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Effect of desert dust storms and meteorological factors on respiratory diseases

To the Editor,

Climate change is a growing global health concern with extreme meteorological, heat, and drought events resulting in increased risks of wildland fires, hurricanes, floods, and desert dust storms.[1] The average global surface temperature recorded from 2011 to 2020 was 1.09°C higher than in 1850–1900.[1] In Turkey, the increase in average temperature is even more pronounced. The annual temperature in 2020 was 1.4°C above the mean temperature recorded between 1981 and 2010.[2]

Studies suggest that annual dust aerosol concentrations have increased worldwide due to increased dust storms.[1,3,4] Turkey is in the Eastern Mediterranean basin and is exposed to dust particles originating from the Sahara and Arabian Peninsula, which are two of the largest dust source regions.[4,5] The southeastern Anatolian provinces including Gaziantep, one of the most heavily polluted cities in Turkey, are close to the Syrian border and face increased frequency of dust storms that originate in the Middle East's deserts and Sahara Desert.[4,5]

For the first time, we investigated the effect of dust storms, daily temperature, and particulate matter with a diameter ≤10 μm (PM_{10}), on mortality, emergency room (ER) visits, and hospitalizations due to asthma, chronic obstructive pulmonary disease (COPD), lower respiratory tract infections (LRTIs), and pulmonary emboli (PE) in Gaziantep, Southeast Turkey. Dust storm, PM_{10}, and climatological records from 1 January 2009 to 31 March 2014 were acquired through local monitoring stations and assessments of satellite photographs. Data on desert dust storms were provided by the Turkish State Meteorological Service (TSMO). Mean aerosol optical depth (AOD) values over land and ocean were used to determine the daily presence and absence of dust storms for the Gaziantep city area. Concurrent records of adult ER visits and hospitalizations due to respiratory diseases were obtained from four hospitals in Gaziantep City. The study population included 646,665 patients with a respiratory disease out of 10,000,000 total admissions reviewed from four hospitals in Gaziantep. In the study population, 22,278 patients were hospitalized, 624,387 were examined and/or treated in the hospital and discharged to their homes. A generalized additive Poisson regression model was designed to investigate the main and lag effects of dust storms, PM_{10} concentrations, and maximum temperatures on respiratory mortality and morbidity by adjusting for possible confounding factors. Data were presented as adjusted odds ratios (ORs) and 95% confidence intervals (CI).

Dust storms increased risk for same-day asthma mortality (OR = 3.98, 95% CI = 1.16–13.61) and same-day ER visits due to asthma (OR = 1.04, 95% CI = 1.01–1.07) and COPD (OR = 1.04, 95% CI = 1.01–1.07). Same-day hospitalizations due to asthma (OR = 1.47, 95% CI = 1.21–1.78), LRTIs (OR = 1.18, 95% CI = 1.07–1.29), 2-day lagged hospitalizations due to COPD (OR = 1.17, 95% CI = 1.01–1.36), and 3-day lagged hospitalizations due to LRTIs (OR = 1.12, 95% CI = 1.02–1.23) were positively associated with dust storms (Figure 1). The maximum temperature was positively associated with lag 2 LRTI mortality (OR = 1.06, 95% CI = 1.01–1.11) but not with mortality due to asthma, COPD, or PE. Similarly, the maximum temperature was weakly and inversely related to ER visits for asthma, COPD, LRTI, and PE. Hospitalizations due to LRTI and PE were positively correlated with maximum temperature, while a negative correlation was observed with asthma and COPD hospitalizations (Figure 2). PM_{10} did not affect pulmonary mortality but was positively associated with asthma, LRTI, and COPD ER visits. Hospitalizations due to asthma and LRTI were weakly associated with increased PM_{10}.

We have demonstrated that desert dust storms and meteorological variables such as ambient temperature and PM pollution can affect pulmonary mortality and morbidity in Gaziantep, Southeast Turkey. These findings are in accordance with previous studies reporting adverse health effects of global climate change on the respiratory system.[1,3,6] Governments and health authorities should develop mitigation strategies and take preventive measures against global climate change and environmental problems such as air pollution.

