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DISCUSSION PAPER SERIES

IZA DP No. 13418

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ABSTRACT

Pandemic Meets Pollution: Poor Air Quality Increases Deaths by COVID-19*

We study the impact of short-term exposure to ambient air pollution on the spread and severity of COVID-19 in Germany. We combine data on county-by-day level on confirmed cases and deaths with information on local air quality and weather conditions and exploit short-term variation in the concentration of particulate matter (PM10) and ozone (O3). We apply fixed effects regressions controlling for global time-varying confounding factors and regional time-invariant confounding factors on the county level, as well as potentially confounding weather conditions and the regional stage of the pandemic. We find significant positive effects of PM10 concentration after the onset of the illness on COVID-19 deaths specifically for elderly patients (80+ years): higher levels of air pollution by one standard deviation 3 to 12 days after developing symptoms increase deaths by 30 percent (males) and 35 percent (females) of the mean. In addition, air pollution raises the number of confirmed cases of COVID-19. The timing of results supports mechanisms of air pollution affecting the severity of already realized infections. Air pollution appears not to affect the probability of infection itself.

JEL Classification: I12, I18, Q53

Keywords: COVID-19, health, air pollution, Germany

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1 Introduction

The novel coronavirus (SARS-CoV-2) has sparked the largest public health and economic crisis in recent history. Up to date, the coronavirus disease 2019 (COVID-19) pandemic with millions of confirmed cases has claimed more than 450,000 deaths globally.[1] World Bank projections of economic damage range up to a 5.2 percent contraction in global GDP, which would be the deepest recession in decades (World Bank, 2020). Other pressing social and economic issues, most notably environmental issues of climate change and pollution, have almost vanished from the public debate and are only slowly re-emerging. In this paper, we show that both environmental pollution and the COVID-19 pandemic are significantly connected: higher levels of local air pollution increase the number of deaths of COVID-19, leading to a more severe course of the pandemic.

Three plausible mechanisms link air pollution to the spread and course of severe acute respiratory infections (SARI) and influenza-like illnesses (ILI). First, long-run exposure to air pollution is linked to medical pre-conditions such as illnesses of the respiratory system that can exacerbate the course of the disease (Analitis et al., 2006; Atkinson et al., 2001; Le Tertre et al., 2002; Dominici et al., 2003; Katsouyanni et al., 2001). Second, short-term exposure to air pollution leads to inflammatory reactions and lower immune responses to new infections (Ciencewicki and Jaspers, 2007; Contini and Costabile, 2020; Martelletti and Martelletti, 2020). Third, higher levels of air pollution increase the ability of viruses for airborne infection by prolonging the time the virus remains in open air (Cui et al., 2003; Frontera et al., 2020). Whether these mechanisms are at play in the case of the new coronavirus remains open to debate.

Against this background, we add to the epidemiological literature empirically linking levels of different air pollutants to the onset and severity of SARI and ILI in general, and the new COVID-19 disease specifically. We study the interaction pattern of short-term exposure to ambient air pollution, measured by levels of particulate matter (PM10) and ozone (O3) in German counties, and the number of newly confirmed cases and deaths of COVID-19. Fixed effects for county and date keep county-specific time-invariant and national time-variant confounding variables constant. We further control for confounding weather conditions and the county-specific state of the pandemic.

We find significant effects of higher air pollution on both the number of deaths as well

[1] Numbers taken from the John Hopkins University Coronavirus Resource Center: `https://coronavirus.jhu.edu/map.html` (last updated: June 22, 2020).

as the number of confirmed cases by day and county. Effects are specifically pronounced for patients aged 80 and above. In this age group, a one standard deviation increase in PM10 (6.3 $\mu g/m^3$) 3-7 days after the onset of illness increases the number of deaths among male patients by 30 percent of the baseline mean of about 1 death per 100k population. Additionally, we find that air pollution after the onset of illness leads to increased numbers of cases which is likely explained by aggravated symptoms swaying patients to get tested.

Our results contribute to a better understanding of the determinants and mechanisms of spread and severity of ILI in general and COVID-19 specifically by isolating the causal effect of air pollution from mere correlations between pollution levels and confirmed deaths and cases. This sets us apart from most of the related literature summarized in the next section which mainly relies on pure cross-sectional or time-series variation and is thus unable to determine a causal link. To the best of our knowledge, there are few studies using a similar approach as ours. Clay et al. (2018) exploit differential timing of the Spanish flu pandemic across US cities to show that contemporary short-term air pollution levels increased the number of deaths. Moghadas et al. (2020) show that US county-by-month influenza-related hospitalizations increase with higher air pollution. Persico and Johnson (2020) show with respect to the COVID-19 pandemic that air pollution, instrumented by a rollback of environmental regulations in the US, increases both cases and case fatality of COVID-19.

We add to these closely related papers in two important ways. First, whether or not results from previous pandemics and/or other regional settings are generalizable to other external settings, both in case of disease and locality, is yet to be determined. To the best of our knowledge, we are the first to provide evidence on the linkage between air pollution and the spread as well as the severity of COVID-19 for Germany. Second, we use fine-grained data on daily pollution levels which allow us to identify critical time windows relative to the onset of the illness when pollution levels most crucially affect the mortality risk. This allows us to discriminate between different mechanisms at play. As effects materialize only after the onset of illness, our results support mechanisms causing inflammatory reactions, reducing the immune response and aggravating symptoms. Our results do not support proposed mechanisms of higher ability of the virus for airborne infections due to higher air pollution. By focusing on short-term changes in the exposure to air pollution, our results do not speak to potential effects through longer-run exposure to air pollution on respiratory preconditions.

Our results imply that moving patients at risk aged 60 and above out of more heavily polluted areas might be a way to reduce the number of deaths by COVID-19. This implication

might be of specific importance when the pandemic unfolds in less-developed world regions where air pollution and associated health risks are of much higher importance, e.g., through the more widespread (indoor) use of fossil fuels for cooking and heating, and where supply of high-quality medical care is constrained. So far, we show substantial effects of air pollution on the severity of COVID-19 in a setting of modest pollution levels in a Western society with sufficient supply of high quality health care, intensive care units and ventilators. There is reason to fear, and the need for additional research, that the effect of air pollution on the severity of COVID-19 will be intensified when these conditions are not met.

The remainder of this paper is organized as follows. Section 2 discusses plausible mechanisms through which higher air pollution might affect onset and severity of COVID-19 and summarizes the related literature. Section 3 describes the data and provides a descriptive overview on the course of the pandemic in Germany. Section 4 describes our empirical approach. Section 5 presents the main empirical results and Section 6 sets forth additional analyses. Section 7 concludes.

2 Background and Literature

Mechanisms. The medical literature has analyzed a number of direct physiological mechanisms how acute air pollution could affect the infection probabilities and severeness of respiratory virus infections, such as influenza and SARS (see Ciencewicki and Jaspers, 2007, for a review). Several authors speculate that these mechanisms could be at play in the case of COVID-19 (Contini and Costabile, 2020; Martelletti and Martelletti, 2020).

Direct physiological mechanisms range from lower initial immune response, reduced ability of macrophages (cells specialized in the detection and destruction of harmful organisms, including viruses), and stronger oxidative stress.[2] Specific to particulate matter (PM) concentration, mice display lower early immune responses when having been exposed to carbon black particles (Lambert et al., 2003). PM-exposed macrophages display lower abilities to devour viruses (Kaan and Hegele, 2003; Becker and Soukup, 1999). Several lab studies show that animals or cell cultures develop stronger oxidative stress and aggravated disease severity after being exposed to PM (Jaspers et al., 2005; Lee et al., 2014).

A second type of mechanisms is related to how air pollution is affecting the airborne transmission of viruses. A number of studies suggests that higher levels of air pollution,

[2] Oxidative stress describes a situation where the production of free radicals exceeds the metabolism's ability to counteract or detoxify and contributes to the pathogenesis of respiratory infections (Liu et al., 2017).

especially PM concentration, increase the ability of viruses for airborne infection and raise the initial viral load (Cui et al., 2003; Frontera et al., 2020). With respect to SARS-CoV-2 it has been shown that the infectious dose and the initial viral load is an important predictor for the severeness of cases (Zheng et al., 2020).

Besides these acute direct effects, air pollution has repeatedly been shown to be strongly linked to medical pre-conditions such as cardiovascular and respiratory diseases that have been found to crucially affect the severeness of COVID-19 (Analitis et al., 2006; Atkinson et al., 2001; Le Tertre et al., 2002; Dominici et al., 2003; Katsouyanni et al., 2001). As we focus in our empirical analysis on the effect of short-term changes in the exposure to pollutants, these long-term effects are not the focus of this study.

Epidemiological evidence. Tracing the outcome of these mechanisms in the field, a number of epidemiological studies proposes empirical links between measures of air quality and case numbers and deaths of respiratory virus infections. With few exceptions, the majority of these studies rely on either pure cross-sectional or regional time series variation. Both approaches limit opportunities to identify a causal link due to potential confounders on both the time and regional level. Spatial variation likely correlates with the presence of obvious confounders, such as population density, public transport usage and age composition. Variation in air pollution over time might be plagued by simultaneity and reversed causality issues. For example, several studies have shown that the reduced economic activity that follows an outbreak has strong effects on air pollution – an obvious challenge for the estimation of a causal effect of the latter on the former.

