Determinants of Health and Labor Market Outcomes

Three Essays in Applied Microeconomics

Anne-Lise Breivik

Thesis for the degree of Philosophiae Doctor (PhD)
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Abstract

This thesis consist of four chapters: One introductory chapter and three chapters that each includes the three papers that are the main part of this thesis. The introductory chapter provides an overview of the determinants health and labor market outcomes, both in a theoretical framework and an overview of the relevant empirical literature. The chapter also provides a discussion on the concept of causality, and the empirical methods used in the three papers, before it concludes with a discussion on the contribution of the thesis with an emphasis on the internal and external validity of the three studies.

The first paper studies the effects of experiencing that a child has a negative health shock on parents' health and labor market outcomes. This paper shows that parents experiencing that their child has a severe health shock between age 5 and 18, have significant and persistent reductions in labor income and employment, and increased use of social security benefits. The underlying health problems are psychological disorders. Heterogeneity analysis by age of the child and type of health shock reveals larger effects of health shocks related to injury, poisoning and other consequences of external causes, such as traumatic head injury, when the children are between age 5 and 12. The results are found using high-quality Norwegian register data and an event study approach.

The second paper examines the impact of increased access to universal childcare on adult health. The results show that affected women increase their use of pregnancy-related healthcare services and sickness absence. However, there is no increase in fertility and no effects on the second generation's birth outcomes, indicating that the women's health is unchanged, but that they have increased their demand for healthcare services. Second, there is a reduction in the use of mental healthcare services, and services related to injuries and social problems, pointing toward improved mental health. Finally, children of employed mothers are driving the effects.

The third paper studies the relationship between short-term air pollution increases and indicators of health and worker productivity. Estimating two-way fixed effects models using geographical and time variation in exposure to particulate matter (PM_{10}) and nitrogen dioxide (NO_2), we find that the number of GP consultations, certified sickness absences, and hospital visits increases in periods with high(er) pollution levels. There is substantial heterogeneity in this relationship. We find some support for previous results showing that pollution affects vulnerable groups like children and elderly negatively. Importantly, however, the largest effects are on school-age children and the working-age population.

Contents

Chapter 1:

Introduction

1 Introduction

In economics, the focus on health has increased substantially during the last decades. There has been an increased focus on empirically investigating how health is shaped throughout the lifecycle, and how this influences other aspects of life, such as educational attainment and labor force participation. There are at least two reasons for the increased focus on health. First, there are increased availability of comprehensive administrative register data on measures of health and health care use. In addition to reducing measurement error and capturing many outcomes, the use of administrative data can help mitigate problems of selective attrition from surveys and the large sample sizes contribute statistical power to detect milder treatments (Almond et al., 2018). Second, economists have long recognized a strong relationship between health and economic outcomes. Measures of health are positively associated with human capital, earnings, income, and wealth. However, the direction of causality in these relationships is unclear. On the one hand, better health can lead to higher productivity and less working time lost to illness, which further incentivizes human capital investment. On the other hand, higher productivity and financial resources can facilitate access to care, avoidance of harmful environmental factors, and access to higher-quality food and drugs (Stephens and Toohey, 2018). It is therefore important to study these associations in a causal setting to learn more about the direction of the causality in the relationships.

This thesis uses microeconometric tools, specifically different types of fixed effects models, and register data covering the entire Norwegian population from 1967–2014 to study how different events shape health and labor market outcomes throughout the lifecycle. A particular focus in the thesis is on the health of the middle-aged, working-age, population, which there has not been a great focus on in the existing literature. The existing literature has typically focused more on vulnerable groups, such as infants, children, and the elderly as the data on these subpopulations often has been more readily available. The thesis consists of three chapters, covering different aspects related to health and labor market outcomes over the lifecycle. The first chapter examines the causal effect of a child's negative health shock on parents' health and labor market outcomes. The second chapter examines the long-term effects of childcare on adult health outcomes. The third chapter examines the relationship between air pollution, health and productivity.

The introductory chapter proceeds as follows. Section 2 presents a theoretical framework on how health is shaped by different inputs. Section 3 reviews the empirical literature on the topics covered in the thesis. In Section 4, I discuss causality. Section 5 presents the methods used in the thesis. In Section 6, I conclude by discussing its contribution. In Section 7, I present summaries of the three papers included in the thesis.

2 Theoretical Framework

In health economics, the benchmark model is the Grossman model, which is a theoretical framework of the demand for the commodity "good health". The model is motivated by the fundamental difference between health as an output and medical care as one of the number of inputs into the production of health, and by the difference between health capital and other forms of human capital (Grossman, 2000).

According to traditional demand theory, each consumer has a utility function that allows him or her to rank alternative combinations of goods and services purchased in the market. Consumers are assumed to select the combination that maximizes their utility function subject to an income or resource constraint. While this theory provides a good explanation of the demand for many goods and services, this is not the case for the demand for medical services. This is because what consumers demand when they purchase medical services are not the services per se but rather better health (Grossman, 2000).

The Grossman model closely relates to human capital theory (Becker, 1964, 1967; Ben-Porath, 1967; Mincer, 1974). According to human capital theory, increases in a person's stock of knowledge or human capital raise his productivity in the market sector of the economy, in which he produces money earnings, and in the nonmarket or household sector, in which he produces commodities that enters his utility function. To realize potential gains in productivity, individuals have an incentive to invest in formal schooling and on-the-job training. The costs of these investments include direct expenses on market goods and the opportunity cost of the time that have competing uses. Becker (1967) and Ben-Porath (1967) used this framework to develop models that determine the optimal quantity of investment in human capital at any age and show how the optimal quantity varies over the lifecycle of an individual and among individuals of the same age (Grossman, 2000).

Although the human capital theory pointed out that health capital is one component of the stock of human capital, Grossman was the first to construct a model of the demand of health capital itself. The reason for this is that Grossman argues that health capital differs from other forms of human capital. His argument is that a person's stock of knowledge affects his market and nonmarket productivity, while his stock of health determines the total amount of time he can spend producing money earnings and commodities. If increases in the stock of health simply increased wage rates, the Grossman model would not be necessary, as one could use Becker's and Ben-Porath's models to study the decision to invest in health (Grossman, 2000). However, the Grossman model is now the benchmark model used to study health investment decisions.

The Grossman model uses the household production function model of consumer behavior to account for the gap between health as an output and medical care as one of many inputs into

its production. The model draws a distinction between fundamental objects of choice, called commodities, which enters the utility function, and market goods and services. Consumers produce commodities with inputs of market goods and services and their own time. Since goods and services are inputs into the production of commodities, the demand for these goods and services is a derived demand for a factor of production. That is, the demand for medical care and other health inputs is derived from the basic demand of health (Grossman, 2000).

There is an important link between the household production theory of consumer behavior and the theory of investment in human capital. Consumers as investors in their human capital produce these investments with inputs of their own time, books, teachers, etc. Thus, some of the outputs of household production directly enter the utility function, while other outputs determine earnings or wealth in a lifecycle context. Health, on the other hand, does both (Grossman, 2000).

In the Grossman model, health includes longevity and illness-free days. Health is a choice variable because it is a source of utility and because it determines income. Consumers have two reasons for demanding health. First, as a consumption commodity it directly enters into the consumers' utility functions. That is, sick days are a source of disutility. Second, as an investment commodity it determines the total amount of time available for market and nonmarket activities. That is, an increase in the stock of health reduces the amount of time lost from these activities, and the monetary value of this reduction is an index of the return to an investment in health. A person start out with an initial stock of health that depreciates with age and increases with investment. Investments are produced by health inputs, such as medical care use, exercise as well as cigarette and alcohol consumption. When the stock of health falls below a certain level, death occurs (Grossman, 2000).

Solving the Grossman model can produce a conditional labor supply function in which labor supply depends on the endogenous health variable (Currie and Madrian, 1999). From an empirical point of view, the main implication of the model is that health must be treated as an endogenous variable (Currie and Madrian, 1999). However, much of the empirical literature treats health as an exogenous variable, in which the implicit assumption is that exogenous shocks to health are the key factors creating variation in health status. Given that current health depends on past decisions and on habits that may be hard to alter, and the imperfect information individuals have about the health production function, this may not be an unreasonable assumption (Currie and Madrian, 1999).

One of the major efforts of the health and labor literature over many decades has been measuring the effect of health on wages, usually by adding health measures to a standard Mincer wage function (Mincer, 1974). Thus, a more complete model of the choices faced by individuals would recognize that investments in health might alter wages. Conversely, wages can affect investments in health, just as they affect educational decisions. Furthermore, an additional

possibility is that wages and labor market activity have a direct effect on health. Exogenous changes in employment or wages can influence health by directly affecting the probability of workplace injury or stress and risk-taking behaviors (Currie and Madrian, 1999). However, the concept of health is similar to the concept of ability in the sense that everyone has some idea of what the term means, but that it is hard to measure. Failure to measure health accurately leads to bias similar to the "ability bias" (Griliches, 1977) in standard human capital models. That is, if healthier individuals are likely to get more education, for example, then failure to control for health in a wage equation will result in over-estimates of the effects of education (Currie and Madrian, 1999).

3 Empirical Evidence

3.1 Health Shocks

Even though the question of the causal effects of children's health shocks on parents' health and labor market outcomes is an important question from a policy perspective, the literature on this question is limited. This may be due to data limitations, and recent developments in the event study approach making it possible to rely on health shocks for identification of causal effects. There are, however, studies that examine related questions in three main strands of the literature.[1]

The first strand examines the effects of health shocks on family members and spouses, and spillovers within families. In a study using Danish register data, Fadlon and Nielsen (2019) examine how health behaviors are shaped through family spillovers by examining the effects of health shocks on family members' consumption of preventive care and health-related behaviors. They find that both spouses and adult children improve their health behaviors immediately after a family member experience a health shock, defined as a non-fatal heart attack or stroke, and that these responses are significant and persistent. Using data from the United States and Denmark, Black et al. (2017) study the effects of having a sibling with a disability and find that this has negative spillovers on the nearest sibling, as siblings have worse student outcomes. They explain these findings with differences in parental allocations of time and financial resources. Coile (2004) examines the effect of health shocks on the labor supply of spouses. She finds that a spouse's health shock elicits only a small labor supply increase for men and no

[1]There is a large body of health literature documenting that experiencing that a child gets a serious illness has a range of negative effects for the family, such as uncertainty and stress (Enskär et al., 1997; Woodgate and Degner, 2002; Björk et al., 2005; Robinson et al., 2006), psychological distress, anxiety and depression (Rosenberg et al. 2013; Norberg and Boman 2008; Katz et al. 2018), financial distress and work disruption (Dussel et al., 2011; Lansky et al., 1979; Bloom et al., 1985; Patterson et al., 2004). There is also evidence of negative mental health effects for parents of other types of child illnesses and shocks, such as acute burns (Hall et al., 2005), traffic injury (Winston et al., 2002), and accidental injuries (Daviss et al., 2000). However, these studies are correlational and do not allow for a causal interpretation of the effects.

significant increase for women.

The second strand examines the effects of child bereavement on parents' outcomes. In a Swedish study, van den Berg et al. (2017) examine the effects of child bereavement on parental labor market outcomes, health, and family outcomes. They find that losing a child has adverse effects on labor income, employment status, marital status, and hospitalization.[2]

The third strand examines the effects of having a health shock on own outcomes. García-Gómez et al. (2013) examine the effects of having a health shock on own employment and income, and find that a health shock, defined as an acute hospital admission, lowers employment probability by 8% and causes a 5% loss of income two years after the health shock. They find no recovery in either employment or income four years later. Their findings also show substantial negative spillovers within the household. The probability that the spouse is working is reduced by around 1 percentage point and spousal income falls by 2.5% two years after the health shock. Halla and Zweimüller (2013) use accidents occurring on the way to and from work as negative health shocks to identify the causal effect of health on labor market outcomes. They find that after initial periods with a higher incidence of sick leave, injured workers are more likely to be unemployed, and a growing share of them leave the labor market via disability retirement, while injured workers who manage to stay in employment incur persistent earnings losses. Also Dano (2005) uses road injuries to investigate the effects on labor market outcomes. She finds negative effects on disposable income for older individuals and for those with lower initial incomes. She also finds a significant negative effect on employment for males. Lindeboom et al. (2016) examine the relationship between health shocks and the onset of disability and employment outcomes, and find that experiencing a health shock increases the likelihood of the onset of disability, while it does not have a direct effect on employment at older ages.

3.2 Childcare

A large body of evidence has shown that early life experiences can affect health throughout the lifecycle (Shonkoff et al., 2009; Conti and Heckman, 2013). Similarly, early childhood programs have been found to affect early childhood conditions and life experiences, including significant impacts on children's long-run health development (D'Onise et al., 2010a,b; Muennig, 2015) and other human capital outcomes (see e.g., Almond and Currie, 2011; Almond et al., 2018). However, despite the predictive power of early life health for adult well-being (Currie et al., 2010; Reilly and Kelly, 2011), evidence on how provision of universal childcare programs

[2]The effect of child bereavement on health has also been studied in the health literature. For example, Rogers et al. (2008) examine the effects of child bereavement on a range of long-term outcomes. They find that bereaved parents report more depressive symptoms, poorer well-being, more health problems, and are more likely to have experienced a depressive episode and marital disruption compared to comparison parents up to 18 years after the death of a child.

affects children's long-run development focuses mainly on cognitive and non-cognitive measures of child development. For example, Havnes and Mogstad (2011) find positive impacts on educational attainment and labor market participation, measured when the children are in their early 30s. In a related paper, the same team of authors find that the positive effects of the childcare expansion are driven by children in the lower and middle part of the earnings distribution, and that the effects are negative for children in the uppermost part (Havnes and Mogstad, 2015). Datta Gupta and Simonsen (2016) find positive effects on children's test scores in ninth grade. Felfe and Lalive (2018) and Cornelissen et al. (2018) find positive effects on school readiness indicators for children of immigrant ancestry. Also Rossin-Slater and Wüst (2019) find positive effects on schooling. On the other hand, Baker et al. (2008) find no effects on cognitive outcomes, but negative effects on children's non-cognitive outcomes, and Datta Gupta and Simonsen (2010) find that compared to home care, being enrolled in preschool does not lead to significant differences in children's non-cognitive outcomes.

Most of the evidence on the health effects of childcare come from studies that focus on childcare programs targeted at disadvantaged children. For example, Conti et al. (2016) examine the effects on health and healthy behaviors of two targeted early childhood interventions, the Perry Preschool Project (PPP) and the Abecedarian Project (ABC). Both interventions randomly assigned enriched environments to disadvantaged children. They find that boys randomly assigned to the treatment group of the PPP have a significantly lower prevalence of behavioral risk factors in adulthood compared to those in the control group, while those who received the ABC intervention have better physical health. The impacts on girls is considerably weaker for both programs, although they find that both the PPP and the ABC substantially improved the adult healthy habits of girls who were randomized to the treatment groups: they engaged in more physical activity, ate more fresh fruit, and drank less alcohol. However, these programs are not only targeted at disadvantaged children, they also include both schooling and a mix of interventions, such as home visits in the PPP, and interventions to improve health, nutrition, and parent involvement in the ABC, making it difficult to directly compare these findings to those from studies of universal childcare effects.

The literature on the effects of large-scale publicly provided universal childcare on health remains scarce. There are, however, one study from Sweden and three studies from Canada investigating health effects of universal childcare. In the Swedish study, van den Berg and Siflinger (2018) examine the effect of a childcare reform that led to considerable cuts in childcare fees for formal public childcare. Children affected by the reform had better physical health, measured as respiratory illnesses, ear diseases, and other childhood illnesses, at ages 4–5 and 6–7, and better developmental and psychological conditions at age 6–7. Baker et al. (2008) and Baker et al. (2019) investigate the introduction of a large-scale subsidized childcare program in Quebec, Canada, in the late 1990s, and find the opposite from van den Berg and Siflinger

(2018); the introduction of the universal childcare program led to negative effects on children's non-cognitive outcomes both in the short term and in the long term, and significant declines in self-reported health and life-satisfaction, as well as behavioral problems and criminal activity among boys in the long term. Haeck et al. (2018) study the same program as Baker et al. (2019) but come to a different conclusion: when allowing for different treatment periods for different cohorts rather than taking an average, they find that the effects fade out in the long term.

3.3 Air Pollution

The recognition that environmental factors can affect human health is not new, and historically much of our understanding about this relationship comes from the health literature. In particular, the fields of toxicology and epidemiology. However, during the last decades the economics literature on the impacts of pollution on health has grown considerably. The economics literature on health effects of air pollution finds that exposure to increased pollution levels can lead to adverse health outcomes (Neidell, 2004; Currie and Neidell, 2005; Currie et al., 2009; Currie and Walker, 2011; Schlenker and Walker, 2016). In addition to causing adverse health outcomes directly, an indirect effect of exposure to pollution is increased sickness absence from work. Such an effect is related to a literature that in recent years has documented that pollution significantly lowers labor productivity in different contexts (Graff Zivin and Neidell, 2012; Arceo and Oliva, 2015; Chang et al., 2016, 2019).

Previous research from a number of fields has documented a relationship between exposure to air pollution and a range of health outcomes, including respiratory illnesses, asthma, cardiovascular illnesses, stroke, and mortality (Pope-III and Dockery, 2006; Brook, 2008; Chay and Greenstone, 2003a,b; Neidell, 2004; Currie and Neidell, 2005; Knittel et al., 2016; Schlenker and Walker, 2016; Bauernschuster et al., 2017; Jans et al., 2018). In economics, the majority of studies focus on infant and child outcomes. See e.g. Neidell (2004), who examines the effect of air pollution levels on child hospitalization for asthma using naturally occurring seasonal variations in pollution within zip codes in California between 1992 and 1998 to ambient pollution levels, or Jans et al. (2018), who look at the effect of ambient air pollution on hospital visits for children aged 0–18 in Sweden. Only a small number of studies have investigated the short-term impacts of air pollution on health of a general population. One example is Schlenker and Walker (2016), who show that daily variation in ground-level airport congestion significantly increases both exposure to carbon monoxide (CO) and hospitalization rates for asthma, respiratory and heart-related problems. Another example is Bauernschuster et al. (2017), who look at short-term hikes in pollution levels using daily variation in public transport strikes in German cities and show that days with strikes significantly increases both particle matter emissions, and hospital admissions related to respiratory problems among young children.

4 Causal Effects

Over the past three decades, much research has been done on the econometric and statistical analysis of causal effects (Imbens and Wooldridge, 2009). Causal effects are defined as comparison of potential outcomes under different treatments on a common set of units (Rubin 1974, 2005).

In search for causal inference, there has during the past decades been a surge of work in economics (especially applied microeconomics) that takes on the language and conceptual framework of randomized experiments. These studies, which are often called natural experiments or quasi-experiments, examine outcome measures for observations in treatment and control groups, with an exogenous source of variation in the explanatory variables that determine the treatment assignment. This exogenous variation can come from policy changes, government randomization or naturally occurring phenomena, such as natural disasters (Meyer, 1995). Meyer (1995) describes quasi-experimental research as "an outburst of work in economics that adopts the language and conceptual framework of randomized experiments." Here, the ideal research design is explicitly taken to be a randomized trial and the observational study is offered as an attempt to approximate the force of evidence generated by an actual experiment (Angrist and Krueger, 1999).

This approach is a more empirical approach than earlier approaches to econometrics, as the economic theory used to interpret data is typically kept at an intuitive level. It rejects the use of structural econometric models because, according to its adherents, such models do not produce credible estimates and impose arbitrary structures onto the data, and they find such structural assumptions "less credible" (Heckman, 2000). According to Heckman (2000), the track record of the structural approach is at best mixed, because economic data, both at the macro and the micro level, has not yielded many stable structural parameters, and the parameter estimates from structural research programs are widely held not to be credible.

The econometric methods that feature most prominently in the quasi-experimental studies are instrumental variables, regression discontinuity methods, and differences-in-differences-style policy analysis (Angrist and Pischke, 2009). These are all methods that are not new, but their use has grown and become more sophisticated since the 1970s. When using instrumental variables today, for example, it is no longer enough to mechanically invoke a simultaneous equation framework, labeling some variables endogenous and others exogenous, which was usual in the 1970s (Angrist and Pischke, 2009). The econometric literature using these methods goes back to early work by economists such as Ashenfelter (1978), Ashenfelter and Card (1985), and LaLonde (1986).

Especially LaLonde (1986) made an important contribution to the program evaluation literature. In his paper, Lalonde compares the results from an econometric evaluation of the

National Supported Work Demonstration with those from a randomized trial. His results show that many of the econometric procedures do not replicate the experimentally determined results. He argues that these results suggest that researchers should be aware of the potential for specification errors in other non-experimental evaluations.

The central problem studied in this literature is that of evaluating the effect of the exposure of a set of units to a program, or treatment, on some outcome. The units are typically economic agents such as individuals, households, markets, firms, counties, states or countries, and the treatments can be job search assistance programs, educational programs, laws or regulations, or environmental exposure. A critical feature of this approach is that, in principle, each unit can or cannot be exposed to the treatment. This literature is therefore focused on settings with observations on units exposed, and not exposed, to the treatment, with the evaluation of the treatment being based on comparison of units exposed and not exposed (Imbens and Wooldridge, 2009). We can of course not observe both outcomes for one unit as the unit can be exposed to only one level of treatment. In a famous paper, Holland (1986) refers to this problem as the "fundamental problem of causal inference".

In several prominent papers, Rubin (1973, 1974, 1977) formulated the now dominant approach to the analysis of causal effects in observational studies. Rubin proposed the interpretation of causal statements as comparisons of so-called potential outcomes: pairs of outcomes defined for the same unit given different levels of treatment. In his approach, models are developed for the pair of potential outcomes rather than solely for the observed outcome.

4.1 The Potential Outcomes Framework

Consider a setting with $i = 1, ..., N$ units. Some of these units receive treatment, while others do not receive treatment. In the potential outcomes framework each unit has two potential outcomes, but only one observed outcome. Potential outcomes are defined as Y_i^1 if the unit receives treatment and Y_i^0 if the unit does not. The state where the unit receives no treatment is called the control state. That is, if unit i receives treatment Y_i^1 will be realized and Y_i^0 will *ex post* be a counterfactual outcome. If, on the other hand unit i does not receive treatment, Y_i^0 will be realized and Y_i^1 will be the *ex post* counterfactual. The treatment status is denoted by D_i, which equals one if the unit receives treatment and zero if it does not. For each unit we observe the following outcome:

$$Y_i = D_i Y_i^1 + (1 - D_i) Y_i^0 \tag{1}$$

The causal effect, δ_i, is given by the difference between the potential outcomes:

$$\delta_i = Y_i^1 - Y_i^0 \tag{2}$$

However, because we cannot observe both potential outcomes for each unit at a given point in time, we cannot calculate the causal effect directly. Researchers are therefore interested in two different parameters (Cunningham, 2018). The first is the average treatment effect:

$$ATE = E[\delta_i] = E[Y_i^1 - Y_i^0] = E[Y_i^1] - E[Y_i^0] \tag{3}$$

The average treatment effect is also unknowable because it requires two observations per unit i, one of which is a counterfactual. Thus, the average treatment effect, ATE, like the individual treatment effect, is not a quantity that can be calculated.

The second parameter of interest is the average treatment effect for the treatment group, ATT. The average treatment effect for the treatment group is the population mean treatment effect for the group of units that received treatment in the first place. In observational data, ATT will generally be different from ATE, and like ATE, it is unknowable because it also requires two observations per unit i:

$$ATT = E[\delta_i|D_i = 1] = E[Y_i^1 - Y_i^0|D_i = 1] = E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1] \tag{4}$$

Because we never observe both potential outcomes for unit i, we must learn about the effects of the treatment by comparing the mean outcomes of those who receive treatment and those who does not. A naive comparison of means by treatment status tells us something about potential outcomes, but maybe not what we want to know (Angrist and Pischke, 2009). The comparisons of mean outcomes conditional on treatment status is formally linked to the average causal effect by the following equation:

$$\underbrace{E[Y_i|D_i = 1] - E[Y_i|D_i = 0]}_{Observed\ difference\ in\ mean\ outcomes} = \underbrace{E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1]}_{ATT} + \underbrace{E[Y_i^0|D_i = 1] - E[Y_i^0|D_i = 0]}_{Selection\ bias} \tag{5}$$

The term:

$$E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1] = E[Y_i^1 - Y_i^0|D_i = 1] \tag{6}$$

is the average causal effect of treatment on those who were treated. This term captures the mean difference between those who receives treatment, $E[Y_i^1|D_i = 1]$ and what would have happened to them had they not received the treatment, $E[Y_i^0|D_i = 1]$. The observed difference in mean outcomes however, has an additional term called selection bias. Selection bias is the mean Y_i^0 between those who are and are not treated. For example, if the treatment is hospitalization and the outcome is health status, because the sick are more likely than the healthy to seek treatment, those who are hospitalized have worse Y_i^0, making the selection bias negative

(Angrist and Pischke, 2009). What solves the problem of selection bias is random assignment of D_i, because random assignment makes D_i independent of the potential outcomes. To see this, note that:

$$E[Y_i|D_i = 1] - E[Y_i|D_i = 0] = E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1] = \\ E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1] \quad (7)$$

where the independence of Y_i^0 and D_i allows us to swap $E[Y_i^0|D_i = 1]$ for $E[Y_i^0|D_i = 0]$ in the second line. Given random assignment, equation 7 simplifies to:

$$E[Y_i^1|D_i = 1] - E[Y_i^0|D_i = 1] = E[Y_i^1 - Y_i^0|D_i = 1] = E[Y_i^1 - Y_i^0] \quad (8)$$

where the effect of randomly-assigned treatment on the treated is the same as the effect of treatment on a randomly chosen unit i. The random assignment has also eliminated selection bias. The goal of most economic research is exactly to overcome selection bias, and therefore estimate the causal effect of treatment.

5 Econometric Methods

5.1 Two-way Fixed Effects Models

The main idea behind identification strategies using fixed effects is to use repeated observations on individuals to control for unobserved and unchanging characteristics that are related to both outcomes and treatment variables (Angrist and Krueger, 1999).

Suppose that we have a panel data set of N individuals and T time periods. Let $D_{i,t}$ and $Y_{i,t}$ represent the treatment indicator and the observed outcome for individual i at time t. The observed $Y_{i,t}$ is either $Y_{0,i,t}$ or $Y_{1,i,t}$ depending on the treatment status (Angrist and Pischke, 2009). Suppose further that:

$$E(Y_{0,i,t}|A_i, X_{i,t}, t, D_{i,t}) = E(Y_{0,i,t}|A_i, X_{i,t}, t) \quad (9)$$

i.e., treatment status is as good as randomly assigned conditional on unobserved individual characteristics A_i, and other observed covariates $X_{i,t}$. The key to fixed effects estimation is that the unobserved A_i does not vary over time t in a linear model for $E(Y_{0,i,t}|A_i, X_{i,t}, t)$:

$$E(Y_{0,i,t}|A_i, X_{i,t}, t) = \alpha + \lambda_t + A_i^{'}\gamma + X_{i,t}\delta \quad (10)$$

Finally, we assume that the causal effect of treatment is additive and constant:

$$E(Y_{1,i,t}|A_i, X_{i,t}, t) = E(Y_{0,i,t}|A_i, X_{i,t}, t) + \rho \tag{11}$$

where ρ is the causal effect of interest. This implies:

$$Y_{i,t} = \alpha_i + \lambda_t + \rho D_{i,t} + \delta X_{i,t} + \epsilon_{i,t} \tag{12}$$

where

$$\alpha_i \equiv \alpha + A_i^{'}\gamma \tag{13}$$

The two-way fixed effects model is estimated by linear regression with time and individual fixed effects:

$$Y_{i,t} = \alpha_i + \lambda_t + \delta D_{i,t} + \beta X_{i,t} + \epsilon_{i,t} \tag{14}$$

Given panel data, the causal effect of treatment on the outcome can be estimated by treating the individual fixed effect α_i and the time fixed effect λ_t as parameters to be estimated. The main assumption of the fixed effects model is that we have a linear, additive functional form to account for the problem of unobserved confounders (Angrist and Pischke, 2009).

5.2 Differences-in-Differences Design

Differences-in-differences is both the most common and the oldest quasi-experimental research design (Goodman-Bacon, 2018). Since the seminal work by Ashenfelter (1978) and Ashenfelter and Card (1985), the use of differences-in-differences methods have become widespread in empirical economics.

In the simplest setting, outcomes are observed for individuals in one of two groups, in one of two time periods. Only individuals in one of the two groups, in the second time period, receive treatment. There are no individuals exposed to the treatment in the first period, and individuals from the control group are never observed to be exposed to the treatment. The average the change in outcomes over time in the control group is subtracted from the change in outcomes over time in the treatment group. This double differencing removes biases in the second period comparisons between the treatment and control group that could be the result of permanent differences between the two groups, as well as biases from comparisons over time in the treatment group that could be the result of time trends unrelated to the treatment (Imbens and Wooldridge, 2007).

Differences-in-differences is a version of fixed effects estimation using data on the group level (the groups can be municipalities, states, countries, etc.). The potential outcomes are defined as $Y^1_{i,s,t}$ for individual i in time period t with treatment, and as $Y^0_{i,s,t}$ for individual i in time period t without treatment. The main feature of the differences-in-differences design is an

additive structure of potential outcomes in the no-treatment group (Angrist and Pischke, 2009). Specifically, we assume that:

$$E(Y_{0,i,s,t}|s,t) = \gamma_s + \lambda_t \tag{15}$$

where s denotes group and t denotes time period. This equation says that in absence of treatment, the outcome is determined by a sum of a time-invariant group effect and a year effect that is common across groups. Let $D_{s,t}$ be a dummy for treatment. Assuming that $E(Y_{1,i,s,t} - Y_{0,i,s,t}|s,t)$ is a constant β, we get:

$$Y_{i,s,t} = \gamma_s + \lambda_t + \beta D_{s,t} + \epsilon_{i,s,t} \tag{16}$$

where β is the causal effect of interest (Angrist and Pischke, 2009). This quantity also equals the estimated coefficient on the interaction of a treatment group dummy and a post-treatment period dummy in the following regression:

$$Y_{i,t} = \alpha + \beta_1 D_i + \beta_2 Post_t + \delta(D \times Post)_{i,t} + \epsilon_{i,t} \tag{17}$$

Many empirical applications of differences-in-differences, however, deviate from the simple differences-in-differences setup and have more than two periods and variation in the timing of treatment (Callaway and Sant'Anna, 2019). The key identifying assumption in the differences-in-differences design is the parallel trends assumption, which says that trends would be the same in the treatment and the control group in the absence of treatment.

5.3 Event Study Design

The event study approach is an extension of the differences-in-differences design, used to estimate the dynamic effects of discrete shocks and non-transient treatments. The event study approach exploits variation in the timing of an individual's treatment, which allows for identification of the path of treatment effects even when there are no pure control individuals. The treated individuals are grouped into cohorts based on when they first receive treatment, and for each cohort, relative time to initial treatment can be defined. The crucial feature of event studies is the presence of multiple cohorts, which serves as a source of identification for estimation. Within a cohort, calendar time and relative time are collinear, so it is not possible to identify dynamic treatment effects separately in the presence of time trends. However, with multiple cohorts it is possible to separate these two sets of effects by comparing the trends in average outcomes between treated cohorts and cohorts that receive treatment at a later point in time, given the parallel trends and no anticipation assumptions. For any given cohort, the differences in trends identify the average treatment effect for this cohort at different relative times (Abraham and Sun, 2019).

Consider a setting with $i = 1, \ldots, N$ individuals and $t = 1, \ldots, T$ time periods. For individual *i*, the observed outcome in year *t* is denoted by $Y_{i,t}$. E_i denotes the year of initial treatment. $Y_{i,t}(e)$ denotes the potential outcome for individual *i* at time *t*. This is only observed when $E_i = e$. The parameter of interest is the average treatment effect on the treated, $ATT_t(e)$:

$$ATT_t(e) \equiv \mathbb{E}[Y_{i,t}(e) - Y_{i,t}(\infty)|E_i = e] \tag{18}$$

where $Y_{i,t}(\infty)$ is the outcome an individual *i* would have at time *t* if counterfactually assigned treatment at time ∞ (i.e. never treated). This is the average difference in $Y_{i,t}$ that is due to being treated at *e* instead of ∞, among those who are treated at *e*.

To compare across cohorts in an event study framework, the calendar time *t* can be changed to relative time index, *l*, which denotes the time periods relative to treatment. For cohort *e*, *l* ranges from $-e$ to $T - e$ since we observe e periods before initial treatment and $T - e$ after initial treatment. The causal parameter of interest, $CATT_{e,l}$, can therefore be defined as:

$$CATT_{e,l} \equiv \mathbb{E}[Y_{i,e+l}(e) - Y_{i,e+l}(\infty)|E_i = e] \tag{19}$$

The treatment effects in the event study approach are estimated by a dynamic linear regression specification with two-way (individual and calendar time) fixed effects, given by the following estimating equation:

$$Y_{i,t} = \alpha_i + \gamma_t + \sum_{l=-3}^{T=3} \mu_l D_{i,t}(l) + \epsilon_{i,t} \tag{20}$$

There are three identifying assumption in the event study approach. The first is the parallel trends assumption. This says that for any two observed cohorts e and e', the change over time they would have had in the absence of treatment is the same. The second is the no anticipation assumption. This says that prior to the onset of treatment outcomes do not depend on the time at which treatment will occur. The third is the assumption of homogenous treatment effects. That says that each cohort experience the same path of treatment effects on average and that $CATT_{e,l}$ at any given *l* is the same across cohorts. Whereas violations of the parallel trends and no anticipation assumptions invalidates the event study, violation of the treatment effect homogeneity assumption makes the event study harder to interpret (Abraham and Sun, 2019).

6 Internal and External Validity

Empirical evidence on any given causal effect is always local, derived from a particular time, place, and research design. A constructive response to the specificity of a given research design is therefore to look for more evidence, so a general picture can emerge (Angrist and Pischke, 2010). Achieving a high degree of internal validity, that is, a high degree of confidence that

what is measured indeed represents a causal phenomenon, is the primary goal of the ex post evaluation problem (DiNardo and Lee, 2011). However, it is important to think about the external validity of a specific setting as well. External validity means that the statistical inferences can be generalized from the population and setting studied to other populations and settings. That is, it says something about the extent to which the results of a study can be generalized.

There are two conditions for internal validity to exist. First, the estimate of the causal effect, which is measured as the coefficient(s) of interest, should be unbiased and consistent. Second, statistical inference is valid, that is, hypothesis tests should have the desired size and confidence intervals should have the desired coverage probability. There are several threats to internal validity, such as omitted variables, functional form misspecification, measurement error, sample selection, simultaneous causality, and heteroscedasticity and/or correlated error terms.

External validity might be invalid if there are differences between the population studied and the population of interest or if there are differences in the settings of the considered populations, e.g., the legal framework or the time of the investigation. As with internal validity, there are several threats to external validity. First, there may be differences in populations. That is, the population from which the sample is drawn might differ from the population of interest. Second, there may be differences in settings, as the setting studied might differ from the setting of interest due to differences in laws, institutional environment and physical environment.

By using well-established econometric methods and large samples from register data covering the entire Norwegian population, hopefully the results found in this thesis have both internal and external validity. However, as Angrist and Pischke (2010) states, it is always important to look for more evidence so that a general picture can emerge.

7 Paper Summaries

7.1 Effects of Children's Health Shocks on Parental Health and Labor Market Outcomes

Experiencing that a child has a serious negative health shock is stressful for parents. Parents may face psychological stress, such as anxiety and depression, due to the uncertainty that follows serious illness in the family (Björk et al., 2005; Hosoda, 2014; Quin, 2005). In addition, the illness of a child may increase the burden of care, increasing time spent on taking care of the child, which may lead to less time for work and other activities. Both these factors may lead to negative labor market outcomes, and adverse mental and physical health for the parents. Despite being an important topic, there is little causal evidence on the relationship between children's negative health shocks and parents' outcomes.

The main question I ask in this paper is whether experiencing that a child has a negative

health shock leads to negative labor market outcomes, such as reduced labor income and employment, for the parents of the child.[3] Second, do these potential negative effects on labor market attachment result in a higher probability of receiving social security benefits? Third, could parents' deteriorating health be a key mechanism for the labor market responses? To make progress on these questions, I use Norwegian register data on individual labor market and health outcomes, covering the period 2006–2014. In the main analysis, the sample consists of families in which a child has a health shock between age 5 and 18. The health shock is defined as having an acute overnight hospital admission, given that the child did not have a hospital admission the year before the shock, ensuring that there is no anticipation the year before the health shock.

To identify the causal effects of children's negative health shocks on parents' health and labor market outcomes, I use an event study approach. The event study approach exploits variation in the timing of a child's health shock. The treated children are grouped into cohorts based on when they have a health shock, and for each cohort, relative time to the initial health shock can be defined. The key feature of the event study is the presence of multiple cohorts, which serves as a source of identification for estimation. Within a cohort, calendar time and relative time are collinear, so it is not possible to identify dynamic treatment effects separately in the presence of time trends. However, with multiple cohorts it is possible to separate these two sets of effects by comparing the trends in average outcomes between treated cohorts and cohorts that are treated at a later point in time, given the parallel trends and no anticipation assumptions. For any given cohort, the differences in trends identify the average treatment effect for this cohort at different relative times (Abraham and Sun, 2019).

The results show that experiencing that a child has a health shock has significant effects on parents' labor market attachment. The effects are immediate and persistent. Specifically, in the year of the health shock, mothers experience a decrease in labor income by 7.6% and fathers a decrease of 3.3%. At the same time, received sick pay increases by 15% for mothers and by 9.5% for fathers. The effects are larger in the long term. Three years after the health shock, mothers have a 19% lower labor income, a 2.1% lower probability of being in employment, and an increased probability of receiving disability benefits of 100%. Fathers have a 18% lower labor income, a 1.8% lower probability of being in employment, and a 70% higher probability of receiving disability benefits, three years after the health shock.

The underlying health problems are psychological disorders. In the year of the health shock, mothers have an increased probability of having a GP consultation related to psychological diagnoses by 7%, and an increased probability of having sickness absence related to psychologi-

[3]Norway has a universal tax-financed social security system, as well as a universal healthcare system. Thus, I do not expect to see direct economic consequences due to increased spending on healthcare. In other settings, parents may need to increase their labor supply to increase their income to pay for healthcare.

cal diagnoses by 18%. The corresponding increases for fathers are 3.6% and 13%, respectively. The effects on the use of healthcare services and sickness absence are not persistent in the long term. This is, however, in line with the expected effects given how the Norwegian social security system is organized, with temporary benefits, such as sickness absence ending after one year, and permanent benefits, such as disability benefits, starting earliest after sick pay ends.

This paper makes several contributions to the literature. First, although the question of the causal effects of children's health shocks on parents' health and labor market outcomes is an important question from a policy perspective, the economics literature on this question is limited.[4] This paper thus contributes to the literature by using high-quality register data and state-of-the-art econometric methods to study this question in a manner that provides causal effects. Second, by considering a wide range of possible outcomes as well as heterogeneity by the type of health shock, I am able to capture a comprehensive picture of the effects on parents' health and labor market outcomes of experiencing that a child has a negative health shock, as well as the potential mechanisms driving the effects. The results in this study can guide policymakers in improving the design of policies to assist parents and to reduce the economic costs of children's health shocks. Third, this study complements the literature studying the effects of health shocks on family members' health behavior, the effects of bereavement on parents' health and labor market outcomes, and the effects of child disability on siblings and parents' health and labor market outcomes, described further in the next section.

7.2 Effects of Universal Childcare on Long-Run Health

(Joint with Emilia Del Bono and Julie Riise)

A large body of evidence has shown that early life experiences can affect health throughout the lifecycle (Shonkoff et al., 2009; Conti and Heckman, 2013). Similarly, early childhood programs have been found to affect early childhood conditions and life experiences, including significant impacts on children's long-run health development (D'Onise et al., 2010a,b; Muennig, 2015) and other human capital outcomes (see e.g., Almond and Currie, 2011; Almond et al., 2018). However, despite the predictive power of early-life health for adult well-being (Currie et al., 2010; Reilly and Kelly, 2011), evidence on the long-term effects of childcare programs on health outcomes is based mainly on small-scale targeted programs (Campbell et al., 2014; Conti et al., 2016), and evidence on how the provision of universal programs affects

[4]There is a large body of health literature documenting that experiencing that a child gets a serious illness has a range of negative effects for the family, such as uncertainty and stress (Enskär et al., 1997; Woodgate and Degner, 2002; Björk et al., 2005; Robinson et al., 2006), psychological distress, anxiety, and depression (Rosenberg et al. 2013; Norberg and Boman 2008; Katz et al. 2018), financial distress and work disruption (Dussel et al., 2011; Lansky et al., 1979; Bloom et al., 1985; Patterson et al., 2004). There is also evidence of negative mental health effects for parents of other types of child illnesses and shocks, such as acute burns (Hall et al., 2005), traffic injury (Winston et al., 2002), and accidental injuries (Daviss et al., 2000). However, these studies are correlational and do not allow for a causal interpretation of the effects.

children's long-run development remains scarce. Evidence of the effects of universal programs on adult health and healthy behavior is particularly limited.

In this paper, we use Norwegian administrative data and examine the long-run health outcomes of children affected by a 1975 reform in Norway, which led to a large-scale expansion of subsidized universal childcare for children three to six years old. More specifically, our main research question is whether, and to what extent, the expansion of universal childcare has long-term effects on adult health outcomes. We examine the effects of the reform on four main outcomes: primary healthcare use, certified sickness absence from work, and somatic and psychiatric specialist healthcare use. The health outcomes are measured in 2006–2014 (primary healthcare and sickness absence) and 2008–2014 (specialist healthcare), which means that the sample of children exposed to the reform are in their prime age, between 30–47, when the outcomes are measured.

Although the childcare reform was planned centrally, the responsibility for childcare was assigned to the municipalities. This led to a staged expansion of childcare coverage across Norway's (at that time) 445 municipalities. We exploit the variation in the expansion of childcare between different municipalities in this period to examine the long-run health effects of childcare. Our empirical strategy follows that of Havnes and Mogstad (2011), using a differences-in-differences approach comparing adult health outcomes of children three to six years old before and after the reform, from municipalities where childcare expanded significantly and municipalities with little or no expansion.

