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Sara Sofie Abrahamsson
Aline Bütikofer
Krzysztof Karbownik

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Swallow This: Childhood and Adolescent Exposure to Fast Food Restaurants, BMI, and Cognitive Ability

Sara Sofie Abrahamsson, Aline Bütikofer, and Krzysztof Karbownik

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ABSTRACT

Using spatial and temporal variation in openings of fast food restaurants in Norway between 1980 and 2007, we study the effects of changes in the supply of high caloric nutrition on the health and cognitive ability of young adult males. Our results indicate that exposure to these establishments during childhood and adolescence increases BMI and has negative effects on cognition. Heterogeneity analysis does not reveal meaningful differences in the effects across groups, including for those with adverse prenatal health or high paternal BMI, an exception being that cognition is only affected by exposure at ages 0--12 and this effect is mediated by paternal education.

Sara Sofie Abrahamsson
Center for Fertility and Health,
Norwegian Institute of Public Health
Postboks 222 Skøyen
Oslo N-0213
Norway
sofiesara.abrahamsson@fhi.no

Aline Bütikofer
Department of Economics
Norwegian School of Economics
Hellev. 30, N-5035 Bergen
Norway
aline.buetikofer@nhh.no

Krzysztof Karbownik
Department of Economics
Emory University
Rich Memorial Building
1602 Fishburne Drive
Atlanta, GA 30322
and CESifo
and also NBER
krzysztof.karbownik@emory.edu

1 Introduction

Obesity is one of the leading causes of preventable morbidity and mortality in Western countries, with the childhood and adolescent period being of particular concern. Excess weight during these critical developmental years is associated with higher incidence of asthma, diabetes, adult cardiovascular problems, increased risk of cancer, and adverse social and economic outcomes in early adulthood (WHO, 2016). It affects both poor and rich countries alike, with the United States having comparable obesity prevalence to Libya (about 30–35 percent) and Norway to Haiti (about 23 percent) (World Obesity Federation, 2022). Thus, the rise in obesity rates over the past five decades is a global phenomenon, and although many different actions are being taken to reduce or reverse it, the forecasts suggest an urgent need to counter the obesogenic environment by addressing vital elements in children's lives such as diet and physical activity (OECD, 2017).

Physiologically, the cause of obesity is excessive food energy intake, while its social and economic foundations have long been disputed (Cawley, 2010), and researchers struggle to provide population-level causal estimates for the hypothesized channels (Keith et al., 2006). Above and beyond genetics and biological factors, two major contributors to the rise in obesity that have been proposed are: (1) food supply and marketing practices that decrease price and increase the consumption of highly processed meals; and (2) declining physical activity (Wareham et al., 2005; Lakdawalla et al., 2005; Chandon and Wansink, 2012; Griffith et al., 2016; Dubois et al., 2018). The former factor in particular has gained attention in both the media and policy circles in recent years. The Royal College of Pediatrics and Childhood Health in the UK, for example, has proposed banning fast food restaurants from opening in close proximity to schools, and multiple jurisdictions worldwide have imposed additional taxes on sodas intended to reduce the consumption of drinks with added sugar (WHO, 2015; Marsh, 2018). Likewise, a proposal is currently under consideration in the UK to ban promotions and discounts on unhealthy food items (Forrest, 2020) and to limit the advertisement of such products (Siddique, 2020). During the COVID-19 pandemic, many US states banned fast food drive-throughs in order to limit the accessibility of unhealthy eating options (Helmer, 2020). Such policies are often motivated by available evidence on the effects of fast food supply (Davis and Carpenter, 2009; Currie et al., 2010), yet this evidence is far from conclusive (Cawley, 2015) and mostly based on US data where many other obesogenic trends are present (Cutler et al., 2003).[1] At the same time, empirical evidence on such restrictions supports the notion that banning fast food outlets decreases their spatial density and thus the supply of unhealthy food options (Brown et al., 2022). In Canada, a ban on the sale of junk food in schools also led to a reduction in the students' BMI (Leonard, 2017). Furthermore, less extreme policy measures such as calorie posting (Bollinger et al., 2011; Restrepo, 2017; Aranda et al., 2021), common sense consumption acts (Carpenter and Tello-Trillo, 2015), advertisement bans (Dubois et al., 2018), and soda taxes (Fletcher et al., 2010; Dubois et al., 2020; Seiler et al., 2021) may also prove effective by either directly changing consumption patterns or indirectly encouraging increases in healthy behaviors.

[1] More extensive research on this topic has been conducted in the fields of medicine and epidemiology, but these papers seldom use quasi-experimental variation, are limited to cross-sectional analyses, and are based on small sample sizes. Hence, we treat much of this literature as descriptive. See Jia et al. (2021) for a recent review.

The aforementioned policy interventions are often based on economic arguments. Given that the average fast food meal consists of more than twice the energy density of a recommended healthy meal (Prentice and Jebb, 2003), and excessive consumption of sugar-sweetened beverages leads to gains in weight (Malik et al., 2013), it seems plausible that changing the cost of access or the availability of such products could lead to health benefits. At the same time, proximity to fast food restaurants facilitates easier access to high-caloric nutrition by lowering both monetary and non-monetary costs. Moore et al. (2009) document that an increased density of fast food outlets in a neighborhood leads to higher consumption of such products and substitution away from a healthy diet. However, the negative health effects of this broader availability might not unravel if healthier options are available and affordable (Niebylski et al., 2015), if consumption patterns differ by demographic group (Dunn et al., 2012), or if positive changes in physical activity occur in parallel (Courtemanche et al., 2021). Ultimately, it is an empirical question of whether increased access to fast food outlets leads to worse health and non-health outcomes. At the same time, it is clear that the consumption of such food items is increasing given the expansion and profits seen in this sector of the economy (NACS, 2018).

In this paper, we ask the following questions to understand the effects of fast food restaurants' expansion on children's well-being: Does an increased supply of fast food outlets lead to worse health outcomes as measured by BMI? Do these negative effects extend beyond health capital and into human capital and cognition? And finally, how homogeneous are the effects across a variety of individual characteristics including the propensity for being overweight and prenatal health?

We answer these questions by leveraging data on all fast food restaurants that opened in Norway between 1980 and 2007 paired with a universe of conscription and education data for all Norwegian males born between 1980 and 1989. Norway is a country where more than 50 percent of all adult males are currently overweight (SSB, 2017), and this rate has increased more than seven-fold, from about 7 percent in the early 1980s when the first Western fast food restaurants opened (FHI, 2017). Our main empirical approach exploits quasi-random variation in changes in access to fast food using a two-way fixed effects methodology. Thus, we compare the outcomes of individuals residing in narrow geographical locations in Norway where a restaurant has and has not been opened, and before versus after its establishment. This is an intent-to-treat effect that estimates the consequences of facilitating access to, rather than consumption of, fast food.

We find that growing up in a neighborhood that has a fast food restaurant increases BMI and the likelihood of being overweight in young adult males. These effects appear economically meaningful given that a mean exposure confers a BMI increase of 1.4% or about 35% of the growth in average BMI between the first and the last cohort observed in our data. The overweight rate increases at about 1.6% per year of exposure to a fast food establishment, which, given the average exposure, likewise amounts to over a third of the growth in overweight rates across the cohorts included in the study. These health effects appear homogeneous across groups and we do not find meaningful heterogeneity by paternal BMI, household socioeconomic status, or the child's neonatal health. However, at least when it comes to the probability of being overweight, the effects are larger when children are exposed at age 13–19 compared to age 0–12.

Parallel to the adverse health effects, we also find declines in cognition of about 0.56

percent of a standard deviation (SD) per year of exposure. These estimates, although quantitatively smaller, suggest a decline in cognitive ability of 4.1% of a SD for the average number of years of exposure. We further find evidence that exposure to fast food restaurants lowers the likelihood of pursuing an academic track in high school. In contrast to the health outcomes, however, we find that cognition is solely affected by early life exposure to fast food at age 0–12. Furthermore, the negative cognitive effects are reduced by approximately half if the father has at least an academic high school degree.

Our findings on BMI and cognitive ability are robust. Point estimates and statistical significance are not materially affected by the choice of econometric specification, estimation sample, definition of treatment distance, or transformations of the dependent variable. We also demonstrate that our results are unlikely to be driven by differential pre-trends or selection by considering an event study and by employing a randomization inference approach that randomizes the set of locations with fast food restaurants while holding their actual number fixed. These tests mitigate concerns that selection or spurious trends are driving our results.

This paper makes three contributions to the existing literature. First, almost all previous research has focused on the health consequences of access to fast food, while we investigate the effects on both young adults' BMI and cognition for the same population. Second, population-level administrative data allow us to conduct extensive heterogeneity analyses, including the potential interactions between access to fast food restaurants and individual measures of fetal health and propensity for obesity. Much of the previous research has relied on smaller-scale administrative or survey data, which do not allow for such detailed analysis. At the same time, understanding the heterogeneity is of particular relevance to policy-makers given the potential for targeted vs. universal interventions (Dubois et al., 2020; Griffith, 2022) and the observed intergenerational associations in obesity (Classen, 2010; Classen and Thompson, 2016). Finally, to the best of our knowledge, this is the first set of causal estimates on access to fast food restaurants in the context of the Nordic countries where, despite their relatively healthy populations, high per capita income, and universal free healthcare, the obesity rates are growing rapidly.

Our main contribution is to the literature on the effects of the supply of fast food on health outcomes. Research in the fields of economics, epidemiology, medicine, and public health have studied this relationship before but the results have been inconclusive, appear to be context specific, and most of this literature focuses on the US (see, e.g., Rosenheck (2008), Papoutsi et al. (2013), Williams et al. (2014), Cawley (2015), and (Jia et al., 2021) for recent reviews). For example, Davis and Carpenter (2009), Currie et al. (2010), and Sanchez et al. (2012) demonstrate that teenagers attending a school nearby a fast food restaurant have elevated BMI and other measures of excess weight. In contrast, Howard et al. (2011), Asirvatham et al. (2019), and Langellier (2012) find no such relationship. Other papers examine exposure at the place of residence, with estimates likewise ranging from increases in BMI (Elbel et al., 2020; Qian et al., 2017) to no effects (Lee, 2012; Dolton and Tafesse, 2022). The literature studying exposure in adulthood is also inconclusive, with Anderson and Matsa (2011) finding no link between fast food restaurants and obesity, while Giuntella (2018) reports a positive association and excess weight gain in pregnant mothers.[2]

[2]There is also some evidence on the effects of convenience stores and supermarkets on obesity. Howard

The majority of studies to date have focused on the US where, for certain demographic groups, fast food and soda are the most easily accessible and cheapest sources of food. Indeed, prior work has shown that poverty is associated with both a higher consumption of fast food meals and increased obesity (Drewnowski and Specter, 2004). This could be driven by either supply-side factors via food deserts and lack of access to higher quality nutrition (Wrigley et al., 2003) or demand-side factors with different demographic groups having different preferences regarding fast food (Allcott et al., 2019). Despite very different institutional and cultural environments, some studies from other countries such as China (Kong and Zhou, 2021), Mexico (Giuntella et al., 2020), and Sweden (Hamano et al., 2017) have likewise found positive associations between fast or Western food and elevated weight.[3] Dolton and Tafesse (2022) is a notable counterexample, however, as they do not find any meaningful effects in their study on fast food access carried out in the UK. Given this limited international evidence, there is a growing interest in studies from countries where healthier food alternatives are readily available and accessible for most people, and where the fast food business is still relatively new, albeit growing at a high rate.

The aforementioned demographic differences in consumption and obesity rates further call for a detailed heterogeneity analysis. Rich administrative data allow us to not only study heterogeneity by parental employment, education, or place of residence, but also to investigate BMI in the context of prenatal and intergenerational health. Ravelli et al. (1976), Velde et al. (2003), and Fall (2011), for example, all document that nutritional deficiencies during the prenatal period – often manifesting through low birth weight (LBW) – lead to obesity problems later in life. For this reason, we also examine the interaction between birth weight and access to fast food. In an intergenerational context, it could be the case that the propensity for obesity is genetically (Comuzzie and Allison, 1998; Rankinen et al., 2006) or socially driven. For example, Stoklosa et al. (2018) document that "impatient time preferences" and the present bias of parents are associated with both their own and their children's increased obesity. Datar et al. (2022) further show in the US that exposure to counties with higher obesity rates increases the likelihood of obesity among less patient, but not among the more patient, adolescents. Although we cannot measure the time preferences of either children or parents, we observe information about their BMI at age 19 for men in both generations. This allows us to document intergenerational elasticity in obesity and

et al. (2011), Zeng et al. (2019b), and Rummo et al. (2020) document that access to convenience/corner stores is associated with increases in the BMI of school age children in California, Arkansas, and New York City, respectively. Furthermore, Courtemanche and Carden (2011) document that the proliferation of Walmart Supercenters in the US can explain up to 10.5% of the rise in obesity since the late 1980s. On the other hand, Zeng et al. (2019a) find no relationship between supermarket openings or closures and weight. Our primary interest in this paper is access to fast food restaurants, but given the prior research in all empirical specifications, we also control for proximity to convenience stores and supermarkets. This also addresses alternative supply channels of sugar-sweetened beverages.

[3]The last of these studies is of particular interest as it is the only analysis addressing a Nordic country. Although the authors find statistically significant associations in some models, this result is not robust to all modeling choices and the paper does not incorporate any plausibly quasi-random variation. We are not aware of any other studies from the Nordic countries that relate fast food supply to health, although, Svastisalee et al. (2012) and Gebremariam et al. (2012) study the relationship between (fast) food outlets and children's diets in Denmark and Norway, respectively. We believe it is particularly relevant to study the Nordic population as it can be thought of as a lower bound for the effects we might expect in other developed countries with lower levels of health capital and healthcare access.

investigate whether access to fast food moderates this association.

