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Hannah Klauber
Mercator Research Center on Global Commons and Climate Change (MCC)

Felix Holub
University of Mannheim

Nicolas Koch
MCC, Potsdam Institute for Climate Impact Research (PIK) and IZA

Nico Pestel
IZA

Nolan Ritter
MCC and PIK

Alexander Rohlf
MCC and PIK

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IZA – Institute of Labor Economics

Schaumburg-Lippe-Straße 5–9
53113 Bonn, Germany

Phone: +49-228-3894-0
Email: publications@iza.org

www.iza.org

ABSTRACT

Killing Prescriptions Softly: Low Emission Zones and Child Health from Birth to School*

We examine the persistence of the impact of early-life exposure to air pollution on children's health from birth to school enrollment using administrative public health insurance records covering one third of all children in Germany. For identification, we exploit air quality improvements caused by the implementation of Low Emission Zones, a policy imposing driving restrictions on high-emission vehicles. Our results indicate that children exposed to cleaner air around birth require less medication for at least five years. The initially latent health response materializes only gradually in lower medication usage, leaving important but subtle health benefits undetected in common measures of infant health.

JEL Classification: policy evaluation, cohort study, air pollution, health, children, Low Emission Zone

Keywords: I18, Q51, Q53, Q58

Corresponding author:
Hannah Klauber
Mercator Research Center on Global Commons and Climate Change (MCC)
Torgauer Str. 19
10829 Berlin
Germany
E-mail: klauber@mcc-berlin.net

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1 Introduction

Poor air quality is a major public health concern worldwide. Various policy interventions have curbed ambient air pollution considerably over recent decades (Shapiro and Walker 2018). To further improve air quality, policymakers in high-income countries increasingly focus on clean air programs in urban areas where motor vehicles are the major source of emissions. For instance, more than 200 European cities with already moderate pollution levels have implemented Low Emission Zones banning emission-intensive vehicles from entering city areas to achieve additional air pollution reductions. However, this raises questions about the returns to investments in such ancillary clean air regulations which impose significant costs while the specific health benefits from slight improvements in air quality remain uncertain. In particular, important latent health effects are likely to remain undetected. First, it is difficult to isolate causal effects in the presence of a health stock that changes slowly over time (Almond and Currie 2011, Currie et al. 2014). Estimates of contemporaneous pollution impacts may substantially underestimate "fully formed" health benefits. Few studies address this issue indirectly by connecting pollution exposure at birth to productivity and earnings in adulthood (Isen et al. 2017). However, there is no quasi-experimental evidence for the long-term relationship between early-life exposure to air pollution and physical health that may affect human capital acquisition and labor market outcomes in later life. Second, in contexts characterized by a combination of high baseline population health, universal access to health care, and comparatively low pollution levels, health benefits are likely to take subtle forms that are hard to detect in the most widely used health measures such as low birth weight, hospitalizations, or mortality.

In this paper, we examine the persistence of the impact of early-life exposure to air pollution on children's health stock from birth to school enrollment. Using quasi-experimental variation in the roll-out of Low Emission Zones (LEZs) in Germany and administrative health insurance data covering one third of the entire population, we test whether children born just before and just after policy-induced reductions in particulate matter concentrations exhibit persistent differences in rarely studied medication usage for up to five years after treatment. We characterize how health benefits accumulate over the course of pre-school childhood and disentangle immediate from longer-term health effects in response to lower pollution levels. By doing so, this paper provides novel evidence on the adjustment trajectory of the health stock in the "middle years" between birth and school enrollment that have not been studied before (Almond et al. 2018). Our findings suggest that moderate improvements in air quality

due to the adoption of LEZs persistently reduce the number and cost of prescriptions for respiratory diseases while results for more commonly used measures of infant health (e.g., birth weight) do not point to significant changes.

Our work draws on a data set and a setting which allow us to gain insight into whether pollution reductions below levels that are already low by the standards of high-income countries have lasting health benefits.[1] Using medical records provided by Germany's largest public health insurer (AOK), we track children from birth to school enrollment and move beyond the usual measures of infant health. Our preferred outcome variables are the cumulative number and the cumulative costs of prescriptions for pharmaceuticals. Prescriptions are a sensitive, real-time, and monetizable health measure that captures subtle effects, e.g. slightly reduced medication requirements manifesting only over a prolonged time frame. While subtle for the individual, these effects apply to a broader population and become large in aggregate. Moreover, because medication alleviates morbidity, its effects are likely undetectable when using the usual indicators such as hospitalizations. The German context is well suited for the analysis of pharmaceutical prescriptions because Germany's universal public health care system covers all prescription costs without any deductibles for minors. Mandatory insurance coverage implies that we observe health care for the general population rather than for a specific group with insurance, as is the case in pioneering studies using pharmaceutical data for the U.S. (Deschênes et al. 2017, Williams and Phaneuf 2019, Deryugina et al. 2019) and China (Barwick et al. 2018).

The identification of causal effects rests on the staggered implementation of LEZs across 49 counties in Germany. *De facto*, LEZs constitute a ban of old, emission-intensive diesel vehicles from city areas.[2] Previous research has shown that LEZs are robust predictors of statistically significant reductions in local concentrations of particulate matter smaller than 10 μm (PM_{10}), which are however moderate in magnitude (Wolff 2014, Gehrsitz 2017, Pestel and Wozny 2019, Margaryan 2021). Thus, we exploit the staggered policy introduction as an instrumental variable for exogenous variation in PM_{10} exposure from motor vehicles. To isolate health effects, we follow Isen et al. (2017) and compare cohorts of children born just before and just after the policy-induced improvements in air quality. That way, cohorts experience different levels of pollution exposure in early life but the same levels throughout

[1] Notably, even prior to LEZ implementation, the annual mean concentration of particles smaller than 10 μm (PM_{10}) of 26.4 $\mu g/m^3$ is close to the corresponding WHO guideline level of 20 $\mu g/m^3$.

[2] Restrictions are generally based on the Euro emission standards for gasoline and diesel vehicles. However, German LEZs have banned very few gasoline vehicles because of the prevalence of catalytic converters that transform pollutants into less toxic gases (Cyrys et al. 2014).

subsequent years. Our sample is limited to children from "non-attainment" counties that violated EU air quality standards regarding PM_{10}. While treated counties introduce LEZs, control counties comprise late-adopters and not-to-date-adopters.

Our research design accommodates important methodological advances in difference-in-differences (DID) settings with staggered policy introduction (Goodman-Bacon 2018). We implement a "stacked DID" estimator (Fadlon and Nielsen 2019, Deshpande and Li 2019) that deals with the challenge of heterogeneous treatment effects inherent in the standard two-way fixed effect DID estimator. The stacked design aligns treatment events by event-time not calendar time. Therefore, it allows us to exploit a richer fixed effect structure: We remove (i) event-time trends caused by policymakers relying on local pollution or socio-economic trends in the years prior to the policy introduction as criteria to decide whether or when to introduce an LEZ, and (ii) time-invariant unobservables that may influence outcomes and the decision if or when to introduce an LEZ. Using this design, we demonstrate that pre-trends run parallel for all outcomes studied.

We produce three key findings. First, we present evidence that the number of prescriptions for children decreases significantly over early childhood in response to a moderate air quality improvement caused by the adoption of LEZs. A one $\mu g/m^3$ reduction in the concentration of PM_{10} *in utero* and in the first year of life decreases the total number of prescriptions for respiratory diseases over the five pre-school years by about 0.55 or by 3.9% in relative terms. The number of prescriptions decreases by up to 6.8% when analyzing the subgroup of pharmaceuticals closely tied to asthma. A decreasing share of children diagnosed with asthma (i.e. the extensive margin) drives the total reduction of asthma drug prescriptions and, thereby, also reduces prescriptions for respiratory diseases in general. However, we also find that children suffering from any kind of respiratory disease require less medication. This additional effect at the intensive margin suggests positive effects on children's respiratory health beyond asthma. Unconditional quantile regressions (Firpo et al. 2009) reveal suggestive evidence for substantial heterogeneity at the intensive margin. With a point estimate of 2.8 fewer prescriptions, children who suffer most from respiratory diseases may benefit nearly fives times as much from LEZs as the average sufferer.

Second, our results indicate that children's health stock adjusts slowly to changes in pollution levels which has rarely been documented because of a lack of real-time health measures. The contemporaneous effect on medication needs during the first year of life is substantially

smaller than the aggregate effect over the first five years of life. In fact, the effect in the first year accounts for only about 6–20% of the cumulative savings over the period before school enrolment. For asthma, the contemporaneous effect in the first year even remains statistically insignificant. It is only from the second year of life that the initially latent health response materializes in fewer children diagnosed with asthma and, thus, fewer total prescriptions. The observed delay in adjustment may also explain why prior studies on LEZs could not provide evidence for improved infant health measured by low birth weight (Gehrsitz 2017, Pestel and Wozny 2019). We corroborate this conjecture by using hospital data on fetal development disorders as alternative outcome variables for which treatment effects remain statistically insignificant.

Third, the identified health benefits yield economically meaningful cost savings for public health insurers. We find that a reduction of one μg/m^3 PM_{10} reduces costs by 10.54 Euro per child in their first five years of life. LEZs reduce PM_{10} levels by 1.37 μg/m^3 so that cost savings are 14.44 Euro per child. With average pre-treatment costs of 218.62 Euro per child, LEZs cause an economically relevant relative cost reduction of 6.6%. With $1,836,434$ children protected by LEZs in their *in utero* period and their first year of life, treatment reduces pharmaceutical spending for children born between 2008 and 2017 over their pre-school years by approximately 26.5 million Euros, or 42.4 million Euros when accounting for positive spillover effects. Because all children benefit from LEZs from their second year of life onward, cost savings originate from a single life year with slightly improved air quality. While these savings represent only a fraction of the total policy benefits, they already account for about 22% (or 35% when accounting for spillovers) of the up-front costs of owners of vehicles that fail to meet LEZ standards calculated by Rohlf et al. (2020). Finding considerable health benefits, even at overall low pollution levels, suggests that reducing pollution can have large positive effects on children's respiratory health in many settings.

This paper makes several contributions. First, our study is an important step forward in credibly estimating the sustained health benefits of lower exposure to air pollution in early life. There is robust evidence for particularly severe impacts of air pollution on infant and fetal health (Chay and Greenstone 2003a, Chay and Greenstone 2003b, Currie and Neidell 2005, Currie et al. 2009, Sanders and Stoecker 2015, Knittel et al. 2016). Motivated by the "fetal origins hypothesis", a few papers investigate the persistent effects of early-life exposure to air pollution on economic outcomes such as human capital formation (Sanders 2012, Bharadwaj et al. 2017) and labor market outcomes (Isen et al. 2017) in the long-run. Relat-

edly, Simeonova et al. (2019) exploit the sequential introduction of Stockholm's congestion charge to demonstrate that health benefits increase with the *duration of exposure* to cleaner air. We add to this literature by presenting evidence for the *persistence of health benefits* from exposure to cleaner air in a fixed period of time, i.e., the period before age one. By doing so, we provide a first quasi-experimental estimate for the dose-response relationship between PM pollution and *physical* health for the "missing middle", i.e., the understudied period between infancy and adulthood. We reveal a slowly adjusting health response over the years between birth and school and demonstrate that commonly used contemporaneous infant health measures may be too crude to detect health benefits from moderate air quality improvements in the context of already low pollution levels. By linking improvements in air quality from banning pervasive high-emission vehicles to health benefits across the child population, the estimates may also come with a higher external validity. The few seminal papers on the health impact of traffic pollution focus on more disadvantaged populations (children living next to highway toll stations, Currie and Walker 2011) or more unique pollution settings (emissions-cheating diesel cars in the U.S., Alexander and Schwandt 2019).[3]

The second feature that sets this study apart is its focus on the use and the costs of pharmaceuticals. Guided by the medical literature (Fanta 2009) and a seminal paper by Deschênes et al. (2017), we argue that direct morbidity and mortality conditions are a function of pollution and compensatory adaptation in terms of drug therapy. Failing to account for the pharmaceutical expenditures means to underestimate the benefits of clean air policies. With the notable exceptions of Deschênes et al. (2017), Williams and Phaneuf (2019), and Deryugina et al. (2019), the effect of air quality on defensive pharmaceutical expenditures remains largely unevaluated. In our setting in Germany, characterized by universal health care access, our finding of significant longer-term reductions in defensive spending is relevant from a public finance perspective. Reduced pharmaceutical expenditures may lower insurance contributions, lower labor costs for employers, and increase net income for households.[4] The quantified policy benefits in terms of persistently lower defensive spending for children's

[3] Alexander and Schwandt (2019) exploit the dispersion of emissions-cheating diesel cars in the U.S. to quantify the morbidity costs of diesel pollution for a broader population of children. However, the U.S. passenger vehicles exhibit only a low single-digit diesel share (only 1.5% of all light duty vehicles in 2014, U.S. DOT 2015). He et al. (2018) provide a careful study in the context of exceptionally high pollution levels, showing a decrease in hospitalizations after the opening of a beltway diverting diesel trucks from passing through São Paulo. In contrast, we examine the link between child morbidity and diesel pollution in the context of German cities where (i) diesel vehicles are pervasive (45.9% in 2016, KBA 2017) and a major source of PM, and (ii) initial pollution levels are low. Both characteristics are widespread in Europe and unaccounted for in prior work.

[4] Presently, health care contributions are set to 14.6% of gross wages equally shared between employers and employees.

medication are a complement to recent estimates of Pestel and Wozny (2019) and Margaryan (2021), indicating that LEZs also lead to contemporaneous reductions in hospital treatments and ambulatory care claims related to cardiovascular and respiratory diseases for all age groups. While hospitalizations and outpatient treatments of adults may also reflect medical histories linked to pollution exposure and confounding influences in the distant past, such confounders can be ruled out for infants observed from birth onward.

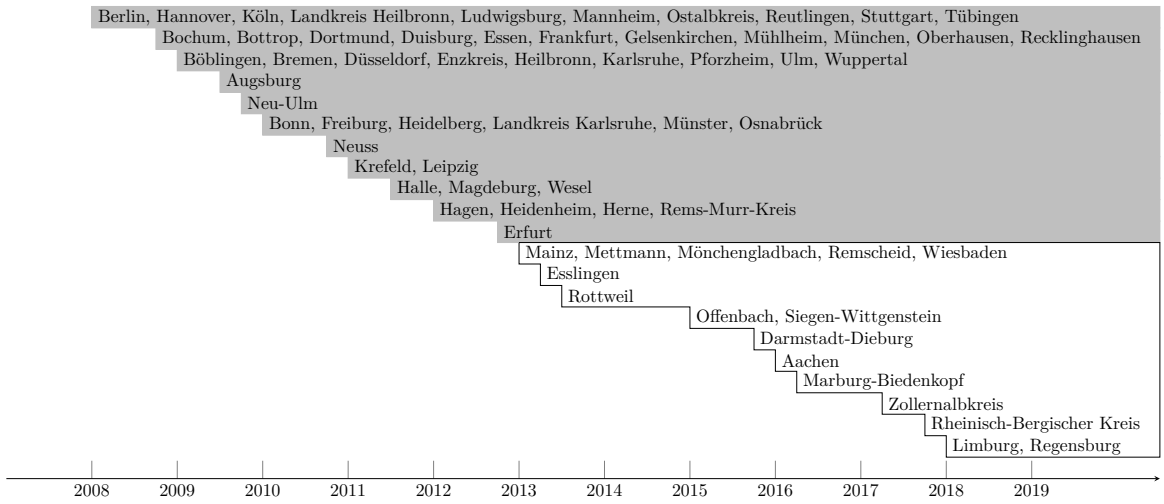
2 Low Emission Zones as a Research Design

2.1 Institutional Background

In Europe, LEZs are the main instrument for cities to meet EU air quality standards which are among the strictest worldwide. To improve air quality and protect public health, the EU enacted several directives that set legally binding limits for criteria pollutants. Since 2005, for example, the annual mean of PM_{10} pollution must not exceed 40 $\mu g/m^3$. Moreover, daily PM_{10} readings must not exceed 50 $\mu g/m^3$ more than 35 times per year at any measuring station (Directive 2008/50/EC). For $PM_{2.5}$, the EU implemented legally binding limits only in 2015. EU Member States that violate these limits face considerable fines. In Germany, the 16 federal states are responsible for compliance with the EU air quality standards. In case of violations, state governments are obliged to develop city-specific Clean Air Plans (*Luftreinhaltepläne*). The implementation of LEZs is by far the most tangible measure to comply with Clean Air Plans. LEZs explicitly target traffic pollution. To date, 65 counties implemented them in a staggered process where the time of introduction of each individual LEZ depended on several idiosyncratic factors. First, the decision-making process usually involves the respective city administrations and city councils as well as other stakeholders, but state governments ultimately have to approve local Clean Air Plans.[5] They may overrule the decisions of local authorities and force cities to implement LEZs. Because of often conflicting interests between state and local policymakers, the length of the decision-making process regarding the introduction of an LEZ varies. Second, NGOs and private citizens frequently appeal to the courts to advocate against or in favor of air quality regulations which creates

[5] At the federal level, regulations first had to (1) establish vehicle emission categories and (2) designate an official road sign for LEZs. This was done in late 2007.

Figure 1: The Staggered Implementation of LEZs



The figure depicts introduction dates for all counties with an LEZ. The eleven implementation waves considered in this paper are marked in grey.

further plausibly exogenous variation in the timing of LEZ introductions. Court rulings based on EU air quality legislation have generally sped up the adoption of LEZs.[6]

Figure 1 shows that in almost every year since 2008 there have been waves of new LEZ introductions. Some LEZs cover entire counties while others rather ban emission-intensive vehicles from inner-cities. To secure access to LEZs, a vehicle must display an appropriately colored windscreen sticker based on EU-wide tailpipe emissions categories. The most emission-intensive diesel vehicles up to Euro1 standards (equivalent to 0.14g PM_{10} per km) are banned from LEZs. Petrol-driven vehicles are banned if they do not have a catalytic converter, which is very rare in Germany. Therefore, LEZs are *de facto* bans of old, emission-intensive diesel vehicles.[7] Police and local public order authorities enforce the policy with penalties for its violation of currently 100 Euros.

We exploit the temporal and spatial variation in the introduction of LEZs to break well-known sources of endogeneity in the link between health and pollution. For instance, local economic conditions not only affect ambient air pollution (Chay and Greenstone 2003b) but also infant

[6]Examples from Wiesbaden and Halle shed light on the different paths leading to the introduction of LEZs. In late 2010, Wiesbaden's city council proposed the introduction of an LEZ which the state government of Hesse rejected. After the appeal of an environmental interest group, Wiesbaden's administrative court ruled that the state government had to approve the LEZ in order to comply with air quality standards as soon as possible (Wiesbadener Tagblatt 2011). In contrast, the city of Halle appealed plans by the state government of Saxony-Anhalt to implement an LEZ. The city council decided to abandon their case against an LEZ after it was made clear that the EU would otherwise penalize the city almost one million Euros per day (Mitteldeutsche Zeitung 2011).

[7]Less emission-intensive vehicles with Euro2 and 3 standard are eligible for red or yellow stickers while cars that meet Euro4 standards are eligible for green stickers.

