Moving to Cleaner Skyes: Age-Specific Effects of Exposure to Pollution on Health and Human Capital*

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Abstract

This paper investigates the long-term and age-specific consequences of early childhood exposure to pollution by exploiting the sharp decline in imported sulfur dioxide in Norway following the enactment of the 1979 Convention on Long-Range Transboundary Air Pollution. We find that cohorts born after 1980 in municipalities experiencing significant reductions in pollution exposure saw greater improvements in education, earnings, and health outcomes at age 30 than those born in municipalities with minimal initial exposure. To further examine age-specific effects, we embed a movers' design in our analysis, leveraging relocations that generate sharp variations in exposure levels—both in magnitude and direction—across different ages. Our findings highlight the critical importance of early-life exposure, irrespective of its duration.

JEL: H23, H51, I14, J18, Q5, Q53, Q58

Keywords: Pollution, Children, Health, Long-term Effects, Environmental Policy

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1 Introduction

Despite decades of emission reductions, air pollution is one of the most significant environmental health risks. A growing literature has documented the causal effects of pollution, for example, on health outcomes including respiratory diseases, hospitalizations, and mortality (e.g. Chay and Greenstone, 2003; Currie and Neidell, 2005; Graff Zivin and Neidell, 2013), cognition (e.g., Ebenstein, Lavy, and Roth, 2016) and labor productivity (e.g., Graff Zivin and Neidell, 2012; Chang et al., 2016, 2019). Most quasi-experimental estimates linking pollution and human capital, labor market, or health outcomes are based on short-run changes in pollution caused by idiosyncratic changes in local wind direction (e.g., Deryugina et al., 2019), daily airport congestion (Schlenker and Walker, 2015), or inversion episodes (e.g., Jans, Johansson, and Nilsson, 2018). However, these identification strategies mainly alter the timing of exposure but fail to identify the cumulative effects of pollution exposure. If the changes in health or human capital outcomes were more gradual than the change in pollution or the effects of pollution accumulate, it may take time for the full consequences to develop, and the short-run effects of the pollution may understate the long-run effects. Moreover, short-term pollution changes enable individuals to adapt their behavior temporarily by spending less time outdoors (Neidell, 2009; Moretti and Neidell, 2011) or adjusting their work schedules (e.g., Hoffmann and Rud, 2022). However, in the long run, such avoidance strategies become significantly more costly.

As pollution exposure is believed to be particularly harmful in utero or to infants when humans undergo stages of rapid development of their central nervous and immune systems, understanding the long-term consequences of childhood pollution exposure is crucial. Grönqvist, Nilsson, and Robling (2020); Isen, Rossin-Slater, and Walker (2017); Black et al. (2019), and Persico (2022) document that exposure to policies limiting pollution exposure early in life improves test scores, education, and income. However, we have limited knowledge of the long-term consequences of childhood pollution exposure on adult health. In addition, measuring the effect of long-run pollution exposure is complicated by the circumstance that human capital and health outcomes might be malleable at different ages, and damages from pollution exposure might affect the vulnerability to future pollution exposure. That is, infants exposed to high levels of air pollution are likely also exposed to the same pollution as toddlers, preschoolers, and school-aged children. Conventional causal estimates often investigate the exposure during a specific period, such as the in utero period, infancy, or old age, and do not necessarily distinguish whether age at exposure and duration of exposure are the main mechanisms behind the estimated effects.[1] If the timing of exposure

[1] Only a few exceptions try to pinpoint which exact month in utero is most crucial (Black et al., 2019).

correlates with socioeconomic status, the potential existence of such sensitive periods implies that the impact of pollution-limiting policies crucially depends on the timing at which they operate and how pollution exposure accumulates over more extended periods. Hence, credibly estimating the long-term effects of childhood pollution exposure is often challenging due to the lack of large-scale data that connects adult outcomes to early-life exposure and provides detailed information on key confounding factors. In addition to data limited data availability, there is a lack of a framework to distinguish age-specific effects of exposure.

This paper aims to provide consistent estimates of the long-term effects of childhood exposure to pollution and identify the ages at which exposure is most harmful. To quantify these effects, we leverage a lasting, exogenous reduction in ambient pollution driven by environmental regulations that significantly improved air quality. Specifically, we examine the sharp decline in sulfur dioxide (SO2) concentrations in Western Europe during the early 1980s following the 1979 Geneva Convention on Long-Range Transboundary Air Pollution (CLRTAP). The agreement initially aimed to tackle acid rain that is caused by SO2 and nitrogen oxides (NOx), brought together scientists and policymakers to solve complex transboundary air pollution problems, and committed (European) countries to substantially reduce sulfur dioxide emissions. Key policies motivated by CLRTAP included a mandate for sulfur scrubbers in coal-fired power plants——a major source of SO2. These national regulations led to significant local improvements in air quality and had a significant impact on infant and elderly mortality (Barreca, Neidell, and Sanders, 2021; Luechinger, 2014). However, the long-term effects of SO2 exposure on human capital and adult health remain poorly understood. Since SO2 is a precursor to fine particulate matter (PM2.5), its impact may extend beyond short-term health outcomes, potentially shaping individuals' long-term health and human capital accumulation. Moreover, because airborne SO2 can travel long distances, the benefits from regulation extend beyond areas around the pollution sources, lowering transboundary pollution even in economies less reliant on fossil fuels and thus not bearing the abatement costs.

Our study focuses on Norway and leverages several unique advantages of this setting. First, access to rich individual-level data covering the entire population allows us to link local air pollution levels over two decades to more than a million children, whom we can track until the age of 30. These individual-level data include measures of human capital, labor market, and health outcomes, enabling us to assess the impact of childhood pollution exposure on a comprehensive set of adult outcomes critical to individual well-being. Our novel measure of transboundary SO2 pollution is based on data collected by the Norwegian Environmental Agency as part of the Program for Monitoring and Evaluation of the Long-range Transmission of Air Pollutants in Europe (EMEP). These data provide annual

pollution levels at the municipal level over several decades. Using multiple independent data sources, we confirm that geographic and temporal variations in the SO2 pollution measures align with observed changes in other measures related to acid rains, such as water quality in lakes and freshwater fish stocks.

Second, we address the challenge of correlated unobservables by focusing on children born in the decade before and after the signing of the 1979 Convention on Long-Range Transboundary Air Pollution. This convention established a framework for international cooperation to reduce sulfur emissions and their transboundary fluxes by at least 30% by 1993 in Western Europe. Since transboundary pollution was the primary source of SO2 exposure for the general Norwegian population, the convention led to a sharp decline in children's SO2 exposure between 1970 and 1990. Moreover, substantial geographic differences in pre-convention SO2 levels——primarily driven by wind patterns, weather systems, and topography——resulted in considerable variation in pollution reduction across municipalities, which we leverage in our empirical analysis.

Third, air pollution and traffic density in Norway have historically been low compared to other countries. Additionally, with 99% of electricity production at the time coming from hydropower, Norway contributed minimally to global SO2 pollution in the 1970s. As a result, the Norwegian economy was largely unaffected by many of the new regulations under the Convention on Long-Range Transboundary Air Pollution, which primarily targeted coal-fired power plants. Consequently, Norway experienced a substantial drop in transboundary pollution exposure without incurring the implementation costs or the (unemployment-related) economic disruptions associated with industrial restructuring. This is particularly important, as environmental regulations often result in job losses (Greenstone, 2002; Walker, 2013), which can independently impact health and child outcomes (Sullivan and von Wachter, 2009; Rege, Telle, and Votruba, 2011; Oreopoulos, Page, and Stevens, 2008). This unique asymmetry of benefitting from the reform without bearing the costs allows us to identify the causal impact of pollution at exposure levels that remain prevalent in many countries today.

Finally, we introduce a novel framework to identify the ages at which childhood exposure to pollution is most harmful. This framework leverages plausibly exogenous and sharp variation in early-life pollution exposure, combining a mover's design (Chetty and Hendren, 2018) with a baseline difference-in-differences approach based on air quality regulations. Specifically, our design exploits differences in the age at which children relocate——both from high- to low-pollution areas and vice versa——allowing us to estimate age-specific effects of pollution exposure on human capital development. While this estimation strategy permits unrestricted correlations between age-specific effect estimates and other model parameters, we impose certain restrictions, including the additive separability of age-specific

effects, exogenous mobility, and exogenous moving ages. We provide a detailed discussion of these identifying assumptions, along with empirical evidence supporting their validity in our setting.