We consider our results to have three main policy implications. First, despite relatively high levels of human and health capital, as well as a more accessible and equitable healthcare system in the Nordic societies, we provide evidence that even in such a setting, the supply of fast food could lead to increased BMI. This is concerning given that many studies on weight reduction find small or no effects (see, e.g., Franz et al. (2007) and Dombrowski et al. (2014) for meta-analyses), that other prevention mechanisms often fail (Roberto et al., 2015), and that the penetration of unhealthy food providers is nonetheless increasing (e.g., in Norway between 1980 and 2007, we observed a five-fold increase in the number of fast food restaurants). Because of these factors, some predictions suggest that within a decade, more than 30% of the Norwegian adult male population could be obese, thus increasing the need for effective policy interventions reducing the obesogenic environment (Lobstein et al., 2022). The obesity rates in other developed countries are predicted at even higher levels. Furthermore, obesity and elevated BMI have been linked to increased healthcare costs (Allison et al., 1999; Cawley and Meyerhoefer, 2012), lower educational achievement (Black et al., 2015), and worse labor market outcomes (Cawley, 2004; Lundborg et al., 2014), thus imposing a direct burden on a country's healthcare system and workforce. This affects the society as a whole as well as potentially increases inequality. Second, we document that fast food access may not only affect health but also cognition, thereby increasing the stakes of a potential lack of counter-measures or regulation of fast food establishments. This extends the literature on the negative consequences of a sugar-rich diet (Gertler and Gracner, 2022) on cognitive outcomes to fast food consumption. Finally, the relative homogeneity of our treatment effects suggests that any interventions or campaigns should target a broad population rather than specific groups e.g., those with a history of obesity in their families (Griffith, 2022).

2 Data

We use Norwegian administrative data on individuals and firms, which makes it possible to link information on exposure to fast food restaurants with individual-level data on health and cognitive outcomes. The data also allow us to track individuals over time and space and facilitate a host of heterogeneity analyses and robustness checks.

2.1 Firm Data

The data on firms come from the Norwegian Register of Business Enterprises and include the exact address, opening year, and, if applicable, the year of closure of all enterprises in Norway. We include the opening and closing down of businesses between 1980 and 2007, and select establishments based on industry codes.[4] A fast food restaurant is defined as a business specializing in serving prepared processed food using counter service at any time of

[4]The system of industry codes is tied to the European Industry Classification System (NACE) and groups industries into five-digit numerical codes. The first four digits are the same across all European countries while the fifth is specific to Norwegian legislation and distinguishes firms according to their most important activity.

the day (code 56.102). This includes traditional Norwegian fast food providers (e.g., sausage stands), Western-style fast food restaurants (e.g., McDonald's), as well as independent kebab, hamburger, and pizza stands, most of which were established after 2000. The first Western-style fast food restaurants opened in the late 1970s in Oslo and marked the arrival of a new food concept. Since then, their number has expanded dramatically, and by 2010, there were over 900 of them.[5] A separate code (56.101) is used for full-service restaurants that offer seating options. Since some fast food restaurants might be classified under this code, we also use it to define treatment. However, to reduce the likelihood of including non-fast food establishments, we only extract the opening and closing-down dates of restaurants with the 56.101 code if their name is linked to fast food establishments.[6]

Figure 1 (panel A) presents the evolution of the fast food market – as indicated by the aforementioned definition – in Norway between 1980 and 2007. In total, our data set includes the openings of 769 and closures of 173 fast food establishments, and each decade we consider is characterized by an increase in new suppliers.[7] For example, 154 new fast food restaurants opened in the decade prior to the first birth cohorts included in our sample (1970–1979), while this figure had risen to 492 in the last decade of our sample (2001–2010). Importantly for our identification, the location of fast food restaurants is not uniform across the country and over time. To illustrate this, Appendix Figure A1 presents Norwegian municipalities with at least one operational fast food supplier in different decades.

Given prior research, it is important to differentiate fast food restaurants from other processed food providers such as supermarkets, grocery stores, convenience stores, and gas stations. For this reason, we geocode the locations as well as the opening and closing down of these suppliers, and use the additional variables as controls in our preferred specification. Convenience stores are defined based on industry code 41.112, gas stations based on industry code 47.300, and grocery stores and supermarkets based on industry code 41.111. The number of convenience stores in Norway increased from 82 in 1980 to 322 in 2007. Equivalent numbers for grocery stores were 965 and 1,236, respectively.

2.2 Individual Level Data

We draw on information from multiple interconnected databases containing individual-level records. The central population register contains annual data on the place of residence (including postcode) and the municipality and postcode of residence at birth, allowing us

[5]The biggest fast food chains in Norway include Burger King, McDonald's, Big Bite, Pizza Hut, Peppes Pizza, Dolly Dimple's, and Subway. In 2000, the market share of McDonald's when it comes to fast food restaurants was 33 percent, and they operated 52 establishments.

[6]Other small-scale fast food restaurants could be coded with either the industry code for traditional restaurants (56.101) or with the industry code for pubs (56.301). We identify these potentially single-site fast food suppliers by extracting all operations including the words "pizza", "hamburger", and "kebab" from the business registry and then manually checking that these establishments were indeed fast food restaurants. Our results are substantively unchanged regardless of whether we include these single-site operating firms or not.

[7]Since in the vast majority of cases, a restaurant that closes down in a specific postcode is almost immediately replaced by another similar outlet, we only refer to "restaurant openings" as a shorthand throughout the paper. Our results are unchanged if we exclude all closures from the coding of the treatment variable.

to assign exposure to fast food restaurants from birth to age 18. Since the major increase in fast food supply takes place after 1980, we limit the sample to individuals born between 1980 and 1989. We exclude individuals born outside of Norway and those who migrate out of the country since we are interested in cumulative exposure from birth to the individual's health assessment at age 18–19. We do not make any further sample restrictions when it comes to individual health and demographic characteristics.[8]

We assign individuals to fast food restaurant exposure according to their geographic proximity, with the idea that closer distance increases accessibility and consumption through lower monetary and non-monetary costs (Moore et al., 2009). We measure the distance as the crow flies between the centroid of the individual's postcode of residence at birth and the exact coordinates of the restaurant. We define treatment at the postcode of residence at birth – rather than contemporaneous postcode of residence – to avoid any issues related to endogenous migration possibly correlated with the changing landscape of fast food supply.[9] Since the average radius of a postcode in Norway is around 400 meters, we use a distance of less than 500 meters (0.5 kilometers (km)) as our most conservative exposure measure. However, we also consider exposures at distances of less than 1,000 meters (1 km) and 2,000 meters (2 km) in the main set of results. We construct our treatment variable of interest, for a given proximity cutoff, as the number of years of exposure to a fast food restaurant, taking values from 0 for never exposed individuals to 19 for those always exposed. Since we do not observe actual consumption, our estimates should be interpreted as reduced-form intent-to-treat effects, but they are nonetheless policy relevant.

Most of our outcome variables come from Norwegian military records, which include weight, height, and cognitive ability assessments for the universe of males. Since military service is only mandatory for males, we necessarily exclude females from the analysis.[10] Before conscripts start service, their medical and cognitive suitability is screened at around the age of 18. In our estimation sample, 73 percent of males are assessed when they turn 18 years of age, 25 percent at the age of 19, 1 percent at the age of 20, and the remaining 2 percent at other ages. Since this examination is compulsory for all men, there is no selection on fitness or ability in the data, a concern that would otherwise arise when using data from countries where military service is voluntary, such as the US.

Our empirical sample includes males born between 1980 and 1989 for whom the out-

[8]Prior research has assigned fast food exposure either at the home or school location level. In Norway, a large majority of children attend local primary and middle schools that are assigned based on strict zoning regulations tied to a home address. For this reason, there is little difference between exposure at the place of residence and at the place of schooling prior to high school. Furthermore, even at the high school level, children in most municipalities attend a local school. There is nevertheless some degree of school choice or grade-based assignments in larger cities. We therefore use the education registry to code exposure to fast food restaurants at high school, and use this variable as an additional control in select regressions.

[9]We are not concerned that people migrate to be closer to fast food restaurants but rather that people in Norway tend to move into cities and areas with increased economic activity, which is precisely where the fast food restaurants are more likely to be located. Thus, we prefer using the postcode of residence at birth as a more conservative measure. In Section 4.2, we present robustness checks where we limit the sample to non-movers. For the remainder of the paper, we use "postcode of birth" as a shorthand for "postcode of residence at birth".

[10]Women are allowed to enroll in the Norwegian military, and for women who do, we have the same information as for men. However, with only a 2 percent participation rate among women during our sample period, it is clearly a selected group.

comes were measured between 1998 and 2007. In the main analysis, we focus on two health outcomes: log BMI and the probability of being overweight, i.e., having a BMI of 25 or more. We multiply these variables by 100 in order to avoid rounding issues when displaying coefficients. In addition, we analyze the effect on cognitive ability, which is measured as a mean score from three IQ tests: arithmetic, word similarities, and figures. We standardize this score by cohort to have a mean of zero and standard deviation of one hundred. To complement the data on cognitive ability, we also investigate an earlier educational outcome – the probability of being enrolled in an academic track in high school. This information comes from a separate data source – the education registry – and is a good indicator for future earnings. Yearly lifetime earnings for men born between 1967 and 1989 are on average NOK 366,088 for those who graduated from an academic track and NOK 331,074 for those who graduated from a vocational track, implying at least a 10% premium for the academic track.[11] In our data, about 98% of students enroll in the first year of high school: 50% enroll in an academic track, 45% enroll in a vocational track, 3% in alternative training plans, and 2% drop out after compulsory education. Among those who continue into high school, 97% enroll the year they turn 16 while 3% enroll at other ages.[12] When analyzing this outcome, we code exposure to fast food restaurants between ages 0 and 16 (rather than 0 and 19 for the outcomes based on the military registry).

Family identifiers in the data allow us to link children with their parents and provide information on family structure and socioeconomic background, including labor force participation, earnings, as well as the education of both mothers and fathers.[13] The military records of fathers – another reason for focusing on children born after 1980 – further allow for a novel analysis where we use the father's BMI as an intergenerational proxy for offspring predispositions, either genetic or environmental, to be overweight. For example, the likelihood that we observe an overweight son is 43% when the father is also overweight compared with just 21% when the father is not. Likewise, intergenerational elasticity in BMI is 0.4, meaning that a 10% increase in the father's BMI increases the son's BMI by about 4% (see Appendix Table A1). This association, although not causal, is almost invariant to including a rich set of controls for both generations, suggesting that parental BMI might meaningfully mediate the effects of access to fast food restaurants.[14] The mean probabilities of being

[11]Note that only about 80% of students who initially enroll in an academic track graduate, while graduation rates for vocational programs are even lower.

[12]Students start high school on-time the year they turn 16, and usually finish within 3 years, but have the right to apply for a high school place until the year they turn 24. The education registry also contains information on school grades, but this data is only available for cohorts born after 1986 (equivalent to about 40% of our sample). For this reason, we choose to study academic track enrollment rather than grades.

[13]In principle, the data structure allows us to use sibling fixed effects as an alternative identification strategy. We do not focus on this type of analysis, however, because families with two male siblings born between 1980 and 1989 represent only a small sub-sample (14% of the sample) that is highly positively selected since wealthier families are more likely to have two or more children. Furthermore, there is limited within-family variation in the treatment variable remaining after including mother-fixed effects, subjecting this strategy to selection-into-identification issues (Miller et al., 2022).

[14]Classen (2010) finds intergenerational BMI elasticities in the US of about 0.35, and while investigating multiple countries, both Classen and Thompson (2016) and Dalton and Xiao (2017) find BMI elasticities of about 0.2. One reason why our elasticity is higher could be that we rely on complete administrative data rather than survey information, which tends to suffer from selection and measurement error biasing the associations towards zero.

overweight for fathers and sons presented in this table further illustrate the policy relevance of the obesity epidemic, with a three times higher likelihood of the younger generation being overweight compared with their parent's generation.

2.3 Descriptive Statistics

We first present descriptive evidence on the expansion of fast food restaurants and parallel changes in BMI as well as cognitive ability. We limit the data to individuals born between 1980 and 1989 in postcodes that at some point had a fast food restaurant within a 30 km radius of their centroid – our primary empirical sample. The left-hand axis of Figure 1 (Panel B) documents the increase in BMI (dashed line) and the right-hand axis the decline in cognitive ability (dotted line) of Norwegian males. The mean BMI increased from 22.5 for the 1980 birth cohort to 23.4 for the 1989 birth cohort, equivalent to 4%. These modest differences in mean BMI over time mask more substantial changes in the shape of the upper-tail of the BMI distribution. To illustrate, over the same period of time, the likelihood of being overweight (BMI > 25) increased from 14 to 18%, or by about 30%, while the likelihood of being obese (BMI > 30) increased from 4 to 7%, or by about 75%. These changes in the tail of the distribution are depicted in Appendix Figure A2, which confirms an outward shift in the upper-tail of BMI among the younger birth cohort. Notably, despite these meaningful increases over time, the mean BMI of those born towards the end of our sample period in 1989, at 23.4, is still somewhat lower than the numbers reported in previous research based on US data (Davis and Carpenter, 2009; Anderson and Matsa, 2011).

Cognitive ability exhibits a similar qualitative pattern, with average raw scores declining from 5.14 for the 1980 birth cohort to 4.86 for the 1989 birth cohort, equivalent to more than 5%. The decline in cognitive ability in recent decades is not only a Norwegian phenomenon and has been documented in multiple other countries (Dutton et al., 2016), but the exact reasons for this drop remain unclear. At the same time, prior research suggests a link between obesity and intelligence (Yu et al., 2010; Belsky et al., 2013), as well as between obesity and skill attainment in early childhood (Cawley and Spiess, 2008). To the extent that increased access to fast food restaurants increases BMI, it could in turn also affect the cognitive ability of these individuals and ultimately play a role in the observed aggregate trends. When discussing the main results, we descriptively explore to what extent the drop in cognitive ability is mediated by the increase in BMI.