Based on cross-sectional variation, a positive link between regional air pollution and the local severeness of the COVID-19 pandemic and related severe acute respiratory and influenza-like virus infections has been shown. For the early and severely hit region of Northern Italy, several cross-sectional studies propose a relationship between high levels of air pollution and COVID-19 cases (Contini and Costabile, 2020; Pansini and Fornacca, 2020). Others have raised concerns that the high level of domestic bio mass fuel usage in developing countries might aggravate the impact of the pandemic (Thakur et al., 2020) – an effect that was already foreshadowed by evidence from the interaction of biomass fuel usage and the Spanish flu pandemic (Clay et al., 2018). For countries besides Italy, Andree (2020) and Cole et al. (2020) report correlations between air pollution and COVID-19 cases across Dutch municipalities. Travaglio et al. (2020) finds positive associations between markers of poor air quality and COVID-19 cases in England. For China, Yongjian et al. (2020) and Yao et al. (2020) describe

positive spatial associations between PM2.5 and PM10 and COVID-19 death rates. Wu et al. (2020) find that small increases in long-term PM2.5 concentration are associated with large increases in the COVID-19 death rate, yet acknowledging the inherent limitations of their study design.

Other studies focus on (regional) time series variation in PM. Several studies show positive time series correlations between daily PM levels and influenza-like illnesses in Chinese regions (Liang et al., 2014; Su et al., 2019; Huang et al., 2016). Chen et al. (2018) show Granger causality between PM2.5 and weekly influenza cases with specifically strong effects on the elderly. Similar relationships have previously been found for SARS (Cui et al., 2003).

Causal Effects of Air Pollution. Few studies have attempted to identify a causal effect of air pollution on the onset and severity of respiratory infections using quasi-experimental study designs. Clay et al. (2018) show that air pollution exacerbated the impact of the Spanish flu by applying fixed effects regressions exploiting the differential timing of the pandemic across US cities. Based on a similar methodological approach as our own, Moghadas et al. (2020) show that county-by-month influenza-related hospitalizations increase with higher air pollution. Persico and Johnson (2020) show with respect to the COVID-19 pandemic that air pollution, instrumented by a rollback of environmental regulations, increases both cases and case fatality of COVID-19 in the US.

3 Data and Descriptives

COVID-19 cases and deaths. We collect data on confirmed cases and deaths of COVID-19 by German counties (*Kreise*) from the official German COVID-19 reporting database by the Robert-Koch-Institut (RKI). In accordance with the Infection Protection Act (*Infektionsschutzgesetz*), the RKI collects daily reports from county-level public health offices on newly detected cases and deaths. Case reports are transmitted to the RKI by 0:00 a.m. on the respective day. The records contain the exact date on which the local public health office became aware of the case and recorded it electronically. For most cases, the data contains additional information on when the patient became ill with clinical symptoms according to the patient's own statement or according to the statement of the treating physician (illness onset). Cases are separately recorded by gender and age group (0–34, 35–59, 60–79, 80+). Daily case counts are regularly updated based on delayed lab confirmations and deaths of earlier recorded cases. The analysis of this paper is based on a snapshot of the data taken on May 26, 2020.

We use population counts of demographic groups to normalize case numbers and deaths to 100K inhabitants by county, gender and age.

Table 1 displays average day-by-county cases and deaths per 100K population, as well as cell population shares for gender $\times$ age groups over the entire window of observation from February 1 until May 26, 2020. The lowest row displays average cases and deaths across all age $\times$ gender groups. On average, we observe 1.7 confirmed cases and 0.08 deaths per day per county, a case fatality rate (i.e., the probability of dying of COVID-19 conditional on being a confirmed case) of 4.7 percent. Germany's comparably low case fatality rate compared to European hotspots such as Spain (14.5 percent) or Italy (11.5 percent)[3] has been explained by several arguments: higher numbers of intensive care beds, higher test intensity and demographic factors such as a younger population living largely separated from their parent's generation (Bayer and Kuhn, 2020).

There is considerable heterogeneity in the number of deaths and confirmed cases by age and gender. Below age 80, case prevalence varies little by age and gender between 1.2 and 2.1 cases per 100K population in a respective demographic group. Only among the very old aged 80 and above we observe a higher prevalence of 2.84 cases per 100K population for males and of 3.06 cases for females. Deaths due to COVID-19 are concentrated among the very old, too. Below age 60, we observe close to zero deaths per 100K per demographic group. Among male patients between 60 and 79 we observe 0.18 daily deaths per 100K population. The resulting case fatality rate in this demographic group is about 11 percent. Female patients of the same age group display a significantly lower case fatality rate of just 4.5 percent. For very old patients above 80 years, deaths are substantially higher: per 100K population, we observe about 1 death per day per county for male and about .7 deaths per day per county for female patients. Associated case fatality rates are 34 percent for male and 23 percent for female patients.

To understand the limits of interpretation of the data, it is important to discuss the testing regime that has been in place during the peak of the pandemic. COVID-19 cases are confirmed after a patient has been positively tested. During the peak of the pandemic, patients in Germany mostly were eligible for scarce testing capacities after having developed symptoms (acute respiratory symptoms and/or loss of sense of taste/smell), and after having been in contact with a confirmed case. Thus, it can be expected that the majority of asymptomatic cases will have remained undetected. Early estimates of the share of symptomatic cases based

[3] Fatality rates are from `https://ourworldindata.org/coronavirus/` (last accessed June 23, 2020).

Table 1: Descriptives: COVID-19 cases and deaths (by county and day)

		Cases per 100K		Deaths per 100K		Pop. share	
Age range	Gender	Mean	SD	Mean	SD	Mean	SD
0–34	Men	1.24	3.49	0	.03	.19	.02
0–34	Women	1.36	3.39	0	.03	.17	.02
35–59	Men	1.88	4.26	.01	.23	.18	.01
35–59	Women	2.13	4.68	0	.15	.17	.01
60–79	Men	1.64	4.56	.18	1.23	.11	.01
60–79	Women	1.34	3.86	.06	.63	.12	.02
80+	Men	2.84	11.62	.97	5.95	.03	0
80+	Women	3.06	13.55	.7	4.58	.04	.01
All	All	1.69	3.14	.08	.34	1	0

Notes: This table summarizes means and standard deviations of confirmed cases and deaths of COVID-19, as well as average population shares of demographic groups separated by age and gender. All numbers averaged across counties. *Source*: RKI and Statistical Office.

on Chinese data of the City of Wuhan range from 5 to 9.2 percent (Read et al., 2020; Nishiura et al., 2020). A population study in the county of Heinsberg in North Rhine-Westphalia, Germany, estimates a 5-fold higher number of infected individuals than the number of officially reported cases (Streeck et al., 2020).

Timeline of the pandemic. First cases of COVID-19 in Germany were registered and contained near Munich, Bavaria, at the end of January 2020. After that, only on February 25 additional cases, which were related to the outbreak in Italy, were detected in in the state of Baden-Wuerttemberg. A first major cluster emerged at the beginning of March in the county of Heinsberg in the state of North Rhine-Westphalia. Shortly after, additional clusters broke out all over the country. German disease control initially reacted by local containment strategies, and federal and state governments stressed that the country was well prepared for a larger outbreak.

On March 11, 2020, on the day the World Health Organization announced the pandemic status of the Corona outbreak, Chancellor Angela Merkel asked the German population to do anything to avoid the further spread of the virus. Schools and public child care as well as national borders were closed on March 15. On March 16, closures of bars, restaurants, churches and shops followed. Federal states reacted with severe mandatory social-distancing measures from March 20 on. Groups outside were restricted to no more than two persons if from different household. In some states leaving one's home was only allowed for important or recreational activities. From March 30 on, visits to hospitals and homes for elderly were