We find that cohorts born after 1980 in municipalities experiencing significant reductions in pollution exposure saw greater improvements in education, earnings, and health outcomes than those born in municipalities with minimal initial exposure. Specifically, individuals from these municipalities exhibited a measurable increase in years of schooling, alongside a statistically significant rise in the likelihood of obtaining a college degree. For men, for whom additional data from the military enlistment are available, we find a notable reduction in the probability of being classified as having low cognitive ability. In addition, our findings suggest that exposure to the policy-induced pollution decline led to a modest yet statistically significant decline in the likelihood of being diagnosed with asthma, heart disease, and thyroid disease among cohorts from municipalities that experienced high levels of sulfur pollution in the 1970s. These findings highlight the long-term human capital and health benefits associated with reduced transboundary air pollution. Importantly, the results of our mover's design difference-in-differences indicate that early exposure is the most crucial independent of the duration of exposure.

Our findings contribute to the broad literature on the effects of childhood pollution exposure on human capital, labor market outcomes, and health. We make four key contributions. First, we examine a wide range of adult outcomes using a consistent approach and population-wide data, at exposure levels that remain relevant today. While previous research has credibly estimated the long-term impact of childhood pollution exposure on human capital or mortality (e.g., Grönqvist, Nilsson, and Robling, 2020; Isen, Rossin-Slater, and Walker, 2017; Black et al., 2019; Barreca, Neidell, and Sanders, 2021), our study extends this by providing comparable estimates for health outcomes measured at similar ages as income. This allows us to better understand the specific health dimensions that may drive the effects on human capital and labor market outcomes. Second, we speak to the literature on international spillover effects of air pollution that primarily focuses on Asian dust (Jia and Ku, 2019; Heo, Ito, and Kotamarthi, 2023) or nuclear fallout from radiation accidents or weapon testing (Almond, Edlund, and Palme, 2009; Black et al., 2019) and extend it by studying transboundary SO2. Third, we provide more evidence on the importance of SO2 regulations on human health outcomes by extending the current work focusing on mortality (Barreca, Neidell, and Sanders, 2021; Luechinger, 2014). Last, and most importantly, we introduce a novel framework to estimate age-specific effects of pollution exposure. Our findings indicate that the in utero period and infancy are more critical than the preschool years. Crucially, this framework is not limited to the context of pollution and can be applied to

identify sensitive periods for other types of investments or shocks, and adds an important tool to the growing literature studying movers to understand health or environmental inequalities (Finkelstein, Gentzkow, and Williams, 2016, 2021; Deryugina et al., 2019; Lyubich, 2021).

2 Background and Measurements

2.1 Convention on Long-Range Transboundary Air Pollution

Achieving international consensus on transboundary environmental issues has historically proven challenging. However, the Convention on Long-Range Transboundary Air Pollution (CLRTAP or Convention, hereafter), established in 1979, is a notable yet often underrecognized success in mitigating transboundary air pollution. Initially signed by 32 Western European nations, alongside the United States and Canada, CLRTAP was initially designed to combat acid rain. Some consequences of acid rain were clearly visible in the 1970s in the form of forest dieback, acidity levels in lakes endangering aquatic wildlife, or dirty and deteriorated surfaces of buildings, which led to a strong political movement. The Convention aimed to mitigate acid rain by controlling sulfur dioxide (SO2) emissions. Among the goals of the Convention was the reduction of sulfur emissions or their transboundary fluxes by at least 30 percent by 1993. Most participating countries reported reaching the goal (Dai, 2005). One of the key policies motivated by CLRTAP was the German Federal Emission Control Act, which mandated scrubbers in coal-fired power plants——a major source of SO2 (Luechinger, 2014). Note that European countries were among the first to regulate SO2 emissions. In the US, the United States Acid Rain Program is part of the Clean Air Act Amendments of 1990 and introduces a cap-and-trade regulation to control SO2 emissions in the US.

The success of the Convention is largely attributable to its strong foundation in scientific coordination, which has enabled evidence-based policymaking and the implementation of targeted regulatory interventions. A key pillar of this scientific framework is the European Monitoring and Evaluation Programme (EMEP), which plays a central role in data collection, analysis, and modeling to support the Convention's objectives. The EMEP data provide the empirical basis for international negotiations and the formulation of legally binding protocols under the Convention and these data are also one primary source of pollution data we use for this paper (see Section 2.2).

Besides tangible and measurable air pollution reductions both locally and transnationally, the Convention has yielded substantial health and environmental benefits. Hence, CLRTAP

serves as a compelling example of effective international environmental cooperation that can lead to substantial improvements in air quality and public health.

Over time, the Convention has evolved into a paradigmatic framework for international environmental governance, effectively bridging the gap between scientific research and policymaking. The Convention has since expanded its scope, incorporating over 51 member states and introducing eight protocols to address various environmental challenges associated with industrialization, agricultural intensification, and fossil fuel consumption. These protocols have targeted pollutants such as ground-level ozone, black carbon, persistent organic pollutants, heavy metals, and particulate matter, with policy measures grounded in rigorous scientific assessments of their health and ecological impacts.

2.2 Measuring Exposure

To measure pollution exposure, we utilize novel data from the Norwegian Institute for Air Research (NILU), which has monitored sulfur deposition since 1971 through a nationwide monitoring system as part of the European Monitoring and Evaluation Programme (EMEP). Both EMEP and NILU conduct detailed monitoring of air and precipitation composition while employing advanced modeling techniques to simulate the long-range dispersion and deposition of airborne pollutants across national borders. Monitoring sulfur deposition has been crucial for understanding the causes, impacts, and trends in acid rain and has played a key role in shaping policy debates.

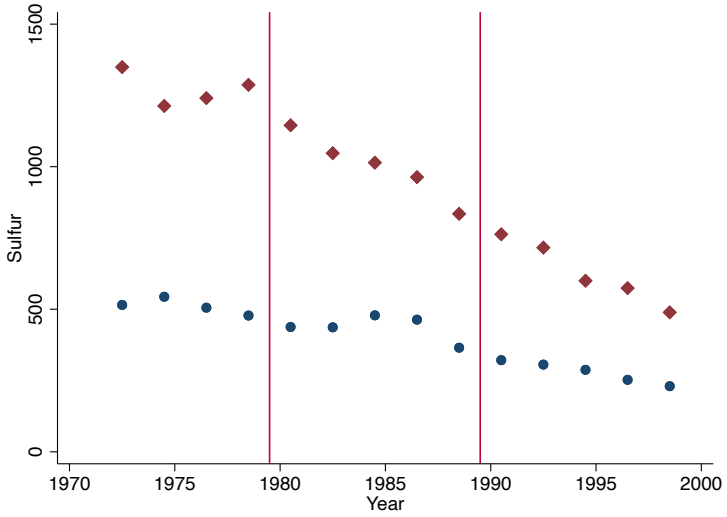
NILU collects pollution data from approximately 30 sites across Norway. The specific locations of these sampling sites and the collection procedures are strictly defined. However, the sampling locations have not always remained identical across the years. To construct a measure of acid exposure for each period and municipality, our main analysis relies on NILU's deposition map for the period 1978–-1982. NILU estimates acid deposition levels by interpolating data from fixed monitoring sites to a regular grid using the kriging method.[2] We estimate acid exposure by assigning each municipality the deposition level from the geographically closest grid point.

It is worth noting that concentrations of particulate matter (e.g., PM2.5 or PM10) are now the most commonly used measures of ambient air quality. However, comprehensive data on particulate matter was not available in the 1970s. Measures of PM2.5 based on satellite imaging generally date back only to 2000 (see, e.g., Currie, Voorheis, and Walker, 2023).

To illustrate the effect of the Convention on transboundary sulfur dioxide (SO2) deposi-

[2]Kriging is a statistical interpolation technique used to estimate unknown values based on neighboring observations. A commonly used alternative in the economics literature—inverse distance weighting—is a special case of kriging.

Figure 1. Annual Transpoundary Sulfur Dioxide Pollution by Region 1970-2000



tion in Norway, we analyze data from five monitoring stations that have been continuously collecting sulfur deposition measurements since 1972. Eight of these stations are located in southern Norway, while three are situated in the north. The precise locations of these monitors are displayed in Figure C1 in Appendix C.

Figure 1 presents annual sulfur dioxide deposition levels. The first vertical line, in 1980, marks the introduction of the Convention, while the second vertical line indicates the last cohort in our individual-level data. In southern Norway, represented by red diamonds, annual sulfur deposition declined from approximately 1,300mg/m^2 to 800mg/m^2 by 1990—–a reduction of about 30%, aligning with the initial target of the program.

In contrast, sulfur deposition levels recorded by the northern monitoring station (blue dots) were consistently lower. This pattern aligns with the fact that prevailing winds carried the majority of sulfur emissions from continental Europe and the United Kingdom toward southern and western Norway. While sulfur deposition also declined in the north, the magnitude of the reduction was significantly smaller. By 2000, the pollution gap between northern and southern Norway had narrowed to just 15% of the initial difference observed in the 1970s.