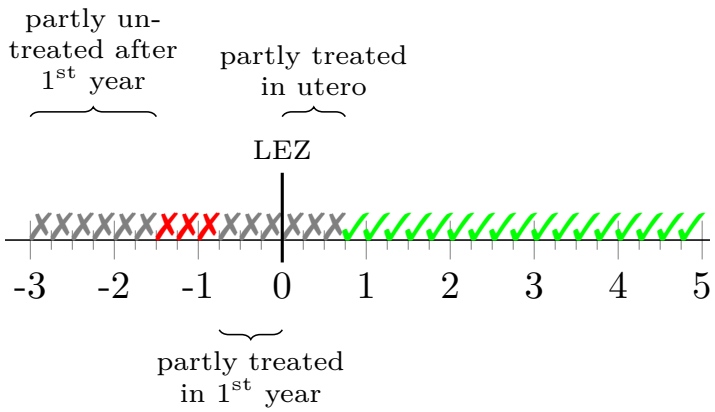
health (Dehejia and Lleras-Muney 2004, Lindo 2011). Therefore, we instrument changes in PM_{10} with the implementation of LEZs. Prior research finds that LEZ implementation decreased local PM_{10} concentrations by 4 – 9% (Wolff 2014, Malina and Scheffler 2015, Gehrsitz 2017, Pestel and Wozny 2019, Margaryan 2021). Our approach of using a policy intervention as an instrumental variable is similar to the identification strategy by Chay and Greenstone (2005), Bento et al. (2015), and Isen et al. (2017) who use county attainment status under the U.S. Clean Air Act as an instrument for changes in pollutant concentrations. To address concerns that LEZs are not introduced randomly but in areas where air quality is deteriorating, we follow Wolff (2014) and include in our sample only non-attainment counties, that failed to meet the PM_{10} limits. Of these counties (i) 65 implemented an LEZ and (ii) 63 have not implemented an LEZ to date despite non-compliance. Only the 49 counties that implement their LEZ until 2012 count towards the treated because we want to be able to follow children after LEZ implementation for five years. Our identifying assumption is that in the absence of LEZ introduction, air pollution and health outcomes in treated counties would have evolved similarly to the control counties.

2.2 Isolation of Early-Exposure Effects

Our goal is to estimate the causal effect of PM exposure in the *in utero* period and the first year of life on later life health outcomes measured before school enrollment. Due to rapid cell proliferation and an intense phase of epigenetic programming, children in the prenatal and immediate postnatal development period are especially vulnerable to the toxicological effects of pollution (Holt 1998, Šrám et al. 2005, Gluckman et al. 2008, Baccarelli and Bollati 2009). The empirical challenge is to isolate the long-term effect of PM exposure before age one from any exposure throughout the subsequent lifetime. We want to compare individuals who are exposed to different levels of PM pollution up to age one but who are exposed to the same levels of PM pollution thereafter.

To this end, we resort to a cohort study design as proposed by Isen et al. (2017). Our analysis compares differences in health outcomes of children born just before and just after LEZ implementation relative to the difference in health outcomes between children from the control group born at the same times. We restrict our sample so that within each county the policy provides the same regulatory protection for all children after age one. Of the cohorts born after LEZ implementation we include all those born at least four quarters post-treatment. LEZs protect these children from conception onward. Of the cohorts born pre-treatment we

Figure 2: Isolating early-exposure effects



The figure shows which cohorts are included in a given implementation wave. The numbers on the timeline measure years with respect to the introduction date defining the implementation wave of interest. The green marks indicate the post-treatment cohorts while the red marks indicate the pre-treatment cohorts.

include those born four to six quarters prior to LEZ implementation. These children are not protected before age one but they are protected shortly thereafter (see Figure 2).[8] We exclude children born in the three quarters prior or post to LEZ implementation from the sample because they are partly treated *in utero* or during their first year of life.

3 Methodology

Our empirical strategy combines the staggered introduction of LEZs in an instrumental variable approach with a cohort design to isolate the persistent impacts of early-life exposure to PM pollution. We first explain the basic difference-in-differences model (DID) underlying our instrumental variable (IV) estimation. We then explain the stacked regression approach that we implement to estimate the DID model. It addresses recent concerns about bias arising from heterogeneous treatment effects in DID settings with staggered policy introduction.

3.1 Difference-in-Differences Model

Our first-stage regression of the IV estimator is a DID regression model specified as

$$P_{ct} = \alpha Treated_{ct} + \sum_{\tau} \delta^{\tau} D_{ct}^{\tau} + W_{ct}'\rho + X_{c\bar{t}}'\pi_t + \gamma_c + \eta_{st} + \nu_{ct} \tag{1}$$

[8]Note that to ensure equal exposure after age one, we would need to exclude all cohorts born more than four quarters prior to LEZ implementation. To avoid limiting ourselves to a single pre-treatment observation of the treated counties, we keep the two cohorts born five and six quarters prior to LEZ implementation in our sample and ensure that this does not influence our results in a robustness check.

where the dependent variable P_{ct} is the mean PM_{10} exposure in $\mu g/m^3$ during the first quarter of life of a cohort born in county c and year-quarter t. The binary variable $Treated_{ct}$ is equal to 1 if county c has implemented an LEZ in quarter t. The indicators D_{ct}^{τ} are equal to 1 if quarter t is τ quarters before or after the quarter of LEZ implementation in county c. Because we include cohorts of children born in the pre- and post-treatment periods defined in Figure 2, $\tau \in \{-6, -5, -4, 4, 5, 6, \ldots, 19\}$ (see Section 2.2).[9] The vector W_{ct}' comprises weather controls in the county of birth c, which are exogenous and, thus, included for every year of life. In some specifications, we also include pre-treatment controls for socio-economic characteristics (e.g. population density, employment, income, and transfers) interacted with year-quarter fixed effects denoted by $X_{c\bar{t}}'$.[10] Observing that the estimated treatment effect changes significantly after allowing for these trends that vary with the levels of the exogenous covariates would suggest that the results are driven by differential trends in pollution across socio-economic characteristics (see Jaeger et al. (2020) and Hoynes et al. (2016) for similar approaches). The county fixed effects γ_c control for time-invariant, unobserved determinants of pollution exposure for children born in county c. The fixed effects η_{st} account for time-varying determinants of pollution exposure that are common to all children born in state s in year-quarter t.[11] The coefficient $\widehat{\alpha}$ provides a DID estimate of the impact of LEZ implementation on quarterly PM_{10} levels at the county level.

In the second stage, predicted values for PM_{10} from Equation (1) serve as an explanatory variable in

$$H_{ct} = \beta \widehat{P_{ct}} + \sum_{\tau} \sigma^{\tau} D_{ct}^{\tau} + W_{ct}' \kappa + X_{c\bar{t}}' \mu_t + \gamma_c + \eta_{st} + \epsilon_{ct} \tag{2}$$

where the coefficient of interest β describes the marginal effect of a one $\mu g/m^3$ LEZ-driven increase in PM_{10} exposure on the average health outcome H_{ct} of a cohort born in county c and year-quarter t. The health outcome is accumulated over the first five years of a child's life and is averaged over the children in cohort ct. We obtain the health outcome through an

[9] For instance, $\tau = 4$ refers to the fourth quarter after LEZ implementation. Similarly, $\tau = -4$ refers to the fourth quarter prior to LEZ implementation. Note, that the number of treated cohorts depends on the implementation timing because our sample only comprises children born between 2006 and 2012.

[10] We generate these controls by interacting year-quarter dummies with terciles of the variables measured in $\bar{t} = 2007$, the year prior to the first LEZ implementations (Barrot and Sauvagnat 2016). Appendix D.1 holds a detailed description of the control variables.

[11] Note that the counties of Berlin, Hamburg, and Bremen are states in their own right. Including state–quarter fixed effects absorbs the variation explained by the treatment in these counties. Therefore, we allocate these three counties to neighboring states, i.e. we assign Berlin to Brandenburg, Hamburg to Schleswig-Holstein, and Bremen to Lower Saxony. We ensure that this allocation does not determine the results by estimating alternative specifications.

auxiliary regression that exploits the available information at the level of the individual child (i.e. gender and area of residence within a county at the five-digit zip code), which controls for some of the observed heterogeneity in individual health when aggregating the data.[12] All regressions are weighted by the number of children in a cohort. We cluster standard errors at the county level, the level at which the treatment is assigned.

The unbiasedness of $\widehat{\beta}$ depends on two crucial assumptions. First, LEZs need to be a strong instrument for changes in particulate matter pollution. We subsequently present evidence for a strong first-stage, showing that PM_{10} levels decline significantly and persistently in response to LEZ implementation. Second, for consistency, it must be the case that LEZ introduction affects health outcomes only via its impact on air pollution. Given that our results are largely insensitive to the choice of controls, and conditional on the full set of fixed effects, we believe that the exclusion restriction holds. While we cannot conclusively show its validity, we conduct various indirect tests: (i) we comprehensively assess whether LEZs shift the composition in the underlying population in LEZ counties, (ii) we re-estimate our IV model for medical conditions unrelated to air pollution, and (iii) we explore whether the compliance costs of LEZs reduce emissions from industrial activity. Finally, the exclusion restriction may be violated if LEZs affect any pollutants other than PM_{10} that also have a direct impact on respiratory health (see discussion in Section 5.5). In fact, we show that LEZs reduce both PM and NO_2 but no other pollutants and argue that our identification strategy suffices to identify the health effects of pollutants from motor vehicles rather than of PM_{10} in particular. Similarly, other studies attribute estimated IV-effects to air pollution more generally rather than to a particular pollutant (Chay and Greenstone 2003a, Currie and Neidell 2005, Arceo et al. 2016, Knittel et al. 2016, Deryugina et al. 2019, Sager 2019, Colmer et al. 2020).

3.2 Stacked Regression Design

To address concerns about bias arising from the combination of potentially heterogeneous treatment effects and the weighting implied by the two-way fixed effects regression in settings with staggered policy introduction (Goodman-Bacon 2018, Athey and Imbens 2018, de Chaisemartin and d'Haultfoeuille 2020), we implement Equation (1) and (2) in a stacked

[12] Aggregating individual level data *via* auxiliary regressions and conducting regressions on the obtained aggregates is a common approach in the literature (e.g. Currie et al. 2015, Isen et al. 2017) and asymptotically equivalent to using the individual level data itself (e.g. Donald and Lang 2007). We provide a detailed description of the procedure in the Appendix B.

regression design (Deshpande and Li 2019, Cengiz et al. 2019, Fadlon and Nielsen 2019). Goodman-Bacon (2018) shows that the two-way fixed effect DID estimator consists of comparisons between all combinations of early treated, late treated, and untreated units. The units that are treated in the middle of the study period have higher weights in the regression than those treated at the beginning or at the end.[13] Because the effectiveness of treatment may vary with policy adoption timing, we have to ensure that the weighting of cohorts does not bias our average treatment effect. For instance, early implemented LEZs may be more effective at reducing PM emissions than the later implemented ones due to the stock of old high-emission vehicles decreasing over time. Our stacked DID design aligns treatment events by event-time, not calendar time. This results in a setting that is equivalent to one where the treatment events occur all at the same time instead of in a staggered fashion. This prevents the unintended weighting of events driven by their timing.[14] In addition, the stacked DID design prevents potential bias from using units as controls that have been treated shortly before and might still be on differential trends.

To implement the stacked DID design we create an individual data set for all LEZ *implementation waves*, i.e. year-quarters in which at least one county implemented an LEZ. For the period between 2008 and 2012, there are 11 distinct LEZ implementation waves. Counties that introduce LEZs in the year-quarter defining the respective implementation wave count towards the treatment group. Counties that do not introduce an LEZ either because they do so at a different point in time or never, are eligible for the control group. The stacked design enables us to refine the selection of the control counties in each wave. Throughout our main analysis we allow only LEZ-counties that implement the policy measure at least four years before or five years after the implementation wave to enter the control group. This selection ensures that we have a balanced control group in each implementation wave and that the control group is not on a differential trend from recent treatment, as we explain in detail in Appendix C.1. We stack the data sets for the 11 implementation waves for a pooled regression.

[13] This is because each comparison is weighted by the size of the subgroup and the variance of treatment. If treatment occurs either early or late, this results in lower treatment variance and, hence, a lower weight.

[14] The variance of treatment no longer biases the weighting (see also Abraham and Sun 2020). Regression weights now mainly depend on the size of the subgroup.

The first and second stage of the IV estimator in the stacked DID regression model are specified as

$$P_{ctj} = \alpha(Treated_{cj} \times Post_{tj}) + \sum_{\tau} \delta_j^{\tau} D_{tj}^{\tau} + \lambda_j D_{cj} + W'_{ctj}\rho + X'_{c\bar{t}j}\pi_t + \gamma_c + \eta_{st} + \nu_{ctj} \quad (3)$$

$$H_{ctj} = \beta \widehat{P_{ctj}} + \sum_{\tau} \sigma_j^{\tau} D_{tj}^{\tau} + \psi_j D_{cj} + W'_{ctj}\kappa + X'_{c\bar{t}j}\mu_t + \gamma_c + \eta_{st} + \epsilon_{ctj} \quad (4)$$

where the dependent variables are now given for a cohort born in county c and year-quarter t for each treatment wave j, respectively. The binary variable $Treated_{cj}$ is equal to 1 if county c introduces an LEZ in implementation wave j. $Post_{tj}$ is a binary variable equal to 1 if quarter t is after the implementation quarter of wave j. For every implementation wave, the indicators D_{tj}^{τ} are equal to 1 if quarter t is τ quarters before or after the quarter of LEZ implementation j. Every wave j also has its own indicator D_{cj} which is equal to 1 if county c introduces an LEZ specifically in that implementation wave.

Our stacked DID design enables us to remove important unobservables that may simultaneously drive treatment selection and outcomes. First, the indicators D_{tj}^{τ} in Equations (3) and (4) remove LEZ wave-specific unobservable trends in event-time that emerge, for instance, if policymakers use local pollution or socio-economic trends in the years prior to implementation as decision criteria for introducing LEZs. Accommodating calendar time effects will not eliminate such pre-trends.[15] Second, by including D_{cj} and allowing its effect to vary by implementation wave, we remove time-invariant differences between treatment and control groups for each LEZ implementation wave j and between different implementation waves.[16] This accounts for time-invariant unobservables that may drive outcomes and selection into LEZ adoption and earlier or later adoption.[17]

Conditional on fixed effects, we consistently show that there are no differences in the trends of air pollution and health outcomes across the treatment and control groups before LEZ implementation. To this end, we estimate event-study specifications of the first stage and the reduced form model. For these specifications, we expand the pre-treatment window of our sample to include all observations up to three years prior to LEZ implementation. Furthermore, we include observations from the three quarters before and the three quarters

[15] One could restrict the effect of the wave-specific event-time binary variables D_{tj}^{τ} to be equal across treatment waves, that means replacing δ_j^{τ} by δ^{τ}. However, we would then only remove the variation in event-time pooled over all implementation waves.

[16] The latter is only true because we include D_{tj}^{τ} as well.

[17] In case we only included one wave-unspecific binary variable $Treated_{cj}$, we would only control for these differences between the pooled treatment and the pooled control group.

after LEZ implementation, so that $\tau \in \{-12, \ldots, 19\}$. To gain precision and prevent noise potentially linked to estimating 32 quarter coefficients, we group the event study coefficients at the year level throughout our paper (see also Appendix C.2).

4 Data

Air Pollution and LEZ Data

The German Environment Agency (UBA) provides data on air pollution for the years 2006 through 2012 for 128 counties that violated EU-wide limits for PM_{10}. Specifically, monitoring stations record daily concentrations of regulated air pollutants particulate matter (PM_{10}), nitrogen dioxide (NO_2), ozone (O_3), and sulphur dioxide (SO_2). For the sample period, there is no consistent data on fine particulate matter ($PM_{2.5}$) concentrations available from UBA because there were no legally binding thresholds for $PM_{2.5}$ before 2015. Stations located at the roadside primarily measure peak pollution exposure from traffic, while the remaining stations measure the permanent exposition to pollution in residential areas (UBA 2019). We combine measurements from both types of stations and interpolate the point measures into county space using Inverse Distance Weighting (Currie and Neidell 2005, Karlsson and Ziebarth 2018). Based on the daily records, we construct a weighted average of quarterly levels of PM_{10}, NO_2, O_3, and SO_2. Following recent studies (i.e. Chay and Greenstone 2003a, Isen et al. 2017), we use weights proportional to the number of monitor observations within a quarter and limit our data to stations with at least 60 measurements per quarter (see Appendix D.2 for a detailed description). UBA also provides data on the exact dates at which LEZs are implemented.

Health Care Data

The health data comes from Germany's largest public health insurer AOK. In Germany, public health care applies to roughly 90% of the population, of which the AOK covers about a third. The remaining 10%, in particular civil servants, self-employed, and high-income employees, are insured privately. Within the population of the publicly insured, the AOK data is the best available base for identifying generalizable effects. The insurance fund covers a population share almost twice as high as is the case with U.S. Medicare and provides a well-balanced representation of all population groups (Jaunzeme et al. 2013).[18] We obtained

[18]For a detailed overview of the representativeness of the AOK population, refer to Appendix D.3.

access to the anonymized data through the "AOK Research Institute" (WIdO). The data is at the level of the individual child and the individual quarter and provides us with information on pharmaceutical prescriptions and in-hospital doctoral care.[19] It holds information on about a third of all children born in Germany, approximately 200,000 annually. The sample includes the full medical records of all children born between January 1, 2006, and December 31, 2012, until the end of 2017, which marks the maximum range of data available at WIdO when conducting this research. This allows for observing all children in the sample from birth until age five. Because our analysis focuses on early childhood pollution effects, we exclude children from our sample who move out of their birth county within the first year of life. Likewise, we exclude children who are not continuously insured with AOK until the age of five. Overall, our sample holds observations on about 1.1 million children across all counties in Germany. The sample of counties underlying our analysis that either implement or consider implementing an LEZ comprises 550,000 children.

The focal point of our analysis are respiratory health outcomes. It is well established in the medical literature (e.g. Li et al. 2003) that PM pollution causes inflammations in the respiratory tract that may irreversibly reduce lung growth and function. In consequence, affected children are more vulnerable to suffer from respiratory problems in general and are at a higher risk of developing severe chronic diseases such as asthma later in life. Because respiratory diseases are primarily treated with pharmaceuticals, we restrict the main analysis to prescriptions. Pharmacies electronically provide health insurers with data on prescriptions (Swart et al. 2005). These data hold information on costs and pharmaceutical substances classified according to the ATC-Code system.[20] In Germany's universal public health care system, all prescription costs are covered by the insurance without any co-payments for children. This applies to over-the-counter drugs as well, as public insurances are legally obligated to bear the expenses for children up to the age of 12 (§34 Abs. 1 Satz 5 SGB V).

Prescription data are not linked to ICD-10 codes that reveal the diagnoses for which pharmaceuticals are prescribed.[21] Therefore, it is necessary to identify the relevant pharmaceutical substances for the therapy of respiratory diseases. To this end, we follow two different

[19] Note, that we only observe stationary hospital treatments that accommodate overnight cases.

