# Short-term ambient air pollution exposure and risk of atrial fibrillation in patients with intracardiac devices

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**Introduction:** Atrial fibrillation (AF) is the most common cardiac arrhythmia and is associated with substantial morbidity and mortality. Short-term exposure to fine particulate matter ($PM_{2.5}$) has been causally linked to higher risk of cardiovascular disease, but the association with atrial fibrillation (AF) is less clear.
**Methods:** We conducted a time-stratified case-crossover study to estimate the association between short-term air pollution levels and risk of AF episodes. The episodes were identified among patients with paroxysmal AF and an intracardiac devices able to register and store AF episodes. We obtained air pollution and temperature data from fixed monitoring stations and used conditional logistic regression to quantify the association of $PM_{2.5}$, particulate matter ($PM_{10}$), nitrogen dioxide ($NO_2$) and ozone ($O_3$) with onset of AF episodes, adjusting for temperature and public holidays.".
**Results:** We analyzed 584 episodes of AF from 91 participants and observed increased risk of AF episodes with $PM_{2.5}$ levels for the 48–72 hours lag (OR 1.05; CI [1.01,1.09] per IQR)] and 72–96 hours (OR 1.05 CI [1.00,1.10] per IQR). Our results were suggestive of an association between $O_3$ levels and AF episodes during the warm season. We did not observe any statistically significant associations for $PM_{10}$ nor $NO_2$.
**Conclusion:** Short-term increases in $PM_{2.5}$ in a low-pollution level environment were associated with increased risk of AF episodes in a population with intracardiac devices. Our findings add to the evidence of a potential triggering of AF by short-term increases in air pollution levels, well below the new WHO air quality guidelines.

**Keywords:** Air pollution, Particulate matter, Ozone, Atrial fibrillation, Case-crossover study

## Introduction

Atrial fibrillation (AF) is the most common cardiac arrhythmia and is associated with substantial morbidity and mortality.[1–3] The prevalence of AF is estimated to between 2% and 4% in the adult population with increasing prevalence with age.[3–5] With an aging population, estimates suggest that the prevalence of AF in adults 55 years and older in Europe will have doubled by 2060.[6]

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Fine particulate matter ($PM_{2.5}$) air pollution has been causally linked to higher risk of cardiovascular disease and the estimated burden of excess disease is substantial.[7–9] An extensive body of literature connects short-term exposure of ambient air pollution to ischemic heart disease but the association is less clear for arrhythmic disease.[7,10] Short-term air pollution exposure has been associated with higher risk of ventricular arrhythmias and cardiac arrest,[11,12] and there is some evidence that short-term $PM_{2.5}$ exposure is associated with electrical changes in the heart in animal models[13] as well as important electrocardiographic predictors of AF in human subjects such as P-wave complexity and PR-duration.[14] Although a conclusive connection between air pollution exposure and AF has yet to be established, a growing body of literature suggests an association with short-term increased risk of AF episodes with increased air pollution levels. A recent meta-analysis reported associations for both short-term exposure of $PM_{2.5}$ and $NO_2$ but not for particulate matter with a diameter of <10 μm ($PM_{10}$) or ozone ($O_3$), with a large heterogeneity between different studies.[15]

### What this study adds

We were able to study the association between air pollution and atrial fibrillation with a high temporal resolution by using data from patients with intracardiac devices. We observed positive association between $PM_{2.5}$ levels and atrial fibrillation episodes even though the air pollution levels were below the new WHO standards. Given the high prevalence of atrial fibrillation and the universal exposure to air pollution this has a significant impact on public health. Our findings add to the growing body of evidence of that there is no clear lower threshold for adverse air pollution effects.

Several previous studies have used hospital admissions for AF as outcome measure with mixed results, with a few studies that observed positive associations for short-term $PM_{2.5}$,[16–19] although others did not.[20–22] This approach has a limited ability to adequately capture cases of AF since it will not include cases of asymptomatic AF that do not lead to a health care visit and makes it difficult to get a correct assessment of when the episode started, given the delay between onset and time of seeking medical care. Taking asymptomatic AF into consideration, we previously conducted a study utilizing data from a large AF-screening study with ambulatory electrocardiogram (ECG) measurements twice daily for 2 weeks and observed an association between increased short-term $PM_{10}$ levels and AF episodes but not for $PM_{2}.5$, $O_3$, or $NO_x$.[23]

