ONLINE APPENDIX

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

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A. Additional Figures and Tables

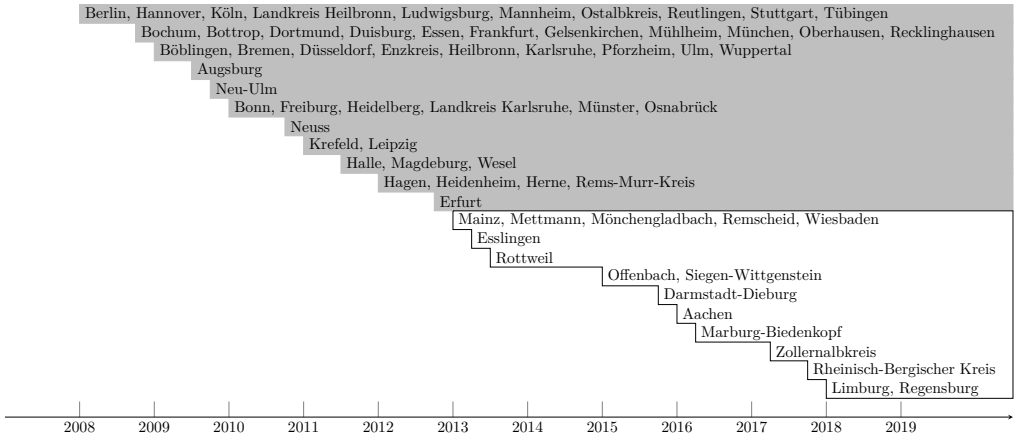


Figure A.1. : The Staggered Implementation of LEZs

Note: The figure shows the implementation dates and counties of all LEZs until 2019. The eleven implementation waves considered in this paper are marked in grey.

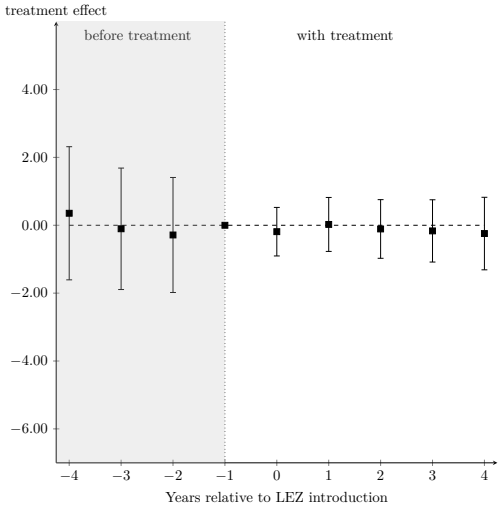


Figure A.2. : The Effect of LEZs on Later-Life PM_{10} Concentrations

Note: The figure presents an event-study that uses the children's average later-life pollution exposure as the dependent variable. Pollution exposure is the mean quarterly PM_{10} concentration in $\mu g/m^3$ in the children's second through fifth year of life. 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, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regression is weighted by county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

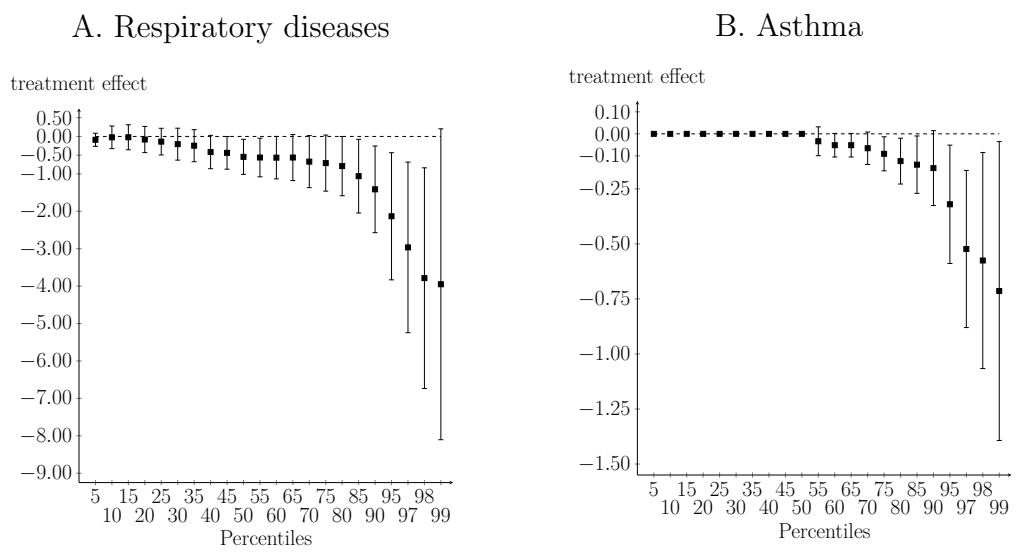


Figure A.3. : Unconditional Quantile Treatment Effects of LEZs

Note: The figure presents coefficients from unconditional quantile regressions (Firpo, Fortin and Lemieux, 2009; Borgen, Haupt and Wiborg, 2021) at the level of the individual child (Appendix I for further information). The dependent variable is either the number of prescriptions for respiratory diseases (Panel A) or for asthma (Panel B) that accumulate over the first five years of a child's life. The severity of suffering increases from left to right. All regressions include birth county and birth state–birth quarter fixed effects. We include weather and socio-economic controls as well as controls for whether there are partial treatments after the first year of pre-school childhood and LEZ stringency increases over the five years of pre-school childhood. We obtained the bars that indicate the 95% confidence interval using 500 bootstrap repetitions and 486,226 observations.

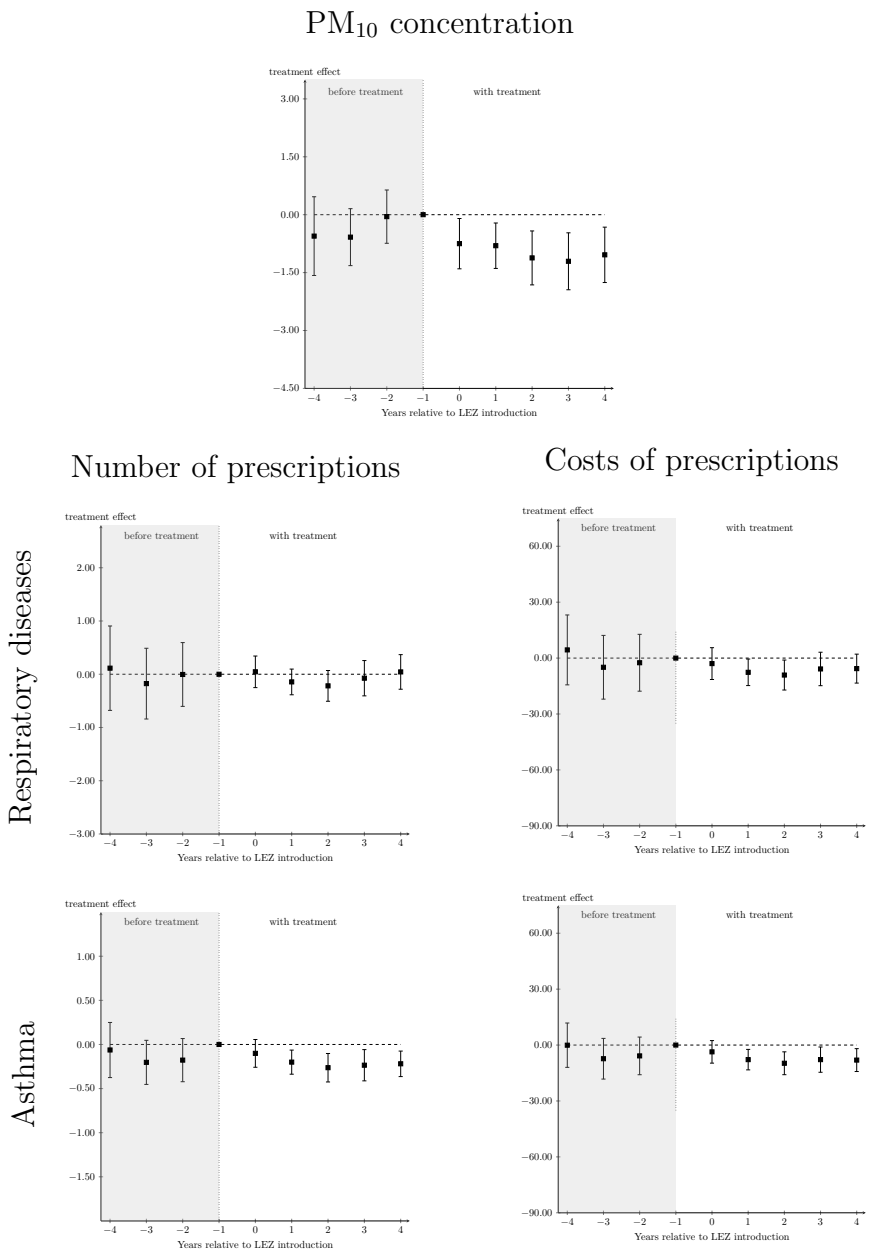
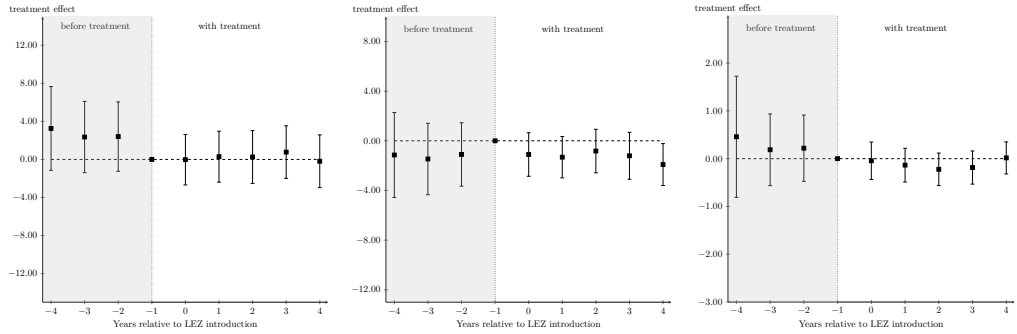


