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ABSTRACT

Background: Daily exposure to air pollution has been shown to increase cardiovascular and respiratory mortality. While increases in short-term exposure to air pollutants at any daily concentrations has been shown to be associated to adverse health outcomes, days with extreme levels, also known as air pollution peaks based on specific thresholds, have been used to implement air quality alerts in various cities across the globe.
Objectives: We aimed at evaluating the potential effects of the Air Quality Alerts (AQA) system on different causes of premature mortality in Paris, France.
Methods: Air quality alerts (AQA) based on particulate matter (PM_{10}) levels and related interventions were implemented in the region of Paris in 2008 and were revised to be more stringent in 2011. In this study, we applied a difference-in-differences (DID) approach coupled with propensity-score matching (PSM) to daily mortality data for the period 2000 to 2015 to evaluate the effects of the Paris AQA program on different causes of premature mortality for the entire population and for adults > 75 years old.
Results: Overall, results did not show evidence of a reduction in mortality of the PM_{10} AQA program when first implemented in 2008 with initial thresholds (80 μg/m^3); DID estimates were slightly above 1 for cardiovascular and respiratory mortality. However, when evaluating the drastic reduction in revised thresholds in 2011 (50 μg/m^3) to trigger interventions, we identified a reduction in cardiovascular (DID = 0.84, 95% CI: 0.755 to 0.930) mortality, but no change in respiratory mortality was detected (DID = 0.97, 95% CI: 0.796, 1.191).
Discussion: Our study suggests that AQA may not have health benefits for the population when thresholds are set at high daily PM_{10} levels. Given that such policies are implemented in many other metropolitan areas across the globe, evaluating the effectiveness of AQA is important to provide public authorities and researchers a rationale for defining specific thresholds and extending the scope of these policies to lower air pollution levels.

1. Introduction

In the last two decades, there has been growing evidence of adverse health outcomes being associated with exposure to air pollution which has led to the implementation of air quality standards in various countries. Chronic exposure to air pollution has been shown to induce systemic inflammation, telomere erosion and oxidative stress leading to chronic respiratory and cardiovascular diseases and mortality (Franchini and Mannucci, 2009; Sesé et al., 2018b, 2018a). Moreover, acute short-term exposure to different pollutants, including both gaseous pollutants (i.e., nitrogen dioxide [NO_2], sulfur dioxide [SO_2] and ozone [O_3]) and particulate matter (PM_{10} and $PM_{2.5}$), may exacerbate existing health conditions, leading to asthma exacerbations, chronic obstructive pulmonary diseases or myocardial infarction, which may cause

premature death (Franchini and Mannucci, 2009; Pope, 2007). While it has been shown that increases at any level of daily exposure to air pollutants are associated with adverse health outcomes including cardiovascular or respiratory mortality (Daniels et al., 2000; Dominici et al., 2002; Papadogeorgou et al., 2019; Schwartz et al., 2002, 2001; Schwartz and Zanobetti, 2000; Yan et al., 2019), an emphasis has been made on days with extreme levels of air pollutants (above specific threshold values), also known as air pollution peaks.

In order to prevent adverse health events associated with air pollution peaks, air quality alerts (AQA) have been implemented in various metropolitan cities worldwide. Such AQA typically involve a large range of measures focused primarily on reducing emissions on days where air pollution concentrations exceed a given threshold. In line with this policies, the World Health Organization (WHO) provides air quality guidelines (World Health Organization (WHO), 2005) for national and local authorities, defining a set of thresholds for common air pollutants (including fine and coarse particles, NO_2 and Ozone).

Despite their widespread deployment in many big cities worldwide (e.g. Los Angeles, Santiago, Beijing, Toronto, Sao Paulo, Hong Kong), evidence about the impacts of such policies on population health is still limited (Chen et al., 2018; Lin and Wei Umanskaya, 2011; Mason et al., 2019, 2020a; Mullins and Bharadwaj, 2015; Neidell, 2009). In an analysis on the impact of driving restriction policies in four different cities (São Paulo, Bogotá, Beijing, and Tianjin), Lin and Wei Umanskaya (2011) concluded that, even if this type of measures could potentially reduce the extreme concentrations of air pollutants, no evidence of an effect on air quality improvement was observed (Lin and Wei Umanskaya, 2011). In contrast, a study evaluating impacts of the AQA program in Santiago, Chile found that the implementation of the program was effective for reducing air pollution and mortality in the short term (Mullins and Bharadwaj, 2015). A recent study assessing the potential effects of the Toronto AQA program found limited benefits on various morbidity and mortality indicators (Chen et al., 2018). Two recent studies in Hong Kong concluded that the AQA program may benefit specific vulnerable population (like children and elderly), but that it did not affect the number of respiratory and cardiovascular hospitalizations in the overall population (Mason et al., 2020b, 2019). Given the heterogeneity in findings, it is important to document the evidence of the effectiveness of such policies in different contexts. Yet, to the best of our knowledge, no study has evaluated the potential health benefits of AQA

systems in a large European city such as Paris, France. Such evidence is critical given the inconsistent findings in other locations and the important differences regarding air pollutions levels, emission sources, and potential actions to mitigate air pollution among and between European cities and cities in North America or Asia.