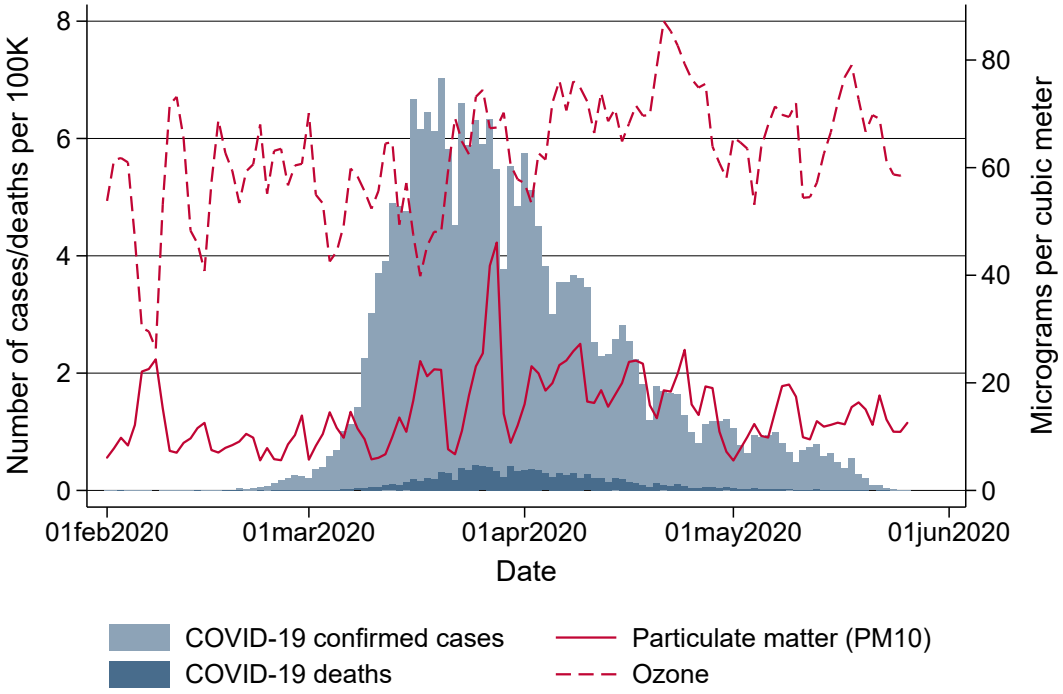

Figure 1: Daily variation in new confirmed cases and deaths and mean air pollution

Note: This graph shows the total number of new confirmed cases and deaths of COVID-19 in Germany as well as the mean concentration of particulate matter (PM10) and ozone (O3) by date. *Source*: RKI and UBA.

prohibited. After numbers of newly infected decreased again, counter-measures were slowly retracted from April 15 on. Since mid-June, schools, kindergartens and most shops are open again.

Figure 1 summarizes the course of the pandemic over our window of observation from February to May 2020. Registered cases are displayed in light-blue, deaths in dark-blue bars. Until June 8, 184,193 cases, 8,674 deaths and about 169,600 recoveries were reported to the RKI. The pandemic has hit German regions differentially. Figure A.1 in the Appendix shows the total number of confirmed cases and deaths per 100K of population as of May 26, 2020. Following first clusters that had emerged in the states of North Rhine-Westphalia, Bavaria and Baden-Wuerttemberg, these states remained the hotspots of the pandemic. Northern and especially Eastern states (with the exception of the city of Berlin) were hit much less severely, with some counties up to beginning of June experiencing less than 50 cumulative cases per 100K population.

Air pollution. We assess local levels of air pollution by particulate matter (PM) which measures the concentration of small airborne particles including dust, dirt, soot, smoke and liquid

droplets. PM may be emitted by natural sources such as bush fires, dust storms, pollens and sea spray, or anthropogenic ("man-made") sources like motor vehicle emissions and industrial processes. In our main empirical analysis, we focus on PM10, i.e., the concentration of particles up to a diameter of 10 μm. We later corroborate our results using PM2.5. Unfortunately, the coverage of PM2.5 measurements in Germany is significantly scarcer than for PM10. For the window of analysis, measurements of these smaller particulates are only available for a selective set of counties. Both measures are highly correlated and lead to similar aforementioned physical reactions of the human body.

We further assess levels of ozone (O3) as a second dimension of air pollution. Ozone arises from reactions under sunlight of nitrogen oxide with so-called 'reactive organic substances'. These substances mainly stem from motor vehicle exhaust and aviation. Ozone leads to similar inflammatory reactions like PM10 (Ciencewicki and Jaspers, 2007). As we focus on the effect of PM10, we treat ozone as a confounder in our regressions. We separately show patterns of the effect of ozone on onset and severity of the disease in the additional analyses.

Data on PM10 and O3 are provided on a daily basis by the air pollution monitoring system of the German Federal Environment Agency (*Umweltbundesamt*, UBA). Data is available on the geo-coded monitor level which allows us to assign levels of air pollution to counties. Specifically, we compute a county's daily pollution level as the inverse-distance weighted mean of all monitors within a radius of 25 km around the county centroid as a proxy for the population center. Figure A.3 displays the coverage of counties through monitors.

Table 2 summarizes county-levels of daily and 5-day averages of air pollution. The 5-days averages are the basis for the empirical analysis. All pollutants are measured in units of $\mu g/m^3$. As in the case of confirmed COVID-19 cases per population, Figure A.2 in the Appendix shows strong heterogeneity in air pollution across counties. PM10 is closely associated with industry agglomeration and population density, with highest levels in Western Germany, concentrated in the highly populated and industrialized regions in North Rhine-Westphalia, Rhineland-Palatinate and Baden-Wuerttemberg following the rivers Rhine, Main and Ruhr which connected the former heavily industrialized regions of Western Germany. Interpreting simple spatial correlations between average levels of air pollution and local onset and severity of the COVID-19 pandemic might lead to erroneous claims about a causal effect of the former on the latter. We will instead focus in our empirical analysis on within-county changes in short-term exposure to air pollution to circumvent this identification problem.

Solid and dashed red lines of Figure 1 display average levels of our main independent of

Table 2: Descriptives: Air pollution and weather (by county and day)

		Unit	Avg. Period	Mean	SD	Min	Max
Air pollution	PM10	$\mu g/m^3$	1 day	14.5	8.5	1	95
	PM10	$\mu g/m^3$	5 days	14.5	6.3	2.2	45
	Ozone	$\mu g/m^3$	1 day	62.3	14.8	2.1	132.5
	Ozone	$\mu g/m^3$	5 days	62.3	11.6	2.3	121
	PM2.5	$\mu g/m^3$	1 day	14.5	5.5	.4	50.6
	PM2.5	$\mu g/m^3$	5 days	8.7	4	1.1	32.9
Weather	Precipitation	mm/m^2	1 day	2	3.9	0	52.1
	Wind speed	m/s	1 day	4.2	2	.9	16.5
	Temperature	$^\circ$ C	1 day	8.4	4.3	-3.5	20.8

Notes: This table summarizes means and standard deviations of average pollution and weather measures across counties. *Source*: UBA and DWD.

interest: levels of PM10 and O3. Both variables display significant day-to-day variation over time. We do not observe trends over time to be affected by the global course of the pandemic. Most noteworthy, we do not observe a reduction in air pollution due to the reduced economic activity and traffic during the lockdown – rather, levels of PM10 even reached a maximum during the peak of the crisis. Reasons for this counter-intuitive development of air pollution lie in several sources. First, traffic-related emissions only contribute about 19 percent to overall emissions of particulate matter PM10, while other sources such as industrial processes, heating and agriculture arguably less affected by the lockdown contribute up to about 70 percent.[4] Further, while economic activity was drastically reduced, economic sectors being hit hardest by the lockdown, such as the hospitality, entertainment industry and retailing, are characterized by rather low levels of emissions. Strongly polluting industries in the manufacturing sector kept up their production during the lockdown.

Weather. Another important reason for the high levels of air pollution experienced during the lockdown lies in the weather conditions which drastically changed in Germany almost simultaneously to the lockdown. Levels of air pollution are heavily influenced by local weather conditions. Wind speed and precipitation reduce the level of air pollution by "washing away" particulate matter. Wind speed and precipitation may also have a direct effect on the virus spread by affecting contact probabilities and virus survival in the air, hence we control for these variables as potential confounders. We use daily local measurements of weather conditions provided by the German Meteorological Services (*Deutscher Wetterdienst*, DWD) as controls

[4] Calculations based on https://www.umweltbundesamt.de/daten/luft/luftschadstoff-emissionen-in-deutschland#ermittlung-der-emissionsmengen (last accessed: June 22, 2020).

in our regressions. Daily county means of weather conditions (precipitation in mm/m^2, wind-speed in m/s and temperature in degree Celsius) are listed in Table 2. Figures A.4 and A.5 in the Appendix highlight how both average precipitation and wind speed dropped to extremely low levels over the entire observation window, partially explaining the high levels of air pollution coinciding with the pandemic.

4 Empirical Approach

To estimate the effect of higher air pollution levels on COVID-19 case numbers and deaths, we compare changes in case and death numbers in relation to changes in short-term exposure to air pollution. Unobserved factors on the county level, date-specific shocks, and time-variant weather conditions and local timing of the pandemic are kept statistically constant.

We estimate the two-way fixed effects model

$$\text{COVID}_{it}^{g} = \alpha_i + \mu_t + \sum_{l=-3}^{3} \beta_l \overline{P}_{i,t-l} + \sum_{l=-3}^{3} \gamma_l \overline{W}_{i,t-l} + \delta \text{CASES}_{i,t-14}^{cum} + \varepsilon_{it}.$$

Here, COVID_{it}^{g} displays the dependent variable, i.e., deaths or confirmed cases in county i for date of illness onset t, normalized to 100K of population by gender-age group g. Fixed effects for the county (α_i) and date (μ_t) keep time-invariant confounding variables on the county level and nation-wide homogeneous time-variant confounders constant. We further control for five-day averages of local weather conditions $\overline{W}_{i,t-l}$ (temperature, wind speed, precipitation, as well as ozone levels). To capture heterogeneous dynamics and timing of the pandemic across counties, we control for lagged cumulative cases per 100K 14 days prior to t ($\text{CASES}_{i,t-14}^{cum}$) to compare counties in similar stages of the pandemic. Standard errors ε_{it} are clustered at the county level.

