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Short-term PM2.5 and Ozone Spikes and Acute Cardiopulmonary Outcomes

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Abstract:
The results show that ozone is related to the all-cause mortality rate on the same day (lag 0). The most valuable indicator is the maximum value of 8 hours in the warm season. $PM_{2.5}$ showed a short delay, which had an impact on the mortality rate and hospitalization at 0-3 points in time. The distribution lag nonlinear model (DLNM) improves these estimates. After mutual adjustment, both pollutants maintained statistical accuracy. High temperatures will amplify the risk, and vulnerable groups such as the elderly and people with low socioeconomic status will be more affected. China's research is consistent with the international lag and seasonal patterns. Although $PM_{2.5}$ in winter is relatively high. The 2021 WHO guidelines provide useful protection benchmarks. Biological mechanism - $PM_{2.5}$. Systemic inflammation and oxidative stress caused by ozone - consistent with the observed effect time. Graded alerts, targeted plans for high-risk populations, and hospital surge programs can reduce harm. Overall, this work provides public health and clinical teams with practical tools to manage cardiopulmonary risks associated with short-term contamination exposure.

Keywords: $PM_{2.5}$; ozone; distributed lag models; emergency demand; public-health operations.

1. Introduction

Modern multi-city time series research makes the time of pollution risk clearer. The effects of ozone come quickly: most deaths and respiratory effects will appear on the same day or the next day (0-1 delay), especially in warmer months [1]. The action time window of $PM_{2.5}$ is usually long, and it will accumulate within 24-48 hours (lag 0-2), and sometimes even longer. This pattern is reflected in different regions, so the WHO's 2021 Air Quality Guidelines will be the annual $PM_{2.5}$. The guidance value is tightened to 5 μg/m³, and the 8-hour ozone level in the peak season is set to 60 μg/m³, pointing out that the current level is both harmful and lacks a clear safety threshold. A large-scale global analysis supports this view: a study conducted by the British Journal of Medicine on 406 locations in 20 countries found that for every 10 μg/m³ increase in ozone concentration, the mortality rate will increase on the same day; a study conducted by the New England Journal of Medicine on 652 cities According to a research report, $PM_{2.5}$ of different climates and income groups. There is a strong short-term lag relationship with mortality [1,2]. A national time series study covering hundreds of cities in China shows the same time and direction, and increases regional differences related to precedents (NOx/VOC) and weather. In short, the risk pattern is stable, but the magnitude of the effect varies depending on the specific situation [3]. This paper discusses a practical problem. Long-term emission control is reasonable for urban investment, but daily choices - issuing alerts, setting up clinical personnel, and customizing information for high-risk groups - often lag behind evidence. The goal is to close that gap by translating what we know about timing, risk magnitude, heterogeneity, and biology into simple, actionable steps that improve health this week, not just by 2030. This work synthesizes rigorous, policy-relevant evidence to support time-sensitive decisions for public-health and clinical teams, with three aims: reduce avoidable healthcare demand; refine and target protective measures; and prioritize residents most vulnerable to pollution-related harm—such as older adults, people with cardiopulmonary disease, and low-socioeconomic-status populations [1].

2. Core Evidence

2.1 Effect Magnitudes and Lag Periods of Pollutant-Cardiopulmonary Associations

Across large, modern multi-city datasets, per-increment risks are modest in magnitude and consistent in direction—the profile expected from a ubiquitous exposure operating on physiologic thresholds. For ozone, the BMJ two-stage time-series reported statistically significant increases in all-cause mortality associated with 10 μg/m³ increases, with the weight of the association at lag0 and most of the signal contained within the same day. The same analysis explored policy counterfactuals and concluded that stricter standards would translate to avoidable mortality, supporting the utility of conservative, health-oriented trigger levels.

For $PM_{2.5}$, high-quality studies repeatedly show positive same-day and short-lag links with mortality and hospitalizations, often summarized over lag0–1 or lag0–3. Meta-analyses and systematic reviews, such as Atkinson et al.'s 2014 work in Thorax and more contemporary pooled analyses, converge on small, positive estimates for all-cause and cardiopulmonary outcomes. These effects are robust across continents after controlling for temperature, humidity, seasonality and long-term trends. Critically, several national cohorts sharpen the inference. In the United States, short-term $PM_{2.5}$ has been linked to cardiovascular hospitalizations in large administrative datasets including Medicaid and Medicare cohorts, with susceptible subgroups showing larger relative increases. Take the deSouza et al. Medicaid study as an example: it found harmful links between daily $PM_{2.5}$ and cardiovascular admissions, extending the evidence beyond the Medicare-aged population [4]. The conclusion is clear: the lag structure is important - the analysis at the lag 0 may miss the impact - health signals will appear even at relatively low concentrations. Operational takeaway is that on ozone days, same-morning communication is key; on $PM_{2.5}$ days, a 48-hour stance better matches the biology and the statistics. Hospitals that staff for today only after a PM peak will miss the next-day trickle-up in cardiopulmonary admissions that many time-series detect.