Figure A.4. : Event-studies for LEZ Effects Excluding the Never Treated

Note: The figure presents event-study coefficients for the effect of LEZs on PM_{10} pollution and the medication of respiratory diseases and asthma for a sample that excludes never treated counties from the control group. The dependent variable is either the average PM_{10} level in $\mu g/m^3$ or the number or the costs 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 three years around treatment to avoid that the control groups for the later treated become very small. The grey shaded areas indicates the pre-treatment periods. The coefficients in the year prior to implementation are normalized to zero. The regressions include county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regressions are weighted by county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

Hospital treatments per 100 children

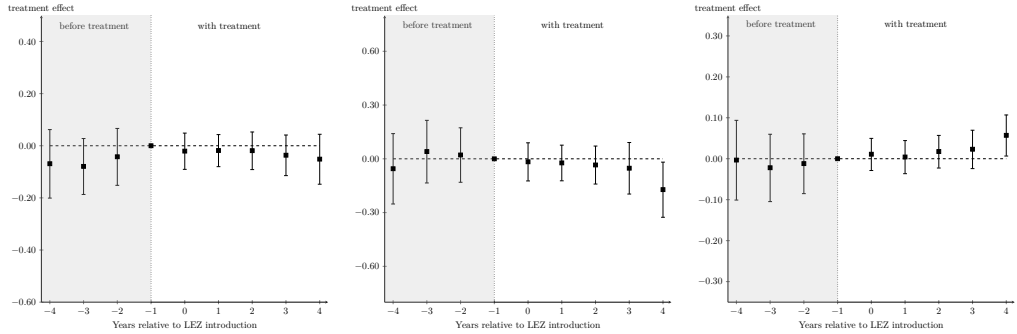


(a) Infectious diseases
mean outcome: 15.132

(b) Head injuries
mean outcome: 6.812

(c) Neurodermatitis
mean outcome: 0.370

Pharmaceutical prescriptions per child



(d) Nausea treatment
mean outcome: 1.15

(e) Wound and pain care
mean outcome: 1.825

(f) Caries prophylaxis
mean outcome: 0.305

Figure A.5. : Event-studies for LEZ Effects on Placebo Outcomes

Note: The figure presents event-study coefficients based on the specification in Equation (F.1) that show how LEZs affect placebo outcomes depending on the time between birth and LEZ implementation in years. The dependent variable is the number of hospital treatments per 100 children (Panel a through c) and the number of prescriptions per child (Panel d through f) that accumulate over the first five years of the children's lives. The gray shaded areas indicate the pre-treatment period. The coefficients in the year prior to implementation are normalized to zero. The regressions include county fixed effects, state–quarter fixed effects, LEZ wave–event time fixed effects, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regressions are weighted by county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

Table A.1—: Excluding Varying Shares of Small LEZs from the Sample

	(1)	*(2)*	*(3)*	*(4)*
	10% smallest LEZs excluded	15% smallest LEZs excluded	20% smallest LEZs excluded	25% smallest LEZs excluded
	A. PM_{10} concentration			
Mean PM_{10}	-1.315	-1.373	-1.470	-1.358
s.e.	(0.346)	(0.349)	(0.351)	(0.357)
mean	28.239	28.431	28.431	28.485
	B. Number of prescriptions			
Respiratory diseases	-0.487	-0.578	-0.507	-0.648
s.e.	(0.218)	(0.221)	(0.221)	(0.233)
mean	14.532	14.54	14.540	14.535
Asthma	-0.291	-0.325	-0.296	-0.350
s.e.	(0.097)	(0.100)	(0.100)	(0.108)
mean	2.558	2.543	2.543	2.504
	C. Costs of prescriptions			
Respiratory diseases	-15.985	-18.560	-16.507	-18.903
s.e.	(6.051)	(6.203)	(5.991)	(6.502)
mean	228.916	228.185	228.185	227.345
Asthma	-14.618	-16.071	-14.521	-16.165
s.e.	(4.752)	(4.872)	(4.781)	(5.037)
mean	77.573	77.559	77.559	76.110
Sample size	12,950	12,893	12,761	12,695

Note: This table reports coefficients when excluding varying shares of small LEZs from our sample. In Column 1, we exclude the 10% of the LEZ-counties that exhibit the smallest population coverage and we sequentially increase the share to 25% in Column 4. The dependent variable in Panel A is the PM_{10} concentration; in Panel B 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; and in Panel C it is the respective costs. The dependent variables in Panels B and C are composition-adjusted for birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regressions are weighted by birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level.

Table A.2—: Summary Statistics for Pollution and Health Outcomes

	(1) mean	(2) sd	(3) min	(4) max	(5) N
Air pollution					
PM_{10} ($\mu g/m^3$)	24.4	6.1	9.0	56.7	2,996
Prescriptions for respiratory diseases					
Number of prescriptions over five years per child	12.9	2.8	5.0	23.8	2,996
Prescription expenditures over five years per child (€)	197.7	50.9	64.9	635.3	2,996
Share of sufferers per cohort (%)	76.5	7.4	38.3	94.6	2,996
Prescriptions for asthma					
Number of prescriptions over five years per child	2.0	0.7	0.3	5.4	2,996
Prescription expenditures over five years per child (€)	62.3	30.4	4.4	489.8	2,996
Share of sufferers per cohort (%)	18.6	5.6	4.0	37.4	2,996
Number of children per cohort	178	190	17	1,593	2,996

Note: The table reports summary statistics for PM_{10} pollution in $\mu g/m^3$ and for cumulative prescriptions over the five years of pre-school childhood linked to a broad group of respiratory diseases and asthma specifically. The variables are defined for our study period between 2006 to 2012 and our sample of 128 German counties that violated EU-wide limits for PM_{10}. Cumulative prescriptions over 5 years 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.