Our main independent variable of interest is a five-day average of PM10 $\overline{P}_{i,t-l}$, measured in lags and leads l relative to date of illness onset t. We define relevant lags and leads l of 17 to 13, 12 to 8 days and 7 to 3 days before the day of illness, a period of 2 days before to 2 days after the date of illness, and leads of 3 to 7 days, 8 to 12 days and 13 to 17 days after the date of illness onset.[5] This window of lags and leads encompasses the typical stages of the disease. The median incubation period between infection and onset of illness has been

[5] Results on daily changes or 3-day averages lead to qualitatively similar, but less precisely estimated patterns and are available on request.

reported to range between 5 to 6 days (WHO, 2020). Several studies report a median time between onset of illness until hospitalization of 4 days (Docherty et al., 2020; Chen et al., 2020). The time between hospitalization and intensive care is estimated as about one day (ISARIC, 2020). Other studies report the median time between onset of illness and pneumonia by 4 days, and by 8 days for acute lung failure (Guan et al., 2020; Li and Ma, 2020). Based on data from New York, on average, deceased patients were hospitalized for 9 days (Cummings et al., 2020).[6]

We remain ex ante agnostic about when exactly to expect the effects during the course of the illness.[7] For the number of deaths, plausible effects could happen both during and before the onset of the infection. Higher initial viral loads, e.g., through higher ability of the virus for airborne infection at high levels of air pollution, have been shown to lead to more severe progressions later in the course of the disease. But also effects after the onset of illness are plausible, when higher air pollution causes inflammatory reactions and causes additional work for already stressed immune systems, increasing the severeness of symptoms.

For the number of cases, at a first glance it appears only plausible to expect effects up to the date of onset of illness (the latest point where an infection could have happened), but not after. Yet, the incomplete testing that hampers the data collection allows for a less obvious channel of air pollution affecting the number of cases even after their onset of illness: as case confirmations rely on infected individuals to seek testing, symptoms aggravated through air pollution after infection and onset of illness can plausibly lead to higher confirmed cases.

5 Main Results

Effect of PM10 on deaths. Figure 2 displays coefficients of 5-day averages of PM10 for lags and leads surrounding the day of onset of illness on the number of deaths per 100K population in a respective demographic group. Coefficients stem from separate regressions on samples split by age group and gender. Pollution levels before the onset of illness appear unrelated to the severity of the pandemic. After the onset of illness, the effect of higher levels of PM10 is heterogeneous across age groups.

[6] See also the SARS-CoV-2 briefing note of the RKI at `https://www.rki.de/DE/Content/InfAZ/N/Neuartiges_Coronavirus/Steckbrief.html` (last accessed: June 26, 2020).

[7] This sets us apart from a common event study design since we cannot rely on a pre-treatment period as comparison group, as we do not ex ante know when the treatment occurs. Instead, we use as comparison period time windows where no effect is yet to be expected (before infection) or no more expected (after the illness).

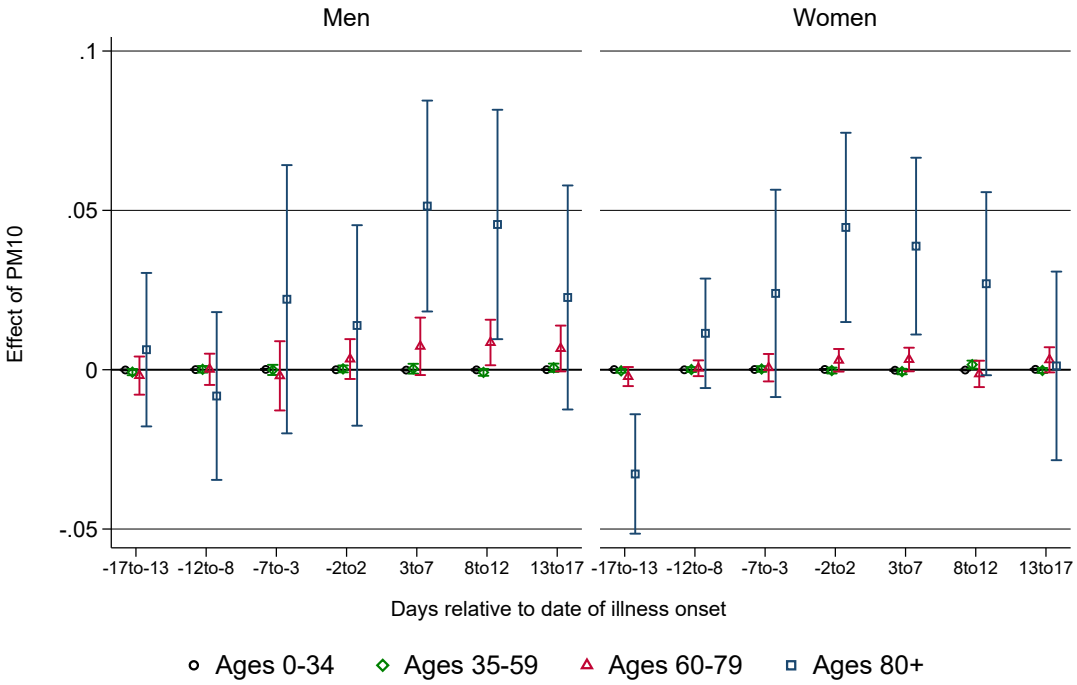

Figure 2: Effect of PM10 on new deaths from COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of PM10 concentration on the number of new deaths from COVID-19. *Source*: RKI, UBA and DWD.

For patients below 60, effect patterns are precisely estimated and remain close to zero. For male patients aged 60–79 years, we observe a significant effect between 8 and 12 days after the onset of illness: A one $\mu g/m^3$ increase in PM10 leads to 0.009 additional deaths per 100K population. Translated into standard deviations, a one standard deviation increase in air pollution 8 to 12 days after the onset of illness leads on average to 0.05 additional deaths per 100K population in a demographic group. Due to the low average death rate in this demographic group, this effect is sizable in relative terms and accounts for an increase of 30 percent of the mean fatality rate of this demographic group. For females, the effect is similar in relative terms, but remains insignificant.

For male patients from 80 years on, we observe a much stronger effect pattern which reaches its peak at 3 to 7 days after the onset of illness. At this point a one $\mu g/m^3$ increase in PM10 leads to 0.051 additional deaths per 100K population. This implies an effect of a one standard deviation increase in air pollution of on average 0.31 additional deaths per 100K population. In relative terms to the higher mean in this group, the effect again accounts for an increase of 30 percent of the mean fatality rate per 100K population of this demographic

group. Because of small sample sizes in this age group, effect sizes are rather imprecisely estimated, 95 percent confidence intervals range from 0.02 to 0.08 additional deaths per one $\mu g/m^3$ increase. For females, the effect pattern is similar. The effect of a one standard deviation increase of PM10 3 to 7 days after the onset of illness for female patients aged 80 and above accounts for a similar increase of about 35 percent of the mean number of deaths.

Effect of PM10 on confirmed cases. Figure 3 summarizes coefficients from regressions of the number of confirmed cases per 100K population on 5-day averages of PM10 surrounding the onset of illness, based on separate samples split by age and gender. Curiously, we do not observe effects of higher air pollution before the onset of illness, i.e., in a period where the infection likely has occurred. Thus, we do not find empirical support for a mechanism of air pollution increasing the ability of the virus for airborne infection. We do find, though, strong effects of higher air pollution after onset of illness for all age groups. Coefficient sizes again increase with age of the patients. For the critical age group of patients aged 80 and above, we find that a one $\mu g/m^3$ higher level of PM10 3 days after the onset of illness increases the number of confirmed cases for male patients by 0.15 cases per 100K population in this demographic group. This translates into an effect of a one standard deviation increase in PM10 of almost one additional confirmed case: an increase of 34 percent at the mean. Effect sizes are comparable for male and female patients.

At first glance, effects on the number of confirmed cases after the onset of illness might appear counter-intuitive. One has to keep in mind though the specific testing regime described above that was in place during the analysis window. Asymptomatic cases remained largely undetected as predominantly symptomatic patients after a contact to a confirmed case were tested. Higher levels of air pollution might have aggravated the severeness of already realized infections, increasing the number of symptomatic compared to asymptomatic cases, swaying people to seek testing, and thus also increasing the number of confirmed cases.