While PM_{10} emissions decreased in the region of Paris in recent years (Site officiel de la Ville de Paris, n.d.), Paris still has the highest PM_{10} concentrations among cities in the European Union (EU) (Kodukula et al., 2018) and 60,000 of its inhabitants are regularly exposed to PM_{10} concentrations above the EU regulatory thresholds (Site officiel de la Ville de Paris, n.d.). AQA based on PM_{10} levels have been implemented in the region of Paris through a prefectural order in December 2007 (arrêté inter-préfectoral n° 2007–21277). Two distinct thresholds based on average PM_{10} daily concentrations have been defined for informational and warning AQA respectively (see details in Fig. 1). When informational AQA are triggered, measures are implemented including public health messages targeting vulnerable populations, traffic and traffic-speed restrictions and reduced fees for public transportation. When warning AQAs are triggered, these measures are reinforced, with public health messages targeting the entire population, more severe driving restrictions, and limitations of some industrial emitters. The initial threshold values for PM_{10} concentration defined in December 2007 was 80 μg/m³ over a period of 24 h for the "informational AQA" and 120 μg/m³ per 24 h for the "warning AQA". These thresholds were then revised in November 2011 (arrêté inter-préfectoral n° 2011–00832) to 50 μg/m³ per 24 h and 80 μg/m³ per 24 h, respectively.

In this study we aimed to evaluate the potential effects of the AQA system on different causes of premature mortality for the entire population and for the most exposed age group (adults > 75 years old) in Paris based on changes in PM_{10} levels. We used a quasi-experimental study design with a difference-in-differences (DID) approach coupled with propensity-score matching (PSM) to quantify the potential effects of the Paris AQA program for PM_{10} on premature mortality.

2. Methods

2.1. Data

Daily mortality data for the region of Paris (corresponding to deaths of people with last known residence in the city of Paris and the crown of
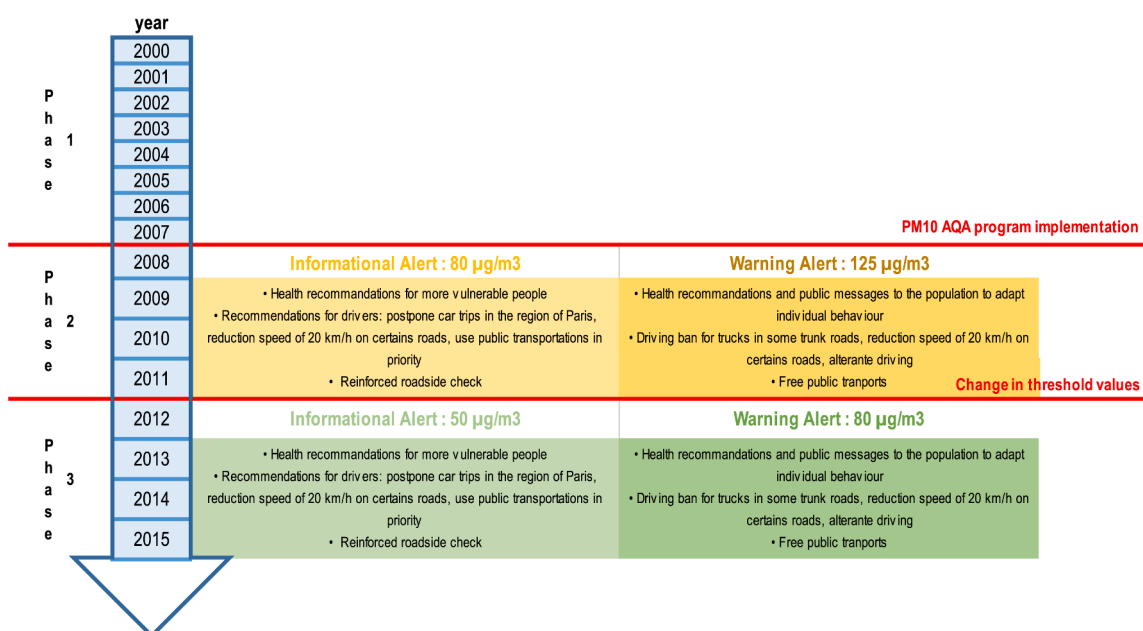

Fig. 1. The PM_{10} Air Quality Alerts (AQA) program implementation steps and measures.

3 counties around Paris) for the period from 2000 to 2015 were obtained from the French National Institute of Health and Medical Research (CepiDC). Daily mortality in the whole population and among people older than 75 years was analyzed for the following causes: all non-accidental mortality (ICD10: A00-R99), cardiovascular causes (ICD10: I00-I99), and respiratory causes (ICD10: J00-J99).