Table A.3—: Event-study Estimates of LEZ Effects on PM_{10} Concentrations and Medication Before and After Policy Implementation

	PM_{10}	Respiratory disease		Asthma	
	(1) ($\mu g/m^3$)	*(2)* cases	*(3)* costs	*(4)* cases	*(5)* costs
			Pre-treatment period		
LEZ treatment ($\theta = -3$)	-0.368	-0.376	1.986	0.123	2.689
	(0.571)	(0.541)	(14.568)	(0.230)	(10.174)
LEZ treatment ($\theta = -2$)	-0.162	-0.414	-5.132	0.062	-4.383
	(0.410)	(0.466)	(14.156)	(0.219)	(10.212)
LEZ treatment ($\theta = -1$)	-0.416	-0.142	1.455	0.096	-1.052
	(0.352)	(0.422)	(12.207)	(0.192)	(8.512)
			Post-treatment period		
LEZ treatment ($\theta = 1$)	-1.576	-0.602	-21.538	-0.324	-18.275
	(0.424)	(0.249)	(7.242)	(0.116)	(5.604)
LEZ treatment ($\theta = 2$)	-1.565	-0.644	-20.217	-0.343	-17.039
	(0.400)	(0.227)	(6.410)	(0.105)	(4.858)
LEZ treatment ($\theta = 3$)	-1.633	-0.802	-26.030	-0.441	-21.453
	(0.481)	(0.234)	(7.167)	(0.108)	(5.357)
LEZ treatment ($\theta = 4$)	-1.699	-0.696	-20.041	-0.404	-18.619
	(0.546)	(0.309)	(8.876)	(0.124)	(6.412)
LEZ treatment ($\theta = 5$)	-1.665	-1.062	-22.781	-0.448	-19.837
	(0.643)	(0.341)	(8.105)	(0.116)	(5.637)

Note: This table reports event-study coefficients underlying Figures 2 and 3. 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 birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. 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 $12,972$.

Table A.4—: Multiple Hypotheses Testing

Table	Col.	Year	Respiratory diseases		Asthma	
			p	$p(BH)$	p	$p(BH)$
			A. Number of prescriptions			
2	1	year 1-5	0.013	0.036	0.001	0.009
2	2	year 1	0.244	0.347	0.731	0.768
2	3	year 2	0.015	0.036	0.010	0.034
2	4	year 3	0.002	0.011	0.000	0.001
2	5	year 4	0.274	0.355	0.008	0.030
2	6	year 5	0.130	0.224	0.020	0.044
			B. Costs of prescriptions			
2	1	year 1-5	0.003	0.014	0.001	0.009
2	2	year 1	0.246	0.347	0.502	0.574
2	3	year 2	0.001	0.009	0.001	0.009
2	4	year 3	0.004	0.019	0.001	0.009
2	5	year 4	0.015	0.036	0.012	0.036
2	6	year 5	0.420	0.504	0.087	0.160
			C. Share of sufferers			
3	1	year 1-5	0.828	0.828	0.017	0.038
3	2	year 1	0.163	0.261	0.736	0.768
3	3	year 2	0.827	0.828	0.243	0.347
3	4	year 3	0.262	0.349	0.000	0.001
3	5	year 4	0.318	0.402	0.070	0.134
3	6	year 5	0.619	0.691	0.002	0.011
			D. Prescriptions per sufferer			
3	1	year 1-5	0.014	0.036	0.335	0.412
3	2	year 1	0.725	0.768	0.478	0.559
3	3	year 2	0.012	0.036	0.066	0.131
3	4	year 3	0.006	0.025	0.064	0.131
3	5	year 4	0.106	0.188	0.160	0.261
3	6	year 5	0.180	0.279	0.257	0.349

This table reports p values for all 48 hypotheses regarding respiratory diseases and asthma tested in Tables 2 and 3. Columns labeled p indicate unadjusted p-values while columns labeled $p(BH)$ indicate p-values adjusted for multiple hypotheses testing by disease and regression method following Benjamini and Hochberg (1995).

Table A.5—: Unconditional Quantile Regression Estimates of the Effect of Early-Life LEZ Exposure on Medication throughout Pre-School Childhood

	A. Respiratory diseases							
	Q-5	Q-10	Q-15	Q-20	Q-25	Q-30	Q-35	Q-40
LEZ	-0.089	-0.023	-0.022	-0.082	-0.138	-0.206	-0.247	-0.417
treatment	(0.089)	(0.154)	(0.171)	(0.179)	(0.182)	(0.217)	(0.219)	(0.227)
	Q-45	Q-50	Q-55	Q-60	Q-65	Q-70	Q-75	Q-80
LEZ	-0.438	-0.545	-0.563	-0.567	-0.563	-0.673	-0.712	-0.792
treatment	(0.223)	(0.239)	(0.263)	(0.289)	(0.314)	(0.355)	(0.382)	(0.404)
	Q-85	Q-90	Q-95	Q-97	Q-98	Q-99		
LEZ	-1.061	-1.413	-2.133	-2.965	-3.787	-3.950		
treatment	(0.502)	(0.591)	(0.867)	(1.163)	(1.504)	(2.119)		
	B. Asthma							
	Q-5	Q-10	Q-15	Q-20	Q-25	Q-30	Q-35	Q-40
LEZ	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000
treatment	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
	Q-45	Q-50	Q-55	Q-60	Q-65	Q-70	Q-75	Q-80
LEZ	0.000	0.000	-0.034	-0.052	-0.052	-0.065	-0.091	-0.124
treatment	(0.000)	(0.000)	(0.033)	(0.027)	(0.027)	(0.038)	(0.039)	(0.053)
	Q-85	Q-90	Q-95	Q-97	Q-98	Q-99		
LEZ	-0.140	-0.156	-0.320	-0.523	-0.575	-0.714		
treatment	(0.067)	(0.087)	(0.137)	(0.182)	(0.250)	(0.346)		

Note: This table presents coefficients from unconditional quantile regressions (Firpo, Fortin and Lemieux, 2009; Borgen, Haupt and Wiborg, 2021) at the level of the individual child (Appendix I for further information). The dependent variable is either the number of prescriptions for respiratory diseases (Panel A) or for asthma (Panel B) that accumulate over the first five years of a child's life. The severity of suffering increases with the percentiles. All regressions include birth-county and birth state–birth quarter fixed effects. We include weather and socio-economic controls as well as controls for whether partial treatments and LEZ stringency increase at least once over the course of the five years of pre-school childhood. The regressions are weighted by birth county–birth quarter cell size. We obtained the bars that indicate the 95% confidence interval using 500 bootstrap repetitions and 486,226 observations.

Table A.6—: The Effect of Early-Life Air Quality Improvements from LEZs on the Medication of Heart Diseases throughout Pre-School Childhood

	Heart diseases	
	(1) Number of prescriptions	*(2)* Costs of prescriptions
LEZ treatment	-0.106	-2.481
s.e.	(0.043)	(0.837)
mean	1.210	21.768

Note: This table reports estimates for the effect of LEZ implementation during the *in utero* period and the first life on the medication for heart diseases. The dependent variable is either the number of prescriptions for heart diseases per child (Column 1) or their costs in Euro per child (Column 2) that accumulate over the first five years of a child's life on average. We consider pharmaceuticals prescribed for heart diseases such as hypertension, ischaemic heart disease, pulmonary heart disease, or heart insufficiency. The dependent variables are composition-adjusted for birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatment and LEZ stringency. 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 12,972.

Table A.7—: The Effect of Early-Life Air Quality Improvements from LEZs on Antibiotic Prescriptions throughout Pre-School Childhood

	Antibiotics	
	(1) Number of prescriptions	*(2)* Costs of prescriptions
LEZ treatment	-0.164	-6.871
s.e.	(0.118)	(5.178)
mean	5.709	118.027

Note: This table reports estimates for the health effect of LEZ implementation during the in utero period and the first life. The dependent variable is either the number of prescriptions for antibiotics per child (Column 1) or their costs in Euro per child (Column 2) that accumulate over the first five years of a child's life on average. We consider the group of antibiotics for systemic use (ATC J01) which are frequently prescribed to children. The dependent variables are composition-adjusted for birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. 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 12,972.