[20] The Anatomical Therapeutic Chemical (ATC) classification system categorizes drugs based on their active ingredients according to the organ or the system on which they act as well as their therapeutic, pharmacological, and chemical properties. It is compiled by the World Health Organization (WHO) and adapted to the German market on an annual basis (Swart et al. 2005).

[21] The ICD-10-Code is an international system for the statistical classification of diseases and related health problems provided by the WHO. Germany uses the extended version ICD-10-GM. Outpatient and inpatient physicians are legally required (§§295 and 301 SGB V) to classify diagnoses accordingly.

approaches. First, we use a publication akin to the Red Book ("Gelbe Liste") to link pharmaceuticals and diagnoses for more than 120,000 drugs. This provides us with a broad group of pharmaceuticals used to treat respiratory diseases. Second, we consider the 20 most often prescribed substances for asthma and chronic obstructive pulmonary disease (ATC R03) in a given year. This returns a small, strict subset of the pharmaceuticals identified in the first approach. Appendix D.4 describes the procedures in detail. Costs of prescriptions are in real values normalized to the fourth quarter of 2017 and account for market price changes, such as expiring patents (see Appendix D.4).

We complement the analysis of respiratory health outcomes with placebo tests. To this end, we consider three outcomes that are independent of air pollution but correlated with socioeconomic status: hospital treatments of injuries of the head (S00-S09), the arm (S40-S49), or several body parts (T00-T07). Additionally, we use hospital data on pregnancy duration and fetal growth (ICD-10 codes P05, P07, and P08) in our analysis of common infant health measures.[22] On the insured themselves, the medical records additionally offer information about the birth dates, gender, and the precise location of residence within a county at the five-digit zip code. Table 1 provides summary statistics of the prescription data for respiratory diseases and pollution levels at the county and quarter level. Overall, the cross-sectional dimension of our data covers 128 counties, while the longitudinal dimension covers 28 quarters from 2006 to 2012. A detailed description is provided below the table.

Additional Data on Weather and County Characteristics

Weather is a strong correlate of pollution and health (Karlsson and Ziebarth 2018). Therefore, we obtain data from the German Weather Service (DWD) on a battery of weather phenomena at the level of the individual weather station as well as in an interpolated grid format. We combine weather, pollution, and health data at the county level. In the regressions, we include linear and quadratic terms of precipitation as well as 12 temperature bins[23] in addition to mean temperature, sunshine duration, relative humidity, pressure, and wind speed. We include additional control variables from the Federal Institute for Research on Building, Urban Affairs and Spatial Development (BBSR) such as socio-economic and demographic characteristics as well as information on the age of mothers giving birth. We

[22] In accordance with common practice, we consider the discharge diagnosis as the main reason for hospitalization (Swart et al. 2005).

[23] Temperature bins count the number of days with temperatures above 0, 5, 10, 15, 20, 25, 29, 30, 31, 32, 33 and 34 degrees Celsius.

Table 1: Summary Statistics of Pollution and Health Outcomes

	(1) mean	(2) sd	(3) min	(4) max	(5) N
Air pollution					
PM_{10} ($\mu g/m^3$)	23.7	6.2	9.0	57.3	3,584
Prescriptions for respiratory diseases					
Number of prescriptions over five years per child	12.7	2.8	4.4	23.8	3,584
Prescription expenditures over five years per child (€)	195.8	54.1	63.6	635.3	3,584
Share of sufferers per cohort (%)	76.1	7.5	38.3	94.6	3,584
Prescriptions for asthma					
Number of prescriptions over five years per child	2.0	0.8	0.1	6.1	3,584
Prescription expenditures over five years per child (€)	62.2	33.9	1.6	489.8	3,584
Share of sufferers per cohort (%)	18.3	6.1	1.3	50.6	3,584
Number of children per cohort	159.3	171.6	10	1,593	3,584

The table reports summary statistics of PM_{10} pollution (measured in $\mu g/m^3$) and of cumulative prescriptions over the five pre-school years linked to a broad group of respiratory diseases and asthma specifically. The variables are defined for our study period from 2006 to 2012 and our sample of 128 German counties that violated EU-wide limits for PM_{10}. Cumulative prescriptions are calculated based on data until 2017. Health measures are in terms of the number or the costs of prescriptions per child. Costs of prescriptions are in real values normalized to the fourth quarter of 2017. The share of sufferers reflects the share of children in the cohort that require at least one prescription for a respiratory disease or asthma, respectively.

use pre-treatment measures of these variables in 2007 and categorize them into terciles. For a detailed description see Appendix D.1.

5 Results

5.1 Ambient Air Pollution

First, we present evidence that the relationship between LEZ implementation and particulate matter levels is strong. Table 2 shows estimates of the effect of LEZ introduction on average quarterly PM_{10} concentrations following Equation (3) for two different sets of control variables that increase in stringency from left to right. All regressions include birth county and birth state–birth quarter fixed effects. Standard errors clustered at the county level are in parentheses. The reported mean outcomes represent weighted averages for the dependent variable in the pre-treatment period over all treated counties with weights equal to the number of children per county.

Our most stringent specification shows that the presence of an LEZ reduces mean quarterly PM_{10} concentrations by about 1.37 $\mu g/m^3$. This estimate is statistically significant at the

0.1% level and robust across control sets. The stability of our treatment effect estimates after including heterogeneous trends that vary with the pre-treatment levels of the socio-economic controls (see Section 3.1) suggests that the LEZ effect is not driven by differential trends in pollution across socio-economic characteristics. Compared to the mean pollution exposure of 26.44 μg/m^3, an LEZ decreases particulate matter by about 5.2%, which is in line with the LEZ literature. An F-statistic of 20.14 provides evidence for a fairly strong first stage relationship. To accommodate remaining concerns about potential bias from weak instruments (Andrews et al. 2019, Lee et al. 2020), we subsequently also report robust Anderson-Rubin (AR) confidence intervals for our IV-estimates.

Figure 3 plots the event-study results for the effect of LEZs on PM_{10}.[24] The post-treatment patterns suggest that LEZs cause a level shift to persistently lower PM_{10} concentrations with a strong immediate effect. Moreover, coefficients prior to treatment are close to zero and statistically insignificant, which is in line with common trends in LEZ and non-LEZ counties in the years preceding the policy interventions. Recall that our reported event study coefficients exploit a rich fixed effect structure to remove (i) event-time trends caused by policymakers relying on local pollution or socio-economic trends as criteria to decide whether and when to introduce an LEZ and (ii) time-invariant unobservables that may drive outcomes and selection into LEZ adoption and earlier or later adoption.

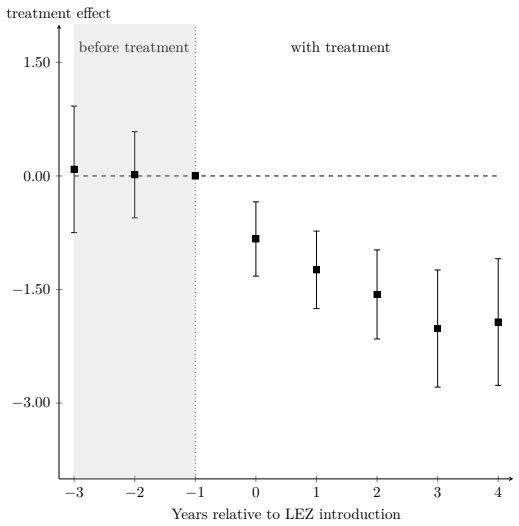
Table 2: The Effect of LEZ Implementation on PM_{10} Concentrations

	First Stage Estimation	
	PM_{10} Pollution $(in\ \mu g/m^3)$	
	(1)	*(2)*
LEZ treatment	-1.30***	-1.37***
	(0.34)	(0.30)
Mean outcome	26.44	26.44
First stage F-statistic	14.25	20.14
Weather controls	x	x
Socio-economic controls		x

This table reports coefficients from two variants of the first stage regression in Equation (3). The dependent variable is the quarterly mean PM_{10} concentration in a given county and year in μg/m^3. Both columns include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is 9,609.
* $p < .05$, ** $p < .01$, *** $p < .001$.

[24] Table A.1 in the Appendix provides the full regression results.

Figure 3: Event-study Estimates of the Effect of LEZ Implementation on PM_{10} Concentrations



The figure presents event-study coefficients from Equation (C.1) that show how LEZs affect the quarterly mean PM_{10} concentration in $\mu g/m^3$ in the years before and after LEZ implementation. The grey shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

5.2 Medication of Respiratory Diseases

In the following, we show the persistent effects of lower PM_{10} pollution on the medication of respiratory diseases. Our analysis comprises two groups of prescriptions to detect health effects of varying severity. To comprehensively capture respiratory health effects, we consider a broad group of pharmaceuticals. To capture effects related to asthma specifically, we consider a subset of pharmaceuticals that is closely linked to the therapy of this chronic disease. For both groups of prescriptions, we provide reduced form estimates that indicate the health effect of LEZ implementation (upper panel) and IV estimates representing the health effect of a one $\mu g/m^3$ increase in PM_{10} levels (lower panel) in Table 3. The dependent variable is either the number of prescriptions (left side) or their costs in Euro (right side) that accumulate on average over the first five years of a child's life.

All regression estimates show that LEZs benefit child health. The magnitude of our results is robust and statistically significant across control sets. For the broad group of respiratory diseases the most stringent IV estimate in column (2), for instance, shows that an LEZ-caused decrease in pollution of one $\mu g/m^3$ *in utero* and in the first year of life reduces the number of medical prescriptions by about 0.55 per child, on average. With a standard error of 0.17, the estimate is statistically significant at the 1% level. Relating the treatment effect to the

Table 3: The Effect of Early-Life PM_{10} Exposure on Medication of Respiratory Diseases throughout Early Childhood

	A. Number of prescriptions		B. Costs of prescriptions (€)	
	(1)	*(2)*	*(3)*	*(4)*
			Reduced Form Estimation	
Respiratory diseases	-0.65***	-0.75***	-10.98**	-14.41**
	(0.17)	(0.18)	(3.58)	(4.32)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	-0.21**	-0.23**	-6.34*	-8.01*
	(0.07)	(0.08)	(2.57)	(3.22)
Mean outcome	2.5	2.5	73.27	73.27
			IV Estimation	
Respiratory diseases	0.50**	0.55**	8.46*	10.54**
	(0.16)	(0.17)	(3.36)	(3.97)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	0.16**	0.17*	4.88*	5.86*
	(0.06)	(0.07)	(2.19)	(2.74)
Mean outcome	2.5	2.5	73.27	73.27
First stage F-statistic	14.25	20.14	14.25	20.14
Weather controls	x	x	x	x
Socio-economic controls		x		x

This table reports reduced form estimates that indicate the health effect of LEZ implementation (upper panel) and IV estimates from Equation (4) representing the health effect of a one $\mu g/m^3$ increase in PM_{10} levels during the *in utero* period and the first life year (lower panel). The dependent variable is either the number of prescriptions per child (left side) or their costs in Euro per child (right side) that accumulate over the first five years of a child's life on average. It refers to either prescriptions for respiratory diseases in general or asthma specifically. The dependent variable is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $9,609$.
$p < .05$, ** $p < .01$, *** $p < .001$.

pre-treatment average of 14.14 prescriptions per child reveals a relative reduction of 3.9%. Likewise, the coefficient in column (4) shows that the costs of prescriptions decrease by 10.54 Euro (4.8%) over the first five years of a child's life on average. The relative reductions are even higher in magnitude when considering the subset of prescriptions for asthma. While the number of prescriptions decreases by about 6.8%, the costs decrease by about 8.0% on average. Moreover, comparing the estimated coefficients for respiratory diseases in general and asthma specifically, we find that about 56% (5.86/10.54) of the cost savings accrue due to changes in chronic asthma diseases, while about 31% (0.17/0.55) of the reduction in the number of prescriptions is attributable to asthma. AR confidence intervals (CIs) for the IV-estimates reported in Table A.2 of the Appendix corroborate that all average effects are positive and significantly different from zero.

Based on the reported results we approximate the total cost savings from LEZ protection during the *in utero* period and the first year of life. The most recent birth statistics tell us that $1,836,434$ children are protected by LEZs *in utero* and in the 12 months after birth. Multiplying this number with 14.41 Euro in cost savings per child, the first reduced form coefficient in column (4), we find that treatment reduces long-run pharmaceutical costs by approximately 26.5 million Euro in children born until 2017.[25] Similarly, the calculation linked to asthma medication highlights cost savings of about 14.7 million Euro.

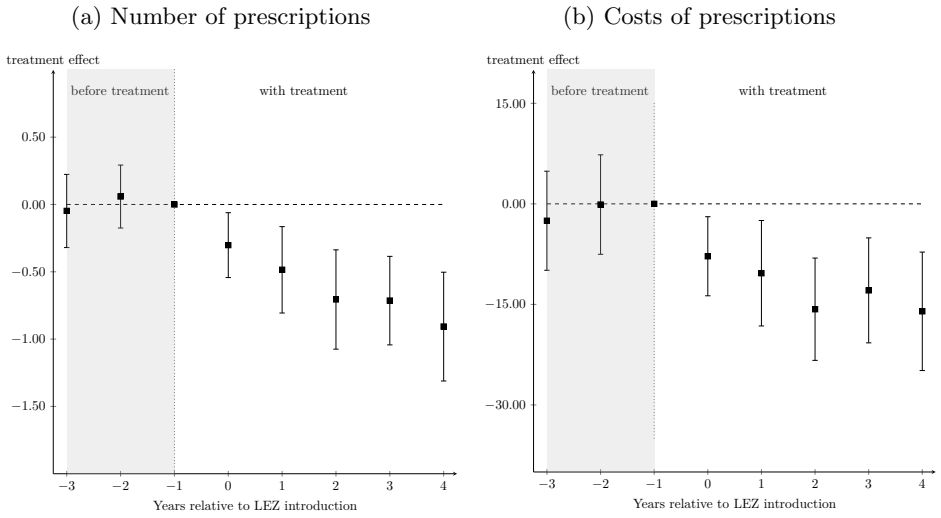
These specific savings represent an important component for a cost-benefit analysis of the policy but only a fraction of the total health benefits that need to be included. In particular, our study considers only new-born children rather than the whole population, purely physical health not accounting for consequential effects on human capital formation, and only prescriptions rather than doctor visits, hospitalizations, and fatalities. Moreover, the estimated savings derive exclusively from early-exposure effects measured until school enrollment. Benefits that persist over half a decade, however, are unlikely to cease suddenly with enrollment. Nonetheless, the estimated 26.5 million Euro already account for about 22% of the up-front costs of owners of vehicles that fail to meet LEZ standards calculated by Rohlf et al. (2020). In combination with other empirically identified LEZ-benefits, namely reduced hospitalizations (Pestel and Wozny 2019), ambulatory care claims (Margaryan 2021), and prescriptions (Rohlf et al. 2020) in the general population, it is realistic to conclude that LEZ-costs can be recovered within a few years. Our IV estimates may also serve to approximate the counterfactual situation of implementing the WHO's PM_{10} guideline of 20 $\mu g/m^3$ already in 2008. Holding constant health benefits at 10.54 Euro per child per $\mu g/m^3$ and assuming all children born into LEZ-counties until 2017 had benefited, the 6.4 $\mu g/m^3$ PM_{10} reduction from the annual mean of 26.4 $\mu g/m^3$ could potentially have reduced longer-run pharmaceutical costs by about 6.4×10.54 Euro $\times$ $2,301,305$ children $\approx$ 155 million Euro.

Figure 4 plots the event-study results for the number and the costs of prescriptions for respiratory diseases.[26] They allow us to examine how LEZs affect long-run health depending on a child's age at the time of exposure. Because the year prior to LEZ implementation is the reference category, we essentially test for differential effects of exposure relative to exposure at age one and older. Therefore, the significant post-treatment decrease in the number and

[25] This back-of-the-envelope calculation does not account for potential differences between those insured with AOK and the general population and is based on the assumption that children born after 2012 benefit equally from the policy.

[26] Table A.1 in the Appendix provides the full regression results.

Figure 4: Event-study Estimates of LEZ Effects on Medication for Respiratory Diseases



The figure presents event-study coefficients based on the specification in Equation (C.1) that show how LEZs affect the medication of respiratory diseases depending on the time between birth and LEZ implementation in years. The dependent variable is the number of prescriptions per child (Panel A) or their costs in Euro per child (Panel B) that accumulate over the first five years of a child's life on average. The gray shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

the costs of prescriptions suggests additional benefits of exposure to cleaner air between conception and age one relative to exposure at age one and later. Also note that significant pre-treatment coefficients could indicate differential benefits from exposure beginning at age two or three relative to exposure at age one or that the common trends assumption is violated. The fact that pre-treatment coefficients are close to zero, suggests that neither of the two scenarios apply. The event-study plots for asthma prescriptions are similar to the ones for respiratory diseases in general (see Figure A.2 in the Appendix).

Disaggregation by Year of Life

Using outcome measures that aggregate medication over the first five years of life neither reveal whether health effects are persistent nor whether their intensity is constant over time. To gain further insights in how early pollution exposure propagates through early life, we analyze the pharmaceuticals prescribed in each of the five years separately in Panel A of Table 4. Because numbers and costs of prescriptions have shown to exhibit very similar behavior with respect to LEZ implementation, we focus on the number of prescriptions from here on.

With regards to respiratory diseases in general, the reduced form and the IV estimations consistently reveal that early pollution exposure persistently and statistically significantly affects medication. For instance, the IV estimates indicate that a one $\mu g/m^3$ decrease in PM_{10} exposure *in utero* and in the first year of life decreases the average number of prescriptions per child in each of the five pre-school years. However, the coefficient in year five is statistically significant only at the 10% level. Contemporaneous reductions in medication needs in the first year of life account for only about 20% of the cumulative savings on prescriptions over the first five years of life (0.11/0.55). With regards to asthma related prescriptions, this share reduces to 6% (0.01/0.17). Moreover, the contemporaneous effect on asthma medication remains statistically insignificant and effects occur only from the second year on. This suggests that it requires time for improvements in chronic diseases to materialize. Note that the sum of the coefficients estimated for each individual pre-school year is identical to the cumulative effect over all five years presented previously and presented again in column (1) of Table 4.