2.3 Sulfur Exposure and Health Consequences

Acid rain was one of the most critical environmental issues during the last decades of the twentieth century. Initially, the research focused on SO2's impact on forests, fisheries, and historical buildings. Little attention was given to its direct impact on human health. How-

ever, acid rain has significant adverse effects on human health, though not through direct exposure to acidic precipitation or contaminated water. Instead, the primary health risk arises from inhaling pollutants associated with acid rain. These airborne pollutants, primarily sulfur dioxide (SO2) and nitrogen oxides (NOx), are byproducts of fossil fuel combustion and industrial emissions. Exposure to these pollutants—especially over time—may cause irritation to the eyes and skin, respiratory illnesses, including chronic bronchitis, pneumonia, and asthma, cardiovascular problems, lower birth weight, and lung cancer. Moreover, once released into the atmosphere, these compounds undergo chemical transformations, leading to the formation of fine particulate matter such as PM2.5, which has been shown to pose serious risks for respiratory and cardiovascular health and cognition in the short run (add citations). Moreover, prolonged exposure to PM2.5 can lead to oxidative stress and inflammation in both the lungs and the body, potentially resulting in vascular issues, atherosclerosis, and heart rhythm disturbances. Continuous, low-level exposure to PM2.5 has effects similar to those of long-term cigarette smoking. Hence, policies lowering the exposure to SO2 as intended by CLRTAP should be beneficial for the health and human capital of affected cohorts. Several studies documented the link between acid rain-related pollution and various health conditions. For example, Barreca, Neidell, and Sanders (2021) documented persistent mortality effects of the Acid Rain Program in the United States for those 35–64 years of age, and (Luechinger, 2014) exploits the mandated desulfurization at power plants to document that lowering the SO2 pollution also lowered infant mortality in Germany.

While Barreca, Neidell, and Sanders (2021) and Luechinger (2014) examine populations located relatively close to pollution sources, our study estimates the consequences of pollution exposure for individuals situated much farther from the regulated plants. To validate the relevance of pollution levels in our setting, we replicate some of their findings on contemporaneous mortality outcomes, demonstrating that the pollution levels we observe had measurable short-term effects.

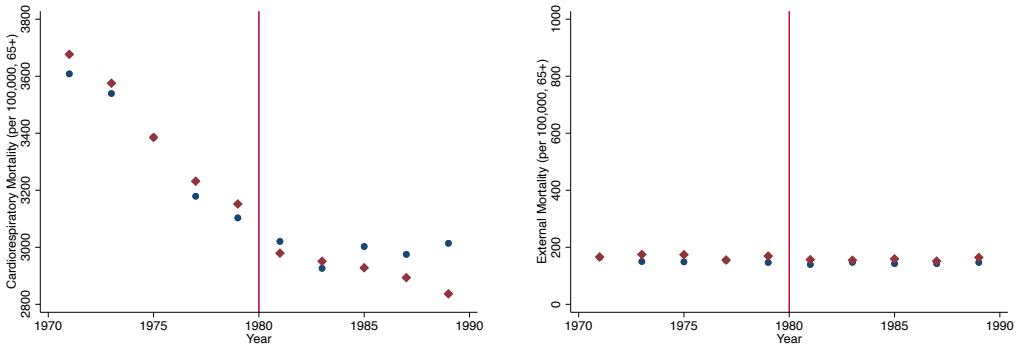
Using the Norwegian Cause of Death Registry, we analyze cardiorespiratory-related deaths, as these are the outcomes most likely to be influenced by pollution exposure. Age-adjusted mortality rates are constructed following the methodology of Barreca, Neidell, and Sanders (2021).[3] To account for potential confounding factors, we conduct a falsification test using external causes of death, such as accidents or homicides. These causes may be affected by factors like selective migration or economic fluctuations but should not be directly influenced by pollution-related health improvements.

Panels (a) and (b) of Figure 2 present the raw plots for cardiorespiratory and external death rates by pre-policy sulfur levels. Red diamonds indicate the average biannual car-

[3] A more detailed description of the data and analysis can be found in Appendix A.

diorespiratory mortality rates for municipalities with sulfur exposures above the median, while blue dots represent municipalities with lower exposure levels. Over time, cardiorespiratory mortality rates declined in both groups, although the gap between them gradually narrowed. Following the introduction of the Convention, mortality rates declined more rapidly in the south, where municipalities had experienced higher sulfur exposure prior to the policy. In contrast, external mortality rates remained stable throughout the period. Unlike cardiorespiratory mortality, external mortality trends moved in parallel for both groups across the entire period. These patterns align with the expectation that sulfur pollution affects mortality rates, but only for causes directly linked to pollution exposure.

Figure 2. Mortality per 1000,000 Ages 65+ by Pre-Policy Pollution Levels 1970-2000



(a) Cardiorespiratory Mortality **(b)** External mortality

3 Empirical Strategy

This study examines the long-term effects of early childhood exposure to airborne pollution on a comprehensive set of adult outcomes. We begin by implementing a cohort-based difference-in-differences analysis, leveraging the 1979 Convention on Long-Range Transboundary Air Pollution, which led to a reduction in transboundary sulfur fluxes. After establishing these long-term consequences, we focus on the age-specific effects of pollution exposure during childhood, identifying critical periods of vulnerability. To achieve this, we analyze children who relocated early in life ("movers") to disentangle the effects of exposure at a specific age from the effect of the overall duration of exposure.

3.1 Basic Design

Our initial analysis leverages the geographic variation in transboundary SO2 exposure before the Convention and the subsequent convergence in imported sulfur levels across regions.

Our identification strategy follows a similar approach to that of Acemoglu and Johnson (2007) and Bleakley (2007). Specifically, we compare long-term economic outcomes across municipalities with varying levels of pre-policy transboundary pollution by estimating the following reduced-form relationship:

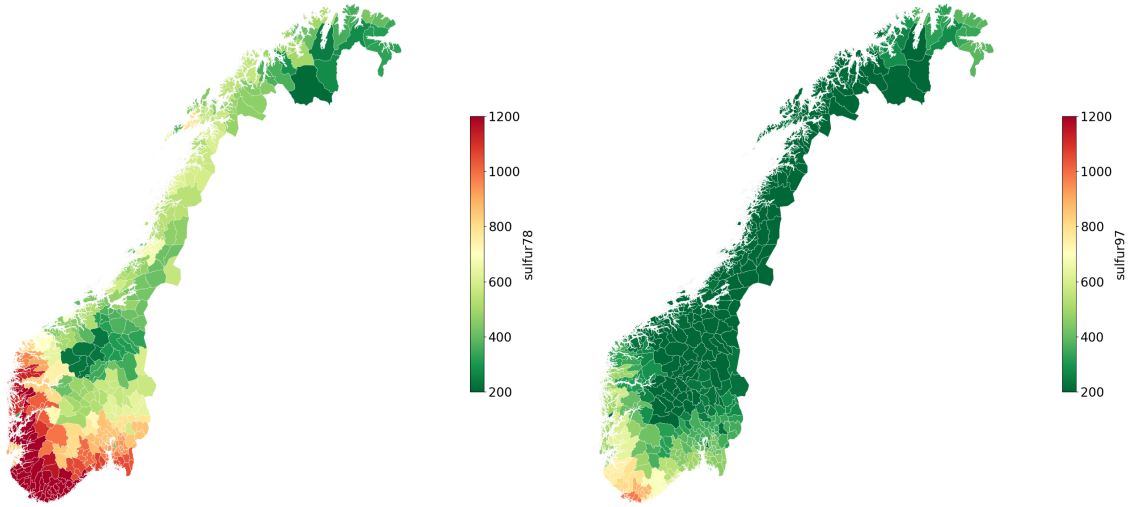
$$Y_{ijc} = \beta_1 Sulfur_j^{78} \times Post_c + X_{ijc}\rho + \delta_j + \gamma_c + \epsilon_{ijc}, \tag{1}$$

where Y_{ijt} denotes the outcomes of interest recorded in adulthood for individual i born in municipality j in year (cohort) c. The main coefficient of interest, β_1, is the interaction term of the pre-policy transboundary pollution ($Sulfur_j^{78}$) in municipality j and a dummy variable ($Post_c$) indicating whether cohort c was born after the Convention. Therefore, β_1 measures the difference in birth cohort trends in outcome Y across individuals living in municipalities with different levels of pre-policy pollution levels. The specification includes the municipality of birth fixed effects (δ_j) and year of birth fixed effects (γ_c). Included in X_{ijc} are individual controls for gender and fathers' education. Standard errors are clustered at the municipality of birth level (Bertrand, Duflo, and Mullainathan, 2004) to account for serial correlation in the outcomes.