The significant effect of air pollution on confirmed cases has important implications for the interpretation of case fatality rates, i.e., deaths divided by cases per population, which are an important and often used indicator for the severity of the pandemic. Higher air pollution positively affects both nominator and denominator of such an indicator for all age groups. Thus, an effect of air pollution on the severity of COVID-19 measured in case fatality rates would be downward-biased if higher air pollution after the onset of illness aggravates symptoms and reduces the number of asymptomatic and thus potentially undetected cases. Figure A.6 in the Appendix supports this argument: case fatality rates for age groups aged 60 and above are

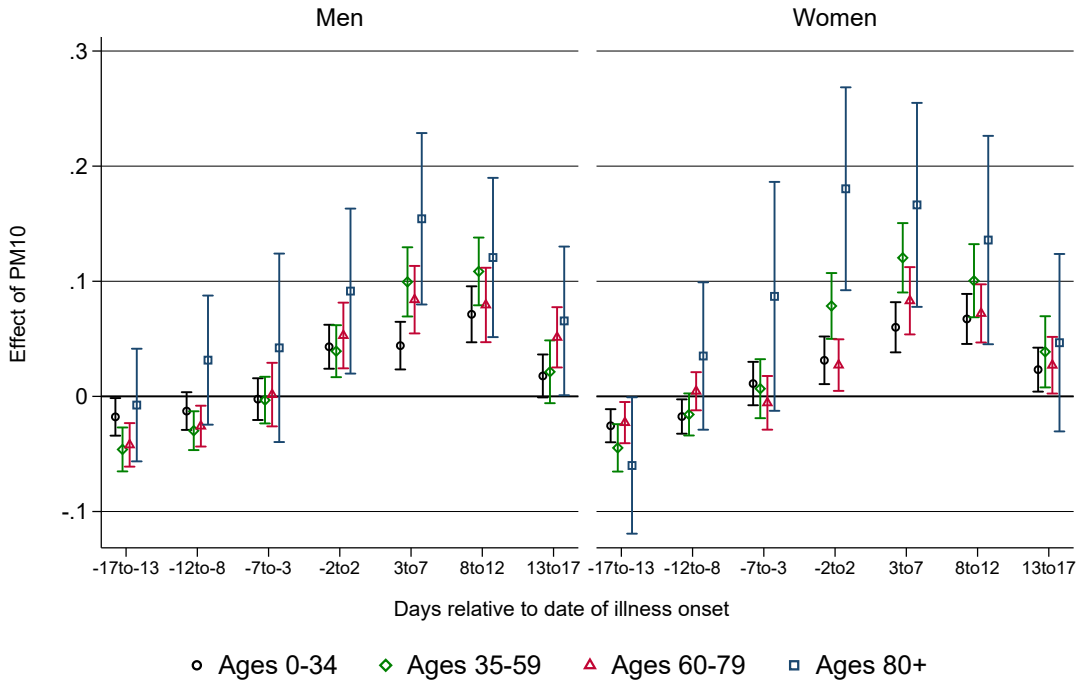

Figure 3: Effect of PM10 on new confirmed cases of COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of PM10 concentration on the number of new confirmed cases of COVID-19. *Source*: RKI, UBA and DWD.

indeed reduced in response to higher air pollution 8–12 days after the onset of illness. Note, though, that this bias does not affect our estimates of the effect on the *absolute* number of deaths per 100K population which we use as our dependent variable throughout the above analysis.

6 Additional analyses

Effects of PM2.5. Some of the related literature on the effect of air pollution has focused on PM2.5 – particulate matter with a diameter up to a fourth of PM10. PM2.5 indeed has a stronger potential to enter deeper into the human respiratory system and might lead to more severe inflammatory reactions. Unfortunately, the measurement of PM2.5 in Germany is less widespread and universal, and data is not as readily available as in the case of PM10. Additionally, as Figure A.7 shows, both measurements are strongly correlated with each other, so that we believe that the effect of PM10 already gives a sufficient approximation of the effect of smaller particulates.

We further estimate the effect of PM2.5 for those counties we were able to secure data

access. Figures A.8 and A.9 in the Appendix summarize the results of these regressions. The results mainly confirm the patterns we have found for PM10 in the main results, yet in a less pronounced way. Because we cannot rule out selectivity in the counties providing PM2.5 measurements, we draw our main conclusions from the results on PM10 described above.

Effects of Ozone. Although resulting from different pollution sources, levels of PM10 and ozone are in general highly correlated with each other. As several studies show (see Ciencewicki and Jaspers, 2007, for an overview), ozone itself has adverse respiratory health effects. While we consider ozone as a confounder to be kept constant in our main regressions summarized above, it is informative to look at coefficient patterns of ozone as an additional mechanism of how air pollution can affect deaths by and confirmed cases of COVID-19.

Figures A.10 and A.11 in the Appendix summarize the respective coefficient patterns resulting from the same regressions as summarized above. In contrast to the strong effects exhibited by PM10, we only observe very small effects of ozone arising between 3 and 7 days after the onset of illness for the oldest age group above 80 years. This effect is only visible for male patients. Similarly, we observe effects of ozone 3 to 7 days on the number of confirmed cases for both male and female patients of the oldest age group. Taken together, it appears that ozone leads to qualitatively similar, but quantitatively less pronounced effects on the onset and severity of COVID-19. Ozone arguably does not add to the proposed mechanism of increasing the ability of the virus for airborne infection. The observed effect on cases and severity therefore supports that our results are mainly explained through additional inflammatory reactions by which higher air pollution aggravates COVID-19 infections.

7 Conclusions

This paper studies the effect of short-term changes in the exposure to air pollution, measured by levels of particulate matter PM10, on the onset and severity of COVID-19 illness. We base our analysis on comprehensive data on confirmed cases and deaths from the official reporting provided by the Robert-Koch Institute, the German disease control authority. We merge this data with county $\times$ 5-day averages of pollutants and potentially confounding weather conditions. To isolate the effect of air pollution from confounding factors, we exploit changes in the 5-day average levels within German counties, keeping constant time-variant global confounders, time-invariant confounders on the county level as well as the regional progress level of the pandemic, approximated by the cumulative number of cases in a county.

Effect patterns are heterogeneous by age groups. Confirmed cases and deaths of persons below age 60 are not affected. Strongest effects are found for at-risk patients aged 80 and above. In this age group, a one standard deviation higher air pollution 3 to 12 days after the onset of illness increases deaths by 30 percent (males) and 35 percent (females) of the mean of the respective demographic group. We further find effects on the number of confirmed cases: a one standard deviation increase in PM10 raises confirmed cases in the group of patients 80 and above by 34 percent of the mean. Effect sizes are comparable for male and female patients.

Our empirical setting allows us to causally interpret the estimated effects, which sets us apart from studies relying on pure cross-sectional or time-series variation in pollution levels. By doing so, we contribute to a better understanding of the determinants of onset and severity of the new COVID-19 illness. Fine-grained daily data on the onset of illness allows us to discriminate between transmitting mechanisms: The timing of the estimated effects, affecting severity only after the onset of illness, supports mechanisms of increased inflammatory reactions causing additional stress for the immune system.

Further, our results show substantial effects in a setting of modest pollution levels in a Western society with sufficient supply of high quality health care, intensive care units and ventilators. There is reason to fear, and the need for additional research, that the effect of air pollution on the severity of COVID-19 will be intensified when the pandemic reaches less-developed world regions where particulate matter pollution and associated health risks are of much higher importance, e.g., through the more widespread (indoor) use of fossil fuels for cooking and heating, and supply of high-quality medical care is constrained. Moving high-risk patients aged 60 and above out of more heavily polluted areas might be a way to reduce the number of deaths by COVID-19.