2.2 Key Methodologies and Pollutant Metrics

Modern short-term air-pollution epidemiology leans on Distributed Lag Non-Linear Models (DLNMs) for a reason. DLNMs jointly estimate a non-linear exposure–response function and a distributed lag structure via cross-basis splines, yielding a full lag–response surface from which day-specific and cumulative risks over a pre-specified window can be derived and thresholds or susceptible lag periods identified. They also reduce classic errors including assuming linearity where there is curvature at low concentrations and misplacing risk into the wrong day via too-narrow lag choices. Fitting only lag0 with a linear term is likely to lead to underestimation, as supported by methodological research in the field. Gasparrini's JSS paper and subsequent vignettes are the canonical references here, and they have reshaped standards for multi-city time-series in both environmental epi and heat-health literature.

For ozone, the warm-season daily maximum 8-hour average is the right tool. It matches inhalation physiology and human activity—people spend long continuous periods outdoors—and it is the principal short-term metric emphasized by WHO 2021[5]. Several large studies restrict the warm season to minimize confounding by wintertime chemistry and to target the period when ozone actually drives risk. This metric-season harmonization is why cross-study comparisons for ozone now read cleaner than a decade ago. A subtle but practical point is that metric choice shapes operations. Alerting schemes using 1-hour ozone peaks as triggers generate noisier public messages and mis-time clinical posture compared to designs keyed to 8-hour max. The latter is far closer to the exposure physiology that links to ED arrivals[6].

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2.3 Mixed Pollution Effects and Interactive Relationships

When $PM_{2.5}$ and ozone are included together, effect sizes in single-pollutant models often attenuate because exposures co-vary via shared sources and meteorology. Attenuation itself is expected. The key question is persistence: whether ozone's same-day and $PM_{2.5}$'s short-lag signals remain statistically precise after mutual adjustment. In large multi-city designs, the answer is frequently yes, though magnitudes vary by season, temperature, and city-specific precursor regimes [1,3].

As for interaction (non-additivity), the epidemiologic picture is mixed. Some recent work—national analyses that explicitly tested $PM_{2.5}$–O_3 co-pollution—reports synergistic patterns for specific endpoints or during extreme heat, but estimates are sensitive to modeling choices and exposure measurement error. The safest reading is that heat can amplify both $PM_{2.5}$ and ozone associations, and that co-exposure on hot days plausibly raises absolute risk more than either pollutant alone [7,8].

2.4 Heterogeneity of Effects and Influencing Factors

Effect sizes are not uniform. Older adults and people with cardiovascular/respiratory disease show larger relative risks for the same exposure increment. Lower-SES neighborhoods often bear higher risk, likely via baseline health, housing, and exposure differences. Season and temperature matter: warm-season ozone is strongest at lag0; heat increases vulnerability for both pollutants, steepening exposure–response slopes. Several multi-country studies report that combined heat and pollution days are among the highest-risk calendar cells [1,4,6,8]. Regional and precursor differences—including NOx/VOC regimes, background photochemistry, and meteorology—explain between-city heterogeneity. Local planning should be built on local data where possible, as these differences can shape the timing and magnitude of health impacts.

2.5 Comparison of Chinese and International Evidence and Policy Context

On direction and timing, China and international results converge. The 252-city national time-series spanning 2013–2017 (over 117 million admissions) reported positive associations between short-term $PM_{2.5}$ and ozone and cause-specific admissions across 14 major and 188 minor disease categories—an unusually granular mapping that moves beyond "respiratory/cardiovascular only." The timing of effects and seasonal patterns in China look much like those in North America and Europe. What differs is size: effect magnitudes vary by region in ways that track pollutant precursors and local weather. Chinese cities often see much higher winter $PM_{2.5}$—often several times above WHO guidance—because of heating emissions and inversions. Even if the relative risks are similar, the higher the baseline, the greater the absolute health impact. Chinese people also spend more time indoors, so in $PM_{2.5}$. During the incident, indoor air cleanliness is particularly important, especially in areas with small indoor and outdoor gaps.

With the increase of evidence, the policy baseline also tends to be stricter. The 2021 guidelines of the World Health Organization shows the 5-year average concentration of $PM_{2.5}$ is reduced to micrograms/cubic meter, and it is recommended that the ozone level for 8 hours in the peak season is 60 micrograms/cubic meter, which reflects the harm at relatively low concentrations and there is no clear safety threshold. Even in the context of backwardness of national laws, these benchmarks now guide many health protection alert systems. In 2024, the European Union has taken a step towards these levels, setting $PM_{2.5}$ by 2030. The five-year average concentration target of 10 micrograms/cubic metre marks an increased regulatory momentum and provides a guarantee for local health institutions to develop stricter and health-first trigger mechanisms.