As highlighted in Bleakley (2007), identifying the impact of pollution reduction depends on three key factors: (i) regional variation in pollution exposure, (ii) the immediate effectiveness of the policy intervention, and (iii) the exogeneity of the program's implementation. During the 1970s, there was considerable regional variation in imported sulfur dioxide pollution. Figure 3 depicts the annual deposition of transboundary sulfur dioxide across municipalities in 1978 to 1997. Due to prevailing wind patterns and the geographical landscape, the highest sulfur deposition levels were typically concentrated in southern municipalities. However, this pattern was not uniform. For instance, the Lofoten area, despite being located in the north, experienced exceptionally high levels of transboundary pollution. This anomaly can be attributed to its position along a winding atmospheric pathway from the UK, further underscoring the substantial spatial variation in imported pollution across the country.

Our strategy capitalizes on the heterogeneity in transboundary pollution levels prior to the Convention. The central assumption is that municipalities with higher initial pollution experienced a greater reduction in pollution and, consequently, derived more substantial benefits from the SO2 regulations compared to municipalities with lower initial pollution levels. This variation in potential benefits forms the basis for a treatment and control framework. This assumption is supported by the evidence presented in Figure 4, which illustrates the municipality-specific reduction in imported pollution between the program years and 1960, plotted against pre-policy pollution levels. The figure reveals a convergence

Figure 3. Geographic Variation in SO2 Deposition Pre- and Post-Policy



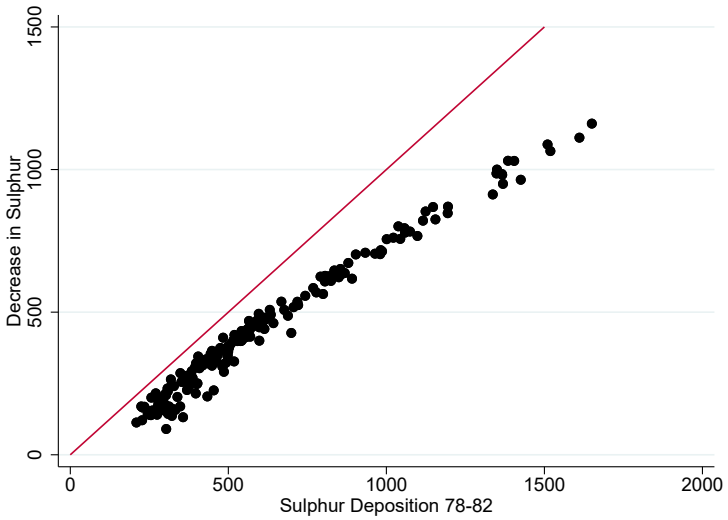
1978 1997

in imported pollution levels across municipalities over time, which aligns with our hypothesis.

Furthermore, the identification strategy relies on the rapid success of international efforts to reduce sulfur emissions. As previously noted, these efforts resulted in substantial reductions in sulfur emissions across Western European countries. The primary goal of the Convention, which was later formalized in an amendment known as the Helsinki Protocol, was to achieve a 30% reduction in sulfur emissions by 1993. While emission data is not available for all the countries participating in the Convention, several case studies examining individual countries provide consistent evidence of swift and effective action. For example, Luechinger (2014) studied sulfur dioxide concentrations in Germany, starting with data from 1985. His findings indicate a significant decline in SO2 concentrations in West Germany, from approximately 40 µg/m^3 to 20 µg/m^3, representing a nearly 50% reduction.[4] The key consideration for our identification, however, is whether the reduction in imported pollution in Norway was both rapid and consistent. Figure 1 supports this, showing a 30% decline in sulfur deposition between 1980 and 1990. Specifically, average deposition levels, measured by four monitoring stations in the south, decreased from 1000 mg/m^2 to 700 mg/m^2. In the north, where transboundary air pollution was less prevalent, deposition levels fell from 500 mg/m^2 to 400 mg/m^2. These patterns highlight the consistent reduction in imported pollution across Norway during the intervention period. Notably, both large and small cities are present in both high- and low-pollution areas.

[4]In East Germany, a similar decline began in 1991, following the fall of the Berlin Wall.

Figure 4. Convergence in SO2 Deposition



Unobserved characteristics that vary by municipality of birth and birth year and correlate with both pollution exposure and later-life outcomes could pose a potential threat to our identification strategy. To address this concern, we include specifications that account for trends in public infrastructure. Specifically, we control for pre-existing local trends by incorporating variables such as the number of inhabitants per doctor in each municipality and the student-teacher ratio in the year an individual enters school. These controls aim to capture variations in access to healthcare and education that may influence outcomes independently of pollution exposure. Ultimately, the validity of our identification strategy hinges on the common trend assumption. To validate this assumption, we present an event-study analysis in Figure 7 in the result section. This more flexible specification allows us to assess whether trends in outcomes were similar across municipalities prior to the intervention, thus strengthening the credibility of our findings.

The identification strategy faces additional challenges. First, our design, which relies on a "dose" or varying treatment intensity, deviates from the canonical difference-in-differences (DiD) approach. Unlike the standard DiD framework, which compares outcomes between treated and untreated groups (difference one), our design evaluates variations in treatment intensity. This approach offers several advantages. It allows for evaluating treatments received by all units, enables the testing of theoretical predictions, and provides insights into the effects of changes in treatment intensity—outcomes that are often more relevant in practice. However, recent literature (Callaway, Goodman-Bacon, and Sant'Anna, 2024) has highlighted potential issues with this design, particularly when employing a two-way fixed effects estimator in the presence of effect heterogeneity across treatment doses. Specifically,

the two-way fixed effects estimator identifies a weighted integral of the average treatment effect parameters, which can obscure heterogeneity and lead to biased estimates. To address these concerns, we employ two complementary approaches. First, we binarize pre-policy imported pollution exposure, enabling us to estimate a canonical difference-in-differences model. Second, we apply a nonparametric DiD estimator, similar to the approach proposed by Callaway, Goodman-Bacon, and Sant'Anna (2024), to account for potential effect heterogeneity and ensure robust estimation of treatment effects.

A further challenge to our identification strategy is the potential for estimates to be influenced by mean reversion. Specifically, income and years of education for post-campaign cohorts might increase independently of the direct effect of pollution on human capital development if pre-policy cohorts exposed to high pollution experienced low productivity due to a mean-reverting shock. To address this concern, we incorporate controls to account for mean reversion. Using data from the 1970 Census, we include the number of individuals with secondary education in each municipality, the natural logarithm of average earnings in each municipality, and the interaction of these variables with individuals' year of birth. These controls help isolate the effects of pollution reduction from any underlying mean-reverting dynamics.

3.2 Mover's Design Difference-in-Differences

To date, our analyses have relied on cohort-based comparisons, operating under the assumption that early-life exposure to air pollution represents a critical "sensitive period" for its impact. As brain plasticity and cell division are very high in utero and in early childhood, it is likely that pollution exposure is harmful to fetuses and small children. This approach has been widely adopted in earlier studies on air pollution exposure both in early childhood (see, e.g., Isen, Rossin-Slater, and Walker, 2017; Klauber et al., 2024) or the in utero period (see, e.g., Currie and Walker, 2011; Almond, Edlund, and Palme, 2009; Black et al., 2019). Focusing on a pre-specified sensitive period is compelling and sometimes practical for application. However, this simplification limits our ability to examine the age-specific damage functions and understand whether and how periods of pollution exposure interact. That is, the basic design does not allow us to distinguish whether the timing or the duration of the exposure matters and assumes that pollution-related health damages at one stage do not change the vulnerability to pollution exposure at subsequent stages.

In this paper, we present a novel framework that leverages plausibly exogenous variation in early-life pollution exposure using a mover's design (Chetty and Hendren, 2018), combined with exogenous policy-induced variation in pollution exposure. The mover's design

exploits differences in the age at which children relocate—both from high- to low-pollution areas and vice versa—to estimate age-specific effects of pollution exposure on human capital development. While the mover's design is a powerful tool, it has faced criticism for relying on strong assumptions regarding the timing of mobility. To address this, our framework embeds the mover's design into a natural experiment setting to relax some of these assumptions and derive testable implications that can validate our modeling assumptions.

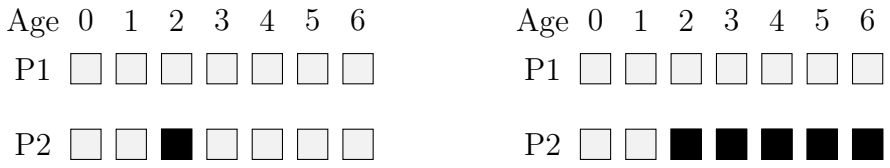
In practice, our approach combines the mover's design with our baseline difference-in-differences framework, implemented through a two-way fixed effects model. This model offers the advantage of accommodating unrestricted correlations between age-specific effect estimates and other parameters in the model. However, it imposes certain restrictions on the interactions of age-specific effects, which can introduce bias into the estimates. For the estimates derived from this framework to be unbiased, three key assumptions must hold: (1) additive separability of age-specific effects, (2) exogenous mobility (parallel trends), and (3) exogenous moving age (parallel trends). In the following subsections, we outline the identifying assumptions, describe how we can test them, and detail the empirical specifications.