Air quality data were collected from the local air quality monitoring network, managed by the regional Air Quality Surveillance Association AirParif (https://www.airparif.asso.fr/), which is the entity in charge of the air pollution data collection and forecast and the one who relays information to the local public authorities and media as soon as an acute air pollution peak is predicted. Daily PM_{10} concentrations were available for the period 2000–2015. Measures of daily PM_{10} exposure for the study area were provided by the network of urban background and *peri-*urban monitors. Complete information about days being predicted to experience an acute PM_{10} pollution peak during the intervention period (2008–2015) was publicly available on the website of AirParif (https://www.airparif.asso.fr/). Daily hourly minimal and maximal temperatures were provided by the French national meteorological service Météo-France for one reference station for the city of Paris (*Montsouris* station).

2.2. Study design and statistical analysis

For the main analysis, three periods were defined according to the PM_{10} AQA-program implementation dates: the pre-intervention period (without any intervention), to which we refer here as Phase 1 (from the 1st of January 2000 to the 31st of December 2007), the first intervention period with the initial threshold values in force, Phase 2 (from the 1th of January 2008 to the 29th of November 2011) and the last intervention period characterized by revised threshold values, Phase 3 (from the 30th of November 2011 to the 31st of December 2015) (see details in Fig. 1).

For each period, eligible days for triggering the AQA measures (i.e. days experiencing an air pollution peak) were identified. Following the approach of AIRPARIF, days that experienced an air pollution peak during Phase 1 (when the AQA program had not been implemented yet so days with air pollution peaks had not been identified by AIRPARIF) were determined by the authors when at least two monitors, one which had to be an urban background monitor, recorded a daily average concentration for PM_{10} exceeding the initial thresholds (i.e. average daily concentration $PM_{10} \geq 80$ μg/m^3). During the two intervention periods (Phase 2 and Phase 3), days with AQA were defined from the aforementioned list provided by AIRPARIF. As "warning alert" peaks were very rare over the study period (i.e., 19 events), we grouped informational and warning alerts together.

A DID approach, coupled with PSM, was used in order to evaluate the effectiveness of both the PM_{10}-AQA program implementation and the change in threshold value in preventing mortality in the region of Paris. We capitalized on the timing of the implementation of AQA and specific threshold values to compare Phase 2 with Phase 1 and then compare Phase 3 with Phase 2.

First, the difference in daily mortality between eligible days (treated group) and non-eligible days (control group) before the PM_{10} AQA program was compared to the difference between AQA days (treated group) and non-AQA days (control group) during the first period after the program implementation (Phase 1 versus Phase 2). In this case, the counterfactual quantity of interest is the difference in daily mortality between AQA and non-AQA days that would have been observed during the Phase 2 if PM_{10} AQA program had not been implemented and that is expected to be equal to the difference observed during the Phase 1. Then, the difference in daily mortality between AQA days and non-AQA days during the first phase of the PM_{10} AQA program was compared to the difference observed during the last period with revised threshold values (Phase 2 versus Phase 3). Here, the counterfactual quantity of interest is the difference in daily mortality between AQA and non-AQA days that would have observed during Period 3 if the threshold values

for the PM_{10} AQA program had not been revised.

Key assumptions of DID analysis are that: i) there is no other policy potentially influencing mortality and air pollution levels above defined thresholds implemented at the same time and; ii) before the intervention, control and treated groups present similar trends in the outcome, which means that the trend in the control group represents a good approximation for the counterfactual trend of the treated group in the absence of the treatment (Abadie and Cattaneo, 2018). In order to control for possible time-varying confounders and make eligible and non-eligible days as comparable as possible, a PSM approach was used to create a panel of matched eligible and non-eligible days to be used in the DID model estimation (Stuart et al., 2014). By optimizing covariate balance between eligible and non-eligible days for a set of a priori identified observed confounders (i.e. temperature, day of the week, months, year, O_3 and PM_{10} concentrations of the previous days), PSM helps make eligible and non-eligible days as similar as possible regarding measured covariates.

All analysis were conducted with *R* and *RStudio* software (R version 3.6.1). Syntax to replicate them are available at this page: https://github.com/aalari/AQAproject.

2.2.1. Propensity score matching

The Propensity Score (PS) is defined as the probability for each observed day to be eligible for the AQA program. A logistic regression was used to estimate the PS as a function of a set of covariates that are likely to impact on the occurrence of an acute PM_{10} pollution peak (Pollution Peak [PP = 1]). Different PS model specifications were tested with different combinations of independent variables and different types of spline functions s() and degrees of freedom (df) for the continuous variables. The model with the lowest AIC value was used, which included the following covariates: day of week (DOW), bank holidays (BH), month and year, smooth functions of the PM_{10} and Ozone (O_3) concentrations levels of the 3 previous days (with 6 df), smooth functions of the mean and the maximum temperature of the day (with 7 df).