We have two main findings. First, women affected by the reform increase their use of pregnancy-related healthcare services and sickness absence. However, there is no increase in fertility and no effects on the second generation's birth outcomes, indicating that the women's health is unchanged, but that they have increased their demand for healthcare services, suggesting a change in health-seeking behavior rather than a change in health. However, in the long run, as these individuals become older, more preventive behavior such as more health check-ups, may translate into better health. A change in behavior could come directly from the practices and habits formed already in childcare, but it is more likely an indirect effect. It is well established that there is a socioeconomic gradient in the use of healthcare services (Monstad et al., 2014; Kaarboe and Carlsen, 2014; Moscelli et al., 2018; Cutler and Lleras-Muney, 2010), and the observed effect can thus be an indirect consequence of the reform, resulting from the identified positive effects on education and income (Havnes and Mogstad, 2011).

Second, there is a reduction in the use of mental healthcare services, and services related to injuries and social problems. The reduction in the use of mental health services could reflect improved mental health or a change in behavior towards less help seeking. There is a reduction both in the probability of visiting a GP and in the use of psychiatric specialist care. The latter is especially indicative of better mental health, as there is high excess demand for men-

tal healthcare in Norway, and individuals are only granted access to these specialist services once the mental health problems have become severe. Together this suggests that formal childcare benefits individuals by improving their mental health in the long run. The routines and pedagogical environment of childcare could strengthen social skills and induce better decision-making and healthier behavior that last into adulthood. Being in a formal childcare institution could also increase the chances of detecting behavioral, social and psychological problems at an early stage and could thus prevent the development of more serious problems. Both of these explanations point to direct effects of childcare, but we are not able to exclude the possibility of an alternative or additional effect related to the already identified increases in education and income.

Our study of the long-term health effects of a universal childcare program contributes to the literature in several ways. First, the majority of studies on the long-term effects of universal childcare focuses exclusively on cognitive and non-cognitive measures of child development.[5] In spite of a considerable number of studies on the effects on human capital development, stringent evidence on the causal effects on health remains scarce. Most of the existing evidence comes of small-scale and/or targeted programs[6], and the literature on effects from large-scale publicly provided universal childcare on health is limited. van den Berg and Siflinger (2018), Baker et al. (2008), Baker et al. (2019), and Haeck et al. (2018) all study health effects of universal programs, but only Baker et al. (2019) and Haeck et al. (2018) focus on long-term outcomes, in which the health aspect is limited to two survey questions about self-reported health. We take advantage of a large, universal reform in combination with highly detailed administrative register data and examine a wide range of adult health outcomes and healthcare use, capturing many aspects of health that have not yet been studied.

7.3 Effects of Air Pollution on Health and Productivity

(Joint with Tor Helge Holmås and Julie Riise)

According to the European Environmental Agency, air pollution has substantial economic impacts: it increases the use of health care services, causes adverse health outcomes, and short-

[5]Havnes and Mogstad (2011) find positive impacts on educational attainment and labor market participation, measured when the children are in their early 30s. In a related paper, the same team of authors find that the positive effects of the childcare expansion are driven by children in the lower and middle part of the earnings distribution, and that the effects are negative for children in the uppermost part (Havnes and Mogstad, 2015). Datta Gupta and Simonsen (2016) find positive effects on children's test scores in ninth grade. Felfe and Lalive (2018) and Cornelissen et al. (2018) find positive effects on school readiness indicators for children of immigrant ancestry. Also Rossin-Slater and Wüst (2019) find positive effects on schooling. On the other hand, Baker et al. (2008) find no effects on cognitive outcomes but negative effects on children's non-cognitive outcomes. Datta Gupta and Simonsen (2010) find that compared to home care, being enrolled in preschool does not lead to significant differences in children's non-cognitive outcomes.

[6]Targeted programs like Head Start, the Perry Preschool Project (PPP), and the Abecedarian Project (ABC) have generated positive long-term effects on outcomes such as behavioral problems, prevalence of chronic conditions, and obesity for their participants (Currie and Thomas, 1995; Carneiro and Ginja, 2014; Conti et al., 2016).

ens people's lives (EEA, 2015). The economics literature on the health effects of air pollution supports this notion and finds that exposure to increased pollution levels can lead to adverse health outcomes (Neidell, 2004; Currie and Neidell, 2005; Currie et al., 2009; Currie and Walker, 2011; Schlenker and Walker, 2016). In addition to causing adverse health outcomes directly, an indirect effect of exposure to pollution is increased sickness absence from work. Such an effect is related to a literature that in recent years has documented that pollution significantly lowers labor productivity in different contexts (Graff Zivin and Neidell, 2012; Arceo and Oliva, 2015; Chang et al., 2016, 2019), but, to the best of our knowledge, this issue has not been studied previously.

The main research question in this paper is whether, and to what extent, short-term air pollution increases from relatively low levels adversely affect health outcomes and productivity in the general population. To answer this question, we explore the relationship between short-term air pollution increases and the use of healthcare services and worker productivity in the period 2011–2014 by examining three main outcomes: general practitioner (GP) consultations, certified sickness absences from work, and acute hospital visits. For all three main outcomes, we examine related diagnoses. Estimating two-way fixed effects models, using geographical and time variation in exposure to particulate matter (PM_{10}) and nitrogen dioxide (NO_2), we find that short-term air pollution increases from relatively low levels adversely affect health outcomes — also for the working age population, in contrast to most other studies, which have not had access to data covering this part of the population. The adverse effects on health outcomes in turn lead to negative impacts on worker productivity, measured as sickness absences from work.

Specifically, the results show positive and statistically significant relationships between PM_{10} and GP consultations and certified sickness absences, driven by diagnoses related to the respiratory system. We also find a positive and statistically significant relationship between NO_2 and acute hospital visits, again driven by diagnoses related to the respiratory system. The results suggest that PM_{10} is related to diseases that can be treated by the GP and sickness absence from work, while NO_2 is related to more serious diseases leading to a hospital visit. The heterogeneity analysis shows that the working-age population and the middle-to-high-income groups are the main drivers of the estimated effects.

We contribute to the literature in several ways. First, our empirical approach allows us to estimate the contemporaneous effects of air pollution on the health and productivity of the general population based on variation in local air pollution. By doing so, we take advantage of highly detailed Norwegian population register data and examine the health responses of the entire population, not only the most fragile groups. Second, because we have data on both GP consultations and hospital visits, we capture both less serious incidences (leading to a GP consultation) and very serious incidences (leading to unplanned hospitalization). Third, since

we have universal access to healthcare in Norway, GP consultations as well as hospitalization is practically free of charge. Any heterogeneity in hospitalization should therefore not be driven by access to health services or the ability to pay for health services in our study. However, this could pose a potential problem for studies conducted, for example, in the United States, where the retired population has access to medical services through Medicare, while not all individuals of working age are covered by health insurance (Schlenker and Walker, 2016). Finally, our framework allows us to control for various potential confounders with detailed data on observable characteristics of the individuals in our sample, such as socioeconomic status, in addition to detailed weather data on precipitation, wind, and temperature.

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Chapter 2:

Effects of Children's Health Shocks on Parental Health and Labor Market Outcomes

Effects of Children's Health Shocks on Parental Health and Labor Market Outcomes*

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Abstract

This paper shows that parents experiencing that their child has a severe health shock between age 5 and 18, have significant and persistent reductions in labor income and employment, and increased use of social security benefits. The underlying health problems are psychological disorders. Heterogeneity analysis by age of the child and type of health shock reveals larger effects of health shocks related to injury, poisoning and other consequences of external causes, such as traumatic head injury, when the children are between age 5 and 12. The results are found using high-quality Norwegian register data and an event study approach.

Keywords: Health shocks, labor productivity, health

JEL Codes: I1, J24

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1 Introduction

Experiencing that a child has a serious negative health shock is stressful for parents. Parents may face psychological stress, such as anxiety and depression, due to the uncertainty that follows serious illness in the family (Björk et al., 2005; Hosoda, 2014; Quin, 2005). In addition, the illness of a child may increase the burden of care, increasing time spent on taking care of the child, which may lead to less time for work and other activities. Both these factors may lead to negative labor market outcomes, and adverse mental and physical health for the parents. Despite being an important topic, there is little causal evidence on the relationship between children's negative health shocks and parents' outcomes.

The main question I ask in this paper is whether experiencing that a child has a negative health shock leads to negative labor market outcomes, such as reduced labor income and employment, for the parents of the child.[1] Second, do these potential negative effects on labor market attachment result in a higher probability of receiving social security benefits? Third, could parents' deteriorating health be a key mechanism for the labor market responses? To make progress on these questions, I use Norwegian register data on individual labor market and health outcomes, covering the period 2006–2014. In the main analysis, the sample consists of families in which a child has a health shock between age 5 and 18. The health shock is defined as having an acute overnight hospital admission, given that the child did not have a hospital admission the year before the shock, ensuring that there is no anticipation the year before the health shock.

To identify the causal effects of children's negative health shocks on parents' health and labor market outcomes, I use an event study approach. There are three main empirical challenges, which motivates my choice of an event study approach. First, there may be selection bias because families that have a child that has a health shock may be different from families that do not have a child that has a health shock. To avoid this problem, I restrict the sample to only those families in which a child has a health shock in the period the available data covers, which is the period 2009–2014. Second, there may be omitted characteristics that correlate with both the probability of having a health shock and the outcomes. To avoid the omitted variables problem, I exploit the long panel data set and use individual fixed effects to control for time-invariant individual characteristics. Third, the child's and parents' health may affect the probability that a child has a health shock at the same time that the child's health shock affects the outcomes of the parents. I address the simultaneity problem by studying the graphical presentation of the paths of the potential outcomes before and after the health shock, ensuring that the timing of the health shock is exogenous.

[1] Norway has a universal tax-financed social security system, as well as a universal healthcare system. Thus, I do not expect to see direct economic consequences due to increased spending on healthcare. In other settings, parents may need to increase their labor supply to increase their income to pay for healthcare.

The event study approach exploits variation in the timing of a child's health shock. The treated children are grouped into cohorts based on when they have a health shock, and for each cohort, relative time to the initial health shock can be defined. The key feature of the event study is the presence of multiple cohorts, which serves as a source of identification for estimation. Within a cohort, calendar time and relative time are collinear, so it is not possible to identify dynamic treatment effects separately in the presence of time trends. However, with multiple cohorts it is possible to separate these two sets of effects by comparing the trends in average outcomes between treated cohorts and cohorts that are treated at a later point in time, given the parallel trends and no anticipation assumptions. For any given cohort, the differences in trends identify the average treatment effect for this cohort at different relative times (Abraham and Sun, 2019).

In the analysis, I first study the effects of the health shock on four main outcomes: sick pay, labor income, employment, and the probability of receiving disability benefits. Second, I study the use of healthcare services and sickness absence from work in total, and related to musculoskeletal and psychological diagnoses, as these diagnoses are related to stress and anxiety (Cho et al., 2003; Edwards et al., 2016; Meints and Edwards, 2018). Third, to examine how the effects evolve over time, and to see whether the effects are persistent, I study both the short-term and the long-term effects of the health shock on parents' outcomes.

The results show that experiencing that a child has a health shock has significant effects on parents' labor market attachment. The effects are immediate and persistent. Specifically, in the year of the health shock, mothers experience a decrease in labor income by 7.6% and fathers a decrease of 3.3%. At the same time, received sick pay increases by 15% for mothers and by 9.5% for fathers. The effects are larger in the long term. Three years after the health shock, mothers have a 19% lower labor income, a 2.1% lower probability of being in employment, and an increased probability of receiving disability benefits of 100%. Fathers have a 18% lower labor income, a 1.8% lower probability of being in employment, and a 70% higher probability of receiving disability benefits, three years after the health shock.

The underlying health problems are psychological disorders. In the year of the health shock, mothers have an increased probability of having a GP consultation related to psychological diagnoses by 7%, and an increased probability of having sickness absence related to psychological diagnoses by 18%. The corresponding increases for fathers are 3.6% and 13%, respectively. The effects on the use of healthcare services and sickness absence are not persistent in the long term. This is, however, in line with the expected effects given how the Norwegian social security system is organized, with temporary benefits, such as sickness absence ending after one year, and permanent benefits, such as disability benefits, starting earliest after sick pay ends.

To shed light on whether there are differences in the effects due to different cause, severity, and timing of the health shock, I assess heterogeneity by the type of health shock given by the

related diagnosis, by the length of the initial hospital stay, and by the age of the child at the time of the health shock. The heterogeneity analysis shows that the effects are mainly driven by health shocks related to injury, poisoning and other consequences of external causes when the children are between age 5 and 12. Specifically, the most common diagnosis is traumatic head injury.

This paper makes several contributions to the literature. First, although the question of the causal effects of children's health shocks on parents' health and labor market outcomes is an important question from a policy perspective, the economics literature on this question is limited.[2] This paper thus contributes to the literature by using high-quality register data and state-of-the-art econometric methods to study this question in a manner that provides causal effects. Second, by considering a wide range of possible outcomes as well as heterogeneity by the type of health shock, I am able to capture a comprehensive picture of the effects on parents' health and labor market outcomes of experiencing that a child has a negative health shock, as well as the potential mechanisms driving the effects. The results in this study can guide policymakers in improving the design of policies to assist parents and to reduce the economic costs of children's health shocks. Third, this study complements the literature studying the effects of health shocks on family members' health behavior, the effects of bereavement on parents' health and labor market outcomes, and the effects of child disability on siblings and parents' health and labor market outcomes, described further in the next section.

The paper proceeds as follows. Section 2 reviews the existing literature on the effects of health shocks in different settings. Section 3 describes the institutional background of the Norwegian social security system, the definition of the health shock, and the data applied. Section 4 outlines the empirical strategy. Section 5 presents the results, and Section 6 presents the robustness checks. Section 7 concludes.

2 Literature Review

Even though the question of the causal effects of children's health shocks on parents' health and labor market outcomes is an important question from a policy perspective, the literature on this question is limited. This may be due to data limitations, and recent developments in the event study approach making it possible to rely on health shocks for identification of causal effects. There are, however, studies that examine related questions in three main strands of the

[2]There is a large body of health literature documenting that experiencing that a child gets a serious illness has a range of negative effects for the family, such as uncertainty and stress (Enskär et al., 1997; Woodgate and Degner, 2002; Björk et al., 2005; Robinson et al., 2006), psychological distress, anxiety, and depression (Rosenberg et al. 2013; Norberg and Boman 2008; Katz et al. 2018), financial distress and work disruption (Dussel et al., 2011; Lansky et al., 1979; Bloom et al., 1985; Patterson et al., 2004). There is also evidence of negative mental health effects for parents of other types of child illnesses and shocks, such as acute burns (Hall et al., 2005), traffic injury (Winston et al., 2002), and accidental injuries (Daviss et al., 2000). However, these studies are correlational and do not allow for a causal interpretation of the effects.

economics literature.[3][4]

The first strand examines the effects of health shocks on family members and spouses, and spillovers within families. In a study using Danish register data, Fadlon and Nielsen (2019) examine how health behaviors are shaped through family spillovers by examining the effects of health shocks on family members' consumption of preventive care and health-related behaviors. They find that both spouses and adult children improve their health behaviors immediately after a family member experience a health shock, defined as a non-fatal heart attack or stroke, and that these responses are significant and persistent. However, having data only on the adult population, they do not examine how health shocks of children and youths affect parents' outcomes. Using data from the United States and Denmark, Black et al. (2017) study the effects of having a sibling with a disability and find that this has negative spillovers on the nearest sibling, as siblings have worse student outcomes. They explain these findings with differences in parental allocations of time and financial resources. Coile (2004) examines the effect of health shocks on the labor supply of spouses. She finds that a spouse's health shock elicits only a small labor supply increase for men and no significant increase for women.

The second strand examines the effects of child bereavement on parents' outcomes. In a Swedish study, van den Berg et al. (2017) examine the effects of child bereavement on parental labor market outcomes, health, and family outcomes. They find that losing a child has adverse effects on labor income, employment status, marital status, and hospitalization.[5]

The third strand examines the effects of having a health shock on own outcomes. García-Gómez et al. (2013) examine the effects of having a health shock on own employment and income, and find that a health shock, defined as an acute hospital admission, lowers employment probability by 8% and causes a 5% loss of income two years after the health shock. They find no recovery in either employment or income four years later. Their findings also show substantial negative spillovers within the household. The probability that the spouse is working

[3]There is a growing literature using the event study approach to examine the effects of a variety of shocks, e.g., the economic consequences of hospital admissions (Dobkin et al., 2018); the health effects of increased access to primary care (Bailey and Goodman-Bacon, 2015); parental layoffs and income losses on children's long-term outcomes (Hilger, 2016); the short-and long-term effects of criminal victimization (Bindler and Ketel, 2019); the long-run effects of a financial windfall on saving behavior (Druedahl and Martinello, 2016); the effects of inheritance on wealth inequality (Nekoei and Seim, 2019).

[4]This question is also related to the literature examining parental investments in children. E.g., Carneiro and Ginja (2016) study the impact of permanent and transitory shocks to income on parental investments in children and find that parental inputs respond to permanent income shocks. Yi et al. (2015) study how children's health shocks affect intra-household resource allocation and the human capital formation of children. They find that compared with the twin sibling who does not suffer from a negative health shock, the twin sibling who does experience a health shock receive more health investment but less educational investment.

[5]The effect of child bereavement on health has also been studied in the health literature. For example, Rogers et al. (2008) examine the effects of child bereavement on a range of long-term outcomes. They find that bereaved parents report more depressive symptoms, poorer well-being, more health problems, and are more likely to have experienced a depressive episode and marital disruption compared to comparison parents up to 18 years after the death of a child.

is reduced by around 1 percentage point and spousal income falls by 2.5% two years after the health shock. Halla and Zweimüller (2013) use accidents occurring on the way to and from work as negative health shocks to identify the causal effect of health on labor market outcomes. They find that after initial periods with a higher incidence of sick leave, injured workers are more likely to be unemployed, and a growing share of them leave the labor force via disability retirement, while injured workers who manage to stay in employment incur persistent earnings losses. Also Dano (2005) uses road injuries to investigate the effects on labor market outcomes. She finds negative effects on disposable income for older individuals and for those with lower initial incomes. She also finds a significant negative effect on employment for males. Lindeboom et al. (2016) examine the relationship between health shocks and the onset of disability and employment outcomes, and find that experiencing a health shock increases the likelihood of the onset of disability, while it does not have a direct effect on employment at older ages.

3 Institutional Background and Data

3.1 The Norwegian Social Security System

Norway has a universal tax-financed social security system. The social security system provides insurances, such as retirement pension and unemployment insurance, and health-related insurances, such as sick pay, temporary social security benefits, and disability insurance. All Norwegians are eligible to apply for the insurances. However, there are requirements of prior income for some of the schemes, such as unemployment benefits and sick pay.

The healthcare system in Norway is a universal system, and consist of primary healthcare services and specialist healthcare services. The primary healthcare services are provided by general practitioners (GPs), and is organized as a list system in which all Norwegian citizens belong to a specific GP's list. The GPs are responsible for providing primary healthcare services, such as consultations, preventive care, and drug prescriptions, and they are the first instance an individual meet when seeking healthcare. The GPs are also responsible for referring patients to specialist healthcare services. The specialist healthcare services are organized in four regional areas, in which each region provides somatic and psychiatric hospital services, which offers both inpatient (overnight stays), and outpatient (day treatments and shorter consultations) services, as well as other services, such as rehabilitation institutions.

Sickness insurance in Norway is mandatory and covers all individuals who have been employed at the same employer for at least four weeks. Generally, workers are entitled to at least three days of self-reported absence per spell, but in some workplaces, workers are entitled to up to eight days. For absences lasting more than three (eight) days, medical certification is required. The first 16 days of absence are covered by the employer, while day 17 onwards is covered by the Norwegian Labor and Welfare Administration. The replacement rate is 100%

up to an amount of 6 G[6] (approximately 66,000 USD in 2019) from the first day of absence up to one year. Parents with one or two children below age 13 are entitled to 10 care days, and parents with three or more children below age 13 are entitled to 15 care days during a year.

After one year of sickness absence, eligible individuals go on to temporary disability insurance, which can at most last up to four years. During this period, the aim is to return the individual to the labor force. If this is not possible, the individual can apply for permanent disability insurance. The disability benefits provides partial earnings replacement for individuals in working age that are unable to work due to physical or mental health problems. The level of disability benefits is determined based on previous earnings.

3.2 Data Sources

The data used in this paper comes from several administrative registers and is merged by individual identification numbers.

Data on the labor market outcomes comes from two registers provided by Statistics Norway: the tax register, which contains information on individual labor and capital income as well as welfare benefits from 1993–2014, and the FD-Trygd register, covering all disability insurance use from 1992–2014. Statistics Norway also provides data on individual background information, including gender, age, immigrant status, and education level, covering the entire resident population of Norway from 1967–2014.

The Control and Distribution of Health Reimbursement database (KUHR) provides information about the use of primary healthcare services from 2006–2014. For each encounter, it provides a report of procedures used and the main diagnosis given by ICPC-2 codes[7]. The Norwegian Patient Registry (NPR) provides data on the use of specialist healthcare services from 2008–2014. It includes all somatic and psychiatric hospital admissions, both inpatient (overnight stays) and outpatient (day treatments and shorter consultations), and information about the related diagnosis given by ICD-10 codes[8]. The Norwegian Labor and Welfare Administration (NAV) provides data on all sickness absence certified by a GP from 1995–2014. For each certified sickness absence spell, the register has information on start and end dates as well as the related diagnosis given by ICPC-2 codes.

[6] G is an inflation-adjusted unit for calculation of social benefits in Norway.

[7] The International Classification of Primary Care (ICPC) is a classification method for primary care encounters. It classifies the patient's reason for the encounter and the related diagnosis, as well as the procedures done by the primary healthcare service.

[8] ICD-10 is the International Statistical Classification of Diseases and Related Health Problems, a medical classification list by the World Health Organization. It contains codes for diseases, signs and symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases.

3.3 Definition of the Health Shock

The health shock is defined as having an acute overnight hospital admission, given that the child did not have a hospital admission the year before the shock, ensuring that there is no anticipation the year before the health shock. Acute unplanned hospital admissions cannot be postponed since immediate treatment is deemed necessary and is often due to serious illnesses, which increases the exogeneity requirement of the health shock.

The children experiencing a health shock spend on average 2.35 nights in hospital after the initial admission. There is a large variation in this number, ranging from one night to 186 nights. Of the children, 89% are admitted from home, 3% are admitted directly from the accident site, and 1.35% from the emergency room. While 4% of the children are admitted to other units in the specialist healthcare services after the initial admission, 95% go home after they are discharged. Overall, 63% of the children have no surgical procedures, 17.7% have one surgical procedure, 8.7% have two, 5% have three, and the rest have between 4–20 surgical procedures. This includes all types of surgeries and procedure done, for example, head injury surgery. A total of 85% of the children have no medical procedures, 8% have one medical procedure, 4% percent have two, and the rest have between 4–20 medical procedures. This includes all examinations and monitoring procedures done.

Table 2 presents summary statistics of the diagnoses related to the health shocks, given by ICD-10 codes. The most common health shock is related to injuries, poisoning and certain other consequences of external causes, accounting for 33% of all health shocks. In this diagnosis group, the most common diagnosis is traumatic head injury, which is defined as something that occurs when an external force injures the brain, accounting for 20% of the diagnoses in this group. The next diagnoses groups are: diseases of the digestive system, in which acute appendicitis is the most common diagnosis, accounting for 12.6% of the health shocks; diseases of the respiratory system, in which pneumonia, acute tonsillitis, and asthma are the most common diagnoses, accounting for 7.9% of the health shocks; infections, which accounts for 3.9% of the health shocks.

Of the health shocks, 1.4% are related to cancer and diseases of blood, and 1.9% are related to mental health problems. These diseases would be very interesting to examine separately, as they may have severe long-term effects. However, due to small sample sizes, this is not possible.

3.4 Outcome Variables

I investigate four main labor market outcomes: sick pay, which is the social benefit received during sickness absence from work; log(labor income), defined as the logarithm of labor income; employment, defined as a dummy variable that is equal to one if an individual have a labor income above 1 G (approximately 11,000$ in 2019) during a year; the probability of re-

ceiving disability benefits, defined as a dummy variable that takes the value one if an individual receives disability benefits during a year. These outcomes are chosen to get a comprehensive picture of the effects of the health shock on parents' labor market attachment. Sickness absence is the first response to a negative health shock for parents. Parents can be on sickness absence up to one year and are then transferred to other programs if they are eligible. The question is whether parents go back to work after the initial shock, or whether they get transferred to other programs and ultimately leave the labor force permanently. To answer this question, I look at labor income, employment, and permanent disability benefits use.

For primary healthcare service use, I examine all GP consultations per year in total, and all emergency room (ER) visits per year in total, as well as GP consultations related to psychological and musculoskeletal diagnoses, as a large body of evidence in the health literature has shown that psychological stress can cause both musculoskeletal diseases, such as headache and pain, and psychological issues, such as stress and anxiety (Cho et al., 2003; Edwards et al., 2016; Meints and Edwards, 2018). For specialist healthcare service use, I examine all somatic hospital admissions per year in total as well as all psychiatric specialist healthcare use per year in total. To capture the intensive and extensive margins of healthcare use, the health outcomes are defined in two ways: as the total number of events per year and as an indicator equal to one if an individual uses the healthcare services at all during a year.

Furthermore, I consider sickness absence per year in total as well as sickness absence related to musculoskeletal and psychological diagnoses separately. As with the health outcomes, the sickness absence outcomes are defined in two ways, capturing both the intensive and extensive margins of sickness absence use: as the total number of sickness absence days per year and as the probability of having sickness absence at all during a year.

I study health shocks that occur in the years 2009–2014. For the primary healthcare service use, sickness absence, and the labor market outcomes the data is available from 2006, making it possible to examine the pre-trends three years before the health shock for all cohorts experiencing a health shock. The data on specialist healthcare service use is available from 2008, limiting the pre-shock observations to one (2009), two (2010), and three (2011–2014).

3.5 Sample Selection and Descriptive Statistics

To construct the main sample, I start with the entire Norwegian population that has at least one somatic hospital admission in the period 2008–2014, which are the years the specialist healthcare data covers. For each year, I keep all children between age 5 and 18. I start at five years old to avoid pregnancy and birth related issues. This is 668,199 children with 3,673,019 hospital admissions in the period 2008–2014. To ensure that I capture severe health shocks, I impose three sample restrictions. First, the child must have an acute hospital admission. Second, the child cannot have a hospital visit the year prior to the health shock. Third, the child

cannot have an acute hospital visit in 2008 because that is the first year the data covers, which means that I cannot ensure that these children did not have a hospital visit the year before the health shock. Of children aged 5–18, 64% do not meet these criteria, and are therefore dropped. Furthermore, I keep only the children that are admitted to an inpatient stay, which is 19% of the sample, and drop those that do not have an overnight stay, which is 21% of the sample. To ensure that I do not capture health shocks related to pregnancy and birth outcomes, I drop all children that has admissions related to the pregnancy diagnosis group, which are 102 children. Finally, 211 of the children have no registered parents, and are therefore dropped. The final sample consists of 53,494 children and their parents.

Table 1 presents summary statistics of the main analysis sample. The mean age at which the children have a health shock is 12 years old, they are on average born in 1999, 46% of the children are girls, and 3% are children of single parents. Approximately 20% of the sample are immigrants. The mothers are on average born in 1970 and the fathers in 1967. The parents have on average approximately 13 years of education in 2008 (the year before the first cohort has a health shock), which corresponds to having a high school degree. In 2008, 8% of the mothers and 3% of the fathers were still in education.

4 Empirical Strategy

The event study approach is an extension of the differences-in-differences design, used to estimate the dynamic effects of discrete shocks and non-transient treatments.[9] The event study approach exploits variation in the timing of an individual's treatment, which allows for identification of the path of treatment effects even when there are no pure control individuals. The treated individuals are grouped into cohorts based on when they first receive treatment, and for each cohort, relative time to initial treatment can be defined. The crucial feature of event studies is the presence of multiple cohorts, which serves as a source of identification for estimation. Within a cohort, calendar time and relative time are collinear, so it is not possible to identify dynamic treatment effects separately in the presence of time trends. However, with multiple cohorts it is possible to separate these two sets of effects by comparing the trends in average outcomes between treated cohorts and cohorts that receive treatment at a later point in time, given the parallel trends and no anticipation assumptions. For any given cohort, the differences in trends identify the average treatment effect for this cohort at different relative times. Abraham and Sun (2019) call these cohort-specific average treatment effects on the treated (CATT). Any convex average of CATT is a causally interpretable estimate of the average treatment effect on the treated (Abraham and Sun, 2019).

[9]The empirical strategy used in this paper relates to a recent literature that examines linear two-way fixed effects models with staggered adoption to treatment (Abraham and Sun, 2019; Borusyak and Jaravel, 2017; Goodman-Bacon, 2018; Callaway and Sant'Anna, 2019; Athey and Imbens, 2018).

4.1 The Event Study in a Potential Outcomes Framework

Following Abraham and Sun (2019) and Novgorodsky and Setzler (2019), I consider a setting with $i = 1, \ldots, N$ individuals and $t = 1, \ldots, T$ time periods, which in my setting are years. For individual *i*, the observed outcome in year *t* is denoted by $Y_{i,t}$. E_i denotes the year of initial treatment. $Y_{i,t}(e)$ denotes the potential outcome for individual *i* at time *t*. This is only observed when $E_i = e$. In my context $Y_{i,t}$ are the parents' health and labor market outcomes, and E_i is the year that a child has a negative health shock. The parameter of interest is the average treatment effect on the treated, $ATT_t(e)$:

$$ATT_t(e) \equiv \mathbb{E}[Y_{i,t}(e) - Y_{i,t}(\infty)|E_i = e] \tag{1}$$

where $Y_{i,t}(\infty)$ is the outcome an individual *i* would have at time *t* if counterfactually assigned treatment at time ∞ (i.e., never treated). This is the average difference in $Y_{i,t}$ that is due to being treated at *e* instead of ∞, among those who are treated at *e*. In my context $\mathbb{E}[Y_{i,t}(2009) - Y_{i,t}(\infty)|E_i = 2009]$ is the difference in parents' outcomes, for example, employment in $t = 2010$ for parents whose child has a negative health shock in 2009 as opposed to parents whose child has a negative health shock at a later point in time. The average treatment effect on the treated (ATT) for a given cohort corresponds to the cohort-specific average treatment effect (CATT) (Abraham and Sun, 2019).

To compare effects across cohorts in an event study framework, the calendar time *t* can be changed to a relative time index, *l*, which denotes the time periods relative to treatment. For cohort *e*, *l* ranges from $-e$ to $T-e$ since we observe e periods before initial treatment and $T-e$ after initial treatment. The causal parameter of interest, $CATT_{e,l}$, can therefore be defined as:

$$CATT_{e,l} \equiv \mathbb{E}[Y_{i,e+l}(e) - Y_{i,e+l}(\infty)|E_i = e] \tag{2}$$

4.2 Identifying Assumptions

For the identification of the $CATT_{e,l}$, three assumptions are necessary (Abraham and Sun, 2019):

Assumption 1. *Parallel trends.* $\mathbb{E}[Y_{i,s}(\infty) - Y_{i,t}(\infty)|E_i = e] = \mathbb{E}[Y_{i,s}(\infty) - Y_{i,t}(\infty)|E_i = e']$ *for all* $e \neq e'$ *and all* $t \neq s$*. That is, for any two observed cohorts* e *and* e'*, the change over time they would have had in the absence of treatment is the same.*

In my setting, parallel trends requires that for parents whose child had a health shock in 2009 and parents whose child had a health shock in 2010–2014, if none of their children had a health shock, they would have experienced the same change in mean outcomes.

Assumption 2. *No anticipation.* $Y_{i,t}(e) = Y_{i,t}(\infty)$*, for all* $t < e$ *and for all e. This says that prior to the onset of treatment, outcomes do not depend on the time at which treatment will occur.*

In my setting, no anticipation requires that, for example, in 2008 parents of children who had a health shock in 2009 did not have different health and labor market outcomes than they would have had if their child had never had a health shock.

Assumption 3. *Treatment effect homogeneity. For each lag of treatment* $l \geq 0, CATT_{e,l}$ *does not depend on cohort* e *and is equal to* $CATT_l$*.*

In my setting, treatment effect heterogeneity can occur for several reasons. If, for example, treatment effects differ with age, and since age is correlated with cohort there will be heterogeneous treatment effects. This is discussed in more detail below.

4.3 Identification Challenges

Whereas violations of the parallel trends and no anticipation assumptions invalidates the event study, violation of the treatment effect homogeneity assumption makes the event study harder to interpret (Abraham and Sun, 2019). There are two main identification challenges with the event study approach given that the parallel trends and no anticipation assumptions hold, which may be corrected for using the methods described below: treatment effect heterogeneity and age and sample composition effects.

4.3.1 Treatment Effect Heterogeneity

When different cohorts have different profiles of dynamic treatment effects, the treatment effect homogeneity assumption is violated. There are several reasons why heterogeneity in treatment effects can arise using an event study design. For example, cohorts may differ in their covariates, which may affect how they respond to treatment. If, for example, treatment effects differ with age and since age is correlated with cohort there will be heterogeneous effects. After controlling for covariates, cohorts may still vary in their response to the treatment if individuals select treatment timing based on treatment effects (Heckman et al., 2006). In addition to these two sources of heterogeneity, treatment effects may vary across cohorts due to calendar time-varying effects (e.g., macroeconomic conditions could affect the effects on labor market outcomes across cohorts) (Abraham and Sun, 2019).

The level of variation used to identify dynamic treatment effects separately from calendar time trends are cohorts. They are therefore also the key level of concern for confounding heterogeneity. In the dynamic two-way fixed effects specification, which is the one I use, lags and leads of the treatment indicator are included as regressors. Abraham and Sun (2019) show that the dynamic specification does not return causally interpretable estimates when treatment

effects are heterogeneous. The estimand associated with a particular lead or lag, *l*, can then be a non-convex average of the $CATT_{e,l}$ from all periods, not just *l*. Thus the interpretation of this estimand as an average treatment effect *l* periods since initial treatment is confounded by two factors: the inclusion of spurious terms reflecting treatment effects from periods other than *l* and the possibility of negative weights on some $CATT_{e,l}$ (Abraham and Sun, 2019).

There are two relevant dimensions of heterogeneity in $CATT_{e,l}$: variation within a given cohort across lags (stationarity and non-stationarity) and within a given lag across cohorts (cross-cohort homogeneity and heterogeneity) (Abraham and Sun, 2019).

Definition 1. Stationarity and non-stationarity. If $CATT_{e,l} = CATT_{e,l'}$ for all lags of treatment $l, l' \geq 0$, then treatment effects for cohort *e* are stationary. If $CATT_{e,l} \neq CATT_{e,l'}$ for all lags of treatment $l, l' \geq 0$, then treatment effects for cohort *e* are non-stationary.

Definition 2. Cross-cohort homogeneity and heterogeneity. For each lag of treatment $l \neq 0$, if $CATT_{e,l}$ does not depend on *e*, then treatment effects are homogenous across cohorts. If for any $l \neq 0$, $CATT_{e,l}$ varies by *e*, then treatment effects are heterogeneous across cohorts.

While stationary effects affect individuals immediately upon treatment and on average persist at the same level for all treated periods, non-stationary effects occurs when there is learning and adoption to the treatment over time. Cross-cohort homogeneity means that each cohort experience the same path of treatment effects on average and that $CATT_{e,l}$ at any given *l* is the same across cohorts. While treatment effects do not need to be the same across cohorts in every lag to hold, for heterogeneity to occur, treatment effects just need to differ across cohorts in one lag (Abraham and Sun, 2019).

The solution to the challenge with confounding due to treatment effect heterogeneity is to examine cohort-specific effects and pooling heterogeneous effects, rather than assuming homogenous effects across cohorts (Abraham and Sun, 2019). The pooled estimate when imposing homogeneity in the estimator can be found by using the cohort stacking approach described in the next section.

4.3.2 Age and Sample Composition Effects

In addition to the challenge with heterogeneous treatment effects, there are also challenges related to sample composition and confounding effects due to unbalance in the covariates of the cohorts who are treated and those who are yet to be treated.

Possible solutions to these challenges are reweighting using propensity score matching and stacking cohorts.

Reweighting using propensity score matching. Abadie (2005) suggests using covariate balancing to correct for anticipation and deviations from parallel trends. By controlling for the

effects of the covariates, the identification is extended to those cases in which observed compositional differences between treated and non-treated cohorts causes non-parallel dynamics in the outcome variables. The parallel trends assumption is then extended to condition on covariates.

Assumption 4. *Parallel trends conditional on the covariates.* $\mathbb{E}[Y_{is}(\infty) - Y_{it}(\infty)|X, E_i = e] = \mathbb{E}[Y_{is}(\infty) - Y_{it}(\infty)|X, E_i = e']$ *for all* $e \neq e'$ *and all* $t \neq s$*. That is, for any two observed cohorts* e *and* e'*, the change over time they would have had in the absence of treatment is the same, conditional on the covariates.*

In practice, to produce estimates of the average treatment effect on the treated conditional on this assumption, Abadie (2005) proposes a weighting scheme using propensity score matching. As a first step, the propensity score, $P(E_i = e|X)$, is estimated. In my setting, I use this approach to adjust for differences in the age of the children at the time of initial treatment, gender, immigrant status, mothers' age at first birth, and parents' education level.

Since the identification is attained after controlling for the effect of some covariates X, it will be required that for each given value of the covariates there is some fraction of the population that remains untreated and can be used as controls.

Assumption 5. *The propensity score.* $P(E_i = e) > 0$ *and with probability one* $P(E_i = e|X) < 1$

This assumption implies that the support for the propensity score for the treated is a subset of the support for the propensity score for the untreated (Abadie, 2005).

Stacking cohorts. Suppose we have several treatment cohort and several control cohorts $e < e' < e''$. When $e = e' - 1 = e'' - 2$, so that e, e', e'' are each in adjacent years, e'' can be used twice as control group, as it can be the control group for e both at $t = e$ and $t = e'$, and the control group for e' at $t = e'$ (Novgorodsky and Setzler, 2019). Consider at setting when $t = e$ and $s < e$:

$$Y_{i,t} = \alpha(e, \{e', e''\}) + \delta(e, \{e', e''\})1_{t=e} + \tau(e, \{e', e''\})1_e + \gamma_{e,s}(e, \{e', e''\})1_{t=e}1_e + \epsilon_{i,t}, \text{ for } E_i \in \{e, e', e''\} \text{ and } t \in \{s, e\} \quad (3)$$

$$Y_{i,t} = \alpha(e', e'') + \delta(e', e'')1_{t=e+1} + \tau(e', e'')1_{e+1} + \gamma_{t=e+1,s}(e', e'')1_{t=e+1}1_{e'} + \epsilon_{i,t}, \text{ for } E_i \in \{e', e''\} \text{ and } t \in \{s, e+1\} \quad (4)$$

Equation (3) identifies $ATT_{t=e}(e)$ when pooling the e', e'' cohorts as control groups, while equation (4) identifies $\gamma_{t=e+1,s}(e', e'') = ATT_{t=e+1}(e)$ using the e'' cohort as the control group. That is, the control group in equation (3), e', is the treatment group in equation (4).

By stacking the data with duplicates and introducing the reference cohort variable, r, it is possible to run a single regression that uses e' as the control group for e but also uses e' as a treatment group in which e'' is its control group. In equation (3), $r = e$ is the reference cohort for both e' and e''. In equation (4), $r = e'$ is the reference cohort for e''. There are thus two copies of the e' observation, but one is coded with $r = e$ and the other is coded with $r = e'$. To estimate $\gamma_{e,s}(e, \{e', e''\})$, the regression that uses e as the treatment group and e', e'' as the control groups with and indicator $1_{r=e}$ is fully interacted, and to estimate $\gamma_{t=e+1,s}(e', e'')$, the regression that use e' as the treatment group and e'' as the control group with an indicator $1_{r=e'}$ is fully interacted. Then both these regressions can be estimated, because they are fully interacted with the 1_r indicators so that this is equivalent to estimating the two regressions separately.

An important aspect of this approach is that the control groups available for different t times will vary. For example, as my health shock data is from 2009–2014, for $e = 2011$, then cohorts $e' \in \{2012, 2013, 2014\}$ will be available for $t = 2011$, $e' \in \{2013, 2014\}$ will be available for $t = 2012$, and $e' \in \{2014\}$ will be available for $t = 2013$. This means that is important to make sure to use the same cohorts that are available at t when estimating the mean at s. If not, the difference $(\mathbb{E}[Y_{i,t}|E_i = e' \text{ or } E_i = e''] - \mathbb{E}[Y_{i,s}|E_i = e' \text{ or } E_i = e''])$ will be affected by composition changes between s and t rather than by only the time effects this term is meant to capture (Novgorodsky and Setzler, 2019). It is therefore important to include individual fixed effects in the estimating equation.

4.4 Estimating Equation

The treatment effects in this event study are estimated by a dynamic linear regression specification with two-way (individual and calendar time) fixed effects, given by the following estimating equation:

$$Y_{i,t} = \alpha_i + \gamma_t + \sum_{l=-3}^{T=3} \mu_l D_{i,t}(l) + \epsilon_{i,t} \tag{5}$$

where *Y* is the outcome of interest, *i* indexes individual and *t* indexes year. α_i is a set of individual fixed effects (i.e. child fixed effects for the first stage outcomes, mother fixed effects for the mothers' outcomes, and father fixed effects for the fathers' outcomes), and γ_t is a set of year fixed effects. $D_{i,t}(l)$ is an indicator for being *l* time periods relative to individual *i's* initial treatment period, $l = 0$. μ_l are the estimated treatment effect coefficients. Standard errors clustered at the level of the individual (child for children's outcomes, mother for mothers' outcomes, and father for fathers' outcomes) are included in all regressions.

In this estimation model, two types of multicollinearities arises. The first arises from the linear dependence between individual (or time) fixed effects and relative time indicators. The second arises from the linear relationship between cohort, calendar time, and relative time

(Abraham and Sun, 2019). This is closely related to the age-cohort-time problem discussed by Borusyak and Jaravel (2017), among others. At least two relative time indicators therefore needs to be excluded. In practice, it is common to exclude more than two relative times from Equation 5, for example, all leads greater than *K* and all lags greater than *L* (Abraham and Sun, 2019). I exclude all $K \geq 3$, all $L \geq 3$, and $l = -2$, which is the relative time that the treatment effects are compared to. The relative time period is set to $l = -2$ so that it is possible to examine that there is no anticipation in period $l = -1$.