Table 4: The Effect of Early-Life Exposure to PM_{10} on Medication of Respiratory Diseases by Year of Life and at the Extensive and the Intensive Margin

	A. Total						B. Extensive Margin						C. Intensive Margin					
	Number of prescriptions per child						Share of sufferers						Number of prescriptions per sufferer					
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*	*(8)*	*(9)*	*(10)*	*(11)*	*(12)*	*(13)*	*(14)*	*(15)*	*(16)*	*(17)*	*(18)*
	year 1-5	year 1	year 2	year 3	year 4	year 5	year 1-5	year 1	year 2	year 3	year 4	year 5	year 1-5	year 1	year 2	year 3	year 4	year 5
							Reduced Form Estimation											
Resp. diseases	-0.75***	-0.15**	-0.23***	-0.20***	-0.10*	-0.08	-0.02**	-0.02*	-0.02*	-0.01*	-0.01*	-0.01	-0.58***	-0.08	-0.18***	-0.18***	-0.07	-0.07
	(0.18)	(0.05)	(0.06)	(0.05)	(0.04)	(0.04)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.16)	(0.05)	(0.04)	(0.05)	(0.04)	(0.04)
Mean outcome	14.14	2.17	3.34	2.94	3.08	2.60	0.79	0.71	0.84	0.81	0.82	0.77	17.69	3.03	3.97	3.63	3.73	3.33
Asthma	-0.23**	-0.02	-0.08**	-0.06**	-0.04*	-0.02	-0.01**	0.00	-0.02*	-0.02***	-0.01	-0.01	-0.33	-0.07	-0.14	-0.06	-0.04	-0.02
	(0.08)	(0.02)	(0.03)	(0.02)	(0.02)	(0.02)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.01)	(0.28)	(0.06)	(0.08)	(0.07)	(0.08)	(0.09)
Mean outcome	2.50	0.50	0.67	0.50	0.46	0.38	0.22	0.24	0.28	0.22	0.20	0.16	11.32	2.05	2.38	2.26	2.31	2.33
							IV Estimation											
Resp. diseases	0.55**	0.11**	0.17**	0.14**	0.07*	0.06	0.01**	0.02*	0.02*	0.01*	0.01*	0.00	0.42**	0.06	0.13**	0.13**	0.05	0.05
	(0.17)	(0.04)	(0.05)	(0.04)	(0.03)	(0.03)	(0.00)	(0.01)	(0.01)	(0.01)	(0.00)	(0.00)	(0.15)	(0.04)	(0.04)	(0.04)	(0.03)	(0.03)
Mean outcome	14.14	2.17	3.34	2.94	3.08	2.60	0.79	0.71	0.84	0.81	0.82	0.77	17.69	3.03	3.97	3.63	3.73	3.33
Asthma	0.17*	0.01	0.06*	0.04**	0.03	0.02	0.01*	0.00	0.02*	0.02**	0.01	0.01	0.24	0.05	0.10	0.04	0.03	0.02
	(0.07)	(0.02)	(0.02)	(0.02)	(0.02)	(0.01)	(0.00)	(0.01)	(0.01)	(0.01)	(0.01)	(0.00)	(0.21)	(0.05)	(0.06)	(0.05)	(0.06)	(0.07)
Mean outcome	2.50	0.50	0.67	0.50	0.46	0.38	0.22	0.24	0.28	0.22	0.20	0.16	11.32	2.05	2.38	2.26	2.31	2.33
Weather controls	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x
SE controls	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x

This table reports reduced form estimates (upper panel) and IV estimates (lower panel) for disaggregated health effect in each of the five pre-school years and at the extensive and the intensive margin. The dependent variable is either the number of prescriptions per child (Panel A), the share of children in the cohort that requires at least one prescription (Panel B), or the number of prescriptions per child diagnosed with a disease (Panel C). It either refers to prescriptions for respiratory diseases in general or asthma specifically and it is either aggregated over the first five years of a child's life or given for each year separately. The dependent variable is composition-adjusted for the birth county–birth quarter cell in panel A. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. The regressions are weighted by birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $9,609$.* $p < .05$, ** $p < .01$, *** $p < .001$.

Disentangling the Extensive from the Intensive Margin

We assess how the extensive and the intensive margin drive the overall treatment effect in Panel B and C of Table 4. The dependent variable in Panel B is the share of children in the cohort that require at least one prescription for either a respiratory disease or asthma specifically (i.e. extensive margin). The dependent variable in Panel C is the average number of prescriptions a child diagnosed with any respiratory disease or asthma specifically requires (i.e. intensive margin).

For respiratory diseases in general, our IV estimate shows that a one $\mu g/m^3$ decrease in PM_{10} pollution *in utero* and in the first year of life reduces the share of sufferers by one percentage point (column 7). This finding is statistically significant at the 5% level. In addition, the lower pollution level decreases the number of prescriptions in children diagnosed with any kind of respiratory disease on average by about 0.42 over the five pre-school years (column 13). For asthma specifically, we find a one percentage point reduction in the share of children newly diagnosed with asthma (column 7). The prescription requirements of children suffering from asthma, however, are not affected in a statistically significant way (column 13), which may reflect that existing chronic diseases need constant treatment. Because the medication of respiratory diseases comprises that of asthma and because we find clear treatment effects at the intensive margin for respiratory diseases in general, our findings suggest that lower PM_{10} levels have positive effects on children's respiratory health beyond asthma. While we cannot positively identify these effects, we are able to make tentative inferences. For example, Beatty and Shimshack (2014) find that air pollution affects upper respiratory infections such as sinusitis and lower respiratory infections such as acute bronchitis or acute bronchiolitis.

Note that the estimated coefficients for the extensive and intensive margin confirm the overall effect presented in column (1) of Table 4. For instance, for asthma the overall effect given by the IV-coefficient in column (1) is approximated by the sum of the product of the extensive margin coefficient in column (7) and the pre-treatment mean at the intensive margin in column (13) and the product of the intensive margin coefficient in column (13) and the pre-treatment mean at the extensive margin in column (7) ($0.01 \times 11.32 + 0.24 \times 0.22 \approx 0.17$).[27] Moreover, analyzing the health effects at the extensive and intensive margin for each year

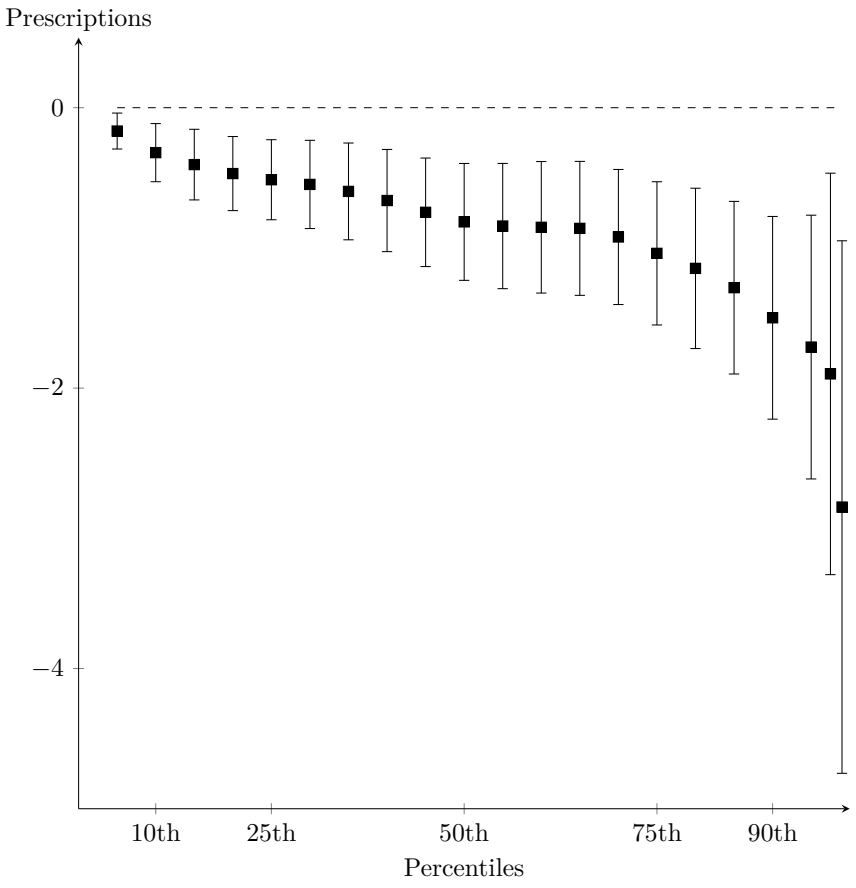
[27] The formal relation is given as $\frac{d(P/N)}{d(x)} = \frac{d(P/S)}{d(x)} \times \frac{S}{N} + \frac{d(S/N)}{d(x)} \times \frac{P}{S}$, where P is the number of prescriptions per child over the five pre-school years, N is the total number of children in the cohort and S is the share of children in the cohort suffering from the disease.

separately, we mostly observe initially latent effects that become prominent only after the first year of life. This finding once more suggests that the health stock adjusts slowly.

Our estimates provide the first dose-response relationship between PM pollution in early-life and health for the years of the "missing middle". While our findings are not directly comparable to related epidemiological and economic research, we provide context for their magnitude in Appendix F. Moreover, we show that our results are robust to controlling the false discovery rate following Benjamini and Hochberg (1995) for the 76 hypotheses we test (Table A.11 in the Appendix).

Because the degree of suffering may vary substantially among children afflicted by respiratory diseases, we deepen our analysis of heterogeneous treatment effects at the intensive margin by

Figure 5: Unconditional Quantile Treatment Effects of LEZs on Medication for Respiratory Diseases



The figures present coefficients from unconditional quantile regressions (Firpo et al. 2009) at the level of the individual child (Appendix E for further information). The dependent variable is the number of prescriptions for respiratory diseases that accumulate over the first five years of a child's life. The severity of suffering increases from left to right. The bars indicate the 95% confidence interval.

applying unconditional quantile regressions (Firpo et al. 2009) at the level of the individual child. For computational tractability, the estimation is based on a reduced-form standard DID.[28] Our estimates in Figure 5 provide suggestive evidence that children who suffer worst from respiratory diseases may benefit the most from LEZs. For example, with 2.8 fewer prescriptions, point estimates for children in the 99th percentile indicate that they may benefit nearly fives times as much from LEZs as the average sufferer with 0.58 prescriptions (Table 4).[29]

5.3 Common Infant Health Measures

Health effects may be subtle if changes in pollution exposure are small and health effects may be latent if the health stock only adjusts slowly. Our results indicate that this could be the case in the context of LEZ implementation. To assess this hypothesis further, we estimate the effect of a one $\mu g/m^3$ decrease in PM_{10} exposure during the prenatal period on fetal development. The outcome variables are postpartum stationary hospitalization due to abnormal birth weight, unusual pregnancy duration, and fetal malnutrition.[30] The estimated treatment effects in Table 5 remain statistically insignificant. Given our findings for respi-

Table 5: Severe Effects of Exposure to PM_{10} During the Perinatal Period on Fetal Development

	IV Estimation		
	Hospital treatments per 1,000 children		
	developmental disorder and malnutrition	short gestation period and low birth weight	long gestation period and high birth weight
	(1)	*(2)*	*(3)*
Mean PM_{10} ($\mu g/m^3$)	-0.03	1.50	0.12
	(0.61)	(2.49)	(0.39)
Mean outcome	2.93	50.22	1.14
Weather controls	x	x	x
Socio-economic controls	x	x	x

This table reports coefficient estimates from three variants of the IV regression in Equation (4). The dependent variable is the number of stationary hospital treatments of three different disorders linked to abnormal fetal development per 1,000 children. It is the composition-adjusted average for a birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, LEZ wave–treated fixed effects, as well as weather and socio-economic controls. The regressions are weighted by birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The first stage F-statistic is 24.39. The sample size is 9,609.
* $p < .05$, ** $p < .01$, *** $p < .001$.

[28] For further information on the unconditional quantile estimator see Appendix E.
[29] Table A.3 in the Appendix features all coefficients and standard errors.
[30] As before, the sample comprises three cohorts born prior to and all cohorts born up to five years subsequent to LEZ implementation. However, we exclude the first three cohorts born after implementation which are only partially protected by LEZs *in utero*.

ratory health, this indicates that infant health measures revolving around hospitalizations immediately postpartum may be too coarse or too focused on the short-term to detect health effects. Moreover, it might explain why prior studies on LEZs could not provide evidence for improved infant health measured in the form of low birth weight (Gehrsitz 2017, Pestel and Wozny 2019).

5.4 Effect Mechanisms

Different mechanisms may explain the effects of LEZs on pollution and health. First, overall traffic could have decreased because of mode switching to public transport. Second, individuals could have substituted their banned vehicles with "greener" vehicles. To assess these two potential channels we draw on additional annual data at the county level provided by the Federal Highway Research Agency (BAST) and the German Federal Motor Transport Authority (KBA). The BAST data provides information on the number of passing vehicles on all freeways (*Autobahnen*) and federal roads (*Bundesstraßen*) recorded by traffic monitors. The KBA data provides information on the number of registered private and commercial passenger vehicles by fuel type (diesel and gasoline) and emission class.

Table 6 shows that LEZ implementation has no statistically significant effect on the traffic volume on an average day (column 1).[31] However, it reveals a shift in the composition of the vehicle fleet. As expected, we observe a significant 20% decrease ($e^{0.18} - 1$) in the number of old diesel vehicles classified as Euro1 and lower that are banned by the LEZs (column 2). Even vehicles with emission classes Euro2 through Euro4 decrease significantly (column 3). The banned vehicles seem to be replaced by used gasoline vehicles with the emission standards Euro2 through Euro4 (column 6). We find no significant changes in the number of the newest diesel and gasoline cars (column 4 and 6).

In combination, these findings provide tentative evidence on the emission source of the policy-induced PM_{10}-reduction. Because overall traffic is not affected, particles from wear and tear of brakes, tires, and road surfaces are unlikely to have changed upon LEZ implementation. The "de-dieselization" of the vehicle fleet, however, suggests that after implementation the same amount of traffic is caused by fewer dirty diesel vehicles and more cleaner gasoline vehicles.[32] Thus, diesel exhaust may be the main driver of the observed PM_{10}-reductions.

[31] Given that LEZs usually cover city centers, it is possible that traffic from within the LEZ shifts to other, unregulated areas of the county. While the data from BAST do not allow a closer examination, Wolff (2014) and Gehrsitz (2017) provide evidence against this hypothesis.

[32] Diesel vehicles with Euro1 may emit up to 140 μg tailpipe PM_{10} per km. Gasoline vehicles with Euro2-4 emit about 1-2 μg PM_{10} per km according to HBEFA.

Table 6: The Effect of LEZ Implementation on Traffic Volume and the Vehicle Fleet

	Traffic volume (vehicles/24 hours)	Diesel vehicles Euro1 and worse	Diesel vehicles Euro2-4	Diesel vehicles Euro5 and better	Gasoline vehicles Euro1	Gasoline vehicles Euro2-4	Gasoline vehicles Euro5 and better
	(1)	*(2)*	*(3)*	*(4)*	*(5)*	*(6)*	*(7)*
LEZ treatment	-0.01	-0.18***	-0.03*	-0.01	0.04	0.02**	-0.01
	(0.02)	(0.02)	(0.01)	(0.05)	(0.06)	(0.01)	(0.15)
Sample size	5,584	6,514	6,514	5,418	6,514	6,514	5,418
Weather controls	x	x	x	x	x	x	x
Socio-economic controls	x	x	x	x	x	x	x

The dependent variable is the number of motor vehicles counted over the 24 hours of an average day in column (1), the number of vehicles with a diesel engine in the emission class Euro1 or worse in column (2), in emission class Euro2 through Euro4 in column (3), and in emission class Euro5 or better in column (4), and the number of vehicles with a gasoline engine in emission class Euro1 in column (5), in emission class Euro2 through Euro4 in column (6), and in emission class Euro5 or better in column (7). The sample in column (4) and (7) is limited to the period after 2008 because the emission class Euro5 was only introduced in 2009. All outcome variables are transformed with the inverse hyperbolic sine function. Accordingly, the percentage change in the outcome variable is given by $(e^{\beta} - 1) \cdot 100$. All regressions include county, state–year, LEZ wave–event time, LEZ wave–treated fixed effects, as well as weather and socio-economic controls. The weather variables comprise only precipitation, temperature, and sunshine duration because they are available for the entire period in which we observe the BAST and KBA data. The regressions are weighted by the average cohort size. Standard errors in parentheses are clustered at the county level.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Toxicological and epidemiological studies suggest that PM from diesel exhaust is particularly harmful because it mainly consists of small particles that can penetrate far into the human body (Krzyżanowski et al. 2005, HEI 2010).

5.5 Robustness Checks

Alternative Control Groups

Changes in the composition of the control group do not alter our results. Our baseline estimates rely on a control group that comprises counties that implement LEZs at some point and counties that violate EU PM_{10} standards but have no LEZ to date. Neither of the event-study plots in Figure 3 or 4 point to differential trends in air pollution and health outcomes across the treatment and control groups before policy introduction. This alleviates concerns that LEZ adoptions are determined by any omitted local conditions or shocks that simultaneously affect air pollution and health outcomes. Nonetheless, we additionally restrict our sample to counties that actually introduce LEZs to ensure that our results are not determined by deliberate selection of counties into treatment. Recall that our full sample comprises 128 counties of which 65 actually implement LEZs; 49 between 2008 and 2012. Figure A.3 in the Appendix shows event-study estimates based on the sample of the 65 ever-adopter counties. Although the restriction to ever-adopters nearly slashes our sample in half, the event-study

plots for the first stage and the reduced form continue showing well-behaved patterns that are similar to those in Figure 3 and 4.

In our preferred DID setting, we observe cohorts born up to five years after treatment and exclude all cohorts from counties that implement LEZs in this time period from the control group in each of the stacked data sets. By choosing a five-year time window after treatment, we are able to identify longer-run effects of LEZ introduction. However, the length of the time window has implications for the composition of the control group. The shorter the window, the more comparable are the control and the treatment group. This is because more control units that introduce an LEZ with close proximity in time are eligible to the control group. In Table A.4 in the Appendix we show that treatment effects remain robust when shortening the time window after treatment sequentially from five to two years.

Spillover effects

Some of the counties in our control group directly neighbor counties implementing an LEZ. These counties may be subject to positive or negative spillovers. This would bias our estimated effects on pollution and health. In particular, we are concerned that drivers may change their routes to circumnavigate the LEZ such that traffic would merely be displaced. In this case, LEZs would have worsened air pollution in neighboring counties and our estimates would overestimate the policy‘s effectiveness. To address this concern, Table 7 replicates the estimations from Tables 2 and 3. The only difference is that we include a binary variable that takes on a value of 1 if a neighboring county implements an LEZ.[33]

We find negative but statistically insignificant policy effects in neighboring counties with regard to either PM_{10} pollution, prescriptions for respiratory diseases, or asthma. In line with these findings, Wolff (2014) and Gehrsitz (2017) show that treatment effects on pollution measuring stations outside of the LEZs are negative but insignificant. However, when controlling for neighboring counties, the effect of LEZs on air pollution within LEZ counties is -2.18 (Table 7) while our main estimate is -1.37 (Table 2). The reduced form effects of LEZs on the number and costs of prescriptions are also higher when controlling for neighboring counties compared to our preferred estimates in Table 3, the only exception being the coefficient for the number of prescriptions for asthma. However, the IV effects remain almost

[33] In alternative tests, we exclude all neighboring counties from each treatment wave in the sample and account for the number of neighboring LEZs when estimating spillover effects. In either case, the results remain almost identical.

