iodised salt.

Figure B.9: Pre-trends in schooling attainment - fathers



This graph plots the trends in the average grade completed of the fathers to the children surveyed in ASER, by their year of birth. Trends are shown by historical goitre endemicity status of the district of residence and prior to the access to iodised salt.

Table B.34: Placebo regression: Effect on village, household and child characteristics

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Mother's Educ	Pucca House	Girl	Gvt Primary School	Anganwadi	Pucca road	Ration shop
Iodised * Endemic	-0.073	-0.014**	0.000**	0.002	-0.015***	-0.004	-0.004
	(0.050)	(0.006)	(0.000)	(0.004)	(0.005)	(0.006)	(0.007)
Observations	1175015	1102800	1237498	1044654	958189	1093464	1091376
R^2	0.545	0.459	1.000	0.938	0.928	0.791	0.741

Robust standard errors clustered on district in parentheses. The following covariates are included:

year of birth, survey year, survey year*year of birth and gender specific linear district trends.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.35: Placebo regression: Effect on village, household and child characteristics with controls

	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	Mother's Educ	Pucca House	Girl	Gvt Primary School	Anganwadi	Pucca road	Ration shop
Iodised * Endemic	0.001	-0.017**	0.000**	0.006	-0.015***	-0.004	0.002
	(0.055)	(0.007)	(0.000)	(0.005)	(0.005)	(0.007)	(0.007)
Pucca	2.279***		0.000	0.001	0.004***	0.036***	0.024***
	(0.039)		(0.000)	(0.001)	(0.001)	(0.003)	(0.003)
HH size	-0.017***	0.009***	0.000***	-0.000	0.000	-0.000	-0.000
	(0.004)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Girl	13.502	0.150		0.296	0.611	-0.846	-0.651
	(8.571)	(0.824)		(0.486)	(0.564)	(0.836)	(0.929)
Gvt Primary School in Vlg	0.036	0.003	0.000		0.140***	0.037***	0.114***
	(0.044)	(0.004)	(0.000)		(0.012)	(0.008)	(0.010)
Vlg has Anganwadi	0.222***	0.017***	0.000	0.118***		0.082***	0.217***
	(0.038)	(0.004)	(0.000)	(0.010)		(0.008)	(0.010)
Vlg is connected to a pucca road	0.410***	0.051***	-0.000	0.014***	0.036***		0.167***
	(0.027)	(0.003)	(0.000)	(0.003)	(0.004)		(0.007)
Vlg has ration shop	0.365***	0.034***	-0.000	0.037***	0.083***	0.148***	
	(0.023)	(0.003)	(0.000)	(0.004)	(0.004)	(0.006)	
Mother's Educ			0.000	0.000	0.001***	0.004***	0.004***
			(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Observations	852979	887174	852979	852979	852979	852979	852979
R^2	0.580	0.473	1.000	0.942	0.930	0.803	0.757

Robust standard errors clustered on district in parentheses. The following covariates are included: year of birth, survey year, survey year*year of birth and gender specific linear district trends.