3.2.1 Identifying the Age-specific Effects

Our objective is to determine the age-specific effects of pollution exposure. That is, we aim to estimate the impact on a child's potential outcomes, on average, if they were exposed to pollution, specifically at age m during childhood and no other year. A mover's design, where a child moves once at age m to a high-pollution area, does not enable us to identify this, but it allows us to estimate the cumulative effects of pollution exposure starting at age m. Two hypothetical experiments illustrate these definitions. Experiment 1 randomly assigns children to pollution exposure only at age m. Experiment 2 randomly relocates children to a different location, starting at age m. Figure 5 illustrates this difference. While random assignment ensures unbiased estimates in both experiments, the results from Experiment 1 sub-figure 5a will yield the age-specific effects for age m, and the results from Experiment 2 in sub-figure 5b will measure the cumulative effects starting at age m. The information captured by the age-specific effects and the cumulative effects is not equivalent, even if unbiased estimates can be identified using randomly assigned children. Below, we discuss the assumptions we must impose to recover the age-specific exposure effect from the cumulative effect estimated using a mover's design.

Our objective is to determine the age-specific effects of pollution exposure. That is, we aim to estimate the impact on a child's potential outcomes, on average, if they were exposed to pollution specifically at age m during childhood and no other year. A mover's design, in which a child moves once at age m to a high-pollution area, does not enable us to identify

Figure 5. Age-Specific and Cumulative Effects



(a) Age-specific Effect **(b)** Cumulative Effect

this directly but allows us to estimate the cumulative effects of pollution exposure starting at age m. Two hypothetical experiments illustrate these definitions. Experiment 1 randomly assigns children to pollution exposure only at age m. Experiment 2 randomly relocates children to a different location, starting at age m. Figure 5 illustrates this difference. While random assignment ensures unbiased estimates in both experiments, the results from Experiment 1 yield the age-specific effects for age m, whereas the results from Experiment 2 measure the cumulative effects starting at age m. The information captured by age-specific effects and cumulative effects is not equivalent, even if unbiased estimates can be identified using randomly assigned children. As mentioned above, previous literature often suggests that age-specific effects are estimated by strictly focusing on exposure in utero or during early childhood periods. Still, it usually does not offer an identification strategy to rule out that cumulative effects and not age-specific effects are estimated. Below, we discuss the assumptions we must impose to recover the age-specific exposure effect from the cumulative effect estimated using a mover's design.

Assumption 1: Additive Separability of Age-Specific Effects.

A core modeling assumption is that long-term outcomes—such as schooling—are additively separable in age-specific effects. In the context of pollution exposure, estimating the cumulative damage over childhood is key. If better air quality provides a positive treatment in each childhood year, the key determinants of outcomes would be both age-specific effects and the total duration of exposure. However, the additive separability assumption imposes a significant restriction by not allowing for interactions between age-specific effects. Such interactions could manifest in two ways: damages from exposure at different ages could either reinforce or compensate for each other.

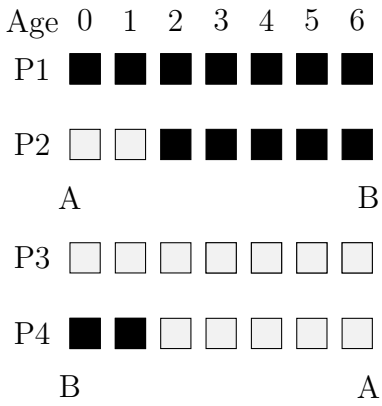
The first scenario, known as dynamic complementarities, suggests that children are more vulnerable to pollution exposure later in childhood if they have already been exposed in an earlier period. In this case, cumulative effects exceed the sum of individual age-specific

effects, as positive interaction terms amplify the harm. For example, consider two children moving to a heavily polluted area: one from a clean environment and the other from a polluted one. Dynamic complementarities would suggest that the child from the more polluted area would experience greater damages from post-move exposure due to heightened vulnerability. The second scenario, the critical age model, suggests that while pollution is harmful at all ages, exposure in a single year may be sufficient to cause full, long-lasting damage. Suppose two children are born—one in a mildly polluted area and another in a heavily polluted area. While the first child has a healthy respiratory system, the pollution that the second child is exposed to results in a disease-prone respiratory system. If both move to a highly polluted area after turning one year old, the first child will suffer negative effects, whereas the second child, having already experienced damage in the first year, will not be further affected. These two examples highlight the limitations of assuming additive separability and underscore the need to formulate testable implications for interaction effects.

Fortunately, our setting allows for empirical testing of interaction effects. Specifically, we investigate whether interactions exist by examining the *direction* of moves. We compare whether absolute changes in long-term outcomes are the same for moves from high- to low-pollution areas and from low- to high-pollution areas. This approach parallels the method used by Card, Heining, and Kline (2013), who tested for interaction effects between firm-specific and individual-specific factors.

To illustrate why testing for symmetry provides evidence of interactions in the impact of air pollution exposure at different ages, consider the stylized example in Figure 6. There are four groups of individuals (P1, P2, P3, and P4), each residing in either polluted place A or the less polluted place B.

Figure 6. Asymmetry



- P1 remains in polluted place A throughout childhood, accumulating an exposure effect of $\beta_{0..6}$.

- P2 moves from the less polluted to the more polluted place in period 2, with a cumulative effect of $\beta_{2..6}$

Under additive separability, the age-specific effect γ_2 for period 2 is simply the difference between $\beta_{0..6}$ and $\beta_{2..6}$. However, if dynamic complementarities exist—where early exposure increases later vulnerability—this difference cannot isolate the age-specific effect.

To address this, we analyze moves in the opposite direction:

- P3 remains in the low-pollution area throughout childhood, accumulating $\beta_{0..0}$ in exposure.
- P4 moves from the polluted to the less polluted area in period 2, with a cumulative effect of $\beta_{0..2}$.

If no interaction effects exist, the absolute difference between $\beta_{0..0}$ and $\beta_{0..2}$ should also yield γ_2, and estimates from high- to low-pollution moves should be comparable in magnitude to those from low- to high-pollution moves. Our data and framework allow us to empirically test whether these estimates are equivalent, which would suggest no interaction effects, or whether we can reject the assumption of additive separability.

Note that the test is not only useful for justifying the assumption of additive separability but also for distinguishing between a scenario with dynamic complementarities and the critical age model.

In section , we present empirical evidence that there are no asymmetries by direction of moves and that the additive separability assumption is likely not violated.

Assumption 1 allows us to recover age-specific exposure effects from cumulative exposure effects. However, this assumption alone does not ensure that cumulative exposure effects are estimated without bias when using observational data. As emphasized in the movers' design literature, the act of moving itself may be endogenous. To address this concern, we draw on insights from the movers' design literature, leveraging variation in moves between high- and low-pollution areas at different ages within observational data. To approximate random assignment, we introduce two additional assumptions for identification.

Assumption 2: Exogenous Mobility (Parallel Trends).

Another key identifying assumption for estimates from a mover's design to be unbiased is that moves are conditionally exogenous. In other words, the decision to move and the choice of destination must not be systematically related to unobserved factors that influence long-term child development outcomes. This would clearly be violated if, for example, only

families with a high socioeconomic background move or if only poorer families move from low-pollution areas to heavily polluted places. While this is a strong assumption, it enables us to compare movers and stayers to identify the cumulative impact. Our data also allow us to test whether movers are selected and whether family background predicts the direction of the move.

Moreover, we can integrate the mover's design regressions with the natural experiment framework discussed in Section 3.1. This provides us with testable implications by examining parallel trends in family background through event study figures.

Nevertheless, this assumption may still be overly strong in practice, as we aim to avoid relying solely on comparisons between movers and stayers. Instead, we adopt a slightly weaker assumption, following a similar approach to Chetty and Hendren (2018), by imposing specific restrictions on the timing of the move.

Assumption 3: Exogenous Moving Age (Parallel Trends).

Assumption 3 permits the possibility that families moving to better areas may differ systematically from those moving to worse areas. However, it requires that the extent of such selection does not vary with the child's age at the time of the move. That is, on the one hand, the assumption is not violated if families with high socioeconomic backgrounds are more likely to move from polluted to clean places as long as this is the same for all age periods. On the other hand, the assumption would be violated if, for example, affluent families move when their kids are one year old and poor families move at age six. Under this assumption, combined with Assumption 1, we can obtain consistent estimates of exposure effects from Equation 2, even with observational data, because the selection effect cancels out when estimating the exposure effect.