Table 7: Air Pollution and Health Effects on Neighboring Counties

	PM_{10} Pollution	Number of prescriptions		Costs of prescriptions	
	($in\ \mu g/m^3$)	Respiratory diseases	Asthma	Respiratory diseases	Asthma
	(1)	*(2)*	*(3)*	*(4)*	*(5)*
[0.2cm]		A. Reduced Form Estimation			
LEZ effect on	-2.18***	-1.09***	-0.24*	-23.10***	-14.34**
LEZ-counties	(0.53)	(0.28)	(0.12)	(6.38)	(5.39)
LEZ effect on	-0.41	-0.16	-0.03	-1.56	-0.54
neighbor-counties	(0.25)	(0.13)	(0.07)	(3.56)	(2.83)
		B. IV Estimation			
PM_{10} Pollution	-	0.49**	0.11	10.34**	6.38*
		(0.15)	(0.06)	(3.47)	(2.73)
Weather controls	x	x	x	x	x
Socio-economic controls	x	x	x	x	x

This table replicates the first stage regression in Table 2 and the reduced form and IV regressions in Table 3. In addition, the treatment effect on counties adjacent to those that implement an LEZ is estimated. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects, as well as weather and socio-economic controls. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is 7,665. * $p < .05$, ** $p < .01$, *** $p < .001$.

unchanged given that the difference in the estimated effects on pollution and health are of similar proportion.

While we cannot recover statistically significant effects on neighboring counties, the higher magnitude of the reduced form treatment effects when controlling for neighboring counties indicates that positive spillover effects are likely present. This conjecture is corroborated when testing whether the policy-induced "de-dieselization" of the vehicle fleet identified in Section 5.4 expands across county borders. In line with Wolff (2014), we find that neighboring counties also exhibit a statistically significant reduction (-0.04^*, $t = -2.06$) in the number of banned diesel cars classified as Euro1 and lower. This effect can be plausibly linked to the fact that LEZs cover city centers where many workplaces and points of interest are located. Individuals living in the neighboring counties have an incentive to ensure that their vehicles allow access.

Overall, the findings alleviate concerns about overestimating and instead mark our main analysis as conservative. In fact, when accounting for positive spillover effects the estimated effectiveness of the policy could be considerably greater. While in Section 5.2 we estimate that treatment reduces long-run pharmaceutical costs by approximately 26.5 million Euro in

children born until 2017, the estimates in Table 7 suggest cost savings of 42.4 million Euro ($1,836,434$ children $\times$ 23.10 Euro).

Accounting for Changes in Population Characteristics

A potential threat to identification in our IV design is that improved air quality might change the composition of the cohorts in LEZ counties, leading to changes in the unobservable characteristics of the children born there. This occurs if the implementation of LEZs changes how individuals move in and out of the county which would invalidate the exclusion restriction. For example, if LEZs either attract good health risks or induce bad health risks to locate to other counties, improvements in child health may result from the changed socio-economic structure of counties rather than from reduced pollution. While there exists no evidence in the literature that LEZs affect the socio-economic structure of counties, we nevertheless report a range of additional estimates that strengthen our confidence that the exclusion restriction holds.

First, we analyze whether LEZ implementation significantly affects how individuals migrate in and out of counties (Appendix Table A.5) and we conduct placebo tests (Appendix Table A.6). To estimate treatment effects on mobility patterns we consider net migration in the overall county population, net migration among families, and the fraction of AOK-insured children moving out of their birth county. As placebo health outcomes we use hospital treatments of injuries of the head, the arm, and of other body parts. All outcomes are typical health issues in children and strongly correlated with socio-economic status (e.g. Faelker et al. 2000, Birken and MacArthur 2004, Yates et al. 2006), while the air pollution literature does not indicate a relationship. Table A.5 and Table A.6 in the Appendix do not reveal any statistically significant policy effects on migration patterns and placebo outcomes. However, because the coefficients are estimated imprecisely, we turn to another indirect test on changes in the socio-economic composition of cohorts.

If unobservable population characteristics adjust gradually over time, comparing treated and untreated cohorts will suffer less bias the closer they are in the time dimension. Therefore, we reuse our previous robustness analysis in which we sequentially reduced the five-year time window after treatment to two years. It is unlikely that population characteristics change markedly within just two years. In the absence of gradual changes in unobserved characteristics, the estimates for the limited samples should be close to those in the main

analysis. Table A.4 in the Appendix shows that treatment effects remain indeed robust as the post-treatment time window decreases.

A particular concern that remains unaddressed by the conducted tests is that LEZs could impose an income shock on households that fail to meet the new standards. The policy requires owners of non-compliant vehicles to invest in new cars or retrofitting. This could come with extraordinary costs, potentially raising maternal stress during pregnancy and thereby adversely affecting the long-term health of unborn children. Such a chain of effects would counteract the LEZ-induced health benefits from air pollution reductions. It would lead to downward bias in our estimates.

Effect on Other Air Pollutants

We use PM_{10} as a measure for PM exposure. By definition, PM_{10} includes particles below 10 μm, including the finer $PM_{2.5}$ particles. We rely on PM_{10} because policymakers in Europe are highly focused on this pollutant, and LEZs explicitly target PM_{10}. Moreover, the EU only set legally binding limits for $PM_{2.5}$ in 2015. To evaluate whether LEZs also decrease $PM_{2.5}$, in a first robustness check, we resort to satellite-based $PM_{2.5}$ estimates from van Donkelaar et al. (2019).[34] This data is available on a fine resolution grid (0.01 degrees) but only at the annual level.[35] Thus, we lose quarterly observations and the corresponding fixed effects. Table 8 shows that the introduction of LEZs reduces mean $PM_{2.5}$ concentrations by about 2%. The relatively modest magnitude compared to that for PM_{10} may reflect attenuation bias from non-classical measurement error.

Our second robustness check is motivated by the fact that diesel vehicles emit significant quantities of nitrogen oxides. In fact, road traffic emissions of nitrogen dioxide (NO_2), which serve as an indicator for different nitrogen oxides, are caused primarily by diesel vehicles.[36] Therefore, we assess whether LEZ implementation also leads to notable changes in ambient NO_2 concentrations using our data from the German air monitoring network. Table 8 shows that LEZs significantly reduce NO_2 by about 3.27 $\mu g/m^3$ (8.0%) on average. This finding is consistent with the fact that LEZs are *de facto* bans of old diesel vehicles.

[34] Data from the German air monitoring network for $PM_{2.5}$ is very limited. We have about 70% fewer observations for $PM_{2.5}$ than for PM_{10}.

[35] van Donkelaar et al. (2019) merge satellite measurements of aerosol optical depth with a particulate transport model, and combine them with data from air monitoring stations to obtain estimates of $PM_{2.5}$ for Europe.

[36] In Germany, about 72.5% of NO_2 emissions from on-road traffic are from diesel vehicles (UBA 2017).

Table 8: The Effect of LEZ Implementation on Different Pollutants

	$PM_{2.5}$	NO_2	O_3	SO_2
	(1)	(2)	(3)	(4)
LEZ treatment	-0.30*	-3.27**	-0.04	-0.12
	(0.12)	(1.07)	(0.56)	(0.25)
Mean outcome	15.51	40.81	39.27	4.3
First stage F-statistic	5.79	9.44	0.01	0.24
Weather controls	x	x	x	x
Socio-economic controls	x	x	x	x

This table reports coefficient estimates for the effect of LEZs on four different air pollutants. The dependent variable is either the mean concentration of $PM_{2.5}$, NO_2, O_3, or SO_2 in $\mu g/m^3$. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $3,466$ in column (1) and $9,609$ in columns (2) through (4).
* $p < .05$, ** $p < .01$, *** $p < .001$.

Because LEZs reduce both PM and NO_2, we cannot conclusively infer that PM_{10} determines our observed health effects alone. Therefore, we caution against interpreting our IV results as a causal estimate of the health effects of PM_{10} as a stand-alone pollutant. Instead, we argue that our IV results represent health effects linked to air pollutants from diesel vehicles. Similarly, other papers generalize their results to air pollution effects (c.p. Chay and Greenstone 2003a, Currie and Neidell 2005, Arceo et al. 2016, Knittel et al. 2016, Deryugina et al. 2019, Colmer et al. 2020). We subsequently show that the policy does not affect other pollutants.

First, we examine whether LEZs have unintended effects on ozone (O_3) concentrations. O_3 is negatively correlated with other local air pollutants, in particular with NO_2 which is one of its precursors. Therefore, we may be concerned that the implementation of LEZs increases O_3 concentrations. However, Table 8 does not provide evidence for an unintended increase in O_3. Second, environmental regulation can have adverse impacts on firm output and productivity. Therefore, we may be concerned that LEZs decrease industrial activity and, thereby, reduce emissions of industrial pollutants, most notably SO_2. To rule out that health effects are subject to this channel, we also estimate effects for SO_2 concentrations. Table 8 does not reveal any statistically significant effects. Because transport only accounts for about 2% of total SO_2 emissions, this robustness check also serves as a placebo test, suggesting that our first-stage results are not driven by confounding factors.

Accounting for Treatment Differences After the First Year of Life

In our main analysis, we compare children who experience different pollution exposure levels *in utero* and over their first year of life but the same exposure levels afterwards. A strict implementation of this comparison requires that we restrict ourselves to cohorts born exactly four quarters prior to LEZ implementation. However, to avoid limiting ourselves to a single pre-treatment observation of the treated, we additionally include the two cohorts born five and six quarters prior to treatment in our main analysis. The drawback of this approach is that it neglects potentially different exposure levels in the second year of the children's lives. However, our event-study estimates in Figure 4 do not indicate additional benefits from exposure at age one relative to exposure at age two and three. As a further robustness check, we limit pre-treatment observations to cohorts born four quarters before implementation. The results in Table A.7 in the Appendix show that our findings are robust with respect to this sample restriction. The reduced form and the IV point estimates are very similar in magnitude to the ones reported in Table 3.

Accounting for Increases in Policy Stringency

Our treatment estimate identifies the effect of LEZ introduction. Upon implementation, LEZs ban the most emission-intensive diesel vehicles with tailpipe emission category Euro1 or lower (no sticker). However, LEZs become more stringent over time so that they eventually also ban vehicles with Euro2 (red sticker) and Euro3 (yellow sticker) standards. This gradual adoption of more stringent restrictions raises the concern that children born after LEZ implementation benefit from cleaner air for longer throughout their pre-school years than children born just before LEZ implementation. This would imply that children in our pre- and post-treatment comparison differ not only in exposure during their *in utero* period and their first year of life. Reassuringly, our event-study results for the number and the costs of prescriptions in Figure 4 provide no evidence that differences in exposure after age one have any additional health benefits. If there were differential benefits from exposure beginning at age two or three relative to exposure at age one, we would expect positive pre-treatment coefficients. Instead, we observe statistically insignificant coefficient estimates close to zero. As a further robustness check we enrich our event study specification for prescriptions in Equation C.1 by eight additional binary variables: $\sum_{y=2}^{5} f_{cy}^{Euro2} + \sum_{y=2}^{5} f_{cy}^{Euro3}$, where f_{cy}^{Euro2} (f_{cy}^{Euro3}) is equal to one if Euro2 (Euro3) vehicles are banned in year of life y of cohort c. These dummy variables should absorb potential additional health benefits in year of life two through five linked to

the two more stringent LEZ regimes banning Euro2 and Euro3 vehicles, respectively. If there were additional benefits from Euro2 and 3 vehicle bans, we would expect post-treatment coefficients to decrease in event time.[37] However, Figure A.4 in the Appendix exhibits very similar patterns to our event study in Figure 4 that lacks the additional dummies capturing changes in stringency. This suggests there are no additional benefits from the increasing stringency of LEZs.

Two-way Fixed Effect DID Estimation

We also estimate the two-way fixed effect equivalent of our stacked difference-in-differences estimator. The coefficient estimates in Table A.8 show similar, robust effects of PM_{10} on child health. However, they tend to be lower in magnitude. For instance, the IV coefficient for respiratory diseases is 0.55 in the stacked DID estimation in column (2) of Table 3 while it is only 0.39 in the two-way fixed effect DID estimation in Table A.8. We expect that part of this attenuation stems from the weighted aggregation of heterogeneous treatment effects revealed in Goodman-Bacon (2018). It may also indicate violations in common trends that result from including already-treated units in the control groups for the newly treated, although they are on differential trends from prior treatment. Also note that it is impossible in the two-way fixed effect setup to include fixed effects that absorb implementation wave-specific unobservables in event-time and time-invariant differences between treatment and control groups within and across implementation waves.

Functional form

We also test the robustness of our results with respect to the functional form. The outcome variables in our baseline specifications are in levels. Using per capita prescriptions as outcome, we implicitly assume that prescriptions per child would have evolved with the same absolute changes in the absence of treatment. However, if prescriptions per child changed at the same rate in the absence of any LEZ intervention instead, the parallel trends assumption would be violated. Although our event-study plots do not reveal pre-trends that differ in a statistically significant manner, we re-estimate our main results with logged outcome variables in Table A.9 in the Appendix. We find that the estimated relative effects are nearly identical to the ones derived from Table 3.

[37] We do not expect shifts in post-treatment patterns because the timing of Euro2 and 3 vehicle bans varies across counties relative to the implementation date.

6 Conclusion

This paper provides a quasi-experimental study that links moderate improvements in air quality in a single year from banning emission-intensive vehicles to substantial health benefits across children's pre-school years. The context of our study are urban counties in Germany, where motor vehicles are a major source of air pollution. Yet, average pre-treatment pollution levels are low. These characteristics are widespread in Europe, so that our results are most likely generalizable. Exploiting unique public health insurance data at the patient level on one million children, we examine whether individuals born just before and just after reductions in PM concentrations caused by the adoption of Low Emission Zones exhibit persistent differences in rarely studied medication use up to five years after treatment. We focus on children's pharmaceutical prescriptions as a sensitive, real-time health measure that overcomes the challenge of capturing health effects that may be both subtle if changes in pollution exposure are moderate and initially latent if the health stock adjusts slowly.

We present strong evidence that the cumulative number and the cumulative costs of pharmaceutical prescriptions over early childhood decrease significantly after LEZ implementations improve air quality. For instance, the number of prescriptions for asthma decreases by 6.8% and their costs decrease by about 8.0% on average for every one $\mu g/m^3$ reduction in PM_{10} concentration. Our findings provide strong support for the notion of health as a stock that changes relatively slowly over time. It is only from the second year of life that the initially latent health response materializes in fewer children diagnosed with asthma and, thus, fewer total prescriptions. Contemporaneous reductions in prescriptions of asthma medication account for less than 6% of the cumulative savings over the first five years of life. This highlights that estimates of contemporaneous pollution impacts may substantially underestimate 'fully formed' health benefits. We identify economically meaningful cost savings for public health insurers. With $1,836,434$ children protected by Low Emission Zones *in utero* and during their first year of life, treatment reduces costs for prescriptions in children born between 2008 and 2017 for respiratory diseases by about 26.5 million Euros over their pre-school years, or 42.4 million Euros when accounting for positive spillover effects. Because we compare children who differ only in their pollution exposure during their *in utero* period and their first year of life, these cost savings originate from a very short period with slightly improved air quality.

Our results inform contentious policy debates. Across Europe, vintage- and fuel-specific driving bans are a widespread and ever more stringent policy intervention. Major metropolitan areas, including Paris, Madrid, and Rome, are even committed to full diesel bans by 2025. Yet, opponents of driving restrictions prominently question whether a narrow focus on diesel bans is a rational choice to effectively improve air quality and public health. Our study seeks to provide first answers by quantifying an important fraction of the reduction in the public health burden accomplished by LEZs - Germany's flagship policy at the local level to comply with air quality standards set by the EU clean air directives. Finding meaningful health improvements, even at low pre-treatment pollution levels, suggests that vintage-specific driving bans that target particularly old and emission intensive diesels can have large and long-lasting positive effects on children's respiratory health in many settings.

Our study cannot assess whether additional restrictions for newer vehicles would yield further health improvements. This is an important policy question for future research. Another research question is how treatment effects progress through life. In our study, health effects are latent in the first year before materializing. It is far from obvious whether health effects persist permanently and how they impede cognitive or non-cognitive skill formation. To address the progression of effects, we would need to follow the children in our study and rerun our estimations at later points in time, for example at the end of elementary school, at the end of high school, and some years into their professional lives.

References

Abraham, S. and L. Sun (2020). Estimating Dynamic Treatment Effects in Event Studies with Heterogeneous Treatment Effects. *Journal of Econometrics*.

Albouy, D. (2009). What Are Cities Worth? Land Rents, Local Productivity, and the Capitalization of Amenity Values. *NBER Working Paper* (14981), pp. 1–32.

Alexander, D. and H. Schwandt (2019). The Impact of Car Pollution on Infant and Child Health: Evidence from Emissions Cheating. *Working Paper No. WP-2019-4*.

Almond, D. and J. Currie (2011). Killing Me Softly: The Fetal Origins Hypothesis. *Journal of Economic Perspectives* 25(3), pp. 153–72.

Almond, D., J. Currie, and V. Duque (2018). Childhood Circumstances and Adult Outcomes: Act II. *Journal of Economic Literature* 56(4), pp. 1360–1446.

Andrews, I., J. H. Stock, and L. Sun (2019). Weak Instruments in Instrumental Variables Regression: Theory and Practice. *Annual Review of Economics* 11(1), pp. 727–753.

Angrist, J. and V. Lavy (2009). The Effects of High Stakes High School Achievement Awards: Evidence from a Randomized Trial. *American Economic Review* 99(4), pp. 1384–1414.

AOK (2018). *Zahlen und Fakten, 2018*. `https://aok-bv.de/imperia/md/aokbv/aok/zahlen/zuf_2018_ppt_final.pdf`. Accessed: 2021-04.

Arceo, E., R. Hanna, and P. Oliva (2016). Does the Effect of Pollution on Infant Mortality Differ Between Developing and Developed Countries? Evidence from Mexico City. *The Economic Journal* 126(591), pp. 257–280.

Athey, S. and G. W. Imbens (2018). Design-based Analysis in Difference-in-differences Settings with Staggered Adoption. *NBER Working Paper* (24963).

Baccarelli, A. and V. Bollati (2009). Epigenetics and Environmental Chemicals. *Current Opinion in Pediatrics* 21(2), pp. 243–251.

Barrot, J.-N. and J. Sauvagnat (2016). Input Specificity and the Propagation of Idiosyncratic Shocks in Production Networks. *The Quarterly Journal of Economics* 131(3), pp. 1543–1592.

Barwick, P. J., S. Li, D. Rao, and N. B. Zahur (2018). The Morbidity Cost of Air Pollution: Evidence from Consumer Spending in China. *NBER Working Paper* (24688).

Beatty, T. K. and J. P. Shimshack (2014). Air Pollution and Children's Respiratory Health: A Cohort Analysis. *Journal of Environmental Economics and Management* 67(1), pp. 39–57.

Benjamini, Y. and Y. Hochberg (1995). Controlling the False Discovery Rate: a Practical and Powerful Approach to Multiple Testing. *Journal of the Royal Statistical Society: Series B (Methodological)* 57(1), pp. 289–300.

Bento, A., M. Freedman, and C. Lang (2015). Who Benefits From Environmental Regulation? Evidence From the Clean Air Act Amendments. *Review of Economics and Statistics* 97(3), pp. 610–622.

Bharadwaj, P., J. G. Zivin, J. T. Mullins, and M. Neidell (2016). Early-Life Exposure to the Great Smog of 1952 and the Development of Asthma. *American Journal of Respiratory and Critical Care Medicine* 194(12), pp. 1475–1482.

Bharadwaj, P., M. Gibson, J. G. Zivin, and C. Neilson (2017). Gray Matters: Fetal Pollution Exposure and Human Capital Formation. *Journal of the Association of Environmental and Resource Economists* 4(2), pp. 505–542.