A few previous studies have also investigated the associations between short-term exposure of ambient air pollution and AF in specific patient populations with continuous ECG monitoring. A study from Boston of patients with implantable cardioverter-defibrillators (ICD), higher 2-hour average $PM_{2.5}$ was associated with an approximate 25% higher risk of AF per interquartile range increase in exposure.[24] Recent studies conducted in China and Italy have shown similar results with increased risk of AF with increased particulate matter levels in device patients.[25,26]

Most of the previous studies have been conducted in urban areas in the United States, England, Italy, China, and Korea, all considered to be medium to highly polluted areas. The evidence for areas with low-pollution levels is more sparse.

This study aims to investigate the short-term association between air pollution and AF episodes in a population with cardiac devices with continuous rhythm monitoring ability in a low-level pollution setting.

## Methods

We conducted a case-crossover study to estimate the association between short-term air pollution levels and trigger of AF episodes. Episodes of AF were identified among patients with paroxysmal AF and an intracardiac device able to register and store AF episodes. We obtained publicly available air pollution and temperature data from fixed monitoring stations. This study was approved by the Stockholm regional ethical board (dnr: 2016/725-31/4–31) and complied with the Declaration of Helsinki. All participants provided written informed consent.

### Patient recruitment

Recruitment of participants for the study was conducted at the arrhythmia out-patient clinic at Danderyd Hospital in Stockholm, Sweden. Participants with an intracardiac device able to sense, record and store intermittent AF episodes (pacemaker, ICD or implantable loop recorder), were recruited during their regular follow-ups. The inclusion criteria included age above 18, a diagnosis of paroxysmal AF and written informed consent to participate. The exclusion criteria included cognitive impairment and chronic/permanent AF (normal sinus rhytm can not be restored). At inclusion, data on AF episodes and total time in AF (AF burden) was extracted from the cardiac devices. We also collected information from the participants and their medical records regarding medical history, medication, extended periods away from home during the study period and smoking status. The participants were followed with data extraction of AF episodes at their regular clinical visits during the study period of 2017 to 2020.

Implanted cardiac devices can detect and store episodes of AF retrieved from the device at follow-up. We collected information on time of onset, duration, and atrial and ventricular rate for all episodes and when available, intracardiac electrograms (EGM). We required detected AF episodes to be longer than 30 seconds, have an atrial rate faster than 200 per minute and at least one

episode per participant with a detected AF linked with a stored EGM to confirm correct device-diagnosed AF.

Longer duration of device detected AF episodes are sometimes incorrectly divided into several shorter episodes by the device due to undersensing. To reduce the risk of this misclassification, we introduced an additional restriction that AF episodes had to be spaced by a period of at least 3 hours from the end of one episode until the start of the next. All episodes occurring during a time period that the participant stated that they were away for an extended period of time (more than 1 month) were excluded.

### Air pollution and temperature data

Air pollution and ambient temperature data were collected from stationary monitoring stations at roof-top level at the center of Stockholm. Hourly measurements of $PM_{2.5}$, $PM_{10}$, $NO_2$, and $O_3$ were recorded from a single monitor for each pollutant and the measurements were reported as micrograms per cubic meter ($\mu g/m^3$). The monitors are operated, and quality checked by the local environmental authority and the data are publicly available. We created 24-hour mean lags for $PM_{2}.5$, $PM_{10}$, $NO_2$, and $O_3$ ranging from 1 to 24 hours (lag0) to 144–168 hours (lag6) from the hourly monitor data.

As exploratory analyses, we created shorter exposure windows down to 1-hour means as well as longer lags 1–72 hours and 1–96 hours for all pollutants. Further, we investigated seasonal differences by dividing the dataset into a warm (May to October) and cold (November to April) period and conducted stratified analyses.

### Statistical analysis

We investigated the potential association between short-term exposure to air pollution and AF episodes using a case-crossover design with a time-stratified referent selection strategy.[27] Specifically, each episode of AF was matched to control periods on the same hour, day of week, and calendar month and year. We assigned distributed lags, lag0 (1–24 hr) to lag6 (144–168 hr), for both case and control periods and all the lags were entered simultaneously in one model (unconstrained distributed lags). For $O_3$, the main analysis was restricted to cases and control occurring during the warm season as $O_3$ levels during the cold season are low due to limited sunlight. The associations between air pollution and atrial fibrillation episodes were quantified with a conditional logistic regression, adjusting for 24-hour mean ambient temperature as a restricted cubic spline with 3 knots and public holidays as a dichotomous variable.