A practical revelation: cities can adjust the prevention and control strategies of alarms or hospitals without waiting for the update of national standards. For the elderly, individuals with pre-existing cardiovascular conditions and people exposed to high temperatures, the WHO 2021 air quality standard is a reasonable reference for internal triggers and patient information transmission, and is also a reliable basis for taking temporary NOx/VOC and combustion control measures in high-risk weather. This is for winter $PM_{2.5}$ Chinese cities with high concentrations are particularly important, and WHO guidance provides more protection for vulnerable groups in these areas.

2.6 Biological Mechanism and Evidence Quality Assessment

Fine particles ($PM_{2.5}$) It can go deep into the lungs and into the blood. They can cause systemic inflammation, endothelial dysfunction, autonomic nerve imbalance and changes that promote thrombosis. These processes may increase the short-term risk of ischemia, arrhythmia, and heart failure decompensation, which is consistent with the lag 0-3 pattern observed in epidemiological studies. In terms of mechanism, $PM_{2.5}$ can activate Toll-like receptor 4 (TLR4) signal conduction, which in turn drives the release of NF-κB pathways and inflammatory cytokines

- which helps explain why $PM_{2.5}$ Exposure is associated with delayed cardiopulmonary events. As a potent oxidant, ozone reacts with airway-surface lipids and proteins to generate reactive oxygen species, producing epithelial injury and increased permeability this activates NF-κB–mediated inflammatory signaling, drives neutrophilic inflammation and cholinergic bronchoconstriction, lowers spirometric indices (FEV_1/PEF), and propagates systemic effects via circulating cytokines, endothelial dysfunction, and pro-thrombotic changes. It reduces lung function, triggers bronchiconstriction, and may produce systemic spillover effects. It can participate in the Nrf2-antioxidant reaction element (ARE) pathway, which coordinates cell defense, but high-concentration ozone exposure will crush the system and produce an acute airway response consistent with the lag pattern of asthma and COPD worsening. These biological pathways are consistent with the time observed in the population data: ozone has a same-day effect, and $PM_{2.5}$ has a short lag effect. Advances in methodology - especially the distribution lag nonlinear model (DLNM) and the conventional control of temperature and humidity - have enhanced the internal validity. Important uncertainties still exist, including the misclassification of exposure (urban monitoring and personal exposure) and residual mixing factors during extreme high temperature events [9–11]. For example, indoor $PM_{2.5}$ may vary depending on housing and ventilation conditions, resulting in deviations in population level estimates; in addition, heat waves often occur at the same time as high ozone levels, which makes it more difficult to completely distinguish between high temperatures and ozone effects.

3. Practical Response Strategies for Pollutant Event Days

3.1 Tired Public Health Alert System

For $FPM_{2.5}$ Events (winter/reverse temperature) should be alerted earlier in the day and last for about 48 hours to cover the lag period 0-1, and cover the part of the lag period 2 in the longer-term event. It is recommended that the elderly and people with heart or lung diseases continue to purify the indoor air (room high-efficiency air filter or central system equipped with MERV-13), close the windows, and avoid vigorous outdoor activities.When outdoor exposure is unavoidable, a properly fitted N95 respirator can reduce inhalation of particulate matter (e.g., $PM_{2.5}$/PM_{10}). However, its effectiveness against gaseous pollutants such as ozone is limited, so exposure timing and avoidance remain essential.In clinical terms, the next day‘s respiratory treatment, heart failure/chronic obstructive pulmonary disease management, and remote triage staffing of patients with high-risk chronic diseases should be marked. For ozone events, a morning alert should be sent so that action can be taken on the same day. Encourage outdoor activities to be arranged in the early morning or evening, launch an asthma action plan, ensure that first-aid inhalers are available at any time, and expand cooling facilities (cooling centers, sunshade transfer areas). In clinical terms, the emergency capacity of the day should be increased, rapid respiratory care channels should be set up in the triage area, and the emergency medical service department should be coordinated to cope with the afternoon peak. Because heat usually amplifies the ozone effect, stronger language and stricter triggers should be used on days of common exposure.

3.2 Clinical Management Strategies for High-Risk Patients

For patients with heart failure or coronary artery disease, daily weight monitoring should be strengthened, sodium intake should be strictly controlled, and the „green-yellow-red“ diuretic scheme should be implemented. Patients should be instructed to wait in well-ventilated indoor areas during peak hours, and consider remote consultations during afternoon activities that last for several days. For patients with asthma/slow obstructive pulmonary disease, their compliance with controlled drugs and the availability of rescue inhalers should be confirmed, the use of fog storage tanks should be checked, and written action plans should be provided. If the alarm persists, remote consultations should be arranged in the same week. These programs are short, repeatable, and synchronized with the lag structure; they are also more economical than the emergency department load.