Birken, C. S. and C. MacArthur (2004). Socioeconomic Status and Injury Risk in Children. *Paediatrics & Child Health* 9(5), pp. 323–325.

Cengiz, D., A. Dube, A. Lindner, and B. Zipperer (2019). The Effect of Minimum Wages on Low-wage Jobs. *The Quarterly Journal of Economics* 134 (2), pp. 1405–1454.

Chay, K. Y. and M. Greenstone (2003a). Air Quality, Infant Mortality, and the Clean Air Act of 1970. *NBER Working Paper* (10053), pp. 1–43.

— (2003b). The Impact of Air Pollution on Infant Mortality: Evidence from Geographic Variation in Pollution Shocks Induced by a Recession. *The Quarterly Journal of Economics* 118(3), pp. 1121–1167.

— (2005). Does Air Quality Matter? Evidence From the Housing Market. *Journal of Political Economy* 113(2), pp. 376–424.

Clark, N. A., P. A. Demers, C. J. Karr, M. Koehoorn, C. Lencar, L. Tamburic, and M. Brauer (2010). Effect of Early Life Exposure to Air Pollution on Development of Childhood Asthma. *Environmental Health Perspectives* 118(2), pp. 284–290. DOI: 10.1289/ehp.0900916.

Colmer, J., D. Lin, S. Liu, and J. Shimshack (2020). Why are Pollution Damages Lower in Developed Countries? Insights from High Income, High-Pollution Hong Kong. *CEP Discussion Paper* (1702).

Currie, J. and M. Neidell (2005). Air Pollution and Infant Health: What Can We Learn from California's Recent Experience? *The Quarterly Journal of Economics* 120(3), pp. 1003–1030.

Currie, J. and R. Walker (2011). Traffic Congestion and Infant Health: Evidence from E-ZPass. *American Economic Journal: Applied Economics* 3(1), pp. 65–90.

Currie, J., M. Neidell, and J. F. Schmieder (2009). Air Pollution and Infant Health: Lessons from New Jersey. *Journal of Health Economics* 28(3), pp. 688–703.

Currie, J., J. Zivin Graff, J. Mullins, and M. Neidell (2014). What Do We Know About Short- and Long-Term Effects of Early-Life Exposure to Pollution? *Annual Review of Resource Economics* 6(1), pp. 217–247.

Currie, J., L. Davis, M. Greenstone, and R. Walker (2015). Do Housing Prices Reflect Environmental Health Risks? Evidence from More than 1600 Toxic Plant Openings and Closings. *American Economic Review* 105(2), pp. 678–709.

Cyrys, J., A. Peters, J. Soentgen, and H.-E. Wichmann (2014). Low Emission Zones Reduce PM10 Mass Concentrations and Diesel Soot in German Cities. *Journal of the Air & Waste Management Association* 64(4), pp. 481–487.

De Chaisemartin, C. and X. d'Haultfoeuille (2020). Two-way Fixed Rffects Rstimators with Heterogeneous Treatment Effects. *American Economic Review* 110(9), pp. 2964–96.

Dehejia, R. and A. Lleras-Muney (2004). Booms, Busts, and Babies' Health. *The Quarterly Journal of Economics* 119(3), pp. 1091–1130.

Deng, Q., C. Lu, C. Ou, L. Chen, and H. Yuan (2016). Preconceptional, Prenatal and Postnatal Exposure to Outdoor and Indoor Environmental Factors on Allergic Diseases/Symptoms in Preschool Children. *Chemosphere* 152, pp. 459–467.

Deryugina, T., G. Heutel, N. H. Miller, D. Molitor, and J. Reif (2019). The Mortality and Medical Costs of Air Pollution: Evidence from Changes in Wind Direction. *American Economic Review* 109(12), pp. 4178–4219.

Deschênes, O., M. Greenstone, and J. S. Shapiro (2017). Defensive Investments and the Demand for Air Quality: Evidence from the NOx Budget Program. *American Economic Review* 107(10), pp. 2958–89.

Deshpande, M. and Y. Li (2019). Who is Screened Out? Application Costs and the Targeting of Disability Programs. *American Economic Journal: Economic Policy* 11(4), pp. 213–48.

Donald, S. G. and K. Lang (2007). Inference with Difference-in-Differences and Other Panel Data. *The Review of Economics and Statistics* 89(2), pp. 221–233.

Fadlon, I. and T. H. Nielsen (2019). Family Health Behaviors. *American Economic Review* 109(9), pp. 3162–3191.

Faelker, T., W. Pickett, and R. J. Brison (2000). Socioeconomic Differences in Childhood Injury: a Population Based Epidemiologic Study in Ontario, Canada. *Injury Prevention* 6(3), pp. 203–208.

Fanta, C. (2009). Drug Therapy: Asthma. *New England Journal of Medicine* 360(10), pp. 1002–1014.

Firpo, S., N. M. Fortin, and T. Lemieux (2009). Unconditional Quantile Regressions. *Econometrica* 77(3), pp. 953–973.

Gehrsitz, M. (2017). The Effect of Low Emission Zones on Air Pollution and Infant Health. *Journal of Environmental Economics and Management* 83, pp. 121–144.

Gluckman, P. D., M. A. Hanson, C. Cooper, and K. L. Thornburg (2008). Effect of in Utero and Early-Life Conditions on Adult Health and Disease. *New England Journal of Medicine* 359(1), pp. 61–73.

Goodman-Bacon, A. (2018). Difference-in-Differences with Variation in Treatment Timing. *NBER Working Paper* (25018), pp. 1–46.

Häussler, B. and A. Höer (2016). *Arzneimittel-Atlas 2018: Der Arzneimittelverbrauch in der GKV*. MWV Medizinisch Wissenschaftliche Verlagsgesellschaft.

He, J., N. Gouveia, and A. Salvo (2018). External Effects of Diesel Trucks Circulating inside the São Paulo Megacity. *Journal of the European Economic Association* 17(3), pp. 947–989.

HEI (2010). *Traffic-related Air Pollution: a Critical Review of the Literature on Emissions, Exposure, and Health Effects*. Health Effects Institute. Panel on the Health Effects of Traffic-Related Air Pollution.

Hoffmann, F. and D. Koller (2015). Different Regions, Differently Insured Populations? Socio-demographic and Health-related Differences Between Insurance Funds. *Gesundheitswesen* 79(1), e1–e9.

Holt, P. G. (1998). Programming for Responsiveness to Environmental Antigens That Trigger Allergic Respiratory Disease in Adulthood Is Initiated during the Perinatal Period. *Environmental Health Perspectives* 106, pp. 795–800.

Hoynes, H., D. W. Schanzenbach, and D. Almond (2016). Long-run Impacts of Childhood Access to the Safety Net. *American Economic Review* 106(4), pp. 903–34.

Isen, A., M. Rossin-Slater, and W. R. Walker (2017). Every Breath You Take - Every Dollar You'll Make: The Long-Term Consequences of the Clean Air Act of 1970. *Journal of Political Economy* 125(3), pp. 848–902.

Jaeger, D. A., T. J. Joyce, and R. Kaestner (2020). A Cautionary Tale of Evaluating Identifying Assumptions: Did Reality TV Really Cause a Decline in Teenage Childbearing? *Journal of Business & Economic Statistics* 38(2), pp. 317–326.

Jaunzeme, J., S. Eberhard, and S. Geyer (2013). Wie repräsentativ sind GKV-Daten? *Bundesgesundheitsblatt-Gesundheitsforschung-Gesundheitsschutz* 56(3), pp. 447–454.

Karlsson, M. and N. R. Ziebarth (2018). Population Health Effects and Health-Related Costs of Extreme Temperatures: Comprehensive Evidence from Germany. *Journal of Environmental Economics and Management* 91, pp. 93–117.

KBA (2017). *Pressemitteilung Nr. 01/2017 - Fahrzeugzulassungen im Dezember 2016 - Jahresbilanz*. `https://www.kba.de/DE/Presse/Pressemitteilungen/2016_2019/2017/Fahrzeugzulassungen/pm01_2017_n_12_16_pm_komplett.html`. Accessed: 2020-09-01.

Khreis, H., C. Kelly, J. Tate, R. Parslow, K. Lucas, and M. Nieuwenhuijsen (2017). Exposure to Traffic-Related Air Pollution and Risk of Development of Childhood Asthma: A Systematic Review and Meta-Analysis. *Environment International* 100, pp. 1–31.

Knittel, C. R., D. L. Miller, and N. J. Sanders (2016). Caution, Drivers! Children Present: Traffic, Pollution, and Infant Health. *Review of Economics and Statistics* 98(2), pp. 350–366.

Krzyżanowski, M., B. Kuna-Dibbert, and J. Schneider (2005). *Health Effects of Transport-Related Air Pollution*. WHO Regional Office Europe, pp. 1–190.

Lee, D. L., J. McCrary, M. J. Moreira, and J. Porter (2020). Valid t-ratio Inference for IV. *arXiv preprint arXiv:2010.05058*.

Li, N., C. Sioutas, A. Cho, D. Schmitz, C. Misra, J. Sempf, M. Wang, T. Oberley, J. Froines, and A. Nel (2003). Ultrafine Particulate Pollutants Induce Oxidative Stress and Mitochondrial Damage. *Environmental Health Perspectives* 111(4), pp. 455–460.

Lindo, J. M. (2011). Parental Job Loss and Infant Health. *Journal of Health Economics* 30(5), pp. 869–879.

Liu, W., C. Huang, Y. Hu, Q. Fu, Z. Zou, C. Sun, L. Shen, X. Wang, J. Cai, J. Pan, Y. Huang, J. Chang, Y. Sun, and J. Sundell (2016). Associations of Gestational and Early Life Exposures to Ambient Air Pollution with Childhood Respiratory Diseases in Shanghai, China: A Retrospective Cohort Study. *Environment International* 92-93, pp. 284–293. ISSN: 0160-4120. DOI: 10.1016/j.envint.2016.04.019.

Malina, C. and F. Scheffler (2015). The Impact of Low Emission Zones on Particulate Matter Concentration and Public Health. *Transportation Research Part A: Policy and Practice* 77, pp. 372–385.

Margaryan, S. (2021). Low Emission Zones and Population Health. *Journal of Health Economics* 76, p. 102402.

Mitteldeutsche Zeitung (2011). Umweltzone startet auch in Halle zum 1. September. 2020-07-09.

Pestel, N. and F. Wozny (2019). Low Emission Zones for Better Health: Evidence from German Hospitals. *IZA Discussion Paper No. 12545*.

Rohlf, A., F. Holub, N. Koch, and N. Ritter (2020). The Effect of Clean Air on Pharmaceutical Expenditures. *Economics Letters* 192, p. 109221.

Sager, L. (2019). Estimating the Effect of Air Pollution on Road Safety Using Atmospheric Temperature Inversions. *Journal of Environmental Economics and Management* 98, p. 102250.

Sanders, N. J. (2012). What Doesn't Kill You Makes You Weaker: Prenatal Pollution Exposure and Educational Outcomes. *Journal of Human Resources* 47(3), pp. 826–850.

Sanders, N. J. and C. Stoecker (2015). Where Have All the Young Men Gone? Using Sex Ratios to Measure Fetal Death Rates. *Journal of Health Economics* 41, pp. 30–45.

Shapiro, J. S. and R. Walker (2018). Why is Pollution from US Manufacturing Declining? The Roles of Environmental Regulation, Productivity, and Trade. *American Economic Review* 108(12), pp. 3814–54.

Simeonova, E., J. Currie, P. Nilsson, and R. Walker (2019). Congestion Pricing, Air Pollution and Children's Health. *Journal of Human Resources*, 0218–9363R2.

Šrám, R. J., B. Binková, J. Dejmek, and M. Bobak (2005). Ambient Air Pollution and Pregnancy Outcomes: a Review of the Literature. *Environmental Health Perspectives* 113(4), pp. 375–382.

Swart, E., P. Ihle, H. Gothe, and D. Matusiewicz (2005). *Routinedaten im Gesundheitswesen: Handbuch Sekundärdatenanalyse: Grundlagen, Methoden und Perspektiven*. 2nd ed. Huber, pp. 1–533.

UBA (2017). *Stickoxid-Belastung durch Diesel-Pkw noch höher als gedacht*. `https://www.umweltbundesamt.de/presse/pressemitteilungen/stickoxid-belastung-durch-diesel-pkw-noch-hoeher`. Accessed: 2019-01.

— (2019). *Luftmessnetz: Wo und Wie wird Gemessen?* `https://www.umweltbundesamt.de/themen/luftmessnetz-wo-wie-wird-gemessen`. Accessed: 2019-01.

U.S. DOT (2015). *Diesel-powered Passenger Cars and Light Trucks*. `https://www.bts.gov/archive/publications/bts_fact_sheets/oct_2015/entire`. Accessed: 2020-09.

Van Donkelaar, A., R. V. Martin, C. Li, and R. T. Burnett (2019). Regional Estimates of Chemical Composition of Fine Particulate Matter Using a Combined Geoscience-Statistical Method with Information from Satellites, Models, and Monitors. *Environmental Science & Technology* 53(5), pp. 2595–2611.

Wiesbadener Tagblatt (2011). Stadt muss Umweltzone einführen; 2011-10-11.

Williams, A. M. and D. J. Phaneuf (2019). The Morbidity Costs of Air Pollution: Evidence from Spending on Chronic Respiratory Conditions. *Environmental and Resource Economics*, pp. 1–33.

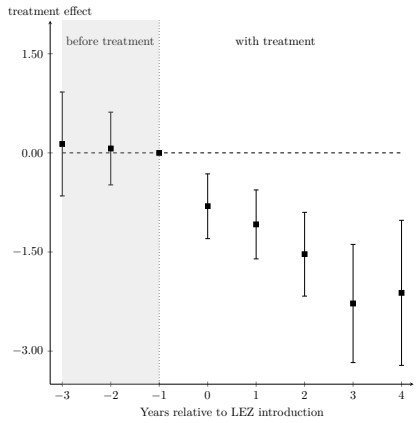
Wolff, H. (2014). Keep Your Clunker in the Suburb: Low-Emission Zones and Adoption of Green Vehicles. *The Economic Journal* 124(578), F481–F512.

Yates, P. J., W. H. Williams, A. Harris, A. Round, and R. Jenkins (2006). An Epidemiological Study of Head Injuries in a UK Population Attending an Emergency Department. *Journal of Neurology, Neurosurgery and Psychiatry* 77(5), pp. 699–701.

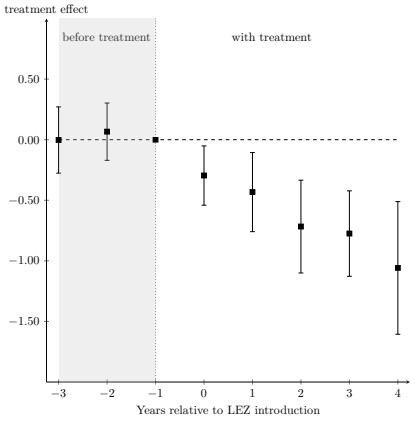
A Additional Figures and Tables

Figure A.1: Event-study Estimates of LEZ Effects - Excluding All Already Treated Counties from the Control Group

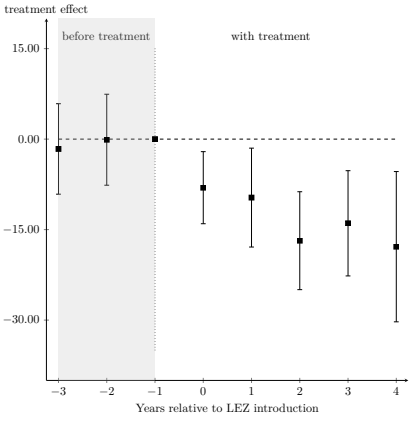
(a) PM_{10}



(b) Number of prescriptions

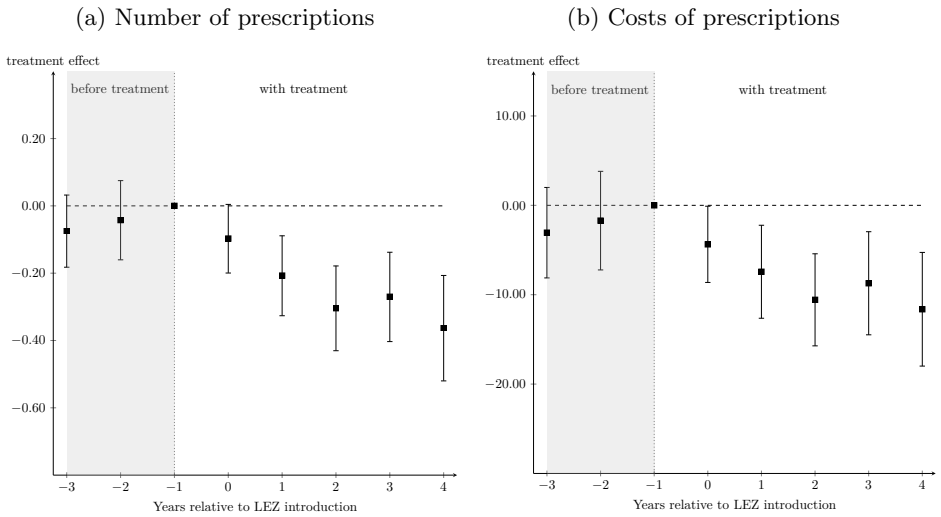


(c) Costs of prescriptions



The figure presents event-study coefficients that show how LEZs affect PM_{10} concentration and the medication of respiratory diseases for a sample that excludes all already treated counties from the control group. The dependent variable is the mean quarterly PM_{10} level (Panel a), the number (Panel b) or the costs (Panel c) of prescriptions that accumulate over the first five years of a child's life on average. The grey shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

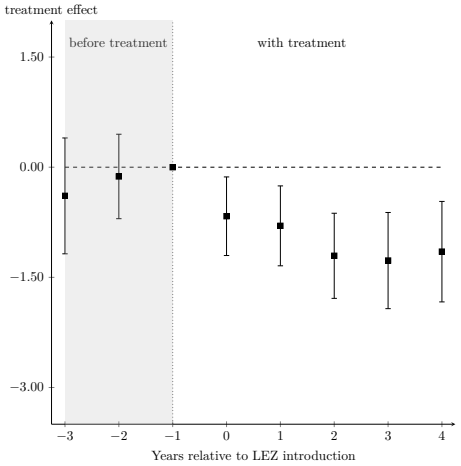
Figure A.2: Event-study Estimates of LEZ Effects on Medication for Asthma



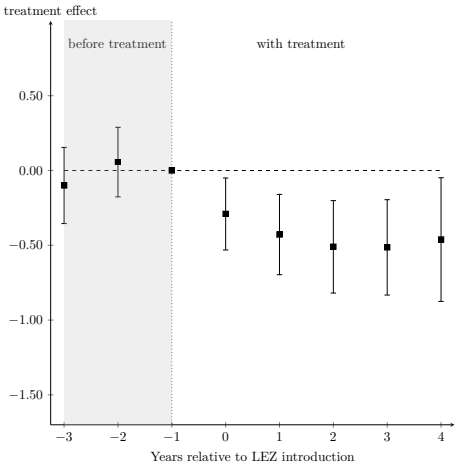
The figure presents event-study coefficients that show how LEZs affect the medication of asthma depending on the time distance between birth and LEZ implementation in years. The dependent variable is the number (Panel a) or the costs (Panel b) of prescriptions that accumulate over the first five years of a child's life on average. The grey shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