* $p < .10$, ** $p < .05$, *** $p < .01$

Table B.36: Falsification checks - The effect on health related outcomes

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	BCG	BCG	Measles	Measles	Vitamin A	Vitamin A	Had Diarrhoea	Had Diarrhoea
Iodised * Endemic	-0.057**		-0.006		-0.006		0.003	
	(0.028)		(0.033)		(0.033)		(0.013)	
Iodised (Both bans) * Endemic		-0.002		-0.013		-0.013		0.008
		(0.012)		(0.016)		(0.016)		(0.006)
Mother's Education	0.009***	0.011***	0.007***	0.010***	0.007***	0.010***	0.000	-0.000
	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Primary school in village	0.021***	0.020***	0.018***	0.018***	0.018***	0.018***	-0.008**	-0.006*
	(0.006)	(0.005)	(0.005)	(0.005)	(0.005)	(0.005)	(0.004)	(0.003)
Girl	-0.012***	-0.015***	-0.009***	-0.012***	-0.009***	-0.012***	-0.006***	-0.006***
	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)	(0.002)
Anganwadi in vlg	0.019***	0.020***	0.011***	0.011***	0.011***	0.011***	0.001	0.003
	(0.005)	(0.005)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.003)
Semi-Pucca	0.037***	0.040***	0.025***	0.030***	0.025***	0.030***	0.001	0.000
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)	(0.002)	(0.002)
Pucca	0.044***	0.053***	0.037***	0.049***	0.037***	0.049***	-0.001	-0.001
	(0.005)	(0.004)	(0.004)	(0.004)	(0.004)	(0.004)	(0.003)	(0.003)
Observations	175567	217804	175507	217711	175507	217711	175170	217861
R^2	0.587	0.633	0.472	0.527	0.472	0.527	0.194	0.192

Robust standard errors clustered on district in parentheses. Data from the DLHS 2 and DLHS 3 is used.

I control for year of birth, interview year, interview year*year of birth linear district trends.

* $p < .10$, ** $p < .05$, *** $p < .01$

Appendix C

3

Table C.1: Effect on stunting (≤-2 HAZ) - OLS, reduced form and IV regressions.

	OLS		Reduced Form		IV	
	(1)	(2)	(3)	(4)	(5)	(6)
Adequately Iodised Salt	-0.090***	-0.037			-0.128*	-0.070
	(0.024)	(0.023)			(0.069)	(0.071)
Far from Gujarat			-0.048	-0.039		
			(0.028)	(0.046)		
Girl		-0.045		-0.043		-0.047*
		(0.027)		(0.027)		(0.024)
Child used ICDS		0.008		0.006		0.009
		(0.022)		(0.022)		(0.021)
Child had diarrhoea recently		-0.001		-0.000		-0.001
		(0.030)		(0.031)		(0.029)
Child had fever recently		0.008		0.008		0.009
		(0.026)		(0.026)		(0.024)
Child had cough recently		-0.006		-0.005		-0.007
		(0.016)		(0.016)		(0.015)
Mother mildly anaemic		-0.013		-0.012		-0.013
		(0.023)		(0.023)		(0.021)
Mother moderately anaemic		0.016		0.016		0.016
		(0.025)		(0.025)		(0.024)
Mother severely anaemic		0.043		0.041		0.044
		(0.079)		(0.079)		(0.075)
Mother: Primary education		0.038*		0.036		0.039**
		(0.021)		(0.021)		(0.019)
Mother: Secondary education		-0.040		-0.041		-0.038
		(0.026)		(0.026)		(0.024)
Mother: Higher education		-0.103*		-0.105**		-0.101**
		(0.050)		(0.050)		(0.047)
Mother eats fish at least weekly		0.042		0.041		0.041*
		(0.025)		(0.025)		(0.023)
Mother heard of one of ORS or TBC or AIDS		-0.038		-0.042		-0.034
		(0.031)		(0.030)		(0.032)
Mother heard of two out of ORS, TBC and AIDS		-0.040		-0.045		-0.036
		(0.032)		(0.032)		(0.033)
Mother heard of ORS and TBC and AIDS		-0.092**		-0.098***		-0.089***
		(0.033)		(0.034)		(0.033)
Unprotected water source		-0.017		-0.015		-0.018
		(0.018)		(0.018)		(0.017)
Mother practices open defecation		0.009		0.010		0.008
		(0.025)		(0.025)		(0.024)
Poorer		0.039		0.037		0.040
		(0.035)		(0.035)		(0.033)
Middle		-0.036		-0.038		-0.036
		(0.025)		(0.025)		(0.023)
Richer		-0.059*		-0.063*		-0.055*
		(0.033)		(0.033)		(0.030)
Richest		-0.105**		-0.113**		-0.099***
		(0.040)		(0.040)		(0.037)
Observations	2850	2477	2850	2477	2850	2477
R^2	0.009	0.135	0.003	0.134	0.007	0.134

Notes: The outcome variable is the probability of stunting. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions in columns 5 and 6. The covariates included in columns 2, 4 and 6 are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.2: Effect on WAZ, - OLS and IV regressions.