Table A.8—: The Effects of LEZs for Different Treatment Time Windows

	(1)	*(2)*	*(3)*
	$[-2, 2]$	$[-3, 3]$	$[-4, 4]$
	A. PM_{10} concentration		
Mean PM_{10}	-0.768	-1.164	-1.316
s.e.	(0.283)	(0.317)	(0.349)
mean	28.384	28.421	28.239
	B.Number of prescriptions		
Respiratory diseases	-0.623	-0.601	-0.770
s.e.	(0.161)	(0.190)	(0.223)
mean	14.358	14.511	14.532
Asthma	-0.180	-0.323	-0.312
s.e.	(0.074)	(0.101)	(0.085)
mean	2.520	2.534	2.558
	C. Costs of prescriptions		
Respiratory diseases	-12.315	-16.427	-20.245
s.e.	(3.968)	(4.996)	(5.012)
mean	223.64	226.787	228.916
Asthma	-6.964	-12.733	-14.951
s.e.	(2.877)	(3.781)	(3.577)
mean	75.093	75.989	77.573
Sample size	8,164	11,564	13,216

Note: This table reports coefficients for shorter treatment time windows. The time window increases sequentially from two years (Column 1) to four years (Column 3) before and after LEZ implementation. The dependent variable in Panel A is the PM_{10} concentration; in Panel B 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; and in Panel C it is the respective costs. The dependent variables in Panels B and C are composition-adjusted for birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. Because the inclusion of implementation wave specific controls for LEZ stringency in each of the five years of pre-school childhood leaves little identifying variation when decreasing the event time window, we slightly relax our specification for this test. For each implementation wave, we replace the stringency controls for the individual years with one binary control that is equal to one if a child experiences an increase in LEZ stringency in any of the five years of pre-school childhood. We acknowledge that this specification absorbs dynamic effects to a somewhat lesser degree than in our main analysis. The regressions are weighted by birth county–birth quarter cell size. Standard errors in parentheses are clustered at the county level.

Table A.9—: Air Pollution and Health Effects on Neighboring Counties

	PM_{10}	Number of prescriptions		Costs of prescriptions	
	(1) $\mu g/m^3$	(2) Respiratory diseases	(3) Asthma	(4) Respiratory diseases	(5) Asthma
LEZ effect on LEZ-counties	-1.260	-0.499	-0.332	-17.766	-16.248
	(0.337)	(0.203)	(0.101)	(5.988)	(4.874)
LEZ effect on neighboring counties	-0.226	-0.074	-0.020	-1.080	-1.187
	(0.105)	(0.045)	(0.018)	(1.058)	(0.823)

Note: This table replicates regressions from Tables 1 and 2. In addition, we present the treatment effect on counties adjacent to those that implement an LEZ. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. 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 $12,972$.

Table A.10—: Balancing Regressions for Socio-economic Control Variables

	(1) Coefficient	(2) Standard error	(3) p	(4) p(BH)
		Migration patterns		
Total net migration	-0.593	0.900	0.510	0.656
Net migration among families	-1.200	0.781	0.124	0.373
Moving AOK children	-0.962	0.735	0.191	0.453
		Socio-economic control variables		
Population density	-5.530	8.145	0.497	0.656
Average age	-0.300	0.068	0.000	0.000
Share of women	-0.006	0.008	0.438	0.656
Share of foreigners	0.090	0.195	0.642	0.680
Women share in foreigners	-0.164	0.235	0.486	0.656
Household income	-11.320	11.462	0.323	0.647
Housing transfers	-0.637	0.498	0.201	0.453
Employment	-0.397	0.240	0.098	0.354
Education	-0.490	0.595	0.411	0.656
Gross Value Added (GVA)	-0.432	0.838	0.606	0.680
GVA share in primary sector	-0.030	0.017	0.085	0.354
GVA share in tertiary sector	1.434	0.652	0.028	0.167
Marriages	-0.241	0.105	0.021	0.167
Share of young mothers	0.113	0.318	0.722	0.722
Share of older mothers	-0.138	0.231	0.552	0.662

Note: This table presents balancing regressions for 20 socio-economic variables. The coefficients in Column (1) show the effect of LEZ implementation on the different annually measured socio-economic outcome variables. Columns (2) and (3) report the respective standard errors and p-values. In Column (4) we additionally report p-values adjusted following Benjamini and Hochberg (1995). All regressions include birth county and birth state–birth quarter fixed effects and we control for LEZ stringency. In line with our main regressions we include weather controls and pre-treatment controls for socio-economic characteristics interacted with year-quarter fixed effects. 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 $3,199$.

B. LEZ implementation patterns

We address concerns that LEZs are not introduced randomly but in areas with deteriorating air quality by limiting our sample to non-attainment counties (following Wolff, 2014). Here we investigate this issue directly in the data. In particular, we implement the following analysis. For each county, we calculate annual average concentrations of PM_{10} for the years 2006 and 2007, i.e., just before the first LEZ is introduced. We then regress a) an indicator that a county belongs to the group of counties that implement an LEZ or b) the year-quarter of the actual LEZ introduction on these two annual averages as well as on the change in PM_{10}.

As Columns (1) to (3) from Table B.1 demonstrate, higher levels of PM_{10} in 2006 or 2007 are associated with a higher likelihood of LEZ introduction, but there is no evidence that counties with rising levels of air pollution are more likely to introduce an LEZ. Columns (4) to (6) suggestively show that counties with higher levels of PM_{10} were not faster to implement LEZs and there also seems to be no relation between the timing of the introduction and prior pollution dynamics. This is also in line with the flat pre-trends in the event study presented in Figure 2.

Table B.1—: LEZ Implementation Patterns

	LEZ treatment group			Year-quarter of introduction		
	(1)	(2)	(3)	(4)	(5)	(6)
PM_{10} in 2007	0.072			0.023		
	(0.008)			(0.249)		
PM_{10} in 2006		0.048			-0.221	
		(0.013)			(0.283)	
Change in PM_{10} 2006 to 2007			0.007			0.043
			(0.016)			(0.232)
Observations	96	92	90	43	40	40

Note: This table shows estimations at the county level. The dependent variable is an indicator that either takes on a value of 1 if a county is treated, or the date of LEZ introduction measured in year-quarters. PM_{10} in $\mu g/m^3$ is measured for a given county and year. Estimates for the constant are omitted. Robust standard errors are in parentheses.

C. Composition-Adjusted Health Outcomes

For the estimation of Equation (3), health outcomes observed at the level of the individual child i are aggregated to the cohort level. 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 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 children's health outcomes on individual-level covariates as well as birth county–birth quarter fixed effects:

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

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 include gender and location of residence within a county at the five-digit zip code. Additionally, Equation (C.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 return the average health outcomes for a birth county–birth quarter cohort after controlling for sex and residence. In line with Isen, Rossin-Slater and Walker (2017), we refer to the predicted cohort means obtained by this approach as composition-adjusted. We use these composition-adjusted outcomes as dependent variables in Equation (3).

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. Albouy, 2009; Angrist and Lavy, 2009; Currie et al., 2015; Isen, Rossin-Slater and Walker, 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, Rossin-Slater and Walker, 2017). Additionally, it requires substantially less computational power.

D. Data

1. Control Variables

Table D.1 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 transformed to terciles and interacted with binary year-quarter variables.

Table D.1—: Control variables

Variable	Definition	Source
	Weather Controls	
- Precipitation	Total precipitation in mm/m^3	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	Avg. wind speed 10m above ground in m/s	DWD
- Relative humidity	Relative humidity 2m above ground in %	DWD
- Pressure	Mean vapor pressure in hPa	DWD
	Socio-economic & Demographic Controls	
- Average age	Average age of 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

Variable	Definition	Source
- 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

2. Aggregating Pollution Measurements

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

For the few counties in our sample that lack a measuring station, we interpolate pollution exposure using Inverse Distance Weighted (IDW) measurements from other counties' stations. Following Karlsson and Ziebarth (2018), we consider all stations within a 60 km (37.5 miles) radius of a given 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 frequently inactive stations, we only consider 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 in-

terpolation. Our results are robust to limiting our sample to counties with own measuring stations. With a coefficient of -1.763 and a standard error of 0.562 the effect of LEZ implementation on PM_{10} exposure during the first year of life increases only slightly in magnitude compared to the estimate in Table 1 (Column 1).