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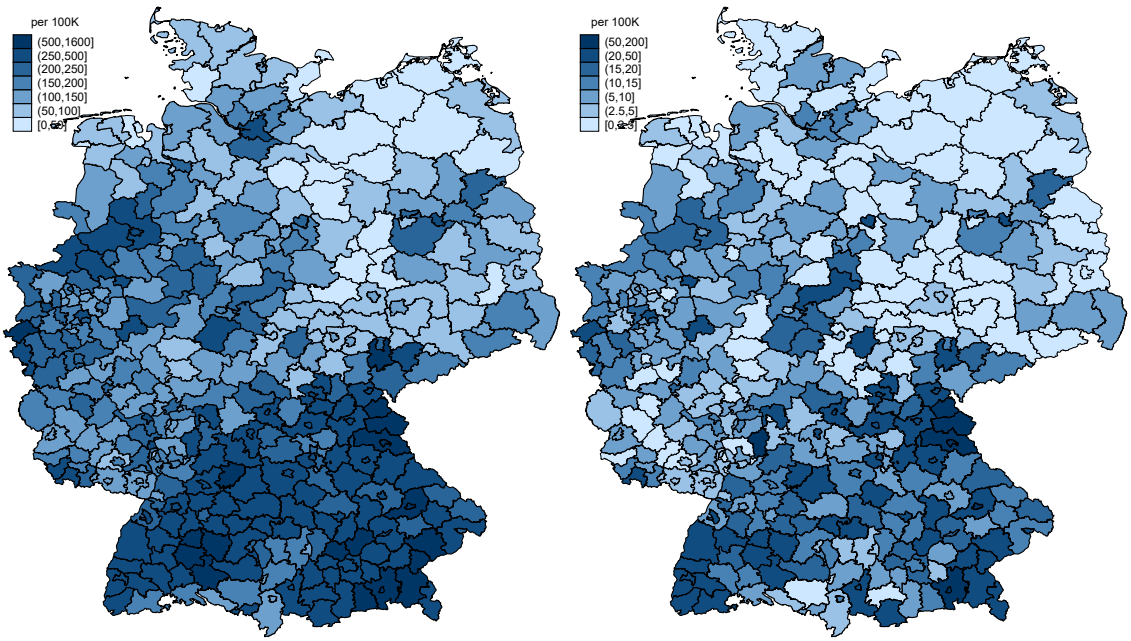
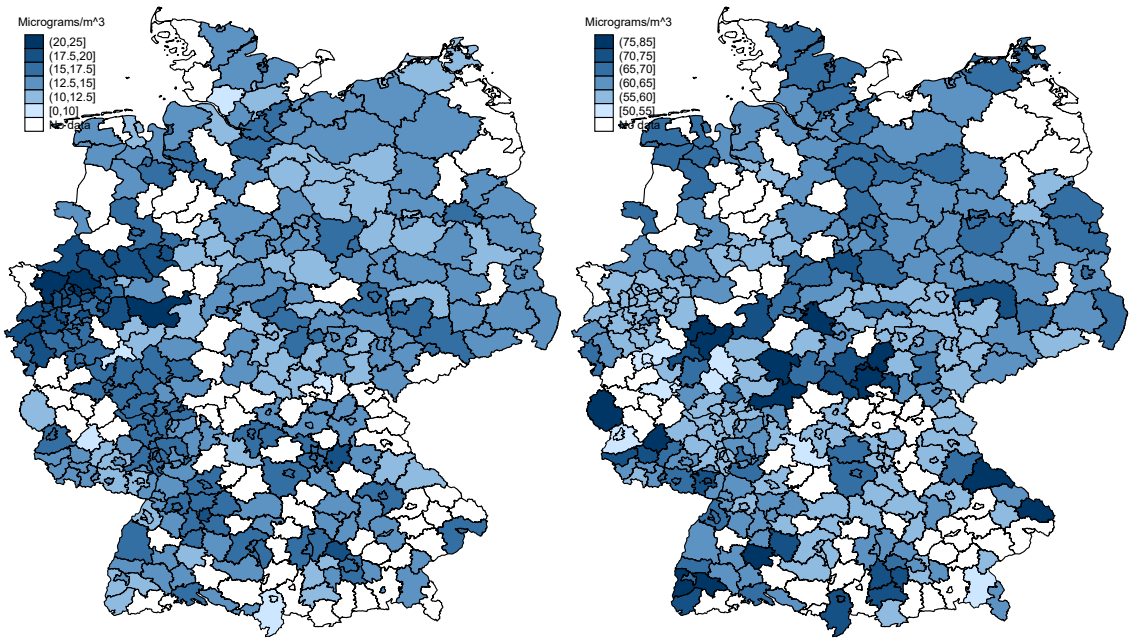
A Appendix



(a) Cases per 100K (b) Deaths per 100K

Figure A.1: Total confirmed cases and deaths of COVID-19 by county

Note: These maps show the total number of confirmed of COVID-19 cases and deaths per 100K population across counties (*Kreise*) as of May 26, 2020. *Source*: RKI.



(a) Particulate Matter (PM10) (b) Ozone

Figure A.2: Mean concentration of air pollutants by county

Note: These maps show the mean concentration of particulate matter (PM10) and Ozone across counties (*Kreise*) over the period 1 Feb to 26 May 2020. *Source*: UBA.

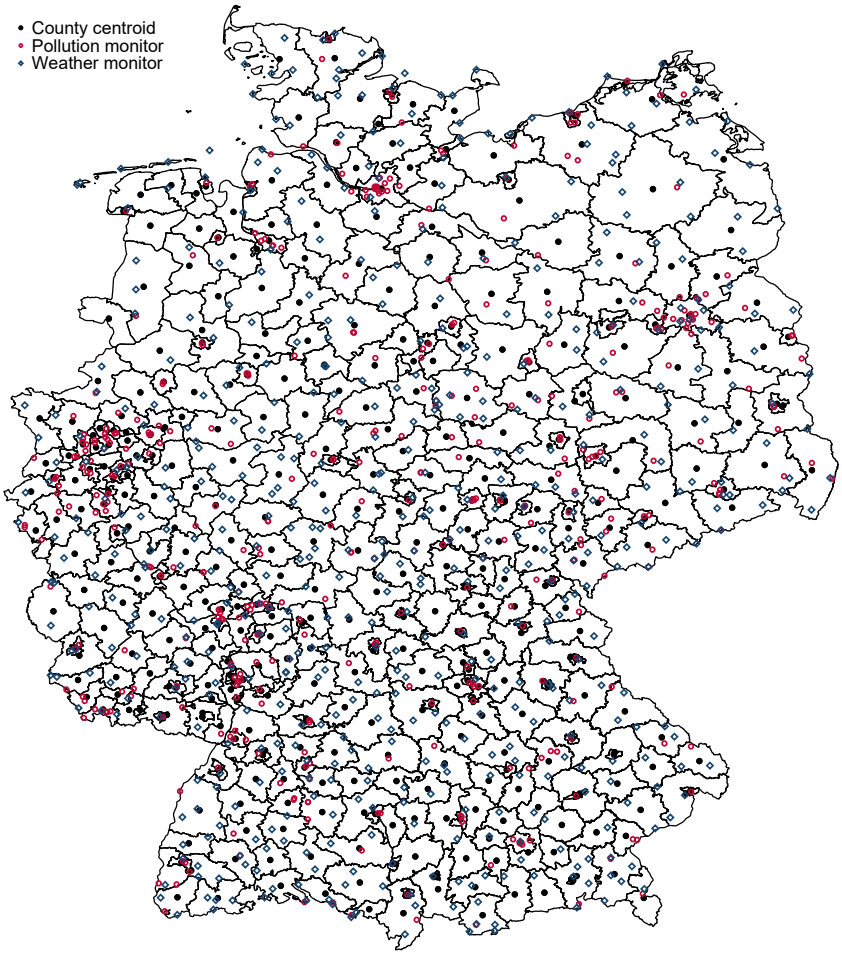


Figure A.3: Air pollution and weather monitor locations

Note: This map shows the locations of air pollution and weather monitors across Germany as well as the geographic centroids of counties (*Kreise*). *Source*: UBA and DWD.

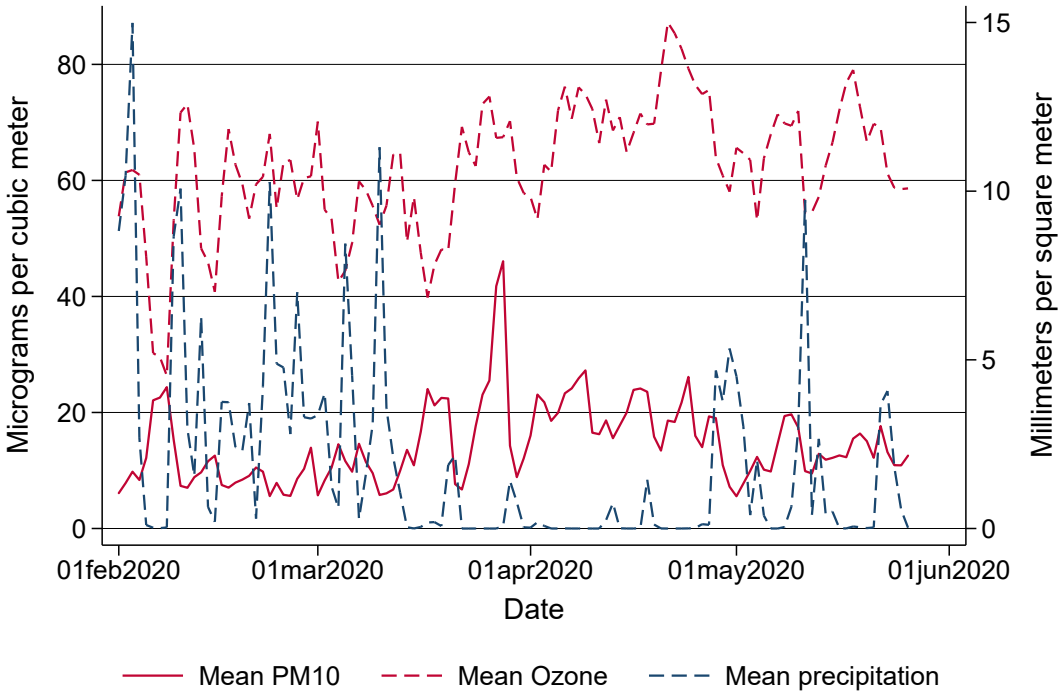


Figure A.4: Air pollution and precipitation

Note: This figure shows daily mean levels of precipitation, ozone and PM10 over the period of analysis. *Source*: UBA and DWD.