$$\text{logit} P[PP=1|X] = \beta_0 + \beta_1 DOW + \beta_2 BH + \beta_3 month + \beta_4 year + s_5(tempmax, 7)$$

$$+ s_6(meantemp, 7) + \sum_{n=1}^{3} s_{7n}(PM10_{lag_n}, 6) + \sum_{n=1}^{3} s_{8n}(O3_{lag_n}, 6) \quad (1)$$

Eligible days were matched with similar non-eligible days having the closest PS value. The matching procedure was optimized by choosing the matching ratio, caliper (the maximal distance in PS values, expressed in standardized units, which is acceptable to match an eligible day with a non-eligible day) and methods for handling ties (deterministically or not, with or without replacement) which provided the lowest standardized mean difference across covariates after matching. The selected matching strategy used a ratio of 1:3, with replacement and no caliper. Details about the matching procedure optimization are provided in the supplementary material.

2.2.2. Difference-in-Differences (DID) model

To obtain the difference in daily mortality during pollution peak days (eligible days) that can be attributable to the implementation of the PM_{10} AQA program and to the change in threshold values, the panel of matched observations was analyzed through two distinct fixed-effect Poisson models using a DID approach comparing Phase 2 to Phase 1 and Phase 3 to Phase 2, respectively. The DID estimator (expressed as a Relative Risk, RR) is the interaction term between the estimated effect of eligible (polluted) versus non-eligible (non-polluted) days and the estimated effect of the pre-intervention versus post-intervention time period in the following model:

$$y_{i,t} \sim P(\lambda_{t,i})$$
$$\log(\lambda_{t,i}) = \beta_1 E_{t,i} + \beta_2 P_{t,i} + \beta_3 E_{t,i} P_{t,i} + \varepsilon_{t,i} \quad (2)$$

In this model, the daily mortality *y* for every day *t* in the matched cluster *i* follows a Poisson distribution having the parameter λ as mean

and variance. *E* represents a binary variable indicating whether the days were eligible or not, whereas *P* indicates whether the days were before or after the intervention (the PM_{10} AQA policy implementation or the change in threshold values). The DID term is the interaction term between these two variables (*EP*) and the corresponding coefficient β_3 provides the estimated change in mortality attributable to the PM_{10} regulatory policy implementation (in the case of the P2 vs. P1 model) or to the change in threshold values (in the P3 vs. P2 model). The same model was applied to estimate the effect of the policy on non-accidental all-cause mortality, cardiovascular mortality and respiratory mortality for the entire population and for adults older than 75. In order to take into account possible lagged effects of the exposure, we also estimated the impact of AQA measures on mortality observed the day following the air pollution episode (day + 1) and two days after (day + 2).

2.2.3. Number of deaths prevented attributable to the AQA program

When an effect on mortality was detected, the estimated RR corresponding to the DID term was used to calculate the attributable fraction of daily prevented deaths (AF) as follows:

$$AF = (\frac{1}{RR} - 1)/\frac{1}{RR} \tag{3}$$

The number of deaths prevented (PD) attributable to the AQA program was then calculated using the following equation:

$$PD = AF \times \overline{MDD} \times ND \tag{4}$$

where $\overline{MDD}$ is the mean observed daily death during the intervention period and *ND* is the number of pollution peaks that occurred during the same period. In order to obtain 95% Confidence Intervals (CIs) for the number of prevented deaths, the bootstrap procedure was used to replicate the analysis on 500 bootstrap samples.

2.3. Sensitivity analyses

As a complementary analysis aiming to provide an estimation of the effect of the AQA program in its entirety, the difference in daily mortality between eligible days and non-eligible days before the PM_{10} AQA program (Phase 1) was compared to the difference between AQA days and non-AQA days during the whole intervention period (Phase 2bis, which correspond to the Phase 2 and Phase 3 together). In order to check the contribution on mortality of the warning AQA days (more rare but corresponding to more extreme exposure conditions), we excluded the warning AQA days and ran the same DID models including informational AQA only.

Furthermore, to check the sensitivity of our results to a possible presence of overdispersion in our data, we used Negative Binomial models to replicate our analysis for the effect on the overall population mortality and we compared these results with the ones obtained with Poisson models. Finally, we also used a linear model with mortality rates per 100,000 inhabitants (obtained by dividing numbers of daily deaths by the local population) as our dependent variable of interest.

3. Results

From the PM_{10} regulatory policy implementation in 2008 until the end of 2015, 122 PM_{10} pollution peaks were recorded in the AirParif database. According to the analysis of the average level of daily PM_{10} concentrations in all urban background monitors and to our eligibility criteria (as used by AIRPARIF), 15 days were defined as eligible to AQA measures during the pre-intervention Phase (2000–2007). Since the AQA program implementation, 103 Informational AQA days and 19 Warning AQA days were recorded.