In practice, to take into account the issues discussed in this section, in the main estimation specification I first match on six covariates: age of the children at the time of initial treatment, gender, immigrant status, mothers' age at first birth, and parents' education level, ensuring that the distribution of these covariates are similar for treatment and control cohorts in each time period before and after the health shock. Second, in a setting utilizing variation in timing of event there may be mechanical differences in age across treated cohorts. I therefore weight by the size of the cohorts using inverse probability weighting (IPW), and the IPW model I use is a logit model. Third, the minimum control gap is set to one. This is the minimum time periods a head that a cohort must receive treatment in order to be included in the control group. The maximum control gap is set to four. This is similar to the minimum control gap, but for the maximum number ahead. The regressions are run on a maximum of seven pre-shock years for the primary healthcare and labor market outcomes (the data on these outcomes is available from 2006–2014), and a maximum of five pre-shock years for the specialist healthcare outcomes (the data on these outcomes is available from 2008–2014). Finally, time period T (2014) is dropped in all the regressions since all the cohorts are treated in the last period.

5 Results

In this section, I first present the results for the main sample of families in which a child has a health shock between age 5 and 18. Second, I present the heterogeneity results on subsamples defined by the children's age at the time of the health shock, the diagnosis related to the health shock, and the severity of the health shock. I present the main results both graphically and in tables with estimation results based on the regression in Equation (5). The figures show estimated coefficients and 95% confidence intervals three years before and three years after the health shock of the child occurs. In the tables showing the effects on the health outcomes, Panel A shows the estimated results on the outcomes defined as the total number of incidents per year, and Panel B shows the estimated effects on the outcomes defined as the probability of an event per year. I examine these two measures of the health outcomes to capture both the intensive and extensive margins of healthcare use and sickness absence.

5.1 First Stage: Children's Health

Figure 1 presents the paths of the estimated coefficients and 95% confidence intervals of the use of three healthcare services for the children experiencing a health shock: GP consultations, ER visits, and somatic hospital visits. The figure shows that there are some statistically significant differences between the treatment and control cohorts in the pre-shock period. However, these differences are small compared to the effects of the health shock. There is a drop in somatic hospital visits the year before the health shock. This is due to the sample restriction that all children included in the sample are restricted on not having a somatic hospital visit the year before the health shock.[10]

Table 3 presents the corresponding estimation results. The results show instantaneous and large effects of the health shock on the three outcomes. As can be seen in Column 1, in the year of the health shock (0), the total number of GP consultations increases by 62%, while the probability of having a GP consultation increases by 28% (16 percentage points from a pre-shock mean of 58%). The number of ER visits increases by 180%, and the probability of having and ER visit increases by 164% (35 percentage points from a pre-shock mean of 21%) (Column 2). The number of somatic hospital visits increases by 556%, and the probability of having a somatic hospital visit by 406% (85 percentage points from a pre-shock mean of 21%) (Column 3). All these effects are statistically significant.

As can be seen from the coefficients for the three years following the health shock, the health shock has persistent health effects for the children. There are positive and statistically significant effects on the total number of GP consultations of 33%, 26%, and 24% in year one, two, and three after the health shock. This is also true for ER visits and somatic hospital visits, with increases of 42%, 40%, and 36% in year one, two, and three for the total number of ER visits, and 239%, 180%, and 170% for the total number of somatic hospital visits.

5.2 Mothers' Health and Labor Market Outcomes

The effects of the health shock on mothers' labor market outcomes are presented in Figure 3 and Table 4, and the effects on the health outcomes are presented in Figure 5 and Table 5. Figure 5 shows the effects on the health outcomes measured as the probability of an event, while Figure A.1 in the Appendix shows the estimated effects on the outcomes measured as the total number of incidents.

Labor market outcomes. Figure 3 presents the estimated coefficients and 95% confidence intervals for the four main outcomes: labor income, employment, sick pay, and probability of disability insurance, based on the regression in Equation (5). The figure first provides a graph-

[10] As a robustness check, I change this restriction to not having a hospital visit three years before the health shock. The reduced form results remain similar. However, due to fewer available cohorts and lower sample size, I will not use this as the baseline. The robustness check is discussed further in Section 6.

ical confirmation of the parallel trends assumption. That is, that the paths of the coefficients in the period before the health shock are flat, ensuring that there are no differences between treatment and control cohorts in the pre-shock period. Second, it shows the estimated effects of the health shock from the year of the health shock up to three years after the health shock. As can be seen from the figure, the health shock has immediate and persistent statistically significant effects up to three years after the health shock on labor income, employment, and sick pay. There are also long-term effects on the probability of receiving disability benefits. As the process of receiving permanent disability benefits takes some time, these effects are expected.

Table 4 shows the corresponding estimated effects. Column 1 shows the effect on labor income, Column 2 on employment, Column 3 on sick pay, and Column 4 on the probability of receiving disability benefits. As can be seen from the coefficient for the year of the health shock (0), in the year of the health shock, mothers experience a decrease in labor income by 7.6%, and a decrease in employment by 0.6%, a decrease of 0.5 percentage points from a pre-shock mean of 80%. At the same time, mothers have an increase in sick pay by 15%.

The effects are persistent up to three years after the health shock, and the treatment effects are increasing over time. Three years after the health shock, mothers have a 19% lower labor income compared to two years before the health shock, and a 2.1% lower probability of being in employment. The amount of sick pay mothers receive are 12% higher compared to two years before the health shock, and they have an increased probability of receiving disability benefits of 100%, a 1 percentage point increase from a pre-shock mean of 1%.

Health care use and sickness absence. As the labor market outcomes may be affected through a deterioration of health, I go on to present the effects of the health shock on mothers' health outcomes. Figure 5 presents the estimated coefficients and 95% confidence intervals for the health and sickness absence outcomes for mothers. The outcomes are measured as the probability of an event. The figure shows that the trends of the outcomes in the pre-shock period are relatively flat, verifying that the parallel trends assumption holds for the mothers' health outcomes. The only exception is somatic hospital visits, in which there are some small differences between treatment and control cohorts in the pre-shock period. Furthermore, the figure show an increase in both GP consultations and sickness absence related to psychological diagnoses in the year of the health shock. There are also statistically significant increases in the probability of having a GP consultation, the probability of having a somatic hospital visit, and sickness absence in total in the years following the health shock. These effects are mainly related to pregnancy diagnoses, and are driven by treated mothers that have more children compared to control mothers in the years following the health shock.

The corresponding estimated effects on the use of healthcare services and sickness absence for mothers are presented in Table 5. In the table, Panel A shows the estimated results on the outcomes measured as the total number of incidents, and Panel B shows the estimated effects

on the outcomes measured as the probability of an event. Columns 1–4 show the effects on the use of primary healthcare services, specifically GP consultations in total, GP consultations related to musculoskeletal diagnoses, GP consultations related to psychological diagnoses, and ER visits. Columns 5 and 6 show the estimated effects on the use of somatic and psychiatric specialist care. Columns 7–9 show the estimated effects on sickness absence in total, and sickness absence related to musculoskeletal and psychological diagnoses.

As can be seen from the coefficient for the year of the health shock (0), the probability of having a GP consultation related to psychological diagnoses increases by 7% in the year of the health shock, an increase of 1.2 percentage points from a pre-shock mean probability of having a GP consultation related to psychological diagnoses of 17%. At the same time, mothers' probability of having sickness absence related to psychological diagnoses increases by 18%, a 0.9 percentage point increase from a pre-shock mean probability of having sickness absence related to psychological diagnoses of 5%. Mothers have an increase in the total number of GP consultation by 2.8% and the total number of somatic specialist healthcare visits by 6.9% the year of the health shock. These effects are mainly driven by pregnancy related diagnoses, and are explained by the fact that mothers have an increased probability of having more children after the child has a health shock. There are no effects of the child's health shock on mothers' use of psychiatric specialist healthcare or ER visits.

Except for the increased number of GP consultations, somatic hospital visits, and sickness absence related to pregnancy, the effects on the use of healthcare services and sickness absence are not persistent in the long term. There are some negative effects on GP consultations and sickness absence related to psychological illnesses in year two and three after the health shock. This is in line with expected effects, as there at the same time is an increased probability of receiving disability benefits.

5.3 Fathers' Health and Labor Market Outcomes

The effects of the health shock on fathers' labor market outcomes are presented in Figure 7 and Table 6, and the effects on the health outcomes are presented in Figure 9 and Table 7. Figure 9 shows the effects on the health outcomes measured as the probability of an event, while Figure A.3 in the Appendix shows the estimated effects on the outcomes measured as the total number of incidents.

Labor market outcomes. Figure 7 presents the paths of the estimated coefficients and 95% confidence intervals for the four main outcomes: labor income, employment, sick pay, and probability of disability insurance, based on the regression in Equation (5). The figure first provides a graphical confirmation of the parallel trends assumption. The pre-shock trends are relatively flat for all the outcomes, ensuring that the parallel trends assumption holds. Second, it shows the estimated effects of the health shock from the year of the health shock up to three

years after the health shock. The figure shows that there are short-term effects on sick pay, and long-term effects on labor income, employment, and the probability of receiving disability benefits.

Table 6 shows the corresponding estimated effects. Column 1 shows the effect on labor income, Column 2 on employment, Column 3 on sick pay, and Column 4 on the probability of receiving disability benefits. As can be seen from the coefficient for the year of the health shock (0), in the year of the health shock, fathers experience a reduction in labor income by 3.3%. The amount of sick pay they receive increases by 9.5%. There is no reduction in the probability of being in employment for fathers in the year of the health shock.

The effects are persistent up to three years after the health shock, and the treatment effects are increasing over time. Three years after the health shock, fathers have an 18% lower labor income compared to two years before the health shock, and a 1.8% lower probability of being in employment. Compared to two years before the health shock, they have an increased probability of receiving disability benefits of 70%. The effect on sick pay is not persistent in the long term for fathers.

Health care use and sickness absence. Figure 9 presents the estimated coefficients and 95% confidence intervals for the healthcare use and sickness absence outcomes for fathers. The outcomes are measured as the probability of an event. First, the figure shows that the trends of the outcomes in the pre-shock period are relatively flat, verifying that the parallel trends assumption holds for the fathers' health outcomes. Second, the figure shows that there are no large effects of the health shock on fathers' healthcare use and sickness absence. Fathers have an increased probability of having a GP consultation related to psychological diagnoses (statistically significant on the 10% level), and an increased probability of sickness absence related to psychological diagnoses in the year of the health shock. There are no long-term effects on the healthcare use and sickness absence for fathers.

The corresponding estimated effects on the use of healthcare services and sickness absence for fathers are presented in Table 7. In the table, Panel A shows the estimated results on the outcomes measured as the total number of incidents, while Panel B shows the estimated effects on the outcomes measured as the probability of an event. Columns 1–4 show the effects on the use of primary healthcare services, specifically GP consultations in total, GP consultations related to musculoskeletal diagnoses, GP consultations related to psychological diagnoses, and ER visits. Columns 5 and 6 show the estimated effects on the use of somatic and psychiatric specialist care. Columns 7–9 show the estimated effects on sickness absence in total, and sickness absence related to musculoskeletal and psychological diagnoses.

In the year of the health shock, fathers have an increased probability of having a GP consultation related to psychological diagnoses by 3.6%, an increase of 0.4 percentage points from a pre-shock mean of 11% (statistically significant on the 10% level). In the year of the health

shock, fathers also have an increase in the probability of having sickness absence related to psychological diagnoses by 13%, an increase of 0.4 percentage points from a pre-shock mean of 3%.

5.4 Heterogeneity

To examine whether there are differences in the effects due to different cause, severity, and timing of the health shock, I assess heterogeneity by the type of health shock given by the related diagnosis, by the length of the initial hospital stay, and by age of the child at the time of the health shock.

5.4.1 Age

The age when the child experiences the health shock may affect the estimated effects because there may be differences between caring for a young child versus an older child, and because the age of the parents at the time of the health shock is different. There is no clear hypothesis on what is most important of these two dimensions. However, to examine whether the effects are different for parents of children that have the health shock at younger ages compared to older ages, I examine effects for children aged 5–12 separately and for children aged 13–18 separately.

Table 8 presents the effects on the number of GP consultations, ER visits, and somatic hospital visits for the children having a health shock in the two age groups separately. For GP consultations and ER visits, the largest effects are found in the group of children aged 13–18, both in the short term and in the long term. For somatic hospital visits, the largest effect in the year of the health shock is for the group of children aged 5–12. The long-term effects are largest in the group of children aged 13–18. The effects on GP consultations are statistically significant different between the two groups in all years, while the effects on ER visits are statistically significant different between the two groups in year one, two, and three after the health shock. For somatic hospital visits, the effects are statistically significant different between the two groups in the year of the health shock and in year three after the health shock.

Table 9 presents the estimation results on labor market outcomes for mothers of children aged 5–12 and of children aged 13–18 separately. As can be seen from the table, the short-term effects on all four outcomes are largest for the group of mothers of children aged 5–12. This is also true for the long-term effects on sick pay and the probability of receiving disability benefits. The long-term effects on labor income and employment are, however, larger for mothers of the group of children aged 13–18. With the exception of the effects on the probability of receiving disability benefits in the year of the health shock and the year after the health shock, there are, however, no statistically significant differences in the effects between the two groups.

Table 10 presents the estimation results on labor market outcomes for fathers of children

aged 5–12 and of children aged 13–18 separately. The effects on all four outcomes are largely driven by the group of fathers of children aged 5–12. The effects on labor income are statistically significant different between the two groups in year two and three after the health shock. This is also the case for the effect on employment in year three after the health shock.

5.4.2 Diagnosis

There may also be heterogeneity with respect to the diagnosis related to the health shock, because there may be different effects of different diagnoses. Some of the diagnoses are more severe and will have more long-term effects compared to the less-severe diagnoses. I therefore examine effects separately in subsamples defined by the four most prevalent diagnosis groups related to the health shocks. The four diagnosis groups examined are: injury, poisoning and certain other consequences of external causes; diseases of the digestive system; diseases of the respiratory system; certain infectious and parasitic diseases. It would be very interesting to examine effects for children that have a health shock related to cancer separately as well, as this is a very serious and possibly a long-term illness. However, due to the small number of health shocks related to cancer, the sample is not large enough to do this.

Table 11 shows the effects of the health shock on the number of GP consultations, ER visits and somatic hospital visits for the children experiencing a health shock for the subsamples defined by the diagnosis related to the health shock. The effects on all three outcomes in the year of the health shock for the group of children experiencing a health shock related to injury, poisoning and certain other consequences of external causes are statistically significantly different from the effects on the groups experiencing health shocks related to digestive illnesses, respiratory illnesses and infections. There are also statistically significant differences in the effects on GP consultations and ER visits between the children experiencing a health shock related to digestive illnesses compared to those experiencing a health shock related to respiratory illnesses in year two and three after the health shock. Finally, there are statistically significant differences in the effects between children experiencing a health shock related to respiratory illnesses compared to children experiencing a health shock related to infections on GP consultations in all years, and on ER visits in year one and three after the health shock.

Table 12 shows the estimated effects of the health shock on mothers' labor market outcomes for the subsamples defined by the diagnosis related to the health shock. Overall, the largest effects are found in the group in which the children experience a health shock related to injury, poisoning and certain other consequences of external causes. However, there are no statistically significant differences in the effects between the four subsamples on any of the outcomes.

Table 13 presents the corresponding estimated effects on fathers' labor market outcomes. The effects on labor income in the subsample in which the children experience a health shock related to digestive illnesses are statistically significantly different from the effects in the sub-

samples in which the health shocks are related to injury, poisoning and certain other consequences of external causes and respiratory illnesses in year one, two, and three after the health shock. There are also statistically significant differences in the effects in the subsample in which the health shock is related to respiratory illnesses compared to infection in the year of the health shock and up to three years after the health shock. When it comes to employment, there are statically significant differences in the effects in the year of the health shock between the subsample in which the children experience a health shock related to respiratory illnesses compared to injuries and infections, and between infections and the three other groups in year three after the health shock. There are no statistically significant differences between the subsamples in the effects on sick pay and the probability of receiving disability benefits.

5.4.3 Severity

Furthermore, I go on to examining heterogeneity by the severity of the health shock, defined by the length of the initial hospital stay. In this heterogeneity analysis, the main sample is divided in two, in which the first subsample consists of children who stay one night in hospital (approximately 60% of the main sample), and the second subsample children who stay longer than one night (approximately 40% of the main sample). In the second subsample, the length of the hospital stay ranges from two nights to 186 nights.

Table 14 shows the estimated effects on the number of GP consultations, ER visits, and somatic hospital visits for the children having a health shock in the two subsamples separately. The effects on hospital visits are statistically significantly different between the two subsamples in the year of the health shock and up to three years after the health shock. The effects on children that stay more than one night are larger than the effects on children that stay one night in both the year of the health shock and subsequent years. As these children most likely have the most severe health problems, these effects are in line with expected effects. There are also statistically significant differences in the effects on GP consultations in the year of the health shock and year one after the health shock, as well as in the effects on ER visits in the year of the health shock.

Table 15 shows the estimated effects of the health shock on mothers' labor market outcomes for the subsamples defined by the length of the hospital stay of the child. With the exception of sick pay in the year of the health shock and year one after the health shock, there are no statistically significant differences in the effects on mothers.

Table 16 presents the corresponding estimated effects on fathers' labor market outcomes. The effects for fathers are similar across the two groups, and there are no statistically significant differences in the effects.

6 Robustness

6.1 Anticipation

Suppose that for all cohorts, the parallel trends assumption holds, but the no anticipation assumption does not hold. That is, for each treated cohort with onset time e, individuals begin adjusting their outcome in anticipation of treatment as of period $e-k, k>0$. For a given cohort e, anticipation is analogous to moving the treatment onset time for all later cohorts k periods into the future.

When there is anticipation the approach described in Section 4 has to be adjusted for a given anticipation period. When stacking control observations for a given reference cohort $r=e$, only $E_i \in \{e'|e'>e+k\}$ can be used. For example, consider the case with five adjacent cohorts, i.e., $e=e'-1=e''-2=e'''-3=e''''-4$ and suppose that all cohorts begin adjusting their outcome in anticipation of being treated two periods prior to treatment, $k=2$. In this case, $\{e', e''\}$ would no longer be included in the set of control groups (Novgorodsky and Setzler, 2019). Thus, for the reference cohort $r=e$, the underlying regression for the $r=e$ subsample is now given by:

$$Y_{i,t} = \alpha(e,\{e''',e''''\}) + \delta(e,\{e'',e''''\})1_{t=e} + \tau(e,\{e''',e''''\})1_e + \gamma_{t=e,s}(e,\{e''',e''''\})1_{t=e}1_e + \epsilon_{i,t}, \\ for\ E_i \in \{e,e''',e''''\} \text{ and } t \in \{s,e\} \quad (6)$$

The estimated effects on the labor market outcomes from regressions in which anticipation in the year before the health shock is controlled for, for mothers are presented in Table A1 and for fathers in Table A2. As can be seen from the tables, the results remain similar to the baseline results when controlling for anticipation one year before the health shock.

6.2 Sample Selection

To ensure that the estimated effects in the main analysis is not affected by the restriction that all children included in the sample are restricted on not having a somatic hospital visit the year before the health shock, I change this restriction to not having a hospital visit three years before the health shock. Due to data limitations, I only include cohorts that have a health shock in 2011, 2012, 2013, and 2014. The sample is thus smaller than the main sample. This also means that I can only examine effects up to two years after the health shock.

The results from estimation on this sample for mothers are presented in Table A3 and for fathers in Table A4. As can be seen from the tables, the results remain similar to the baseline results when changing the restriction, ensuring that it is not the sample restriction that is driving the results.

7 Conclusion

In this paper, I examine the effects of a child's negative health shock on parental health and labor market outcomes using an event study approach. The event study approach exploits variation in the timing of the health shock to identify the causal effects of the health shock. The children experiencing a health shock in the same year are grouped into a cohort, and the crucial feature of event studies is the presence of multiple cohorts, which serves as a source of identification for estimation. The data used in the analysis comes from Norwegian administrative registers, covering the period 2006–2014. In the main analysis, the sample consists of families in which a child has a health shock between age 5 and 18. To examine whether there are differences in the effects due to different causes and timing of the health shock, I assess heterogeneity by type of health shock given by the related diagnosis, by length of hospital stay, and by age of the children at the time of the health shock.

The results show that parents experience significant and persistent reductions in labor income and employment, as well as increased use of social security benefits as a result of the child's negative health shock. The underlying health effects relates to psychological disorders. The effects are mainly driven by health shocks related to injury, poisoning and certain other consequences of external causes, such as traumatic head injury, when the children are between age 5 and 12.

This paper contributes to the literature by using high-quality data and state-of-the-art econometric methods to study the effects of a child's negative health shock on parental health and labor market outcomes in a manner that provides causal effects. By considering a wide range of possible outcomes as well as heterogeneity by the type of health shock, I am able to capture a comprehensive picture of the effects on parents' health and labor market outcomes of experiencing that a child has a negative health shock, as well as potential mechanisms driving the effects. The results from this study can guide policymakers in improving the design of policies to assist parents and to reduce the economic costs of children's health shocks.

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8 Tables and Figures

Table 1: Descriptive Statistics: Main Sample

	Mean	**St. Dev.**
Age at health shock	12.09	[4.27]
Female	0.46	[0.50]
Born in Norway	0.79	[0.40]
Birth year	1999	[4.62]
Single parent	0.03	[0.17]
Mother's birth year	1970	[6.53]
Mother born in Norway	0.81	[0.39]
Years of education in 2008, mother	13.98	[3.14]
Mother in education in 2008	0.08	[0.27]
Father's birth year	1967	[7.21]
Father born in Norway	0.82	[0.38]
Years of education in 2008, father	13.72	[2.98]
Father in education in 2008	0.03	[0.18]
Number of children	53,238	
Observations	479,547	

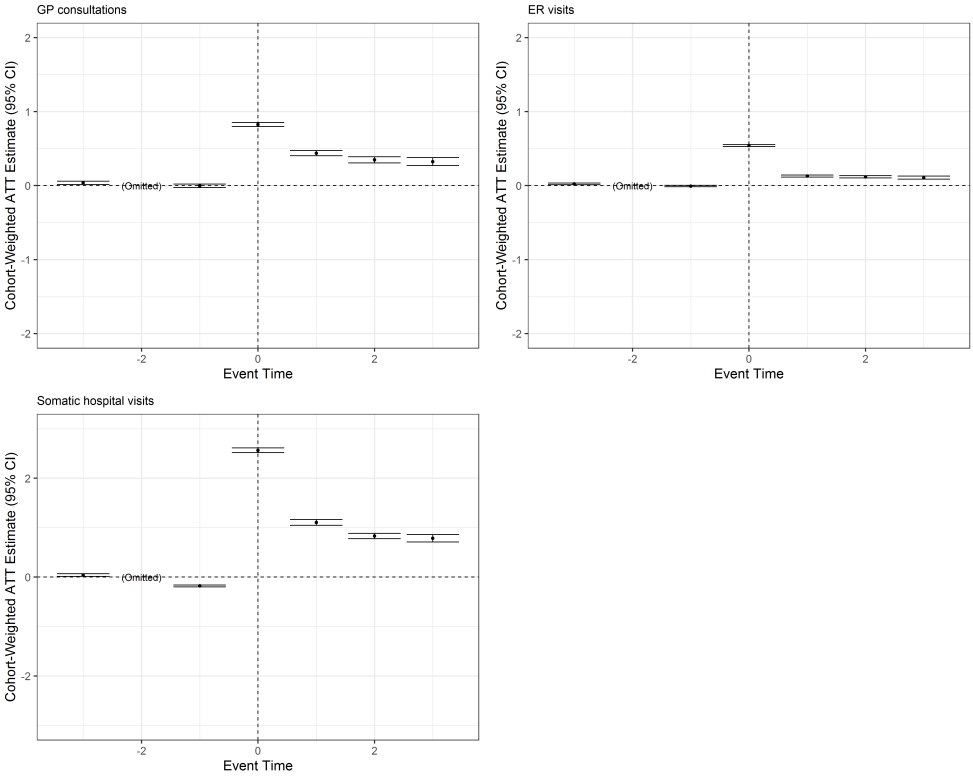
Note: This table presents the summary statistics of the background characteristics of the main analysis sample.

Table 2: Descriptive Statistics: Distribution of the Diagnoses Related to the Health Shocks

	Frequency	**Percentage**
Infectious and parasitic diseases	3243	3.89
Neoplasms and diseases of the blood	735	1.38
Metabolic diseases	1691	3.17
Mental and behavioural disorders	994	1.87
Diseases of the nervous system	1238	2.32
Diseases of the eye and ear	331	0.62
Diseases of the circulatory system	798	1.50
Diseases of the respiratory system	4182	7.85
Diseases of the digestive system	6719	12.61
Diseases of the skin	866	1.63
Diseases of the musculoskeletal system	1046	1.96
Diseases of the genitourinary system	2593	4.87
Conditions originating in the perinatal period	1	0.00
Congenital malformations, deformations and chromosomal abnormalities	120	0.23
Abnormal clinical and laboratory findings	7477	14.03
Injury, poisoning and certain other consequences of external causes	20125	32.77
External causes of morbidity and mortality	3	0.00
Factors influencing health status and contact with health services	1108	2.08
Missing diangosis	13	0.02
Number of children	53,283	

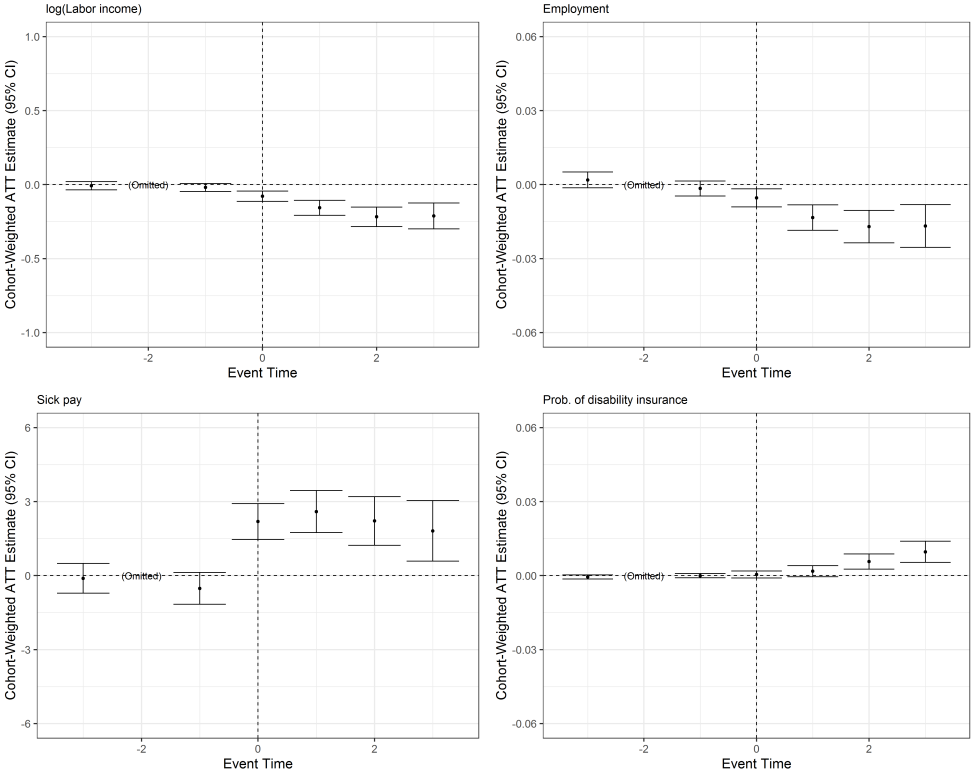
Note: This table presents the summary statistics of the diagnoses related to the health shocks. The diagnoses are based on ICD-10 codes.

Figure 1: First Stage: Children's Health



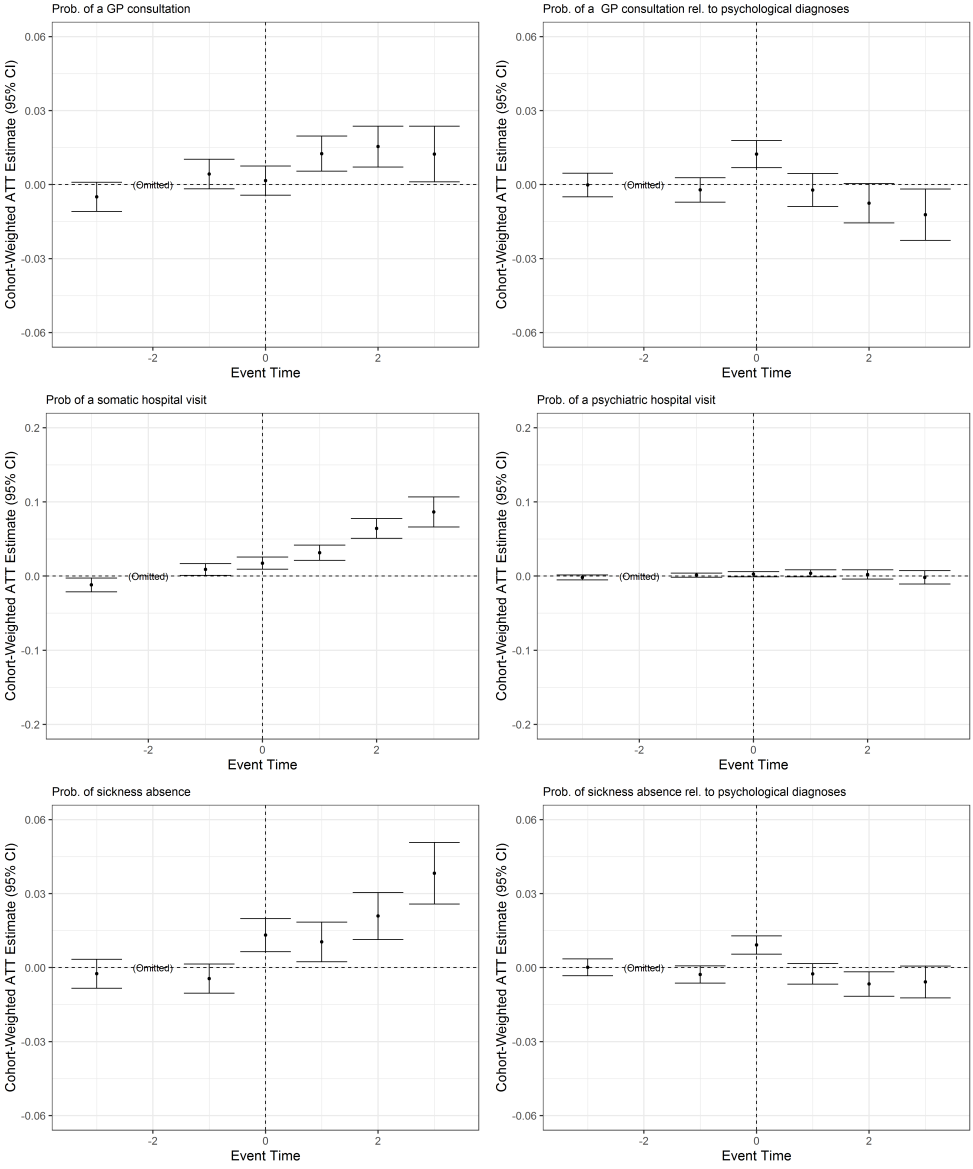
Note: This figure shows the pre-trends three years before and the estimated cohort-weighted effects up to three years after the health shock on the use of three healthcare services for the children experiencing a health shock: general practitioner consultations, emergency room visits, and somatic hospital visits. The omitted reference time is two years before the health shock.

Figure 3: Mothers' Labor Market Outcomes



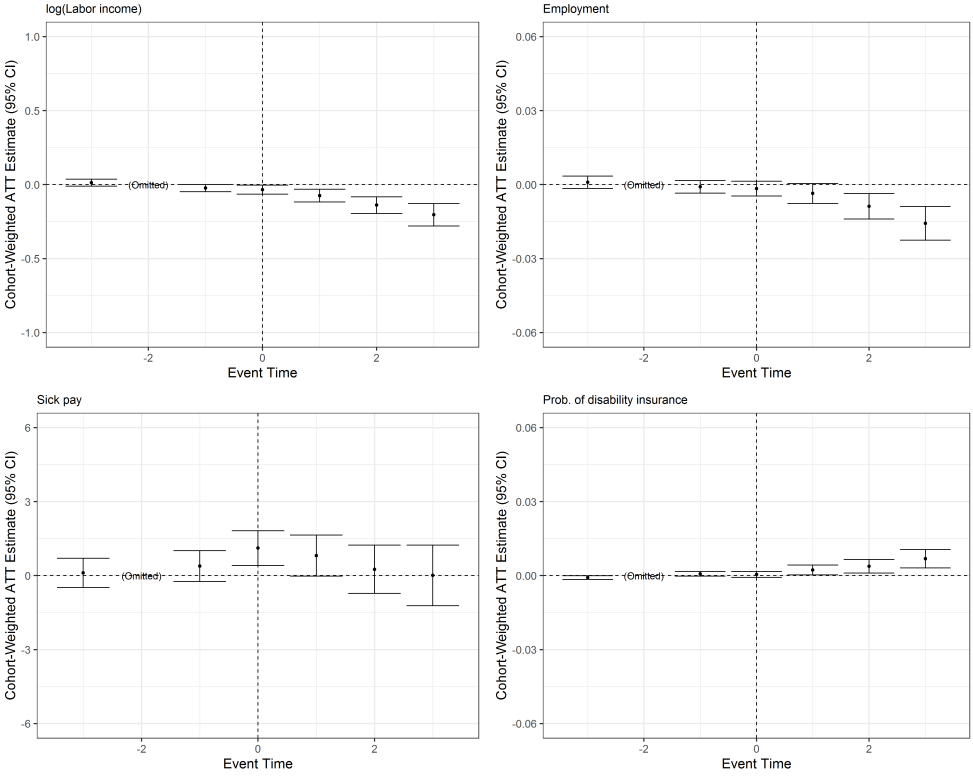
Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the labor market outcomes for mothers of the children experiencing a health shock. The omitted reference time is two years before the health shock.

Figure 5: Mothers' Health Outcomes



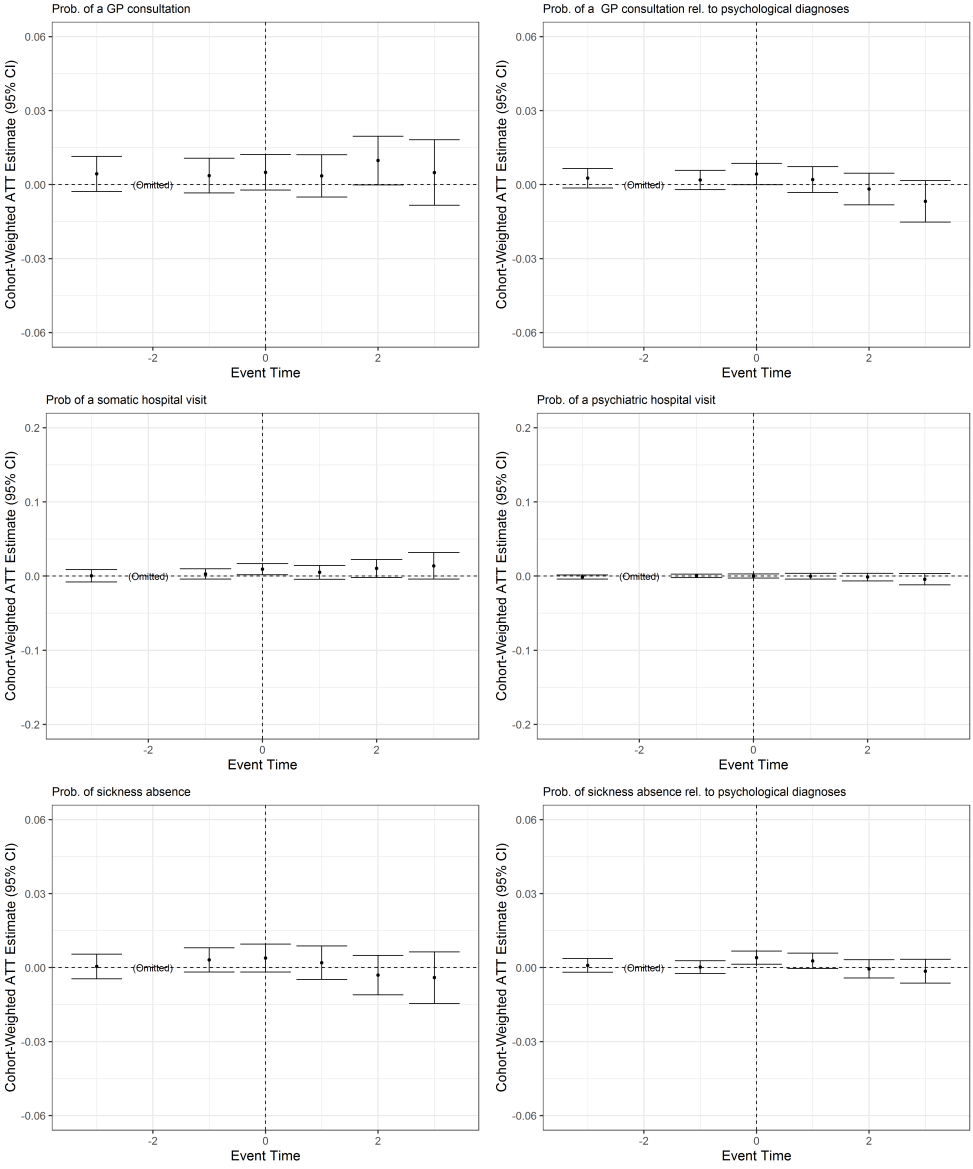
Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the health outcomes for mothers of the children experiencing a health shock. The outcomes are defined as the probability of an event per year. The omitted reference time is two years before the health shock.

Figure 7: Fathers' Labor Market Outcomes



Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the labor market outcomes for fathers of the children experiencing a health shock. The omitted reference time is two years before the health shock.

Figure 9: Fathers' Health Outcomes



Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the health outcomes for fathers of the children experiencing a health shock. The outcomes are defined as the probability of an event per year. The omitted reference time is two years before the health shock.