A comparison of Panels A and B of Figure 1 suggests a positive (negative) relationship between the expansion of fast food restaurants and BMI (cognitive ability). There were 156 fast food restaurants in 1980, 229 in 1989, and 769 in 2007. Thus, the last exposure year we consider represents approximately a five-fold and 3.5-fold increase in supply compared to the 1980 and 1989 levels, respectively. At the same time, the BMI of exposed cohorts increased by 4% while cognitive ability declined by more than 5%. The main goal of our paper is to investigate to what extent these time-series relationships reflect causality.

To further contextualize our setting, Table 1 provides individual-level summary statistics of the various outcome and control variables. Column 1 provides information on the full population of Norwegian males, while Column 2 limits the sample to males who at any time have been exposed to a fast food restaurant within 30 km of their place of birth. We consider the sample in Column 2 as our baseline population of interest, which we then divide into

treatment and control groups depending on their proximity to a fast food outlet. Almost 90% of Norwegian males born between 1980 and 1989 have at some point (between ages 0 and 19) been exposed to at least one fast food restaurant within 30 km of their place of birth, and this high exposure rate stems from the fact that the majority of the Norwegian population lives in urban agglomerations, which is precisely where fast food restaurants tend to locate. Despite this, there are no meaningful demographic differences (Panel C of Table 1) between the samples in Columns 1 and 2, which should increase the external validity of our estimates.

The subsequent columns in Table 1 divide the estimation sample into two subgroups: males who at some point had a fast food restaurant within 2 km of their place of birth (Column 3) and those who never had access at such close proximity (Column 4). On average, the former group has somewhat better educated and richer parents, which makes sense given that more affluent people in Norway tend to cluster closer to the urban core where fast food restaurants tend to locate. At the same time, we do not observe any striking differences in parental age at birth. Somewhat surprisingly, we find that people residing closer to fast food restaurants appear to be healthier and have higher cognitive scores compared to those residing further away, which contradicts the time-series evidence presented in Figure 1. On the other hand, this might simply reflect sorting along the socioeconomic dimensions documented in Panel C, with the children of more affluent parents having more favorable outcomes. These somewhat contradictory descriptive patterns are therefore likely to reflect the endogeneity of both family and firm choices with respect to geographical location, therefore motivating the need for our quasi-experimental design.

3 Empirical Approach

We are interested in estimating the effect of access to fast food restaurants on the health and ability of young Norwegian males. To overcome the potential endogeneity issues discussed above, we utilize a two-way fixed effects identification strategy.[15] As such, we exploit the quasi-random variation in the opening of fast food restaurants across different narrow geographical locations in Norway and over time. We estimate the following reduced-form equation:

$$Y_{ipt} = \alpha + \gamma \text{Years of exposure}_{pt} + \beta X_{ipt} + \lambda_p + \theta_t + \varepsilon_{ipt} \quad (1)$$

where Y_{ipt} are the outcomes of interest for individual i born in postcode p in year t; Years of exposure$_{pt}$ measures the number of years an individual would have been exposed to a fast food restaurant based on their postcode of birth; X_{ipt} is a set of individual and family controls (mother's education, mother's income, mother's age and marital status at birth, father's education, father's income, father's age at child's birth, the individual's birth order) as well as postcode-specific characteristics (number of years of access to a supermarket or a grocery store and number of years of access to a convenience store); λ is a set of postcode of birth fixed effects to control for time-invariant location characteristics; and θ

[15] Several previous papers have employed an instrumental variable strategy using the distance to the nearest highway (from the place of residence or school) as an instrument for access to fast food. In the context of Norway, where the highway network is very limited and only a small percentage of fast food restaurants are located close to major highways, this estimation strategy is not appropriate.

is a set of birth cohort fixed effects to control for common time specific shocks.[16] In our baseline empirical sample, we include all individuals within a radius of 30 km from a fast food restaurant based on the centroid of their postcode of birth.[17] We cluster the standard errors at the municipality of birth.[18]

The coefficient of interest, γ, is the per-year effect of proximity to a fast food restaurant on the outcome variables of interest. It is identified by variation in the time and location of restaurants' openings under two assumptions. First, that these events are not perfectly correlated with other unobserved determinants of health and cognition. To ensure this, we have studied a variety of reforms and laws implemented over the time period in question that may have affected our outcomes, but we did not find that these are correlated with the establishment of fast food restaurants.[19] Second, we assume that the treated locations would have had the same health and cognitive outcomes as nearby control locations in the absence of the treatment. In Section 4.1, we provide suggestive evidence that this parallel trends assumption is likely to hold.

Under these two assumptions, γ can be interpreted as a causal intention-to-treat (ITT) effect of proximity to a fast food restaurant in the postcode of birth. First, the treatment is defined for all individuals born in a specific year and location, but not all of these individuals would have regularly dined at these restaurants. In our data, we cannot observe individual-level consumption, which would have allowed us to compute the treatment-on-the-treated effects of fast food. Our reduced-form estimates should therefore be viewed through the lens of easier access to fast food rather than its direct consumption. We nonetheless believe that there is a strong first-stage relationship between the presence of restaurants and the demand

[16] Postcode-specific characteristics included in X_{ipt}, such as indicators for supermarkets, are measured at the same proximity as Years of exposure$_{pt}$. Thus, if we define treatment at a 1 km radius, we code it in the same way for both fast food restaurants and other food suppliers included in X_{ipt}. We cannot observe the size of a supermarket in our data to further stratify these into "regular" vs. "mega" (e.g., Walmart or ICA Maxi) types.

[17] We test both the sensitivity of our choice of proximity to a fast food restaurant (treatment group definition) in Appendix Figure A4 and the choice of radius for inclusion in the empirical sample in Appendix Figure A5.

[18] Although larger than a postcode, municipalities are the smallest governmental units in Norway and they are largely responsible for the planning of local business development. We cluster standard errors at these larger units to allow for potential correlation across postcodes within a municipality. There are 430 municipalities in our empirical sample. Our conclusions are unchanged if we cluster standard errors at the postcode level.

[19] These reforms include extensions of the maternity leave from 18 to 24 weeks in three stages for individuals born after May 1, 1987, June 1, 1988, and April 1, 1989; a school choice reform in Oslo in 1997, which affected cohorts born after 1981 in Oslo; and childcare subsidies for low-income households. The maternity leave reforms had no effect on children's school outcomes (Dahl et al., 2016) or mothers' health outcomes (Bütikofer et al., 2021), and there is no difference in proximity to fast food restaurants among families that were treated or not treated by these changes. The school choice reform had no effects on student outcomes but lowered house prices to some extent (Machin and Salvanes, 2016). In one of the robustness checks, we directly control for exposure to these reforms and our results remain unchanged. The families of children born in the latter half of our sample had access to means-tested childcare subsidies, an intervention that was shown to increase student performance (Black et al., 2014). Since fast food restaurants are more likely to open in more affluent postcodes (Table 1), if anything, this reform could bias our estimates towards zero. Given the similarity of the estimate with and without control variables, however, we are not concerned that this is a major confounder.

for fast food since these suppliers would otherwise go out of business. In stark contrast, the profits of fast food restaurants in Norway have increased over time (Moe, 2019; Foss, 2011). Prior literature also supports this assumption (Moore et al., 2009; Svastisalee et al., 2012). Second, to avoid endogenous sorting on factors correlated with the treatment, we assign the treatment at the time and place of residence at birth rather than using a contemporaneous place of residence, which could potentially be endogenous.

We also estimate an event study model to verify the parallel trends assumption:

$$Y_{ipt} = \alpha + \sum_{s=-7, s\neq -1}^{20} \gamma_s 1(y - F_p^* = s) + \lambda_p + \theta_t + \varepsilon_{ipt} \tag{2}$$

where Y_{ipt} are the outcomes of interest for individual i born in postcode p in year t, while y is the year when an individual undergoes military service screening and thus when our health and cognitive ability outcomes are measured. In this equation, s indicates the time periods relative to exposure, while the regressors of interest are dummy variables defined by $1(y - F_p^* = s)$. These dummy variables take on a value of one for each event time s. Our reference period, $s = -1$, is the opening of a fast food restaurant in close proximity to the postcode of birth one year after an individual underwent military screening. The event study plots omit $s = -7$ and $s = 20$, which are binned endpoints.[20] Event times $-6 \leq s \leq -2$ denote pre-trends relative to $s = -1$, while event times $0 \leq s \leq 19$ are treatment effects where greater values imply both longer exposure and exposure at an earlier age. Thus, one caveat with the interpretation of our results is that we cannot differentiate between the time of exposure and length of exposure as is common in studies relying on cohort variation. Spatially, akin to the preferred specification based on Equation 1, we define treated individuals as those whose centroid of the postcode of birth is within 2 km of a fast food restaurant, while individuals at a distance of 2–30 km are considered a control group.[21]

[20] Our results are invariant to not binning the endpoints and reporting estimates for $-6 \leq s \leq 19$. When we use academic track as an outcome, the highest value of s we consider is 16 since this is the age at which children decide on their high school tracks. We do not include control variables in the event study for ease of interpretation and to conform with recent recommendations in the literature (Sun and Abraham, 2021). The results are almost identical if we include the vector of controls used in Equation 1.

[21] Our treatment of interest is the number of years exposed to a fast food restaurant at close proximity, which is a discrete variable taking values between 0 and 19. It represents cumulative exposure to fast food from birth until the outcome measurement, which is similar to e.g., Hollingsworth et al. (2022) who study the effects of cumulative exposure to lead by specific grades on test scores. Given the structure of our treatment variable, we are unable to implement any of the modern difference-in-differences designs proposed by Borusyak and Jaravel (2017); De Chaisemartin and d'Haultfoeuille (2020); Goodman-Bacon (2021); Callaway and Sant'Anna (2021); Sun and Abraham (2021); Athey and Imbens (2022), as these methods require binary treatment variables taking the value of one in the first year of treatment and all subsequent years. Since our outcomes are measured at a single point in time (at ages 18/19 or at age 16) while our exposure/treatment is measured over multiple years, we cannot straightforwardly convert our setting to a single binary treatment variable. When we use a binary treatment variable – defined as any exposure year between the age of 0 and 19 – the estimates become less precise and we lose statistical significance in select specifications. We also re-estimated our preferred models dividing the data into two time periods: "early" and "late" openings delineated by year 1995 which is roughly a mid-point of our exposure timeline. For log BMI the effects are larger for the later (point estimate of 0.251 with a standard error of 0.074) compared with the earlier period (point estimate of 0.130 with a standard error of 0.048) while the reverse is true for

Before presenting the main results, we first investigate to what extent the location of fast food restaurants is correlated with observable characteristics. We already know, based on the discussion of Table 1 in Section 2.3, that there is some degree of spatial sorting and that more affluent individuals are more likely to live in close proximity to fast food suppliers, presumably because they can afford housing in the urban core. In Table 2, we formalize this conjecture through regression analysis. In particular, we correlate pre-determined postcode characteristics (Columns 2 and 3) or changes in these pre-determined characteristics (Column 4 and 5) with indicators for the postcode centroid being within 2 km of a fast food restaurant opening before 1980 (Columns 2 and 4) or before 1985 (Columns 3 and 5). First and foremost – and critical to our identification strategy – fast food restaurants do not appear to locate in either places where the local population has a higher BMI or in places where BMI has been increasing prior to the opening. In other words, the treatment does not appear to be correlated with lagged values of one of our outcomes of interest, hinting that the parallel trends assumption is likely to hold (as we confirm in Section 4.1). Second, this analysis confirms aforementioned anecdotal evidence that fast food retailers in Norway tend to concentrate in cities and the urban core – we find positive and statistically significant associations with population, age, and education. Third, the imbalances, to the extent they exist, appear quantitatively small. Even the larger coefficients from Column 3 imply sorting of between 0.001% (statistically insignificant for BMI) and 2.6% (statistically insignificant for rural indicator), while the statistically significant coefficients are in the range of 0.02% to 1.1%.[22]

In Appendix Table A2, we further probe the relationship between neighborhood characteristics and our treatment of interest. In line with Pei et al. (2019), we regress postcode-level average characteristics on an indicator for a postcode having a fast food restaurant in the first year an individual in our sample can be treated and all years thereafter (Column 2), and on the postcode centroid being within 2 km of a fast food restaurant in the first year an individual in our sample can be treated and all years thereafter (Column 4).[23] An observation in these regressions is postcode of birth-by-birth year cell, and we include both postcode of birth and birth year fixed effects. We do not find large or consistently signed coefficients in this analysis – only 3 out of 16 estimates are statistically significant at conventional levels, while 14 out of 16 imply an imbalance of less than 5% compared to the pre-treatment mean. Overall, we conclude that, considering the net of the known factors determining the location of food suppliers (or retailers more broadly), there is limited evidence of sorting that could invalidate our quasi-experimental two-way fixed effects design. Since the balance is not perfect, however, we further probe the sensitivity of our results in Sections 4.1 and 4.2 and find that the results are robust.

cognitive ability (estimates of -0.734 (0.303) and -0.375 (0.345) for early and late periods, respectively). This is consistent with event studies presented in Figure 2 which is the best evidence we can offer in support of the identifying assumptions.

[22]Coefficients in Columns 4 and 5 should not be compared to the means presented in Column 1 since they are based on regressions where changes in characteristics are used as outcomes. Given that a change in population is reflected in 1000s of individuals, these point estimates are likewise reasonably small.

[23]For example, if a fast food restaurant opened in a given postcode in 2003, we assign 0 to birth cohorts 1980–1984 and 1 to birth cohorts 1985–1989. This is because, for example in 2001, individuals aged 17 and older could still be treated by the opening. In our sample, we observe 2,328 postcodes for birth cohorts 1980–1989 and exposure years 1980–2007 as in the main analysis.