	OLS		IV	
	(1)	(2)	(3)	(4)
	WAZ	WAZ	WAZ	WAZ
Adequately Iodised Salt	0.475***	0.043	0.706	-0.418
	(0.104)	(0.050)	(0.461)	(0.292)
Girl		-0.056		-0.111
		(0.072)		(0.070)
Child used ICDS		-0.092*		-0.038
		(0.049)		(0.075)
Child had diarrhoea recently		-0.101*		-0.088*
		(0.049)		(0.046)
Child had fever recently		-0.183**		-0.160**
		(0.074)		(0.071)
Child had cough recently		0.104		0.019
		(0.063)		(0.074)
Mother mildly anaemic		-0.089**		-0.013
		(0.042)		(0.039)
Mother moderately anaemic		-0.207***		-0.096*
		(0.055)		(0.056)
Mother severely anaemic		-0.388***		-0.311**
		(0.128)		(0.154)
Mother: Primary education		-0.029		0.035
		(0.075)		(0.076)
Mother: Secondary education		0.122*		0.022
		(0.065)		(0.080)
Mother: Higher education		0.264**		-0.001
		(0.119)		(0.122)
Mother eats fish at least weekly		-0.020		0.057
		(0.050)		(0.079)
Mother heard of one of ORS or TBC or AIDS		0.155*		0.187*
		(0.088)		(0.101)
Mother heard of two out of ORS, TBC and AIDS		0.206***		0.219**
		(0.072)		(0.101)
Mother heard of ORS and TBC and AIDS		0.324***		0.284***
		(0.090)		(0.099)
Unprotected water source		0.048		0.118
		(0.051)		(0.073)
Mother practices open defecation		-0.046		-0.077
		(0.084)		(0.081)
Poorer		-0.068		0.140**
		(0.084)		(0.067)
Middle		0.166*		0.160**
		(0.086)		(0.079)
Richer		0.249**		0.199**
		(0.099)		(0.084)
Richest		0.418***		0.205*
		(0.140)		(0.111)
Constant	-1.501***	-3.770***	-1.665***	-1.196***
	(0.068)	(0.446)	(0.279)	(0.415)
Observations	2784	2420	2784	2420
R^2	0.028	0.347	0.022	0.143

Notes: The outcome variable is WAZ. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in columns 3 and 4. The covariates included in columns 2 and 4 are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.3: Effect on HAZ - OLS and TSLS regressions for girls and boys separately