Paroxysmal AF is clinically and arbitrarily defined as AF episodes that are shorter than 7 days and spontaneously convert to sinus rhythm. In reality, AF patients typically progress from paroxysmal, to persistent, to permanent AF as atrial fibrosis and distention continuously increases, making them less likely to revert to normal sinus node conduction.[28] In our exploration of the potential for air pollution to trigger the onset of AF, we used a operational definition restricting our analyses to participants with an AF burden below 30% (i.e., less than 30% of the follow-up time spent in AF) in the main analysis assuming a saturation effect of air pollution at higher levels of AF burden.

In a sensitivity analysis, we explored a more stringent cutoff for AF burden to 10%. Further, we tested the robustness of using a single monitor by restricting the analyses to participants living closer than the median distance of 15.5 km from the monitor.

For all statistical testing a 2-sided $P$ value of 0.05 was considered statistically significant. All statistical analyses were conducted with Stata16 (StataCorp LP, College Station, Texas, USA) or R v4.1.1 (The R Foundation for Statistical Computing, Vienna, Austria).

## Results

In total, 125 participants with an acceptable implanted device were recruited and followed between 2017 and 2020. Ninety-one participants contributed data for the final analysis after excluding participants with an AF burden of more than 30% and consecutive AF episodes with a time gap less than 3 hours. The majority (84%) of the participants had a dual-chamber pacemaker with an atrial and ventricular lead (Table 1). About 60% of the participants were men and the mean age was 77 years. More than half of the participants had a diagnosis of hypertension. The total number of AF episodes included in the analyses was 584, with a mean (SD) of 6.4 (6.1) episodes per participant, ranging from 1 to 30 with the 95th percentile at 16 episodes. The median distance from the participants home address to the urban background fixed air pollution monitor was 15.5 km. Seventy-five percent lived within 22 km, and the total range was 2.3 km to 49 km. The excluded participants had a mean burden of AF of 42% and with similar mean age and sex distribution as the included participants. The median number of AF epsiodes for the excluded participants was 4 (IQR 2–8).

Stockholm is an area with relatively low air pollution levels compared with other European cities. The mean 24-hour $PM_{2.5}$ levels during the study period was 4.7 (IQR 3.6). Distribution of all studied air pollutants and ambient temperature is described in Table 2. $PM_{2.5}$ and $PM_{10}$ showed a correlation of 0.68 and $O_3$ and $NO_2$ showed a negative correlation of –0.17 during the warm season (Table 2).

Our main analysis investigating the association between air pollution and AF episodes with an unconstrained distributed lag model showed more pronounced associations for short-term $PM_{2.5}$ levels and for the intermediate lags, that is, 48–72 hours (OR 1.05 CI [1.01,1.09] per IQR) and 72–96 hours (OR 1.05 CI [1.00,1.10] per IQR), although the shorter and longer lags showed no associations. We observed a different pattern for $O_3$, with a stronger association for 1–24 hours (OR 1.12 [0.84, 1.48] per IQR) and 24–48 hour (OR 1.13 CI [0.99, 1.29] per IQR) compared with the longer lags. The distributed lag model did not show any clear associations for $PM_{10}$ nor $NO_2$ (Figure 1). We explored shorter exposure windows down to 1-hour averages and longer exposure windows (1–72 hour and 1–96 hour) for all the pollutants but did not find any statistically significant associations (Supplementary table 1; http://links.lww.com/EE/A193).

We conducted a stratified seasonal analysis for the warm (May to October) and cold (November to April) season. For $PM_{2.5}$ exposure, the association was slightly more pronounced for the cold season with a similar pattern to the main analysis, cold season $PM_{2.5}$ 48–72 hour (OR 1.06 CI [1.01,1.11] per IQR) and 72–96 hour (1.06 CI [1.00,1.12] per IQR) (Supplementary table 2; http://links.lww.com/EE/A193). We did not observe a clear associations between $PM_{2.5}$ and AF episodes during the warm season but the pattern of the association was similar to the cold season, albeit with less pronounced and non significant associations (Supplementary table 2; http://links.lww.com/EE/A193). As with the main analysis, we did not observe any conclusive associations for $NO_2$ and $PM_{10}$. Correlation of the different air pollutants and ambient temperature during the warm and cold season is available in Supplementary table 3 (http://links.lww.com/EE/A193).