4 Results

4.1 Main Results

Table 3 presents estimates of γ from Equation 1 for a 0.5 km (Columns 1 and 4), 1 km (Columns 2 and 5) and 2 km (Columns 3 and 6) radii around the closest fast food restaurant (approximately 0.31, 0.62, and 1.24 miles, respectively). The treatment variable – the number of years exposed to a fast food restaurant based on postcode and time of birth – varies from 0 (for never exposed) to 19 (for always exposed). The coefficients, therefore, represent the effect of an additional year of exposure to a fast food restaurant on log BMI and the probability of being overweight (Panel A), as well as cognitive ability and the probability of being in an academic track in high school (Panel B).[24]

Focusing on the health outcomes first, we find that close proximity to fast food restaurants increases both BMI and the probability of being overweight at ages 18–19. The point estimates increase somewhat when we expand the radius but are generally statistically indistinguishable from each other and all are statistically significant at conventional levels. The point estimate of 0.198 implies that an additional year of exposure increases BMI by approximately 0.2%. Given that the average exposure in the data is slightly over 7 years, this coefficient translates to an effect size of 1.4%.[25] We find larger effects for the upper-tail outcome – the probability of being overweight – at 1.6% per year relative to the pre-treatment sample mean, or more than an 11% increase for the average number of years that individuals are exposed in our sample. These effect sizes are meaningful given that the average BMI and overweight rate in the last cohort in the sample (1989) were 4 and 30% higher compared with the 1980 cohort, respectively. Thus, using this back-of-the-envelope calculation, it appears that we can attribute a non-trivial fraction (approximately one-third) of the increase in weight observed across cohorts in Norway to exposure to fast food restaurants.

Although we cannot observe individual level fast food consumption and thus we interpret our estimates as an intent-to-treat, we also consider the minimum detectable effects we could expect based on what we know regarding the consumption patterns and a caloric value of the fast food meals. In order to gain 1 kg of additional weight individuals need to eat about 7000 kcal extra while a Big Mac Meal – the most sold and popular fast food product in Norway – contains 1120 kcal according to McDonald's (McDonald's, 2023). On the other hand, the recommended dinner time caloric intake for young males is only 400-700 calories (Folkhälsoguiden, 2023). Bugge Bahr (2023) reports that approximately 60% of Norwegian 15-24 year olds eat "American fast food" at least once per month. Therefore, given our estimates we would expect a weight gain of $0.6 \times 12 \times 0.06 \times 7.26 = 3.14$ kg on top of the pre-treatment mean of 74.64 kg, while our estimates in Appendix Table A3 imply increase in weight of 1.04 kg due to average exposure of the treated group to fast food.[26] This

[24] As noted above, our treatment variable varies from 0 (for never exposed) to 16 (for always exposed) when we consider high school academic track as an outcome.

[25] We include never-treated individuals in this calculation. Conditional on ever being exposed to a fast food restaurant at 2 km before age 19, the average number of years of exposure is 13.5, which would yield an effect size of about 2.7%.

[26] Specifically we multiply 0.6 (fraction of youth consuming at least one meal a month) times 12 months times 0.06 (excess kilograms for eating a single Big Mac Meal rather than a recommended caloric intake: $\frac{1120-700}{7000}$ kcal) times 7.26 (average number of years exposed in the full sample). The gain of 1.04 kg comes

conservative approach, as Bugge Bahr (2023) only reports extensive margin consumption, would account for about a third of our treatment effect. On the other hand, if we are willing to assume that at least some of the 60% of youth eat fast food as often as three times a month, then this would match almost exactly with our preferred estimates. Thus, we view the results in Panel A of Table 3 as non-negligible and consistent with fast food exposure effects rather than other parallel changes in unhealthy behaviors.

Our estimates confirm prior findings from the US on the negative health consequences of exposure to fast food restaurants at either place of residence (Qian et al., 2017) or school (Currie et al., 2010), while extending them to Norway – a country with not only substantially lower obesity rates but also lower fast food access and socioeconomic inequalities. Qian et al. (2017) find that an additional fast food restaurant within half a mile of a child's place of residence increases their BMI z-score by 7.9% of a standard deviation. This is very similar to our estimates, which – reflected in standard deviations as presented in Appendix Table A3 – would imply a 1.3% of an SD effect per year, or 9.4% of a SD for the average exposure time in our sample. Most other US studies focus on exposure at school rather than at place of residence. For example, Currie et al. (2010) find that a fast food restaurant within 0.1 miles of middle school increases the likelihood of a child being obese by at least 5.2%. In our setting, we chose to investigate the likelihood of being overweight rather than obese because our population is healthier, and even the former rate of 23% is lower than the average probability of being obese in the California data at almost 33%. When we estimate the effects on the obesity rate, at a 2 km radius, our effect size is a 2.9% higher probability per year of exposure (Appendix Table A3). Likewise, Davis and Carpenter (2009) find that Californian students whose schools are within 0.5 miles of a fast food restaurant have increased probabilities of being overweight and obese by 6 and 7%, respectively. In Arkansas, Alviola IV et al. (2014) find an increase in obesity rates of 5.8% per additional restaurant within a 1 mile radius of a school.[27] Overall, we view our results as largely consistent with a number of previous studies from the US, which found adverse health effects of exposure to fast food restaurants, despite a very different institutional, healthcare, economic, and cultural settings.

Prior research suggests that proper childhood nutrition could affect education and test scores (see, e.g., Anderson et al., 2018; Bütikofer et al., 2018; Lundborg et al., 2022; Gertler and Gracner, 2022), while some epidemiological studies have attempted to directly link obesity and intelligence (see, e.g., Yu et al., 2010; Belsky et al., 2013). Thus, going beyond health outcomes, we also investigate whether access to fast food restaurants affects cognitive ability. The first three columns in Panel B of Table 3 suggest reductions in cognitive ability of up to 0.56 percent of a standard deviation per one additional year of exposure to a fast food restaurant, or a 0.041 SD for the average number of years of exposure. Strikingly, these estimates are very close to the effects found from contracting with healthy meal vendors in California, where children's test scores increased by a 0.03 to 0.04 SD (Anderson et al., 2018).[28] We likewise find negative effects on the probability of academic track enrollment.

from multiplying our treatment effect of 0.143 (Column 4 of Appendix Table A3) by 7.26.

[27]Since our postcodes are quite small, there is rarely more than one fast food restaurant per postcode. In fact, only 6% of the postcodes in our sample have more than one fast food vendor. Thus, in Norway, we cannot analyze intensive margin treatment akin to Alviola IV et al. (2014).

[28]Considering unstandardized scores as an outcome (Appendix Table A3), we find a point estimate of

The point estimate for our preferred radius of 2 km implies a reduction in academic track enrollment of 0.5% per year of exposure, or 3.0% for the average exposure. We view these cognitive and schooling effects as quantitatively meaningful and as important from a policy perspective since they show that an increased supply of fast food could also lead to lower cognition beyond its negative health effects.[29]

The validity and interpretation of the aforementioned effects critically depend on both parallel trends and absence of correlated shocks assumptions. In Figure 2, we therefore provide event study estimates based on Equation 2 where we define the treated group using the 2 km cutoff. The top row of this figure focuses on health outcomes (Panels A and B) while the bottom row presents event studies for cognitive ability (Panel C) and high school academic track attendance (Panel D). Irrespective of the outcome, we do not find any evidence of pre-trends. Out of the 20 estimated pre-treatment coefficients, none are statistically significant at conventional levels, and for no outcome we can detect statistically significant pre-trends. At the same time, for the health outcomes, we find clearly increasing treatment effects in the post-periods, implying both higher BMI and increased probability of being overweight. Both effects grow with the length (and age) of exposure. The cognitive effects are somewhat more muted, but we still estimate statistically significant post-opening treatment effects that are consistent with the results presented in Table 3. Importantly, from the perspective of our heterogeneity analysis, results in Panels C and D suggest that cognitive and schooling effects are concentrated among those who were exposed for a longer period of time and at a younger age.

4.2 Robustness Checks

The results presented in Table 3 imply that boys growing up in close proximity to fast food restaurants have a higher BMI and lower cognitive ability in young adulthood. In Figure 2, we have shown that these effects are not driven by differential pre-trends, which is our main testable identifying assumption. In this section, we present a multitude of additional robustness checks. In particular, we ensure that our results are unaffected by the choice of estimation sample, the definition of treatment, econometric specification, and transformation of the dependent variables. We also present the results from a randomization inference test.

First, Table 4 presents a variety of alternative specifications for our preferred exposure radius of 2 km, with each outcome in a separate panel. Column (1) replicates our main results from Table 3 to ease the comparisons. Column (2) drops all control variables and estimates the model with only postcode and cohort fixed effects. To the extent that our variation is quasi-random, the inclusion of additional controls should not substantially affect the results. Column (3) excludes Oslo, the capital, which is a major population and commerce center. In

-0.010, which based on pre-treatment mean yields an effect size of 0.2% per year or 1.45% for an average exposure. Since the decline in cognitive ability in our empirical sample between 1980 and 1989 birth cohorts is 0.28 points or 5.4%, our estimate could account for about 27% of this decline.

[29]Controlling for log BMI in Appendix Table A4 does not affect the effects on ability or the probability of attending an academic high school track despite a negative and statistically significant association between log BMI and either of these outcomes. Coefficients on log BMI are -0.076 (p-value < 0.001) and -0.102 (p-value < 0.001) when we consider cognitive ability scores and academic track probability as outcomes, respectively. This means that the effects on cognitive outcomes are largely orthogonal (and plausibly additive) to any effects that could have operated through access to fast food restaurants increasing BMI.

fact, almost 10% of the population in Norway resided in the city during the 1980s, while 18% of fast food restaurant openings in our sample are observed in Oslo. Column (4) addresses a concern that regional trends simultaneously driving the supply of fast food restaurants, unhealthy behaviors, and economic activity could be driving the observed increases in BMI. We do this by controlling for linear municipality-specific time trends.[30] Column (5) addresses concerns related to the measurement of BMI and cognitive ability at different ages. These outcomes are measured at age 18 for 73% of the sample and age 19 for 25% of the sample, and as both outcomes might change with age, we control for age at measurement.[31] Column (6) directly addresses the concern of reforms that might be co-timed with the expansion in the supply of fast food restaurants, as discussed in Section 3. At the individual level, we therefore control for the two aforementioned reforms: (a) maternity leave and (b) increased school choice within the municipality of Oslo.[32] In Column (7), we control for exposure at high school level, which for those students who are able to choose which school they attend, could be different from exposure at their place of residence. Finally, in Column (8), we exclude single-site restaurants, which we have coded based on a search of phrases such as "pizza", "hamburger", and "kebab" among establishments with industry codes 56.101 and 56.301. Since this was done manually, our concern here is that those outlets could have more measurement error. Irrespective of the exact specification, the coefficients remain largely unchanged both in terms of magnitude and statistical significance, thus supporting the robustness of our preferred specification.

Second, we conduct a randomization inference test where we randomly allocate fast food restaurant openings from our sample at the postcode and cohort level. We repeat this exercise 1,000 times for each outcome and plot the resulting coefficient distribution together with our preferred estimates at a 2 km radius from Table 3. This exercise addresses both the possibility of spurious correlations and provides empirically driven alternative p-values for exact sharp nulls (Young, 2019). Appendix Figure A3 presents these results for our four outcomes of interest. Here, the vertical black line denotes the preferred coefficients from Table 3, the gray shaded area depicts the 95% confidence intervals around these coefficients, and the orange areas present the distributions from the estimates when we randomly assign exposure to fast food restaurants. In all cases, the placebo distributions are bell-shaped and centered around zero as expected if there was indeed no sorting or spurious correlations. Furthermore, the black vertical lines are always outside of the simulated distributions, implying empirical p-values of less than 0.001. Taking a more conservative view, the distributions for BMI, the probability of being overweight, and cognitive ability do not even overlap with 95% confidence intervals of our preferred estimates.

Third, we vary the distance definitions of both the treatment group and the inclusion in

[30] We do not control for post-code specific time trends as this is geographic unit close to the definition of our treatment. See Meer and West (2016) for a discussion on why inclusion of such time trends is econometrically problematic.

[31] When considering academic track as an outcome, we control for the age at which students enter high school since 3% do not start immediately after completing their compulsory education at age 16.

[32] We do not have information about the low-income childcare subsidies at the individual level, but address this by controlling for parental income. A comparison of Columns (1) and (2), with and without additional controls (including parental income), suggests that these do not materially affect our results – if anything, the results with controls are on the conservative side.

the empirical sample. Our preferred estimates define treatment at a 2 km proximity to a fast food restaurant, and we include all individuals between 2 and 30 km from the restaurant as a control group. In Table 3, we have already documented that the results are not sensitive to smaller radii. In Appendix Figure A4, we further document that they are stable when we increase the radius up to around 5–8 km, but at larger distances, the effect fades out and becomes statistically insignificant; except for cognitive ability. This is consistent with the declining likelihood of (frequently) using a restaurant that is further away from one's place of residence. Thus, we posit that distance and consumption are inversely related, which would give us the pattern of results depicted in Appendix Figure A4. Another concern is that we include either too few or too many individuals in our empirical sample, which is then divided into treatment and control groups. In Appendix Figure A5, we present the results where we vary the inclusion cutoff from 5 to 100 km while keeping the definition of the treatment group at a 2 km radius from a fast food outlet.[33] To the extent that individuals beyond a 30 km radius should never be affected by any restaurant openings we consider when defining treatment, our results should not change, and this is precisely what the estimates in Appendix Figure A5 imply.

Fourth, there could be a concern that our results are downward biased due to spillovers. It is clear that access to fast food restaurants does not change discretely at a 2 km radius and individuals included in the control group – e.g., between a 2 and 3 km radius – are thus also likely to be treated to some degree. To address this issue, we re-estimated our main results while defining the treatment group as in Table 3 at 0–2 km, and the control group as those between 5 and 30 km, while dropping individuals in the "donut" between the treatment and control group cutoffs. Panel B of Appendix Table A5 presents these results, which are substantively unchanged, ensuring that our main results are not downward biased due to meaningful spatial spillovers. This is also consistent with evidence from Appendix Figure A4, where we observe relatively constant effects up to the 5 km definition of treatment.