It is uncommon that a monitor measures all five key pollutants (PM_{10}, SO_2, CO, O_3, NO_2). While the pollutants a monitor measures are usually fixed, the number of monitors varies over time. 21% of the monitors that are located in one of the counties we study are discontinued during the sample period, while newly added account for 17% of all monitors. To examine the potential issue of strategic placement, we implement the following analysis. We regress quarterly PM_{10} at a monitor located in a sample county on an indicator for being added or being discontinued as well as on a county-by-year-by-quarter fixed effect. Monitors that were discontinued recorded on average 0.48 $\mu g/m^3$ less PM_{10}. However, this point estimate is not significantly different from 0 ($t = 0.60$). In contrast, newly introduced monitors recorded on average 3.72 $\mu g/m^3$ ($t = 4.35$) more PM_{10} than other monitors in the same county. This speaks against the strategic placement of new monitors as we would expect PM_{10} readings below a county's average.

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 share of individuals insured with AOK is slightly higher in the southeastern than in the northwestern states and that the insured exhibit a lower socioeconomic status than the population of all publicly insured on average (Jaunzeme, Eberhard and Geyer, 2013; Hoffmann and Koller, 2015). On the other hand, per capita expenditures on medical treatments and prescribed pharmaceuticals exhibit a comparable magnitude across public health insurances.[28]

Table D.2 compares characteristics of the AOK-population to those of all publicly insured. AOK is historically the largest public health insurer and the default provider. Consequently, AOK covers the largest fraction of the publicly insureds. While we cannot rule out that an extension of our findings beyond the studied population may be biased, those insured with

[28]Obtaining a health insurance plan is mandatory in Germany and the mandating of public health insurance base coverage, premiums, and contractual details by law ensures that these aspects are equivalent across providers.

Table D.2—: Comparing the AOK-Population to All Publicly Insured

	(1) AOK population	(2) Total publicly insured population
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 individuals	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€

Note: This table reports summary statistics for the AOK population and all publicly insured. 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. Statistics on medical expenditures per insured person are provided by AOK (2018).

AOK comprise a well-balanced sample of individuals from all subgroups (Jaunzeme, Eberhard and Geyer, 2013) and constitute the best available base for identifying effects generalizable to a vast majority of the German population.

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 ISO 9001:2015 certified Vidal MMI Germany GmbH, which serves as a source of information for medical and pharmaceutical professionals (Vidal MMI, n.d.). 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 identify 150 pharmaceutical substances that are prescribed for respiratory diseases. We follow the same procedure to identify

pharmaceutical substances that are relevant in the therapy of heart diseases and diseases considered in our placebo analysis. 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 (IGES, 2019).[29] 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).

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.

[29]IGES's website and their latest published report Häussler and Höer (2016) provide additional information regarding the underlying data and aggregation methodologies.

E. Additional Robustness Checks

1. Exposure at Older Ages

Our analysis makes strong assumptions about the importance of pollution exposure prior to age one based on the broad evidence that this is a critical development phase for children (Holt, 1998; Šrám et al., 2005; Gluckman et al., 2008; Baccarelli and Bollati, 2009; Almond, Currie and Duque, 2018). To ensure, that this early-life focus is justified in the context of our analysis, we run additional regressions isolating the pollution exposure effects not in the *in utero* period and during the first year of life as in our main analysis but in the years two through five of the children's life. Table E.1 in the Appendix confirms that exposure before age one is associated with a clear health response while health effects from LEZ exposure in the later years of life remain consistently statistically insignificant.

2. Accounting for Treatment Differences After the First Year of Life

We aim to compare children who experience different levels of pollution exposure *in utero* in their first year of life but the same exposure levels afterward. To achieve this, we control for partial treatments after the first year of life in our main analysis by including binary variables f_t^p (see Section II.B). An alternative approach that we implement here is to restrict the pre-treatment observations in our sample to cohorts born exactly four quarters prior to LEZ implementation. These cohorts benefit from cleaner air after their first year of life. Thus, they differ only in pollution exposure in the preceding period compared to the children born after policy implementation. The results in Table E.2 show that our findings are robust with respect to this sample restriction. Although smaller in magnitude, the estimated coefficients confirm our main analysis's findings.

Table E.1—: The Effect of LEZ Exposure Conditional on Children's Age

	Number of prescriptions		Costs of prescriptions	
	(1) Respiratory diseases	*(2)* Asthma	*(3)* Respiratory diseases	*(4)* Asthma
	A. Early-life exposure			
LEZ treatment *in utero* & year 1	-0.508 (0.205)	-0.335 (0.101)	-17.895 (6.014)	-16.390 (4.901)
	B. Later-life exposure			
LEZ treatment in year 2	0.030 (0.208)	0.022 (0.087)	-3.339 (5.417)	-3.005 (3.855)
LEZ treatment in year 3	0.431 (0.417)	-0.032 (0.179)	5.397 (12.662)	-1.698 (9.458)
LEZ treatment in year 4	-0.092 (0.301)	-0.161 (0.152)	-12.399 (10.119)	-12.689 (8.705)

Note: This table reports the effect of LEZs conditional on children's age at the time of exposure. Panel A presents the isolated effects of exposure *in utero* and during the first year of life that we estimate in our main analysis (Table 2). Panel B presents the isolated exposure effects in the second through fourth year of life. The dependent variable is either the number of prescriptions per child or their costs in Euro per child. It is accumulated over the first five years of a child's life on average and refers to either prescriptions for respiratory diseases in general or asthma specifically. The dependent variable is composition-adjusted for birth county–birth quarter cell size. All regressions include birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include controls for weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. Controls for partial treatments are included for all years other than the one of interest, e.g. we include bivariate controls for the years one, three, four, and five when estimating the exposure effect for the second year. Just as in our main analysis, we exclude partially treated cohorts in the year of exposure and interest, e.g. we exclude the three cohorts born in the quarters five through seven before LEZ implementation when estimating the exposure effect during the second year. 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 12,972 in Panel A and B, 13,188 in Panel C, and 13,632 in Panel D.

Table E.2—: The Effect of LEZs when Limiting Pre-treatment Observations to Cohorts Born Four Quarters Before Implementation

	A. Number of prescriptions		B. Costs of prescriptions	
	(1) Respiratory diseases	*(2)* Asthma	*(3)* Respiratory diseases	*(4)* Asthma
LEZ treatment	-0.940	-0.309	-18.643	-14.012
s.e.	(0.268)	(0.100)	(7.376)	(5.209)
mean	14.059	2.468	214.483	72.202

Note: This table reports coefficients that indicate the health effect of LEZ implementation during the *in utero* period and the first life year when we use only cohorts born four quarters prior to LEZ implementation as pre-treatment observations of the treated. The dependent variable is either 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. Moreover, it refers to either prescriptions for respiratory diseases in general or asthma specifically. The dependent variable is composition-adjusted for the size of 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. We include weather and socio-economic controls as well as controls for LEZ stringency. Because the inclusion of the implementation wave-specific controls for LEZ stringency in each of the years one through five leaves little identifying variation when including only one pre-treatment observation per county, we slightly relax our specification for this test. For each implementation wave, we replace the stringency controls for the individual years with one binary control that is equal to one if a child experiences a more stringent LEZ regime until age five. We acknowledge that this specification absorbs dynamic effects to a somewhat lesser degree than in our main analysis. The regressions are weighted by the size of the birth county–birth quarter cell. Standard errors in parentheses are clustered at the county level. The sample size is $6,224$.