In a sensitivity analysis, we excluded participants with an AF burden of more than 10%. The results for $PM_2.5$ and $O_3$ showed a similar pattern but with larger confidence intervals due to a smaller sample size (Supplementary table 4; http://links.lww.com/EE/A193). For $O_3$, the 24–48 hour exposure windows reached statistical significance. We did not observe any significantly different results for $PM_{10}$ and $NO_2$. When restricting the analyses to participants living closer to the monitor we observed stronger effect estimates for $PM_2.5$ for the 48–72 and 72–96 hour exposures. The associations for $PM_{10}$, $O_3$, and $NO_2$ were overall unchanged but with wider confidence intervals. Results are presented in Supplementary table 5 (http://links.lww.com/EE/A193).

Due to the limited sample size of our final dataset, we did not conduct any subgroup analyses of participants with different comorbidities. Results expressed for a 10 µg/m³ increase in $PM_{2.5}$, $PM_{10}$, $NO_2$, and $O_3$ are provided in Supplementary table 6 (http://links.lww.com/EE/A193).

## Discussion

In this case, crossover study investigating short-term associations between ambient air pollution levels and the risk of AF episodes in a population with intracardiac devices, we observed an increased risk for the lagged $PM_{2.5}$ levels. Results for other pollutants were less clear. Our results were suggestive of an association between $O_3$ levels and AF episodes with a borderline statistically significant during the warm season. We did not observe any statistically significant associations for $PM_{10}$ nor $NO_2$.

**Table 1.**

**Demographic and clinical characteristics of the 91 participants included in the final analyses**

| Characteristic | |
|---|---|
| Age, yr (SD) | 77.6 (7.8) |
| Female sex, no. (%) | 35 (38.5%) |
| Body-mass index, no. (SD) | 26.3 (4.4) |
| Smoking status, no. (%) | |
|   Never | 38 (41.8%) |
|   Current | 3 (3.3%) |
|   Former | 47 (51.6%) |
| eGFR, mean mL/min/1.73 m² (SD) | 53.7 (17.8) |
| Medical History, no. (%) | |
|   Heart failure | 18 (19.8%) |
|   Hypertension | 54 (59.3%) |
|   Diabetes mellitus | 13 (14.3%) |
|   Stroke or TIA | 10 (11.0%) |
|   Ischemic heart disease | 25 (27.5%) |
| Medication at inclusion, no. (%) | |
|   Beta-blocker | 56 (61.5%) |
|   Digoxin | 2 (2.2%) |
|   ACE inhibitor or angiotensin II receptor blocker | 45 (49.5%) |
|   Mineralocorticoid-receptor antagonist | 8 (8.8%) |
|   Antiarrhythmic drugs | 2 (2.2%) |
|   Platelet inhibitor | 7 (7.7%) |
|   Oral anticoagulation | 62 (68.2%) |
| Device type, no. (%) | |
|   Pacemaker | 75 (83.3%) |
|   Implantable cardioverter defibrillator | 4 (4.4%) |
|   Cardiac resynchronization therapy | 9 (10.0%) |
|   Implanterbar loop recorder | 2 (2.2%) |
| Atrial fibrillation episodes | |
|   Number per participant, median (IQR) | 4 (7) |
|   Atrial fibrillation burden, % (SD) | 4.6% (7.6%) |

ACE indicates angiotensin-converting enzyme; eGFR, estimated glomerular filtration rate; TIA, transient ischemic attack.

**Table 2.**

**Summary statistics of 24-hour mean air pollutants and ambient temperature levels**

| Air pollutants and temperature | Mean | 25th | 50th | 75th | Correlation[a] | | | |
|---|---|---|---|---|---|---|---|---|
| | | | | | $PM_{2.5}$ | $PM_{10}$ | $O_3$[b] | $NO_2$ |
| $PM_{2.5}$, µg/m³ | 4.7 | 2.1 | 3.4 | 5.7 | | | | |
| $PM_{10}$, µg/m³ | 11.8 | 5.6 | 9.2 | 14.9 | 0.68 | | | |
| $O_3$[b], µg/m³ | 53.1 | 38.8 | 52.1 | 67.1 | 0.26 | 0.43 | | |
| $NO_2$, µg/m³ | 11.2 | 4.4 | 8.2 | 14.2 | 0.18 | 0.25 | –0.17 | |
| Temperature, °C | 8.1 | 1.5 | 6.4 | 13.3 | 0.05 | 0.13 | 0.05 | –0.35 |