Table 3: First Stage: Children's Health

	GP consultations (1)	**ER visits** (2)	**Somatic spec. care** (3)
PANEL A: Total #			
-3	0.037***	0.023***	0.041***
	(0.012)	(0.005)	(0.014)
-1	-0.002	-0.007	-0.180***
	(0.012)	(0.005)	(0.011)
0	0.826***	0.542***	2.560***
	(0.014)	(0.006)	(0.025)
1	0.438***	0.128***	1.100***
	(0.017)	(0.007)	(0.030)
2	0.349***	0.119***	0.830***
	(0.021)	(0.008)	(0.028)
3	0.324***	0.108***	0.784***
	(0.028)	(0.011)	(0.038)
Pre-shock mean	[1.32]	[.30]	[.46]
PANEL B: Probability of			
-3	0.012***	0.008***	0.006
	(0.004)	(0.003)	(0.004)
-1	-0.012***	-0.007**	-0.083***
	(0.004)	(0.003)	(0.003)
0	0.160***	0.345***	0.852***
	(0.004)	(0.004)	(0.003)
1	0.046***	0.051***	0.260***
	(0.004)	(0.004)	(0.005)
2	0.018***	0.046***	0.215***
	(0.005)	(0.005)	(0.006)
3	-0.009	0.039	0.211***
	(0.007)	(0.006)	(0.009)
Pre-shock mean	[.58]	[.21]	[.21]
Observations	479,547	479,547	372,981

Note: This table shows the estimated cohort-weighted effects based on Equation 5 of the health shock on the health of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Panel A shows the results from estimations on outcomes defined as the total number of incidents per year, and Panel B shows the results from estimations on outcomes defined as the probability of an event per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 4: Main Results: Mothers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income) (1)	Employment (2)	Sick pay (3)	Prob. of disability (4)
-3	-0.008	0.002	-0.110	-0.001
	(0.014)	(0.002)	(0.306)	(0.000)
-1	-0.020	-0.002	-0.521	-0.000
	(0.014)	(0.002)	(0.329)	(0.000)
0	-0.079***	-0.005***	2.190***	0.000
	(0.018)	(0.002)	(0.371)	(0.001)
1	-0.157***	-0.013***	2.600***	0.002
	(0.026)	(0.003)	(0.436)	(0.001)
2	-0.218***	-0.017***	2.210***	0.006***
	(0.034)	(0.003)	(0.502)	(0.002)
3	-0.212***	-0.017***	1.810***	0.010***
	(0.045)	(0.004)	(0.626)	(0.002)
Pre-shock mean	[285,286]	[.80]	[14.86]	[.01]
Observations	478,458	478,458	478,458	478,458

Note: This table shows the estimated cohort-weighted effects based on Equation 5 of the health shock on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 5: Main Results: Mothers' Primary Health Care Use, Sickness Absence and Specialist Health Care Use

	GP consultations				Specialist care		Sickness absence		
	Total (1)	Musculoskeletal (2)	Psychological (3)	ER visits (4)	Somatic (5)	Psychiatric (6)	Total (7)	Musculoskeletal (8)	Psychological (9)
PANEL A: Total #									
-3	-0.022	0.003	0.004	0.009*	-0.114***	-0.008	0.407	0.070	-0.021
	(0.023)	(0.012)	(0.011)	(0.005)	(0.031)	(0.052)	(0.465)	(0.281)	(0.247)
-1	0.015	0.002	-0.022*	-0.011*	0.059*	0.017	-0.410	0.225	-0.322
	(0.023)	(0.011)	(0.012)	(0.005)	(0.029)	(0.033)	(0.480)	(0.283)	(0.252)
0	0.092***	0.004	0.017	-0.000	0.087***	0.067*	1.100**	0.240	0.581**
	(0.025)	(0.013)	(0.013)	(0.005)	(0.030)	(0.040)	(0.511)	(0.285)	(0.253)
1	0.158***	0.003	-0.022	-0.002	0.160***	0.022	1.125**	0.211	-0.299
	(0.031)	(0.016)	(0.017)	(0.006)	(0.038)	(0.052)	(0.596)	(0.325)	(0.292)
2	0.280***	0.006	-0.072***	-0.001	0.408***	0.030	2.100***	0.375	-0.612*
	(0.038)	(0.019)	(0.020)	(0.008)	(0.051)	(0.072)	(0.713)	(0.391)	(0.344)
3	0.355***	0.019	-0.076***	0.008	0.467***	0.025	3.020***	0.074	-0.728*
	(0.052)	(0.026)	(0.025)	(0.011)	(0.079)	(0.097)	(0.914)	(0.534)	(0.428)
Pre-shock mean	[3.34]	[.77]	[.56]	[.26]	[1.27]	[.60]	[23.93]	[6.70]	[5.19]
PANEL B: Probability of									
-3	-0.005**	0.005	-0.000	0.000	-0.012**	-0.002	-0.002	-0.001	0.000
	(0.003)	(0.003)	(0.002)	(0.003)	(0.005)	(0.002)	(0.003)	(0.002)	(0.002)
-1	0.004	-0.003	-0.002	-0.007**	0.009**	0.001	-0.004	-0.000	-0.003
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.001)	(0.003)	(0.002)	(0.002)
0	0.002	-0.003	0.012***	-0.001	0.017***	0.003	0.013***	-0.000	0.009***
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.002)	(0.003)	(0.002)	(0.002)
1	0.013***	-0.000	-0.002	-0.000	0.032***	0.004	0.010**	0.002	-0.003
	(0.004)	(0.004)	(0.003)	(0.004)	(0.005)	(0.002)	(0.004)	(0.002)	(0.002)
2	0.015***	0.000	-0.008*	0.002	0.064***	0.002	0.021***	0.003	-0.007***
	(0.004)	(0.005)	(0.004)	(0.004)	(0.007)	(0.003)	(0.005)	(0.003)	(0.003)
3	0.012**	0.000	-0.012**	0.010*	0.087***	-0.002	0.038***	0.004	-0.006*
	(0.006)	(0.007)	(0.005)	(0.006)	(0.010)	(0.005)	(0.006)	(0.004)	(0.003)
Pre-shock mean	[.79]	[.31]	[.17]	[.18]	[.36]	[.05]	[.24]	[.07]	[.05]
Observations	478,458	478,458	478,458	478,458	372,134	372,134	478,458	478,458	478,458

Note: This table shows the estimated cohort-weighted effects based on Equation 5 of the health shock on health outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Panel A shows the results from estimations on outcomes defined as the total number of incidents per year, and Panel B shows the results from estimations on outcomes defined as the probability of an event per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 6: Main Results: Fathers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income) (1)	Employment (2)	Sick pay (3)	Prob. of disability (4)
-3	0.013	0.001	0.111	-0.001**
	(0.012)	(0.001)	(0.303)	(0.000)
-1	-0.023*	-0.001	0.387	0.000
	(0.012)	(0.001)	(0.317)	(0.000)
0	-0.034**	-0.002	1.120***	0.000
	(0.016)	(0.002)	(0.360)	(0.001)
1	-0.075***	-0.004*	0.809*	0.002**
	(0.022)	(0.002)	(0.426)	(0.001)
2	-0.139***	-0.009***	0.257	0.004***
	(0.029)	(0.003)	(0.497)	(0.001)
3	-0.204***	-0.016***	0.008	0.007***
	(0.039)	(0.003)	(0.628)	(0.002)
Pre-shock mean	[495,292]	[.88]	[11.77]	[.01]
Observations	466,497	466,497	466,497	466,497

Note: This table shows the estimated cohort-weighted effects based on Equation 5 of the health shock on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 7: Main Results: Fathers' Primary Health Care Use, Sickness Absence and Specialist Health Care Use

	GP consultations				Specialist care		Sickness absence		
	Total (1)	Musculoskeletal (2)	Psychological (3)	ER visits (4)	Somatic (5)	Psychiatric (6)	Total (7)	Musculoskeletal (8)	Psychological (9)
PANEL A: Total #									
-3	-0.001	-0.013	0.012	0.008*	-0.024	0.007	0.112	0.138	-0.008
	(0.018)	(0.010)	(0.009)	(0.004)	(0.025)	(0.026)	(0.371)	(0.247)	(0.187)
-1	0.022	-0.003	0.005	0.004	0.022	-0.000	0.591	0.146	0.034
	(0.019)	(0.010)	(0.009)	(0.005)	(0.022)	(0.025)	(0.374)	(0.256)	(0.181)
0	0.014	-0.021*	0.014	0.008*	0.061**	-0.016	-0.003	-0.423*	0.246
	(0.020)	(0.012)	(0.010)	(0.004)	(0.029)	(0.030)	(0.408)	(0.253)	(0.182)
1	0.048*	-0.014	0.020*	-0.001	0.019	-0.007	-0.015	-0.489	0.308
	(0.026)	(0.014)	(0.013)	(0.006)	(0.033)	(0.037)	(0.481)	(0.300)	(0.215)
2	0.071**	0.000	0.007	-0.003	0.040	-0.098**	-0.366	-0.064	-0.120
	(0.032)	(0.017)	(0.016)	(0.007)	(0.040)	(0.046)	(0.569)	(0.365)	(0.256)
3	0.040	-0.008	0.008	-0.008	0.081	-0.065	-0.276	-0.319	-0.131
	(0.042)	(0.022)	(0.022)	(0.009)	(0.059)	(0.066)	(0.739)	(0.482)	(0.343)
Pre-shock mean	[1.98]	[.62]	[.31]	[.20]	[.82]	[.37]	[15.04]	[5.78]	[2.66]
PANEL B: Probability of									
-3	0.004	0.002	0.003	0.006*	0.000	-0.001	0.000	0.002	0.001
	(0.004)	(0.003)	(0.002)	(0.003)	(0.004)	(0.001)	(0.003)	(0.002)	(0.001)
-1	0.004	-0.000	0.002	0.002	0.003	0.000	0.003	0.001	0.000
	(0.004)	(0.003)	(0.002)	(0.003)	(0.004)	(0.001)	(0.003)	(0.002)	(0.001)
0	0.005	-0.003	0.004*	0.004	0.009**	0.000	0.004	-0.001	0.004***
	(0.004)	(0.003)	(0.002)	(0.003)	(0.004)	(0.001)	(0.003)	(0.002)	(0.001)
1	0.004	-0.001	0.002	-0.003	0.005	-0.000	0.002	-0.002	0.003*
	(0.004)	(0.004)	(0.003)	(0.003)	(0.005)	(0.002)	(0.003)	(0.002)	(0.002)
2	0.010*	-0.003	-0.002	-0.005	0.010*	-0.001	-0.003	-0.001	-0.001
	(0.005)	(0.005)	(0.003)	(0.004)	(0.006)	(0.003)	(0.004)	(0.003)	(0.002)
3	0.005	-0.012*	-0.007	-0.010*	0.014	-0.004	-0.004	-0.000	-0.001
	(0.007)	(0.006)	(0.004)	(0.005)	(0.009)	(0.004)	(0.005)	(0.004)	(0.002)
Pre-shock mean	[.61]	[.26]	[.10]	[.15]	[.26]	[.03]	[.15]	[.06]	[.03]
Observations	466,497	466,497	466,497	466,497	362,831	362,831	466,497	466,497	466,497

Note: This table shows the estimated cohort-weighted effects based on Equation 5 of the health shock on health outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Panel A shows the results from estimations on outcomes defined as the total number of incidents per year, and Panel B shows the results from estimations on outcomes defined as the probability of an event per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 8: Heterogeneity - Ages 5–12 and Ages 13–18: Children's Health

	GP consultations		**ER visits**		**Somatic spec. care**	
	5–12	13–18	5–12	13–18	5–12	13–18
	(1)	(2)	(3)	(4)	(5)	(6)
-3	0.151***	-0.072***	0.067***	-0.019***	0.029	0.052***
	(0.018)	(0.015)	(0.009)	(0.006)	(0.021)	(0.018)
-1	-0.033*	0.027	-0.018**	0.004	-0.125***	-0.231***
	(0.017)	(0.016)	(0.008)	(0.006)	(0.016)	(0.014)
0	0.728***	0.920***	0.535***	0.548***	2.710***	2.420***
	(0.019)	(0.021)	(0.009)	(0.008)	(0.039)	(0.031)
1	0.305***	0.566***	0.088***	0.166***	1.100***	1.110***
	(0.022)	(0.025)	(0.010)	(0.009)	(0.050)	(0.036)
2	0.169***	0.523***	0.050***	0.184***	0.784***	0.872***
	(0.028)	(0.031)	(0.012)	(0.011)	(0.040)	(0.040)
3	0.052	0.587***	0.007	0.204***	0.635***	0.928***
	(0.037)	(0.040)	(0.016)	(0.014)	(0.043)	(0.059)
Pre-shock mean	[1.39]	[1.26]	[.37]	[.23]	[.48]	[.45]
Observations	236,916	242,631	236,916	242,631	184,268	188,713

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by age of the children at the time of the health shock, on the health of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Outcomes are defined as the total number of incidents per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 9: Heterogeneity - Ages 5–12 and Ages 13–18: Mothers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)		**Employment**		**Sick pay**		**Prob. of disability**	
	5–12	13–18	5–12	13–18	5–12	13–18	5–12	13–18
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
-3	-0.042*	0.023	-0.002	0.005**	0.027	-0.286	-0.000	-0.001
	(0.021)	(0.018)	(0.002)	(0.002)	(0.434)	(0.426)	(0.000)	(0.001)
-1	-0.024	-0.011	-0.001	-0.003	-0.214	-0.717	0.001	-0.001
	(0.020)	(0.018)	(0.002)	(0.002)	(0.470)	(0.457)	(0.001)	(0.001)
0	-0.096***	-0.061***	-0.007**	-0.004*	2.840***	1.600***	0.002*	-0.001
	(0.026)	(0.023)	(0.003)	(0.002)	(0.537)	(0.505)	(0.001)	(0.001)
1	-0.191***	-0.126***	-0.016***	-0.011***	3.350***	1.730***	0.004***	-0.001
	(0.038)	(0.034)	(0.004)	(0.003)	(0.608)	(0.606)	(0.001)	(0.002)
2	-0.247***	-0.197***	-0.019***	-0.015***	3.090***	1.430**	0.009***	0.002
	(0.050)	(0.044)	(0.005)	(0.004)	(0.709)	(0.712)	(0.002)	(0.003)
3	-0.194***	-0.245***	-0.015**	-0.017***	2.940***	0.644	0.011***	0.007**
	(0.067)	(0.058)	(0.007)	(0.006)	(0.888)	(0.886)	(0.002)	(0.004)
Pre-shock mean	[272,106]	[298,172]	[.79]	[.80]	[15.22]	[14.50]	[.01]	[.02]
Observations	236,538	241,920	236,538	241,920	236,538	241,920	236,538	241,920

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by age of the children at the time of the health shock, on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table 10: Heterogeneity - Ages 5–12 and Ages 13–18: Fathers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)		**Employment**		**Sick pay**		**Prob. of disability**	
	5–12	13–18	5–12	13–18	5–12	13–18	5–12	13–18
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
-3	0.015	0.016	0.002	0.000	0.009	0.240	-0.001*	-0.001
	(0.017)	(0.017)	(0.002)	(0.001)	(0.414)	(0.422)	(0.000)	(0.001)
-1	-0.021	-0.031*	-0.001	-0.001	0.239	0.477	0.000	0.001
	(0.017)	(0.017)	(0.002)	(0.002)	(0.451)	(0.439)	(0.000)	(0.001)
0	-0.044**	-0.032	-0.003	-0.000	1.460***	0.638	0.000	0.001
	(0.021)	(0.021)	(0.002)	(0.002)	(0.504)	(0.490)	(0.001)	(0.001)
1	-0.101***	-0.059	-0.006*	-0.002	0.631	1.030*	0.003**	0.001
	(0.031)	(0.030)	(0.003)	(0.003)	(0.596)	(0.589)	(0.001)	(0.002)
2	-0.217***	-0.080***	-0.015**	-0.004	-0.114	0.488	0.005***	0.002
	(0.040)	(0.039)	(0.004)	(0.004)	(0.686)	(0.712)	(0.002)	(0.002)
3	-0.306***	-0.121***	-0.023**	-0.010**	-0.267	0.157	0.008***	0.004
	(0.054)	(0.053)	(0.005)	(0.005)	(0.866)	(0.882)	(0.002)	(0.003)
Pre-shock mean	[491,565]	[498,949]	[.89]	[.86]	[11.51]	[12.01]	[.01]	[.02]
Observations	231,075	235,422	231,075	235,422	231,075	235,422	231,075	235,422

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by age of the children at the time of the health shock, on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table 11: Heterogeneity - Diagnoses: Children's Health

	GP consultations				ER visits			
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)	Injury (5)	Digestive (6)	Respiratory (7)	Infections (8)
-3	0.024	-0.014	0.201***	0.106**	0.018**	-0.020	0.094***	0.105***
	(0.018)	(0.031)	(0.049)	(0.052)	(0.008)	(0.013)	(0.025)	(0.024)
-1	-0.018	0.006	-0.027	-0.003	-0.004	-0.014	-0.002	0.012
	(0.018)	(0.032)	(0.047)	(0.051)	(0.007)	(0.013)	(0.022)	(0.022)
0	0.437***	0.884***	1.020***	1.360***	0.411***	0.688***	0.651***	0.745***
	(0.020)	(0.039)	(0.056)	(0.064)	(0.009)	(0.017)	(0.027)	(0.028)
1	0.363***	0.404***	0.289***	0.580***	0.116***	0.110***	0.084***	0.138***
	(0.026)	(0.048)	(0.067)	(0.077)	(0.011)	(0.018)	(0.030)	(0.031)
2	0.360***	0.367***	-0.035	0.249***	0.113***	0.134***	-0.010	0.074*
	(0.031)	(0.058)	(0.087)	(0.096)	(0.013)	(0.023)	(0.038)	(0.038)
3	0.339***	0.448***	-0.168	0.302**	0.104***	0.139***	-0.026	0.160***
	(0.040)	(0.078)	(0.119)	(0.135)	(0.016)	(0.028)	(0.051)	(0.056)
Pre-shock mean	[1.18]	[1.23]	[1.64]	[1.48]	[.26]	[.25]	[.43]	[.33]
Observations	181,125	60,471	37,638	29,187	181,125	60,471	37,638	29,187
	Somatic hospital visits							
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)				
-3	0.039*	-0.019	0.072	0.060				
	(0.020)	(0.033)	(0.059)	(0.076)				
-1	-0.162***	-0.205***	-0.167***	-0.108***				
	(0.015)	(0.026)	(0.046)	(0.041)				
0	2.940***	1.570***	1.640***	1.780***				
	(0.029)	(0.042)	(0.062)	(0.096)				
1	0.728***	0.659***	0.596***	0.642***				
	(0.028)	(0.063)	(0.076)	(0.098)				
2	0.543***	0.549***	0.505***	0.513***				
	(0.033)	(0.062)	(0.094)	(0.106)				
3	0.586***	0.659***	0.450***	0.661***				
	(0.059)	(0.082)	(0.143)	(0.174)				
Pre-shock mean	[.39]	[.40]	[.67]	[.51]				
Observations	140,875	47,033	29,274	22,701				

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the diagnoses related to the health shocks, on the health of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Outcomes are defined as the total number of incidents per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 12: Heterogeneity - Diagnoses: Mothers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)				**Employment**			
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)	Injury (5)	Digestive (6)	Respiratory (7)	Infections (8)
-3	0.019	0.018	-0.029	-0.007	0.005*	-0.008*	-0.004	0.003
	(0.022)	(0.040)	(0.054)	(0.059)	(0.003)	(0.004)	(0.006)	(0.007)
-1	-0.006	-0.002	-0.060	-0.079	-0.002	-0.004	-0.003	-0.001
	(0.021)	(0.039)	(0.052)	(0.058)	(0.002)	(0.004)	(0.006)	(0.007)
0	-0.067**	-0.037	-0.178***	-0.148**	-0.006**	-0.012**	-0.004	-0.008
	(0.027)	(0.049)	(0.066)	(0.073)	(0.003)	(0.005)	(0.007)	(0.008)
1	-0.161***	-0.121*	-0.278***	-0.133	-0.013***	-0.018**	-0.008	-0.008
	(0.039)	(0.068)	(0.093)	(0.109)	(0.004)	(0.007)	(0.010)	(0.011)
2	-0.243***	-0.159*	-0.307**	-0.156	-0.014***	-0.027***	-0.008	-0.014
	(0.052)	(0.089)	(0.126)	(0.144)	(0.005)	(0.009)	(0.013)	(0.015)
3	-0.241***	-0.143	-0.263	-0.135	-0.013*	-0.027**	-0.008	-0.012
	(0.067)	(0.121)	(0.167)	(0.194)	(0.007)	(0.012)	(0.017)	(0.020)
Pre-shock mean	[292,194]	[291,012]	[270,811]	[279,909]	[.81]	[.80]	[.77]	[.79]
	Sick pay				**Prob. of disability**			
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)	Injury (5)	Digestive (6)	Respiratory (7)	Infections (8)
-3	0.351	-1.180	-1.210	-2.550**	-0.001	-0.003**	-0.000	0.001
	(0.479)	(0.848)	(1.090)	(1.250)	(0.001)	(0.001)	(0.001)	(0.002)
-1	-0.064	-0.437	-0.229	-0.512	-0.000	0.000	0.000	0.002
	(0.529)	(0.949)	(1.130)	(1.370)	(0.001)	(0.001)	(0.002)	(0.002)
0	2.070***	-0.645	0.349	0.376	0.000	-0.001	0.001	0.003
	(0.600)	(0.981)	(1.240)	(1.570)	(0.001)	(0.002)	(0.003)	(0.003)
1	1.660**	0.030	-0.311	3.330**	-0.000	-0.001	0.002	0.005
	(0.663)	(1.160)	(1.530)	(2.130)	(0.002)	(0.003)	(0.004)	(0.005)
2	2.420***	0.199	1.840	2.910	0.002	0.005	0.005	0.007
	(0.792)	(1.390)	(1.910)	(2.190)	(0.002)	(0.005)	(0.006)	(0.006)
3	2.670***	-0.533	-3.720	6.870**	0.006**	0.009	0.004	0.017*
	(0.982)	(1.790)	(2.460)	(3.100)	(0.003)	(0.006)	(0.008)	(0.009)
Pre-shock mean	[14.20]	[14.89]	[15.63]	[16.17]	[.01]	[.01]	[.01]	[.01]
Observations	180,729	60,336	37,575	29,106	180,729	60,336	37,575	29,106

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the diagnoses related to the health shocks, on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table 13: Heterogeneity - Diagnoses: Fathers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)				Employment			
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)	Injury (5)	Digestive (6)	Respiratory (7)	Infections (8)
-3	-0.005	-0.011	0.048	0.058	-0.000	-0.005	0.002	0.003
	(0.020)	(0.034)	(0.048)	(0.049)	(0.002)	(0.004)	(0.005)	(0.005)
-1	-0.019	-0.071**	-0.111**	0.038	-0.000	-0.006*	-0.013***	0.006
	(0.020)	(0.034)	(0.046)	(0.048)	(0.002)	(0.003)	(0.004)	(0.005)
0	-0.029	-0.081**	-0.174***	0.095	-0.002	-0.003	-0.016***	0.008
	(0.026)	(0.040)	(0.058)	(0.061)	(0.002)	(0.004)	(0.005)	(0.006)
1	-0.098***	-0.093	-0.230***	0.101	-0.005	-0.001	-0.014**	0.012
	(0.036)	(0.059)	(0.079)	(0.082)	(0.003)	(0.005)	(0.007)	(0.008)
2	-0.156***	-0.096	-0.373***	0.034	-0.010**	-0.006	-0.019**	0.007
	(0.047)	(0.074)	(0.104)	(0.114)	(0.004)	(0.007)	(0.009)	(0.011)
3	-0.249***	-0.131	-0.351**	-0.223	-0.023***	-0.001	-0.025**	-0.024
	(0.063)	(0.101)	(0.141)	(0.157)	(0.006)	(0.009)	(0.012)	(0.016)
Pre-shock mean	[503,286]	[503,403]	[473,756]	[496,160]	[.88]	[.88]	[.87]	[.87]
	Sick pay				**Prob. of disability**			
	Injury (1)	Digestive (2)	Respiratory (3)	Infections (4)	Injury (5)	Digestive (6)	Respiratory (7)	Infections (8)
-3	0.421	0.765	0.496	-2.250**	-0.001	0.001	-0.002	-0.001
	(0.496)	(0.826)	(1.100)	(1.130)	(0.001)	(0.001)	(0.001)	(0.001)
-1	1.010**	0.784	-2.440**	-0.443	0.000	0.002	-0.000	0.001
	(0.497)	(0.862)	(1.200)	(1.350)	(0.001)	(0.002)	(0.001)	(0.002)
0	1.840**	-0.550	-1.620	-0.922	0.001	0.001	-0.001	0.001
	(0.588)	(0.934)	(1.280)	(1.460)	(0.001)	(0.002)	(0.002)	(0.002)
1	1.510	-1.120	-0.735	1.110	0.004**	-0.000	0.005	0.003
	(0.659)	(1.140)	(1.540)	(1.780)	(0.002)	(0.003)	(0.003)	(0.004)
2	1.740	-1.120	-2.010*	1.290	0.005**	0.000	0.008*	0.005
	(0.781)	(1.330)	(1.860)	(2.250)	(0.002)	(0.004)	(0.005)	(0.005)
3	-0.015	1.280	-0.943	1.720	0.009***	0.001	0.012**	0.013**
	(0.972)	(1.780)	(2.240)	(2.780)	(0.003)	(0.006)	(0.007)	(0.006)
Pre-shock mean	[11.45]	[11.31]	[12.19]	[12.35]	[.01]	[.01]	[.01]	[.01]
Observations	176,634	58,644	36,486	28,305	176,634	58,644	36,486	28,305

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the diagnoses related to the health shocks, on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table 14: Heterogeneity - Length of Hospital Stay: Children's Health

	GP consultations		ER visits		Somatic spec. care	
	1 night	> 1 night	1 night	> 1 night	1 night	> 1 night
	(1)	(2)	(3)	(4)	(5)	(6)
-3	0.026*	0.056***	0.027***	0.017**	0.044***	0.036
	(0.015)	(0.019)	(0.007)	(0.008)	(0.016)	(0.024)
-1	-0.019	0.026	-0.011*	0.000	-0.182***	-0.177***
	(0.015)	(0.019)	(0.006)	(0.008)	(0.012)	(0.019)
0	0.663***	1.080***	0.528***	0.565***	2.090***	3.300***
	(0.018)	(0.024)	(0.008)	(0.010)	(0.023)	(0.051)
1	0.393***	0.503***	0.129***	0.127***	0.749***	1.650***
	(0.022)	(0.028)	(0.009)	(0.011)	(0.031)	(0.061)
2	0.322***	0.391***	0.124***	0.111***	0.621***	1.140***
	(0.027)	(0.035)	(0.011)	(0.013)	(0.028)	(0.057)
3	0.307***	0.345***	0.098***	0.123***	0.618***	1.040***
	(0.036)	(0.046)	(0.014)	(0.018)	(0.044)	(0.070)
Pre-shock mean	[1.35]	[1.28]	[.30]	[.28]	[.44]	[.49]
Observations	293,661	185,886	293,661	185,886	228,403	144,578

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the length of the initial hospital stay, on the health of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. Outcomes are defined as the total number of incidents per year. * p<0.1, ** p<0.05, *** p<0.01.

Table 15: Heterogeneity - Length of Hospital Stay: Mothers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)		Employment		Sick pay		Prob. of disability	
	1 night	> 1 night	1 night	> 1 night	1 night	> 1 night	1 night	> 1 night
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
-3	-0.024	0.019	0.001	0.004	-0.234	0.063	-0.000	-0.001
	(0.018)	(0.022)	(0.002)	(0.003)	(0.390)	(0.475)	(0.001)	(0.001)
-1	-0.026	-0.011	-0.002	-0.002	-0.460	-0.460	0.000	-0.000
	(0.017)	(0.022)	(0.002)	(0.003)	(0.424)	(0.498)	(0.001)	(0.001)
0	-0.086***	-0.069**	-0.005**	-0.007**	0.882*	4.350***	-0.000	0.001
	(0.022)	(0.028)	(0.002)	(0.003)	(0.463)	(0.590)	(0.001)	(0.001)
1	-0.155***	-0.158***	-0.011***	-0.017***	1.750***	3.930***	0.001	0.003
	(0.033)	(0.040)	(0.003)	(0.004)	(0.546)	(0.721)	(0.001)	(0.002)
2	-0.194***	-0.257***	-0.015***	-0.022***	2.140***	2.280***	0.004**	0.007***
	(0.043)	(0.053)	(0.004)	(0.005)	(0.645)	(0.787)	(0.002)	(0.003)
3	-0.209***	-0.213***	-0.018***	-0.015**	0.878	3.010***	0.007***	0.013**
	(0.056)	(0.071)	(0.006)	(0.007)	(0.799)	(1.000)	(0.003)	(0.004)
Pre-shock mean	[285,795]	[284,480]	[.80]	[.79]	[14.98]	[14.67]	[.01]	[.01]
Observations	293,076	185,382	293,076	185,382	293,076	185,382	293,076	185,382

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the length of the initial hospital stay, on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

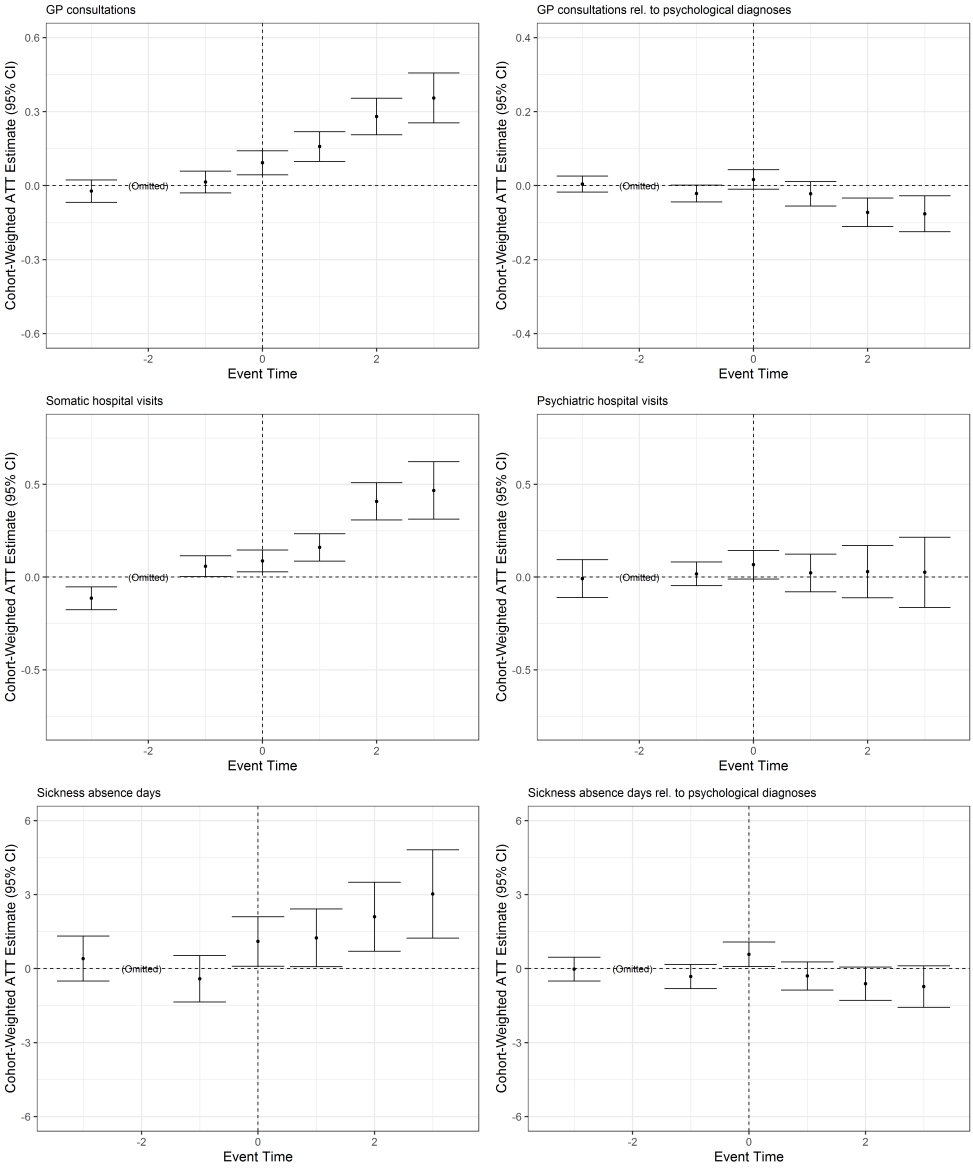
Table 16: Heterogeneity - Length of Hospital Stay: Fathers' Social Insurance Use and Labor Market Outcomes

	log(Labor income)		**Employment**		**Sick pay**		**Prob. of disability**	
	1 night	$>$ 1 night	1 night	$>$ 1 night	1 night	$>$ 1 night	1 night	$>$ 1 night
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
-3	0.023	-0.004	-0.000	0.003	-0.212	0.534	-0.001	-0.001
	(0.015)	(0.020)	(0.002)	(0.002)	(0.376)	(0.495)	(0.000)	(0.001)
-1	-0.026	-0.020	-0.001	-0.002	0.409	0.278	0.000	0.001
	(0.016)	(0.020)	(0.002)	(0.002)	(0.400)	(0.510)	(0.001)	(0.001)
0	-0.037**	-0.043	-0.002	-0.002	0.572	1.830***	0.000	0.000
	(0.019)	(0.026)	(0.002)	(0.003)	(0.446)	(0.602)	(0.001)	(0.001)
1	-0.101***	-0.043	-0.005*	-0.003	0.394	1.270*	0.002**	0.002
	(0.027)	(0.036)	(0.003)	(0.003)	(0.535)	(0.698)	(0.001)	(0.002)
2	-0.152***	-0.134***	-0.011***	-0.007*	0.232	0.016	0.004**	0.004**
	(0.035)	(0.049)	(0.003)	(0.004)	(0.631)	(0.805)	(0.002)	(0.002)
3	-0.232***	-0.187***	-0.015***	-0.020***	0.001	0.022	0.006**	0.009***
	(0.047)	(0.065)	(0.004)	(0.006)	(0.792)	(1.010)	(0.002)	(0.003)
Pre-shock mean	[494,501]	[496,544]	[.88]	[.87]	[11.82]	[11.69]	[.01]	[.01]
Observations	285,885	180,612	285,885	180,612	285,885	180,612	285,885	180,612

Note: This table shows the estimated cohort-weighted effects based on Equation 5, for subsamples defined by the length of the initial hospital stay, on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

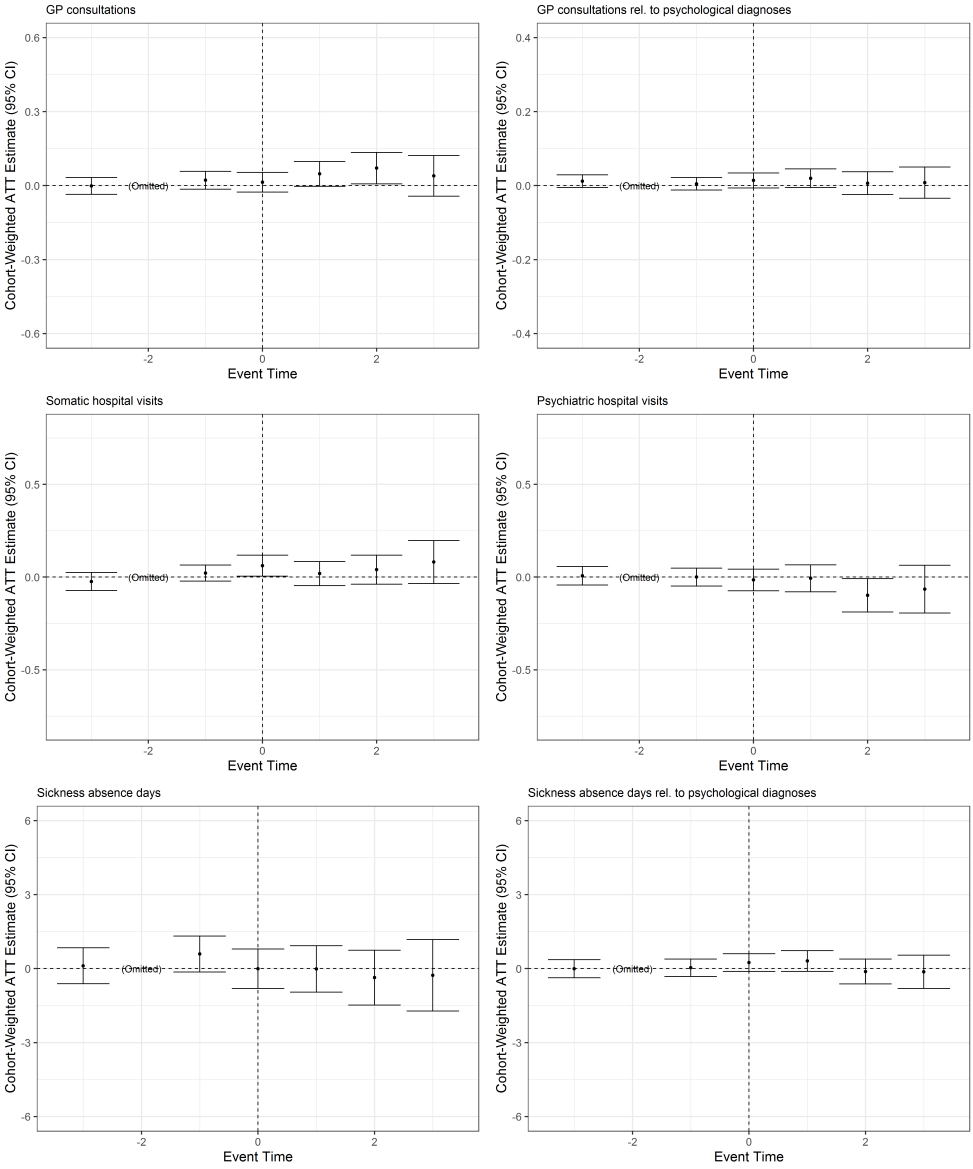
A Appendix

Figure A.1: Mothers' Health Outcomes



Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the health outcomes for mothers of the children experiencing a health shock. The outcomes are measured as the total number of incidents per year.

Figure A.3: Fathers' Health Outcomes



Note: This figure shows the pre-trends three years before, and the estimated cohort-weighted effects up to three years after the health shock on the health outcomes for fathers of the children experiencing a health shock. The outcomes are measured as the total number of incidents per year.

Table A1: Robustness - Anticipation: Mothers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income) (1)	Employment (2)	Sick pay (3)	Prob. of disability (4)
-3	-0.007	0.002	-0.115	-0.001
	(0.014)	(0.002)	(0.304)	(0.000)
-1	-0.019	-0.002	-0.509*	-0.000
	(0.013)	(0.002)	(0.326)	(0.000)
0	-0.075***	-0.008***	2.000***	0.001
	(0.022)	(0.002)	(0.415)	(0.001)
1	-0.145***	-0.013***	2.270***	0.004***
	(0.031)	(0.003)	(0.505)	(0.001)
2	-0.171***	-0.016***	1.060*	0.007***
	(0.044)	(0.004)	(0.617)	(0.002)
3	-0.233***	-0.022***	-0.185	0.013***
	(0.066)	(0.006)	(0.894)	(0.003)
Pre-shock mean	[285,286]	[.80]	[14.86]	[.01]
Observations	478,458	478,458	478,458	478,458

Note: This table shows the estimated cohort-weighted effects when controlling for anticipation the year before the health shock, of the health shock on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table A2: Robustness - Anticipation: Fathers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income)	Employment	Sick pay	Prob. of disability
	(1)	(2)	(3)	(4)
-3	0.015	0.001	0.105	-0.001**
	(0.013)	(0.001)	(0.301)	(0.000)
-1	-0.025**	-0.001	0.331	0.001
	(0.012)	(0.001)	(0.314)	(0.000)
0	-0.040**	-0.002	1.040**	0.001*
	(0.019)	(0.002)	(0.411)	(0.001)
1	-0.092***	-0.004*	0.592	0.003***
	(0.027)	(0.002)	(0.484)	(0.001)
2	-0.196***	-0.011***	0.933	0.006***
	(0.037)	(0.003)	(0.610)	(0.002)
3	-0.235***	-0.009*	-0.158	0.009***
	(0.054)	(0.004)	(0.825)	(0.002)
Pre-shock mean	[495,292]	[.88]	[11.77]	[.01]
Observations	466,497	466,497	466,497	466,497

Note: This table shows the estimated cohort-weighted effects when controlling for anticipation the year before the health shock, of the health shock on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table A3: Robustness - Sample Selection: Mothers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income)	Employment	Sick pay	Prob. of disability
	(1)	(2)	(3)	(4)
-3	-0.030	-0.001	-0.375	-0.000
	(0.022)	(0.002)	(0.508)	(0.001)
-1	-0.021	0.002	-0.266	-0.001
	(0.021)	(0.002)	(0.550)	(0.001)
0	-0.086***	-0.003	2.470***	-0.001
	(0.027)	(0.003)	(0.607)	(0.001)
1	-0.157***	-0.012***	3.520***	-0.001
	(0.043)	(0.004)	(0.772)	(0.002)
2	-0.214***	-0.016**	3.310***	0.003
	(0.067)	(0.007)	(1.120)	(0.003)
Pre-shock mean	[301,324]	[.80]	[15.16]	[.01]
Observations	230,130	230,130	230,130	230,130

Note: This table shows the estimated cohort-weighted effects when the sample of children is restricted to not having a hospital visit three years before the health shock, of the health shock on labor market outcomes for mothers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * p<0.1, ** p<0.05, *** p<0.01.

Table A4: Robustness - Sample Selection: Fathers' Social Insurance Use and Labor Market Outcomes

	Labor market		**Social insurance**	
	log(Labor income)	Employment	Sick pay	Prob. of disability
	(1)	(2)	(3)	(4)
-3	-0.007	0.000	-0.103	-0.001
	(0.019)	(0.002)	(0.486)	(0.001)
-1	-0.031	0.001	0.387	0.000
	(0.019)	(0.002)	(0.483)	(0.001)
0	-0.024	0.001	1.520***	0.001
	(0.025)	(0.002)	(0.561)	(0.001)
1	-0.063*	-0.001	1.010	0.002
	(0.038)	(0.004)	(0.724)	(0.002)
2	-0.063	-0.006	-0.313	0.003
	(0.060)	(0.005)	(1.100)	(0.003)
Pre-shock mean	[518,699]	[.88]	[11.74]	[.01]
Observations	223,785	223,785	223,785	223,785

Note: This table shows the estimated cohort-weighted effects when the sample of children is restricted to not having a hospital visit three years before the health shock, of the health shock on labor market outcomes for fathers of the children experiencing a health shock, each event time from three years before the health shock to three years after the health shock. The omitted reference time is two years before the health shock. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Chapter 3:

Effects of Universal Childcare on Long-Run Health

Effects of Universal Childcare on Long-Run Health*

Anne-Lise Breivik† Emilia Del Bono‡ Julie Riise§

Abstract

This paper examines the impact of increased access to universal childcare on adult health. The results show that affected women increase their use of pregnancy-related healthcare services and sickness absence. However, there is no increase in fertility and no effects on the second generation's birth outcomes, indicating that the women's health is unchanged, but that they have increased their demand for healthcare services. Second, there is a reduction in the use of mental healthcare services, and services related to injuries and social problems, pointing toward improved mental health. Finally, children of employed mothers are driving the effects.

Keywords: Universal childcare, health, pregnancy, health behaviors, mental health

JEL Codes: J13, H40, I1

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1 Introduction

A large body of evidence has shown that early life experiences can affect health throughout the lifecycle (Shonkoff et al., 2009; Conti and Heckman, 2013). Similarly, early childhood programs have been found to affect early childhood conditions and life experiences, including significant impacts on children's long-run health development (D'Onise et al., 2010a,b; Muennig, 2015) and other human capital outcomes (see e.g., Almond and Currie, 2011; Almond et al., 2018). However, despite the predictive power of early-life health for adult well-being (Currie et al., 2010; Reilly and Kelly, 2011), evidence on the long-term effects of childcare programs on health outcomes is based mainly on small-scale targeted programs (Campbell et al., 2014; Conti et al., 2016), and evidence on how the provision of universal programs affects children's long-run development remains scarce. Evidence of the effects of universal programs on adult health and healthy behavior is particularly limited.

In this paper, we use Norwegian administrative data and examine the long-run health outcomes of children affected by a 1975 reform in Norway, which led to a large-scale expansion of subsidized universal childcare for children three to six years old. More specifically, our main research question is whether, and to what extent, the expansion of universal childcare has long-term effects on adult health outcomes. We examine the effects of the reform on four main outcomes: primary healthcare use, certified sickness absence from work, and somatic and psychiatric specialist healthcare use. The health outcomes are measured in 2006–2014 (primary healthcare and sickness absence) and 2008–2014 (specialist healthcare), which means that the sample of children exposed to the reform are in their prime age, between 30–47, when the outcomes are measured.

The childcare reform was introduced by the Norwegian Government as a response to a growing demand for childcare driven by increased female entry into the labor market during the 1960s and 1970s. The aim of the reform was to create arenas for child development as well as to free up labor market reserves among mothers. A number of children were already in informal care, and it turned out the reform did not lead to increased labor market participation among mothers (Havnes and Mogstad, 2011a). The main effect of the reform was thus a shift from informal to formal care for children three to six years old.

Although the childcare reform was planned centrally, the responsibility for childcare was assigned to the municipalities. This led to a staged expansion of childcare coverage across Norway's (at that time) 445 municipalities. We exploit the variation in the expansion of childcare between different municipalities in this period to examine the long-run health effects of childcare. Our empirical strategy follows that of Havnes and Mogstad (2011b), using a differences-in-differences approach comparing adult health outcomes of children three to six years old before and after the reform, from municipalities where childcare expanded significantly and

municipalities with little or no expansion.

We have two main findings. First, individuals affected by the reform are more likely to have certified sick leave, and they increase their use of both primary and specialist healthcare. Second, the use of mental health services is reduced. With data on healthcare use, it can be difficult to disentangle changes in health from changes in behavior. However, we apply detailed data on diagnoses and procedures to get a better understanding of the possible mechanisms.