3. 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 E.3 show effects of LEZ-induced improvements in air quality on child health that are robust only at the 10% level of statistical significance. However, they tend to be lower in magnitude. For instance, the coefficient for respiratory diseases is 0.50 in the stacked DID estimation in column (1) of Table 2 while it is only 0.39 in the two-way fixed effect DID estimation in Table E.3. We expect that part of this attenuation stems from the weighted aggregation of heterogeneous treatment effects revealed in Goodman-Bacon (2018). Also note that the two-way fixed effect setup does not allow 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. Likewise, the binary controls for partial treatments and LEZ stringency cannot be included for each implementation wave separately.

Table E.3—: Two-Way Fixed Effect Estimation of LEZ Effects

	A. Number of prescriptions		B. Costs of prescriptions	
	(1) Respiratory diseases	*(2)* Asthma	*(3)* Respiratory diseases	*(4)* Asthma
LEZ treatment	-0.390	-0.169	-9.175	-7.639
s.e.	(0.223)	(0.092)	(5.337)	(3.845)
mean	14.532	2.558	228.916	77.573

Note: This table replicates our main results in Table 2 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. The dependent variable is composition-adjusted for the size of the birth county–birth quarter cell. All regressions include birth county and birth state–birth quarter fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. 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 $2,670$.

4. Effects on Other Air Pollutants

We use PM_{10} as a measure for air pollution because European policymakers are highly focused on it, and LEZs explicitly target PM_{10}. Moreover, the EU only set legally binding limits for $PM_{2.5}$ in 2015. By definition, PM_{10} includes particles below 10 μm such as the finer $PM_{2.5}$ particles. To evaluate whether LEZs decrease $PM_{2.5}$ specifically, in a first robustness check, we resort to satellite-based $PM_{2.5}$ estimates from van Donkelaar et al. (2019).[30] This data is available on a fine resolution grid of 0.01 degrees but only at an annual level.[31] Thus, we lose quarterly observations and the corresponding fixed effects. Table E.4 shows that we lack the statistical power to identify statistically significant reductions in mean $PM_{2.5}$ concentrations from LEZs based on these data.

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) are caused primarily by diesel vehicles.[32] Therefore, we assess whether LEZs also impact ambient NO_2 concentrations using our data from the German air monitoring network. Table E.4 shows that LEZs significantly reduce NO_2 by about 2.0 $\mu g/m^3$ (4.8%) on average. This

Table E.4—: The Effect of LEZs on Additional Air Pollutants

	(1)	(2)	(3)	(4)
	$PM_{2.5}$	NO_2	O_3	SO_2
LEZ treatment	-0.121	-2.362	0.069	0.186
s.e.	(0.097)	(1.031)	(0.450)	(0.205)
mean	15.591	42.631	41.238	4.900

Note: This table reports coefficients for the effect of LEZs on four additional 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. We include weather and socio-economic controls as well as controls for LEZ stringency. 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 3,981 in Column (1) and 12,972 in Columns (2) through (4).

[30]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}.

[31]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.

[32]NO_2 serves as an indicator for different nitrogen oxides. In Germany, about 72.5% of NO_2 emissions from on-road traffic are from diesel vehicles (UBA, 2017*b*).

finding is consistent with the fact that LEZs are *de facto* bans of old diesel vehicles. However, it means that we cannot conclusively infer that PM_{10} exclusively determines the health effects we observe. Similar to other papers, we attribute our results to effects of air pollution in general (c.p. Chay and Greenstone, 2003*a*; Currie and Neidell, 2005; Arceo, Hanna and Oliva, 2016; Knittel, Miller and Sanders, 2016; Deryugina et al., 2019; Colmer et al., 2020).

We subsequently show that the policy does not affect pollutants other than PM or NO_2. 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. Second, environmental regulation can adversely impact firms' output and productivity. Therefore, we might be concerned that LEZs decrease industrial activity and, thereby, reduce emissions of industrial pollutants, most notably SO_2. Table E.4 does not reveal any statistically significant effects on O_3 or SO_2 concentrations.[33]

5. 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 E.5. We find that the estimated relative effects are smaller in magnitude but comparable to the ones derived from Table 2. However, only the coefficients for the number and costs of prescriptions for asthma (Column 2 and 4) remain statistically significant at conventional levels.

[33]Because transport only accounts for about 2% of total SO_2 emissions, this robustness check also serves as a placebo test, suggesting that our results in Table 1 are not determined by confounding factors.

Table E.5—: The Effect of LEZs on Log-Transformed Medication Outcomes

	A. Number of prescriptions		B. Costs of prescriptions	
	(1) Respiratory diseases	*(2)* Asthma	*(3)* Respiratory diseases	*(4)* Asthma
LEZ treatment	-0.021	-0.107	-0.058	-0.157
s.e.	(0.019)	(0.046)	(0.031)	(0.076)
mean	14.532	2.558	228.916	77.573

Note: This table replicates our main results in Table 2 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. The dependent variable is composition-adjusted for birth county–birth quarter cell size and 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 birth county, birth state–birth quarter, LEZ wave–event time, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. 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 $12,972$.

F. Stacked DID Design

1. Defining the Control Group

For our stacked analysis we define an event-time window, i.e. the period in which we observe treatment effects before and after LEZ implementation, and an exclusion window, i.e. the period in which counties must not implement an LEZ themselves to be eligible for the control group.

Our event-time window covers the period up to four years prior and five years subsequent to LEZ implementation. This choice is based on the dates of the implementations of the LEZs, the availability of data, and the observation of cumulative benefits over five years. Our exclusion window is set to the same period. This implies that we only allow our control group to hold LEZ-counties that do not implement the policy measure at least four years before or five years after the treatment wave for which they serve as control units. 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 A.1).

The selection of this exclusion window results from two considerations. First, we expect and show that an LEZ's treatment effect on PM concentration levels off immediately after implementation. In other words, the policy induces a level shift in pollution, but treated counties are not on a differential trend after treatment. Otherwise, they would be unsuitable to serve as control units. Second, we want to have a balanced control group throughout event time, where control counties are not subject to treatment themselves during the period in which they are compared to the treated.

To rule out that our choice of the exclusion window determines our results, we provide estimates for alternative specifications of the exclusion-window as robustness checks. We also show that our results are almost identical if we exclude all already-treated from the control group to allow for persistent dynamics (see Figure F.1).