Fifth, we verify that our results are not driven by how we define the outcome variables measuring health and cognition. Appendix Table A3 presents the results for alternative dependent variables: BMI (Column 1), BMI z-score (Column 2), probability of being obese (Column 3), weight in kilograms (Column 4), height in centimeters (Column 5), and raw cognitive ability score (Column 6). These are in contrast to using the log BMI, probability of being overweight, and standardized cognitive ability score in Table 3. Irrespective of the outcome, we always find that access to fast food restaurants positively affects proxies for increased weight. Column 5 further shows that the BMI effects are not moderated by parallel increases in height. If anything, this point estimate is negative. This is interesting given the literature linking nutrition and height (see, e.g., Grasgruber et al., 2014) as well as nutrition and educational achievement (see, e.g., Bütikofer et al., 2018; Lundborg et al., 2022). We do acknowledge, however, that the estimate is not very precise and we cannot rule out negative effects as large as 0.4 cm for an average exposure to fast food restaurants. This potential cumulative negative effects is about two-thirds of the positive height effect found in Lundborg et al. (2022) and certainly consistent with fast food restaurants decreasing average nutritional value of consumed meals. Likewise, we find negative effects on the unstandardized

[33]This means that at 5 km, the treatment group is 0–2 km and the control group is 2–5 km, while at 100 km, the treatment group is 0–2 km and the control group is 2–100 km.

measure of cognitive ability. We therefore conclude that our results are not sensitive to how we proxy for lower health and cognitive ability.

Sixth, we assign treatment based on the postcode of residence at birth, which we view as the most exogenous location from the perspective of potentially treated children. This renders our estimates as an intend-to-treat effect and removes any potential endogeneity due to migration. As noted before, we are not concerned with households sorting based on access to fast food but rather with sorting on characteristics correlated with access to fast food, such as urbanicity. To ensure that our results are not driven by this choice, we focus on non-movers only in Panel C of Appendix Table A5. In particular, we restrict the sample to individuals with the same postcode between (1) birth and age 6 (first panel), (2) birth and age 12 (second panel), and (3) birth and age 18 (third panel), which is approximately when we measure the outcomes in the military registers. Regardless of the exact age cutoff, our point estimates are largely similar. We lose statistical significance in a few specifications, but this is mostly due to inflated standard errors and much smaller sample sizes rather than point estimates converging to zero. We conclude that our conservative approach of assigning exposure at the postcode of birth is not driving the results.

Our final robustness check verifies that the results are not driven by always treated individuals. This could be a concern if these people are different from the overall population given that their parents chose to live in a location that already had a fast food outlet nearby prior to the child's birth (rendering even our assignment of treatment based on the postcode of residence at birth potentially endogenous). Panel D of Appendix Table A5 presents the results when excluding always-treated individuals from the sample. If anything, these coefficients are somewhat larger than our preferred point estimates. This makes sense if always exposed individuals are less sensitive to marginal changes in the fast food supply. We conclude that including always-treated individuals in our sample does not represent a major empirical concern in relation to the results.

Overall, we consider our preferred estimates to be remarkably robust across a multitude of estimation and sample permutations. Thus, we consider the coefficients in Table 3 to reliably represent the causal effects of proximity to a fast food restaurant on young adult men's health and cognitive ability.

4.3 Heterogeneity

The effects of access to fast food on obesity can vary across a wide range of demographic characteristics including gender, race, and socioeconomic background (see, e.g., Currie et al., 2010). In this section, we therefore analyze whether the effects documented in Table 3 differ by socioeconomic background (proxied by the father's education and employment status of the parents), urbanicity, health endowments at birth, and the father's BMI in early adulthood.[34] In each case, we execute the heterogeneity analysis through a model with interactions, meaning that we expand Equation 1 to include the number of years of exposure within 2 km of a fast food restaurant, a variable of interest in the heterogeneity analysis (either indicator or continuous), and an interaction between those two variables. The

[34]Because our outcomes are limited to men, we cannot study differences by gender. Furthermore, Norway has insufficient racial diversity to investigate this dimension.

interaction term documents whether our treatment effect is different across the heterogeneity dimension in question. We do not include any auxiliary control variables beyond birth postcode and birth year fixed effects in this analysis.

Allcott et al. (2019) suggest that there are differences in the demand for healthy food between poor and rich households. We therefore study whether children from a low socioeconomic background are more affected by their proximity to fast food restaurants than children with richer parents.[35] Panels A and B of Table 5 stratify the sample by paternal education and parental employment.[36] We do not find any sizable, statistically significant, or consistently signed differences in our treatment effects across these groups when we consider health outcomes. This is despite the fact that overweight rates are 34% higher in families where the father has no high school or only vocational high school education, compared with families in which the father has at least an academic high school education. On the other hand, for both cognitive outcomes, there is a clear mediation of the fast food restaurants' effect by paternal education. In households where fathers do not have an academic high school degree, sons have 0.67% of an SD lower cognitive ability and 0.36 percentage points lower probability of choosing an academic track in high school per year of exposure to a fast food restaurant. These penalties are reduced to only 0.34% of an SD and 0.19 percentage points in families where the father has an academic high school degree, corresponding to about 50%.[37]

Since fast food restaurants in Norway tend to locate in cities and urban areas, we also investigate differential effects of being born in the top 10 biggest cities in Norway (Panel C). These children could potentially have even easier access to fast food due to either a higher density of suppliers or more efficient public transport. We do not find any statistically significant differences in this dimension.[38] Interestingly, individuals living in big cities have lower BMI and higher cognitive outcomes, which makes sense given that much of the country's white-collar economic activity is concentrated in these areas.

Another question is whether exposure to fast food restaurants matters differentially for younger vs. older children. On the one hand, at younger ages, parents arguably have more control over what their children eat. Thus, we view early age exposure as primarily driven by parental choices while exposure during teenagehood is driven by both child and parental choices – and perhaps to a larger degree by the former than the latter. On the other hand, to the extent that parents allow fast food in their children's diet, this could be more consequential for a rapidly developing human body and brain in early childhood.

[35] Unlike in the US, fast food in the Norwegian context might be thought of as more of a luxury good because it is often more expensive than relatively accessible unprocessed food. Therefore, it is also plausible that the effects could be more pronounced in higher-SES families that can more easily afford such consumption.

[36] Another channel through which parental employment could affect obesity is traditional gender norms where a stay-at-home mother might do the cooking for the family, thereby limiting the reliance on food consumption outside of the household.

[37] We also examined the effects of exposure to fast food restaurants by quintiles of household income. The point estimates are larger in the bottom quintiles but we lack statistical power to reject the equality of coefficients across quitiles. For this reason we prefer reporting stratification by education and parental employment which are binary variables.

[38] Statistically insignificant interaction terms in Panel C further alleviate the concern that a 2 km radius could facilitate differential consumption patterns in larger vs. smaller areas.

Appendix Table A6 presents these results.[39] As regards health outcomes, the estimates are not statistically different across the two age groups but they do suggest somewhat larger effects from exposure in teenagehood when the youth can make more independent nutritional choices. When it comes to cognitive ability, the effects are only significant in the early years.[40] This is consistent with evidence on critical periods and brain development (Heckman, 2007; Berg et al., 2014), as well as with the notion that proper nutrition might matter, especially for young children (Anderson et al., 2018; Bütikofer et al., 2018; Lundborg et al., 2022). These results are also broadly consistent with the event studies presented in Figure 2.

Returning to Table 5, we now move to two novel heterogeneity analyses. Thanks to population-level registry data, we can ask whether the effects of access to fast food restaurants are mediated by prenatal health endowments as well as by the intergenerational propensity for elevated BMI. First, since in utero health and nutrition (often proxied by birth weight) have long-term consequences (Black et al., 2007; Figlio et al., 2014), individuals with poorer prenatal health might be particularly vulnerable to changes in later life nutrition. Using birth weight as a marker of neonatal health, we analyze whether pre-determined health endowments are compensated or reinforced by subsequent negative nutritional shocks. Panel D of Table 5 shows that children who were born with a higher birth weight have a higher BMI and improved cognitive ability. However, we do not find any statistically significant or sizable interaction between birth weight and access to fast food restaurants.[41]

Second, we ask whether the consequences of easier access to fast food are different for males whose fathers had a high BMI (Panel E). For a subset of our sample (62 percent), we know the BMI of the fathers at the age of 18/19 from their military records (the same data source we use to measure our outcomes for young adults), and can therefore use this information about predispositions for being overweight.[42] Although the father's BMI at the age of 18/19 is a very good predictor of their son's BMI (positive), as well as their cognition and schooling (negative), we do not find any statistically significant interactions between access to fast food restaurants and paternal BMI. If anything, some of the coefficients have the opposite sign to what we had expected. However, quantitatively speaking, these differences are small compared to both estimates of the father's BMI and the years of exposure.

[39] In this case, we do not use interaction terms but instead split the exposure variable from Equation 1 into two exposure variables: number of years of exposure at ages 0–12 and number of years of exposure at ages 13–19. We acknowledge the limitation that children exposed at younger ages are also likely exposed at older ages and we cannot therefore differentiate age at exposure from the length of exposure.

[40] Again, controlling for log BMI in Appendix Table A4 does not alter the results on ability. Our takeaway from this exercise is that the decline in cognitive ability cannot be explained by the orthogonal increase in BMI.

[41] We have also verified that this result is not driven by our method of measuring prenatal health. Using an indicator for low birth weight, likewise, does not yield any sizeable or statistically significant interaction terms. The fact that higher birth weight children have a higher BMI is in contrast with some prior research suggesting that lower prenatal health leads to increased BMI (Gluckman et al., 2007).

[42] To keep the sample constant, we opted to run the heterogeneity analysis on the full sample. We additionally control for an indicator of missing data on the father's BMI, as well as its interaction with the number of years of exposure to fast food restaurants (not shown). Results are similar when we restrict the sample to those with available data on the father's BMI, although the standard errors increase. Furthermore, our preferred estimates for the effects of the number of years of exposure to fast food restaurants in this sub-sample are similar to those reported in Table 3.

5 Conclusions

Our findings suggest that in a country like Norway where healthier food options are both available and affordable, the negative causal effects of access to high-caloric nutrition provided by fast food restaurants are still present and could have contributed to the increase in obesity rates among adolescents in recent decades. We further document that increased access to this type of food, which likely also leads to increased consumption, could have negative effects on cognitive ability. Back-of-the-envelope calculations suggest that increased penetration of fast food suppliers could be responsible for as much as 35% of the increase in average BMI and 27% of the decrease in average cognitive ability for the cohorts in our sample. These are much larger shares than that documented in the US with respect to Walmart Supercenters (Courtemanche and Carden, 2011) and fast food exposure in middle school (Currie et al., 2010), which may not be that surprising given lower average BMI levels, limited other obesogenic factors, and easier access to healthy substitutes in Norway. In contrast to some previous studies, we do not find meaningful heterogeneity in these effects in our setting when considering health outcomes. For cognitive outcomes, we find differences by age at exposure and by the father's education. Our results are generally robust to alternative specifications and support the identifying assumptions.

Nevertheless, given that our point estimates can explain much less than a half of the increases in BMI and declines in cognitive ability observed in Figure 1, it is worth asking what other factors could be contributing to these trends. One potential contributor to obesity and related chronic diseases has been the significant shift to unhealthy diets at home, in particular with respect to calories from sugar, refined carbohydrates, and fat (see, e.g., Cutler et al., 2003). Yet another factor could be an increased sedentary lifestyle. Focusing on adults, Griffith et al. (2016) suggest that the increase in obesity is to a lesser degree due to an increase in calories consumed and is rather caused by the decline in the strenuousness of work and daily life. Moreover, Aguiar et al. (2021) demonstrate using time-use data that since 2004, younger men (21–30 years) in the US have shifted their leisure activities to video gaming and other recreational computer activities, and conclude that innovations in these leisure activities explain an important share of the decline in the labor market activity of younger men relative to older men. Over 70 percent of Norwegian adolescents spend more than three hours each day in front of a TV, computer, or smartphone screen outside of school (Bakken, 2022). Hence, a shift in leisure activities among children from more active forms of recreation to video gaming/social media might also contribute to the increase in BMI.

Notwithstanding these additional channels, which are plausibly responsible for the remaining share of the increase in BMI, we view our results as an important addition to the literature on the effects of fast food supply on health, particularly the health of young adults. We also complement this literature by documenting adverse effects on cognition that appear to be largely orthogonal to the health effects. Furthermore, we note that fast food regulation might be more policy-actionable and effective than attempts at altering consumption behaviors at home or exercise habits (Leonard, 2017; Griffith, 2022; Xiang et al., 2022). Indeed, some governments are currently looking into imposing stricter regulations on both advertising and the location of fast food outlets.