A total number of 622,268 non-accidental deaths were observed during the whole study period in the general population, whose 386,202 were among adults > 75 years old (2000–2015). In the entire

population, 154,894 deaths were from cardiovascular causes and 39,899 from respiratory causes, while 119,464 cardiovascular deaths and 31,653 respiratory deaths were recorded for the elderly. This represented an average of 106.5 non-accidental deaths per day in the whole population (66.1 among the elderly), and a daily number of 26.5 (20.4 for the elderly) and 6.8 (5.4 for the elderly) deaths for cardiovascular and respiratory causes, respectively (see more details in **Table A1**). Descriptive statistics for all days and for each period are presented in **Table A1** for both non-eligible days (n = 5707) and eligible days (n = 122).

The PSM procedure provided a new set of 548 matched observations, composed by 137 pairs, where each eligible day matched with 3 non-eligible days. The analysis of the standardized mean differences in the set of unmatched observations highlighted a remarkable imbalance on all covariates between the two groups before matching. The final matching algorithm allowed to reduce the standardized mean difference and considerably improved covariate balance on all covariates in the final set of matched observations (**Table A2** and **Figure A1**).

When comparing eligible days and non-eligible days between periods and in only matched days (Table 1), we observed that during the pre-intervention period the average daily non-accidental and cause-specific mortality for non-eligible days was higher than for eligible days, in the overall population as well as among people older than 75 years. This changed during Phase 2, when the average daily mortality for AQA days become slightly higher than for similar matched non-AQA days. Finally, Phase 3 was characterized by a remarkable reduction in average cardiovascular mortality for the overall population and among elderly people during AQA days; average numbers of daily cardiovascular mortality in both groups were smaller for AQA days than for matched non-eligible days. Even if respiratory mortality for AQA days didn't decrease during this Phase compared to the Phase 2, its average number for AQA days was still slightly smaller than for matched non-AQA days. Non-accidental mortality was also more important during non-AQA days, but only for people older than 75 years old.

In order to test DID assumption on whether control and treated groups present similar trends in the outcome, the average number of deaths before the policy implementation were plotted for both eligible and matched non-eligible days. Very similar trends were observed for cardiovascular and respiratory deaths but not for non-accidental deaths (**Figure A2**). For this reason, our main analysis finally focused on cardiovascular and respiratory mortality and results concerning the effect of the PM_{10} AQA program on non-accidental mortality are only presented in Supplementary Materials.

We did not find evidence of an effect of the PM_{10} AQA program on overall-population mortality (Table 2) when first implemented in 2008 with initial thresholds. The DID estimators for the effect on cardiovascular and respiratory mortality were 1.10 (95% CI: 0.94 to 1.29) and 0.94 (95% CI: 0.69 to 1.30), respectively (see Table 2). Similar results were found for elderly when comparing Phase 2 to Phase 1 (see Table 3).

When assessing the impact of changes in PM_{10} thresholds in 2011 (Phase 3 vs. Phase 2) in the overall population, we identified a reduction in cardiovascular mortality (DID = 0.84, 95% CI: 0.75 to 0.93), which corresponds to 386 (95% CI: 35 to 729) prevented cases. A weaker and more imprecise effect was observed on respiratory mortality. A decrease in cardiovascular (DID = 0.81, 95% CI: 0.72 to 0.91) mortality was also detected among adults older than 75 years, which prevented 348 (95% CI: 79 to 617) cardiovascular deaths in this age group during Phase 3 (Table 2 **and Table A4**).

The analysis of the effect of the PM_{10} AQA program on mortality at day + 1 and day + 2 didn't detect a change in mortality during Phase 2 when compared to Phase 1 (**Tables from A5 to A8**). A decrease in non-accidental mortality at day + 1 was estimated for Phase 3 compared to Phase 2 in the overall population, as well as among elderly people, but no change was found for cardiovascular and respiratory mortality (**Tables A5 and A6**). No effect of the PM_{10} AQA program on mortality at day + 2 was detected during Phase 3 compared to Phase 2, in the overall

Table 1
Mean (and Standard Deviation) of mortality and environmental variables in the 3 Phases studied (pre-intervention Phase, first intervention Phase and last intervention Phase) according to the air pollution status (Non-eligible days, Eligible days). Only matched days (n = 548) were considered.

	Before PM$_{10}$ Regulation (P1)		First PM$_{10}$ Regulation (P2)		Last PM$_{10}$ Regulation (P3)	
	Non-Eligible Days (n = 41)	Eligible Days (n = 15)	Non-AQA Days (n = 59)	AQA Days (n = 23)	Non-AQA Days (n = 311)	AQA Days (n = 99)
Entire Population						
Non-acc Deaths	121.15	116.67	112.81	114.78	112.73	113.38
Cardio Deaths	33.78	31.47	27.64	30.04	26.86	24.94
Respi Deaths	7.95	7.93	6.68	7.61	8.75	8.13
≥ 75 years old						
Non-acc Deaths	78.20	74.27	74.39	75.00	74.48	73.94
Cardio Deaths	26.07	25.33	22.49	24.74	21.21	19.85
Respi Deaths	6.20	6.00	4.92	6.35	7.45	6.85
Temperatures						
Minimum	2.13	1.77	3.94	2.23	3.42	3.35
Maximum	10.06	9.59	12.98	8.86	10.58	11.33
Mean	5.92	5.25	8.34	5.54	7.00	7.34

P1: Phase 1 (pre-intervention period), from the 1st of January 2000 to the 31st of December 2007.
P2: Phase 2 (first intervention period), from the 1th of January 2008 to the 29th of November 2011.
P3: Phase 3 (last intervention period), from the 30th of November 2011 to the 31st of December 2015.
AQA: Air Quality Alert

population nor among people older than 75 years (**Tables A7 and A8**).