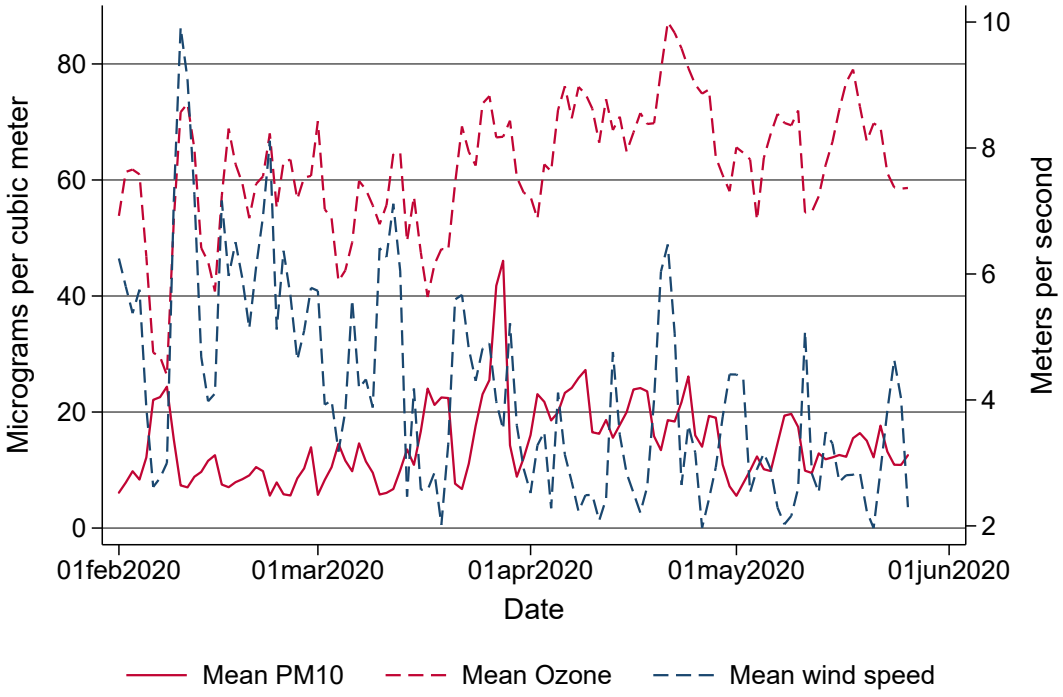


Figure A.5: Air pollution and windspeed

Note: This figure shows daily mean wind speed, ozone and PM10 over the period of analysis. *Source*: UBA and DWD.

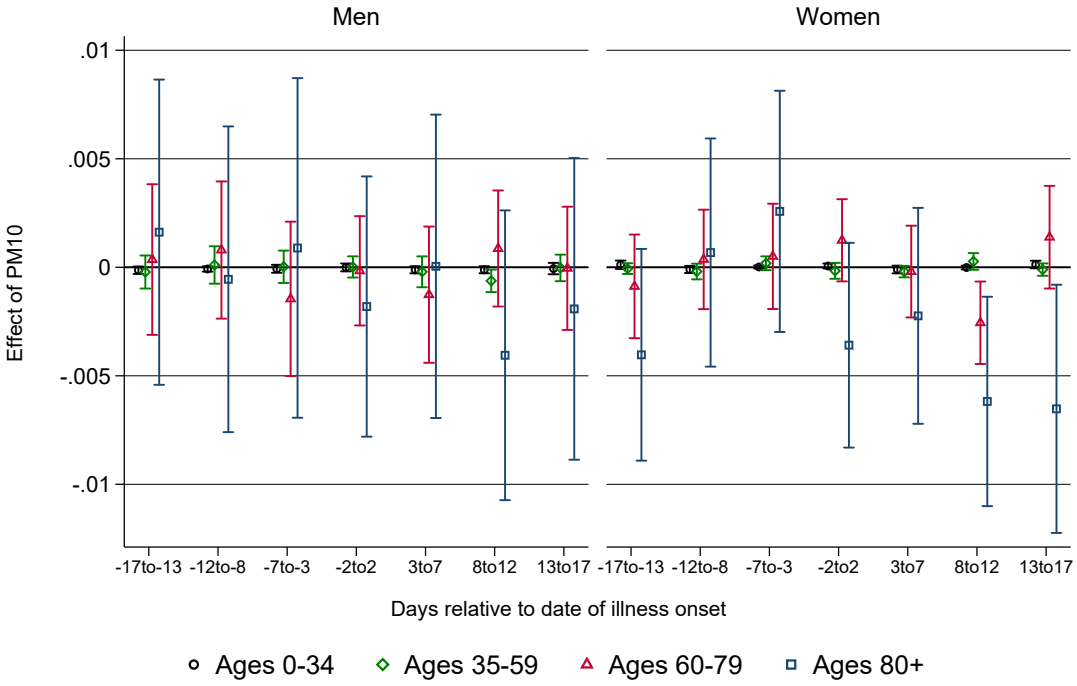


Figure A.6: Effect of PM10 on case fatality rate of COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of PM10 concentration on the case fatality rate of COVID-19. *Source*: RKI, UBA and DWD.

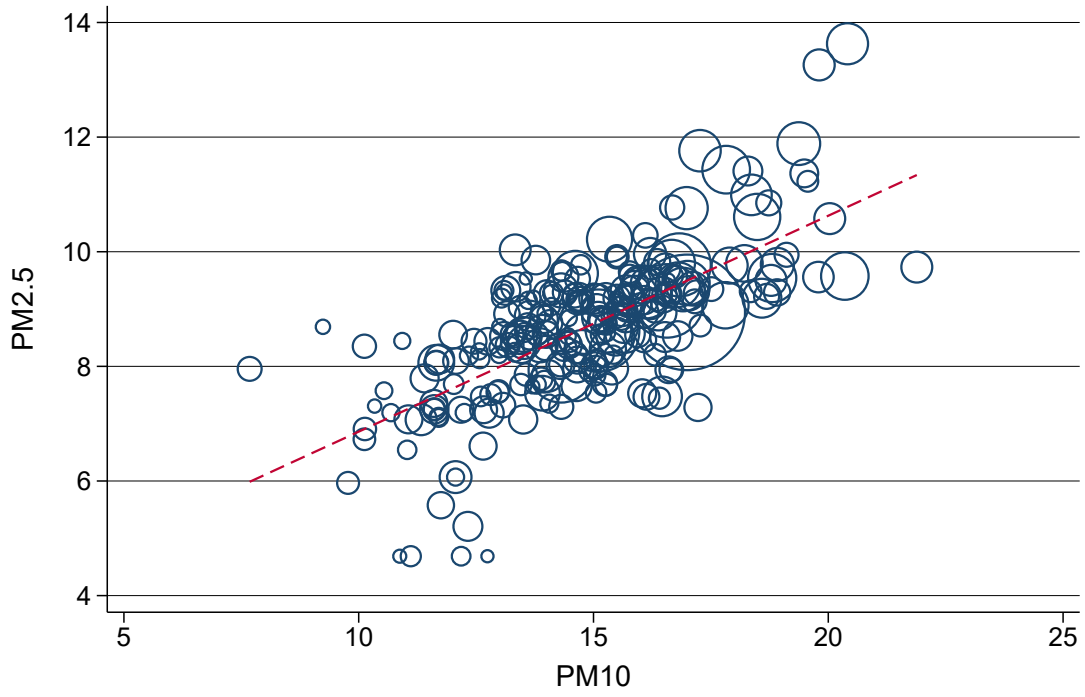


Figure A.7: Correlation between PM2.5 and PM10

Note: This graph visualizes the relationship between measures of PM2.5 and PM10 weighted by county population size.
Source: UBA.

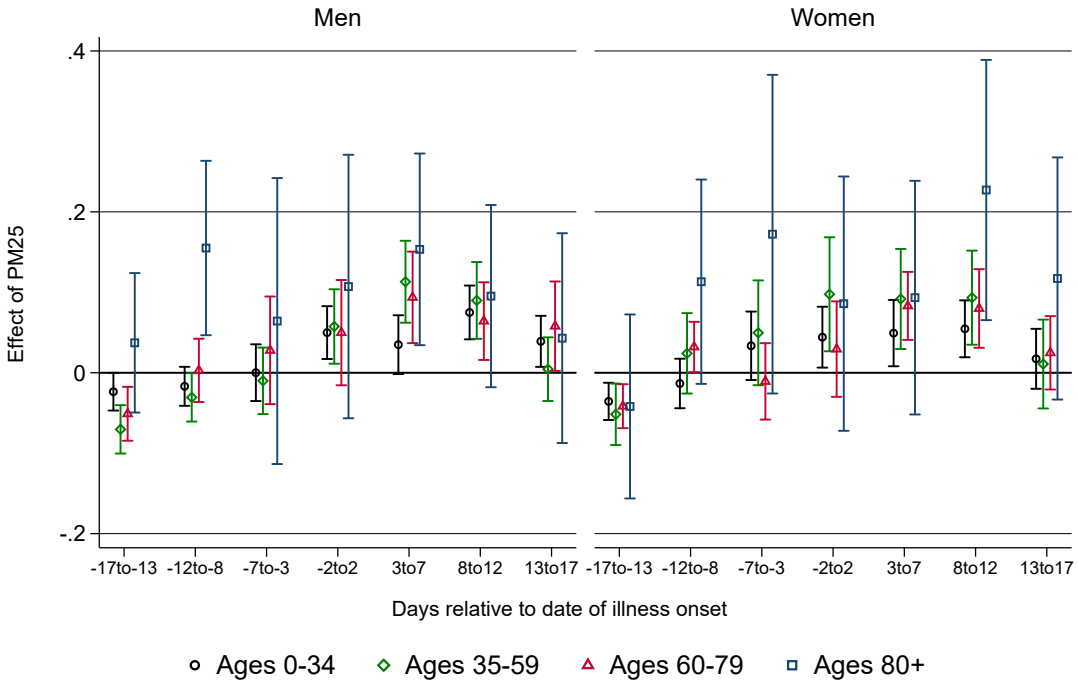


Figure A.8: Effect of PM2.5 on new confirmed cases of COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of PM2.5 concentration on the number of new confirmed cases of COVID-19. *Source*: RKI, UBA and DWD.