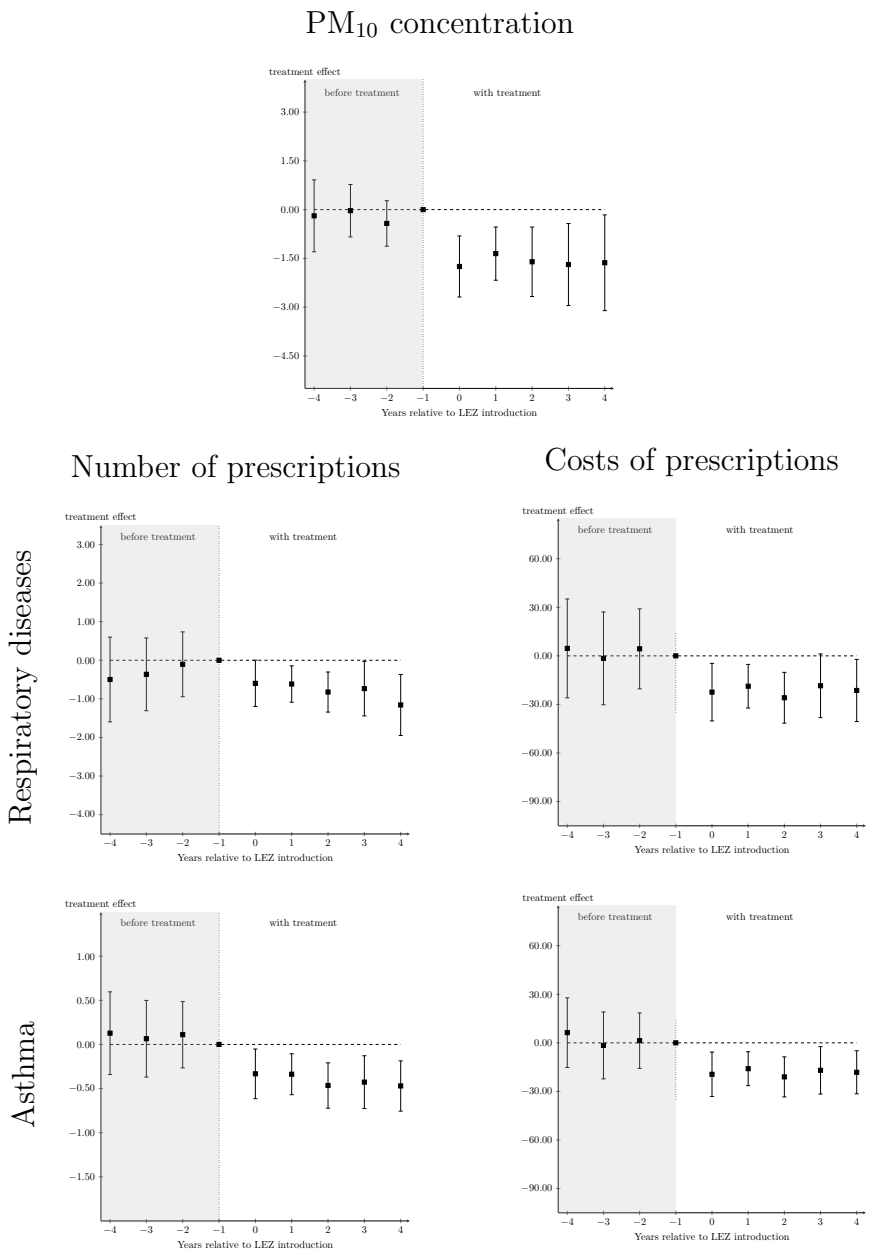


Figure F.1. : Excluding Already Treated Counties from the Control Group

Note: This figure presents event-study coefficients that show how LEZs affect PM_{10} concentrations and the medication of respiratory diseases for a sample that excludes already treated counties from the control group. The dependent variable is either the average PM_{10} level in $\mu g/m^3$ or the number or the costs 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, and LEZ wave–treated fixed effects. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regressions are weighted by county–quarter cell size. Standard errors are clustered at the county level. Confidence intervals refer to the 5% level of significance.

2. Event-Study Specification

We estimate event-study specifications of our stacked DID model. The regression equation becomes

$$Y_{ctj} = \sum_{\tau} \theta_{\tau}(Treat_{cj} \times D^{\tau}_{tj}) + W'_{ctj}\delta + X'_{c\bar{t}j}\pi_t + \lambda_j Treat_{cj} + f^p_{ctj} + f^s_{ctj} + \gamma_c + \gamma_{tj} + \gamma_{st} + \eta_{ctj} \tag{F.1}$$

where the parameter of interest is θ_{τ}. It either captures the marginal effect of LEZs on the mean PM_{10} exposure in early-life or pre-school health status of children born in year τ prior or post to treatment (see Figure 1). 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_{τ} estimates in event time. The main difference between the standard two-way fixed effect event study and our dynamic estimator in Equation (F.1) is that we eliminate time-invariant unobservables both within and between LEZ implementation waves by including $\lambda_j Treat_{cj}$ as well as wave-specific event-time trends that do not appear in calendar time by including γ_{tj}. Just as in our main specification, we include implementation wave specific controls for partial treatments (f^p_{ctj}) and LEZ stringency (f^s_{ctj}). However, including wave specific partial treatment controls in each of the years two through five leads to a high degree of collinearity that prevents us from estimating coefficients for the individual pre-treatment period. Therefore, we slightly relax our event study specification compared to our main specification and replace the four partial treatment controls with one binary control that is equal to one if a child is partially treated in the years two through five for each implementation wave. The adjustment applies only to regressions where the dependent variable is the number or the costs of prescriptions that accumulate over the children's five years of pre-school childhood. This is because we do not include partial treatment controls that refer to a period after the outcome variable is measured and pollution exposure refers to the first year of the children's life only. Concerning the prescription outcomes, we acknowledge that our event-study specification may absorb effects from partial treatments to a somewhat lesser degree than in our main analysis. We believe this to be tolerable because the estimated coefficients for the pre-treatment period indicate that the relaxed specification still suffices to absorb potential differences linked to partial treatments after the first year of life.

G. Instrumental Variable Analysis

Throughout our analysis we report reduced form estimates of the effect of LEZ implementation on PM_{10} concentrations and medication of respiratory diseases. We complement this analysis by providing an instrumental variable (IV) estimation. In Table G.1 we present coefficients that represent the effect of a 1 $\mu g/m^3$-reduction in early exposure to PM_{10} induced by the implementation of an LEZ on the number and the costs of prescriptions for respiratory disease that accumulate over the five years of pre-school childhood.

In line with our main analysis, we find that the number and the costs of required prescriptions decreases as children's pollution exposure in early-life declines. For instance, a 1 $\mu g/m^3$-reduction in PM_{10} exposure *in utero* and during the first year of life, decreases the number of prescriptions required for respiratory diseases in general by 0.46 and for asthma specifically by 0.30 (Panel A). We can compare these effects to the reduced form results from our main analysis, by dividing the treatment effect of LEZs on the prescription outcomes in Table 2 by the treatment effect of LEZs on the PM_{10} level in Table 1. For instance, for the number of prescriptions for asthma we would expect that for every 1 $\mu g/m^3$ decrease in PM_{10} pollution, the number of required prescriptions reduces by 0.25 (0.335/1.317) which is close to the corresponding coefficient 0.30 in G.1. The IV analysis also confirms that the effects exist at both the intensive and the extensive margin.

The unbiasedness of the IV estimates depends on two crucial assumptions. First, LEZs need to be a strong instrument for changes in particulate matter pollution. In Section IV.A we present evidence for a strong first-stage, showing that PM_{10} levels decline significantly and persistently in response to LEZ implementation. Moreover, we include weak-instrument-robust inference in Table G.1. The reported Anderson-Rubin confidence intervals corroborate that all average effects are positive and significantly different from zero. Second, for consistency, it must be the case that LEZ introduction affects health outcomes only via its impact on air pollution. While we cannot conclusively show the validity of this assumption, we conduct a number of robustness checks in Section IV.E. We do not find any evidence that LEZ implementation affects the characteristics of the population or any health outcomes other than those linked to air pollution. However, we do find evidence that the policy's effects are not limited to particulate matter pollution. In fact, we show that LEZs reduce both PM and NO_2 but no other pollutants. Therefore, we argue that our identification strategy suf-

fices to provide an upper-bound estimate for the dose-response relationship between PM_{10} and child health. Because policy measures are hardly ever limited to a single pollutant, an upper bound is the best possible approximation. Nevertheless, it is highly relevant for cost-benefit analyses that provide the basis for policy decisions. Similarly, other studies attribute estimated IV-effects to air pollution more generally rather than to a particular pollutant (Chay and Greenstone, 2003*a*; Currie and Neidell, 2005; Arceo, Hanna and Oliva, 2016; Knittel, Miller and Sanders, 2016; Deryugina et al., 2019; Sager, 2019; Colmer et al., 2020).