In the sensitivity analyses, when we considered only two periods (grouping Phase 2 and Phase 3 together), we did not find evidence of an effect of the PM$_{10}$ AQA program on mortality (**Table A9** and **Table A10**). When considering only Informational AQA (and thus excluding warning AQA) for both period comparisons, we found similar conclusions for cardiovascular mortality (**Table A11** and **Table A12**). However, when removing the warning AQA days, we did not detect any more decreases (even imprecise) in respiratory mortality associated with the change in thresholds that took place in Phase 3, in the overall population or among elderly. This suggests that, while the number of warning AQA events was relatively rare, such interventions seem to be an important contributor to benefits in respiratory mortality.

Both linear and negative binomial models confirmed results obtained by Poisson regression models concerning cardiovascular mortality (**Table A13** and **Table A14**). Estimations about the effect of the change in threshold values on respiratory mortality were still characterized by a certain degree of uncertainty, with imprecise DID estimates indicating a reduction in the difference of respiratory mortality between AQA days and non-AQA days during Phase 3 compared to Phase 2 in the Poisson and Linear models estimations, but a positive and very imprecise DID estimator in negative binomial model estimations.

4. Discussion

This paper evaluated the effectiveness of the AQA program for PM$_{10}$ on mortality in the region of Paris. We found that while the initial version of the AQA in 2008 did not seem to reduce mortality during PM$_{10}$ peak days, the decrease in thresholds to trigger AQA occurred in 2011 led to a decrease in cardiovascular mortality for the overall population and among people older than 75 years. Given that such policies are implemented in many metropolitan areas across the globe, these findings are important to understand the potential impact of AQA in other locations.

The AQA system implemented in the region of Paris includes a range of actions. For the first "informational alert" level, a public announcement relayed by local press and media is addressed to the population with a number of recommendations for adapting personal behavior. These public messages strongly suggest people to limit practices that contribute to emissions (heating, the use of certain types of engines, biomass combustion), they target some identified industrial facilities, and they provide health recommendations for avoiding behaviors that can increase the exposure to air pollution (typically, outdoor activities). Evaluation of the effectiveness of these types of measures encouraging voluntary actions has been contrasted in the literature. In his analysis of the impact of air quality warning system in the Los Angeles area, Neidell (2009) found that people were responsive to air quality alerts by decreasing their participation in outdoor activities (Neidell, 2009). On the contrary, a survey conducted on a random sample of individuals from Portland and Houston in 2005 and 2006 revealed that air quality alerts had limited impact on population awareness (Semenza et al., 2008).

Other type of measures based on voluntary implication, like driving self-restrictions, have been shown to have limited effectiveness. When traffic restrictions rely on voluntary reductions in vehicle usage, they not only appear to be ineffective (Noonan, 2014), but they may also exacerbate average daily traffic levels by persuading residents to leave the polluted city to go to the countryside, as pointed out by Tribby et al. (2013) in Salt Lake City (Tribby et al., 2013). Based on these results, we might conclude that population lack of compliance for voluntary actions are a major obstacle in implementing effective air quality alert polices. However, a similar lack of effect of traffic reductions policies was also observed in cities where driving restrictions were based on coercion. In Mexico City, vehicles are banned one week-day per week on the basis of the last digit of the license plate and violators are fined and will have their vehicle impounded. However, no evidence of an air quality

Table 2
Results of the Difference-in-Differences (DID) Poisson models. DID estimates provide the effect of the PM_{10} Air Quality Alert program implementation (P2 vs. P1 models) and of the change in threshold values (P3 vs. P2 models) on health outcomes in the entire population in the region of Paris, France.

Period	Outcome	Estimate*	Value	95% IC			
2 vs. 1	Cardiovascular Deaths	Period	0.81	[	0.670	,	0.988	]
		Eligibility	1.04	[	0.912	,	1.178	]
		DID	1.10	[	0.938	,	1.292	]
	Respiratory Deaths	Period	1.00	[	0.690	,	1.455	]
		Eligibility	1.03	[	0.799	,	1.325	]
		DID	0.94	[	0.687	,	1.300	]
3 vs. 2	Cardiovascular Deaths	Period	1.05	[	0.874	,	1.257	]
		Eligibility	1.14	[	1.035	,	1.249	]
		DID	0.84	[	0.755	,	0.930	]
	Respiratory Deaths	Period	1.12	[	0.815	,	1.570	]
		Eligibility	0.96	[	0.802	,	1.160	]
		DID	0.97	[	0.796	,	1.191	]

* Period (P) represents the average difference in the outcome of interest between the two periods (for all types of days eligible or not); Eligibility (E) represents the average difference in the outcome of interest between eligible and non-eligible days for the entire study period.