More use could reflect a need to compensate for negative health effects of childcare. However, it turns out that the increase is driven by affected women and by healthcare use related to normal pregnancies. There is no change in timing of fertility, in the use of services related to high-risk pregnancies, or in the use related to other diagnoses, pointing toward a change in health-seeking behavior rather than a change in health. There is also no change in the next generations' birth outcomes or type of birth. However, in the long run, as these individuals become older, more preventive behavior such as more health check-ups, may translate into better health. A change in behavior could come directly from the practices and habits already formed in childcare, but perhaps it is more likely an indirect effect. It is well established that there is a socioeconomic gradient in the use of healthcare services (Monstad et al., 2014; Kaarboe and Carlsen, 2014; Moscelli et al., 2018; Cutler and Lleras-Muney, 2010), and the observed effects can thus be an indirect consequence of the reform, resulting from the identified positive effects on education and income (Havnes and Mogstad, 2011b).

Similarly, the reduction in the use of mental health services could reflect improved mental health or a change in behavior towards less help seeking. There is a reduction both in the probability of visiting a GP and in the use of psychiatric specialist care. Especially, the latter is indicative of better mental health, as there is high excess demand for mental healthcare in Norway, and individuals are only granted access to these specialist services once the mental health problems have become severe. Together this suggests that formal childcare benefits individuals by improving their mental health in the long run. The routines and pedagogical environment of childcare could strengthen social skills and induce better decision-making and healthier behavior that last into adulthood. Being in a formal childcare institution could also increase the chances of detecting behavioral, social, and psychological problems at an early stage and could thus prevent the development of more serious problems. Both of these explanations point to direct effects of childcare, but we are not able to exclude the possibility of an alternative or additional effect related to the already identified increases in education and income.

Based on previous literature that has identified heterogeneous effects of childcare along dimensions such as gender and family background, we investigate heterogeneity by gender, and three different measures of family background: mothers' education level, mothers' employment status, and household income. Women, as expected, drive the estimated effects on the use of healthcare services related to pregnancy, while we are not able to identify any gender

differences with respect to the effects on the use of mental healthcare. Socioeconomic status measured by mothers' education level and household income does not seem to matter much, neither for the use of mental healthcare nor for the use of pregnancy-related services. Interestingly, we find that children of employed mothers are driving the effects. Knowing that the reform implied a shift from informal care arrangements to formal ones, and that it had no effect on mothers' labor supply, this makes sense: the children of working mothers are the ones more likely to take up the reform, and thus they are the ones most likely to be directly affected by it.

Our study of the long-term health effects of a universal childcare program contributes to the literature in several ways. First, the majority of studies on the long-term effects of universal childcare focuses exclusively on cognitive and non-cognitive measures of child development.[1] In spite of a considerable number of studies on the effects on human capital development, stringent evidence on the causal effects on health remains scarce. Most of the existing evidence comes from small-scale and/or targeted programs[2], and the literature on effects from large-scale publicly provided universal childcare on health is limited. van den Berg and Siflinger (2018), Baker et al. (2008), Baker et al. (2019), and Haeck et al. (2018) all study health effects of universal programs, but only Baker et al. (2019) and Haeck et al. (2018) focus on long-term outcomes, in which the health aspect is limited to two survey questions about self-reported health. We take advantage of a large, universal reform in combination with highly detailed administrative register data and examine a wide range of adult health outcomes and healthcare use, capturing many aspects of health that have not yet been studied.

The paper proceeds as follows. Section 2 establishes the foundation of the study by reviewing the relationship between health and childcare. Section 3 provides background information about the 1975 childcare reform and the organization of formal childcare in the period examined. Section 4 describes the data applied. Section 5 describes and discusses the empirical strategy. Section 6 presents our results, and Section 7 presents the robustness checks, before we conclude in Section 8.

[1] Havnes and Mogstad (2011b) find positive impacts on educational attainment and labor market participation, measured when the children are in their early 30s. In a related paper, the same team of authors find that the positive effects of the childcare expansion are driven by children in the lower and middle part of the earnings distribution, and that the effects are negative for children in the uppermost part (Havnes and Mogstad, 2015). Datta Gupta and Simonsen (2016) find positive effects on children's test scores in ninth grade. Felfe and Lalive (2018) and Cornelissen et al. (2018) find positive effects on school readiness indicators for children of immigrant ancestry. Also Rossin-Slater and Wüst (2019) find positive effects on schooling. On the other hand, Baker et al. (2008) find no effects on cognitive outcomes but negative effects on children's non-cognitive outcomes. Datta Gupta and Simonsen (2010) find that compared to home care, being enrolled in preschool does not lead to significant differences in children's non-cognitive outcomes.

[2] Targeted programs like Head Start, the Perry Preschool Project (PPP), and the Abecedarian Project (ABC) have generated positive long-term effects on outcomes such as behavioral problems, prevalence of chronic conditions, and obesity for their participants (Currie and Thomas, 1995; Carneiro and Ginja, 2014; Conti et al., 2016).

2 Childcare and Health

2.1 Previous Findings on Childcare and Health

Most of the available evidence on the health effects of childcare comes from studies that focus on childcare programs targeted at disadvantaged children. For example, Conti et al. (2016) examine the effects on health and healthy behaviors of two targeted early childhood interventions, the Perry Preschool Project (PPP) and the Abecedarian Project (ABC). Both interventions randomly assigned enriched environments to disadvantaged children. They find that boys randomly assigned to the treatment group of the PPP have a significantly lower prevalence of behavioral risk factors in adulthood compared to those in the control group, while those who received the ABC intervention have better physical health. The impacts on girls is considerably weaker for both programs, although they find that both the PPP and the ABC substantially improved the adult healthy habits of girls who were randomized to the treatment groups: they engaged in more physical activity, ate more fresh fruit, and drank less alcohol. However, in contrast to our setting, these programs are not only targeted at disadvantaged children; they also include both schooling and a mix of interventions, such as home visits in the PPP and interventions to improve health, nutrition, and parent involvement in the ABC, making it difficult to directly compare these findings to those from other studies of childcare effects.

The literature on the effects of large-scale publicly provided universal childcare on health remains scarce. The studies that are most closely related to our study were conducted in Sweden and Canada, respectively. In the Swedish study, van den Berg and Siflinger (2018) examine the effect of a childcare reform, which led to considerable cuts in childcare fees for formal public childcare. Children affected by the reform had better physical health, measured as rates of respiratory illnesses, ear diseases, and other childhood illnesses, at ages 4–5 and 6–7 and better developmental and psychological conditions at age 6–7. Baker et al. (2008) and Baker et al. (2019) investigate the introduction of a large-scale subsidized childcare program in Quebec, Canada, in the late 1990s and find the opposite from van den Berg and Siflinger (2018); the introduction of the universal childcare program led to negative effects on children's non-cognitive outcomes both in the short term and in the long term, significant declines in self-reported health and life-satisfaction, as well as behavioral problems and criminal activity among boys in the long term. Haeck et al. (2018) study the same program as Baker et al. (2019) but come to a different conclusion: when allowing for different treatment periods for different cohorts rather than taking an average, they find that the effects fade out in the long term. Importantly, the childcare program rolled out in Quebec encompassed long hours and large groups of children, and the quality of care is not directly comparable with that of the Swedish or Norwegian universal childcare programs.

2.2 Early Investments and Dynamic Complementarities

Recent evidence from both biological and social sciences point to the importance of the early years in building the foundations for lifelong health.

Investments in early childhood have been shown to have high returns (Knudsen et al., 2006). There is considerable evidence from psychology and neuroscience showing that learning is easier in early childhood than later in life, making investments in human capital in this period relatively more rewarding. The earlier such investments are made, the longer the payoff period becomes. In addition, investments in human capital have dynamic complementarities, implying that learning begets learning (Carneiro and Heckman, 2003). The literature on early-life interventions suggests both direct and indirect channels through which early childhood experiences affect long-run health outcomes (Conti et al., 2016). Childhood conditions and interventions such as formal childcare arrangements, can directly affect adult health, both because early health conditions are quite persistent throughout the lifecycle (Millimet and Tchernis, 2019) and because early traits are determinants of lifestyle, which in turn can affect long-term health (Conti and Heckman, 2010).

Attending childcare can also increase cognitive skills and lead to increased ability to understand and control the environment as well as better education outcomes, which typically make the children more efficient health producers in adulthood. Moreover, better health practices learned in childcare can form the basis for healthy behavior later in life and lead to better adult mental and physical health. In formal childcare, the professional staff at the childcare center can also help to detect health problems earlier and suggest preventive health measures that could reduce the likelihood of health problems at older ages. Evidence on the social determinants of health suggests that a strategy of prevention is more effective than treatment later in life (Marmot and Wilkinson, 2006), recognizing the dynamic nature of health capital formation and viewing policies aimed at shaping early-life environments as effective tools for promoting health (Conti and Heckman, 2014).

2.3 Physical Health

The health literature provides evidence that attending childcare is associated with a range of illnesses, such as asthma and other respiratory illnesses (Rantala et al., 2015; Illi et al., 2001; Ball et al., 2000; Busse et al., 2010; Nafstad et al., 2005), overweight, obesity, blood pressure and mortality, as well as healthcare use and health-affecting behaviors, such as smoking and exercising (D'Onise et al., 2010a,b). A limit of most of these studies, however, is their lack of empirical strategies that allow for causal interpretations.

Increased access to childcare may affect physical health through several channels. On the one hand, attending childcare means interacting with a group of children at an early age. According to the so-called hygiene hypothesis (Strachan, 1989), early exposure to other children

may first increase the incidence of infections but then decrease the rate later when the immunization process has finished, resulting in fewer infections, asthma, and allergies in older age. If the hygiene hypothesis holds, sending more children to childcare, making them highly exposed to infectious agents early in life, could reduce the number of children developing asthma and other allergies, or on a more moderate level, reduce the severity of asthma-related problems. This is a particularly interesting example since asthma is the leading chronic condition among children and is known to be one of the leading causes of pediatric emergency room use, hospitalization, and school absence (Currie, 2009). On the other hand, an increase in infections may cause increased use of antibiotics. Evidence from the medical literature links antibiotics use at an early age to several negative later-life health conditions, such as asthma and obesity (Neuman et al., 2018).

2.4 Mental Health

There is a possible association between attending childcare and mental health illnesses (D'Onise et al., 2010a,b). On the one hand, childcare, which typically involves several children and routines not specifically tailored to each individual child, can be stressful, and the children's cortisol levels have been found to be higher in childcare than at home (Vermeer and Groeneveld, 2017). A further stress factor is the separation from primary caregivers. Studies of stress response pathways have shown that the environment can become biologically embedded in the body in ways that can affect (also through latent pathways) health across the life course of an individual. The mechanisms through which adverse conditions early in life induce changes in brain structure are not yet fully known, but there are indications that these environmental stressors can affect epigenetic programming of long-term changes in neural development and behaviors (Conti and Heckman, 2013).

On the other hand, if children in childcare are exposed to a stimulating environment that supports cognitive and non-cognitive development, this may have positive effects on their mental health. First, an environment that stimulates communication, creativity, and motor skills has been shown to enhance a child's prospects in life. Second, professionals at the childcare center can support parents in identifying developmental and mental health problems and can help find strategies for dealing with them earlier than would have been the case if the child did not attend childcare (Heckman and Masterov, 2007). This could ultimately lead to fewer mental health problems in adulthood.

2.5 Education and Health

A more indirect mechanism through which childcare can affect long-run health is through potential effects on socioeconomic determinants, such as education, employment, and income (Heckman et al., 2010) — factors that can also have an independent effect on health, as doc-

umented by a large body of literature (Deaton, 2003; Heckman and Mosso, 2014; Lochner, 2011). Havnes and Mogstad (2011b) use the same reform as us and find that subsidized childcare had positive effects on children's educational attainment and labor market participation as well as negative effects on welfare dependency, indicating that any effect we find on long-run health could also come indirectly through the improved socioeconomic status of the affected children. Unfortunately, we do not have data that allow us to investigate the short- and mid-term effects on health. Our focus is therefore on the total effect of childcare on long-run health outcomes, but along the way, we investigate potential channels that point in the direction of both direct and indirect effects.

3 Institutional Background

3.1 The 1975 Childcare Reform

The Kindergarten Act, passed by the Norwegian Parliament in June 1975, introduced universal subsidized childcare for children three to six years old in Norway. The act regulated the authorization, operation, and supervision of formal childcare institutions across the country. According to the act, the municipalities were responsible for building and operating childcare facilities, while the Norwegian Ministry of Administration and Consumer Affairs held the responsibility for the overall regulations of formal childcare.

The background for the political process that led to the 1975 childcare reform was the entry into the labor market of married women with children, causing a growing demand for out-of-home childcare. In the years prior to the childcare reform of 1975, there was little supply of formal childcare, and most families had to rely on informal childcare arrangements for their childcare. In a nationwide survey from 1968 conducted by Statistics Norway, families were asked questions regarding childcare, including which type of care they currently used, if the mother was not taking care of the child. Out of these, 35% were looked after by relatives, 20% were in play parks, 15% were looked after by maids, 10% by nannies, and 7% by friends and neighbors. Only 14% were in formal care (enrolled in a childcare institution). On average, among parents with children three to six years of age, 32% stated a demand for formal childcare. Among employed women, this number was over 90% (Norwegian Ministry of Administration and Consumer Affairs, 1972).

Before the 1975 reform, the focus of the formal childcare system was on children with special needs. The arguments for making the program universal at the time were to create positive arenas for child development and to free further labor market reserves among mothers (Norwegian Ministry of Administration and Consumer Affairs, 1972). The reform created a significant positive shock to the supply of formal childcare. The aim of the reform was to reach 125,000 childcare places by 1981 (Norwegian Ministry of Administration and Consumer

Affairs, 1972), and municipalities with relatively low childcare coverage rates were awarded the highest subsidies. After the reform, each municipality was required to draw up a program in which they stated the pace of expansion, the types of operation, and the extent of the childcare activities. For the operation, the municipalities received state subsidies per child. The state also granted municipalities subsidies for the construction of childcare institutions and covered up to 85% of the construction costs. The prerequisite for the grant was that the childcare institution fulfilled the requirements of the law (Balke, 1979).

In 1976, 51% of the municipalities had no childcare institutions, 24% had one, and 9% had two institutions (NSD, 1976). The coverage rate increased substantially in the years following the reform. By 1979, the average national childcare coverage rate increased to above 28 percent from a coverage rate of less than 10 percent for children aged three to six in 1975. This corresponds to more than a doubling of total childcare places over this period. In the analysis, we follow Havnes and Mogstad (2011b) and focus on the childcare expansion from 1976 to 1979, which likely reflects the sudden increase in the supply of childcare places because of the reform instead of a spike in the local demand for childcare (Havnes and Mogstad, 2011b).

As documented by Havnes and Mogstad (2011a), the 1975 Kindergarten Act caused center-based care to crowd out informal care, and led to almost no net increase in maternal labor supply. The results we present can therefore be thought of as consequences of moving from informal care, rather than parental care, into relatively high-quality formal care for the affected children. The fact that the reform had little if any effect on maternal employment also means that it is unlikely that increased family income is the driving factor behind our results.[3]

3.2 Organization and Contents of Childcare in the 1970s

Organization. In the 1970s, childcare institutions in Norway were jointly financed by the central government, the municipalities, and the parents and were run by either the municipalities, public institutions, private organizations, or private firms under the supervision of the municipality. All officially approved childcare institutions, public or private, received operating subsidies from the central government, determined based on the number and age of children and the amount of time they spent in childcare. In 1976, 56% of the childcare institutions were owned by the municipalities, 7% by the regional or state government, 20% by private organizations, and 5% by churches. The rest of the childcare institutions were owned by parents (4.8%), private firms (3.5%), foundations (0.6%), housing cooperatives (0.3%), and other unspecified owners (2.7%). The majority of institutions were open during normal working hours (8am–4pm). In 1976, 13.8% of the childcare institutions were open less than 30 hours per week, 19.5% were open between 30–39 hours per week, 32.5% between 40–49, 26.6%

[3]The reform could still affect disposable income if the price of informal care was different from that of formal care. Unfortunately, we do not have data on the price or use of informal care before the reform.

between 50–55, and 7.4% over 55 hours per week (NSD, 1976).

Eligibility. All children were eligible for a slot, which was in general allocated according to length of time on the waiting list and the child's age. Only under special circumstances could a child get priority on the waiting list (Leira, 1992). In 1976, 13% of the children in childcare were children of single parents, 50% were children of working parents, 4% were children of parents in education, and 2% were children of parents who, due to illness, could not take care of the children during the day. A total of 14.7% of the children were in child care 6–15 hours per week, 37.6% 16–30 hours per week, 32% 31–40 hours per week, and 15% more than 40 hours per week (NSD, 1976).

Staff and Requirements. An educated preschool teacher supervised the day-to-day management, and there were federal requirements on the educational content and activities, group size, staff skill composition, and physical environment, regardless of ownership. In 1976, 32.6% of the employees in the childcare institutions were managers and preschool teachers (30% of these had approved preschool education), 25% were interns, 23% were assistants, 4% were children nurses, and 15% had unspecified positions (NSD, 1976).

Educational content. A social pedagogy tradition dominated the childcare programs. In practice, this meant that children were supposed to develop social, language, and physical skills mainly through play and informal learning. The preschool teacher education at the time can inform us more about the content of the childcare programs.

According to the study plans, the preschool teachers' main task was to create an environment in the childcare institution that promoted children's physical and mental development and ensured their well-being, safety, and health. Through their education, preschool teachers learned about children's behavior and ways of reaction. In addition, there was a special focus on physical, emotional, social, cognitive, moral, and identity development. There was also a focus on group dynamics, particularly on the socialization process, roles and expectations, forms of leadership, conflict and conflict resolution, and the role of the child in the family (Lærerutdanningsrådet, 1979).

In addition to being responsible for the educational content, the preschool teachers were responsible for the organization of the daily activities in childcare. They were also responsible for the preparation and assessment of 1) daily, weekly, and annual programs covering children at different stages of development, 2) children who needed special help and support, and 3) use of music, drama, movement, art, and literature (Lærerutdanningsrådet, 1979).

Meals. Meals were an important part of the daily program. The children ate together once or twice per day. In the toddler group, children often ate hot food. Otherwise, the food was usually simple, with meals consisting of bread, milk, and fruit. In the full-day institutions, half-day staff often ran the kitchen. The children usually sat at small tables with four to six children

and preferably an adult. In educational terms, the meal was important for the well-being of the children. The children learned to eat by themselves, they participated in preparation and cleaning up after the meal, and the dining situation was meant to be a pleasant social situation that increased the children's well-being and allowed for conversation and contact (Balke, 1979). In 1976, 56% of the childcare institutions had no food payments, 29% had low food costs (less than 40 NOK), while 15% had high food costs (40 NOK or more) (NSD, 1976).

Sleep. In general, individual needs were supposed to dictate sleep arrangements. In groups in which most children needed rest, the children typically lay on mats while the preschool teacher read or sang or they listened to music. There was also some individualization of rest hours if not all children needed rest. Typically, those who needed it slept in a quiet corner, while the rest of the children had a quiet reading hour (Balke, 1979).

Outdoor activities. Regarding time spent outdoors, the principle was that all children should be outside in fresh air during the bright part of the day all year around (Balke, 1979), and this has been a guiding principle for Norwegian childcare for decades.

Health. Although there was a strong focus on strengthening healthy behaviors and children's non-cognitive skills, the childcare programs did not offer any health services, and they had no access to health personnel. There was a universal vaccination program with very high coverage rates in place in Norway at the time, but these services were distributed to all children through mother and child health centers (Bütikofer et al., 2019), and as such they were not a part of the childcare system directly or indirectly.

4 Data

Data on the rollout of universal childcare in Norway is linked with individual administrative data from several sources described in this section.

4.1 Municipal Childcare Coverage Rates

The Norwegian Centre for Research Data provides childcare data through their municipality database. From this register, we have information on the annual number of children in formal childcare by the age of the child from 1973 and onwards, for each of the municipalities, 445 in total. The register also provides information on the total number of children at different ages, allowing us to calculate childcare coverage rates as the number of children attending formal childcare from age three to six over the total number of children in this age group.[4]

[4]Individual data on childcare attendance would be preferable since it makes it possible to identify take-up of the reform precisely, but such information is unfortunately not available for this period.

4.2 Municipality Characteristics

The Norwegian Centre for Research Data provides data on municipality characteristics for the period 1973–1985. From this register, we obtain information on family structure, unemployment, revenues (including central government transfers), total expenditures, expenditures on primary school, as well as a number of political variables.

4.3 Demographic Background Variables

Individual background information comes from administrative registers provided by Statistics Norway covering the entire resident population of Norway from 1967–2014. From these registers, we obtain data on gender, immigrant status, birth date, municipality of birth and residence, as well as on educational attainment and earnings. The registers also contain individual identification numbers that allow us to match children to their parents and siblings. We merge this data to the health registers described below using the individual identification numbers.

4.4 Health Data

Primary healthcare. The Control and Distribution of Health Reimbursement database (KUHR) provides detailed information about the use of primary care services and is available for the years 2006–2014. In Norway, the primary healthcare system is list based, which means that all Norwegian citizens belong to a specific GP's list. The GPs are responsible for providing primary healthcare services as well as referring patients to specialist healthcare services. The GPs in this system are financed by a mix of capitation (a lump sum per patient on their list — on average 30% of their income) combined with fee for service (on average 70% of the income). The GPs are required to report all the services they provide to each patient. For each consultation, they send an invoice to the Health Economics Administration (HELFO), which includes the patient's personal ID number, a report of the procedures used in the consultation, and a classification of the main diagnosis given by ICPC-2 codes[5] linked to the consultation. All this information is then stored in the KUHR database.

Specialist healthcare. Information about the use of specialist healthcare services comes from the Norwegian Patient Registry (NPR), available from 2008–2014. This register contains data on all admissions to somatic and psychiatric hospitals, both inpatient (overnight stays) and outpatient (day treatments and shorter consultations). For all admissions, the register provides information about the related diagnoses, both main and secondary, given by ICD-10 codes[6].

[5]The International Classification of Primary Care (ICPC) is a classification method for primary care encounters. It classifies the patient's reason for the encounter and the related diagnosis as well as the procedures done by the primary healthcare service.

[6]ICD-10 is the International Statistical Classification of Diseases and Related Health Problems, a medical classification list by the World Health Organization. It contains codes for diseases, signs and symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases.

Sickness Absence. The Norwegian Labor and Welfare Administration (NAV) provides data on sickness absence. This register contains every sickness absence spell certified by a general practitioner and the related diagnoses from 1995–2014. For each certified sickness absence spell, we have information on the start and end dates, and the related diagnosis from ICPC-2 codes. In Norway, sickness insurance is mandatory and covers all individuals who have been employed at the same employer for at least four weeks. Generally, workers are entitled to at least three days of self-reported sickness absence per spell, but in some workplaces, workers are entitled to up to eight days. For absences lasting more than three (eight) days, medical certification is required. The employer covers the first 16 days of absence, while the Norwegian Labor and Welfare Administration covers day 17 onwards. The replacement rate is 100% up to an amount of 6 G[7] (approximately 66,000 USD in 2019) from the first day of absence up to one year. At first thought, it may seem natural to condition sickness absence on working, but as the reform has had effects on labor market participation, this would be an endogenous measure, and we choose to use the unconditional measure of sickness absence.

Birth Outcomes for the Second Generation Children. The Norwegian Medical Birth Registry provides birth records for all Norwegian births over the period 1967 to 2014. The birth records contain information on date of birth, type of birth, gestational length, and a range of variables describing infant health at birth.[8] We use this register to look at birth outcomes of the second generation affected and to construct health measures characterizing the municipalities at the time around the reform.

4.5 Outcome Variables

The adult health outcomes are defined in two ways. First, we define the outcomes as the total number of events in the period 2006–2014 (2008–2014 for specialist healthcare services). These outcomes measure how many times an individual uses different healthcare services and how many days of sickness absence an individual has, measuring healthcare use at the intensive margin. Secondly, we define the outcomes as an indicator variable that is equal to one if an individual had any use of health services or any sickness absence during the period of 2006–2014 (2008–2014 for the specialist healthcare services), measuring the probability of experiencing any such event. This measure thus picks up changes at the extensive margin. The adult health outcomes are measured when the individuals in our sample are between 30 and 47 years old.

We start by examining all incidents in total (visits and sickness absence) and then continue to break these down by major groups of diagnoses based on findings from the studies described in Section 2. More precisely, we examine health care use and sickness absences related to

[7] G is an inflation-adjusted unit for calculation of social benefits in Norway.

[8] The birth records also provide information about maternal health during pregnancy and birth, but for the period we study the quality of this information is relatively poor, as there are many missing observations for mothers. We have as a result chosen not to use these.

metabolic, cardiovascular, musculoskeletal, respiratory, psychological, injury, social problems, and pregnancy diagnoses separately. For a detailed definition of the health outcomes, see Tables A11 and A12 in the Appendix.

The birth outcomes of the second generation that we examine are birth weight, the probability of having a birth weight below 1500 gram, below 2500 grams, and above 4000 grams. We also examine appearance, pulse, grimace, activity, and respiration (APGAR) scores and gestational length. In addition, we examine type of birth — spontaneous, induced, or caesarean section — and three types of caesarean section: elective, acute, and unspecified.

4.6 Sample Selection and Descriptive Statistics

To construct the baseline sample, we start with the whole population of individuals born in Norway in 1967–1976. This sample consists of 572,840 individuals. We then exclude all individuals that died before 2006 (1% of the above sample). To avoid outliers, we exclude individuals whose mothers gave birth before age 16 and after age 49, which is 332 individuals. The final sample consists of 566,914 individuals from 370,349 mothers.

Table 1 shows the background characteristics of the individuals in our sample. We divide the sample into three groups based on exposure to the reform. Pre-reform cohorts were not exposed to the reform and are defined as those born in the period 1967–1969. Phase-in cohorts are born in the period 1970–1972, and they were somewhat exposed to the reform. Post-reform cohorts, which were fully exposed to the reform, are born in the period 1973–1976. The background characteristics are rather similar for all three groups, indicating that none of these characteristics are likely to explain any differences in outcomes between the cohorts. A total of 49% of the children in the sample are female, and 6% are immigrants. The mothers on average gave birth for the first time at age 23, while fathers were on average 26 years old at first birth. The parents on average had around 12 years of schooling, which corresponds to completing high school, when the child was two years old. The children have on average one older sibling. The only background characteristic that differs between the three groups is the share of the sample that moved between treatment and control municipalities in the post-reform period. For the pre-reform cohort the share is 4%, for the phase-in cohorts the share is 6%, and for the post-reform cohorts it is 9%. However, in the main estimation specification, we control for all the background characteristics. We also exclude all individuals who move between treatment and control municipalities in a robustness check, and the results remain similar to the baseline results.

Table 2 shows characteristics, such as years of education, earnings, employment, municipality expenditures and revenues, population characteristics, and politic of treatment and control municipalities in 1976. As can be seen from the table, there were no large differences between treatment and control municipalities in any of the municipality characteristics in 1976. To en-

sure that this is the case for the whole post-reform period, we also investigate municipality characteristics time trends from 1973 to 1985. These trends are presented in Figures A.1–A.7 in the Appendix.

5 Empirical Strategy

We follow Havnes and Mogstad (2011b) and apply a reduced form model that exploits the differential increase in childcare coverage across municipalities, in combination with differential exposure to the reform across cohorts.[9][10] Specifically, we compare adult health outcomes of children who were three to six years old before and after the reform, from municipalities where the expansion of childcare coverage were above and below the national median.

As shown in Figure 1, there was large geographical variation in childcare coverage rates in the post-reform period. We use this variation to define our treatment and control municipalities. Municipalities where the increase in childcare coverage rates was above the median in the post-reform period (1976–1979) are defined as treatment municipalities, while control municipalities had increases in childcare coverage rates below the median in the post-reform period. Figure 2 shows the development of childcare coverage rates in treatment (solid line) and control (dotted line) municipalities over time. In the pre-reform period, the levels are more or less the same on average and follow the same trend. Then, following from our definition of treatment and control municipalities, treated municipalities experience a larger increase in coverage rates until around 1979, before the trends again become parallel. The cohorts in our sample are divided into three groups. Pre-reform cohorts were not exposed to the reform and are defined as those born in the period of 1967–1969. Phase-in cohorts are born in the period of 1970–1972, and they were somewhat exposed to the reform. Post-reform cohorts, which were fully exposed to the reform, are born in the period of 1973–1976.

5.1 Main Specification

The main estimating equation is given by:

$$\begin{aligned} Y_{ijt} = \beta_1 + \beta_2 Treat_j + \beta_3 \left(Phase - in_t \times Treat_j\right) \\ + \beta_4 \left(Post_t \times Treat_j\right) + \beta_5 X_i + \theta_j + \gamma_t + \epsilon_{ijt} \end{aligned} \tag{1}$$

[9] Our main results are not directly comparable with those of Havnes and Mogstad (2011b), as we cut the sample differently. In contrast to Havnes and Mogstad (2011b), we include 1) children of unmarried mothers, 2) individuals moving between municipalities, and 3) more municipalities than they do. In our robustness checks, we run regressions on samples more similar to that of Havnes and Mogstad (2011b).

[10] A worry could be that it is not random which municipalities experienced the largest increases in coverage rates, and that this non-randomness could in turn affect our outcomes. An alternative could therefore be to use the pre-reform levels as exogenous predictors of post-reform growth. However, given that around 67% of the municipalities had no formal childcare institutions before the reform, there is not enough variation in the pre-reform levels. Instead, we investigate the assumption of parallel trends for a number of municipality characteristics, and there are no indications of other characteristics driving our results. We also include robustness checks, in which we correct for differential trends in outcomes in various ways.

where *Y* is the health outcome of interest, for child *i*, residing in municipality *j* in 1976, and turning three years old in year *t*. $Treat_j$ is a dummy variable equal to 1 if child *i* lives in a treatment municipality in the post-reform period. $Phase-in$ and $Post$ are dummy variables equal to 1 when $t \in [1973, 1975]$ and $t \in [1976, 1979]$, respectively. The vector of covariates *X* includes parents' education when the child is two years old, the parents' age at first birth, number of siblings, birth order, the child's gender and immigrant status, and an indicator of relocation between treatment and control municipalities. θ_j is a set of municipality-specific fixed effects, and γ_t is a set of cohort-specific fixed effects. Thus, the municipality fixed effects absorb unobservable determinants of the long-run outcomes, which are fixed at the municipality level, while common time shocks are controlled for by the cohort fixed effects. Standard errors are clustered at the level of municipality of residence in 1976. As in Havnes and Mogstad (2011b), Baker et al. (2008), and others, we interpret β_3 and β_4 as intention-to-treat (ITT) effects, as we do not have information on actual use of childcare and can only estimate reduced form effects.

5.2 Identifying Assumptions

For identification it is essential that the change in the health outcomes of interest for the included cohort groups (pre, phase-in, and post-reform) would have been the same in municipalities with high and low growth in childcare coverage in the absence of the reform. Municipality fixed effects are included in the main specification, and so time-invariant municipality characteristics that may be correlated with both the growth in coverage rates and health outcomes will not bias our estimates. However, there may be time-varying municipality characteristics that the fixed effects do not capture. To address this, we show that post-reform growth in childcare coverage was not correlated with other factors at the municipality level that could potentially affect our outcome variables. Figures A.1 and A.3 in the Appendix display the development of municipality characteristics, such as family structure, unemployment, revenues (including central government transfers), total expenditures, expenditures on primary school, and health expenditures in the period from 1973 to 1985, in treatment and control municipalities. These graphs are all based on municipality data. Unfortunately, there is no information about the general health of the population in the municipality database, and so we use administrative register data from different sources to construct measures of municipality health. Figure A.5 in the Appendix displays the development in municipality characteristics such as income, share of population with higher education, birth weight of babies born, and number of live births. Finally, in Figure A.7 we show the development in a number of political variables measured at municipal elections (every 4th year). The general impression from all these figures is that there is no substantial difference between the development in the two sets of municipalities that could explain our results.

When evaluating the introduction of a reform, there is always a worry about confounding the effects of the reform with those from other reforms or changes taking place in the same period. However, except for a reform from 1977, which introduced paid maternity leave and extended the period of job protection, we have found no other reforms that were introduced in the same period. An extension in maternity leave could possibly influence family size and in turn matter for child development. However, this reform did not lead to changes in family size, and more importantly, it was implemented at a national level and should therefore be controlled for by the cohort fixed effects (Carneiro et al., 2015).

5.3 Multiple Hypothesis Testing

We estimate the effects on many outcomes and consequently correct the p-values for multiple hypothesis testing. We use the Benjamini and Hochberg (1995) q-values method described in Anderson (2008), in which we control for the false discovery rate (FDR), or the proportion of rejections that are "false discoveries" (type 1 errors). We group outcomes into families based on the level of healthcare services and how the outcomes are defined. For example, primary healthcare use related to physical health defined as the total number of incidents over the period examined is one family of outcomes.

6 Results

6.1 Physical Health

Table 3 presents the ITT effects of the 1975 reform, estimated as described in Equation 1, on the use of healthcare services related to physical health.[11] We organize the table into three panels representing primary care (Panel A), specialist care (Panel B), and sickness absence (Panel C). In each panel, the first row presents effects on outcomes defined as the total number of incidents over the period examined (i.e., 2006–2014 for primary care and sickness absence, and 2008–2014 for specialist care), while the second row shows effects on outcomes defined as the probability of an event over these periods. In this way, we capture effects both on the intensive and extensive margins of healthcare use and sickness absence. For each outcome, we look at all incidents (Column 1), and based on the discussion in Section 2, we examine specific diagnosis groups. Columns 2 to 7 show effects on metabolic, cardiovascular, musculoskeletal, respiratory, and pregnancy diagnoses.[12]

When looking at Table 3, the general picture is that there are few effects of the reform on the use of healthcare services related to physical health. There are, however, a couple of exceptions. First, the use of healthcare services and sickness absences related to pregnancies increase

[11] Due to limited space, we only present coefficients for the post-cohorts. Phase-in estimates are similar, but generally smaller and not statistically significant. They are available on request.

[12] For detailed definitions of the outcomes, see Table A11 and Table A12 in the Appendix.

as a result of the reform (Column 6). The number of GP consultations related to pregnancies increases by 7% (0.17 from a pre-reform mean of 2.39). For sickness absence related to pregnancies, we observe increases at both the intensive and the extensive margin: the number of sickness absence days increase by 27% (0.73 from a pre-reform mean of 2.67), while the probability increases by 17% (0.5 percentage points from a pre-reform mean of 3%). There is not a directly corresponding effect on the use of somatic specialist services related to pregnancies and childbirth (Panel B, Column 6). However, the total number of contacts with the specialist care increase by 3%, and this increase is driven by a set of pregnancy-related check-ups and controls in the category "Factors influencing health status and contact with health services" (The Z-category in the ICD-10 codes). When adjusting for multiple hypothesis testing, the effects on sickness absence are the only ones that survive. Second, in addition to the pregnancy related effects, we find a reduction in the probability of GP consultations related to respiratory diagnoses by 0.7% and an increase in the probability of sickness absence related to cardiovascular diagnoses by 4%. None of these survives multiple hypothesis testing.

6.1.1 Pregnancy Related Effects, Focusing on Females in the Sample

Table 4 presents the results of the model in which we interact the reform effects with gender. The table confirms that women drive all the pregnancy-related effects.[13] The observed increases in the use of healthcare services and sickness absences related to pregnancies can be thought of as preventive or precautionary visits and sickness absence, or they may reflect some underlying negative development in the health of fertile women and their babies. We therefore continue to investigate whether the observed increase in healthcare use is reflecting a change in behavior, a change in risk factors, or a change in the health status of mothers and their babies. In this quest, we base the analysis on the sample of women.

Type of Pregnancy. First, we examine whether the increased use of healthcare services related to pregnancies is driven by specific types of pregnancies or pregnancy-related activities in Table 5. Panel A depicts effects on the use of primary care and sickness absence, while panel B depicts effects on the use of specialist care. It turns out that normal pregnancies are the main drivers of the observed effects on pregnancy-related use of healthcare services. We find no effects on the use of either healthcare services or sickness absence related to high-risk pregnancies and no effect on antenatal screening. For normal pregnancies, however, the number of GP consultations increases by 45% (0.34 from a pre-reform mean of 0.75), while the probability of having a GP consultation increases by 11% (1.4 percentage points from a pre-reform mean

[13] Note that we do not have exact information on the take-up of childcare, only the increase in childcare coverage in each municipality. Any heterogeneous effects could therefore come from one group being more affected by the reform than the other is, but it could also come from differences in take-up. In the case of girls versus boys, there is no reason to believe that any group attended childcare more or less than the other group or that their take-up of the reform differed, but when looking at heterogeneity by family background later, it will be important to keep this in mind.

of 13%). Similarly, the total number of visits related to supervision of normal pregnancies in specialist healthcare increases by 67% (0.05 from a pre-reform mean of 0.07), while the probability of such visits increases by 60% (1.8 percentage points from a pre-reform mean of 3%). The pattern of normal pregnancies driving the results is also reflected in the effects on sickness absence. The probability of sickness absence related to a normal pregnancy increases by 30% (0.9 percentage points from a pre-reform of 3%), and there is an increase of 47% in the number of sickness absence days (0.01 from a pre-reform of 0.03).

Fertility. A reasonable hypothesis could be that we observe an increase in the use of healthcare services and sickness absence related to pregnancies because the treated group are having more children or they are having children later in life, compared to the control group. To check this, we estimate effects of the reform on age at first birth, the number of children, and the probability of having a child at different age cut-offs, namely, age 20, 25, 30, 35, and 40, which is the highest age at which we can observe post-reform cohorts in the data. The results are presented in Table 6, which shows that there are no changes in any of these outcomes for the treated group compared to the control group.[14] The increased use of healthcare services and sickness absence are therefore not likely driven by changes in fertility, either in number or in age.

Birth Outcomes for the Second Generation. A large body of literature has shown that there are positive correlations between health, education, and income (Deaton, 2003; Heckman and Mosso, 2014; Lochner, 2011). At the same time, Havnes and Mogstad (2011b) have shown that increased access to childcare has statistically significant positive effects on educational attainment, attachment to the labor force, and income when the children exposed to the reform are in their 30s. To investigate whether the increased use of healthcare services and sickness absence have preventive effects and improve the health of the babies of the individuals exposed to the reform or whether individuals exposed to the reform have higher education and simply demand more health services in general, we examine birth outcomes for the next generation of children (i.e., the children of the individuals exposed to the 1975 childcare reform).

Specifically, we examine birth weight, the probability of very low birth weight, low birth weight, and high birth weight as well as APGAR scores and gestation. In addition, we examine outcomes related to the type of birth. The estimated effects on these outcomes are presented in Table 7 and Table 8. Mostly, there are no effects of the reform on the babies in the second generation. There are some indications of slightly better birth outcomes, represented by a statistically significant decrease in the probability of high birth weight by 4.5% (0.9 percentage

[14] Havnes and Mogstad (2011b) find reductions in fertility measured in 2006, around the time when the sample was on average 30 years old. We find traces of the same effect in our study at age 30, but we compare outcomes at specific ages rather than at a certain point of time, and the effect seems to be very little robust when looking at different ages.

points from a pre-reform mean of 20%), as well as a statistically significant positive effect on the APGAR score of 0.5%. However, these improvements come at a margin that has few meaningful health consequences.

6.2 Mental Health and Risky Behavior

Table 9 presents the effects on the use of health services related to mental health and risky behavior. We organize the table into two panels representing primary care (Panel A) and specialist care (Panel B). In each panel, the first row presents effects on outcomes defined as the total number of incidents over the period examined (i.e., 2006–2014 for primary care and 2008–2014 for specialist care), while the second row shows effects on outcomes defined as the probability of an event over these periods.

Columns 1–3 in Panel A show that those who are affected by the reform have reductions in the probability of GP consultations related to psychological diagnoses and symptoms by 1.2% (0.7 percentage points from a pre-reform mean of 57%), in GP consultations related to injuries by 1.9% (0.6 percentage points from a pre-reform mean of 33%) and in GP consultations related to social problems by 2% (0.7 percentage points from a pre-reform mean of 34%), respectively. Also in the specialist care, there is a reduction in the probability of admissions related to injuries by 3.3% (0.7 percentage points from a pre-reform mean of 21%), as shown in Column 3 in Panel B. All of these reductions are at the extensive margin, which means that the probability of going at all is reduced. We observe no change in the frequencies for those going. For specialist psychiatric care, however, we observe effects only at the intensive margin. We find that the reform led to a 10% reduction in the number of psychiatric specialist care visits (0.4 from a pre-reform mean of 3.88), as can be seen in Column 4 in Panel B. When correcting for multiple hypothesis testing, only the results on GP visits related to mental health and risky behavior survive. We find no statistically significant effects on local ER visits or acute admissions to specialist care (somatic or psychiatric).

Other outcomes of interest in this context are mortality (including suicides), accidents, and self-harm. The means of these outcomes are very close to zero, and we have therefore not been not able to identify any effects on these outcomes. The results are available upon request.

In Table 10, we investigate whether there are differences in the impacts between boys and girls affected by the reform. In contrast to what we find regarding the use of somatic healthcare, we find no gender differences in the use of mental healthcare and healthcare related to risky behavior.

6.3 Heterogeneity by Family Background

We now turn to examining heterogeneity using three different measures of family background: mothers' education level, mothers' employment status, and household income. All these in-

dicators are based on data from 1975 to ensure that we are conditioning the heterogeneity on pre-reform conditions. One reason that governments offer subsidized universal childcare is to counter differences in school readiness between children from different family backgrounds and create equal opportunities for all children. Previous research based on the reform we study finds that childcare had positive effects on children with low socioeconomic status, while it had no or disadvantageous effects on children with high socioeconomic status on long-term educational attainment and labor market attachment (Havnes and Mogstad, 2015). In other settings, girls and children with low-educated parents have been shown to benefit the most from attending childcare (Almond and Currie, 2011), and children of immigrants have been shown to experience higher returns from early childcare attendance in terms of overall school readiness than native children (Felfe and Lalive, 2018; Cornelissen et al., 2018).

In Tables A1–A6, we present heterogeneity by mothers' education level, mothers' employment status, and household income. The general picture is that there are very few statistically significant or systematic differences between the estimated ITT effects of children from different family backgrounds. As we do not have exact information on the take up of childcare, only the increase in childcare coverage in each municipality, we are not able to establish whether this is because there are in fact no differences in how the groups were affected or whether possible differences in take-up and effects cancel each other out. That said, an interesting pattern is that children of employed mothers display stronger ITT effects on both the general level of sickness absence and on sickness absences related to pregnancies than do children of non-working mothers. The same pattern appears when looking at the use of psychiatric specialist services and services related to injuries (which is our indicator of risky behavior): children of employed mothers are driving the effects. Knowing that the reform implied a shift from informal care arrangements to formal ones, and that it had no effect on mothers' labor supply, this makes sense because the children of working mothers are more likely to take up the reform and thus be directly affected by it.