[a]Pearson Correlation Coefficient.
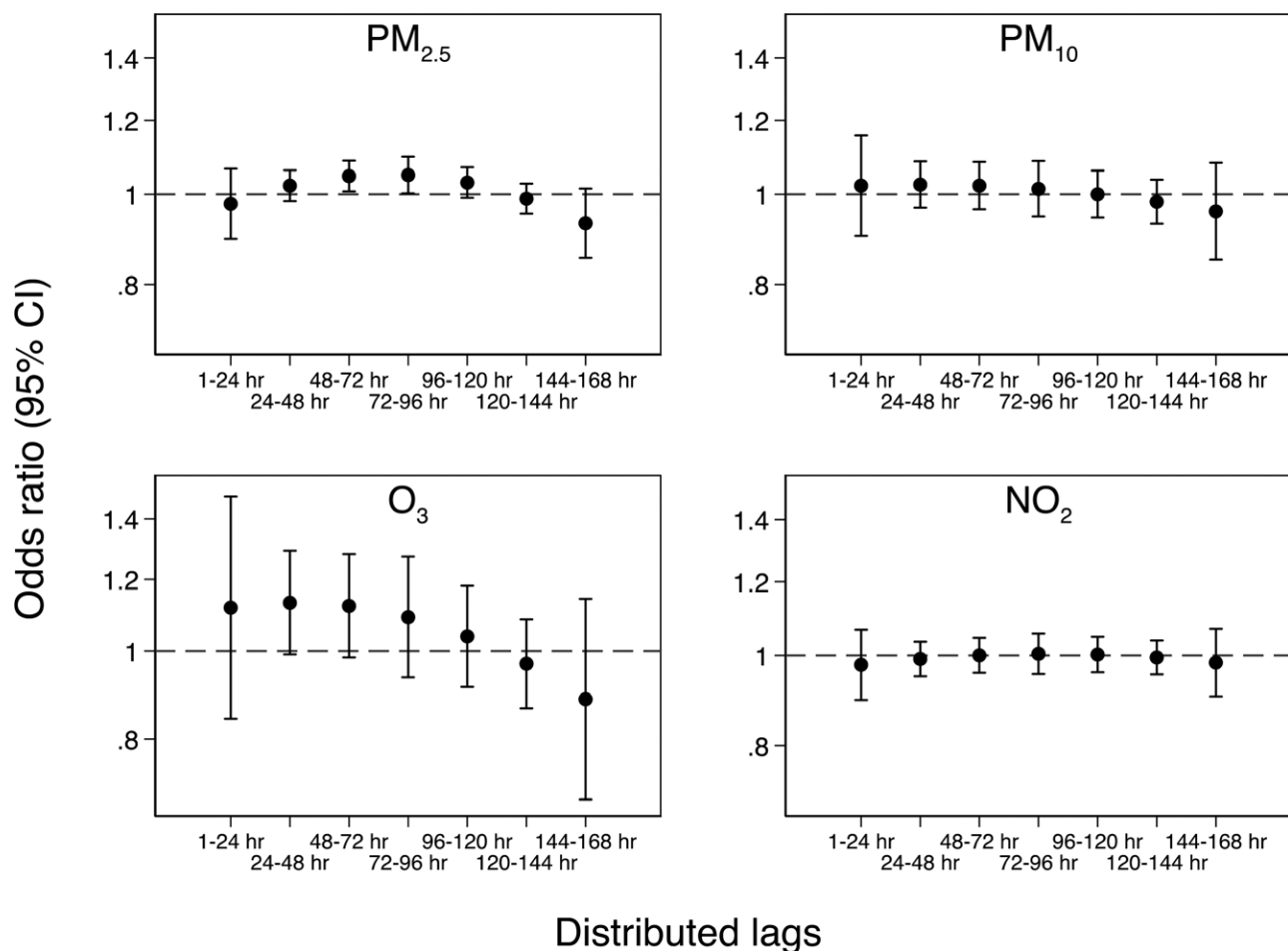[b]Warm-season average May–October.