Similar to the discussion above for Assumption 2, we also exploit the policy reform to weaken the assumption a little and introduce testable implications. The combination of methods allows to estimate age-specific exposure effects even if there are varying selection effects based on the child's age at the time of the move, as long as the selection effects for each age at the move remain constant for children born before and after the Convention. This framework aligns with the common trend assumption commonly used in the difference-in-differences literature.

3.2.2 Specification

With the discussion above, we estimate analogous regression coefficients for children whose parents move at each age m from 0 to 6, using the following specifications:

$$Y_{ijkmc} = \sum_{m=0}^{6} b_m[I(m_i = m) \times \Delta Sul_{jk}^{78} \times Post_c] + \gamma_{mc} + \theta_{jkm} + \tau_{jkc} + \epsilon_{ijkmc} \tag{2}$$

where Y_{ijkmc} denotes the outcomes of interest recorded in adulthood for individual i born in municipality j in year c and lived in municipality k at age of 6. $I(m_i = m)$ is a dummy variable that indicates at which age an individual moved from municipality j to k. $\Delta Sulfur_{jk}^{78}$ measures the difference in sulfur exposure before the policy change. $Post_c$ indicates whether the individual was born before 1980. Our primary goal in estimation is to identify the age-specific effects (b_m). In the baseline model, we control for the age of moving by cohort fixed effects γ_{mc}, the age of moving by migration flow fixed effects θ_{jkm}, and cohort by migration flow fixed effects τ_{jkc}. θ_{jkm} ensures the comparison is always between children who move between the same places at the same age, and τ_{jkc} ensures the comparison is always between children who move between the same places in the same cohort. In contrast, γ_{mc} ensures the comparison does pick up the cohort difference and the difference caused by the move at different ages regardless of the moving direction. ϵ_{ijkmc} captures family inputs and other determinants of children's outcomes.

4 Data

We compile our data from various sources by linking aggregate data on sulfur dioxide exposure with individual administrative records. Our primary data source is the Norwegian Registry Data, a comprehensive administrative dataset covering the Norwegian population up to 2020. The dataset integrates information from multiple administrative registers, including the central population register, the family register, the medical birth registry, the education register, the tax and earnings register, military session data, and various healthcare registers. The data provide detailed information on birthplace and residence at each age, educational attainment, labor market status, earnings, birth weight, healthcare utilization, military session results, and a range of demographic variables, as well as family-related information.

4.1 Main sample

Our full analysis sample includes all children born in the 1970–1989 birth cohorts. We focus on children whose parents can be identified and for whom parental information is available. The earlier cohorts in our sample were born before the Convention, while the later cohorts were born during the implementation of policies aimed at reducing sulfur dioxide exposure. The central population register is the core dataset that allows us to link information from various registers using a unique personal ID and parental IDs. We use the municipality of birth to link pollution exposure data, as described in Section 2.2. The tax register also collects annual address information, which we use to verify the municipality of birth recorded in the birth register. In Section 6.2, we will further discuss how we utilize residence data at each age. Additionally, we extract birth weight information from the birth register to measure health at birth.

Since 1979, educational institutions have reported educational attainment directly to Statistics Norway on an annual basis, minimizing measurement errors due to misreporting. We use two measures of educational achievement: (1) an indicator variable for whether an individual received an academic high school diploma and (2) the total number of years of education completed. On average, 60% of men and 51% of women in our sample have pursued post-compulsory education. The average number of years of education is 15 for men and 14.4 for women.

IQ scores, weight, and height data are obtained from Norwegian military records. In Norway, military service was mandatory for men, meaning we have data for males only, typically recorded at age 18 or 19. The percentage of males completing military service remained high until 1989, when it declined from 90% to 70%. To reduce the impact of selection bias in military service, we exclude the 1989 birth cohort from our analysis.

Data on visits to General Practitioners (GPs) and Emergency Rooms (ERs) come from the Control and Payment of Health Refunds database (KUHR). Covering the period from 2006 to 2016, this database requires GPs and ERs to submit consultation records, including ICPC-2 codes, which classify patients' medical conditions and the nature of the care provided. Each ICPC code consists of an initial letter indicating the affected body system, followed by two numbers denoting the type of assessment or intervention. Using these ICPC-2 codes, we construct chronic condition indicators following Finkelstein, Gentzkow, and Williams (2021).[5] Our analysis focuses on cardiorespiratory-related conditions, including asthma, depression, chronic heart disease, and hypothyroidism.

Notably, we do not analyze labor market earnings, as the youngest individuals in our

[5]We map ICD-10 codes to ICPC-2 codes based on guidelines from the Directorate of e-Health in the Norwegian Ministry of Health and Care Services.

sample are not yet 30 years old by the end of our data period. This presents a challenge because Norway has the oldest student population in Europe, with a significant share (25%) of students over the age of 30. As a result, many individuals, particularly those in the post-reform cohorts, are still enrolled in education at the ages when labor market outcomes would typically be assessed. Furthermore, Bhuller, Mogstad, and Salvanes (2017) finds that college-educated workers experience more rapid earnings growth throughout most of their careers and have significantly higher annual earnings after age 27. Given these factors, we do not study the effects on earnings.

4.2 Movers Sample

Our main sample consists of approximately 1,064,647 children for whom we have annual residence municipality data from age 0 to 6. We divide the full sample into permanent residents (stayers) and movers. We define "stayers" as individuals whose residence remains the same between ages 0 and 6. "Movers" are individuals whose residence changed at least once before age 7. In our sample, 67% are stayers, meaning their municipality of residence did not change before age 6. The remaining 33% moved at least once during this period, with 56% of them (equivalent to 21% of the total sample) being one-time movers. Our mover design focuses exclusively on children who changed their municipality of residence once.

4.3 Municipality-level Data

As discussed in Section 3, we examine cardiorespiratory-related deaths. To construct this variable, we use the death register, which covers all deaths in Norway from 1967 to 2019 and includes information on the cause of death, classified according to ICD-10 codes. The data also provide details such as age at death and municipality of residence. Following Barreca, Neidell, and Sanders (2021), we construct age-adjusted mortality rates for three age groups— all ages, under 35, 35–64, and 65 and over— for each municipality. We focus on cardiorespiratory-related deaths, as these outcomes are most likely to be affected by PM2.5 exposure. As a falsification test, we also construct external death rates (e.g., accidents, murders), which may be influenced by factors such as selective migration and economic fluctuations but are unlikely to be affected by pollution-related health improvements.

4.4 Sample Statistics

Table 1 provides summary statistics of the outcome and control variables.

Table 1. Summary statistics

	Mean	Std. dev.	Obs
Panel A: full sample			
Schooling	14.7	3.41	1064647
With college degree	0.36	0.48	1064647
Low ability	0.17	0.38	481119
Panel B: mover sample			
Schooling	14.9	3.46	356224
With college degree	0.38	0.48	356224

5 Empirical Results

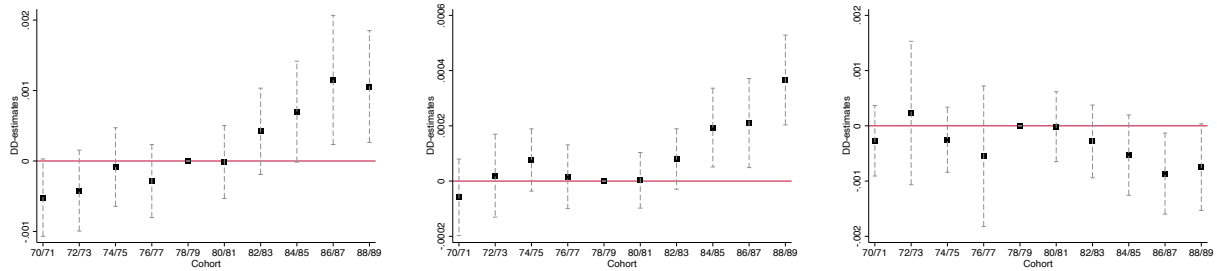
5.1 Long-Term Consequences on Human Capital Development

In this section, we analyze the long-term effects of exposure to the acid rain program. First, we present estimates from the event study

$$Y_{ijc} = \sum_{c=70}^{89} \zeta_c Sulfur_j^{78} \times 1[\tau = c] + X_{ijc}\rho + \delta_j + \gamma_c + \epsilon_{ijc}, \tag{3}$$

where ζ_c is a cohort-specific coefficient on acid rain exposure. This specification allows us to study the relationship between long-term outcomes and pre-policy and imported pollution across cohorts. Cohorts who were born in 1980 or earlier were conceived before the start of the policy.