3.3 Hospital Surge Capacity Planning

The surge capacity of the hospital should follow the third-level strategy associated with the air quality index (AQI) and the lag curve of each pollutant, and clear operating steps should be developed at each step. At the first level (AQI: moderate to unhealthy for sensitive groups), the hospital should notify the respiratory treatment department in advance to reserve flexible beds for patients who may be admitted to the hospital, and ensure that the oxygen, nebulizer and inhaler storage are sufficient and can be quickly deployed at any time. Under the second level (unhealthy), the response strategy should be changed from „preparing“ to „active buffering“: keep staffed beds to prevent bottlenecks, expand the coverage of respiratory therapy in each shift, enhance the telemetry capacity of cardiopulmonary monitoring, and deploy a mobile breath-

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ing team to cope with the usual high in the afternoon and evening. The surge in peak hours. Under level 3 (very unhealthy), scarce respiratory resources are retained by postponing elective cases competing for radiotherapy time or monitoring beds, and special respiratory fast lanes are established in the triage to shorten the outpatient treatment time for acute asthma, chronic obstructive pulmonary disease and heart failure symptoms. Timing is the hinge: maintain Level-2 measures for roughly 24 hours after a $PM_{2.5}$ peak to cover its short-lag (same-day to next-day) effects; for ozone, act the day of the elevation—when symptoms cluster in the afternoon and evening—and taper the following morning unless sustained heat lingers, in which case keep surge measures in place because heat reliably amplifies ozone-related risk.

3.4 Application of WHO 2021 Guidelines

The 2021 WHO values are derived from a systematic review and a modified GRADE approach, explicitly taking into account low levels of risk and uncertainty. Using them as internal triggers and patient-facing thresholds is reasonable in clinical and public health settings and is consistent with emerging policy trends in the European Union [12]. This is especially important for Chinese cities, because the $PM_{2.5}$ level in Chinese cities in winter often exceeds national standards, so WHO guidelines become more appropriate benchmarks for the protection of vulnerable groups.

3.5 Data-Driven Evaluation and Feedback Loops

After each activity day, a 24 to 72-hour review will be conducted to evaluate the number of emergency room visits, RT utilization, boarding time, EMS operating time and call center business volume by ICD group. These data will mark AQI and pollutant indicators to build a local dose response model. This not only supports the trigger mechanism of specific cities, but also helps to prove the rationality of small investment. For example, in the month of high $PM_{2.5}$ concentration in winter, installing dozens of HEPA devices in community centers can bring rich returns. Over time, the city‘s own data-based comprehensive DLNM will become the most powerful reference for stakeholders.

4. Conclusion

This study integrates our current short-term ozone and $PM_{2.5}$ Understand and transform it into measures that can be applied in practice by the health system. Ozone has a rapid impact - most of the effects appear on the same day (lag 0-1), and the maximum value of 8 hours in the warm season can best track the risk - and the lag time of $PM_{2.5}$ is relatively short (about 0-3), which is why the emergency concentration usually lasts until the next day before reaching a high level. Seize the right time to need DLNM, when ozone and $PM_{2.5}$. When modeling together, each of them is still important; high temperatures will push the risk. The burden is uneven: the elderly, people with heart disease or lung disease, outdoor workers, and communities of low socio-economic status are hit harder. The results of China‘s multi-city research are consistent with the global model in terms of lag and seasonality, but in winter $PM_{2.5}$. The baseline is relatively high. Even if there is a lag in the legal limit, it is recommended to use the WHO 2021 level in local practice. Biology is consistent with statistics: $PM_{2.5}$. It can cause systemic inflammation, endothelial dysfunction and the risk of blood clotting, and the oxidative stress of ozone can cause inflammation of the respiratory tract and bronchoconstriction - these mechanisms are similar to the observed picture of „front-loaded ozone and short lag particles“. In terms of action, the graded early warning system for specific pollutants, clear guidance for vulnerable groups („exercise before 9:00 a.m. or after 7:00 p.m.“; „carrying palliative drugs on days with severe ozone pollution“), and the hospital surge plan linked to the air quality index (AQI) and the expected lag window And based on the trigger level of the WHO, it can reduce avoidable cardiopulmonary visits and provide reliable short-term response plans for policymakers - especially in high-exposure cities in China. The next step: improve the personal exposure assessment, better distinguish between high temperature and other mixed factors, and constantly adjust triggers and response measures based on local data to ensure that the response plan remains real-time and localized.

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