Table G.1—: The Effect of Early-Life PM_{10} Exposure on Medication of Respiratory Diseases throughout Pre-School Childhood

	(1) Respiratory diseases	*(2)* Asthma
	A. Number of prescriptions	
PM_{10} mean	0.457	0.301
s.e.	(0.224)	(0.130)
mean	14.532	2.558
CS_{AR}	[0.102 - 1.579]	[0.119 - 1.034]
F_{AR} (p-value)	6.124 (0.015)	11.041 (0.001)
	B. Costs of prescriptions	
PM_{10} mean	16.111	14.756
s.e.	(7.195)	(6.248)
mean	228.916	77.573
CS_{AR}	[5.527 - 54.909]	[5.926 - 49.820]
F_{AR} (p-value)	8.855 (0.004)	11.186 (0.001)
	C. Extensive Margin: Share of sufferers	
PM_{10} mean	-0.001	0.015
s.e.	(0.007)	(0.008)
mean	0.801	0.228
CS_{AR}	[-0.023 - 0.016]	[0.003 - 0.057]
F_{AR} (p-value)	0.047 (0.828)	5.729 (0.018)
	D. Intensive Margin: Prescriptions per sufferer	
PM_{10} mean	0.462	0.344
s.e.	(0.228)	(0.345)
mean	17.908	11.161
CS_{AR}	[0.099 - 1.597]	[-0.541 - 1.409]
F_{AR} (p-value)	5.982 (0.016)	0.931 (0.337)

Note: This table reports instrumental variable estimates that indicate the health effect of PM_{10} exposure during the *in utero* period and the first life year. The dependent variable is either the number of prescriptions per child (Panel A), their costs in Euro per child (Panel B), the share of children in the cohort that require at least one prescription (Panel C), or the number of prescriptions per child with at least one prescription (Panel D). It is accumulated over the first five years of a child's life on average. Moreover, it refers to either prescriptions for respiratory diseases in general or asthma specifically. The dependent variable is composition-adjusted for the size of 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. We include weather and socio-economic controls as well as controls for partial treatments and LEZ stringency. The regressions are weighted by the size of the birth county–birth quarter cell. Standard errors in parentheses are clustered at the county level. The sample size is $12,972$. The table also reports weak-instrument-robust inference. The Anderson-Rubin-confidence sets (CS_{AR}) provide robust confidence intervals with a coverage probability of 95%. The F-distributed Anderson-Rubin-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.

H. Comparison to the Literature

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

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 IV estimates for the share of sufferers to these results. Our estimate for asthma in Column (2) in Panel B of Appendix Table G.1 implies a substantially larger risk ratio of 1.066 at the mean, which is outside of the meta-study's 95% confidence interval. Our result is also greater than the implied odds ratio from studies limited to children aged up to six, which suggest an odds ratio of 1.045, but is now contained in the 95% confidence interval calculated by Khreis et al. (2017).

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 Stockholm's 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 the number of prescriptions for asthma (3.3), expenditures (5.4), and the share of sufferers (1.9) are comparable.[34] The 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 ex-

[34]The calculations are based on the IV estimates for asthma in Column (2) of Appendix Table G.1. These point estimates are then multiplied with the mean PM_{10} exposure and divided by the mean of the outcome to obtain elasticities.

posure, but mainly focus on $PM_{2.5}$.[35] 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. 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.

[35]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.

I. Unconditional Quantile Regression

We estimate the effect of LEZs across the unconditional distribution of the number of prescriptions for respiratory diseases and asthma using the residualized unconditional quantile regression approach by Borgen, Haupt and Wiborg (2021). In a first step, we estimate a common auxiliary regression at the level of the individual child with treatment status as the dependent variable and which includes birth county, birth state–birth quarter fixed effects, weather and socio-economic controls as well as controls for whether there are partial treatments after the first year of pre-school childhood and LEZ stringency increases over the five years of pre-school childhood. We retain the residuals from this regression.

In a second step, we regress our health outcome on the residuals obtained in the first step using the unconditional quantile estimator suggested by (Firpo, Fortin and Lemieux, 2009). Their approach uses the re-centered influence function (RIF) defined in Equation (I.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.

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

The expected value of the RIF equals the quantile of the unconditional distribution.[36] By the law of iterated expectations and integration over the conditional mean, the unconditional quantile q_θ can be expressed as

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

where X is the vector of covariates that in our case only include the residual from the first step and F_X is the marginal distribution function of X. To obtain the marginal treatment effects on the unconditional quantile q_θ, we

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

take the sample quantile $\hat{q_\theta}$ and retrieve the density $\hat{f_H}$ using a Gaussian kernel method.[37] To obtain $\widehat{RIF}$, we substitute both into Equation (I.1). Second, 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:

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

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 estimation times are prohibitively long when using a stacked design, we limit our quantile regression analysis to a standard two-way fixed effect estimation knowing that some caveats may apply. For example, Section IV.E shows that the two-way DID estimator leads to results smaller in magnitude compared to our stacked DID estimator. Table A.5 in the Appendix features all coefficients and standard errors which are bootstrapped using 500 repetitions and clustered at the county level.

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

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Appendix References

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

Alexander, Diane, and Hannes Schwandt. 2019. "The Impact of Car Pollution on Infant and Child Health: Evidence from Emissions Cheating." *Working Paper No. WP-2019-4.*

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

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

Arceo, Eva, Rema Hanna, and Paulina 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): 257–280.

Barwick, Panle Jia, Shanjun Li, Deyu Rao, and Nahim Bin Zahur. 2018. "The Morbidity Cost of Air Pollution: Evidence from Consumer Spending in China." *NBER Working Paper 24688.*

Beatty, Timothy K.M., and Jay P. Shimshack. 2014. "Air Pollution and Children's Respiratory Health: A Cohort Analysis." *Journal of Environmental Economics and Management*, 67(1): 39–57.

Benjamini, Yoav, and Yosef 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): 289–300.

Bharadwaj, Prashant, Joshua Graff Zivin, Jamie T. Mullins, and Matthew 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): 1475–1482.

Borgen, Nicolai T, Andreas Haupt, and Øyvind N Wiborg. 2021. "A New Framework for Estimation of Unconditional Quantile Treatment Effects: The Residualized Quantile Regression (RQR) Model." *Working Paper.*

Chay, Kenneth Y., and Michael Greenstone. 2003. "Air Quality, Infant Mortality, and the Clean Air Act of 1970." *NBER Working Paper 10053*, 1–43.

Colmer, Jonathan, Dajun Lin, Siying Liu, and Jay Shimshack. 2020. "Why are Pollution Damages Lower in Developed Countries? Insights from High Income, High-Pollution Hong Kong." *CEP Discussion Paper1702.*

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

Currie, Janet, Lucas Davis, Michael Greenstone, and Reed Walker. 2015. "Do Housing Prices Reflect Environmental Health Risks? Evidence from More than 1600 Toxic Plant Openings and Closings." *American Economic Review*, 105(2): 678–709.

Deryugina, Tatyana, Garth Heutel, Nolan H Miller, David Molitor, and Julian Reif. 2019. "The Mortality and Medical Costs of Air Pollution: Evidence from Changes in Wind Direction." *American Economic Review*, 109(12): 4178–4219.

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

Firpo, Sergio, Nicole M. Fortin, and Thomas Lemieux. 2009. "Unconditional Quantile Regressions." *Econometrica*, 77(3): 953–973.

Goodman-Bacon, Andrew. 2018. "Difference-in-Differences with Variation in Treatment Timing." *NBER Working Paper 25018*, 1–46.

Häussler, Bertram, and Ariane Höer. 2016. *Arzneimittel-Atlas 2018: Der Arzneimittelverbrauch in der GKV.* MWV Medizinisch Wissenschaftliche Verlagsgesellschaft.

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

Isen, Adam, Maya Rossin-Slater, and W. Reed 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): 848–902.

Jaunzeme, Jelena, Sveja Eberhard, and Siegfried Geyer. 2013. "Wie repräsentativ sind GKV-Daten?" *Bundesgesundheitsblatt-Gesundheitsforschung-Gesundheitsschutz*, 56(3): 447–454.

Karlsson, Martin, and Nicolas R. Ziebarth. 2018. "Population Health Effects and Health-Related Costs of Extreme Temperatures: Comprehensive Evidence from Germany." *Journal of Environmental Economics and Management*, 91: 93–117.

Khreis, Haneen, Charlotte Kelly, James Tate, Roger Parslow, Karen Lucas, and Mark Nieuwenhuijsen. 2017. "Exposure to Traffic-Related Air Pollution and Risk of Development of Childhood Asthma: A Systematic Review and Meta-Analysis." *Environment International*, 100: 1–31.

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

Sager, Lutz. 2019. "Estimating the Effect of Air Pollution on Road Safety Using Atmospheric Temperature Inversions." *Journal of Environmental Economics and Management*, 98: 102250.

Simeonova, Emilia, Janet Currie, Peter Nilsson, and Reed Walker. 2019. "Congestion Pricing, Air Pollution and Children's Health." *Journal of Human Resources*, 0218–9363R2.

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