Figure 7. Event studies



(a) College **(b)** Schooling **(c)** Low Ability

Figure 7 presents the event study figures. There are two main findings: First, we see that trends leading up to the Convention between treatment and control groups are nearly identical for all three outcomes. We view this as evidence in support of the identifying assumption in the model that trends in outcomes between treatment and control groups

would have evolved similarly except through the change in policy. Second, in the years after the Convention, we see a mean shift in years of schooling, college degrees, and low ability (only for males).

In the second step, we study the long-term effects of the change in pollution exposure by estimating Equation 1. Estimates of the variable of interest (β_1) are given for various outcomes and specifications in Tables 2 and 3. Each cell contains estimates from a separate regression. We find a significant increase in years of schooling among those cohorts exposed to the acid rain policy in municipalities with high levels of imported sulfur pollution from 1970 to 1979. In addition, the probability of graduating with a college degree increased significantly. For males, where we can access the military registry, we see a significant decrease in the probability of being classified as low ability. In particular, the coefficients indicate that, on average, a one-standard-deviation decrease in imported sulfur exposure leads to a three percent increase in the likelihood of going to college and a 0.5 percent increase in the years of schooling relative to the pre-policy mean.

Table 2. Education outcome

Outcome	(1) College	(2) Schooling	(3) Low Ability (only male)
Panel A:			
Post × Sulfur78	0.011***	0.058***	-0.0025*
	(0.0033)	(0.022)	(0.0015)
Mean of dep. var.	0.36	13.73	0.066
Observations	1064647	1064647	463631

We control for municipality and cohort fixed effects and controls for trends in local public infrastructure and local exposure to other shocks. As pointed out by Bleakley (2007), the estimated effect could also be caused by mean reversion across municipalities. That is, if a temporary shock led to high sulfur dioxide pollution levels and lower economic productivity before the implementation of the Convention, we could observe improvements in education, earnings, and health outcomes after the program, even in the absence of the direct effect of the pollution control measures on these outcomes. The specification, therefore, additionally controls for the average percentage of inhabitants with a high school diploma in each municipality in 1970 interacted with the year of birth.

5.2 Long-Term Consequences on Health

Beyond improvements in human capital, individuals may also experience long-term health benefits from reduced exposure to pollution in early life. These health gains could manifest

as lower risks of chronic diseases and improved well-being in adulthood.

To explore this, we leverage detailed administrative data from General Practitioners (GPs) and Emergency Rooms (ERs) recorded in the Control and Payment of Health Refunds database (KUHR). Our focus is on cardiorespiratory-related conditions, including asthma, depression, heart failure, and hypothyroidism. To the best of our knowledge, this study provides the first empirical evidence linking early-life exposure to pollution with long-term health outcomes.

Table 3. Health Outcomes

Outcome	(1) Asthma	(2) Depression	(3) Heart	(4) Thyroid	(5) Cancer
Panel A:					
Post × Sulfur78	-0.0029**	0.00076	-0.00030**	-0.0027**	0.000043
	(0.0014)	(0.0019)	(0.00013)	(0.0013)	(0.0019)
Mean of dep. var.	0.0927	0.348	0.00124	0.0755	0.00857
Observations	1072743	1072743	1072743	1072743	1072743

Our findings indicate a small but statistically significant reduction in the likelihood of being diagnosed with asthma, heart disease, and thyroid disease among cohorts exposed to the policy in municipalities with high levels of pre-policy pollution (see Table 3). Note that, due to data limitations, health measures for cohorts born before the policy capture outcomes at somewhat older ages. However, health issues such as asthma, heart disease, and thyroid disease are likely prevalent already at younger ages.

Additionally, we linked our data to the Cancer Register and found no causal evidence linking early-life pollution exposure to cancer diagnoses until age 30. This suggests that while pollution reductions had meaningful impacts on respiratory and cardiovascular health, they may not have influenced longer-latency diseases such as cancer within the observed time frame.

6 Effects of Age-Specific Exposure

So far, the results presented follow the design outlined in Section 3.2, where we compare different cohorts before and after the policy across varying pre-policy pollution levels. In this section, we leverage variation from movers to understand the mechanisms of early-life exposure and disentangle the importance of early exposure from the duration of exposure. Section 6.1 examines moving patterns using detailed administrative data to support the movers' design. We then present the results from the movers' design and discuss their

implications in Section 6.2. Finally, Section 6.3 provides additional validation exercises to assess our model further.

6.1 Moving Pattern

We begin our analysis by examining the relationship between migration patterns and policy effects, focusing on four key measures of migration: (i) whether children moved before age seven, (ii) the educational attainment of fathers in migrating households, (iii) whether the destination had lower pollution exposure, and (iv) whether the move occurred before age three. We estimate equation (1) for these four outcomes, with the last three analyses restricted to families who moved at least once before the child turned 9 years.

Figure X in the Appendix presents event study figures, while Table 4 summarizes the results. In the first column, we analyze whether families relocated across municipalities at least once before their child's seventh birthday. The data reveal that over one-third of children experienced a move, forming the sample used in columns 2–4. Fathers' education level, measured in years of schooling, serves as a proxy for socioeconomic status. Pollution change is defined as the difference in SO2 exposure between the birth municipality and the municipality of residence at age 6, using 1978 pollution levels (prior to the Convention). The third column investigates whether the destination was affected by the policy, while the fourth examines whether the timing of the move was influenced.

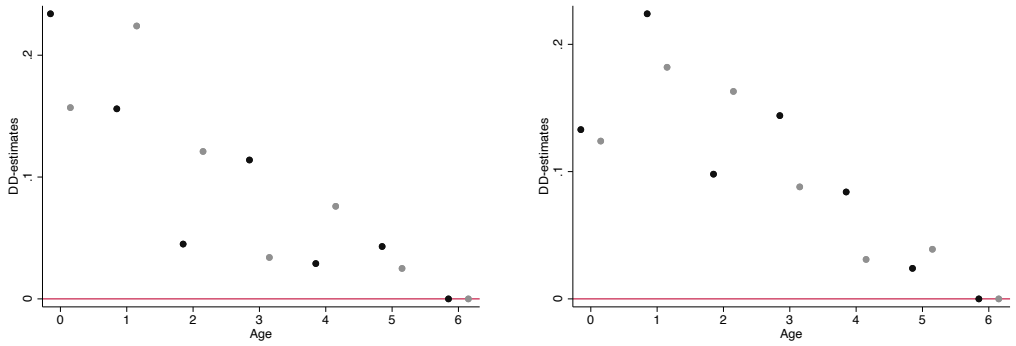
Table 4. Moving Pattern

Outcome	(1) move (0/1)	(2) fathers' schooling	(3) pollution change	(4) move before age 3
Panel A:				
Sulfur78 × Post	-0.0033	0.057	-0.000038	-0.0015
	(0.0042)	(0.071)	(0.0031)	(0.0058)
Mean of dep. var.	0.38	11.0	0.31	0.49
Observations	1064647	375808	375808	375808
Sample	all children	mover	mover	mover

6.2 Is Moving Age Exogenous?

We started the analysis by exploring the movers' design, focusing on the cohorts before and after the policy separately. If the exogenous moving age itself can remove all the selection bias and the assumptions used in Chetty and Hendren (2018), estimates from the pre-policy cohort will be equivalent to the estimates from our proposed model. Estimates from the post-policy cohort, therefore, can be thought of as a placebo test.

Figure 8. Age-Specific Effects from Movers' Design Only

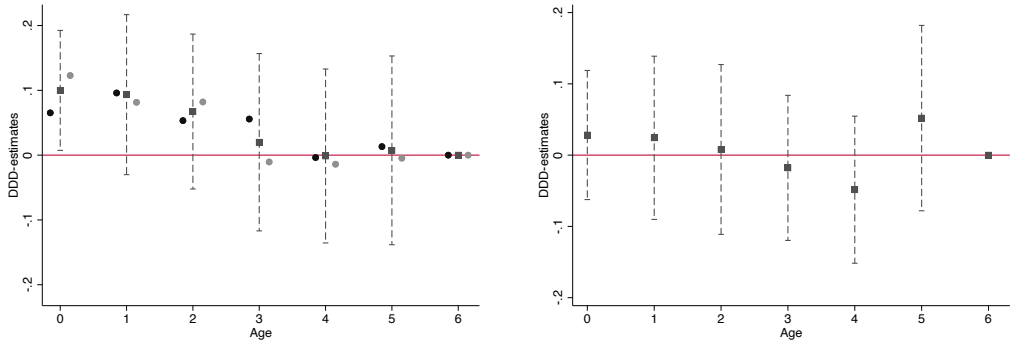


(a) Years of Schooling **(b)** Fathers' education

Figure 8, Panel (a) plot estimates of b_m from movers' design similar to Chetty and Hendren (2018), where we only explore the variation in the timing of the move. Black dots mark the estimates using pre convention data, while gray dots mark the estimates using post convention data. In Panel (b), we look at fathers' education, which is a placebo test similar to Eshaghnia (2023). The figure implies that fathers' education of the movers across municipalities in the destination decreases by 0.3% per year of age of children when their parents move.