	Girls		Boys	
	(1)	(2)	(3)	(4)
	OLS	TSLS	OLS	TSLS
Adequately Iodised Salt	0.090	0.762*	0.192*	0.632*
	(0.180)	(0.439)	(0.101)	(0.362)
Child used ICDS	-0.064	-0.073	-0.076	-0.087
	(0.105)	(0.101)	(0.144)	(0.135)
Child had diarrhoea recently	-0.046	-0.031	0.024	0.014
	(0.133)	(0.120)	(0.135)	(0.123)
Child had fever recently	0.107	0.122	-0.151	-0.171*
	(0.141)	(0.124)	(0.096)	(0.091)
Child had cough recently	-0.057	-0.016	0.179	0.182
	(0.126)	(0.105)	(0.158)	(0.151)
Mother mildly anaemic	-0.110	-0.091	-0.108	-0.104
	(0.129)	(0.114)	(0.106)	(0.095)
Mother moderately anaemic	-0.120	-0.087	-0.240***	-0.266***
	(0.157)	(0.140)	(0.083)	(0.078)
Mother severely anaemic	-0.399	-0.483*	-0.315	-0.308
	(0.292)	(0.261)	(0.302)	(0.270)
Mother: Primary education	-0.119	-0.155	-0.151	-0.172
	(0.123)	(0.110)	(0.156)	(0.143)
Mother: Secondary education	0.284*	0.238	-0.037	-0.050
	(0.164)	(0.156)	(0.108)	(0.103)
Mother: Higher education	0.837*	0.724*	0.099	0.102
	(0.409)	(0.391)	(0.269)	(0.248)
Mother eats fish at least weekly	-0.027	0.003	-0.256*	-0.259**
	(0.104)	(0.111)	(0.124)	(0.113)
Mother heard of one of ORS or TBC or AIDS	0.356	0.302	0.049	-0.010
	(0.215)	(0.206)	(0.196)	(0.197)
Mother heard of two out of ORS, TBC and AIDS	0.375	0.318	0.107	0.036
	(0.225)	(0.220)	(0.197)	(0.199)
Mother heard of ORS and TBC and AIDS	0.453*	0.391*	0.306	0.250
	(0.250)	(0.237)	(0.208)	(0.190)
Unprotected water source	-0.174	-0.141	0.025	0.023
	(0.108)	(0.098)	(0.108)	(0.100)
Mother practices open defecation	-0.065	-0.059	0.058	0.088
	(0.152)	(0.135)	(0.156)	(0.153)
Poorer	-0.245	-0.250	-0.289**	-0.329***
	(0.206)	(0.201)	(0.129)	(0.119)
Middle	0.018	0.019	0.123	0.090
	(0.185)	(0.184)	(0.206)	(0.191)
Richer	0.124	0.048	0.275*	0.221*
	(0.196)	(0.183)	(0.143)	(0.128)
Richest	0.479*	0.344	0.634***	0.549***
	(0.234)	(0.210)	(0.189)	(0.165)
Constant	-3.487**	-3.975***	-2.668**	-3.272***
	(1.622)	(1.435)	(1.038)	(1.173)
Kleibergen-Paap F-statistic		22.650		24.805
Observations	1216	1216	1261	1261
R^2	0.199	0.176	0.189	0.181

The outcome variable is HAZ estimated separately for girls (columns 1-2) and boys (columns 3-4). The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions in the IV regressions in columns 2 and 4. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.4: Effect of salt with some iodine on HAZ - OLS, first stage, reduced form and IV regressions.