Table 3
Results of the Difference-in-Differences (DID) Poisson models. DID estimates provide the effect of the PM_{10} Air Quality Alert program implementation (P2 vs. P1 models) and of the change in threshold values (P3 vs. P2 models) on health outcomes in adults older than 75 years in the region of Paris, France.

Period	Outcome	Estimate	Value	95% IC			
2 vs. 1	Cardiovascular Deaths	Period	0.84	[	0.675	,	1.045	]
		Eligibility	1.07	[	0.931	,	1.240	]
		DID	1.10	[	0.918	,	1.312	]
	Respiratory Deaths	Period	0.89	[	0.591	,	1.374	]
		Eligibility	0.95	[	0.715	,	1.276	]
		DID	1.10	[	0.765	,	1.575	]
3 vs. 2	Cardiovascular Deaths	Period	1.03	[	0.843	,	1.264	]
		Eligibility	1.17	[	1.058	,	1.303	]
		DID	0.81	[	0.722	,	0.911	]
	Respiratory Deaths	Period	1.24	[	0.864	,	1.808	]
		Eligibility	1.04	[	0.847	,	1.276	]
		DID	0.94	[	0.750	,	1.172	]

improvement was found; the total number of vehicles in circulation actually increased because of the purchase of additional vehicles with the aim of circumventing the ban (Davis, 2008). Lin and Wei Umanskaya (2011) also highlighted that no benefit was observed in four major cities where driving restrictions were applied (São Paulo, Bogotá, Beijing, and Tianjin) and suggested that temporal shifting of driving might occur when the restrictions concern only a limited period of time (Lin and Wei Umanskaya, 2011). In Paris, traffic and traffic-speed restrictions constitute the main measure implemented during the acute pollution peaks within the framework of the AQA system, which is enforced by the police.

Beyond the effectiveness of these policies on air quality, few studies address the question of their effects on population health. Our study adds to this literature by providing an example of an AQA system that was initially ineffective on population health but for which changing to a more stringent threshold led to population health benefits. The existing literature shows contrasted findings of the epidemiological impact of these policies. For example, in their analysis of the impact of the AQA program in the city of Santiago, Mullins and Bharadwaj (2015) found that the set of mandatory measures activated by acute pollution peaks (driving restriction, shutdown of major fixed sources of emission, prohibition of biomass combustion and public messages) led to a reduction in ambient concentrations of particulate matter as well as a reduction in mortality among the elderly (Mullins and Bharadwaj, 2015). The stringency of some of the restrictions adopted in Santiago compared to other cities may explain these results. On the contrary, a large population-based cohort study conducted in Toronto by Chen et al. (2018) revealed that AQA allowed a reduction in asthma-related emergency-department visits but were ineffective in preventing mortality or cardiovascular morbidity (Chen et al., 2018). A recent paper evaluating the air quality health index alert program warnings in Hong Kong found that

the program was effective in slightly reducing some types of acute cardiovascular hospitalizations in the elderly population, but little or no effect on emergency hospital admissions for other types of cardiovascular diseases was found (Mason et al., 2020a). An evaluation of the effect of the same program on respiratory morbidity found that the air quality health index policy was associated with a reduction in hospital admissions for respiratory tract infections, especially among children, but did not have an impact on other respiratory diseases, or overall hospitalizations for respiratory causes (Mason et al., 2019). The contrary findings observed in our results when a different threshold is used to activate the AQA in Paris may help to explain the diverse findings observed in the literature considering health impacts of these policies. This highlights the importance of evaluating these policies, including their progressive change over time, to understand their value in decreasing air pollution related health burden.

As no health benefits were observed in the first implementation of the air quality alert system, our study suggested that these types of policies may not have the envisioned health benefits if the selected thresholds are too elevated, particularly in a context of a relatively low levels of air pollution. Policies based on air quality alerts trigged by excessively high air quality thresholds contradict the epidemiological evidence that there is no discernable threshold for which air pollution exposure does not pose a risk to human health. Furthermore, some studies reported supralinear dose–response relationships between air pollution exposure and health, which indicates that the effect is greater at lower concentrations (Apte et al., 2015; Liu et al., 2019; Lu et al., 2015; Papadogeorgou et al., 2019; Samoli et al., 2001; Zhang et al., 2017). In our study no evidence was found for an effect of the PM_{10} AQA program at the initial threshold values for both the overall population and the most exposed age group of people aged>75 (with positive rather than negative point estimates for the DID estimates) but lowering the

threshold values led to a reduction in cardiovascular and respiratory mortality. These results suggest that the highest proportion of deaths attributable to air pollution may stem from low levels of air pollution exposure, so policy interventions intended only to address high-exposure episodes (like the initial phase of the PM_{10} AQA program in Paris) may have little or no public health benefit. This is likely because concentrations below the current air-quality standards may still be hazardous for population health.