6.3 Results From the Movers' Design in a Natural Experiment

Figure 9. Age-Specific Effects



(a) Years of Schooling **(b)** Fathers' Education

Panel (a) of Figure 9 plots estimates of b_m from Equation 2. As discussed in Section 3.2, looking at mover in two directions enables us to shed light on the interaction of exposures in different ages. Therefore, we also estimate the model for two sub-samples separately:

children who moved to an area with higher pollution ($\Delta \geq 0$) and children who moved in the opposite direction($\Delta \leq 0$). The estimates of b_m are marked using black dots and gray dots, respectively. This leads to the third pattern in Figure 9, which is not much difference can be observed from estimates from two sub-samples. Although estimates are not identical from the two samples throughout all ages, we can't statistically reject their equivalence. Under Assumption 1, this enables us to identify the age-specific impacts from the cumulative impacts. The results also suggest that the interaction of exposures, either complementarity or substitution, in different ages, is limited in the setting of the current paper.

6.4 Exposure Effects And Critical Age Effects

As pointed out in Chetty and Hendren (2018), a critical age model cannot be distinguished from an exposure effect model in a sample of one-time movers because a child's age at the move is perfectly collinear with his or her duration of exposure to the new area. Chetty and Hendren (2018) approached the question by exploring the families who moved multiple times, where the collinearity is broken. However, this requires a stronger assumption that families' moving decisions are not endogenous to these multiple movers. Using multiple times, movers also limits the external validity of the results, as they might be different from families that only moved once. Another practical challenge is that the sample size will be even smaller if we are interested in a shorter age period, which is 7 years in our setting.

Our approach by examining asymmetric moves opens another opportunity to distinguish between the critical age and exposure mechanisms, which is essentially a test of the complementarity of shocks at different ages. For example, suppose that moving to a better neighborhood improves a child's network of friends with a probability that falls with age at the move and that once one makes new contacts, they last forever. In this model, neighborhood effects would decline with a child's age at move (as in Figure IV), but the duration of exposure to a better area would not matter for long-term outcomes. Alternatively, if better neighborhoods offer positive treatment (such as better schooling) in each year of childhood, the key determinant of outcomes would be the total duration of exposure rather than the specific age at which a child moves.

7 Conclusion

This study provides consistent estimates of the long-term effects of childhood exposure to pollution and identifies the critical ages at which such exposure is most harmful. By lever-

aging an exogenous reduction in ambient sulfur dioxide (SO) concentrations—driven by stringent environmental regulations in Western Europe during the early 1980s—we demonstrate the lasting consequences of early-life pollution exposure on human capital formation and adult health. Given that SO is a precursor to fine particulate matter (PM2.5), its effects likely extend beyond short-term health outcomes, influencing long-term socioeconomic trajectories.

Focusing on Norway, we take advantage of comprehensive individual-level data covering the entire population, linking pollution exposure during childhood to a rich set of adult outcomes observed up to age 30. Our study benefits from a novel measure of transboundary SO2 pollution, based on high-resolution environmental monitoring data from the Norwegian Environmental Agency's EMEP program. By corroborating pollution variations with independent ecological indicators—such as water quality in lakes and freshwater fish stocks—we ensure the robustness of our exposure measure.

Our findings underscore the far-reaching implications of early-life pollution exposure, highlighting its potential to shape long-term well-being through channels related to education, labor market participation, and health. Furthermore, our results suggest that policies targeting air quality improvements can yield substantial intergenerational benefits, even for populations situated far from pollution sources. These insights reinforce the importance of continued environmental regulation and international cooperation in mitigating the long-term societal costs of pollution. Future research should explore the broader economic and demographic impacts of transboundary air pollution, shedding further light on the mechanisms through which environmental policy can promote equitable and sustainable development.

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Appendix A Contemporaneous Effects

We also examine the contemporaneous effects of the Convention. Unlike the long-term analysis, which focuses on broader trends, the short-term analysis relies on municipality-level data. To assess the immediate impact of the program on cardiorespiratory mortality rates, we estimate the following reduced-form specification:

$$Y_{jt} = \beta_1 Sulfur_j^{78} \times Post_t + X_{jt}\rho + \delta_j + \gamma_t + \epsilon_{jt} \tag{4}$$

where Y_{jt} represents the cardiorespiratory mortality rate in municipality j in year t. X_{jt} is a vector of municipality-level pre-convention demographic characteristics interacted with year t, γ_t are year-fixed effects, and δ_j are municipality-fixed effects. The key variable of interest, β_1, captures the interaction between pre-policy imported pollution levels ($Sulfur_j^{78}$) in municipality j and a dummy variable ($Post_t$) indicating whether year t falls after the implementation of the regulatory policies.

A.1 Mortality

To causally identify the impact of pollution on mortality, we rely on common trends across municipalities with different pre-policy pollution levels. To assess the validity of this assumption, we begin by examining the trend of age-adjusted mortality. We divide all municipalities into two groups—those above and those below the median sulfur pollution level in 1978. Under our definition, we assume that high-exposure municipalities experienced more considerable emissions reductions due to the Convention. This assumption is supported by the monitoring data presented in Figure 1.

Panels (a) and (b) of Figure 2 display raw trends in cardiorespiratory and external mortality rates by pre-policy sulfur levels. Cardiorespiratory mortality rates declined over time, with the gap between the two groups remaining largely stable. After the Convention, mortality rates decreased more rapidly in the South, where municipalities had higher pre-policy exposure than in the North. In contrast, external mortality rates remained stable throughout the period. Unlike cardiorespiratory mortality, external mortality trends moved in parallel for both groups over the entire period. These patterns align with the expectation that sulfur pollution influences mortality but primarily through pollution-related causes.

Next, we quantify the magnitude of this effect. First, we estimate a generalized difference-in-differences (DiD) model using Equation 4. Column (1) of Table A1 presents estimates of β_1 for mortality at all ages, Column (2) for mortality for individuals below 35 years, Column (3) for individuals aged 35—64 years, and Column (4) focus on mortality among individuals

65 and older. Panel A reports estimates for cardiorespiratory causes, while Panel B serves as a sort of placebo test and focuses on external mortality such as accidents and homicides. While cardiorespiratory death remains unchanged for individuals younger than 35 years, the estimates are significant for groups 35 and older and all-age mortality. The results indicate, for example, that a one-standard-deviation reduction in pollution leads to a 0,01% decrease in cardiorespiratory mortality among individuals ages 35-64. Mortality from external causes is not significantly affected by the Convention.

In summary, our key findings suggest that the Convention reduced imported sulfur, leading to a convergence of sulfur pollution levels across regions. Although we lack a direct measure of airborne PM2.5—the most harmful pollutant associated with acid rain—our findings reveal a strong relationship between sulfur levels and cardiorespiratory mortality rates. This suggests that our sulfur measure serves as a reasonable proxy for imported airborne pollution.

Table A1. Exposure to Sulfur Pollution and Age-Adjusted Mortality

	(1) All ages	(2) under 35	(3) 35–64	(4) 65 and over
Panel A: Cardiorespiratory				
Post × Sulfur78	-0.025***	-0.00049	-0.0061**	-0.015**
	(0.0074)	(0.00032)	(0.0028)	(0.0064)
Mean of dep. var.	520.7	2.03	80.9	456.7
Observations	8440	8440	8440	8440
Panel B: External				
Post × Sulfur78	0.0028	0.00070	0.00059	0.0015
	(0.0030)	(0.0014)	(0.0014)	(0.0019)
Mean of dep. var.	59.2	16.5	19.0	23.7
Observations	8440	8440	8440	8440

Appendix B

Table B1. Education outcome - robustness

Outcome	(1) College	(2) Schooling	(3) Low Ability (only male)
Panel A: Binarized exposure			
Post × High sulfur78	0.026***	0.057*	-0.0030
	(0.0056)	(0.030)	(0.0020)
Mean of dep. var.	0.36	14.73	0.066
Observations	1064647	1064647	463631
Panel B: Nonparametric			

Table B2. Health outcome - robustness

Outcome	(1) Asthma	(2) Depression	(3) Heart	(4) Thyroid	(5) Cancer
Panel A:					
Post × High Sulfur78	-0.0034*	0.0031	-0.00045***	-0.0014	-0.00012
	(0.0019)	(0.0024)	(0.00016)	(0.0013)	(0.00031)
Mean of dep. var.	0.0928	0.352	0.00123	0.0776	0.00858
Observations	1072743	1072743	1072743	1072743	1072743

Appendix C

Figure C1. Location of Monitors