	OLS		First Stage		Reduced Form		IV	
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
	HAZ	HAZ	Adequately Iodised Salt	Adequately Iodised Salt	HAZ	HAZ	HAZ	HAZ
Inadequately iodised salt	-0.093	-0.019					-0.401	-0.602
	(0.090)	(0.064)					(0.359)	(0.488)
Far from Gujarat			-0.348***	-0.380***	0.140	0.229		
			(0.056)	(0.055)	(0.131)	(0.212)		
Girl		0.100		0.061		0.100		0.136
		(0.197)		(0.039)		(0.197)		(0.170)
Child used ICDS		0.046		-0.059**		0.054		0.019
		(0.135)		(0.021)		(0.133)		(0.127)
Child had diarrhoea recently		0.052		-0.025		0.051		0.036
		(0.066)		(0.025)		(0.067)		(0.072)
Child had fever recently		-0.135		-0.008		-0.136		-0.141
		(0.118)		(0.023)		(0.118)		(0.109)
Child had cough recently		0.183*		0.067**		0.179*		0.219**
		(0.090)		(0.030)		(0.087)		(0.105)
Mother mildly anaemic		-0.096		0.000		-0.098		-0.098
		(0.156)		(0.022)		(0.156)		(0.152)
Mother moderately anaemic		-0.116		0.017		-0.119		-0.108
		(0.100)		(0.024)		(0.100)		(0.097)
Mother severely anaemic		0.030		0.094		0.030		0.087
		(0.257)		(0.071)		(0.256)		(0.232)
Mother: Primary education		0.005		0.007		0.004		0.008
		(0.151)		(0.034)		(0.151)		(0.138)
Mother: Secondary education		0.107		-0.006		0.107		0.103
		(0.109)		(0.024)		(0.109)		(0.100)
Mother: Higher education		0.574		0.034		0.556		0.576
		(0.365)		(0.101)		(0.362)		(0.364)
Mother eats fish at least weekly		0.022		0.004		0.032		0.035
		(0.163)		(0.031)		(0.167)		(0.154)
Mother heard of one of ORS or TBC or AIDS		-0.035		-0.010		-0.037		-0.043
		(0.168)		(0.047)		(0.168)		(0.167)
Mother heard of two out of ORS, TBC and AIDS		0.025		-0.028		0.026		0.009
		(0.176)		(0.048)		(0.177)		(0.174)
Mother heard of ORS and TBC and AIDS		0.062		-0.020		0.074		0.062
		(0.172)		(0.047)		(0.171)		(0.174)
Unprotected water source		-0.018		0.064*		-0.027		0.012
		(0.065)		(0.036)		(0.062)		(0.065)
Mother practices open defecation		-0.042		0.016		-0.037		-0.027
		(0.135)		(0.048)		(0.135)		(0.143)
Poorer		0.179		0.002		0.178		0.179
		(0.182)		(0.028)		(0.182)		(0.172)
Middle		0.167		0.077**		0.165		0.211
		(0.145)		(0.033)		(0.146)		(0.148)
Richer		0.327*		0.055		0.326*		0.360**
		(0.188)		(0.058)		(0.188)		(0.168)
Richest		1.010***		0.094		1.016***		1.073***
		(0.233)		(0.066)		(0.237)		(0.255)
Constant	-1.035***	-5.906***	1.505***	2.309***	-1.205***	-5.990***	-0.601	-4.599***
	(0.120)	(1.084)	(0.052)	(0.190)	(0.068)	(1.048)	(0.510)	(1.548)
Kleibergen-Paap F-statistic					-		38.632	47.186
Observations	2011	1825	2011	1825	2011	1825	2011	1825
R^2	0.001	0.140	0.099	0.204	0.001	0.141	-0.007	0.118

Notes: The outcome is HAZ. The consumption of inadequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions in column 7 and 8. The covariates included in specifications 2, 4, 6 and 8 are described in subsection 3.5 but are not shown due to space restrictions. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.5: Effect on fertility and infant mortality - OLS and IV regressions

	Fertility		Infant Mortality	
	(1)	(2)	(3)	(4)
	OLS	IV	OLS	IV
Adequately Iodised Salt	0.011	0.056	-0.005	-0.055
	(0.018)	(0.069)	(0.008)	(0.041)
Mother mildly anaemic	-0.008	-0.008	0.014	0.013
	(0.016)	(0.015)	(0.010)	(0.009)
Mother moderately anaemic	0.018	0.018	0.006	0.006
	(0.023)	(0.022)	(0.006)	(0.006)
Mother severely anaemic	0.080*	0.078*	0.053***	0.055***
	(0.046)	(0.044)	(0.016)	(0.016)
Mother: Primary education	-0.000	-0.002	0.006	0.008
	(0.031)	(0.029)	(0.012)	(0.011)
Mother: Secondary education	0.007	0.005	-0.012	-0.008
	(0.034)	(0.033)	(0.009)	(0.009)
Mother: Higher education	0.053	0.048	0.008	0.013
	(0.047)	(0.046)	(0.031)	(0.030)
Mother eats fish at least weekly	-0.032	-0.031	0.013*	0.014**
	(0.023)	(0.022)	(0.006)	(0.006)
Mother heard of one of ORS or TBC or AIDS	0.061	0.056	0.002	0.004
	(0.048)	(0.044)	(0.022)	(0.022)
Mother heard of two out of ORS, TBC and AIDS	0.063	0.058	0.002	0.005
	(0.049)	(0.044)	(0.028)	(0.028)
Mother heard of ORS and TBC and AIDS	0.063	0.059	-0.006	-0.001
	(0.041)	(0.037)	(0.026)	(0.025)
Unprotected water source	0.063**	0.065***	0.023**	0.019**
	(0.024)	(0.023)	(0.008)	(0.008)
Mother practices open defecation	0.014	0.015	0.003	0.003
	(0.027)	(0.027)	(0.012)	(0.011)
Poorer	0.007	0.005	0.000	0.003
	(0.029)	(0.028)	(0.007)	(0.007)
Middle	0.062**	0.060**	0.008	0.011
	(0.029)	(0.028)	(0.011)	(0.011)
Richer	0.024	0.019	-0.024***	-0.017**
	(0.033)	(0.033)	(0.006)	(0.008)
Richest	-0.062	-0.070	-0.054***	-0.042**
	(0.052)	(0.050)	(0.014)	(0.020)
Kleibergen-Paap F-statistic		37.871		100.835
Observations	2482	2482	3395	3395
R^2	0.218	0.217	0.128	0.120