These AQA programs include a range of short-term measures targeting primary individuals' behavior, which is not particularly expensive and more politically acceptable, but they affect pollution concentrations only in the short run. Longer-term policies to be addressed to a larger range of systemic actors should be developed (improvements in public transportation, reorganization of urban spaces in order to improve priority route for public transports or non-pollutant transports like bicycles, mitigation of the direct effects of air pollution through healthy city development), and enforced actions addressed to major stationary emitters should be undertaken, such as taxation or industrial upgrading (Guan et al., 2016).

Our study used 15 years of follow-up data and applied quasi-experimental methods, capitalizing on defined thresholds as natural experiments to determine the causal relationship between the PM_{10} AQA program and changes in mortality. Nevertheless, we would like to point out some limitations. First, the process of air-pollution peak identification carried out by AIRPARIF is based on air pollution forecasts. Therefore, false negative and false positive air pollution peaks might have occurred during the intervention period, which might introduce a bias in our analysis. Moreover, the method to monitor PM_{10} changed in France in 2006, and data collected before 2007 underestimated the volatile part of PM_{10}. Our analysis focuses on PM_{10} and mortality, as AQA program for other pollutants (in particular Ozone and NO_2) could not be studied. The AQA program for these two air pollutants started in 1999 in the region of Paris and we did not have access to data before this date, so we could not examine a comparison period. In future studies, it will be possible to employ distinct identification strategies to evaluate the potential effectiveness of such policies on NO_2 and O_3 by using for example a regression discontinuity design that will capitalize on the arbitrary choice of thresholds while using only data in the period where such policies are implemented, as done elsewhere (Chen et al., 2018). Moreover, the air quality standards defined for O_3 and NO_2 are based on hourly average values, while our data consisted of daily average concentrations. For these reasons we could not take into consideration the effect of joint pollution events involving other air pollutants, which disregards that air pollution is a complex mixture resulting from chemical, physical and biological interactions among air pollutants and the effects attributed to single air pollutants may be influenced by the toxicity of the combination of several of them (WHO, 2013). Second, we could not differentiate between the levels of alert because of the very few number of warning-alert pollution peaks. Nevertheless, our sensitivity analysis carried out by excluding warning-alert peaks confirmed most of our results, suggesting that the contribution of warning-alert peaks on mortality is marginal. The only exception concerned the respiratory mortality during Phase 3, for which the sensitivity analysis did not indicate a mortality reduction, even imprecise, as estimated in the main analysis. Due to the small number of AQA days that occurred during our study period, we could not take into account differences in AQA episode severity; indeed, we could not consider a possible super-linear shape of the relationship between air pollution exposure and mortality, which describes a more important effect on mortality at lower pollutant concentrations (Apte et al., 2015; Papadogeorgou et al., 2019).

It is also important to mention that our study covers the period through 2015 and that more stringent measures have been adopted in the framework of the AQA program in Paris after 2016 (arrêté inter-préfectoral n° 2016–01383). Indeed, further studies with more recent data are needed to address the effect of these new measures on population health. Finally, the study context is a country characterized by low emissions and low annual mean temperature, therefore our results may not be generalizable to other contexts with heavier pollution or different PM composition.

5. Conclusions

Our study suggests that public policies grounded on a system of air quality alerts based on arbitrary threshold values for triggering anti-pollution measures may have limited impact on population health if the selected thresholds are excessively high. This highlights the implications of selecting inappropriate thresholds for the activation of air quality alerts and potential ineffectiveness when these thresholds correspond to pollution concentrations that remain high from an epidemiological perspective. It also demonstrates the need for continuous evaluation of these policies to understand their value in promoting population health. No level of air pollution is harmless (Daniels et al., 2000; Dominici et al., 2002; Papadogeorgou et al., 2019; Schwartz et al., 2002, 2001; Schwartz and Zanobetti, 2000; Yan et al., 2019), and our results indicate that only activating policies based on stringent standards of pollution concentration may be beneficial. Finally, emergency measures aimed to tackle acute pollution episodes should be accompanied by public policies for longer-term measures to reduce the air pollution exposure and protect population health.

CRediT authorship contribution statement

Anna Alari: Methodology, Software, Formal analysis, Writing - original draft. **Lara Schwarz:** Writing - original draft, Writing - review & editing. **Léo Zabrocki:** Writing - review & editing. **Géraldine Le Nir:** Data curation, Writing - review & editing. **Basile Chaix:** Conceptualization, Methodology, Writing - review & editing. **Tarik Benmarhnia:** Conceptualization, Methodology, Supervision, Writing - original draft.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2021.106583.

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