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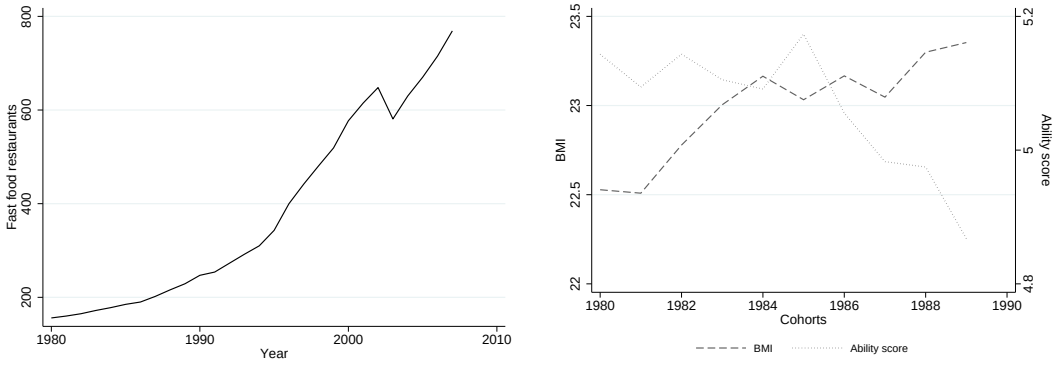
6 Figures and Tables

Figure 1: Trends in Number of Fast Food Restaurants, BMI, and Cognitive Ability

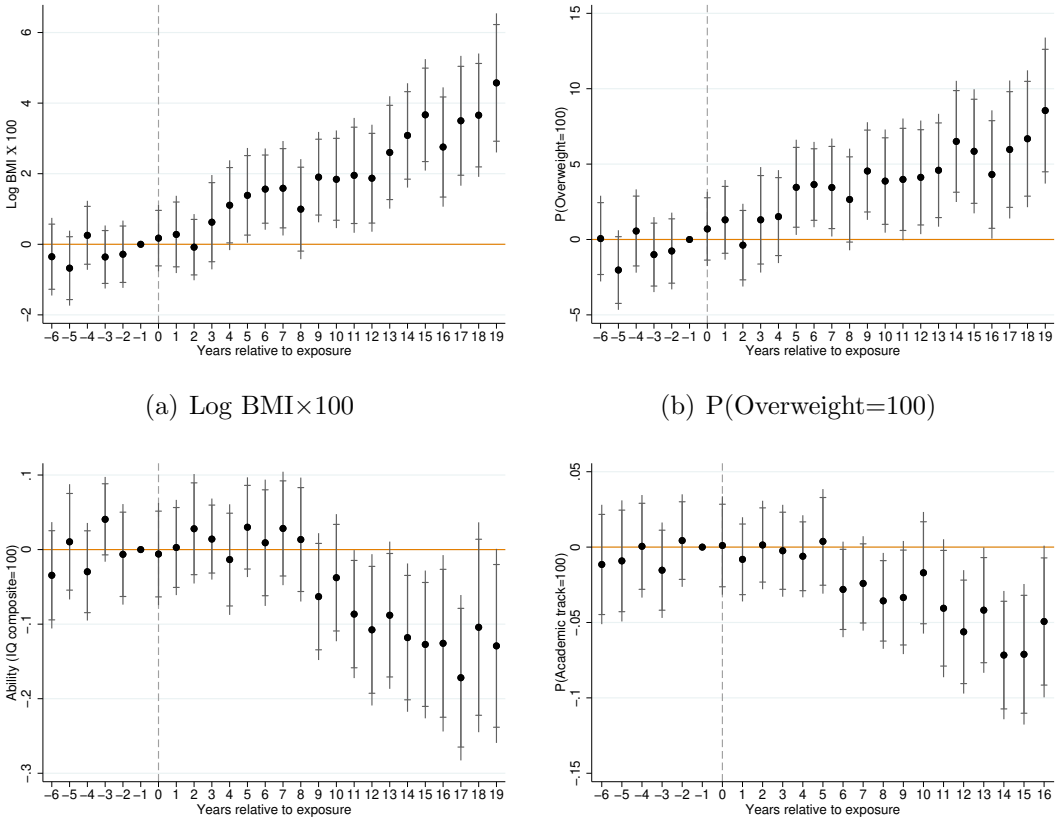


(a) Fast food restaurants

(b) BMI and cognitive ability

Notes: Panel A of this figure documents the increase in the number of fast food restaurants in the period 1980–2007. Panel B of this figure documents trends in Body Mass Index (BMI) and cognitive ability (IQ) scores of Norwegian male recruits by birth cohort (1980–1989) for those born in a postcode that at some point between 1980 and 2007 had a fast food restaurant within a 30 km radius of the postcode centroid.

Figure 2: Event Study Estimates of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes



(a) Log BMI×100

(b) P(Overweight=100)

(c) Ability(IQ composites)×100

(d) P(Academic track=100)

Notes: Each figure is from a separate regression based on Equation 2. We denote 95% confidence intervals with spikes and 90% confidence intervals with whiskers. The treatment group is defined as a 0–2 km distance while the control group is defined as a 2–30 km distance. The sample includes individuals born 1980–1989 who at some point between 1980 and 2007 had access to at least one fast food restaurant within a 30 km radius from the centroid of the postcode of their place of residence at birth. All specifications include a full set of birth postcode and birth year fixed effects and no additional control variables. Standard errors are clustered at the municipality of birth.

Table 1: Descriptive Statistics

		All males within 30km radius		
	Full sample (1)	Estimating sample (2)	Ever treated (3)	Never treated (4)
	Panel A: Outcome variables			
Log BMI×100	312.23	312.03	311.77	312.34
	[15.67]	[15.61]	[15.53]	[15.70]
BMI	22.99	22.94	22.88	23.02
	[3.90]	[3.87]	[3.84]	[3.91]
P(Overweight=100)	22.09	21.67	21.14	22.30
	[41.48]	[41.20]	[40.83]	[41.63]
Standardized ability×100	0.00	0.67	4.83	-4.19
	[100.00]	[99.96]	[100.47]	[99.14]
Raw ability score	5.09	5.09	5.15	5.01
	[1.72]	[1.72]	[1.73]	[1.71]
P(Academic track=100)	51.74	52.12	54.91	48.87
	[49.97]	[49.96]	[49.76]	[49.99]
	Panel B: Treatment variable			
Years exposed to fast food restaurants	6.41	7.26	13.48	0.00
	[7.98]	[8.12]	[6.21]	[0.00]
Years exposed to fast food restaurants before starting high school	5.04	5.71	10.60	0.00
	[6.74]	[6.91]	[6.06]	[0.00]
	Panel C: Individual level characteristics			
Birth order	1.84	1.82	1.76	1.90
	[0.94]	[0.93]	[0.89]	[0.96]
Mother's age at birth	26.66	26.68	26.75	26.60
	[5.00]	[4.96]	[4.89]	[5.05]
Father's age at birth	29.32	29.50	29.49	29.50
	[6.13]	[5.54]	[5.48]	[5.61]
Parents married at birth	0.78	0.79	0.77	0.80
	[0.42]	[0.41]	[0.42]	[0.40]
Years of education mother	12.43	12.45	12.62	12.25
	[2.42]	[2.43]	[2.48]	[2.34]
Years of education father	12.81	12.85	12.62	12.59
	[2.70]	[2.71]	[2.48]	[2.58]
Mother's income/1000	112.33	112.76	118.61	105.95
	[74.06]	[74.87]	[79.00]	[69.13]
Father's income/1000	243.52	247.63	258.97	234.41
	[169.72]	[175.25]	[206.12]	[129.13]
Observations	177790	156699	84421	72278

Notes: The sample is based on all males from birth cohorts 1980–1989 with valid military assessment outcomes. Panel A presents the means of outcomes, Panel B the means of treatment variables, and Panel C the means of individual-level characteristics. Standard deviations in square brackets. Column (1) presents values for all males while Columns (2)–(4) present values for all males with a birth postcode centroid within a 30 km radius of a fast food restaurant opened between 1980 and 2007. Column (3) displays the means for individuals exposed to a fast food restaurant at some point within 2 km of their place of residence at birth. Column (4) displays the means for individuals never exposed to a fast food restaurant within 2 km of their place of residence at birth.

Table 2: Postcode-Level Correlates of the Opening of Fast Food Restaurants

		1977 postcode characteristics		Changes in postcode characteristics	
	Mean in 1977	Opening before 1980	Opening before 1985	Opening before 1980	Opening before 1985
	(1)	(2)	(3)	(4)	(5)
Log BMI × 100	311.21	0.000	0.001	0.001	0.003
		(0.001)	(0.002)	(0.003)	(0.004)
Age	30.86	0.003*	0.006***	0.039**	0.043**
		(0.002)	(0.002)	(0.016)	(0.018)
Population	1.48	0.012***	0.017***	0.329***	0.485***
		(0.002)	(0.003)	(0.064)	(0.072)
Education	10.33	0.042***	0.062***	-0.071	0.028
		(0.013)	(0.014)	(0.066)	(0.084)
Log income	10.53	-0.056	-0.100*	0.070	0.011
		(0.047)	(0.051)	(0.071)	(0.077)
Rural	0.19	-0.005	-0.005	0.006	0.002
		(0.006)	(0.009)	(0.005)	(0.006)
R^2		0.064	0.109	0.053	0.081
Observations		1898	1898	1898	1898

Notes: Each column represents a separate linear probability regression model of the likelihood of a fast food restaurant opening before 1980 (Columns 2 and 4) or before 1985 (Columns 3 and 5) within 2 km of the postcode's centroid in relation to postcode characteristics measured in 1977 (Columns 2 and 3) or changes in these characteristics between 1977 and 1979 (Columns 4 and 5). Population is given in thousands, log income is the log of average income in NOK thousands, education is years of education, and rural is a dummy for whether the postcode is situated in a municipality counted as rural or not. Standard errors are clustered at the municipality level. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table 3: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes

	≤0.5km (1)	≤1km (2)	≤2km (3)	≤0.5km (4)	≤1km (5)	≤2km (6)
	Panel A: Health outcomes					
	Log BMI×100			P(Overweight=100)		
Years exposed	0.128**	0.181***	0.198***	0.246**	0.280***	0.350***
	(0.053)	(0.046)	(0.041)	(0.124)	(0.103)	(0.100)
Observations	156699	156699	156699	156699	156699	156699
Pre-treatment mean	312.14	312.21	312.34	21.91	22.05	22.31
	Panel B: Ability and education outcomes					
	Ability (IQ composites)×100			P(Academic track=100)		
Years exposed	-0.393	-0.528**	-0.558**	-0.325**	-0.359***	-0.256**
	(0.282)	(0.243)	(0.223)	(0.148)	(0.136)	(0.127)
Observations	142434	142434	142434	156561	156561	156561
Pre-treatment mean	-1.59	-2.71	-4.19	50.86	50.25	49.24

Notes: Each point estimate is from a separate regression of an outcome variable on number of years of exposure to a fast food restaurant at a given distance from the centroid of the postcode of an individual's place of residence at birth. Outcomes are: log BMI (Columns 1 to 3 of Panel A), probability of being overweight (Columns 4 to 6 of Panel A), standardized cognitive ability scores (Columns 1 to 3 of Panel B), and probability of enrolling in an academic track in high school (Columns 4 to 6 of Panel B). All outcome variables are multiplied by 100. We consider three distances when defining the treatment group: $\leq$ 0.5 km (Columns 1 and 4), $\leq$ 1 km (Columns 2 and 5), and $\leq$ 2 km (Columns 3 and 6). The sample includes individuals born 1980–1989 who at some point between 1980 and 2007 had access to at least one fast food restaurant within a 30 km radius of the centroid of the postcode of their place of residence at birth. All specifications include a full set of birth postcode fixed effects and birth year fixed effects. Each regression controls for years of exposure to grocery stores and convenience stores within the indicated distance. Additional control variables include: mother's education, mother's income, mother's age at birth, parents' marital status at birth, father's education, father's income, father's age at birth, and the individual's birth order. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table 4: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Sensitivity Analyses

	Main estimates (1)	No controls (2)	Excluding Oslo (3)	Municipality specific time trend (4)	Controlling for age at measurement (5)	Other reforms (6)	Controlling for exposure at high school (7)	Dropping single site restaurants (8)
Log BMI×100	0.198***	0.202***	0.181***	0.203***	0.198***	0.187***	0.197***	0.163***
	(0.041)	(0.039)	(0.042)	(0.041)	(0.041)	(0.040)	(0.041)	(0.045)
Observations	156699	156699	148933	156699	156699	156699	156699	134183
P(Overweight=100)	0.350***	0.396***	0.322***	0.358***	0.348***	0.327***	0.347***	0.321***
	(0.100)	(0.095)	(0.107)	(0.101)	(0.100)	(0.100)	(0.100)	(0.115)
Observations	156699	156699	148933	156699	156699	156699	156699	134183
Ability (IQ composites)×100	-0.558**	-0.824***	-0.594**	-0.545**	-0.560**	-0.548**	-0.534**	-0.748***
	(0.223)	(0.248)	(0.234)	(0.221)	(0.223)	(0.222)	(0.226)	(0.275)
Observations	142434	142434	135310	142434	142434	142434	142434	142434
P(Academic track=100)	-0.256**	-0.434***	-0.227*	-0.257**	-0.244*	-0.221*		-0.405***
	(0.127)	(0.127)	(0.132)	(0.128)	(0.126)	(0.124)		(0.144)
Observations	156561	156561	148815	156561	156561	156561		134056

Notes: Each point estimate is from a separate regression of an outcome variable on the number of years of exposure to a fast food restaurant at a 2 km distance from the centroid of the postcode of an individual's place of residence at birth. Each outcome is in a separate row: log BMI (Row 1), probability of being overweight (Row 2), standardized cognitive ability scores (Row 3), and probability of enrolling in an academic track in high school (Row 4). All outcome variables are multiplied by 100. Column 1 replicates preferred specifications from Table 3. Column 2 drops all control variables except for birth postcode and birth year fixed effects. Column 3 drops Oslo municipality. Column 4 includes municipality-specific linear time trends. Column 5 controls for age at measurement of the outcome. Column 6 controls for maternity leave and Oslo school choice reforms at individual level. Column 7 controls for exposure to fast food restaurants at the attended high school address (this is not available for high school track since this choice is made prior to attendance). Column 8 drops all the hand-coded single-site restaurants from industry codes 56.101 and 56.301. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