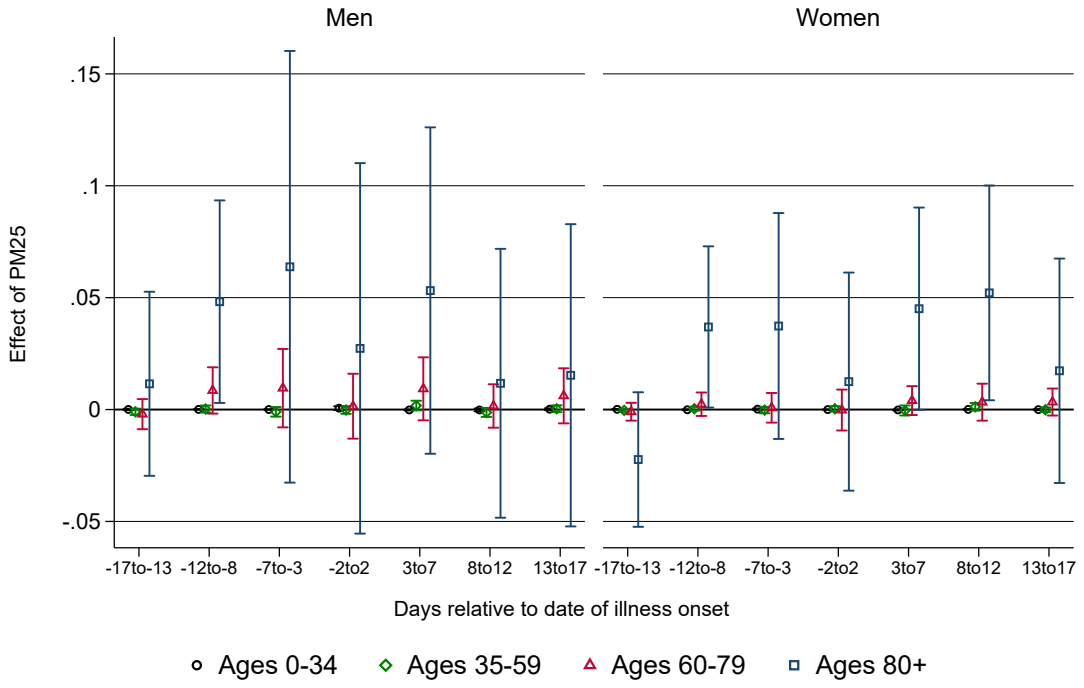


Figure A.9: Effect of PM2.5 on new deaths from COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of PM2.5 concentration on the number of new deaths from COVID-19. *Source*: RKI, UBA and DWD.

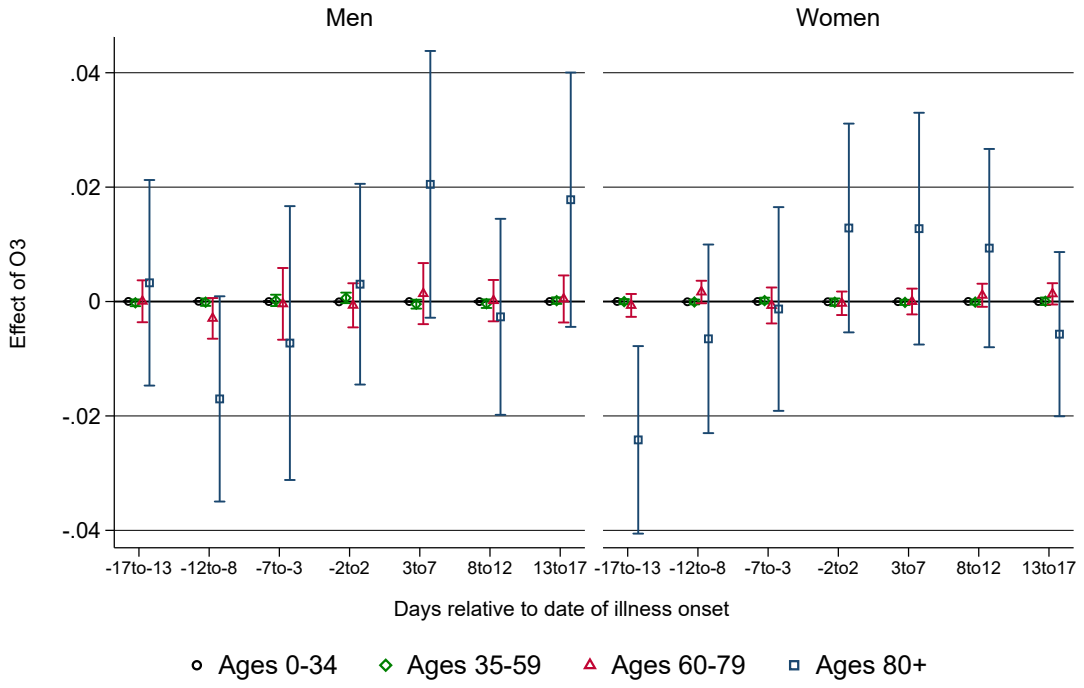


Figure A.10: Effect of Ozone on new deaths from COVID-19

Note:This graph shows the point estimates and the 95 percent confidence intervals of the effect of ozone concentration on the number of new deaths from COVID-19. *Source*: RKI, UBA and DWD.

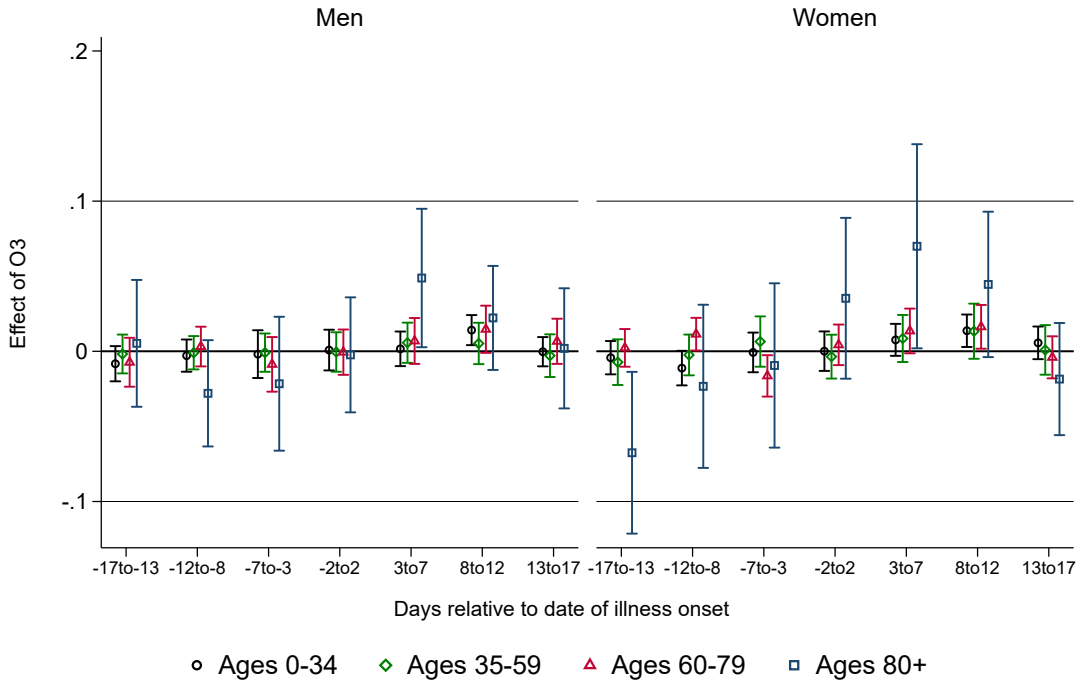


Figure A.11: Effect of Ozone on new confirmed cases of COVID-19

Note: This graph shows the point estimates and the 95 percent confidence intervals of the effect of ozone concentration on the number of new confirmed cases of COVID-19. *Source*: RKI, UBA and DWD.