Figure A.3: Event-study Estimates Excluding the Never Treated

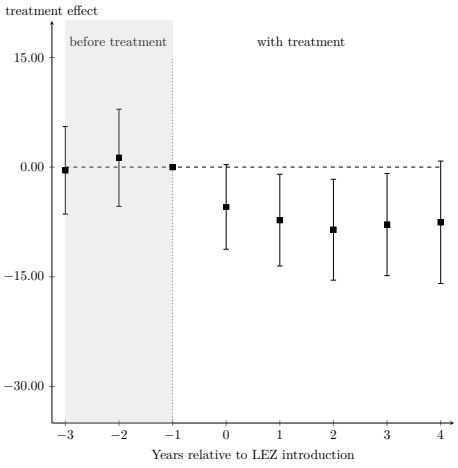
(a) PM_{10}



(b) Number of prescriptions

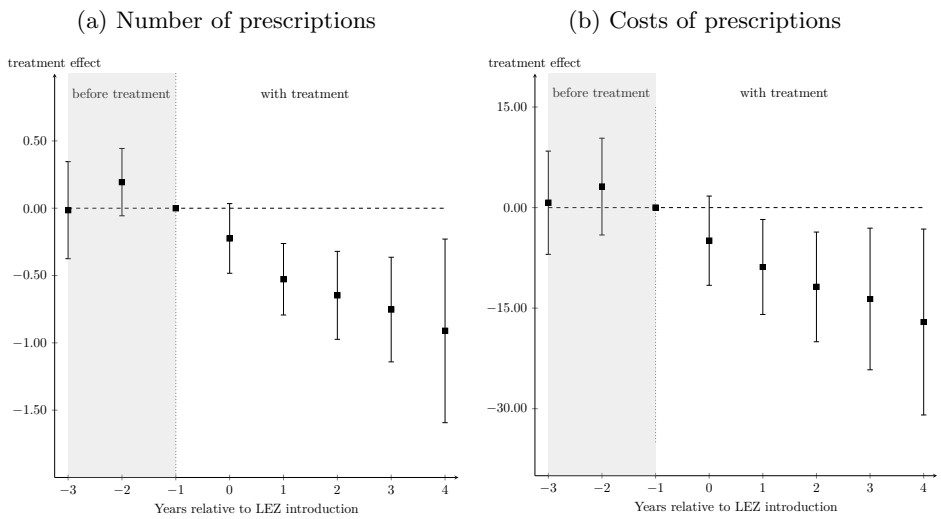


(c) Costs of prescriptions



The figure presents event-study coefficients that show how LEZs affect PM_{10} pollution and the medication of respiratory diseases for a sample that excludes all never treated counties from the control group. The dependent variable is the average PM_{10} level in $\mu g/m^3$ (Panel a), the number (Panel b), or the costs (Panel c) of prescriptions that accumulate over the first five years of a child's life on average. We reduce the time window that defines our control group to 4 years post-treatment and 3 years pre-treatment to avoid that the control groups for the later treated become very small. The grey shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

Figure A.4: Event-study Estimates of LEZ Effects - Accounting for Increases in Policy Stringency



The figure presents event-study coefficients that show how LEZs affect the medication of respiratory diseases based on equation C.1 enriched by eight additional dummies $\sum_{y=2}^{5} f_{cy}^{Euro2} + \sum_{y=2}^{5} f_{cy}^{Euro3}$, which should absorb potential additional health benefits in life years two to five linked to the two more stringent LEZ regimes banning Euro 2 and Euro 3 vehicles, respectively. The dependent variable is either the number (Panel a) or the costs of prescriptions (Panel b) that accumulate over the first five years of a child's life on average. The grey shaded area indicates the pre-treatment period. The coefficient in the year prior to implementation is normalized to zero. The regression includes county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, LEZ wave–treated fixed effects as well as weather and socio-economic controls. It is weighted by the county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

Table A.1: Event-study Estimates – The Effect of LEZ Implementation on PM_{10} Concentrations and Health Outcomes by Year

	PM_{10} Pollution ($\mu g/m^3$)	Number of prescriptions	Costs of prescriptions
	(1)	*(2)*	*(3)*
LEZ treatment ($\theta = -2$))	0.026	-0.079	-3.094
	(0.411)	(0.137)	(3.659)
LEZ treatment ($\theta = -1$))	0.007	0.034	-0.455
	(0.284)	(0.116)	(3.694)
LEZ treatment ($\theta = 1$))	-0.896***	-0.304*	-8.006**
	(0.254)	(0.122)	(2.969)
LEZ treatment ($\theta = 2$))	-1.269***	-0.480**	-10.13*
	(0.249)	(0.166)	(3.969)
LEZ treatment ($\theta = 3$))	-1.613***	-0.674***	-14.897***
	(0.289)	(0.189)	(3.852)
LEZ treatment ($\theta = 4$))	-2.040***	-0.675***	-12.34**
	(0.387)	(0.17)	(3.967)
LEZ treatment ($\theta = 5$))	-1.929***	-0.788***	-14.192**
	(0.388)	(0.200)	(4.243)
Weather controls	x	x	x
Socio-economic controls	x	x	x

This table reports estimated event-study coefficients underlying Figure 3 and Figure 4. The dependent variables are the average PM_{10} level in $\mu g/m^3$, the number or the costs in Euro of prescriptions that accumulate over the first five years of a child's life on average, respectively. The dependent variables are composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. The regressions are weighted by the birth county–birth quarter cell size. They are based on an expanded pre-treatment window including all observations up to three years prior to LEZ implementation. Furthermore, observations from the 3 quarters before and the 3 quarters after LEZ implementation are included. The resulting sample size is 19,290. Standard errors in parentheses are clustered at the county level.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.2: Anderson Rubin Confidence Sets

	A. Number of prescriptions		B. Costs of prescriptions (€)	
	Respiratory diseases			
CS_{AR}	[0.25 - 1.03]	[0.28 - 1.04]	[3.01 - 19.31]	[4.07 - 22.07]
F_{AR}	15.11	16.72	9.41	11.10
p-value	0.0001	0.0000	0.0022	0.0009
	Asthma			
CS_{AR}	[0.06 - 0.33]	[0.06 - 0.34]	[1.05 - 11.44]	[1.20 - 13.48]
F_{AR}	8.83	9.04	6.07	6.18
p-value	0.0030	0.0026	0.0138	0.0129
Weather controls	x	x	x	x
Socio-economic controls		x		x

This table reports weak-instrument-robust inference for the IV-estimates in Table 3. The AR-confidence sets (CS_{AR}) provide robust confidence intervals with a coverage probability of 95%. The F-distributed AR-statistic (F_{AR}) and its p-value test the null hypothesis that the coefficient of the endogenous variable PM_{10} in the structural equation is equal to zero.

Table A.3: Unconditional Quantile Regression Estimates of the Effect of Early-Life Exposure to LEZs on Respiratory Prescriptions throughout Childhood

	(1) Q-5	*(2)* Q-10	*(3)* Q-15	*(4)* Q-20	*(5)* Q-25	*(6)* Q-30	*(7)* Q-35
LEZ treatment	-0.166^{*}	-0.310^{**}	-0.406^{**}	-0.470^{***}	-0.514^{***}	-0.547^{***}	-0.597^{***}
	(0.065)	(0.106)	(0.129)	(0.135)	(0.146)	(0.161)	(0.176)
	(8) Q-40	*(9)* Q-45	*(10)* Q-50	*(11)* Q-55	*(12)* Q-60	*(13)* Q-65	*(14)* Q-70
LEZ treatment	-0.662^{***}	-0.746^{***}	-0.814^{***}	-0.844^{***}	-0.853^{***}	-0.861^{***}	-0.922^{***}
	(0.186)	(0.197)	(0.213)	(0.228)	(0.239)	(0.244)	(0.246)
	(15) Q-75	*(16)* Q-80	*(17)* Q-85	*(18)* Q-90	*(19)* Q-95	*(20)* Q-97.5	*(21)* Q-99
LEZ treatment	-1.039^{***}	-1.146^{***}	-1.284^{***}	-1.498^{***}	-1.707^{***}	-1.899^{**}	-2.848^{**}
	(0.261)	(0.292)	(0.314)	(0.369)	(0.480)	(0.730)	(0.969)

This table reports regression coefficients from unconditional quantile regressions. The dependent variable is the number of prescriptions for respiratory diseases that accumulate over the first five years of a child's life. All regressions include birth county and birth state–birth quarter fixed effects. Standard errors in parentheses are clustered at the county level. The sample size is $556,898$. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.4: The Effect of Early-Life PM_{10} Exposure – Different Post-Treatment Time Windows

	(1) $\Delta = 2$	*(2)* $\Delta = 3$	*(3)* $\Delta = 4$
		A. First Stage Estimation	
Mean PM_{10}	-1.14***	-1.53***	-1.55***
	(0.3)	(0.3)	(0.3)
Mean outcome	26.44	26.44	26.44
		B. Reduced Form Estimation	
Respiratory diseases	-0.44**	-0.50**	-0.62***
	(0.16)	(0.17)	(0.17)
Mean outcome	14.14	14.14	14.14
Asthma	-0.16*	-0.21**	-0.22**
	(0.07)	(0.07)	(0.07)
Mean outcome	2.5	2.5	2.5
		C. IV Estimation	
Respiratory diseases	0.39*	0.32*	0.40**
	(0.16)	(0.13)	(0.12)
Mean outcome	14.14	14.14	14.14
Asthma	0.14*	0.13**	0.14**
	(0.07)	(0.05)	(0.05)
Mean outcome	2.5	2.5	2.5
First stage F-statistic	14.50	25.92	27.19
Sample size	6,922	8,727	9,575
Weather controls	x	x	x
Socio-economic controls	x	x	x

This table reports coefficient estimates for shorter post-treatment windows. The time window increases sequentially from two years (column 1) to four years (column 3). Panel A presents coefficients from first stage, Panel B the coefficients from reduced form and Panel C coefficients from IV estimations. The dependent variable in Panel A is the PM_{10} concentration; in panel B and C it is the number of prescriptions for respiratory diseases in general or asthma specifically that accumulate over the first five years of a child's life on average. The dependent variable in Panel B and C is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.5: The Effect of LEZ Implementation on Migration Patterns

	(1) Total net migration	(2) Net migration among families	(3) Movers among AOK children
LEZ treatment	0.66	-0.07	-0.56
	(0.47)	(0.55)	(0.33)
Mean outcome	2.93	-1.86	9.44
Weather controls	x	x	x
Socio-economic controls	x	x	x

This table reports coefficient estimates for the effect of LEZ implementation on county migration. The dependent variable in column (1) is net migration per 1,000 inhabitants of the total county population. The dependent variable in column (2) is net migration per 1,000 inhabitants of the total county population younger than 18 years old and 30 to 50 years old. The dependent variable in column (3) is the fraction of AOK-insured children moving out of the birth county after their first and before their sixth year of life in percent. All columns include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. Observations of LEZ counties in the three quarters prior and subsequent to implementation are included. The regressions are weighted by the birth county–birth quarter cell size. Standard errors are clustered at the county level and are in parentheses. The sample size is $12,865$. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.6: The Effect of Early-Life PM_{10} Exposure on Placebo Health Outcomes (per 1,000 children)

	(1) Arm injuries	(2) Head injuries	(3) Several injuries
		A. Reduced Form	
LEZ treatment	0.58	-0.97	-0.09
	(1.04)	(4.64)	(0.11)
Mean outcome	5.7	68.81	0.12
		B. IV Estimation	
PM_{10} mean	-0.43	0.71	0.06
	(0.75)	(3.39)	(0.08)
Mean outcome	5.7	68.81	0.12
Weather controls	x	x	x
Socio-economic controls	x	x	x

This table reports reduced form estimates (Panel A) and IV estimates (Panel B) for three different placebo health outcomes. The dependent variable is either the number of stationary hospital treatments of arm injuries, head injuries or injuries involving several body parts, that accumulate over the first five years of a child's life on average and per 1,000 children. The dependent variable is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects as well as weather and socio-economic controls. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $9,609$.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.7: The Effect of Early-Life PM_{10} Exposure when limiting pre-treatment observations to cohorts born four quarters before implementation

	A. Number of prescriptions		B. Costs of prescriptions (€)	
	(1)	*(2)*	*(3)*	*(4)*
			Reduced Form Estimation	
Respiratory diseases	-0.63**	-0.78**	-10.85*	-16.02*
	(0.21)	(0.25)	(4.70)	(6.70)
Mean outcome	13.94	13.94	213.11	213.11
Asthma	-0.30***	-0.31**	-9.29**	-13.12**
	(0.08)	(0.1)	(3.53)	(4.87)
Mean outcome	2.46	2.46	71.96	71.96
			IV Estimation	
Respiratory diseases	0.49*	0.49*	8.48	10.2*
	(0.22)	(0.21)	(4.51)	(5.13)
Mean outcome	13.94	13.94	213.11	213.11
Asthma	0.23*	0.20*	7.26*	8.35*
	(0.09)	(0.08)	(3.59)	(3.9)
Mean outcome	2.46	2.46	71.96	71.96
First stage F-statistic	9.05	12.75	9.05	12.75
Weather controls	x	x	x	x
Socio-economic controls		x		x

This table reports reduced form estimates (Panel A) and IV estimates (Panel B) for health effects when we use only cohorts born four quarters prior to LEZ implementation as pre-treatment observations of the treated. The dependent variable is the number of prescriptions that accumulate over the first five years of a child's life on average. It refers to either prescriptions for respiratory diseases in general or asthma specifically. The dependent variable is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $7,893$.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.8: The Effect of Early-Life PM_{10} Exposure on Medication of Respiratory Diseases throughout Early Childhood - Two-Way Fixed Effect Estimation

	A. Number of prescriptions		B. Costs of prescriptions (€)	
	(1)	*(2)*	*(3)*	*(4)*
		Reduced Form Estimation		
Respiratory diseases	-0.50***	-0.52**	-9.13**	-11.83**
	(0.15)	(0.18)	(2.97)	(3.80)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	-0.18**	-0.23**	-6.04**	-8.43**
	(0.06)	(0.07)	(2.14)	(2.67)
Mean outcome	2.5	2.5	73.27	73.27
		IV Estimation		
Respiratory diseases	0.41**	0.39*	7.53*	8.84**
	(0.16)	(0.15)	(3.03)	(3.32)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	0.15**	0.17**	4.99*	6.30**
	(0.06)	(0.06)	(2.09)	(2.35)
Mean outcome	2.5	2.5	73.27	73.27
First stage F-statistic	13.26	17.23	13.26	17.23
Weather controls	x	x	x	x
Socio-economic controls		x		x

This table replicates our main results in Table 3 using two-way fixed effect estimation. The dependent variable is either the number (panel A) or the costs in Euro (panel B) of prescriptions that accumulate over the first five years of a child's life on average. It refers to either prescriptions for respiratory diseases in general or asthma specifically. In each panel, coefficients from reduced form and IV estimations are presented. The dependent variable is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county and birth state–birth quarter fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $2,904$.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.9: The Effect of Early-Life PM_{10} Exposure on Medication of Respiratory Diseases throughout Early Childhood - Logged outcomes

	A. Number of prescriptions		B. Costs of prescriptions (€)	
	(1)	*(2)*	*(3)*	*(4)*
	Reduced Form Estimation			
Respiratory diseases	-0.05**	-0.06***	-0.06**	-0.07**
	(0.02)	(0.02)	(0.02)	(0.02)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	-0.09**	-0.10**	-0.11*	-0.12*
	(0.03)	(0.04)	(0.05)	(0.05)
Mean outcome	2.5	2.5	73.27	73.27
	IV Estimation			
Respiratory diseases	0.04**	0.04**	0.05**	0.05*
	(0.01)	(0.01)	(0.02)	(0.02)
Mean outcome	14.14	14.14	218.62	218.62
Asthma	0.07**	0.07*	0.08*	0.09*
	(0.02)	(0.03)	(0.04)	(0.04)
Mean outcome	2.5	2.5	73.27	73.27
First stage F-statistic	14.25	20.14	14.25	20.14
Weather controls	x	x	x	x
Socio-economic controls		x		x

This table replicates our main results in Table 3 using logged outcome variables. The dependent variable is either the number (panel A) or the costs in Euro (panel B) of prescriptions that accumulate over the first five years of a child's life on average. It refers to either prescriptions for respiratory diseases in general or asthma specifically. In each panel, coefficients from reduced form and IV estimations are presented. The dependent variable is composition-adjusted for the birth county–birth quarter cell. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $9,609$.* $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.10: The Effect of LEZ Implementation on PM_{10} Concentrations - no IDW interpolation

	First Stage Estimation	
	PM_{10} Pollution $(in\ \mu g/m^3)$	
	(1)	*(2)*
LEZ treatment	-1.30**	-1.50***
	(0.41)	(0.40)
Mean outcome	27.26	27.26
First stage F-statistic	10.27	14.12
Weather controls	x	x
Socio-economic controls		x

This table replicates our main results in Table 2 when we do not interpolate the pollution data but include only counties with own measuring stations in the sample. The dependent variable is the quarterly mean PM_{10} concentration in a given county and year in $\mu g/m^3$. All columns include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. Weather and socio-economic controls are added sequentially moving from left to right. The regressions are weighted by the birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level. The sample size is $8,286$. * $p < .05$, ** $p < .01$, *** $p < .001$.

Table A.11: Multiple Hypotheses Testing

			respiratory dieseases				asthma			
			reduced form		IV		reduced form		IV	
outcome	table	specification	p	$p(BH)$	p	$p(BH)$	p	$p(BH)$	p	$p(BH)$
prescriptions	3	2	0.000	0.002	0.002	0.011	0.003	0.015	0.012	0.035
costs	3	4	0.001	0.011	0.009	0.027	0.014	0.039	0.034	0.071
precriptions year 1	4	2	0.002	0.011	0.003	0.015	0.423	0.465	0.429	0.465
precriptions year 2	4	3	0.000	0.002	0.002	0.011	0.001	0.011	0.010	0.030
precriptions year 3	4	4	0.000	0.002	0.002	0.011	0.003	0.015	0.008	0.026
precriptions year 4	4	5	0.018	0.044	0.029	0.062	0.036	0.071	0.053	0.092
precriptions year 5	4	6	0.055	0.093	0.092	0.139	0.225	0.289	0.247	0.302
share of sufferers	4	7	0.007	0.026	0.008	0.026	0.008	0.026	0.018	0.044
share of sufferers (year 1)	4	8	0.022	0.053	0.016	0.043	0.945	0.945	0.945	0.945
share of sufferers (year 2)	4	9	0.041	0.077	0.036	0.071	0.023	0.053	0.044	0.079
share of sufferers (year 3)	4	10	0.043	0.077	0.039	0.075	0.001	0.010	0.003	0.015
share of sufferers (year 4)	4	11	0.025	0.056	0.035	0.071	0.064	0.106	0.078	0.123
share of sufferers (year 5)	4	12	0.244	0.302	0.276	0.327	0.068	0.110	0.100	0.149
prescriptions per sufferer	4	13	0.000	0.006	0.007	0.026	0.243	0.302	0.259	0.312
prescriptions per sufferer (year 1)	4	14	0.123	0.167	0.156	0.204	0.290	0.339	0.309	0.356
prescriptions per sufferer (year 2)	4	15	0.000	0.002	0.005	0.019	0.091	0.139	0.104	0.150
prescriptions per sufferer (year 3)	4	16	0.000	0.003	0.004	0.015	0.420	0.465	0.427	0.465
prescriptions per sufferer (year 4)	4	17	0.102	0.149	0.119	0.164	0.617	0.656	0.621	0.656
prescriptions per sufferer (year 5)	4	18	0.114	0.161	0.148	0.197	0.801	0.823	0.801	0.823

This table reports p values for all 76 hypotheses regarding respiratory diseases and asthma tested in Tables 3 and 4. Columns labeled p indicate unadjusted p-values while columns labeled $p(BH)$ indicate p-values adjusted for multiple hypotheses testing following Benjamini and Hochberg (1995).