7 Robustness

7.1 Specification Checks

To ensure that our results are not merely a product of choices made regarding specification and sample selection, we run a number of robustness checks, presented in Tables A7 and A8. Overall, the estimates across the different specifications are relatively similar to the baseline results. In both tables, Column 1 shows the baseline results for comparison.

In Table A7, we check the robustness of the results with respect to sample selection and alternative treatment definitions. In the baseline sample, we include children from families that move between treatment and control municipalities in the post-reform period (1976–1979), but

those moving between treatment and control municipalities may bias our estimate of the impact of the reform. In Column 2, we therefore exclude all individuals that move between treatment and control municipalities in the post-reform period. Another sample selection decision was to include children of mothers who were not married in 1975. To ensure that this is not driving our results and to provide a more direct comparison with Havnes and Mogstad (2011b), we run regressions in which we exclude children of mothers who were not married in 1975. We cannot distinguish between cohabitant and single parents in our data from this period, so these children may either have parents that are single or cohabiting. The results from this regression are presented in Column 3. The results do not change when we exclude movers or children of unmarried mothers.

Furthermore, to ensure that our results are not driven by outlier municipalities with a very small population size or the larger cities that were often organized slightly differently than smaller municipalities, we exclude all municipalities with less than 1000 inhabitants (20 municipalities) in Column 4 and the three largest cities (cities with a population size above 100,000 at that time) in Column 5. The results are very similar to the baseline results.

In Columns 6–8, we define the treatment variable and treatment groups in different ways. In Column 6, we merge the phase-in cohort group and post-reform group into one group. As can be expected, the coefficients are a bit smaller with these definitions, as the phase-in cohorts were less exposed to the reform than the post-reform cohorts. We also lose some precision when combining these cohorts into one group. In Column 7, we exclude the phase-in cohort group from the sample entirely. The results using this sample are similar to the baseline results, but again we lose some precision. In Column 8, we use the increase in childcare coverage as a continuous treatment variable instead of splitting the municipalities into treatment and control groups at the median of the childcare coverage rate increase in the post-reform period. Estimated coefficients as they are reported in the table are not directly comparable with those from the main specification, but the coefficients have the same signs, and the findings on psychiatric specialist care remain robust.

In Table A8, we first present results from a specification in which we estimate Equation 1 without the set of individual controls X in Column 2 to check whether our estimates are subjects to selection bias. Although we have graphically examined several municipality characteristics and will also estimate Equation 1 with municipality-specific time trends in Columns 3–5, a concern could be that the time trend in children's outcomes differ by, for example, parent's education, while there are also systematic differences in parental education levels between treatment and control municipalities. The results remain the same when we use this approach, confirming that this type of selection bias is not an issue.

Second, to allow the outcomes in treatment and control municipalities to follow different time trends, we estimate municipality-specific time trends. We first use data on cohorts not

exposed to reform, that is, individuals born before 1970, to obtain slope estimates for each municipality from the pre-reform period and extrapolate the estimated pre-reform time trends to the post-reform period. In Column 3, we include a linear municipality-specific time trend, and in Column 4, we include a quadratic municipality-specific time trend. Finally, in Column 5, we use a direct municipality-specific trend, in which we interact municipality dummies with birth year of the cohorts. The results are robust to the inclusion of all types of trends.

7.2 Placebo Reform

When estimating the effects of a reform using a differences-in-differences approach, there is always the concern that the estimated effects may reflect differential time trends between treatment and control municipalities instead of actual effects of the reform. In addition to including municipality-specific time trends as a robustness check, we therefore examine the identification assumption of a common time trend between treatment and control municipalities in the absence of the reform by performing a placebo-analysis on children from the pre-reform cohorts. Estimated effects on cohorts born in 1968 and 1969, relative to the 1967 cohort, are presented in Table A9. The results show that none of the placebo effects are significant.

7.3 Family Fixed Effects

The baseline specification does not fully account for family-specific unobserved factors that could affect our estimates. In order to account for this, we add family fixed effects and re-estimate Equation 1 on a sample of siblings. Using this approach, we can compare siblings who grew up in the same family but had different exposure to the reform, allowing us to control for time-invariant family heterogeneity. The results from this estimation are presented in Table A10. The sample includes all children with at least one sibling and in which at least one sibling is not exposed to the reform (i.e., the pre-reform cohorts, born in 1967–1969) and at least one is (i.e., the phase-in or post-reform cohorts, born in 1970–1976). In Column (1), we show estimates from the main specification on the baseline sample for comparison. In Column (2), we estimate the main specification on the sibling sample. In Column (3), we include family fixed effects using the sibling sample. The effects on psychiatric healthcare are robust when we use the main specification on the sibling sample, but we lose precision and do not find statistically significant effects on somatic specialist care. When we include the family-specific fixed effects, we lose even more precision, and although the coefficients remain similar to those in the main specification, no effects are any longer statistically significant.

8 Conclusion

In this paper, we examine the long-run health outcomes of children affected by a reform that led to a large-scale expansion of subsidized universal childcare for children three to six years old in

Norway in the late 1970s. Before the reform, formal childcare was severely constrained, while in the years after the reform, formal childcare coverage rates grew substantially. As shown by Havnes and Mogstad (2011a), the childcare reform did not cause increased female labor market participation. Our results must therefore be interpreted as the effects of going from informal to formal care arrangements.

We have two main findings. First, women affected by the reform increase their use of pregnancy-related healthcare services and sickness absence. However, there is no increase in fertility and no effects on the second generation's birth outcomes, indicating that the women's health is unchanged, but that they have increased their demand for healthcare services, suggesting a change in health-seeking behavior rather than a change in health. However, in the long run, as these individuals become older, more preventive behavior such as more health check-ups, may translate into better health. A change in behavior could come directly from the practices and habits formed already in childcare, but it is more likely an indirect effect. It is well established that there is a socioeconomic gradient in the use of healthcare services (Monstad et al., 2014; Kaarboe and Carlsen, 2014; Moscelli et al., 2018; Cutler and Lleras-Muney, 2010), and the observed effect can thus be an indirect consequence of the reform, resulting from the identified positive effects on education and income (Havnes and Mogstad, 2011b).

Second, there is a reduction in the use of mental healthcare services, and services related to injuries and social problems. The reduction in the use of mental health services could reflect improved mental health or a change in behavior towards less help seeking. There is a reduction both in the probability of visiting a GP and in the use of psychiatric specialist care. The latter is especially indicative of better mental health, as there is high excess demand for mental healthcare in Norway, and individuals are only granted access to these specialist services once the mental health problems have become severe. Together this suggests that formal childcare benefits individuals by improving their mental health in the long run. The routines and pedagogical environment of childcare could strengthen social skills and induce better decision-making and healthier behavior that last into adulthood. Being in a formal childcare institution could also increase the chances of detecting behavioral, social and psychological problems at an early stage and could thus prevent the development of more serious problems. Both of these explanations point to direct effects of childcare, but we are not able to exclude the possibility of an alternative or additional effect related to the already identified increases in education and income.

The heterogeneity analysis shows that women, as expected, drive the pregnancy-related effects, while we are not able to identify any gender differences on the effects on the use of mental healthcare services. We find that children of employed mothers are driving the effects. Knowing that the reform implied a shift from informal care arrangements to formal ones — and that this had no effect on mothers' labor supply — this makes sense: the children of working

mothers are the ones more likely to take up the reform and thus the ones most likely to be directly affected by it.

Our findings are relevant for ongoing policy debates in the US, Canada, and many European countries about a possible move towards subsidized, universally accessible childcare or preschool. In addition, they add to the discussion of how authorities can control the soaring healthcare costs in many countries. For example, the focus of the discussion is often on the provision of healthcare to cure or improve the health of patients. However, an increased focus on ways to prevent diseases, for example, by promoting healthy behavior can potentially save the society and individuals large costs. Chronic diseases represent a large share of the increased healthcare costs in many countries. Such conditions are the main causes of premature death, and managing them effectively requires that patients make lifestyle changes by adhering to healthy behaviors. However, while prevention is crucial for lifelong health, changing behavior in adulthood may be challenging (Conti et al., 2016), and early-life interventions like childcare may prove to be more efficient behavior shifters. Similarly, mental health problems are among the main drivers of the increasing trend of young people on long-term social security benefits across OECD countries. If such problems can be detected and treated, or even prevented early in life, this has the potential to save both individuals and society from non-negligible costs.

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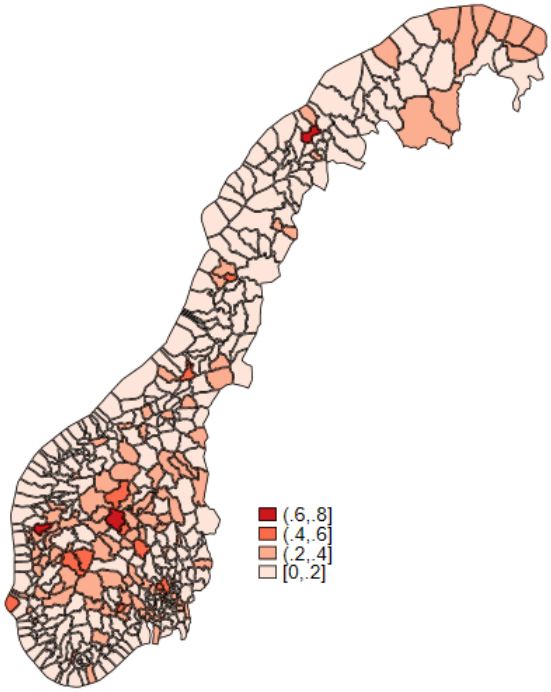
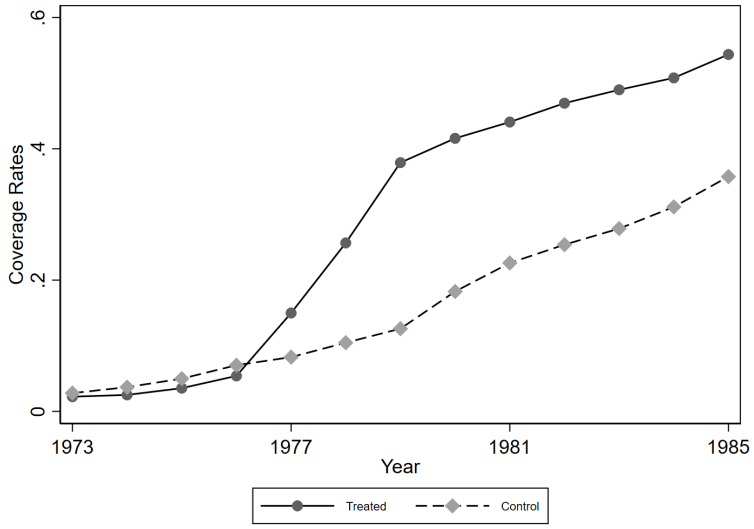
9 Figures and Tables

Figure 1: Average Childcare Coverage Rates in the Post-Reform Period (1976–1979)



Note: This map shows the variation in average childcare coverage rates across Norway's 445 municipalities in the post-reform period (1976–1979). Municipalities with the strongest red color have the highest childcare coverage rates.

Figure 2: Childcare Coverage Rates in Norway in 1973–1985 for Children Ages 3–6 in Treated and Control Municipalities



Note: This figure shows childcare coverage rates in 1973–1985 for children ages 3–6 in treated municipalities (municipalities with above median childcare coverage growth in the post-reform period (1976–1979)) and control municipalities (municipalities with below median childcare coverage growth in the post-reform period (1976–1979)).

Table 1: Descriptive Statistics

	Pre-reform cohorts		**Phase-in cohorts**		**Post-reform cohorts**	
	Mean	St.Dev.	Mean	St.Dev.	Mean	St.Dev.
Female	0.49	[0.50]	0.49	[0.50]	0.49	[0.50]
Immigrant	0.06	[0.23]	0.06	[0.23]	0.05	[0.23]
Mother's age at first birth	23.02	[3.97]	22.90	[3.79]	23.03	[3.79]
Mother's education when child is 2 y. o.	11.71	[2.01]	11.84	[2.04]	11.77	[1.96]
Father's age at first birth	26.27	[5.24]	25.89	[4.97]	25.82	[4.77]
Father's education when child is 2 y. o.	12.28	[2.52]	12.29	[2.44]	12.14	[2.24]
Older siblings	1.10	[1.23]	0.99	[1.16]	0.89	[1.05]
Relocated	0.04	[0.19]	0.06	[0.23]	0.09	[0.28]
Observations	194564		188750		183600	

Note: This table presents background characteristics of the individuals in the sample. Pre-reform cohorts are born between 1967–1969, phase-in cohorts are born between 1970–1972, and post-reform cohorts are born between 1973–1976. Standard deviations in brackets.

Table 2: Descriptive Statistics for Treatment and Control Municipalities in 1976

	Treatment		Control	
	Mean	St.Dev.	Mean	St.Dev.
Childcare coverage rate	0.0540	[0.0927]	0.0703	[0.0982]
Years of education, males	11.2982	[0.4674]	11.3000	[0.3761]
-, females	11.0780	[0.3092]	11.0308	[0.2592]
Earnings, males	52276	[7505]	53879	[7342]
-, females	23106	[4463]	23732	[4340]
Employment, males	0.8040	[0.0611]	0.8190	[0.0666]
-, females	0.4089	[0.0977]	0.4284	[0.0982]
Expenditures (1000 NOK/capita)				
Total	8798.62	[2505.35]	8382.39	[1450.16]
Primary school	1768.69	[604.92]	1697.54	[507.93]
Healthcare	302.73	[443.07]	326.71	[403.87]
Transfers and Revenues (1000 NOK/capita)				
Total	8798.61	[2505.35]	8382.45	[1450.14]
Primary school	835.09	[371.78]	785.98	[356.67]
Taxes	3520.12	[955.95]	3516.51	[864.05]
Population				
Total	9118	[34910]	8936	[12304]
Married	0.4673	[0.0308]	0.4665	[0.0358]
Divorced	0.0112	[0.0064]	0.0118	[0.0064]
Immigrant	0.0096	[0.0096]	0.0101	[0.0091]
0 to 6 years old	0.1051	[0.0182]	0.1099	[0.0174]
7 to 10 years old	0.0680	[0.0104]	0.0710	[0.0106]
11 to 18 years old	0.1302	[0.0136]	0.1326	[0.0133]
Females: 19 to 35 years old	0.1051	[0.0190]	0.1108	[0.0174]
-:36 to 55 years old	0.1005	[0.0102]	0.1011	[0.0100]
Males: 19 to 35 years old	0.1205	[0.0151]	0.1250	[0.0143]
-: 19 to 35 years old	0.1077	[0.0088]	0.1068	[0.0099]
Politics				
Registered voters	6302	[25780]	5937	[8284]
-, female	0.4896	[0.0185]	0.4937	[0.0175]
Election participation	0.7235	[0.0580]	0.7117	[0.0565]
-, female	0.7087	[0.0665]	0.6995	[0.0637]
Female elected representatives	0.1501	[0.0805]	0.1392	[0.0648]
Socialist vote share	0.3881	[0.1702]	0.4107	[0.1676]
Socialist mayor	0.3198	[0.4675]	0.3812	[0.4868]
Female mayor	0.0090	[0.0947]	0.0224	[0.1484]
N	222		223	

Note: This table presents characteristics of treatment and control municipalities in 1976. Standard deviations in brackets.

Table 3: Main Results: Physical Health

		Related diagnoses				
	All	Metabolic	Cardiovascular	Musculoskeletal	Respiratory	Pregnancy
	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A: Primary healthcare use						
- #	0.249	-0.026	0.001	-0.026	-0.033	0.168*
	(0.154)	(0.057)	(0.067)	(0.061)	(0.057)	(0.101)
Adj. p-value	.374	.781	.990	.781	.781	.374
Pre-reform mean	[21.70]	[2.62]	[2.82]	[6.56]	[3.88]	[2.39]
- Prob.	0.001	-0.005	-0.004	-0.003	-0.005*	-0.000
	(0.001)	(0.003)	(0.004)	(0.003)	(0.003)	(0.007)
Adj. p-value	.567	.353	.355	.355	.353	.997
Pre-reform mean	[.96]	[.44]	[.48]	[.79]	[.73]	[.44]
PANEL B: Specialist healthcare use						
- #	0.207**	0.004	-0.004	0.006	-0.005	0.035
	(0.096)	(0.023)	(0.009)	(0.025)	(0.008)	(0.027)
Adj. p-value	.219	.860	.860	.860	.860	.675
Pre-reform mean	[6.99]	[.41]	[.30]	[.94]	[.18]	[.13]
- Prob.	0.007**	0.002	0.004**	0.002	0.000	0.005
	(0.003)	(0.002)	(0.002)	(0.003)	(0.001)	(0.003)
Adj. p-value	.127	.497	.127	.541	.935	.198
Pre-reform mean	[.72]	[.06]	[.10]	[.25]	[.06]	[.04]
PANEL C: Sickness absence						
- #	2.170	0.021	-0.153	-0.191	0.009	0.727***
	(1.510)	(0.142)	(0.134)	(0.687)	(0.157)	(0.274)
Adj. p-value	.444	.956	.444	.956	.956	.058
Pre-reform mean	[146.00]	[2.09]	[3.04]	[44.70]	[3.85]	[2.67]
- Prob.	-0.002	0.001	-0.001	-0.005	-0.001	0.005**
	(0.003)	(0.001)	(0.001)	(0.003)	(0.001)	(0.002)
Adj. p-value	.522	.408	.408	.396	.408	.064
Pre-reform mean	[.54]	[.02]	[.03]	[.29]	[.05]	[.03]
N	566911	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, Panel B shows specialist healthcare use, and Panel C shows sickness absence. In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table 4: Heterogeneity by Gender

		Related diagnoses				
	Total	Metabolic	Cardiovascular	Musculoskeletal	Respiratory	Pregnancy
	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A: Primary healthcare use						
I. ITT (girl)	0.489**	-0.092	-0.036	-0.035	-0.050	0.223*
	(0.198)	(0.061)	(0.069)	(0.079)	(0.065)	(0.124)
II. ITT (boy)	-0.118	-0.024	-0.040	-0.043	-0.081	-0.045
	(0.150)	(0.060)	(0.064)	(0.066)	(0.061)	(0.057)
p-value (I=II)	.016	.377	.960	.938	.698	.011
PANEL B: Specialist healthcare use						
I. ITT (girl)	0.382***	-0.016	0.004	0.010	-0.000	0.049
	(0.134)	(0.025)	(0.010)	(0.031)	(0.011)	(0.047)
II. ITT (boy)	-0.127	0.017	-0.018	-0.016	-0.003	0.001
	(0.119)	(0.024)	(0.011)	(0.022)	(0.009)	(0.004)
p-value (I=II)	.007	.252	.178	.441	.882	.324
PANEL C: Sickness absence						
I. ITT (girl)	3.087	0.017	-0.197	-0.475	0.148	0.965**
	(2.152)	(0.225)	(0.148)	(0.866)	(0.228)	(0.487)
II. ITT (boy)	0.468	-0.000	-0.112	0.351	-0.015	-0.006
	(1.399)	(0.113)	(0.163)	(0.674)	(0.147)	(0.053)
p-value (I=II)	.272	.945	.696	.432	.516	.050
N	566911	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, Panel B shows specialist healthcare use, and Panel C shows sickness absence. In all three panels, the first row shows the estimated ITT effects for girls, while the second row shows the estimated ITT effect for boys. The outcomes are defined as the total number of incidents over the period. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table 5: Pregnancy Related Healthcare Use

	GP consultations		Sickness absence	
	Normal preg.	High-risk preg.	Normal preg.	High-risk preg.
	(1)	(2)	(3)	(4)
PANEL A: Primary healthcare use and sickness absence				
- #	0.335***	0.035*	0.014***	0.003
	(0.088)	(0.020)	(0.004)	(0.004)
Adj. p-value	.001	.131	.001	.422
Pre-reform mean	[.75]	[.15]	[.03]	[.03]
- Prob.	0.014***	0.003	0.009***	0.002
	(0.005)	(0.004)	(0.003)	(0.003)
Adj. p-value	.009	.547	.009	.547
Pre-reform mean	[.13]	[.05]	[.03]	[.03]
	Somatic spec. care			
	Total preg.	Normal preg.	High-risk preg.	Antenatal screening
	(1)	(2)	(3)	(4)
PANEL B: Specialist healthcare use				
- #	0.242	0.047*	-0.009	0.010
	(0.200)	(0.025)	(0.020)	(0.030)
Adj. p-value	.438	.244	.707	.707
Pre-reform mean	[.85]	[.07]	[.06]	[.08]
- Prob.	0.023***	0.018*	-0.000	0.006
	(0.006)	(0.011)	(0.006)	(0.015)
Adj. p-value	.001	.187	.986	.906
Pre-reform mean	[.06]	[.03]	[.02]	[.04]
N	278553	278553	278553	278553

Note: Panel A shows primary healthcare use and sickness absence, and Panel B shows specialist healthcare use. In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table 6: Fertility

	Age at first child (1)	**Child by 20** (2)	**Child by 25** (3)	**Child by 30** (4)	**Child by 35** (5)	**Child by 40** (6)
PANEL A: Total #						
ITT Post	0.077	-0.001	-0.002	-0.008	-0.008	0.000
	(0.050)	(0.003)	(0.010)	(0.013)	(0.016)	(0.017)
Adj. p-value	.859	.859	.859	.859	.955	.915
Pre-reform mean	[26.40]	[.12]	[.58]	[1.25]	[1.76]	[1.97]
PANEL B: Probability of						
ITT Post		0.000	0.000	-0.008*	-0.002	-0.002
		(0.003)	(0.005)	(0.005)	(0.003)	(0.004)
Adj. p-value		.832	.832	.352	.832	.832
Pre-reform mean		[.11]	[.41]	[.69]	[.82]	[.87]
N	278553	278553	278553	278553	278553	246150

Note: Panel A shows results from estimations on outcomes defined as the total number of incidents over the period examined, and Panel B shows results from estimations on outcomes defined as probabilities over the period examined. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. The number of observations is smaller in Column 6 because not all individuals in the sample have turned 40 yet. * p<0.1, ** p<0.05, *** p<0.01.

Table 7: Birth Outcomes for Second-Generation Children

	Birth weight (1)	**Low BW** (2)	**Very low BW** (3)	**High BW** (4)	**Gestation** (5)	**APGAR 5** (6)	**APGAR 5>7** (7)
ITT Post	-8.038	-0.001	-0.000	-0.009***	0.023	0.053***	0.002
	(5.484)	(0.002)	(0.001)	(0.003)	(0.021)	(0.012)	(0.001)
Adj. p-value	.335	.783	.783	.015	.380	.001	.380
Pre-reform mean	[3515]	[.06]	[.01]	[.20]	[39.40]	[9.27]	[.98]
N	546476	546476	546476	546476	522586	542485	542485

Note: The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. The number of observations are lower in Columns 5–7 because there are missing observations on the APGAR score and gestation in the medical birth registry. * p<0.1, ** p<0.05, *** p<0.01.

Table 8: Type of Birth for Second-Generation Children

	Type of birth			**Type of caesarean**		
	Spontaneous (1)	Induced (2)	Caesarean (3)	Elective (4)	Acute (5)	Unspecified (6)
ITT Post	0.003	-0.002	-0.003	-0.011	0.010	0.000
	(0.005)	(0.004)	(0.002)	(0.009)	(0.009)	(0.004)
Adj. p-value	.710	.801	.513	.513	.513	.916
Pre-reform mean	[.81]	[.13]	[.06]	[.38]	[.59]	[.04]
N	548540	548540	548540	78062	78062	78062

Note: The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table 9: Main Results: Mental Health and Risky Behavior

	GP consultations			ER visits	
	Psychological (1)	Injuries (2)	Social prob. (3)	Total (4)	Psychological (5)
PANEL A: Primary healthcare use					
- #	-0.009	-0.008	-0.027	0.028	0.014
	(0.082)	(0.014)	(0.056)	(0.030)	(0.010)
Adj. p-value	.911	.790	.790	.790	.790
Pre-reform mean	[5.09]	[.81]	[1.77]	[1.63]	[.21]
- Prob.	-0.007*	-0.006**	-0.007**	-0.005	0.002
	(0.003)	(0.003)	(0.004)	(0.003)	(0.002)
Adj. p-value	.090	.090	.090	.177	.368
Pre-reform mean	[.57]	[.32]	[.34]	[.59]	[.11]
	Somatic spec. care			**Psych. spec. care**	
	Acute (1)	Psych. acute (2)	Injuries (3)	Total (4)	Acute (5)
PANEL B: Specialist healthcare use					
- #	0.028	0.003	-0.014	-0.404**	-0.068
	(0.032)	(0.004)	(0.010)	(0.185)	(0.054)
Adj. p-value	.396	.396	.345	.150	.345
Pre-reform mean	[1.27]	[.02]	[.43]	[3.88]	[.25]
- Prob.	0.005	-0.000	-0.007**	-0.004	-0.002
	(0.004)	(0.001)	(0.003)	(0.003)	(0.001)
Adj. p-value	.378	.931	.171	.289	.289
Pre-reform mean	[.42]	[.01]	[.21]	[.11]	[.04]
N	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, and Panel B shows specialist healthcare use. In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses and comparison means in brackets. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

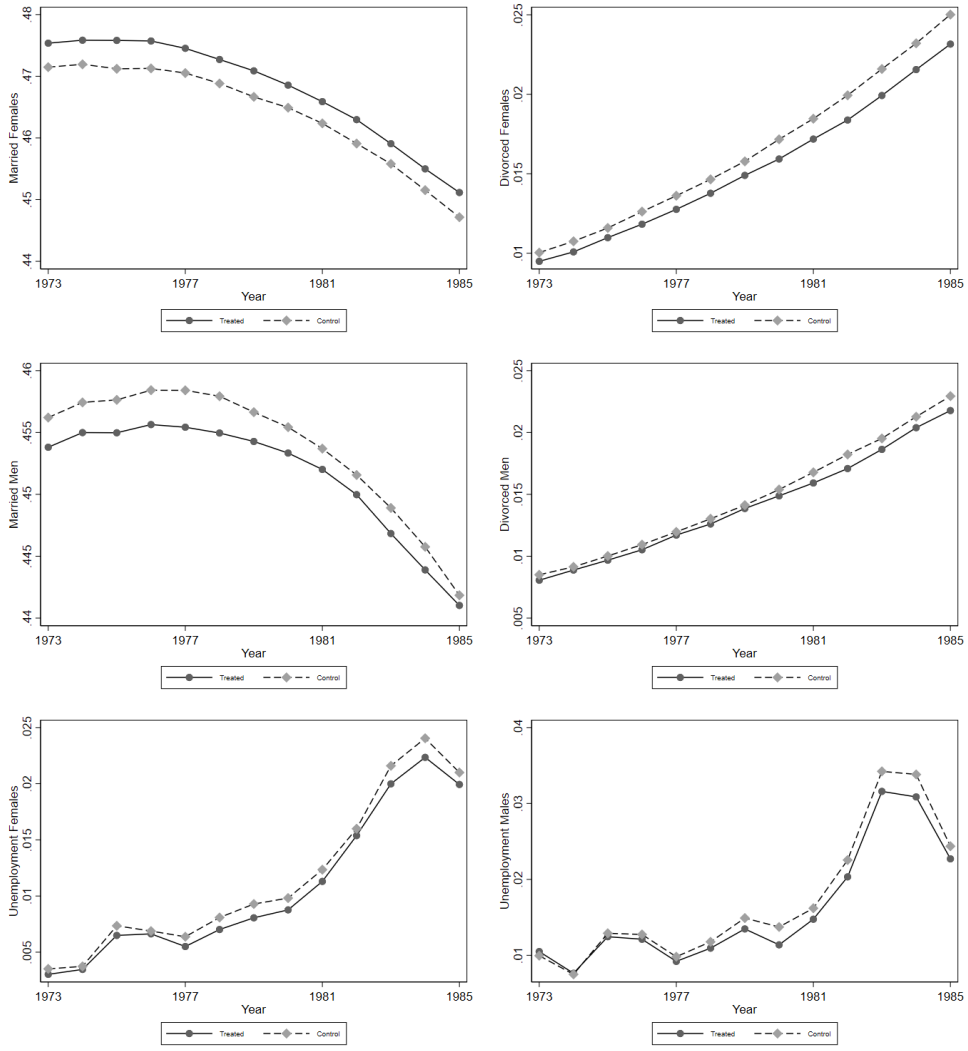
Table 10: Heterogeneity by Gender

	GP consultations			ER visits	
	Psychological	Injuries	Social prob.	Total	Psychological
	(1)	(2)	(3)	(4)	(5)
PANEL A: Primary healthcare use					
I. ITT (girl)	-0.000	0.005	-0.076	-0.005	0.022**
	(0.093)	(0.016)	(0.060)	(0.027)	(0.009)
II. ITT (boy)	-0.104	-0.028	-0.044	0.011	0.002
	(0.102)	(0.018)	(0.060)	(0.033)	(0.014)
p-value (I=II)	.472	.193	.657	.693	.154
	Somatic spec. care			**Psych. spec. care**	
	Acute	Psych. acute	Injuries	Total	Acute
	(1)	(2)	(3)	(4)	(5)
PANEL B: Specialist healthcare use					
I. ITT (girl)	0.055	0.002	-0.024**	-0.365*	-0.002
	(0.047)	(0.002)	(0.011)	(0.215)	(0.030)
II. ITT (boy)	-0.002	0.008	-0.009	-0.318	-0.094*
	(0.029)	(0.006)	(0.011)	(0.215)	(0.054)
p-value (I=II)	.242	.264	.337	.867	.105
N	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, and Panel B shows specialist healthcare use. In both panels, the first row shows the estimated ITT effects for girls, while the second row shows the estimated ITT effect for boys. The outcomes are defined as the total number of incidents over the period. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

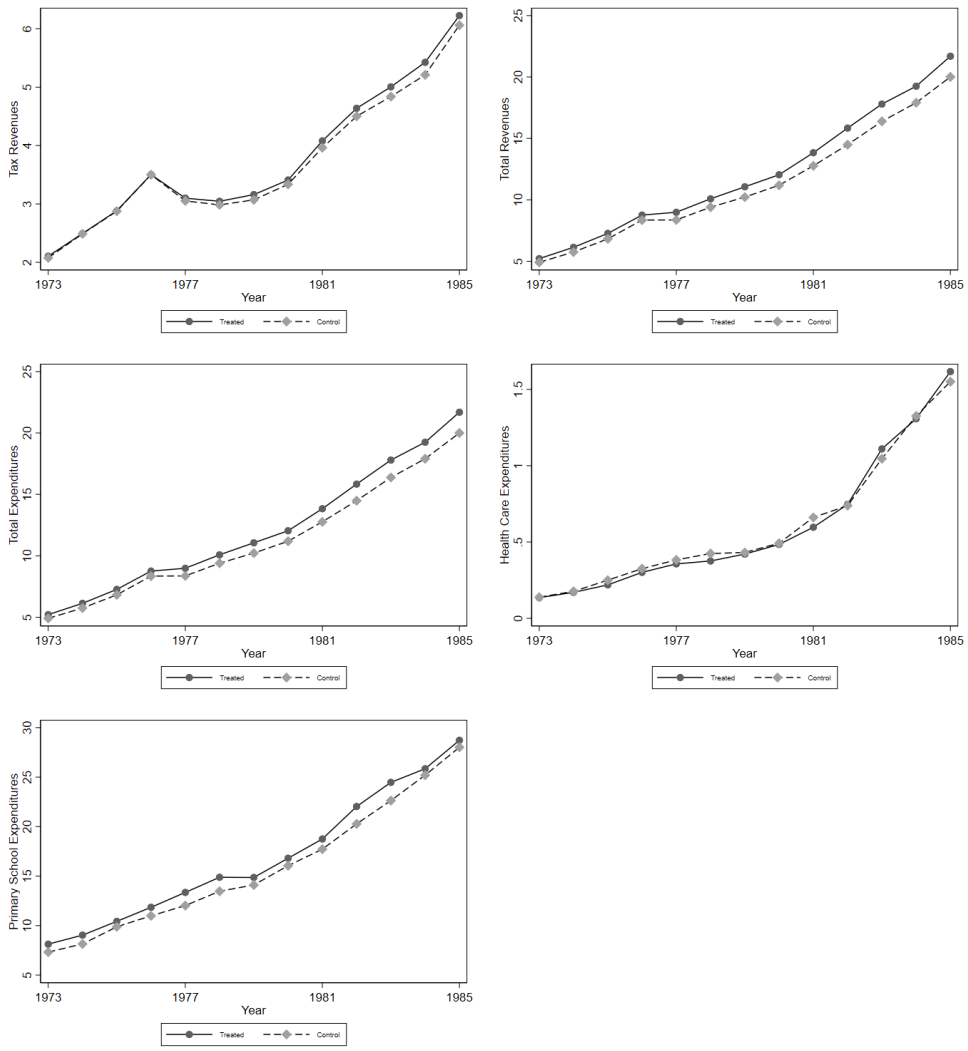
A Appendix

Figure A.1: Municipality Characteristics 1973–1985



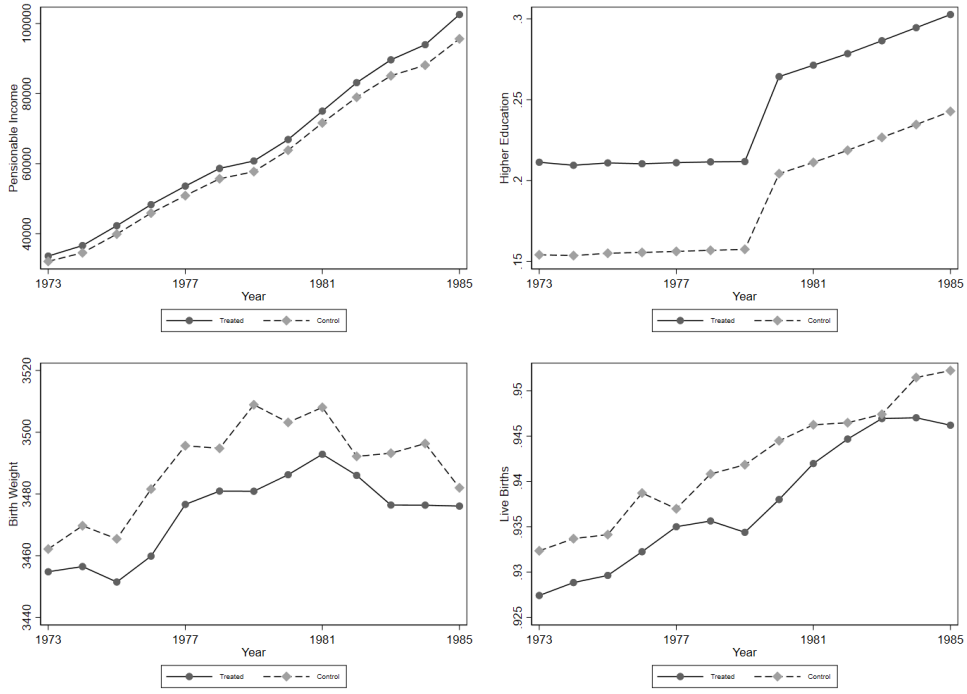
Note: These figures show trends in municipality characteristics in treated municipalities and control municipalities in 1973–1985. Data source: Norwegian Centre for Research Data (NSD).

Figure A.3: Municipality Characteristics 1973–1985 (Continued)



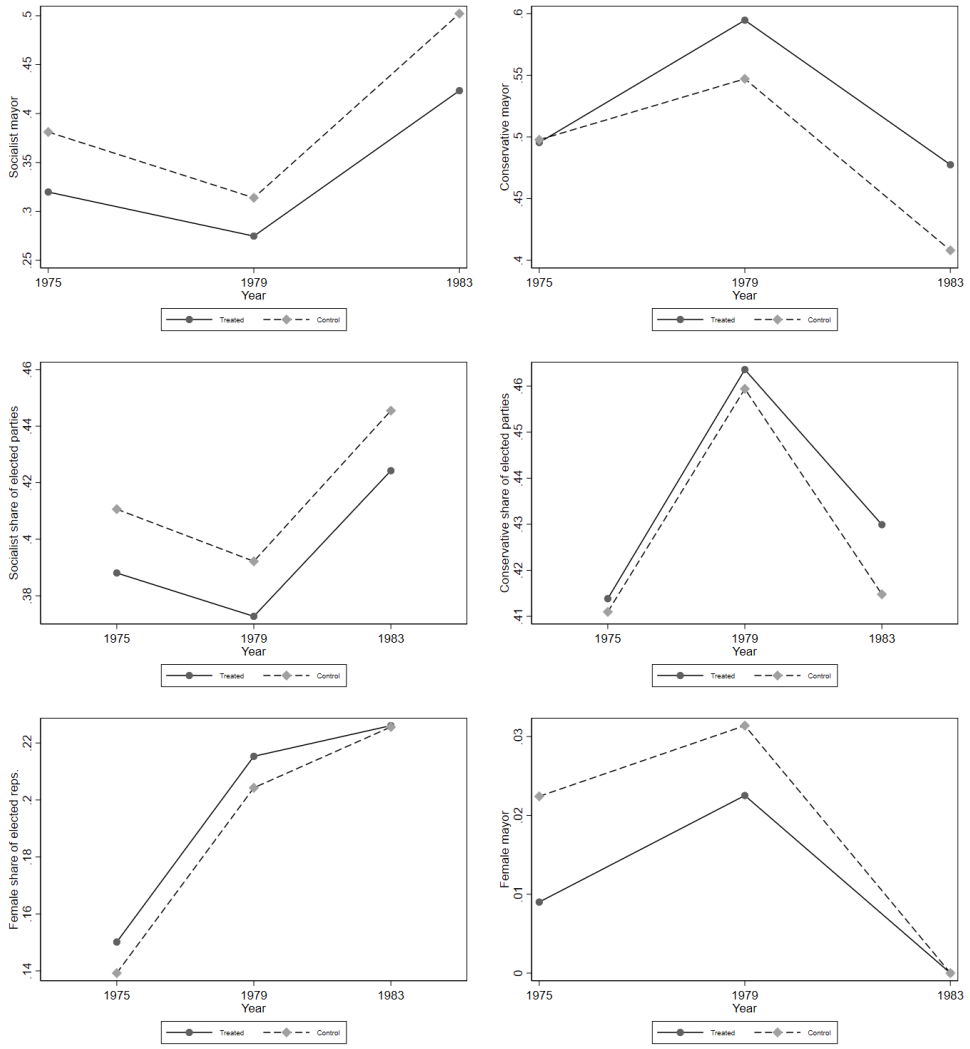
Note: These figures show trends in municipality characteristics in treated municipalities and control municipalities in 1973–1985. Data source: Norwegian Centre for Research Data (NSD).

Figure A.5: Municipality Characteristics 1973–1985 (Continued)



Note: These figures show trends in municipality characteristics in treated municipalities and control municipalities in 1973–1985. Data source: Norwegian administrative registers.

Figure A.7: Municipality Characteristics 1973–1985 (Continued)



Note: These figures show trends in municipality characteristics in treated municipalities and control municipalities in 1973–1985. Data source: Norwegian Centre for Research Data (NSD).