**Figure 1.** Associations between air pollution levels and atrial fibrillation episodes with odds ratio and 95% confidence intervals for distributed lags of $PM_{2.5}$, $PM_{10}$, $O_3$, and $NO_2$ ranging from 1–24 hour to 144–168 hour per IQR change in air pollution levels ($PM_{2.5}$ = 3.6, $PM_{10}$ = 9.3, $O_3$ = 28.3, and $NO_2$ = 6.2). $O_3$ analysis stratified to warm season. The association is adjusted for temperature as a cubic spline and public holidays as a dichotomous variable.

Our current results are by and large in line with previously results from an AF-screening study, we conducted in Stockholm among 75-year olds using intermittent twice daily ECG measurements over 2 weeks. In that study, we observed positive associations for both $PM_{2.5}$ and $PM_{10}$, reaching statistical significance only for $PM_{10}$ and positive yet not statistically significant association with $O_3$.[23] The current study, however, includes patients with cardiac devices, hence more likely to have a higher background risk of AF episodes. More importantly, the use of a continuous device in the current study improves the efficiency of the study design.

There are a few previous studies investigating the association between air pollution and AF in similar patient populations with pacemakers or ICDs. Link et al.[24] reported positive but nonsignificant associations for 24-hour mean of $PM_{2.5}$ and observed statistically significant associations for 2-hour moving averages of $PM_{2.5}$ in a population of ICD-patients in Boston. Liu et al. observed positive associations for daily $PM_{2.5}$ and $PM_{10}$ levels in 100 participants with either pacemaker or ICD in Beijing for daily averages and for 2-day lagged exposure. The risk estimates were stronger for the shortest lag.[25] More recently, Gallo et al.[26] investigated the association between $PM_{2.5}$ and burden of AF in participants with intracardiac devices observed an increased risk with increased exposure for the participants with few cumulative AF episodes, in other words in participants with lower AF burden. These previous results are in line with our association of $PM_{2.5}$. However, we observed a more pronounced association for longer lag periods of 48–72 and 72–96 hours compared with shorter and nonlagged exposure. One of the studies reported

lags up to 3 days, with all lags showing positive significant associations[25] whereas two did not report any distributed lags[24,26] making direct comparisons for longer lags difficult. Apart from the difference in lag structure of the associations, there is a wide range of reported effect estimates between the different studies, with Link et al. reporting approximately 28% increased odds compared with 14% in our study and 3.8% by Liu et al. per 10 µg/m³ $PM_{2.5}$ for 24-hour exposure windows. Whether the reported associations for different lags and risk estimates reflects a different outcome data extraction methodology, analysis methods, particle composition or a difference in biologic effects is not clear. Further, different background levels of air pollution, more than 10 times lower in Boston and Stockholm than Beijing, might influence the association with higher increase per increment in lower levels if the concentration response curve is nonlinear, similar to other health outcomes.[29]

Although our null results for $PM_{10}$ are in line with a few previous studies[20,21] several others have shown positive and significant associations,[18,25,30] making conclusions regarding this fraction less certain. Several other studies have also investigated the association between $O_3$ and AF, some with positive associations[31] but the majority have not observed any statistically significant associations[16,20,21,24,25] and a recent meta-analysis did not observe an association.[15] $O_3$ may, however, have an added importance in Stockholm where the levels have continued to be comparatively high while particulate levels have decreased during the last decades.[32]

Although seasonal analyses reduced the sample size and limited our ability to observe any associations, a few exploratory

analyses suggested stronger associations for $PM_{2.5}$ during the colder season. Different composition of $PM_{2.5}$ during different seasons may explain part of the seasonal effect. In contrast, and as expected, associations between $O_3$ and AF during the cold season were weaker and less clear compared with the warm season. This is consistent with higher production of $O_3$ levels during the warm season as a secondary pollutant created by the reaction between NOx and $O_2$ in the presence of sunlight.

In summary although the evidence for $PM_{10}$ and $O_3$ is less clear, our results with associations between $PM_{2.5}$ and AF episodes are in line with several previous studies although the levels of $PM_{2.5}$ in Stockholm during our study period was considerably lower with a mean of 4.7 µg/m³. For comparison the mean reported in Link et al.[24] was 8.4 µg/m³, Gallo et al. 16.0 µg/m³[26] and for Liu et al.[25] 91.4 µg/m³. Importantly, our results suggest a potential harmful effect on AF of $PM_{2.5}$ with levels below the updated WHO air quality guidelines recommending 24-hour means below 15 µg/m³.[33]

### Mechanisms

AF is believed to develop through a progressive remodeling of the atria driven by inflammation and fatty acid deposition. Over time, the formation of fibrosis results in disturbed electrical properties that increases the risk of AF.[3] This process might be affected by long-term exposure to air pollution, but the potential triggering effect must be due to a different pathway.