Notes: The outcome variable in columns 1-2 is the number of children a woman has given birth to during the past 3 years prior to the survey. The outcome for specifications shown in colmns 3-4 is infant mortality - the likelihood that a child, conditional on being born at least one year prior to the survey, dying within one year of birth. The OLS regressions are shown in columns 1 and 3 and IV regressions are displayed in columns 2 and 4. The consumption of adequately iodised salt is instrumented with the indicator variable for residing far from Gujarat in the IV regressions. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. Child level covariates are not included in columns 1-2 and postnatal child variables are not included in columns 3-4. The omitted reference categories for the covariates displayed in the table are: mother has no anaemia, mother has no education, mother has not heard of ORS, TBC or AIDS and poorest wealth quintile. Robust standard errors are clustered on state and are shown in parenthesis. * $p <$ .10, ** $p <$ .05, *** $p <$ .01.

Table C.6: Reduced form placebo regressions: Variables previously used as covariates

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
	Mother practices open defecation	Unprotected water source	Mother's level of health knowledge	Mother's Age	Mother eats fish at least weekly	Mother has some education	Mother's anemia status	Child had diarrhoea recently	Child used ICDS
Far from Gujarat	-0.043	0.305***	-0.491***	-1.364**	-0.247**	0.036	0.062	0.071	-0.263***
	(0.067)	(0.092)	(0.091)	(0.618)	(0.097)	(0.043)	(0.126)	(0.045)	(0.089)
Constant	1.383***	-0.427**	1.689***	15.697***	0.521**	0.395***	0.948**	0.399*	1.321***
	(0.147)	(0.205)	(0.363)	(1.479)	(0.205)	(0.136)	(0.353)	(0.201)	(0.242)
Observations	2476	2476	2476	2476	2476	2476	2476	2476	2476
R^2	0.532	0.239	0.408	0.579	0.422	0.449	0.107	0.117	0.233

Notes: This table shows the reduced form placebo regressions with the outcomes being variables previously used as covariates in the main regressions. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.7: Reduced form placebo regressions: Child health

	(1)	(2)	(3)	(4)	(5)
	Child received vitamin A-supplementation	Child received iron supplementation	Ever received vaccination	Child has anaemia	Birth Weight
Far from Gujarat	0.010	-0.079***	0.029	-0.093	-0.674
	(0.053)	(0.024)	(0.072)	(0.060)	(136.661)
Constant	1.124***	0.084	1.271***	1.040***	1894.675***
	(0.179)	(0.082)	(0.207)	(0.134)	(638.536)
Observations	2427	2469	1256	1300	756
R^2	0.263	0.106	0.373	0.151	0.259

Notes: This table shows the reduced form placebo regressions for child health related outcomes. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.