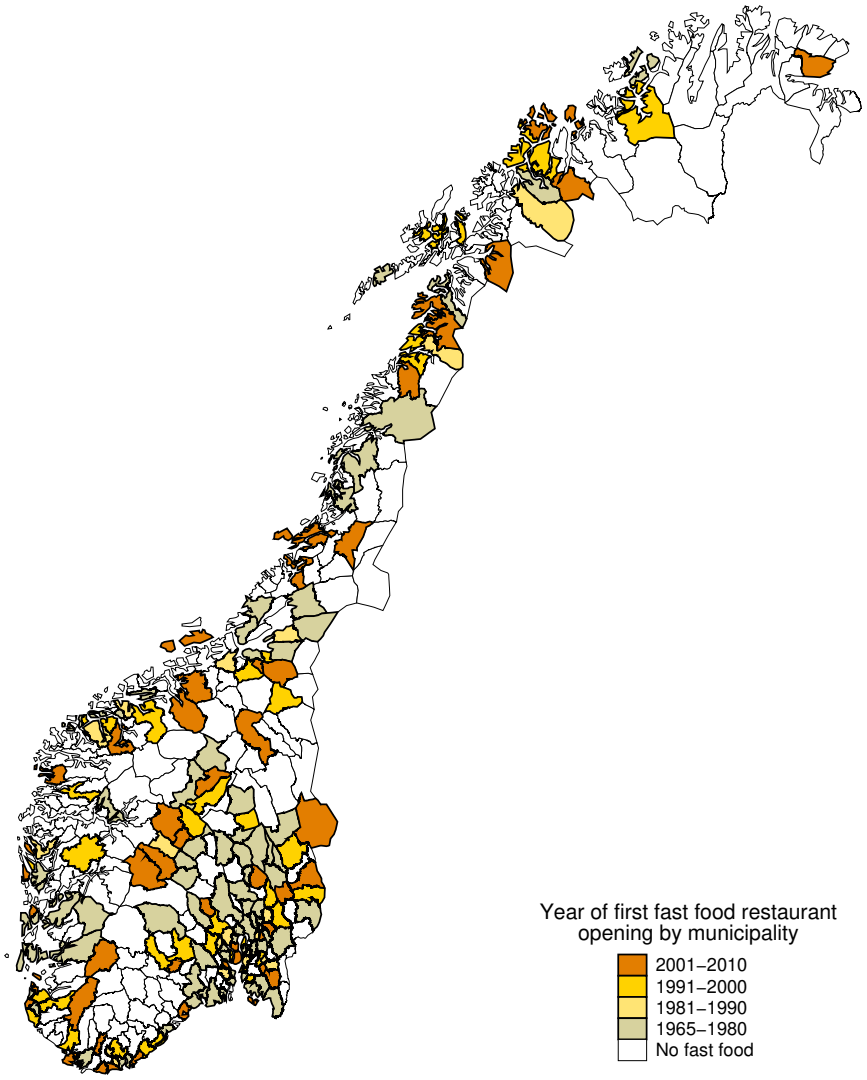
Table 5: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Heterogeneity

	Log BMI×100 (1)	P(Overweight=100) (2)	Ability (IQ composites×100) (3)	P(Academic track=100) (4)
	Panel A: Father's education			
Years exposed	0.197***	0.331***	-0.667***	-0.361***
	(0.041)	(0.101)	(0.245)	(0.133)
Years exposed × father	-0.005	0.010	0.325***	0.167***
academic high school	(0.012)	(0.028)	(0.087)	(0.048)
Father academic high school	-1.965***	-5.318***	52.527***	29.201***
	(0.180)	(0.456)	(1.183)	(0.684)
Observations	156699	156699	142434	156561
	Panel B: Full time working parents			
Years exposed	0.199***	0.348***	-0.719***	-0.406***
	(0.042)	(0.103)	(0.243)	(0.129)
Years exposed ×	0.018	0.065	-0.021	-0.014
full time working parents	(0.016)	(0.040)	(0.090)	(0.048)
Full time working parents	-0.150	-1.113	27.154***	15.698***
	(0.271)	(0.683)	(1.727)	(0.884)
Observations	156699	156699	142434	156561
	Panel C: 10 biggest cities			
Years exposed	0.169***	0.291***	-0.482*	-0.210
	(0.040)	(0.100)	(0.256)	(0.137)
Years exposed × city	0.032	0.032	-0.270	-0.097
	(0.019)	(0.052)	(0.204)	(0.104)
City	-1.634*	-4.729**	19.047***	15.241***
	(0.910)	(2.285)	(3.228)	(3.662)
Observations	156699	156699	142434	156561
	Panel D: Birth weight			
Years exposed	0.210***	0.385***	-1.116***	-0.533***
	(0.050)	(0.137)	(0.333)	(0.205)
Years exposed ×	-0.000	-0.000	0.000	0.000
birth weight	(0.000)	(0.000)	(0.000)	(0.000)
Birth weight	0.002***	0.003***	0.014***	0.005***
	(0.000)	(0.001)	(0.001)	(0.001)
Observations	156699	156699	142434	156561
	Panel E: Father's log BMI			
Years exposed	0.464**	0.855*	-0.715	-0.690
	(0.212)	(0.502)	(1.200)	(0.688)
Years exposed ×	-0.093	-0.180	-0.024	0.078
father's log BMI	(0.067)	(0.161)	(0.385)	(0.216)
Father's log BMI	40.564***	76.345***	-22.948***	-12.141***
	(0.728)	(1.789)	(4.913)	(1.914)
Observations	156699	156699	142434	156561

Notes: Each column in each panel is from a separate regression of an outcome variable on the number of years of exposure to a fast food restaurant at a 2 km distance from the centroid of the postcode of an individual's place of residence at birth, the heterogeneity dimension variable considered, and the interaction between these two variables. The heterogeneity variables include: father's academic education indicator (Panel A), indicator for both parents working (Panel B), indicator for birth in the 10 biggest Norwegian cities (Panel C), birth weight in grams (Panel D), and father's log BMI (Panel E). The specifications are otherwise akin to those in Table 3 but exclude all the control variables except for birth postcode and birth year fixed effects. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

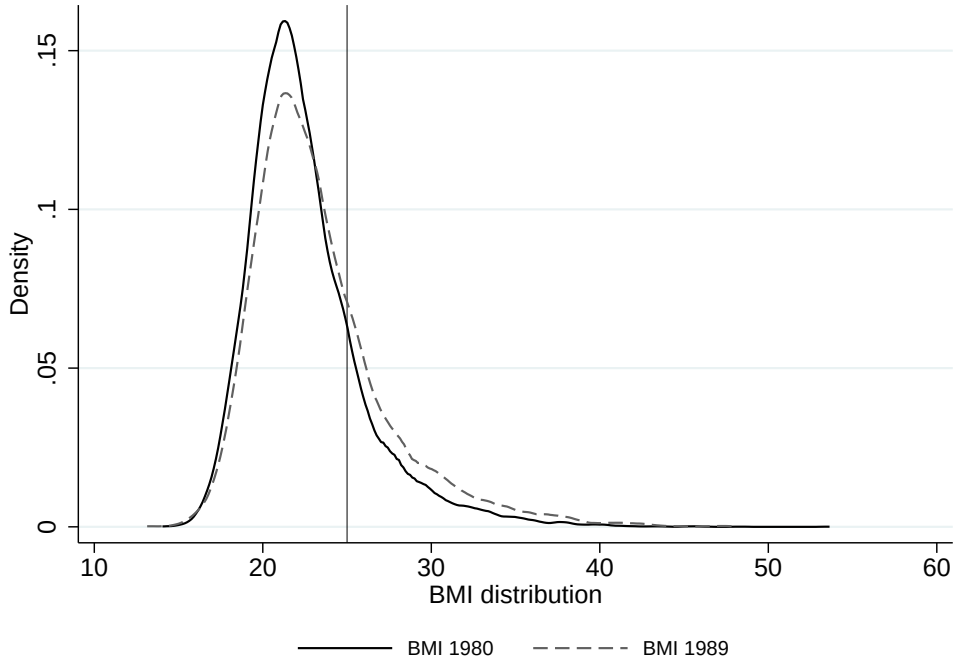
For Online Publication: Online Appendix

Figure A1: Openings of Fast Food Restaurants Openings over Time and by Municipality



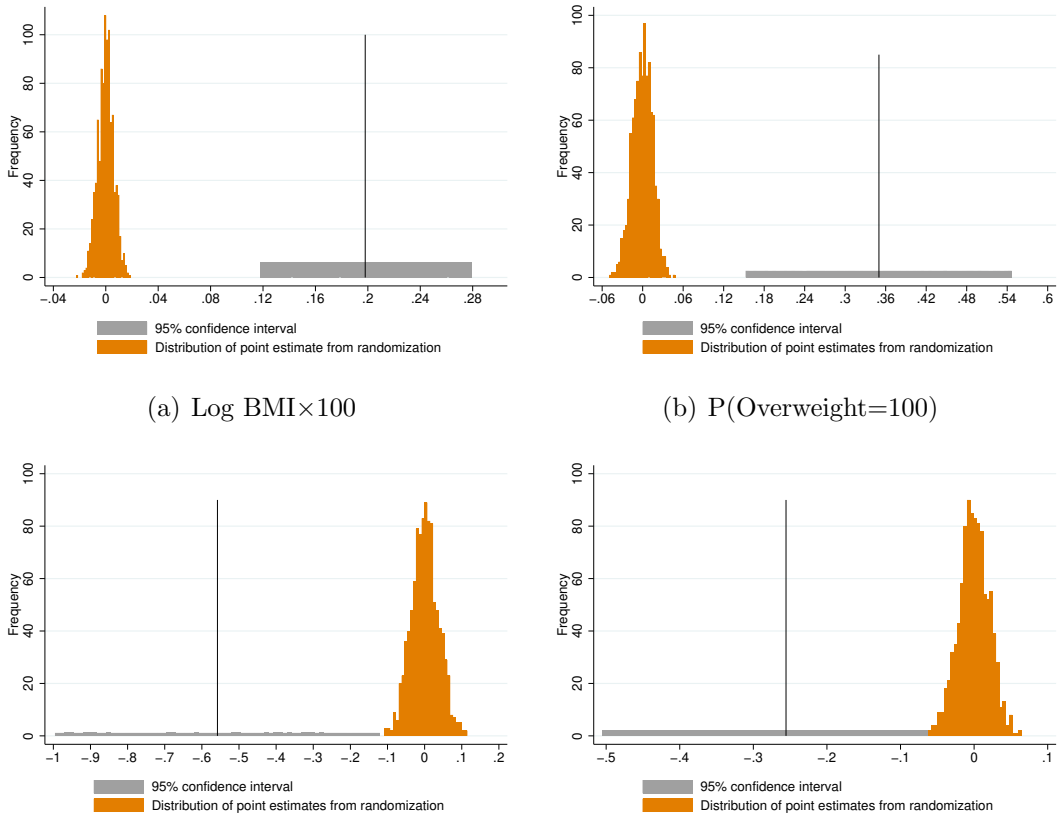
Notes: The map displays Norway's 428 municipalities. The different colors indicate when the first fast food restaurant opened in these municipalities. There are no fast food restaurants in the white municipalities during the period of interest.

Figure A2: Distribution of BMI by Birth Cohort



Notes: The figure plots the distribution of BMI for men born in 1980 and 1989 in a postcode that at some point between 1980 and 2007 had a fast food restaurant within a 30 km radius of the centroid of the postcode of an individual's place of residence at birth. The vertical line marks the threshold for overweight (BMI>25).

Figure A3: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Randomization Inference Analysis



(a) Log BMI×100

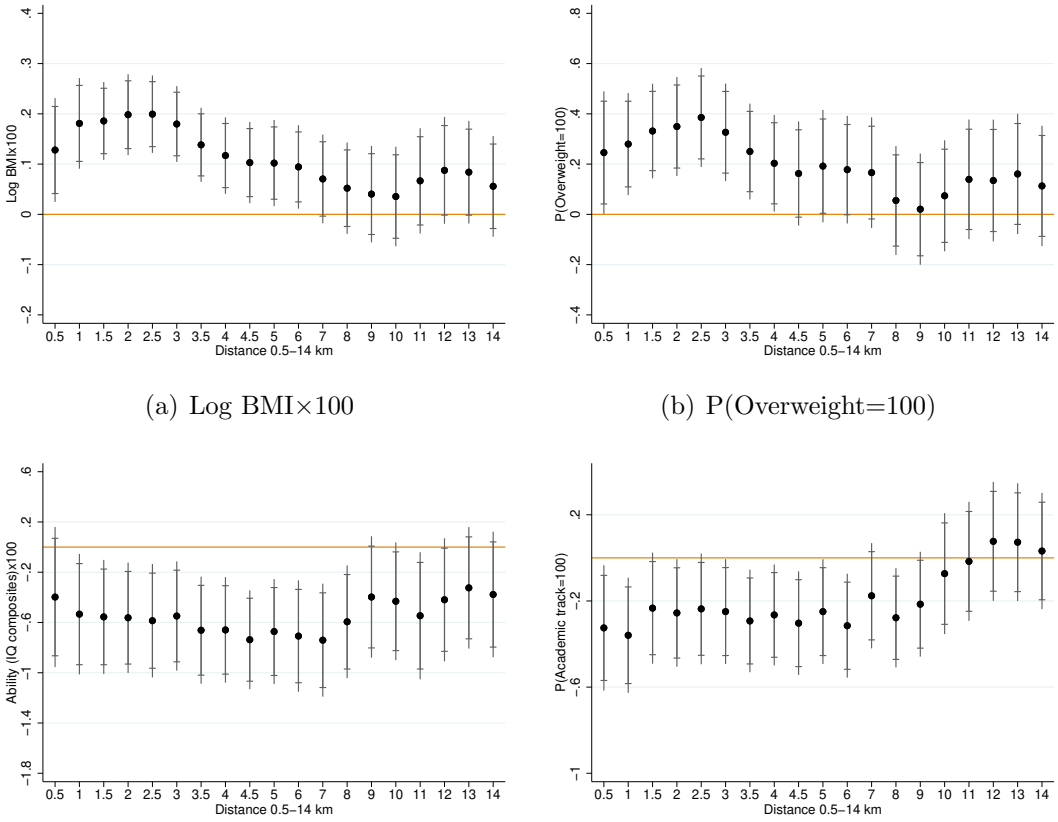
(b) P(Overweight=100)

(c) Ability(IQ composites)×100

(d) P(Academic track=100)

Notes: The figure shows the distributions (orange histograms) of 1,000 coefficients from a randomization test for log BMI (Panel A), probability of being overweight (Panel B), cognitive ability (Panel C), and probability of enrolling in an academic high school track (Panel D). All outcome variables are multiplied by 100. In order to generate the randomization inference distributions of estimates, we randomly allocate postcodes of restaurant openings while holding the number of fast food outlet openings in each year identical to the one observed in the true data. We then compute the number of years exposed given these randomly allocated treatments and re-estimate our preferred specification from Table 3. The vertical black line depicts our preferred estimates from Table 3 while the gray shaded areas indicate 95% confidence intervals around these estimates.