Table A1: Heterogeneity by Mothers' Education

		Related diagnoses				
	All	Metabolic	Cardiovascular	Musculoskeletal	Respiratory	Pregnancy
	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A: Primary healthcare use						
I. ITT (high school or more)	0.301	0.103	0.100	0.044	0.030	0.111
	(0.213)	(0.063)	(0.061)	(0.089)	(0.069)	(0.094)
II. ITT (less than high school)	0.170	-0.101*	-0.079	-0.054	-0.081	0.044
	(0.141)	(0.054)	(0.065)	(0.059)	(0.057)	(0.084)
p-value (I=II)	.615	.012	.040	.386	.197	.540
PANEL B: Specialist healthcare use						
I. ITT (high school or more)	0.163	0.015	-0.008	0.016	-0.005	0.022
	(0.180)	(0.027)	(0.015)	(0.036)	(0.014)	(0.031)
II. ITT (less than high school)	0.086	-0.002	-0.007	-0.008	-0.001	0.016
	(0.094)	(0.022)	(0.009)	(0.022)	(0.009)	(0.021)
p-value (I=II)	.697	.559	.949	.545	.856	.836
PANEL C: Sickness absence						
I. ITT (high school or more)	6.091***	0.167	0.114	0.973	0.192	-0.004
	(2.121)	(0.207)	(0.226)	(0.819)	(0.244)	(0.533)
II. ITT (less than high school)	0.826	-0.011	-0.236*	-0.303	0.043	0.450*
	(1.585)	(0.149)	(0.134)	(0.642)	(0.163)	(0.269)
p-value (I=II)	.055	.469	.203	.230	.597	.448
N	566911	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, Panel B shows specialist healthcare use, and Panel C shows sickness absence. In all three panels, the first row shows the estimated ITT effects for children of mothers who have higher education, while the second row shows the estimated ITT effect for children of mothers who have completed less than high school. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table A2: Heterogeneity by Mothers' Education

	GP consultations			ER visits	
	Psychological (1)	Injuries (2)	Social prob. (3)	Total (4)	Psychological (5)
PANEL A: Primary healthcare use					
I. ITT (high school or more)	0.054	0.037*	0.046	-0.060*	0.004
	(0.111)	(0.020)	(0.056)	(0.035)	(0.011)
II. ITT (less than high school)	-0.065	-0.024*	-0.089	0.033	0.016
	(0.076)	(0.014)	(0.056)	(0.029)	(0.012)
p-value (I=II)	.359	.021	.073	.050	.464
	Somatic spec. care			**Psych. spec. care**	
	Acute (1)	Psych. acute (2)	Injuries (3)	Total (4)	Acute (5)
PANEL B: Specialist healthcare use					
I. ITT (high school or more)	-0.013	0.001	-0.025*	-0.200	-0.062
	(0.056)	(0.003)	(0.013)	(0.283)	(0.062)
II. ITT (less than high school)	0.037	0.006	-0.011	-0.360*	-0.046
	(0.030)	(0.005)	(0.010)	(0.204)	(0.040)
p-value (I=II)	.367	.303	.378	.675	.838
N	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, and Panel B shows specialist healthcare use. In both panels, the first row shows the estimated ITT effects for children of mothers who have higher education, while the second row shows the estimated ITT effect for children of mothers who have completed less than high school. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A3: Heterogeneity by Mothers' Employment Status

		Related diagnoses				
	All	Metabolic	Cardiovascular	Musculoskeletal	Respiratory	Pregnancy
	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A: Primary healthcare use						
I. ITT (Mother employed)	0.166	-0.062	-0.029	-0.051	-0.075	0.087
	(0.157)	(0.053)	(0.061)	(0.059)	(0.052)	(0.085)
II. ITT (Mother not employed)	0.200	-0.047	-0.056	-0.016	-0.047	0.078
	(0.199)	(0.073)	(0.074)	(0.089)	(0.078)	(0.103)
p-value (I=II)	.893	.852	.741	.734	.739	.920
PANEL B: Specialist healthcare use						
I. ITT (Mother employed)	0.119	-0.015	-0.005	-0.008	0.003	0.018
	(0.105)	(0.023)	(0.009)	(0.020)	(0.008)	(0.024)
II. ITT (Mother not employed)	0.123	0.032	-0.011	0.006	-0.010	0.036
	(0.147)	(0.029)	(0.014)	(0.039)	(0.014)	(0.028)
p-value (I=II)	.982	.134	.726	.737	.412	.486
PANEL C: Sickness absence						
I. ITT (Mother employed)	3.172*	-0.034	-0.125	0.287	0.112	0.697**
	(1.755)	(0.163)	(0.133)	(0.713)	(0.176)	(0.295)
II. ITT (Mother not employed)	-1.231	0.089	-0.219	-0.737	-0.026	-0.047
	(2.079)	(0.209)	(0.209)	(0.897)	(0.233)	(0.367)
p-value (I=II)	.102	.648	.706	.367	.626	.095
N	566911	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, Panel B shows specialist healthcare use, and Panel C shows sickness absence. In all three panels, the first row shows the estimated ITT effects for children of mothers who are in employment, while the second row shows the estimated ITT effect for children of mothers who are not in employment. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table A4: Heterogeneity by Mothers' Employment Status

	GP consultations			ER visits	
	Psychological (1)	Injuries (2)	Social prob. (3)	Total (4)	Psychological (5)
PANEL A: Primary healthcare use					
I. ITT (Mother employed)	-0.075	-0.014	-0.069	-0.008	0.005
	(0.078)	(0.013)	(0.053)	(0.023)	(0.009)
II. ITT (Mother not employed)	-0.006	-0.007	-0.042	0.025	0.027
	(0.129)	(0.021)	(0.072)	(0.051)	(0.018)
p-value (I=II)	.656	.781	.724	.576	.175
	Somatic spec. care			**Psych. spec. care**	
	Acute (1)	Psych. acute (2)	Injuries (3)	Total (4)	Acute (5)
PANEL B: Specialist healthcare use					
I. ITT (Mother employed)	0.022	0.003	-0.026***	-0.421**	-0.104**
	(0.028)	(0.003)	(0.010)	(0.190)	(0.040)
II. ITT (Mother not employed)	0.032	0.008	0.003	-0.183	0.061*
	(0.053)	(0.007)	(0.013)	(0.243)	(0.033)
p-value (I=II)	.832	.451	.050	.391	.000
N	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, and Panel B shows specialist healthcare use. In both panels, the first row shows the estimated ITT effects for children of mothers who are in employment, while the second row shows the estimated ITT effect for children of mothers who are not in employment. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table A5: Heterogeneity by Household Income

		Related diagnoses				
	All	Metabolic	Cardiovascular	Musculoskeletal	Respiratory	Pregnancy
	(1)	(2)	(3)	(4)	(5)	(6)
PANEL A: Primary healthcare use						
I. ITT (HH income Q4)	0.134	0.026	-0.027	-0.046	-0.005	0.131
	(0.245)	(0.067)	(0.068)	(0.100)	(0.071)	(0.082)
II. ITT (HH income Q1)	-0.013	-0.094	0.001	-0.031	-0.108	0.038
	(0.262)	(0.093)	(0.105)	(0.112)	(0.101)	(0.132)
p-value (I=II)	.636	.263	.814	.923	.376	.496
PANEL B: Specialist healthcare use						
I. ITT (HH income Q4)	0.300**	0.024	0.012	-0.010	-0.033**	0.049*
	(0.144)	(0.026)	(0.015)	(0.030)	(0.014)	(0.028)
II. ITT (HH income Q1)	0.307*	-0.026	0.026*	0.005	0.019	0.038
	(0.181)	(0.034)	(0.015)	(0.049)	(0.016)	(0.024)
p-value (I=II)	.975	.214	.538	.779	.017	.722
PANEL C: Sickness absence						
I. ITT (HH income Q4)	4.163*	0.467**	0.095	-0.178	0.178	-0.045
	(2.416)	(0.221)	(0.224)	(0.831)	(0.266)	(0.437)
II. ITT (HH income Q1)	1.499	-0.260	-0.150	0.260	-0.030	0.884**
	(2.927)	(0.263)	(0.212)	(1.197)	(0.278)	(0.401)
p-value (I=II)	.449	.031	.395	.756	.572	.096
N	566911	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, Panel B shows specialist healthcare use, and Panel C shows sickness absence. In all three panels, the first row shows the estimated ITT effects for children from households with income in the highest quartile, while the second row shows the estimated ITT effect for children from households with income in the lowest quartile. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table A6: Heterogeneity by Household Income

	GP consultations			ER visits	
	Psychological (1)	Injuries (2)	Social prob. (3)	Total (4)	Psychological (5)
PANEL A: Primary healthcare use					
I. ITT (HH income Q4)	-0.133	-0.007	-0.044	0.045	0.024
	(0.131)	(0.019)	(0.060)	(0.035)	(0.018)
II. ITT (HH income Q1)	-0.082	-0.048*	-0.062	-0.039	0.007
	(0.125)	(0.026)	(0.100)	(0.048)	(0.018)
p-value (I=II)	.780	.212	.869	.195	.536
	Somatic spec. care			**Psych. spec. care**	
	Acute (1)	Psych. acute (2)	Injuries (3)	Total (4)	Acute (5)
PANEL B: Specialist healthcare use					
I. ITT (HH income Q4)	0.015	0.014*	-0.016	-0.500	-0.107*
	(0.045)	(0.008)	(0.013)	(0.316)	(0.064)
II. ITT (HH income Q1)	0.081	-0.002	-0.014	0.017	0.013
	(0.051)	(0.004)	(0.015)	(0.317)	(0.059)
p-value (I=II)	.307	.113	.896	.236	.172
N	566911	566911	566911	566911	566911

Note: Panel A shows primary healthcare use, and Panel B shows specialist healthcare use. In both panels, the first row shows the estimated ITT effects for children from households with income in the highest quartile, while the second row shows the estimated ITT effect for children from households with income in the lowest quartile. The outcomes are defined as the total number of incidents over the period. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * p<0.1, ** p<0.05, *** p<0.01.

Table A7: Robustness

	Baseline (1)	Excl. movers (2)	Excl. non-married (3)	No small munics. (4)	No large cities (5)	One post cohort (6)	No phase-in cohort (7)	Continuous treatment variable (8)
GP consultations								
- #	0.249	0.262*	0.234	0.252	0.315*	0.196	0.250	0.129
	(0.154)	(0.153)	(0.161)	(0.155)	(0.171)	(0.136)	(0.155)	(0.544)
- Prob.	0.001	0.000	0.001	0.001	0.001	0.002	0.001	-0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.002)	(0.001)	(0.001)	(0.005)
Sickness absence								
- #	2.170	1.844	1.686	1.994	2.031	1.519	2.246	1.790
	(1.510)	(1.464)	(1.662)	(1.511)	(1.633)	(1.137)	(1.496)	(5.697)
- Prob.	-0.002	-0.004	-0.002	-0.003	-0.002	0.000	-0.002	-0.006
	(0.003)	(0.003)	(0.004)	(0.003)	(0.004)	(0.003)	(0.003)	(0.013)
Somatic spec. care								
- #	0.207**	0.174*	0.200**	0.208**	0.209*	0.191**	0.204**	0.306
	(0.096)	(0.095)	(0.100)	(0.096)	(0.115)	(0.081)	(0.096)	(0.404)
- Prob.	0.007**	0.006*	0.006*	0.007**	0.005	0.006**	0.007**	0.021*
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)	(0.003)	(0.003)	(0.012)
Psych. spec. care								
- #	-0.404**	-0.404**	-0.487***	-0.412**	-0.308	-0.264*	-0.397**	-0.903
	(0.185)	(0.178)	(0.182)	(0.186)	(0.205)	(0.144)	(0.186)	(0.585)
- Prob.	-0.004	-0.004*	-0.005*	-0.004	-0.003	-0.001	-0.004	-0.019**
	(0.003)	(0.002)	(0.003)	(0.003)	(0.002)	(0.002)	(0.003)	(0.008)
N	566911	533403	516161	564980	471270	566911	378161	566911

Note: In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. Col. 1 shows the baseline estimates. In Col. 2, individuals who move between treatment and control municipalities in the post-reform period are excluded. In Col. 3, children of non-married mothers are excluded. In Col. 4, we exclude observations from municipalities with under 1000 inhabitants, while in Col. 5, observations from the three largest cities are excluded. In Col. 6–8, we explore different treatment definitions. The outcome variables are defined in Table A11 and Table A12 in the Appendix. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A8: Robustness (Continued)

			Trend versions		
	Baseline (1)	No ind. controls (2)	Linear estimated pre-trend (3)	Quadratic estimated pre-trend (4)	Linear direct trend (5)
GP consultations					
- #	0.249	0.177	0.202	0.234	0.228
	(0.154)	(0.187)	(0.165)	(0.158)	(0.159)
- Prob.	0.001	0.001	0.001	0.001	0.001
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Sickness absence					
- #	2.170	1.687	2.288	2.215	1.406
	(1.510)	(1.590)	(1.523)	(1.518)	(1.454)
- Prob.	-0.002	-0.003	-0.003	-0.003	-0.004
	(0.003)	(0.004)	(0.003)	(0.003)	(0.003)
Somatic spec. care					
- #	0.207**	0.190*	0.191**	0.201**	0.179*
	(0.096)	(0.101)	(0.096)	(0.096)	(0.098)
- Prob.	0.007**	0.006*	0.006*	0.007**	0.004
	(0.003)	(0.003)	(0.003)	(0.003)	(0.004)
Psych. spec. care					
- #	-0.404**	-0.451**	-0.374**	-0.385**	-0.426**
	(0.185)	(0.199)	(0.187)	(0.184)	(0.186)
- Prob.	-0.004	-0.005	-0.003	-0.004	-0.004
	(0.003)	(0.003)	(0.003)	(0.003)	(0.003)
N	566911	566914	563555	563555	566911

Note: In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. Col. 1 shows the baseline estimates. In Col. 2, we estimate Eq. 1 without ind. control variables. In Col. 3–5, municipality-specific time trends are included. The outcome variables are defined in Table A11 and Table A12 in the Appendix. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A9: Robustness: Placebo Reform

	GP consultations (1)	**Sickness absence** (2)	**Som. spec. care** (3)	**Psych. spec. care** (4)
PANEL A: Total #				
ITT 1968	0.229	-0.455	-0.120	-0.028
	(0.248)	(2.413)	(0.168)	(0.277)
ITT 1969	0.380	-1.945	0.086	-0.174
	(0.283)	(2.174)	(0.147)	(0.274)
ITT Phase-in	0.349	0.079	0.165	-0.196
	(0.225)	(1.856)	(0.134)	(0.204)
ITT Post	0.454**	1.362	0.196	-0.472**
	(0.210)	(2.103)	(0.132)	(0.237)
Pre-reform mean	[21.7]	[146]	[6.99]	[3.88]
PANEL B: Probability of				
ITT 1968	-0.002	0.005	0.004	0.003
	(0.002)	(0.005)	(0.005)	(0.003)
ITT 1969	-0.003	-0.003	0.004	-0.003
	(0.002)	(0.005)	(0.005)	(0.003)
ITT Phase-in	0.000	0.004	0.007	0.001
	(0.002)	(0.004)	(0.005)	(0.003)
ITT Post	-0.001	-0.001	0.010*	-0.004
	(0.002)	(0.004)	(0.005)	(0.003)
Pre-reform mean	[.96]	[.542]	[.721]	[.112]
N	566911	566911	566911	566911

Note: Panel A shows results from estimations on outcomes defined as the total number of incidents over the period examined, while panel B shows results from estimations on outcomes defined as probabilities over the period examined. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality fixed effects are included in all regressions. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A10: Robustness: Family Fixed Effects

	Full sample Baseline spec. (1)	Sibling sample Baseline spec. (2)	Sibling sample Family FE (3)
GP consultations			
- #	0.249	0.333	0.312
	(0.154)	(0.263)	(0.365)
- Prob.	0.001	0.001	0.001
	(0.001)	(0.002)	(0.004)
Sickness absence			
- #	2.170	0.341	3.223
	(1.510)	(2.494)	(3.917)
- Prob.	-0.002	-0.007	0.001
	(0.003)	(0.006)	(0.009)
Somatic spec. care			
- #	0.207**	0.208	0.225
	(0.096)	(0.182)	(0.300)
- Prob.	0.007**	0.008	0.009
	(0.003)	(0.006)	(0.010)
Psych. spec. care			
- #	-0.404**	-0.575**	-0.537
	(0.185)	(0.275)	(0.409)
- Prob.	-0.004	-0.008*	-0.007
	(0.003)	(0.004)	(0.006)
N	566911	192690	192690

Note: In rows named "#" the outcomes are defined as the total number of incidents over the period examined, while in rows named "Prob." the outcomes are defined as the probability of an incident occurring at all within the period examined. The outcome variables are defined in Table A11 and Table A12 in the Appendix. Standard errors clustered at the level of municipality of residence in 1976 are reported in parentheses. Controls, as listed in Table 1, as well as cohort and municipality/family fixed effects are included in all regressions. * $p<0.1$, ** $p<0.05$, *** $p<0.01$.

Table A11: Definition of Primary Healthcare and Sickness Absence Outcomes

Outcome	Definition/diagnosis group	Source
Primary healthcare		
Total	Total number of GP consultations	ICPC-2
General	A	ICPC-2
Metabolic	T	ICPC-2
Cardiovascular	K	ICPC-2
Musculoskeletal	L	ICPC-2
Respiratory	R	ICPC-2
Pregnancy	W	ICPC-2
Psychological	P	ICPC-2
Sickness absence		
Total	Total number of sickness absence days	ICPC-2
General	A	ICPC-2
Metabolic	T	ICPC-2
Cardiovascular	K	ICPC-2
Musculoskeletal	L	ICPC-2
Respiratory	R	ICPC-2
Pregnancy	W	ICPC-2
Psychological	P	ICPC-2
GP consultations related to pregnancy		
Normal pregnancy	W01, W02, W03, W05, W17, W18, W19, W21, W27, W28, W29, W78, W781, W82, W90, W94, W95	ICPC-2
High-risk pregnancy	W70, W71, W72, W73, W75, W76, W80, W81, W84, W85, W91, W92, W93, W96, W99	ICPC-2
Contraceptives	W10, W11, W12, W13, W14, W15, W79, W83	ICPC-2
Sickness absence related to pregnancy		
Normal pregnancy	W01, W02, W03, W05, W17, W18, W19, W21, W27, W28, W29, W78, W781, W82, W90, W94, W95	ICPC-2
High-risk pregnancy	W70, W71, W72, W73, W75, W76, W80, W81, W84, W85, W91, W92, W93, W96, W99	ICPC-2
Risky behavior		
GP cons rel. to injuries	A80, A81, A82, A84, A85, A86, A87, A88, A89, B76, B77, D79, D80, F75, F76, F79, H76, H77, H78, H79, L72, L73, L74, L75, L76, L77, L78, L79, L80, L81, L96, N79, N80, N81, S12, S13, S14, S15, S16, S17, S18, S19, U80, W75, X82, Y80	ICPC-2
ER visits	Total number of ER visits	ICPC-2

Note: This table gives an overview of the primary healthcare use and sickness absence outcomes, which are all based on the ICPC-2 codes.

Table A12: Definition of Specialist Healthcare and Birth Outcomes

Outcome	Definition/diagnosis group	Source
Specialist healthcare		
Total	Total number of somatic visits	ICD-10
General	Z	ICD-10
Metabolic	E	ICD-10
Cardiovascular	I	ICD-10
Musculoskeletal	M	ICD-10
Respiratory	J	ICD-10
Pregnancy	O	ICD-10
Psychiatric	Total number of psychiatric visits	ICD-10
Hospital visits related to pregnancy		
Total	Z32, Z33, Z34, Z35, Z36	ICD-10
Normal pregnancy	Z34	ICD-10
High-risk pregnancy	Z35	ICD-10
Antenatal screening	Z36	ICD-10
Birth outcomes next generation		
Birth weight	Continuous in gram	Medical Birth Registry
Low birth weight	Birth weight below 2500 gram	Medical Birth Registry
Very low birth weight	Birth weight blow 1500 gram	Medical Birth Registry
High birth weight	Birth weight above 4000 gram	Medical Birth Registry
APGAR 5	Continuous (1–10)	Medical Birth Registry
APGAR 5 >7	Dummy= 1 if APGAR 5 $>$ 7	Medical Birth Registry
Gestation	Continuous, measured in weeks	Medical Birth Registry
Type of birth		
Spontaneous	Dummy 1/0	Medical Birth Registry
Induced	Dummy 1/0	Medical Birth Registry
Caesarean	Dummy 1/0	Medical Birth Registry
Type of caesarean		
Elective	Dummy 1/0	Medical Birth Registry
Acute	Dummy 1/0	Medical Birth Registry
Unspecified	Dummy 1/0	Medical Birth Registry
Risky behavior		
Som. spec. care, injury	S	ICD-10

Note: This table gives an overview of the specialist healthcare outcomes based on ICD-10 codes and birth outcomes based on variables from the medical birth registry.

Chapter 4:

Effects of Air Pollution on Health and Productivity

Effects of Air Pollution on Health and Productivity*

Anne-Lise Breivik† Tor Helge Holmås‡ Julie Riise§

Abstract

This paper examines the relationship between short-term air pollution increases and indicators of health and worker productivity. Estimating two-way fixed effects models using geographical and time variation in exposure to particulate matter (PM_{10}) and nitrogen dioxide (NO_2), we find that the number of GP consultations, certified sickness absences, and hospital visits increases in periods with high(er) pollution levels. There is substantial heterogeneity in this relationship. We find some support for previous results showing that pollution affects vulnerable groups like children and elderly negatively. Importantly, however, the largest effects are on school-age children and the working-age population.

Keywords: Air pollution, health, labor productivity

JEL Codes: Q53, I1, J24

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1 Introduction

According to the European Environmental Agency, air pollution has substantial economic impacts: it increases the use of health care services, causes adverse health outcomes, and shortens people's lives (EEA, 2015). The economics literature on the health effects of air pollution supports this notion and finds that exposure to increased pollution levels can lead to adverse health outcomes (Neidell, 2004; Currie and Neidell, 2005; Currie et al., 2009; Currie and Walker, 2011; Schlenker and Walker, 2016). In addition to causing adverse health outcomes directly, an indirect effect of exposure to pollution is increased sickness absence from work. Such an effect is related to a literature that in recent years has documented that pollution significantly lowers labor productivity in different contexts (Graff Zivin and Neidell, 2012; Arceo and Oliva, 2015; Chang et al., 2016, 2019), but, to the best of our knowledge, this issue has not been studied previously.

The main research question in this paper is whether, and to what extent, short-term air pollution increases from relatively low levels adversely affect health outcomes and productivity in the general population. To answer this question, we explore the relationship between short-term air pollution increases and the use of healthcare services and worker productivity in the period 2011–2014 by examining three main outcomes: general practitioner (GP) consultations, certified sickness absences from work, and acute hospital visits. For all three main outcomes, we examine related diagnoses. Estimating two-way fixed effects models, using geographical and time variation in exposure to particulate matter (PM_{10}) and nitrogen dioxide (NO_2), we find that short-term air pollution increases from relatively low levels adversely affect health outcomes — also for the working age population, in contrast to most other studies, which have not had access to data covering this part of the population. The adverse effects on health outcomes in turn lead to negative impacts on worker productivity, measured as sickness absences from work.

Measuring and assessing the impacts of air pollution is a demanding exercise for at least two reasons. First, air pollution is not randomly assigned to individuals. Individuals with preferences for clean air may self-select into neighborhoods with better air quality, and individuals who live in polluted areas may have worse health for reasons unrelated to pollution, such as socioeconomic status (Chay and Greenstone, 2005). The individual preferences for clean air may also co-vary with unobservable determinants of health, such as tastes, interests, and practices including smoking and exercising. Alternatively, emission sources tend to be located in urbanized areas, and individuals living there might have higher levels of education and better health. Second, it is difficult to measure whether individuals respond to higher pollution levels with increasing avoidance behavior. Being able to disentangle these factors is especially important for external validity. As a response to these challenges, recent research has estimated

the causal effects of pollution on health using natural experiments and econometric methods, such as instrumental variables or fixed effects models (Chay and Greenstone, 2003a,b; Neidell, 2004; Currie and Neidell, 2005; Currie et al., 2009).

To address these challenges, we apply two-way fixed effects models in which we exploit time and geographical variation in pollution levels. An advantage of our setting is that we explore short-term pollution increases at the week level. Furthermore, as our sample covers the whole population, we can split the sample into different age groups in order to investigate avoidance behavior. The most vulnerable age groups, namely, children aged 0–5 and the elderly are the ones that can most easily avoid pollution in the short term, as they do not have to go to school or work and typically spend less time outdoors. We can also explore the non-random assignment of pollution by splitting the sample by socioeconomic status.

Two aspects of the biological effects of exposure to air pollution further complicate the estimation of health effects of ambient air pollution (Graff Zivin and Neidell, 2013). First, while some pollutants have linear effects, others can have non-linear effects or even contain threshold levels where adverse health effects only appear after the pollution reaches a certain level. Second, there may be heterogeneity in responses to given levels of pollution for different groups of the population (apart from the differences in exposure and avoidance behavior discussed above). We address these key aspects of pollution exposure by comparing results from different specifications of the regression model.

The pollutants examined in this paper are PM_{10} and NO_2. PM_{10} consists of a mixture of solid and liquid particles suspended in the air. Identified effects of particulate matter include difficulty breathing, impaired lung function, respiratory and cardiovascular diseases, such as asthma, stroke, and heart attack, and increased mortality. NO_2 is a gas that is primarily emitted into the air from the burning of fuels. It typically forms from emissions from cars, trucks, and transportation vehicles, power plants, and off-road equipment. Inhalation of air with high concentrations of NO_2 can irritate airways in the respiratory system, lower resistance to respiratory infections, and have adverse respiratory impacts, including airway inflammation and increased respiratory symptoms.

In the main specification, we apply dummy variables indicating whether PM_{10} and NO_2 levels in a week are above the annual mean concentration levels recommended by the international air quality guidelines. The average pollution levels across weeks are below these annual thresholds. Given that we apply these thresholds at the week level, we are studying the effects of increased pollution levels at relatively low levels. Recommended daily threshold levels are much higher. We first examine GP consultations, sickness absence, and acute hospital visits in total. Second, we follow the established knowledge on the health effects of air pollution and examine GP consultations, sickness absence, and hospital visits related to respiratory and cardiovascular diagnoses separately. We find positive and statistically significant relationships

between PM_{10} and GP consultations and between PM_{10} and certified sickness absences, driven by diagnoses related to the respiratory system. We also find a positive and statistically significant relationship between NO_2 and acute hospital visits, again driven by diagnoses related to the respiratory system.

In the second part of the analysis, we examine whether air pollution exposure has heterogeneous effects across different groups of the population. Specifically, we examine heterogeneity by age, gender, education level, and income. The results show that the working-age population and the middle-to-high-income groups are the main drivers of the estimated effects.

The analysis continues by examining whether the relationship between air pollution and health is linear or non-linear, as some studies suggest a linear relationship between air pollution and adverse health outcomes, while others suggest that there is a threshold level below which no adverse effects are found (Stoeger et al., 2006). In one specification, we therefore include continuous pollution variables. When we use this approach, the results are similar to the main results when it comes to PM_{10}, but the point estimates are smaller. The treatment is also smaller, so these effects are as expected. When it comes to NO_2, there are no effects when we use the continuous pollution variable. This may indicate that there are effects of NO_2 only at the higher end of its distribution. In a second specification, we include continuous pollution variables and second-order polynomials of the pollution variables. Using this approach, we find evidence of nonlinearity in the relationship between NO_2 concentrations and the use of healthcare services.

A number of decisions have been made with respect to the empirical specification, and in order to check that they are not decisive for the results, we apply a number of robustness checks. Different cut-offs for the assignment of pollution to individuals, no GP-specific time trends, and quadratic GP-specific time trends are evaluated. Furthermore, we trim the dataset to test whether outliers of the pollution distributions drive the results. The baseline results are robust to these checks.

Finally, to address remaining potential threats to identification, we run a number of placebo analyses. The medical and epidemiological research on the effects of PM_{10} and NO_2, and air pollution in general, is not yet very mature. Therefore, it is difficult to identify diagnoses or health conditions that we can be certain are not affected by air pollution. We thus apply placebo diagnoses that we are fairly confident are not associated with air pollution, such as diagnoses related to the genitourinary system and male genital diagnoses.[1] Except for a negative effect of PM_{10} on GP consultations related to male genital diagnoses, we find no effects on these outcomes.

[1] One potential placebo test would be to use planned hospital stays, which in theory should not be affected by random pollution levels, instead of acute stays, which are assumed to be related to contemporaneous air pollution. However, planned hospital stays are often moved if incidents that are more serious need priority. Planned procedures could also be pushed forward if they become more urgent due to high pollution.

We contribute to the literature in several ways. First, our empirical approach allows us to estimate the contemporaneous effects of air pollution on the health and productivity of the general population based on variation in local air pollution. By doing so, we take advantage of highly detailed Norwegian population register data and examine the health responses of the entire population, not only the most fragile groups. Second, because we have data on both GP consultations and hospital visits, we capture both less serious incidences (leading to a GP consultation) and very serious incidences (leading to unplanned hospitalization). Third, since we have universal access to healthcare in Norway, GP consultations as well as hospitalization is practically free of charge. Any heterogeneity in hospitalization should therefore not be driven by access to health services or the ability to pay for health services in our study. However, this could pose a potential problem for studies conducted, for example, in the United States, where the retired population has access to medical services through Medicare, while not all individuals of working age are covered by health insurance (Schlenker and Walker, 2016). Finally, our framework allows us to control for various potential confounders with detailed data on observable characteristics of the individuals in our sample, such as socioeconomic status, in addition to detailed weather data on precipitation, wind, and temperature.

The paper proceeds as follows. Section 2 provides information on the effects of pollution and a review of previous literature on the effects of air pollution on health and productivity. Section 3 describes the data applied. Section 4 outlines the empirical approach. Section 5 presents the results, and Section 6 presents the robustness checks, before we conclude in Section 7.

2 Air Pollution, Health, and Productivity

2.1 Previous Literature

Previous research from a number of fields has documented a relationship between exposure to air pollution and a range of health outcomes, including respiratory illnesses, asthma, cardiovascular illnesses, stroke, and mortality (Pope-III and Dockery, 2006; Brook, 2008; Chay and Greenstone, 2003a,b; Neidell, 2004; Currie and Neidell, 2005; Knittel et al., 2016; Schlenker and Walker, 2016; Bauernschuster et al., 2017; Jans et al., 2018). In economics, the majority of studies focus on infant and child outcomes (see e.g., Neidell (2004), who examines the effect of air pollution levels on child hospitalizations for asthma using naturally occurring seasonal variations in pollution within zip codes in California between 1992 and 1998 to ambient pollution levels, or Jans et al. (2018), who look at the effect of ambient air pollution on inpatient and outpatient hospital visits for children aged 0–18 years in Sweden).

In contrast to these studies, we examine the short-term effects of air pollution increases on the population in all age groups. Only a small number of studies have investigated the short-term impacts of air pollution on the health of a general population. One example is

Schlenker and Walker (2016). They show that daily variation in ground-level airport congestion significantly increases both exposure to carbon monoxide (CO) and hospitalization rates for asthma, respiratory, and heart-related problems. Another example is Bauernschuster et al. (2017), who look at short-term hikes in pollution levels using daily variation in public transport strikes in German cities and find significantly increased particle matter emissions and hospital admissions related to respiratory problems among young children on days with strikes. These studies, however, only have data on hospital admissions and therefore most likely only capture the most severe illnesses induced by exposure to air pollution.

Although there is an established relationship between air pollution and health, the exact biological reactions to most air pollutants are not yet fully understood. In the next sections, we briefly sum up the current state of knowledge related to the two pollutants we study, PM_{10} and NO_2.[2]

2.2 Particulate Matter

Of the two pollutants examined in this paper, PM is believed to be most harmful to human health (Brook, 2008). It consists of a mixture of solid and liquid particles suspended in the air. The particles vary in size (common classifications are PM_{10}, $PM_{2.5}$, and PM_1, referring to the diameter size of the particles), composition, and origin. The particles we study in this paper, PM_{10}, are derived primarily from suspension or resuspension of dust, soil, or other earth crust materials from roads, farming, volcanoes, and windstorms. Sea salts, pollen, molds, spores, and other plant parts are also commonly found in PM_{10} (Pope-III and Dockery, 2006).[3] Although PM_{10} is not directly emitted from traffic, traffic is an important reason that PM_{10} is resuspended in the air (WHO, 2003). PM_{10} can penetrate the thoracic region of the respiratory system and cause physical problems in the mouth, nose, and trachea. Moreover, PM_{10} can induce an inflammatory response in the lungs or activate the autonomic nervous system through sensory receptors on the alveolar surface, causing effects on the cardiovascular system (Miller et al., 2012). Identified effects of particulate matter thus include difficulty breathing, impaired lung function, respiratory and cardiovascular diseases, such as asthma, stroke, and heart attack, and increased mortality. International air quality guidelines recommend that the annual mean concentrations of PM_{10} should not exceed 20 $\mu g/m^3$, while the 24-hour mean is set at 50 $\mu g/m^3$ (WHO, 2006).[4]

[2] We study these particular pollutants because they have been measured for a relatively long period of time in several places in Norway. This study extends the existing knowledge by examining how (at what levels) these pollutants work as well as some aspects of their societal impact (demand and use of health services as well as effects on labor market productivity).

[3] In contrast, the finer particles ($PM_{2.5}$ and PM_1) are derived primarily from direct emissions from combustion processes, such as vehicle combustion of gasoline and diesel, wood burning, and industrial processes. Relative to the larger particles, $PM_{2.5}$ can be inhaled deeper into the lungs and remains suspended for longer periods (Pope-III and Dockery, 2006).

[4] Norwegian guidelines follow the international guidelines on both PM_{10} and NO_2.

2.3 Nitrogen Dioxide

NO_2 is a gas that is primarily emitted into the air from burning fuel. It typically forms from emissions from cars, trucks, and transportation vehicles, power plants, and off-road equipment. It is difficult to disentangle the health effects from NO_2 emissions from those related to PM_{10} emissions since NO_2 concentrations are often highly correlated with levels of other ambient pollutants, either being suspended by the same sources or related through complex atmospheric reactions. In the data used in this paper, the two pollutants are correlated, with a correlation coefficient of 0.46. Another challenge related to estimating the health effects of NO_2 is the fact that NO_2 is typically very locally concentrated; it does not travel over distances to the extent that for example particular matter does. The majority of existing studies only have data for relatively large catchment areas, while we use a relatively short radius of 5 km. Epidemiological evidence links NO_2 exposure to irritation in the lungs, which lowers resistance to respiratory infections, in addition to adverse respiratory impacts, including airway inflammation and increased respiratory symptoms (EPA, 2016). International air quality guidelines recommend that the annual mean concentrations of NO_2 should not exceed 40 $\mu g/m^3$, while the 24-hour mean is set at 200 $\mu g/m^3$ (WHO, 2006). For both NO_2 and PM_{10}, we use the annual mean thresholds in the main estimation specification.

2.4 Heterogeneity

2.4.1 Age

The negative effects of air pollution affect different groups of the population differentially. Susceptible groups, such as infants, children, the elderly, and individuals with pre-existing health conditions, are more vulnerable to exposure to air pollution compared to healthy adults.

Childhood is a critical time for the formation of important body systems. At the same time, evidence shows that health throughout the human lifecycle can be affected by early-life experiences (Shonkoff et al., 2009; Conti and Heckman, 2013). Early-life health also affects long-term outcomes, such as human capital accumulation, labor force participation, and earnings (Almond and Currie, 2011a,b). Because of this, air pollution can have far greater impacts during this period than later in life. Children are especially susceptible to air pollution compared to adults because their immune systems and lungs are not fully developed and they spend more time outdoor, where the concentrations of air pollution are generally higher. Furthermore, children inhale a higher volume of air than adults do, and lifetime exposure to pollution is close to contemporaneous exposure for children (Schwartz, 2004; Bateson and Schwartz, 2008; WHO, 2005).

The elderly are also more vulnerable to exposure to air pollution because they typically have reduced lung function, which occurs as a natural part of aging. Moreover, pre-existing diseases

may determine susceptibility, and exposure to air pollutants may be fatal due to co-morbidity. Elderly people are also more likely to suffer from chronic diseases, and there is evidence that co-existing chronic lung, heart, or circulatory conditions may worsen because of exposure to pollution (Simoni et al., 2015). For example, Deschênes et al. (2017) find that the largest effect on mortality due to variation in NO_x exposure occurs among individuals aged 75 and older, while Schlenker and Walker (2016) find that individuals over age 65 are more vulnerable to changes in CO exposure.

Evidence in the economics literature on the health effects of air pollution on the working-age population remains scarce. However, there is evidence suggesting that air pollution can have negative impacts on labor productivity, which may also be related to health (Neidell, 2017).

2.4.2 Socioeconomic Status

The relationship between socioeconomic status (SES) and health is one of the most robust findings in social science (Currie and Stabile, 2003). Findings show that lower income populations often are disproportionately exposed to and impacted by air pollution (Hsiang et al., 2019). For example, in a study from Indonesia, Jayachandran (2009) finds that there is a large difference in the effects of pollution on mortality between richer and poorer areas.

2.5 Productivity

The literature on the health effects of air pollution has recently been complemented by studies documenting that pollution lowers labor productivity in different contexts (Graff Zivin and Neidell, 2012; Arceo and Oliva, 2015; Chang et al., 2016, 2019). Adverse health effects of exposure to pollution can influence labor market productivity through two different channels. First, sickness related to air pollution exposure may lead to work absenteeism, either through total absence from work or by a reduction in the hours worked. Any resulting changes in labor market productivity would in this case be due to changes in labor supply. Second, workers may become less productive while at work due to the negative health effects of air pollution. Currently, the second channel is the one the literature has focused upon most. Chang et al. (2016) investigate the effect of air pollution on the productivity of workers in a pear-packing factory and find that an increase in particulate matter $(PM_{2.5})$ significantly decreases worker productivity. Similarly, Graff Zivin and Neidell (2012) find that a change in average ozone (O_3) exposure results in a significant reduction in agricultural worker productivity. Similar effects have been found also for indoor workers; Chang et al. (2019) investigate the effect of pollution on worker productivity in two call centers in China. Using measures of each worker's daily output linked to daily measures of pollution and meteorology, they find that higher levels of air pollution decrease worker productivity, measured as the number of calls that workers

complete each day. In contrast to these studies, we focus on the first channel, investigating the relationship between levels of pollution and certified sickness absence.

Related to the literature on labor productivity are studies that examine the effects of air pollution on cognitive performance and human capital accumulation. Ebenstein et al. (2016) show that particulate matter ($\mathrm{PM}_{2.5}$) exposure is associated with a significant decline in student performance during exams. Roth (2016) use readings of indoor PM_{10} to examine the effects of pollution on student performance on exams. He finds that increased levels of PM_{10} have statistically significant negative effects on test scores. Our results showing adverse health effects of PM_{10} can possibly explain one mechanism by which cognitive performance is lowered due to pollution. However, it is unclear whether health is a potential channel for lower cognitive ability or whether health is affected due to the lower cognitive ability.[5]

3 Data

3.1 Pollution and Weather Data

The Norwegian Institute of Air Research (NILU) provides comprehensive data on air pollution. According to NILU, monitoring stations should be located to "provide information about places where the population is believed to be exposed to the greatest concentrations of pollution averaged over a calendar year" (Hak, 2015). The locations of these pollution monitors are shown as blue dots in Figure 1. The map shows that pollution monitors are spread across the country, covering urban and rural areas, and that there are many pollution and weather stations in close proximity to the GPs' locations.

We use the weekly average level of pollution from each pollution monitor, taking the mean of the daily values.[6] To assign these weekly values of pollution to individuals, we have to make an important assumption. We do not have the exact address of each individual and thus use the address of his or her GP office as an approximation. This is based on several studies of the GP system in Norway showing that individuals choose GPs that are located either close to their home or their work (Godager, 2012; Luraas, 2003). Starting out with the GP offices, we measure the distance to each pollution monitor, using geographical coordinates of the pollution monitors and the general practitioners. We then construct a catchment area for pollution by drawing a circle with a radius of 5 km around each GP and calculate a weighted average of the weekly pollution level in each area using data from all pollution monitors within the circle, weighting by the distance from the monitor to the GP office. The locations of all GP offices in

[5]Research on other types of environmental hazards include climate change (Deschênes and Greenstone, 2007; Deschênes et al., 2009; Deschênes and Greenstone, 2011), environmental toxins (Reyes, 2007; Currie and Schmieder, 2009; Grönqvist et al., 2018), radiation (Almond et al., 2009; Black et al., 2019), and effects of measures to curb air pollution (Deschênes et al., 2017; Walker, 2013; Mullins and Bharadwaj, 2014; Tanaka, 2015).

[6]For both pollutants, the daily measure is the 24-hour average. We use weekly averages to keep the amount of data at a manageable level.

Norway are shown as black triangles in Figure 1.

In general, PM_{10} and NO_2 are measured routinely over the whole period at most of the stations. However, not all monitors have GP offices in close proximity. The data from some monitors are therefore not included in the analysis. In the main specification, we have data on PM_{10} and NO_2 from 35 pollution monitors, covering the patient lists of 1174 GPs.

Because weather is a key determining factor of pollution levels, and it could simultaneously have independent effect on health (Deschênes et al., 2009), we include weather controls in all our specifications. The weather data come from the Norwegian Meteorological Institute. For each pollution monitor, we calculate a weighted average of the weekly temperature, precipitation, and wind speed using all weather monitors within a circle with a radius of 25 km, where the weight is the distance from the pollution monitor to the weather monitor. The locations of the weather monitors are shown as red dots in Figure 1.

3.2 Health Data

The health data come from different administrative registers covering the entire resident population of Norway from 2011 to 2014: Hospital records from the Norwegian Patient Registry (NPR), data on GP consultations, visits to the emergency room (ER) and sickness absence from the Control and Distribution of Health Reimbursement database (KUHR), and information on the GPs and their patient lists from the GP database.

In Norway, all individuals belong to the patient list of a certified Norwegian GP. These GPs are the first doctor individuals meet when they seek healthcare, as specialist care requires a referral from a GP. The GPs in this system are financed by a mix of capitation (a lump sum per patient on their list — on average 30% of their income) combined with fee for service and copayment (on average 70% of the income). For this system to work, the GPs must routinely report on all the services they provide to each patient. For each consultation, they send an invoice to the Health Economics Administration (HELFO), which includes the patient's personal ID number, a classification of the main diagnosis given by ICPC-2 codes (ICPC-2 codes[7]) linked to the consultation, and a list of procedures used in the consultation. The issuance of a sickness certificate, for example, is reported with a specific code. Data from GP invoices are included in KUHR, which is the one we use. KUHR thus provides information on all actions the GP has taken for each patient. The GP consultations in this database are conducted either in the GPs offices or in the ER.

Information about hospital visits comes from the NPR. This register contains data on all admissions to somatic and psychiatric hospitals, both inpatient (overnight stays) and outpatient (day treatments and shorter consultations). For all admissions, information about the main and

[7]The International Classification of Primary Care (ICPC) is a classification method for primary care encounters. It classifies the patient's reason for the encounter and the related diagnosis as well as the procedures done by the primary healthcare service.

secondary diagnoses is available, all given by ICD-10 codes[8] related to each incidence.

From the GP database, we obtain background information on each GP: age and gender of the GP, which individuals are on each GP's list, the number of patients on the GP's list, and the zip code of the location of the GPs offices. We merge the data from NPR, KUHR, and the GP database with individual population data from Statistics Norway to get information about the patients on each GP's list, including age, gender, education level, and income, to generate the final data set.

3.3 Outcome Variables

From the KUHR database, we obtain seven outcome variables: all GP consultations in total, GP consultations related to respiratory and cardiovascular diagnoses, as well as all GP consultations in the ER, all sickness absences certified by GPs, and certified sickness absences related to respiratory and cardiovascular diagnoses. From the NPR, we obtain three outcome variables: all acute hospital visits in total (both inpatient and outpatient admissions) as well as acute hospital visits related to respiratory illnesses and cardiovascular illnesses.

For the purpose of placebo analyses, we include several diagnoses that we do not expect to be affected by contemporaneous air pollution. From the KUHR database, we obtain GP consultations and certified sickness absences related to male genital diagnoses. From the NPR, we obtain hospital visits related to diseases of the genitourinary system. Because the medical and epidemiological research on effects of PM_{10} and NO_2, and air pollution in general, is not yet very mature, it is difficult to identify diagnoses or health conditions that we can be certain are not affected by air pollution. We examine these placebo diagnoses since we are relatively sure that they are not associated with air pollution.

All outcomes are measured as weekly totals of all patient lists within the catchment area defined above. These weekly totals are separated into subgroups by age (ages 0–5, 6–18, 19–39, 40–69, and 70+) and gender. The outcomes are thus measured as weekly totals for each GP patient list group (i.e., 10 groups for each GP patient list).

3.4 Sample Selection and Descriptive Statistics

The main sample consists of 2,093,468 GP patient list groups. However, there are some missing pollution and weather observations. The final sample used in the analysis therefore consists of 1,643,121 GP patient list groups. Table 1 provides summary statistics for the two pollutants (Panel A), the main outcome variables (Panel B), characteristics of the GPs' patient lists (Panel C), and the weather controls (Panel D). The mean pollution levels are 32.63 $\mu g/m^3$ and 19.28

[8]ICD-10 is the International Statistical Classification of Diseases and Related Health Problems, a medical classification list by the World Health Organization. It contains codes for diseases, signs and symptoms, abnormal findings, complaints, social circumstances, and external causes of injury or diseases.