Table C.8: Reduced form placebo regressions: Pregnancy related outcomes

	(1) Birth at facility	(2) Number of ANC visits	(3) Iron supplementation during pregnancy	(4) Months of breastfeeding	(5) Infant Mortality
Far from Gujarat	-0.140**	-2.462*	0.075	0.128	0.018
	(0.053)	(1.271)	(0.116)	(0.275)	(0.012)
Child used ICDS	-0.108**	-0.190	0.080*	0.075	
	(0.044)	(0.252)	(0.040)	(0.153)	
Child had diarrhoea recently	-0.011	-0.507	-0.061	0.008	
	(0.039)	(0.359)	(0.038)	(0.145)	
Child had fever recently	-0.000	-0.096	0.072	-0.200	
	(0.045)	(0.342)	(0.048)	(0.157)	
Child had cough recently	0.059**	0.191	0.039	-0.012	
	(0.026)	(0.381)	(0.030)	(0.139)	
Mother mildly anaemic	-0.076*	0.104	-0.017	0.114	0.003
	(0.039)	(0.231)	(0.033)	(0.104)	(0.008)
Mother moderately anaemic	-0.038	-0.408	-0.046	-0.095	0.022
	(0.055)	(0.274)	(0.041)	(0.155)	(0.014)
Mother severely anaemic	0.035	-2.000	-0.018	-0.820	0.053*
	(0.088)	(1.326)	(0.172)	(0.528)	(0.027)
Mother: Primary education	0.011	-0.277	-0.021	0.110	0.006
	(0.070)	(0.221)	(0.048)	(0.208)	(0.013)
Mother: Secondary education	0.004	0.338	0.099*	0.426	-0.013
	(0.062)	(0.410)	(0.051)	(0.259)	(0.011)
Mother: Higher education	-0.029	-0.202	0.151**	0.655**	-0.031
	(0.077)	(0.650)	(0.060)	(0.308)	(0.024)
Mother eats fish at least weekly	-0.001	0.052	0.037	0.062	-0.001
	(0.037)	(0.397)	(0.041)	(0.105)	(0.007)
Mother heard of one of ORS or TBC or AIDS	0.221**	0.644	0.305*	0.692	0.002
	(0.106)	(0.762)	(0.156)	(0.541)	(0.012)
Mother heard of two out of ORS, TBC and AIDS	0.209**	0.899	0.258*	0.709	-0.004
	(0.094)	(0.866)	(0.135)	(0.472)	(0.012)
Mother heard of ORS and TBC and AIDS	0.194**	1.223	0.338**	0.795	-0.008
	(0.091)	(0.896)	(0.122)	(0.469)	(0.014)
Unprotected water source	-0.038	0.303	-0.076**	0.067	-0.002
	(0.031)	(0.322)	(0.031)	(0.059)	(0.010)
Mother practices open defecation	-0.122**	0.034	-0.042	0.140	0.011
	(0.047)	(0.425)	(0.030)	(0.129)	(0.013)
Poorer	0.255***	0.633	-0.018	0.040	0.010
	(0.065)	(0.525)	(0.055)	(0.130)	(0.008)
Middle	0.187**	0.686	-0.072	-0.049	-0.004
	(0.066)	(0.474)	(0.064)	(0.156)	(0.012)
Richer	0.197**	1.279*	-0.093	-0.085	-0.022
	(0.087)	(0.721)	(0.068)	(0.181)	(0.013)
Richest	0.255**	2.598***	-0.055	-0.421	-0.018
	(0.100)	(0.828)	(0.072)	(0.262)	(0.022)
Girl	-0.015	0.226	0.049	0.058	-0.005
	(0.047)	(0.354)	(0.032)	(0.162)	(0.015)
Constant	0.372	4.349	0.693**	19.554***	0.594***
	(0.409)	(2.859)	(0.257)	(1.210)	(0.097)
Observations	756	749	752	756	4882
R^2	0.375	0.349	0.278	0.892	0.113

Notes: This table shows the reduced form placebo regressions for pregnancy related outcomes. The covariates included in all specifications are described in subsection 3.5 but are not shown due to space restrictions. Robust standard errors are clustered on state and are shown in parenthesis. * $p < .10$, ** $p < .05$, *** $p < .01$.