Figure A4: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Varying Definition of Treatment Group Radius



(a) Log BMI×100

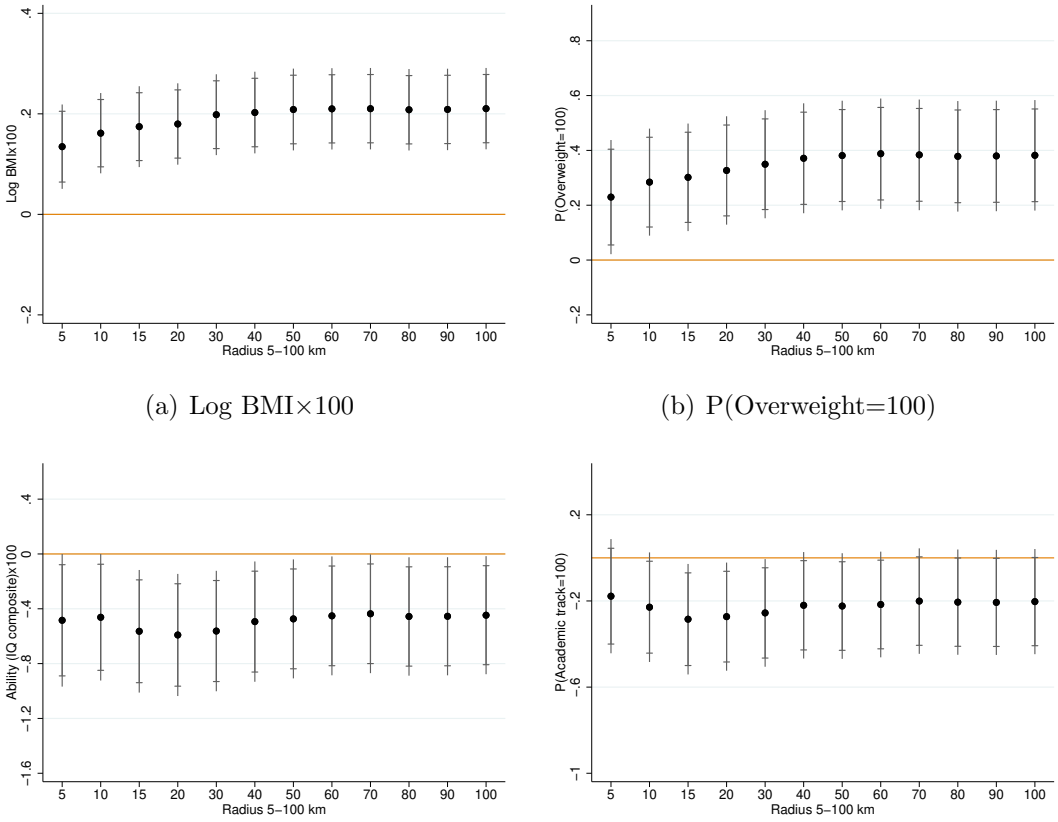
(b) P(Overweight=100)

(c) Ability(IQ composites)×100

(d) P(Academic track=100)

Notes: Each panel of this figure shows robustness checks where we vary the radius defining the treatment group. Outcomes are: log BMI (Panel A), probability of being overweight (Panel B), cognitive ability (Panel C), and probability of enrolling in an academic high school track (Panel D). All outcome variables are multiplied by 100. Each point estimate (circles) and 95% (spikes) and 90% (whiskers) confidence intervals come from a separate regression. The sample includes individuals born 1980–1989 who at some point between 1980 and 2007 had access to at least one fast food restaurant within a 30 km radius of the centroid of the postcode of their place of residence at birth. The definition of treatment group radius varies every 0.5 km from 0.5 km to 5 km, and every 1 km from 5 to 14 km. In each case, the control group is the complement distance up to 30 km. Each regression controls separately for years of exposure to grocery stores and convenience stores within the indicated distance. Additional control variables are mother's education, mother's income, mother's age at birth, parents' marital status at birth, father's education, father's income, father's age at birth, and the individual's birth order. The horizontal orange line denotes zero. Standard errors are clustered at the municipality of birth.

Figure A5: Effect of Proximity to Fast Food Restaurants at 2 km on Health, Ability, and Education Outcomes: Varying Radius for Inclusion in Empirical Sample



(a) Log BMI×100

(b) P(Overweight=100)

(c) Ability(IQ composites)×100

(d) P(Academic track=100)

Notes: Each panel in this figure shows robustness checks where we vary the radius defining inclusion in the analysis sample. Each point estimate is from a separate regression of an outcome variable on the number of years of exposure to a fast food restaurant at a 2 km distance from the centroid of the postcode of an individual's place of residence at birth. Circles denote point estimates, spikes denote 95%, while whiskers denote 90% confidence intervals. Outcomes are: log BMI (Panel A), probability of being overweight (Panel B), cognitive ability (Panel C), and probability of enrolling in an academic high school track (Panel D). The samples include individuals born 1980–1989 who at some point between 1980 and 2007 had access to at least one fast food restaurant within a 5–100km radius of the centroid of the postcode of their place of residence at birth. All specifications include a full set of birth postcode fixed effects and birth year fixed effects. Each regression controls separately for years of exposure to grocery stores and convenience stores at a 2 km distance. Additional control variables are mother's education, mother's income, mother's age at birth, parents' marital status at birth, father's education, father's income, father's age at birth, and the individual's birth order. Standard errors are clustered at the municipality of birth.

Table A1: Intergenerational Transmission of BMI and Likelihood of Being Overweight

	Log BMI×100 (1)	P(Overweight=100) (2)
	Panel A: Without control variables	
Log BMI father×100	0.399***	
	(0.007)	
P(Father overweight=100)		0.217***
		(0.007)
Mean father	306.92	7.45
Mean son	312.35	22.34
Observations	121847	121847
	Panel B: With control variables	
Log BMI father×100	0.396***	
	(0.007)	
P(Father overweight=100)		0.217***
		(0.007)
Observations	121847	121847
	Panel C: With control variables and 30km radius	
Log BMI father×100	0.400***	
	(0.006)	
P(Father overweight=100)		0.211***
		(0.007)
Mean father	306.92	7.51
Mean son	312.03	21.67
Observations	97166	97166

Notes: Each point estimate comes from a separate regression of the son's BMI on paternal BMI (Column 1) or the son's likelihood of being overweight on paternal likelihood of being overweight (Column 2). Panel A presents univariable associations, Panel B adds father, mother, and child's control variables, and Panel C limits the sample to households where the son's birth postcode was within 30 km of the opening of a fast food restaurant between 1980 and 2007. All regressions include 1980–1989 birth cohorts for sons and 1950-1972 birth cohorts for fathers. Additional control variables are mother's education, mother's income, mother's age at birth, parents' marital status at birth, father's education, father's income, father's age at birth, and the individual's birth order. The means of the father's and son's BMI, as well as ther probability of being overweight, are defined as means in the empirical samples. Standard errors are clustered at the son's municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table A2: The Effect of the Establishment of Fast Food Restaurants on Postcode Characteristics

	Pre-treatment mean	Treated	Pre-treatment mean	Treated 2km
	(1)	(2)	(3)	(4)
Family size	2.77	0.091	2.82	0.018
		(0.102)		(0.034)
Observations		14577		14577
Mother's age	26.87	1.217***	26.72	0.052
		(0.452)		(0.171)
Observations		14577		14577
Father's age	29.64	0.528	29.57	0.322
		(0.820)		(0.204)
Observations		14577		14577
Married parents	0.76	-0.002	0.77	-0.016
		(0.060)		(0.015)
Observations		14577		14577
Mother's education	11.96	0.230	11.80	-0.161
		(0.186)		(0.120)
Observations		14577		14577
Father's education	12.05	0.087	11.89	-0.225*
		(0.241)		(0.119)
Observations		14577		14577
Mother's income	117378	979.709	109011.3	946.665
		(7313.229)		(3027.298)
Observations		14577		14577
Father's income	244885.8	-16326.974	230267.3	-12845.194***
		(15783.950)		(4428.408)
Observations		14577		14577

Notes: Each coefficient comes from a separate regression where the outcome variable is the postcode-by-cohort level average of a given characteristic. All regressions include birth postcode and birth year fixed effects and no additional controls. The treatment variable in Column (2) is an indicator for a fast food restaurant opening in the postcode of birth in years that could affect specific cohorts. The treatment variable in Column (4) is an indicator for the centroid of the postcode of birth being within 2 km of a fast food restaurant opening in years that could affect specific cohorts. These variables take the value of one for the first cohort where individuals might be treated by an opening and all subsequent cohorts. Standard errors are clustered at municipality level. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table A3: Effect of Proximity to Fast Food Restaurants on Health and Ability: Alternative Measures of Outcomes

	BMI	BMI z-score	P(Obesity=100)	Weight	Height	Ability
	(1)	(2)	(3)	(4)	(5)	(6)
Years exposed	0.049***	0.013***	0.173***	0.143***	-0.019	-0.010**
	(0.010)	(0.003)	(0.052)	(0.037)	(0.021)	(0.004)
Observations	156699	156699	156699	156699	156699	142434
Pre-treatment mean	23.02	0.01	6.04	74.64	179.97	5.00

Notes: Each point estimate comes from a separate regression. Samples and econometric specifications are based on Column 3 from Panel A of Table 3 for Columns 1 to 5, and are based on Column 3 of Panel B of Table 3 for Column 6. Dependent variables are BMI in levels (Column 1), standardized mean 0 and SD 1 BMI (Column 2), probability of being obese multiplied by 100 (Column 3), weight in kilograms (Column 4), height in centimeters (Column 5), and unstandardized ability scores from military records (Column 6). Obesity is defined as BMI > 30. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table A4: Effect of Proximity to Fast Food Restaurants on Ability and Education Outcomes: Controlling for Log BMI

	Ability (IQ composites)×100 (1)	P(Academic track=100) (2)
	Panel A: Main estimates	
Years exposed	-0.548**	-0.236*
	(0.223)	(0.127)
Observations	142434	156561
	Panel B: Age heterogeneity	
Years exposed 0-12	-0.958***	-0.282**
	(0.282)	(0.138)
Years exposed 13-19	0.096	
	(0.377)	
Observations	142434	156561

Notes: Point estimates in each panel and each column come from separate regressions. Panel A replicates results from Columns 3 and 6 of Panel B of Table 3 while controlling for the child's log BMI. Panel B replicates results from Panel B of Table A6 while controlling for the child's log BMI. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table A5: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Additional Sensitivity Analyses

	Log BMI×100 (1)	P(Overweight=100) (2)	Ability (IQ composites) ×100 (3)	P(Academic track=100) (4)
	Panel A: Main estimates			
Years exposed	0.198***	0.350***	-0.558**	-0.256**
	(0.041)	(0.100)	(0.223)	(0.127)
Observations	156699	156699	142434	156561
	Panel B: Control group residing in 2–5 km radius excluded			
Years exposed	0.205***	0.351***	-0.561**	-0.255**
	(0.041)	(0.101)	(0.224)	(0.128)
Observations	139674	139674	126942	139548
	Panel C: Non-movers different age cutoffs			
Age 6	0.238***	0.443***	-0.440	-0.403**
	(0.050)	(0.116)	(0.267)	(0.162)
Observations	100476	100476	91409	100407
Age 12	0.307***	0.526***	-0.593*	-0.262
	(0.062)	(0.151)	(0.305)	(0.173)
Observations	75679	75679	68746	75628
Age 18	0.262***	0.319	-0.795*	
	(0.080)	(0.197)	(0.441)	
Observation	49202	49202	44843	
	Panel D: Always treated excluded			
Years exposed	0.218***	0.366***	-0.791***	-0.412***
	(0.052)	(0.123)	(0.270)	(0.131)
Observations	120231	120231	109523	120140

Notes: Each point estimate is from a separate regression of an outcome variable on the number of years of exposure to a fast food restaurant at a 2 km distance from the centroid of the postcode of an individual's place of residence at birth. Each outcome is in a separate column: log BMI (Column 1), probability of being overweight (Column 2), standardized cognitive ability scores (Column 3), and probability of enrolling in an academic track in high school (Column 4). All outcome variables are multiplied by 100. Panel A replicates the results from Columns 3 and 6 of Panel A, and Columns 3 and 6 of Panel B of Table 3. Panel B drops a bandwidth between 2 and 5 km from the control group, creating a donut regression with the treatment group defined as 0–2km and control group defined as 5–30 km. Panel C limits the sample to children whose postcode of residence at birth and postcode of residence up to age 6 (Row 1), up to age 12 (Row 2), and up to age 18 (Row 3) are identical. Panel D drops individuals who were always exposed to a fast food restaurant based on their postcode of residence at birth. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.

Table A6: Effect of Proximity to Fast Food Restaurants on Health, Ability, and Education Outcomes: Heterogeneity by Age

	(1)	(2)
	Panel A: Health outcomes	
	Log BMI×100	P(Overweight=100)
Years exposed age 0-12	0.178***	0.261*
	(0.050)	(0.137)
Years exposed age 13-19	0.225***	0.531***
	(0.074)	(0.165)
Observations	156699	156699
P-value from F-statistic	0.625	0.256
	Panel B: Ability and education outcomes	
	Ability (IQ composites)×100	P(Academic track=100)
Years exposed age 0-12	-0.965***	-0.300**
	(0.282)	(0.138)
Years exposed age 13-19	0.079	
	(0.376)	
Observations	142434	156561
P-value from F-statistic	0.026	

Notes: Point estimates in each panel and each column come from separate regressions. Each regression (except for the academic track) divides the treatment into number of years of exposure to a fast food restaurant at ages 0–12 and at ages 13–19. Since we measure academic track selection at age 16, we limit this analysis (Column 2 of Panel B) to number of years of exposure to a fast food restaurant at ages 0–12. Except for this change, the econometric specification is the same as in Columns 3 and 6 of Panels A and B of Table 3. Standard errors are clustered at the municipality of birth. Significance levels: *** 1% level, ** 5% level, * 10% level.