There is some evidence from previous studies to support that autonomic tone responses[34,35] and oxidative stress[36] increases the risk of triggering AF and data from experimental exposure studies suggests that acute exposure to air pollution is associated with electrical disturbances in the atria, possibly preceding AF.[14] Our results indicated a lagged response to $PM_{2.5}$ exposure, suggestive of a delayed effect more in line with an inflammatory or translocating mechanism. The association between $O_3$ and AF was most pronounced for lag0 and lag1 (i.e., 1–24 and 24–48 hours before the episode) suggesting a more direct effect. However, our findings are only suggestive and further studies are needed to determine a conclusive pathophysiological pathway.[37]

### Strength and limitations

A potential limitation of this study is the use of a single fixed monitor for exposure classification. This will likely introduce some degree of exposure misclassification, but we expect it to be nondifferential and on average bias the result toward the null and decreased statistical power. An alternative approach would be averaging over multiple monitors or allocating the monitor believed to be closest to the participant. However, the approach with averaging over several monitors would significantly decrease the variability of the exposure and allocating separate monitors may introduce spatial misclassification with strong assumptions on participants whereabouts. Previous studies[38] give some support of using a single monitor with strong correlations between monitor $PM_{2.5}$ levels and personal exposure. However, we acknowledge that $NO_2$ and $PM_{10}$ probably have a higher spatial variability, given the relation to road traffic, and the exposures are not fully captured by a single urban background monitor. This misclassification might decrease our ability to observe an association and might partly explain the lack of an association for $PM_{10}$ and $NO_2$. Further, the included patient population have a greater comorbidity than the general population of people with AF and the generalizability may therefore be somewhat reduced. However, the source population for our study has substantial morbidity and mortality related to AF and is therefore important to study with results that may have implications on patient recommendations and public health policy.

This study has several strengths. Utilizing data from patients with intracardiac devices allows for inclusion of both symptomatic and asymptomatic episodes of AF greatly improving the outcome classification over studies using hospital records. Further, the temporal resolution of the outcome is high with data on starting point and duration for the episodes and this coupled with hourly measurements of air pollution gives us the ability to investigate both short and longer exposure windows with a high degree of certainty. A further strength is the use of a case-crossover model that adjusts for time independent confounders by design and with a time-stratified reference selection long-term seasonal trends are adjusted for as well. Finally, outcome classification relied on collected data by the devices and we corroborated the diagnosis of AF by at least one EGM per participant. The device algorithms are designed to be able to adapt the pacemaker function to the underlying rhythm and potential electrical disturbances and are therefore constructed to reduce outcome misclassification by accurately diagnosing AF. However, we cannot exclude that some episodes of AF went undetected and similarly that some cases of normal heart rhythm were incorrectly classified as AF.[39] This misclassification is most likely nondifferential in regards to air pollution exposure and would limit our ability to observe a true association by increasing random error.

## Conclusion

Short-term increases in $PM_{2.5}$ in a low-pollution level environment were significantly associated with increased risk of AF episodes in a population with intracardiac devices. The associations were lagged with the most pronounced association for the 48–72 hours exposure window. A positive association was observed for $O_3$ levels and AF risk but did not reach statistical significance in our main analysis. Our findings add to the evidence of a potential triggering of AF by short-term increases in air pollution levels, well below the levels new WHO air quality guidelines of 15 µg/m³ for daily $PM_{2.5}$ as well as the annual mean levels of 5 µg/m³ Given the high prevalence of AF and the universal exposure to air pollution, this has a potential impact on public health and highlights the need for further population-level approaches for air pollution reduction and primary prevention of disease.

## Conflicts of interest

G.W. reports a relationship with Health Effects Institute (Boston, MA) that includes: consulting or advisory and reports a relationship with Google, LLC (Mountain View, CA) that includes: consulting or advisory. The remaining authors declare that they have no conflicts of interest with regard to the content of this report.

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