$\mu g/m^3$ for NO_2 and PM_{10}, respectively.[9] They are both below the international guidelines on annual mean concentrations, and as shown in Figure 3, both pollutants display clear seasonal patterns, as the levels of both PM_{10} and NO_2 tend to be higher during the winter months. The correlation between the two pollutants is 0.46. Figure 2 shows that both distributions are skewed to the left, as most observations are in the lower end of the distributions.

As described above, the outcomes are measured as weekly totals of all individuals on the list of each GP having at least one pollution monitor within 5 km of its office location. These weekly totals are further separated into subgroups by age and gender. For each GP, we have a list of patients divided into ten groups (5 age groups, 2 genders), and we use, for example, the total number of GP consultations for each of these groups in each week as one outcome. The mean number of patients in each group is 129. The variation in size between the groups is large: while the smallest group includes only one patient, the largest group includes 1281 patients. Each GP patient group has on average 6.52 GP consultations, 1.10 certified sickness absence spells, and 0.83 acute hospital visits per week.

In the main sample, 50% of the individuals are female, and the mean age of the individuals is 35, 69% are born in Norway, and they earn on average 468,628 NOK per year. A total of 17% have finished primary school, 32% have finished high school, and 51% have higher education. The average wind speed per week is 3.20 m/s, weekly average precipitation is 4.17 mm, and weekly average temperature is 6.89°C.

4 Empirical Strategy

The inherent endogenous relationship between air pollution and health poses two main challenges. The first is accounting for time-varying omitted variables that are potentially correlated with both ambient air pollution and health. The most likely example of such a factor is the weather. We address this problem by controlling for temperature, wind, and precipitation in all specifications. Moreover, people can respond to increased pollution levels through avoidance behavior. We cannot control for this in our setting. However, if contemporaneous avoidance behavior is both positively related to pollution levels and lowers the likelihood of adverse health outcomes, failing to account for this will give us lower bound estimates of the true effects (Neidell, 2004). Individuals can, as mentioned above, also self-select into locations based on pollution levels. However, our focus on short-term variation in pollution limits concerns regarding residential sorting.[10] Finally, since we account for time-invariant features of a GP's

[9] In comparison, previous studies have examined daily levels of PM_{10} between 25-40 $\mu g/m^3$, and NO_2 levels between 20.5-45 $\mu g/m^3$ (Neidell, 2004; Currie and Neidell, 2005; Schlenker and Walker, 2016; Bauernschuster et al., 2017). In some European countries in 2009, country-average PM_{10} exposure levels in urban areas varied from 10-14 $\mu g/m^3$ (Iceland, Estonia, Finland, and Ireland) to 58-61 $\mu g/m^3$ (Turkey and Bosnia and Herzegovina). A two- to three-fold between-city variation in exposure levels was observed in some countries (WHO, 2011).

[10] Existing research on residential sorting is largely based on average pollution levels over longer time periods (Chay and Greenstone, 2005; Currie et al., 2015).

patient list with fixed effects, we are able to capture time-invariant observed and unobserved factors within a GP patient list that can affect health.

4.1 Main Specification

The main estimating equation is given by:

$$Y_{iat} = \beta_1 + \beta_2 PM_{10at} + \beta_3 NO_{2at} + \beta_4 X_{iat} + \beta_5 W_{at} + \delta_t + \theta_i + \tau_i t + v_{iat} \quad (1)$$

where Y_{iat} is the outcome of interest, *i* indexes GP patient list group, *a* indexes pollution catchment area, and *t* indexes week. PM_{10at} and NO_{2at} indicate ambient pollution levels, and X_{iat} is a set of observable characteristics of the individuals in GP patient list group *i*, including gender, age, years of education, and earnings. W_{at} represents weather controls, including weekly average precipitation, temperature, and wind. θ_i is a set of GP-specific fixed effects, and δ_t is a set of week fixed effects (one for each of the 208 weeks that our data covers). To distinguish the effects of increased levels of pollution from differential secular trends in health on the different GP patient lists, we include GP-specific linear time trends in all specifications $\tau_i t$. Regression estimates are weighted by the number of patients in each GP patient list group, and standard errors are clustered at the GP level.

In the main specification, the pollution variables are dummy variables indicating whether pollution levels reach the recommended threshold for annual mean pollution concentrations given in the Norwegian air quality guidelines.[11] These guidelines set the threshold levels at 20 $\mu g/m^3$ for PM_{10} and at 40 $\mu g/m^3$ for NO_2. The threshold levels are above, but relatively close to the mean pollution levels in our data (19.28 $\mu g/m^3$ and 32.63 $\mu g/m^3$ for PM_{10} and NO_2, respectively). The coefficients of interest are β_2 and β_3, which indicate the relationship between pollution levels reaching the recommended threshold and the outcome variables.

4.2 Heterogeneity

As described in Section 2, previous studies have found heterogeneity in the relationship between air pollution and health. We follow up on these studies and examine whether exposure to air pollution affects different population groups differently. We compare effects for five age groups, both genders, groups with a high and a low share of low educated individuals, and income quartiles by running separate regressions for each group.

4.3 Alternative Pollution Measures

To test the model specification and explore model dynamics, we supplement the baseline regression with two alternative models. First, we examine whether the relationship between air

[11] An alternative could be to use the 24-hour mean, which is set at 50 $\mu g/m^3$ for PM_{10} and at 200 $\mu g/m^3$ for NO_2. However, since our pollution variables are given by weekly means, this is not the most relevant threshold, since it is highly unlikely that all days in a certain week have pollution levels above the 24-hour threshold.

pollution and health is linear or non-linear. A priori, it is not clear whether we should expect increased levels of pollution in general or reaching certain threshold levels to be most harmful for human health. Some studies suggest a linear relationship between air pollution and adverse health outcomes, while others suggest the existence of a threshold level below which no adverse effects are found (Stoeger et al., 2006). To investigate this, we first run regressions in which we include continuous pollution variables. The coefficients of interest, β_2 and β_3, now show the effect of a 1 $\mu g/m^3$ increase in pollution levels on the outcomes. Second, to further explore possible non-linear effects of exposure to air pollution we use a specification in which we include continuous pollution variables and second-order polynomials of the pollution variables.

4.4 Multiple Hypothesis Testing

Because we estimate the effects on many outcomes based on the same variation, we correct p-values for multiple hypothesis testing. We use the Benjamini and Hochberg (1995) q-values method described in Anderson (2008), controlling for the false discovery rate (FDR) or the proportion of rejections that are "false discoveries" (type 1 errors). We include all outcomes in one table in the same group of outcomes and do multiple hypothesis testing.

5 Results

5.1 Main Results

The first set of results showing the relationship between pollution and the main outcomes for the main sample are presented in Table 2. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The first row shows the estimated effects of PM_{10} and the second row the estimated effects of NO_2. Each entry in the table corresponds to a separate regression, and the outcome variables are measured as weekly aggregates within the GP patient list groups described above.

GP consultations. Column 1 shows estimates of the relationships between the pollutants and GP consultations in total, while Columns 2–3 show the relationships between the pollutants and GP consultations related to respiratory and cardiovascular diagnoses. Column 4 displays the relationships between the pollutants and GP consultations in the ER.

The results show positive and statistically significant relationships between PM_{10} and GP consultations in total, GP consultations related to respiratory diagnoses, and GP consultations in the ER. In weeks when PM_{10} exceeds the threshold level, the total number of GP consultations increases by 1.9% (0.12 from a mean of 6.52 consultations), and the number of GP consultations related to respiratory diagnoses increases by 2.6% (0.02 from a mean of 0.89 consultations). At the same time, the number of ER visits increases by 1.5% (0.01 from a mean of 0.54 visits) (statistically significant at the 10% level). There are no statistically significant

effects of NO_2 on GP consultations.

Sickness absence. Columns 5–7 show the relationships between the pollutants and sickness absence in total, and related to respiratory and cardiovascular diagnoses. The relationships between the pollutants and the sickness absence outcomes are examined using the sample of the working-age population (ages 19–69).

We find positive and statistically significant relationships between PM_{10} and sickness absence in total and between PM_{10} and sickness absence related to respiratory diagnoses. As can be seen in Column 5, the total number of certified sickness absence spells increases by 4.7% (0.05 from a mean of 2.74 spells) in weeks when the PM_{10} level exceeds the threshold level. Sickness absence spells related to respiratory diagnoses increase by 5.6% (0.01 from a mean of 0.44 spells) in weeks when the PM_{10} level exceeds the threshold level (Column 6).

There are also statistically significant effects at the 10% level on sickness absence related to cardiovascular diagnoses both in weeks when the PM_{10} level exceeds the threshold level and in weeks when NO_2 exceeds the threshold level. In weeks when PM_{10} exceeds the threshold level, sickness absence related to cardiovascular diagnoses increases by 2.5%, and in weeks when the NO_2 level exceeds the threshold level, sickness absence related to cardiovascular diagnoses increases by 3.3%. These effects do not, however, persist when we adjust p-values for multiple hypothesis testing.

Hospital visits. Columns 8–10 show the relationships between the pollutants and acute hospital visits in total, acute hospital visits related to respiratory diagnoses, and acute hospital visits related to cardiovascular diagnoses.

The results show statistically significant and positive relationships between NO_2 and the total number of acute hospital visits as well as between NO_2 and acute hospital visits related to respiratory diagnoses. The result in Column 8 shows that in weeks when NO_2 exceeds the threshold level the total number of acute hospital visits increases by 3.8% (0.04 from a mean of 0.83 visits). In Column 9, we see that the number of acute hospital visits related to the respiratory system increases by 5.8% (0.003 from a mean of 0.05 visits) in weeks in which NO_2 exceeds the threshold level. When we adjust the p-value for multiple hypothesis testing, this estimate does not remain statistically significant.

The estimate in the first row of Column 8 shows that there is a statistically significant negative relationship between PM_{10} and the total number of acute hospital visits. In weeks when the PM_{10} level exceeds the threshold level, the number of total hospital visits decreases by 1.3%. However, when adjusting the p-value for multiple hypothesis testing, this estimate does not remain statistically significant. Nevertheless, the effect may be negative because there is an increase in the number of GP consultations and sickness absences at the same time. This may indicate that the individuals receive the treatment they need from the GP and staying at

home and thus have a reduced need for a hospital visit. When we investigate the relationship between pollution and the main outcomes in different age groups in the heterogeneity analysis below, we find that the negative effect on hospital visits is driven by the working-age population (ages 40–69), which supports this notion.

Overall, the main results show that both PM_{10} and NO_2 is harmful to the health of the individuals in the main sample. The results further suggest that PM_{10} is related to diseases that can be treated by the treatment given by the GP and by sickness absence from work, while NO_2 is related to more serious diseases resulting in a hospital visit. Finally, the main underlying diagnosis is respiratory illness. As shown by the adjusted p-values in Table 2, the main results hold when we correct for multiple hypothesis testing.

5.2 Heterogeneity

Age. The results of the regressions run for each age group separately are presented in Table 3. Panel A shows the effects of pollution on the main outcomes on infants and children (ages 0–5), Panel B shows the effects on children and youth (ages 6–18), Panel C and D show the effects on the working-age population (ages 19–69), and Panel E shows the effects on the elderly (ages 70+).

The table shows clear indications of heterogeneity with respect to age in the relationship between air pollution and the use of health services, and we report on those that are statistically different from each other here. School-aged children go to the GP more often in weeks when the NO_2 level reaches the threshold level, while we observe no such effect for toddlers. The most striking pattern, however, is that the working age population (ages 19–39 and 40–69) seems to be strongest affected: they have more GP consultations than the other groups in periods with higher PM_{10}, and they experience more acute hospital visits in periods with higher NO_2 levels. School-aged children also experience more acute hospitals admissions in these periods, but to a much smaller degree than the working age population. Both the GP consultations and hospital visits are likely related to symptoms of worse health in these periods, but an additional factor related to GP consultations is that the working-age population needs the GP to certify sickness absence from work.

Gender. Table 4 presents results from the regressions on the subsamples defined by gender. Panel A shows the effects of pollution on the main outcomes on women, and Panel B shows the effects of pollution on men. The effects are similar across the two groups, and we find no statistically significant differences in the estimated effects.

Education. Table 5 presents the results from regressions on the subsamples defined by education level. We calculate the share of low-educated patients within each group (10 groups defined by combinations of age and gender) on the GP lists and divide the groups into two: those with a share of low-educated individuals above and below the median. For children in-

cluded in the sample, the education level is given by their parents' education levels. In Table 5, Panel A shows the effects of pollution on the main outcomes on patient list groups with a high share of individuals with low education, and Panel B shows the effects of pollution on patient list groups with a low share of individuals with low education. The effects of PM_{10} on hospital visits in total and on hospital visits related to respiratory diagnoses are statistically significantly different between the two groups. This is also the case for the effects of NO_2 on hospital visits in total.

Income quartiles. The results from the regressions run for each income quartile separately are presented in Table 6. Panel A shows the effects on income quartile 1 (0–343,966 NOK). Panel B shows the effects on income quartile 2 (343,967–438,167 NOK). The effects on quartile 3 (438,168–549,924 NOK) are displayed in Panel C, while Panel D shows the effects on income quartile 4 (549,925–11,470,973 NOK). For children included in the sample, the income level is given by their parents' income levels. The effect of PM_{10} on GP consultations on income quartile 2 is statistically significantly different from the effects on income quartile 1. This is also the case for the effect of PM_{10} on GP consultations related to cardiovascular diagnoses for income quartile 2 compared to the effects on income quartile 3, and on sickness absence in total for income quartile 2 compared to income quartiles 3 and 4. In addition, there are statistically significant differences in the effects of NO_2 on hospital visits related to respiratory diagnoses for income quartile 1 compared to income quartile 4.

5.3 Alternative Pollution Measures

Table 7 shows the results from estimations using the continuous pollution variables. That is, the results show the effect of a 1 $\mu g/m^3$ increase in pollution levels. The results are similar to the main results when it comes to PM_{10}, but the point estimates are smaller. The treatment is also smaller, so these effects are as expected. When it comes to NO_2, there are no effects when we use the continuous pollution variable. This may indicate that NO_2 only has effects at the higher end of its distribution.

The results show that when PM_{10} increases by one standard deviation, the number of GP consultations related to respiratory diagnoses increases by 2%, and the number of ER visits increases by 1.7%. The total number of certified sickness absence spells increases by 2% when PM_{10} increases by one standard deviation, and sickness absence spells related to respiratory diagnoses increase by 5% when PM_{10} increases by one standard deviation. There is also a decrease in the total number of hospital visits by 4% when PM_{10} increases by one standard deviation.

To further explore possible non-linear effects of exposure to air pollution, we use a specification in which we include continuous pollution variables and second-order polynomials of the pollution variables. As can be seen in Table 8, there is evidence of nonlinearity in the relation-

ship between NO_2 concentrations and the use of healthcare services, while there is no evidence of nonlinearity in the relationship between PM_{10} and the use of healthcare services or in that between both PM_{10} and NO_2 and sickness absence.

5.4 Placebo Diagnoses

To ensure that the estimated effects are due to air pollution, we run regressions on placebo diagnoses. If this is not the case, we would expect to see effects also on health service use related to diagnoses that are presumably not affected by air pollution. In order to test this, we estimate the effects of the pollutants on GP consultations and certified sickness absence related to the male genital system as well as hospital visits related to the genitourinary system. As can be seen in Table 9, except for a negative effect of PM_{10} on GP consultations related to male genital diagnoses, we find no effects on these outcomes.

6 Robustness Checks

Several of the decisions made with respect to the estimation strategy could potentially be crucial for the results. To ensure that our results are not driven by particular choices, we therefore run several robustness checks, which are presented in Table 10. Overall, the results in the robustness checks are similar to the baseline results presented in Column 1.

In Columns 2–3, we test how sensitive the baseline results are to the inclusion of different versions of time trends. In the baseline specification presented in Column 1, we include a linear GP-specific time trend. In Column 2, the specification includes no time trend, while in Column 3, the specification includes a quadratic GP-specific time trend. Except for the estimated effects of PM_{10} on hospital visits when there is no trend included in the specification, the results remain similar to the baseline results. The fact that this coefficient is different when there is no trend may suggest that this relationship does not fit with a linear or quadratic time trend approximation.

In Columns 4–5, we examine the robustness of the distance cutoff of the pollution catchment area in the baseline specification. In Column 4, the distance cutoff is 3 km, and in Column 5 it is 10 km. There are some differences in the estimated relationships using the different catchment areas. When the distance cutoffs are 3 km and 10 km, there is no longer a statistically significant relationship between PM_{10} and GP consultations. The point estimates of sickness absence are also smaller when the cutoff is 10 km. The rest of the results remain similar to the baseline results.

In Columns 6–8, we examine how outliers in the pollution distributions affect the results by reducing the sample by 1, 5, and 10 percent of the top of the pollution distributions. The results remain robust when we do this robustness check, showing that outliers in the pollution distributions do not drive the results.

7 Conclusion

In this paper, we use geographical and time variation in exposure to PM_{10} and NO_2 to estimate two-way fixed effects models of the relationship between air pollution and three main outcomes: GP consultations, hospital visits, and sickness absence from work. We also examine incidences related to the cardiovascular and respiratory systems separately.

We find positive and statistically significant relationships between PM_{10} and GP consultations and certified sickness absences, driven by diagnoses related to the respiratory system. We also find a positive and statistically significant relationship between NO_2 and acute hospital visits, again driven by diagnoses related to the respiratory system. The results suggest that PM_{10} is related to diseases that can be treated by the GP and sickness absence from work, while NO_2 is related to more serious diseases leading to a hospital visit. The heterogeneity analysis shows that the working-age population and the middle-to-high-income groups are the main drivers of the estimated effects.

Our findings build upon the existing evidence from both the economics and medical literature on the relationship between air pollution and health outcomes by providing evidence that air pollution affects both a range of health outcomes and productivity of the working-age population in a setting with relatively low pollution levels. Importantly, significant negative effects on both the health and productivity of the working-age population suggest that previous studies that have focused on children and the elderly may have overlooked important effects on one part of the population and hence underestimated the overall costs of air pollution. Taken together, our findings suggest that air pollution well below air quality guidelines has significant effects of the general population, and that society as a whole could benefit from stricter air pollution control. Specifically, since the two pollutants examined in this analysis are strongly related to road dust and car use, policies that could reduce traffic, such as tolls and taxes on the use of cars, will likely have positive effects on people's health, reduce sickness insurance payments, and free up capacity for other patients in the healthcare system.

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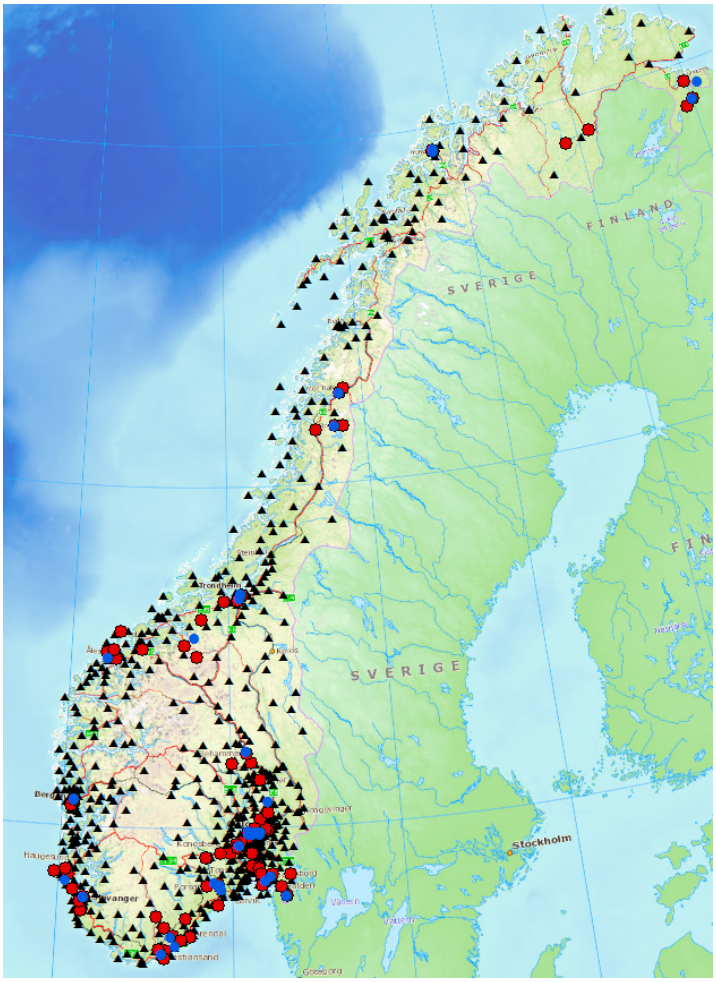
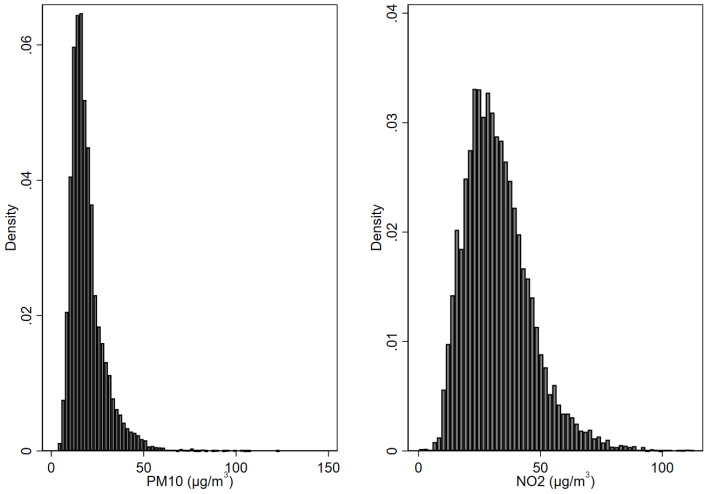
8 Figures and Tables

Figure 1: Norway



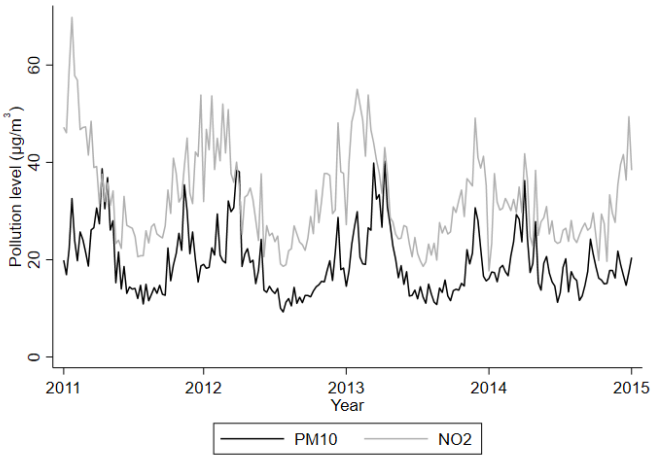
Note: Location of pollution monitors (blue dots), weather monitors (red dots), and the general practitioners (black triangles).

Figure 2: Distributions of the Pollutants



Note: Distributions of the pollutants. The level of pollution is given on the x-axis. The pollutants are measured as weekly mean levels, taking the mean of the daily values. For both pollutants, the daily measures we use are the 24-hour average.

Figure 3: Seasonal Variation in Pollution Levels.



Note: Seasonal variation in the pollutants over the four years that our data cover.

Table 1: Descriptive Statistics

	Mean	St.Dev.	Min	Max
Panel A: Pollutants				
Weekly average PM_{10} $\mu g/m^3$	19.28	9.36	3.47	123.52
Weekly average NO_2 $\mu g/m^3$	32.63	13.67	0.24	113.08
Panel B: Main outcome variables				
GP consultations (#)	6.52	7.53	0.00	146.00
Sickness absence (#)	1.10	2.18	0.00	46.00
Hospital visits (#)	0.83	1.46	0.00	28.00
Panel C: Patient group characteristics				
Male	0.50	0.50	0.00	1.00
Age	35.06	27.65	0.00	95.00
Share with primary school	0.17	0.13	0.00	1.00
Share with high school	0.32	0.14	0.00	1.00
Share with higher education	0.51	0.22	0.00	1.00
Share of natives	0.69	0.21	0.00	1.00
Mean earnings	468216	194191	194	11470973
Number of patients	129.04	112.09	1.00	1281.00
Panel D: Weather controls				
Weekly average wind speed (m/s)	3.20	1.41	0.13	11.80
Weekly average rain (mm)	4.17	3.95	0.00	38.88
Weekly average temperature °C	6.89	7.10	-16.56	23.10
Observations	2,093,721			

Note: Descriptive statistics for the sample. The dependent variables are measured as weekly group aggregates for GP patient groups. The list characteristics are the means in the GP patient list groups.

Table 2: Main Results

	GP consultations				**Sickness absence**			**Hospital visits**		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PM_{10}	0.124***	0.023***	0.007	0.008*	0.052***	0.010**	0.003*	-0.011*	-0.001	0.001
	(0.024)	(0.006)	(0.006)	(0.004)	(0.013)	(0.004)	(0.002)	(0.007)	(0.001)	(0.001)
Adj. p-value	.001	.001	.291	.104	.001	.047	.127	.127	.544	.459
NO_2	0.043	-0.003	0.001	0.006	0.027	0.000	0.004*	0.035***	0.003**	0.000
	(0.033)	(0.008)	(0.007)	(0.004)	(0.018)	(0.005)	(0.002)	(0.009)	(0.001)	(0.001)
Adj. p-value	.326	.915	.915	.302	.302	.915	.302	.001	.915	.084
Mean	[6.52]	[.89]	[.89]	[.54]	[2.72]	[.44]	[.12]	[.83]	[.05]	[.04]
N	1642921	1642921	1642921	1642921	662976	662976	662976	1642921	1642921	1642921

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The first row shows the effects of PM_{10} and the second row shows the effects of NO_2. The regressions on sickness absence are based on the sample of the working-age population. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors clustered at the GP level are reported in parentheses and comparison means in brackets. * p<0.1, ** p<0.05, *** p<0.01.

Table 3: Heterogeneity: Age Groups

	GP consultations				Sickness absence			Hospital visits		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PANEL A: Infants and children (0–5)										
PM_{10}	0.003	-0.005	-0.000	0.003				-0.000	0.003	0.000
	(0.016)	(0.009)	(0.001)	(0.006)				(0.004)	(0.002)	(0.000)
NO_2	-0.006	-0.015	0.000	-0.009				0.004	-0.004	-0.000
	(0.020)	(0.013)	(0.001)	(0.008)				(0.005)	(0.002)	(0.000)
Mean	[1.84]	[.68]	[.01]	[.46]				[.24]	[.06]	[.00]
N	327006	327006	327006	327006				327006	327006	327006
PANEL B: Children and youth (6–18)										
PM_{10}	0.027*	0.006	0.002	0.006				0.001	0.001	-0.000
	(0.014)	(0.007)	(0.001)	(0.004)				(0.005)	(0.001)	(0.000)
NO_2	0.056***	0.009	-0.000	0.000				0.015**	-0.001	-0.000
	(0.016)	(0.007)	(0.001)	(0.005)				(0.006)	(0.001)	(0.000)
Mean	[2.12]	[.49]	[.01]	[.34]				[.33]	[.01]	[.00]
N	326203	326203	326203	326203				326203	326203	326203
PANEL C: Working-age population (19–39)										
PM_{10}	0.178***	0.039***	0.002	0.015*	0.064***	0.017**	0.003*	0.004	0.001	-0.001
	(0.038)	(0.011)	(0.004)	(0.009)	(0.017)	(0.007)	(0.001)	(0.011)	(0.001)	(0.001)
NO_2	0.035	0.002	0.002	0.014	0.026	0.009	0.003	0.056***	-0.001	0.001
	(0.059)	(0.013)	(0.005)	(0.009)	(0.025)	(0.008)	(0.002)	(0.015)	(0.002)	(0.001)
Mean	[9.76]	[1.28]	[.24]	[.88]	[2.61]	[.47]	[.04]	[1.21]	[.02]	[.01]
N	331710	331710	331710	331710	331710	331710	331710	331710	331710	331710
PANEL D: Working-age population (40–69)										
PM_{10}	0.142***	0.020*	0.011	0.005	0.040**	0.003	0.004	-0.032**	-0.001	-0.002
	(0.044)	(0.011)	(0.013)	(0.006)	(0.016)	(0.006)	(0.004)	(0.013)	(0.002)	(0.003)
NO_2	0.051	-0.013	-0.011	0.006	0.022	-0.007	0.005	0.042**	0.003	0.007**
	(0.056)	(0.015)	(0.017)	(0.007)	(0.020)	(0.006)	(0.004)	(0.017)	(0.002)	(0.003)
Mean	[13.40]	[1.51]	[2.09]	[.74]	[2.84]	[.40]	[.20]	[1.45]	[.05]	[.11]
N	331266	331266	331266	331266	331266	331266	331266	331266	331266	331266
PANEL E: The eldery (70+)										
PM_{10}	0.067**	0.011	0.023	-0.003				-0.020	0.001	0.001
	(0.031)	(0.007)	(0.017)	(0.005)				(0.014)	(0.003)	(0.005)
NO_2	0.032	0.012	0.004	-0.001				-0.017	-0.001	0.001
	(0.035)	(0.009)	(0.020)	(0.006)				(0.016)	(0.003)	(0.004)
Mean	[5.33]	[.46]	[2.09]	[.28]				[.92]	[.07]	[.13]
N	326736	326736	326736	326736				326736	326736	326736

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors are clustered at the GP level. * p<0.1, ** p<0.05, *** p<0.01.

Table 4: Heterogeneity: Gender

	GP consultations				Sickness absence			Hospital visits		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PANEL A: Women										
PM_{10}	0.150***	0.018**	-0.001	0.008	0.063***	0.009	-0.000	-0.007	-0.000	-0.001
	(0.033)	(0.009)	(0.006)	(0.005)	(0.018)	(0.007)	(0.002)	(0.009)	(0.001)	(0.001)
NO_2	0.073*	0.001	0.004	0.011*	0.036	0.005	0.003	0.041***	-0.000	0.001
	(0.044)	(0.011)	(0.008)	(0.006)	(0.024)	(0.008)	(0.003)	(0.012)	(0.001)	(0.001)
Mean	[7.78]	[1]	[.85]	[.57]	[3.42]	[.54]	[.11]	[.90]	[.04]	[.04]
N	822298	822298	822298	822298	331933	331933	331933	822298	822298	822298
PANEL B: Men										
PM_{10}	0.101***	0.028***	0.015*	0.007	0.042***	0.011**	0.007**	-0.015*	0.001	-0.001
	(0.025)	(0.007)	(0.007)	(0.006)	(0.015)	(0.005)	(0.003)	(0.009)	(0.001)	(0.002)
NO_2	0.005	-0.006	-0.003	0.001	0.016	-0.003	0.006	0.028**	0.001	0.005**
	(0.035)	(0.009)	(0.010)	(0.005)	(0.022)	(0.006)	(0.004)	(0.011)	(0.001)	(0.002)
Mean	[5.26]	[.77]	[.93]	[.51]	[2.02]	[.33]	[.13]	[.76]	[.04]	[.06]
N	820623	820623	820623	820623	331043	331043	331043	820623	820623	820623

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The regressions on sickness absence are based on the sample of the working-age population. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors are clustered at the GP level. * p<0.1, ** p<0.05, *** p<0.01.

Table 5: Heterogeneity: Education

	GP consultations				Sickness absence			Hospital visits		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PANEL A: High share of low educ.										
PM_{10}	0.108***	0.028***	0.015*	0.001	0.033*	0.003	0.002	-0.027***	-0.001	-0.002
	(0.035)	(0.008)	(0.009)	(0.005)	(0.020)	(0.006)	(0.003)	(0.009)	(0.001)	(0.002)
NO_2	0.066	0.006	-0.006	0.005	0.030	-0.000	0.001	0.010	0.001	0.003
	(0.048)	(0.010)	(0.012)	(0.006)	(0.028)	(0.007)	(0.004)	(0.011)	(0.001)	(0.002)
Mean	[7.88]	[.94]	[1.36]	[.59]	[2.76]	[.41]	[.14]	[.97]	[.05]	[.08]
N	782919	782919	782919	782919	293271	293271	293271	782919	782919	782919
PANEL B: Low share of low educ.										
PM_{10}	0.151***	0.020**	0.004	0.016***	0.060***	0.016**	0.005**	0.005	0.002*	0.000
	(0.030)	(0.009)	(0.006)	(0.006)	(0.016)	(0.006)	(0.002)	(0.009)	(0.001)	(0.001)
NO_2	0.047	-0.008	0.003	0.007	0.029	0.002	0.005*	0.069***	-0.001	0.003*
	(0.043)	(0.011)	(0.008)	(0.006)	(0.022)	(0.008)	(0.003)	(0.012)	(0.001)	(0.002)
Mean	[5.16]	[.84]	[.42]	[.49]	[2.69]	[.46]	[.10]	[.70]	[.03]	[.02]
N	860002	860002	860002	860002	369705	369705	369705	860002	860002	860002

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The regressions on sickness absence are based on the sample of the working-age population. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors are clustered at the GP level. * p<0.1, ** p<0.05, *** p<0.01.

Table 6: Heterogeneity: Income Quartiles

	GP consultations				Sickness absence			Hospital visits		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PANEL A: Income quartile 1										
PM_{10}	0.141***	0.005	0.010	0.015	0.039*	-0.000	0.000	-0.008	0.000	-0.001
	(0.042)	(0.012)	(0.008)	(0.010)	(0.024)	(0.009)	(0.002)	(0.011)	(0.002)	(0.002)
NO_2	0.072	0.002	-0.001	0.007	0.050*	0.014	0.004	0.015	-0.002	0.000
	(0.056)	(0.012)	(0.010)	(0.010)	(0.029)	(0.010)	(0.003)	(0.016)	(0.002)	(0.002)
Mean	[7.73]	[.87]	[1.22]	[.59]	[2.73]	[.44]	[.05]	[1.02]	[.05]	[.07]
N	389040	389040	389040	389040	167393	167393	167393	389040	389040	389040
PANEL B: Income quartile 2										
PM_{10}	0.167***	0.040***	-0.013	0.001	0.100***	0.019**	0.002	-0.020	-0.001	0.001
	(0.046)	(0.012)	(0.010)	(0.008)	(0.022)	(0.008)	(0.003)	(0.013)	(0.002)	(0.002)
NO_2	0.050	-0.000	0.012	0.012	0.011	-0.003	0.001	0.036**	-0.001	-0.000
	(0.070)	(0.016)	(0.013)	(0.009)	(0.035)	(0.011)	(0.004)	(0.015)	(0.002)	(0.002)
Mean	[7.47]	[1.01]	[.85]	[.61]	[2.95]	[.49]	[.10]	[.90]	[.04]	[.05]
N	393136	393136	393136	393136	206486	206486	206486	393136	393136	393136
PANEL C: Income quartile 3										
PM_{10}	0.112***	0.012	0.021*	-0.001	0.028	0.003	0.003	-0.017	-0.000	-0.003
	(0.042)	(0.011)	(0.012)	(0.007)	(0.026)	(0.010)	(0.005)	(0.013)	(0.002)	(0.002)
NO_2	0.020	-0.013	0.015	0.011	0.019	-0.004	0.007	0.031*	-0.000	0.006**
	(0.054)	(0.013)	(0.015)	(0.007)	(0.029)	(0.011)	(0.005)	(0.017)	(0.002)	(0.003)
Mean	[5.46]	[.84]	[.65]	[.50]	[2.89]	[.47]	[.16]	[.69]	[.04]	[.04]
N	408546	408546	408546	408546	138455	138455	138455	408546	408546	408546
PANEL D: Income quartile 4										
PM_{10}	0.082**	0.024**	0.013	0.015**	0.029	0.011	0.009*	-0.003	0.002*	0.000
	(0.037)	(0.011)	(0.012)	(0.006)	(0.019)	(0.007)	(0.005)	(0.013)	(0.001)	(0.003)
NO_2	0.027	-0.001	-0.006	-0.005	0.021	-0.008	0.005	0.048***	0.004*	0.006*
	(0.042)	(0.013)	(0.014)	(0.006)	(0.022)	(0.008)	(0.006)	(0.016)	(0.002)	(0.003)
Mean	[5.43]	[.83]	[.83]	[.47]	[2.21]	[.33]	[.20]	[.72]	[.03]	[.05]
N	452199	452199	452199	452199	150642	150642	150642	452199	452199	452199

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The income level in income quartile 1 is 0–343,966 NOK, in quartile 2 it is 343,967–438,167 NOK, in quartile 3 it is 438,168–549,924 NOK, and in quartile 4 it is 549,925–11,470,973 NOK. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors are clustered at the GP level. * p<0.1, ** p<0.05, *** p<0.01.

Table 7: Alternative Pollution Measures: Continuous Pollution Variable

	GP consultations				**Sickness absence**			**Hospital visits**		
	Total (1)	Respiratory (2)	Cardiovascular (3)	ER (4)	Total (5)	Respiratory (6)	Cardiovascular (7)	Total (8)	Respiratory (9)	Cardiovascular (10)
PM_{10}	0.003*	0.002***	-0.001	0.001***	0.002***	0.001***	-0.000	-0.002***	-0.000**	0.000
	(0.002)	(0.000)	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. p-value	.141	.001	.259	.003	.008	.003	.503	.001	.442	.032
NO_2	0.001	-0.001	0.000	-0.000	0.000	-0.000	0.000	-0.000	0.000	-0.000**
	(0.002)	(0.000)	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Adj. p-value	.664	.618	.785	.916	.782	.664	.618	.916	.197	.618
Mean	[6.52]	[.89]	[.89]	[.54]	[2.72]	[.44]	[.12]	[.83]	[.05]	[.04]
N	1642921	1642921	1642921	1642921	662976	662976	662976	1642921	1642921	1642921

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the continuous pollution variables. The first row shows the effects of PM_{10} and the second row shows the effects of NO_2. The regressions on sickness absence are based on the sample of the working-age population. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors clustered at the GP level are reported in parentheses and comparison means in brackets. * p<0.1, ** p<0.05, *** p<0.01.

Table 8: Alternative Pollution Measures: Non-Linearity

	GP consultations (1)	**Sickness absence** (2)	**Hospital visits** (3)
PM_{10}	0.0057	0.0029	-0.0009
	(0.004)	(0.002)	(0.001)
PM_{10}^2	-0.0000	-0.0000	-0.0000
	(0.000)	(0.000)	(0.000)
NO_2	0.0089**	0.0026	-0.0085***
	(0.004)	(0.002)	(0.001)
NO_2^2	-0.0001**	-0.0000	0.0001***
	(0.000)	(0.000)	(0.000)
Mean	[6.52]	[2.72]	[.83]
N	1642921	662976	1642921

Note: Each column is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The first row shows the effects of PM_{10} and the third row shows the effects of NO_2. Row two and four show the effects of second-order polynomials of the pollutants. The regressions on sickness absence are based on the sample of the working-age population. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors clustered at the GP level are reported in parentheses and comparison means in brackets. * p<0.1, ** p<0.05, *** p<0.01.

Table 9: Placebo Diagnoses

	GP consultations	**Sickness absence**	**Hospital visits**
	Male genital	Male genital	Genitourinary system
	(1)	(2)	(3)
PM_{10}	0.001	0.001	0.001
	(0.002)	(0.001)	(0.001)
Adj. p-value	.487	.487	.487
NO_2	-0.005**	0.000	-0.000
	(0.002)	(0.001)	(0.001)
Adj. p-value	.041	.887	.887
Mean	[.09]	[.01]	[.02]
N	1642921	662976	1642921

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. The first row shows the effects of PM_{10} and the second row shows the effects of NO_2. Controls, as listed in Table 1, week and GP fixed effects, as well as linear time trends are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors clustered at the GP level are reported in parentheses and comparison means in brackets. * p<0.1, ** p<0.05, *** p<0.01.

Table 10: Robustness

		Trend versions		**Distance cutoffs**		**Sample selection**		
	Baseline	No	Quadratic	3 km	10km	90%	95%	99%
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
GP consultations								
PM_{10}	0.124***	0.124***	0.124***	0.001	0.002	0.131***	0.134***	0.136***
	(0.024)	(0.024)	(0.024)	(0.002)	(0.001)	(0.024)	(0.025)	(0.026)
NO_2	0.042	0.042	0.042	0.002	0.003*	0.041	0.058*	0.052
	(0.033)	(0.033)	(0.033)	(0.002)	(0.002)	(0.033)	(0.035)	(0.037)
Sickness absence								
PM_{10}	0.052***	0.052***	0.052***	0.050***	0.025**	0.053***	0.046***	0.048***
	(0.013)	(0.013)	(0.013)	(0.015)	(0.011)	(0.013)	(0.013)	(0.013)
NO_2	0.027	0.027	0.027	0.001	0.032**	0.027	0.024	0.025
	(0.018)	(0.018)	(0.018)	(0.022)	(0.015)	(0.018)	(0.018)	(0.018)
Hospital visits								
PM_{10}	-0.011*	0.022***	-0.011*	-0.002***	-0.001***	-0.013**	-0.017**	-0.021***
	(0.007)	(0.006)	(0.007)	(0.001)	(0.000)	(0.007)	(0.007)	(0.007)
NO_2	0.035***	0.120***	0.035***	0.002***	-0.001**	0.038***	0.040***	0.037***
	(0.009)	(0.009)	(0.009)	(0.001)	(0.000)	(0.009)	(0.009)	(0.009)
N	1642921	1642921	1642921	1233196	2315237	1613341	1498761	1370244

Note: Each parameter is from a separate regression of GP patient list group outcomes (weekly counts of outcomes per GP patient list group) on weekly pollution levels in the period 2011–2014. The table shows the results from estimations using the dummy variables indicating whether the pollution levels are above the guideline threshold levels. For each outcome, the first row shows the effects of PM_{10} and the second row shows the effects of NO_2. The regressions on sickness absence are based on the sample of the working-age population. Column 1 shows the baseline estimates. In Columns 2–3 we include no and GP-specific quadratic time trends, respectively. In Columns 4–5 we use different catchment area cut-offs. In Columns 6–8, we cut the sample by 10, 5, and 1% of the top pollution distributions. Controls, as listed in Table 1, week and GP fixed effects are included in all regressions. Regression estimates are weighted by the number of patients in each GP patient list group. Standard errors are clustered at the GP level. * p<0.1, ** p<0.05, *** p<0.01.

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