B Composition-Adjusted Health Outcomes

For the estimation of Equation (4), health outcomes observed at the level of the individual child (H_{ict}) are aggregated to the cohort level (H_{ct}). We define a cohort by its birth county c as well as its birth year and birth quarter t. However, additional information at the level of the individual such as the individual's sex or the precise location of residence within a county at the five-digit zip code is available. To exploit this information, we conduct auxiliary regressions that are commonly used in the literature (e.g. Currie et al. 2015). In a first step, we regress the children's health outcomes on individual-level covariates as well as birth county–birth quarter fixed effects:

$$H_{ict} = I'_{ict}\zeta + \phi_{ct} + \xi_{ict} \tag{B.1}$$

where the dependent variable H_{ict} is the accumulated health outcome over the first five years of life for individual i born in county c and year and quarter t. I'_{ict} is a vector of individual-level covariates that includes gender and location of residence within a county at the five-digit zip code. Additionally, Equation (B.1) controls for a full set of birth county–birth quarter indicators ϕ_{ct}. Their coefficient estimates $\widehat{\phi_{ct}}$ are orthogonal to the covariates at the individual level. In other words, they give the average health outcomes for a birth county–birth quarter cohort after controlling for gender and residence. In line with Isen et al. (2017), we refer to the predicted cohort means obtained by this approach as composition-adjusted. We use the composition-adjusted outcomes as the dependent variable in Equation (4).

The use of composition-adjusted group means is asymptotically equivalent to using the individual level data (e.g. Donald and Lang 2007) if the sampling variance of the composition-adjusted group estimates is taken into account. In accordance with other studies (e.g. Angrist and Lavy 2009, Albouy 2009, Currie et al. 2015, Isen et al. 2017), we estimate all regressions by weighted least squares using the number of individuals in each birth county–birth quarter cell as weights. This is assumed to be a reasonable approximation of weighting by inverse sampling variance. Compared to running regressions on the individual level data, the estimation of models collapsed to the level of variation ensures that tests are of correct size given serial correlation in the within-group errors (Isen et al. 2017). Additionally, it requires substantially less computational power.

C Stacked DID Design

C.1 Control Group Definition

In our main analysis our control group only holds LEZ-counties that implement the policy measure at least four years before or five years after the treatment wave for which they serve as control units. The selection of this exclusion window results from two considerations. First, we expect that the LEZ treatment effect on PM concentrations levels off one year after implementation. Throughout the first year after implementation treated counties may be on a differential trend and are therefore not suited to serve as control units. Second, we want to have a balanced control group. Given our choice of an event time window of up to three years prior and five years subsequent to LEZ implementation in the event study specification, we thus determine the exclusion window to start one year earlier (-4 to +5 years). For instance, the county Mainz serves as a control unit for Mannheim because it implements its own LEZ more than five years later, in 2013. Likewise, Mannheim serves as a control unit for the county Hagen because it implemented its own LEZ already four years earlier (see Figure 1).

The above selection is subject to an important trade-off. The longer the event time window, the longer we can observe deviations from the parallel trend assumption and effects of LEZs post implementation. The shorter the time window the higher the statistical power and the higher the number of LEZ adopters that serve as control observations. Our preferred choice of the event time window is based on the dates of the implementations of the LEZs, the availability of data, and the observation of cumulative benefits over five years. To rule out that this choice drives our results, we provide estimates for alternative time-window specifications in the robustness analysis. We also robustify our assumption, that the treatment effect on PM concentrations levels off one year after implementation. Our results are almost identical if we exclude all already-treated from the control group to allow for persistent dynamics (see Figure A.1).

C.2 Event-Study Specification

We estimate event-study specifications of our stacked DID model. The first stage becomes

$$P_{ctj} = \sum_{\upsilon} \theta_{\upsilon}(Treated_{cj} \times D^{\upsilon}_{tj}) + \sum_{\tau} \delta^{\tau} D^{\tau}_{tj} + \lambda_j D_{cj} + W'_{ctj}\rho + X'_{c\bar{t}j}\pi_t + \gamma_c + \eta_{st} + \omega_{ctj} \qquad \text{(C.1)}$$

$$\text{with} \quad \upsilon = \lfloor \frac{\tau}{4} \rfloor$$

The parameter of interest is θ_{υ}. It captures the marginal effect of LEZs on the mean PM_{10} concentration in year υ prior or post to treatment. We set $\theta_0 = 0$ so that the year prior to LEZ implementation is the reference category. The event-study figures presented in this paper plot the θ_{υ} estimates in event time. The main difference between the standard two-way fixed effect event study and our dynamic estimator in Equation (C.1) is that we eliminate time-invariant unobservables both within and between LEZ implementation waves by including D_{cj} as well as wave-specific event-time trends that do not appear in calendar time by including D^{τ}_{tj}.

We also estimate an event study specification that explores the treatment effect dynamics for the long-run health outcomes. Replacing the dependent variable in Equation (C.1) with H_{ctj} results in a reduced-form event study model. This specification allows us to examine how LEZs affect long-run health depending on a child's age at the time of exposure. Because the year prior to LEZ implementation is the reference category, we essentially test for differential effects of exposure relative to exposure at age one and older.

D Data

D.1 Control Variables

The following table gives an overview of the county-specific control variables used in the estimations. We observe cohorts over a five-year period and we include weather controls for all of these years. The 2007 values of socio-economic demographic controls are categorized in terciles and interacted with year-quarter dummies in the regressions.

Variable	Definition	Source
	Weather Controls	
Precipitation	Sum of the precipitation height in mm	DWD
Sunshine	Total sunshine duration in hours	DWD
Temperature	Mean, minimum and maximum temperature, 12 separate terms that count the number of days with temperatures above 0, 5, 10, 15, 20, 25, 29, 30, 31, 32, 33 and 34 degree Celsius	DWD
Wind	Average windspeed 10 m above ground in m/s	DWD
Relative humidity	Relative humidity at 2 m above ground in %	DWD
Pressure	Mean vapor pressure in hpa	DWD
	Socio-economic Demographic Controls	
Average age	Average age of the county population	BBSR
Population density	Residents per km^2	BBSR
Migration in	People moving out of county per 1,000 inhabitants	BBSR
Migration out	People moving into county per 1,000 inhabitants	BBSR
Moving AOK children	Share of AOK-insured children moving out of county	WIdO
Women share	Female to male population ratio	BBSR
Share of foreigners	Percentage of people without German citizen-ship	BBSR
Women share in foreigners	Share of female foreigners among foreigners	BBSR
Employment	Employees subject to social insurance contributions per 100 inhabitants of working age	BBSR
Gross Value Added (GVA)	Total gross value added in 1,000 Euro per employed person	BBSR
GVA share in primary sector	Share of gross value added in the primary sector in %	BBSR
GVA share in tertiary sector	Share of gross value added in the tertiary sector in %	BBSR
Household income	Average household income in Euro per inhabitant	BBSR
Housing transfers	Number of households receiving housing benefits, per 1,000 households	BBSR
Education	Share of students graduating with higher education entrance qualification	BBSR
Marriages	Marriages per 1,000 inhabitants 18 years and older	BBSR
Share of young mothers	Births of mothers in the age group 15 to under 20 years per 1,000 women in the age group	BBSR
Share of older mothers	Births of mothers 40 years and older per 1,000 women aged 40 to under 45	BBSR

D.2 Aggregating the Pollution Data

We aggregate the pollution data by averaging the daily PM_{10} readings of all measuring stations in a county and quarter. We weight each observation by the number of station readings in that period (c.p. Chay and Greenstone 2003a, Isen et al. 2017).

In the few counties in our sample without measuring stations, we interpolate pollution exposure from surrounding stations using Inverse Distance Weighting (IDW). Following Karlsson and Ziebarth (2018), we consider all stations within a 60 km (37.5 miles) radius of the county's centroid. We then calculate the weighted average using both the number of station measurements and the inverse distance of the monitors to the centroid as weights.

To avoid fluctuations in pollution levels linked to stations not being active regularly, we generate the quarterly averages including only stations with at least 60 measurements. Moreover, to avoid bias from interpolating pollution levels from treated to nearby untreated counties, we only use stations outside of LEZ counties for the interpolation. We show that our results are robust when we do not interpolate the pollution data and include only counties with own measuring stations in the sample in Table A.10.

D.3 AOK Data

While the AOK population is representative of a large proportion of the publicly insured across Germany, it is not fully representative. Different studies demonstrate that the proportion insured with AOK is slightly higher in the southeastern states than in the northwest and that the insured persons exhibit a lower socioeconomic status than the population of all publicly insured people on average (Jaunzeme et al. 2013, Hoffmann and Koller 2015). On the other hand, per capita expenditures on medical treatments and prescribed pharmaceuticals exhibit a comparable magnitude across insurance funds. Table D.1 presents some of the differences between the AOK-population and the total publicly insured population. While we cannot rule out that an extension of our findings beyond the studied population is biased, AOK data comprise a well-balanced sample of people from all subgroups (Jaunzeme et al. 2013) and constitute the best available base for identifying effects generalizable to a vast majority of the German population.

Table D.1: Differences Between the AOK-Population and the Total Publicly Isured Population

	AOK population	**Total publicly insured population**
	(1)	(2)
Proportion by population characteristics		
Low socioeconomic status	35.8%	21.9%
Medium socioeconomic status	57.5%	62.6%
High socioeconomic status	6.6%	15.5%
With migrant background	22.9%	14.3%
Active smokers	34.7%	30.9%
Obese people	19.9%	16.6%
Moderate/bad self-assessed health condition	37.1%	30.7%
Diagnosed with a cardiovascular disease	39.9%	38.1%
Medical expenditures per person		
Hospital treatments	1,122€	1,001€
Pharmaceuticals and medical aids	537€	518€
Doctoral examinations	551€	539€

The table reports summary statistics on the AOK population and the total publicly insured population. The statistics on the population characteristics are from the analysis of Hoffmann and Koller 2015 and are standardized with regard to the age and gender of the overall population. The statistics on the medical expenditures per insured person are provided by AOK (AOK 2018).

D.4 Prescription Data

The identification process of pharmaceutical substances that are relevant in the therapy of respiratory diseases and asthma specifically is as follows:

i) Pharmaceuticals for Respiratory Diseases

We use a publication akin to the Red Book called "Gelbe Liste" by the ISO 9001:2015 certified Medizinische Medien Informations GmbH, which serves as a source of information for medical and pharmaceutical professionals. For more than 120,000 drugs, it links ATC-code classified pharmaceutical substances to ICD-10-code classified clinical diagnoses. By linking ATC to ICD codes, we can determine for which diseases different pharmaceutical substances are commonly prescribed. From the registered information we draw 6,479 unique links, of which we select only those related to respiratory diseases (150 substances). While this approach is comprehensive, it suffers from the drawback that it may also cover substances generically administered for a broad variety of diseases.

ii) Pharmaceuticals for Asthma

Additionally, we define a smaller list of pharmaceuticals that are closely tied to asthma. To this end, we consult annually updated lists of the substances prescribed most often for asthma in a given year, that is substances in the ATC category R03. The lists are prepared by IGES institute for the years 2006 to 2017.[38] In our analysis, we consider only prescriptions of the 20 most often prescribed substances in the year the prescription is issued. Note, that the top 20 substances cover almost the entire market of substances prescribed for asthma and COPD, however, they may not include substances prescribed in rare cases. The pharmaceuticals identified according to this procedure represent a strict subset of those compiled in approach i).

The prescription costs are adjusted to allow for intertemporal comparisons as if the average cost per prescription had not changed. In other words, we take both inflation but also ATC-specific market price changes, such as expiring patents, into account. To this end, we calculate ATC-specific price indices normalized to the fourth quarter of 2017 using available prescription data for all children in Germany. Based on the generated price indices we adjust the prescription costs observed in our sample to real values, before aggregating them to the cohort level.

E Unconditional Quantile Regression

We estimate an unconditional quantile regression (Firpo et al. 2009) to flexibly estimate LEZ treatment effects across the unconditional distribution of our health outcomes. The approach is based on the use of the re-centered influence function (RIF) defined in Equation (E.1). It is the sum of the influence function (IF) and the θth quantile of the unconditional distribution of the health variable H denoted as q_θ. The IF indicates the marginal influence of an observation H_i on the quantile q_θ. It is determined by f_H, the empirical density function evaluated at q_θ, and by the indicator $1(h \leq q_\theta)$ which is equal to 1 if H_i is below or equal to q_θ. Thus, an observation's influence is negative if its health status lies below and positive if it lies above the health status at the θth quantile.

$$RIF(H_i, q_\theta) = q_\theta + IF(H_i, q_\theta) = q_\theta + \frac{\theta - \mathbb{1}(H_i \leq q_\theta)}{f_H(q_\theta)} \tag{E.1}$$

[38] More information on the underlying data and aggregation methodologies are provided on the IGES website and in the latest published report Häussler and Höer (2016).

The expected value of the RIF equals the quantile of the unconditional distribution.[39] By the law of iterated expectations and integration over the conditional mean, the unconditional quantile q_θ can be expressed as

$$q_\theta = E[RIF(H_i, q_\theta)] = E[E[RIF(H_i, q_\theta)|X_i]] = \int E[RIF(H_i, q_\theta)|X_i]dF_X \tag{E.2}$$

where X is the vector of covariates and F_X is the marginal distribution function of X. To obtain the marginal treatment effects on the unconditional quantile q_θ, we take the sample quantile $\hat{q_\theta}$ and retrieve the density $\hat{f_H}$ using a gaussian kernel method.[40] To obtain $\widehat{RIF}$, we substitute both into Equation (E.1). Secondly, we apply RIF-OLS regression to obtain the coefficients representing the marginal *ceteris paribus* effect of an infinitesimal shift in the distribution of the covariates X on the unconditional θth quantile of H:

$$\hat{\beta}_\theta = (\sum_{i=1}^{N} X_i' X_i)^{-1} \sum_{i=1}^{N} X_i' \widehat{RIF}(H_i, \hat{q_\theta}) \tag{E.3}$$

The identifying assumption is that in the absence of treatment, the change in the health outcome at each quantile would have been the same in the treatment and the control group. Because endogenous regressors cannot be addressed by the conventional unconditional quantile regression framework, and because estimation times are prohibitively long when using a stacked design, we limit our quantile regression analysis to reduced form estimations using a standard DID design knowing that some caveats may apply. For example, Section 5.5 shows that a standard-two-way DID estimator leads to results slightly smaller in magnitude compared to our stacked DID estimator. We regard our unconditional quantile estimates as suggestive evidence that children who suffer worst from respiratory diseases may benefit the most from LEZs. Table A.3 in the Appendix features all coefficients and standard errors which are bootstrapped using 1,000 repetitions and clustered at the county level.

F Comparison to the Literature

To provide context for the magnitude of our findings, we compare our estimates to related epidemiological and economic research that focuses on asthma.

[39] $E[RIF(H_i, q_\theta)] = E[q_\theta] + \frac{\theta - E[\mathbb{1}(H_i \leq q_\theta)]}{f_H(q_\theta)} = q_\theta + \frac{\theta - \theta}{f_H(q_\theta)} = q_\theta$

[40] $\hat{f_H}(\hat{q_\theta}) = \frac{1}{N \cdot b_H} \cdot \sum_{i=1}^{N} K_H(\frac{H_i - \hat{q_\theta}}{b_H})$, where K_H is the kernel function and b_H is a positive scalar bandwidth.

In a meta-study, Khreis et al. (2017) summarize the available epidemiological research on the impact of early life exposure to air pollution on the prevalence of asthma in children. Overall, the research suggests odds ratios of 1.025 for associations between PM_{10} and asthma at any age. Taking the odds ratio as an approximation of relative risk, we can compare the magnitude of our estimates for the share of sufferers to these results. Our estimate for asthma in column (7) in Table 4 implies a risk ratio of 1.045 at the mean, which is outside the meta-study's 95% confidence interval for studies that consider children from age three to young adults up to age 21. However, odds ratios from analyses limited to the ages three to six come very close to our estimates (Clark et al. (2010): 1.068, Deng et al. (2016): 1.048, Liu et al. (2016): 1.029).

Bharadwaj et al. (2016) estimate the lasting effect of exposure to the 1952 Great Smog of London on asthma development. In principle, our research designs are similar as the authors compare children born just before and just after air quality changes. However, our estimates are based on a slight improvement in air quality that by no means is comparable to the variation induced by the extreme impact of the "killer fog" which at least doubled childhood asthma rates.

Economic studies focus on contemporaneous improvements in child health. Using the case of the Stockholm congestion charge, Simeonova et al. (2019) show that persistently lower PM_{10} exposure reduces asthma-related hospital admissions of children below six years of age with an implied elasticity of 3.7. The elasticities we estimate for asthma drug prescriptions (1.8), expenditures (2.1), and the share of sufferers (1.2) are smaller.[41] This difference could be attributed to the fact that Simeonova et al. (2019) examine contemporaneous benefits of persistently improved air quality over a longer time period, while we study longer run health benefits from exposure to cleaner air in a single year.

Other economic studies consider short-run variations in air pollution exposure, but mainly focus on $PM_{2.5}$.[42] Alexander and Schwandt (2019) study the impact of emissions cheating by car manufacturers on $PM_{2.5}$ and child health outcomes. Their estimates imply that a one $\mu g/m^3$ increase in $PM_{2.5}$ increases asthma related hospital admissions of children aged four and younger by 0.42 per $1,000$. Evaluated at the reported means, the elasticity is 3.01.

[41] The calculations are based on the IV estimates for asthma in columns (2) and (4) of Table 3 and in column (7) of Table 4, respectively. These point estimates are then multiplied with the mean PM_{10} exposure and divided by the mean of the outcome to obtain elasticities.

[42] Beatty and Shimshack (2014) is a notable exemption. Based on data from young children in England, they relate respiratory treatments for children to monthly PM_{10} exposure. The estimated coefficient on PM_{10} is, however, statistically insignificant but would imply an elasticity of only 0.1.

Barwick et al. (2018) study changes in health-related consumption in China for $PM_{2.5}$ using data on bank card transactions. They estimate that a 10 $\mu g/m^3$ decrease in $PM_{2.5}$ reduces health spending in children's hospitals by 1.13%, implying an elasticity of 0.06.