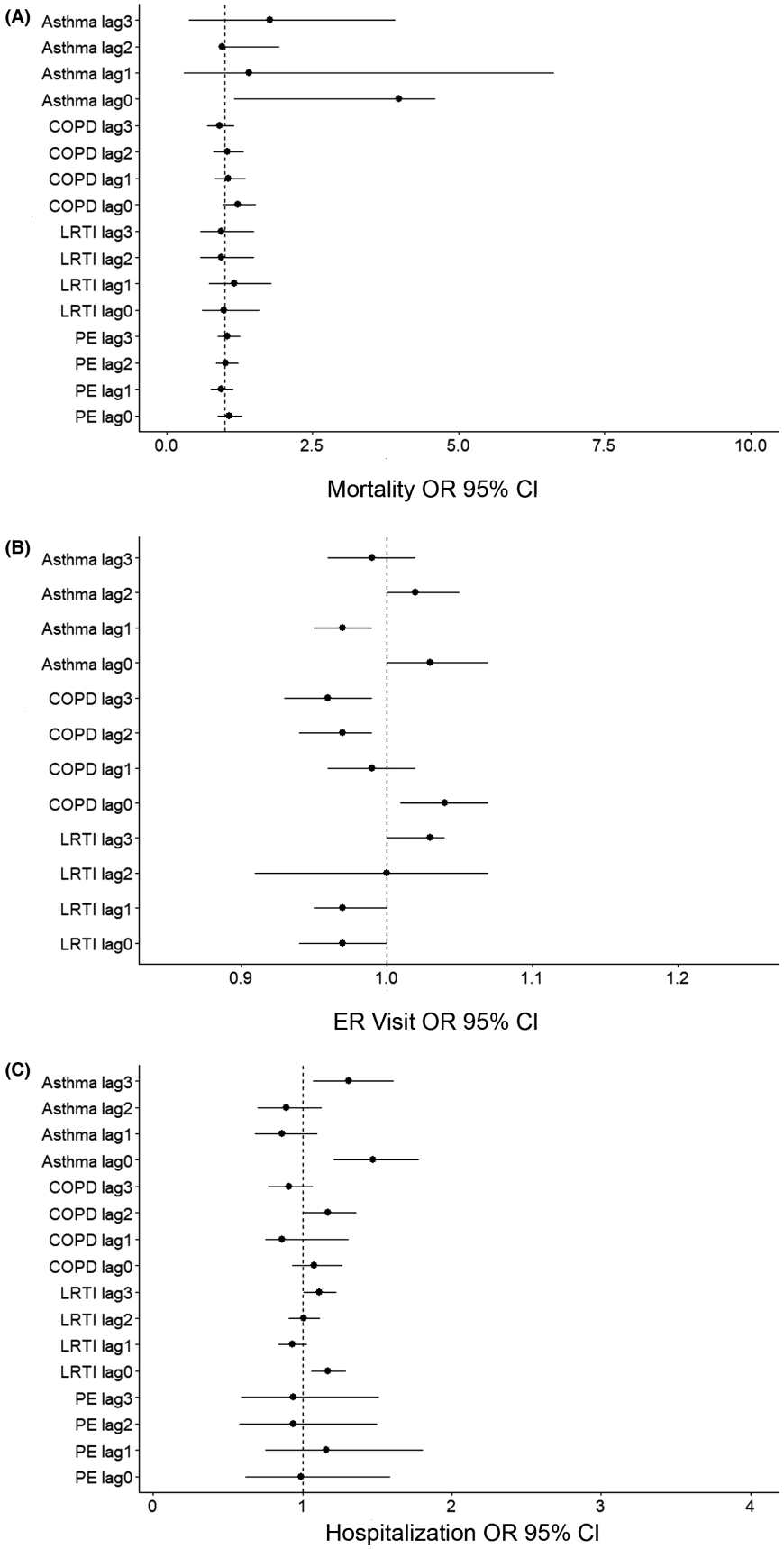

FIGURE 1 Effects of dust storms on respiratory indices at a lag time of 0-3 days. Results of generalized additive Poisson models for predicting number of deaths (A), emergency room (ER) visits (B), and hospitalization (C) due to asthma, chronic obstructive pulmonary disease (COPD), lower respiratory tract infections (LRTI), and pulmonary emboli (PE)

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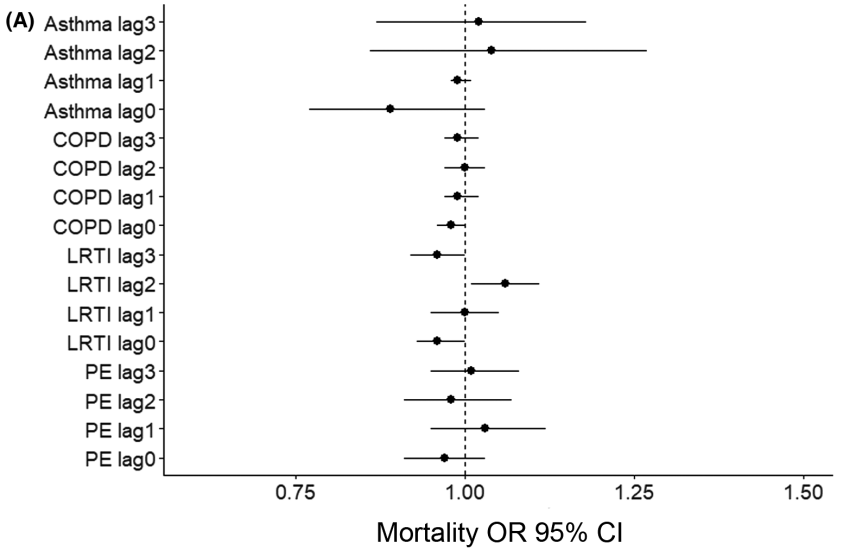

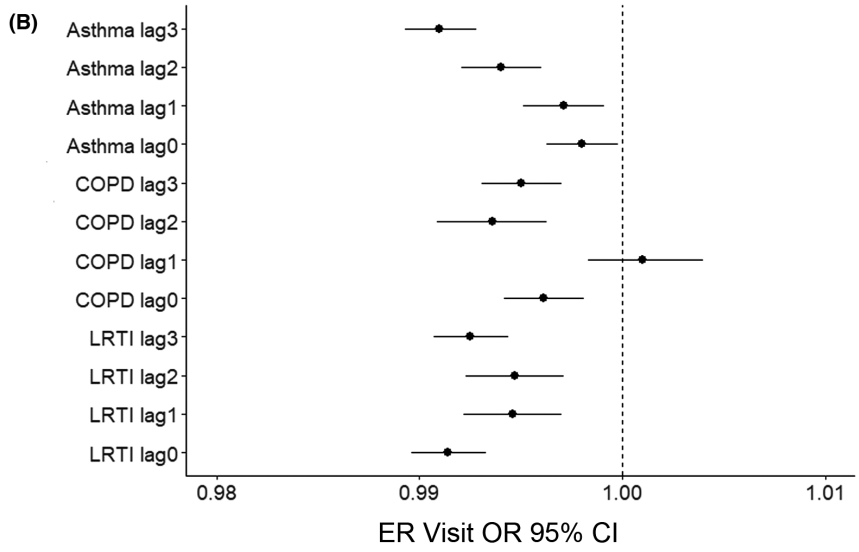

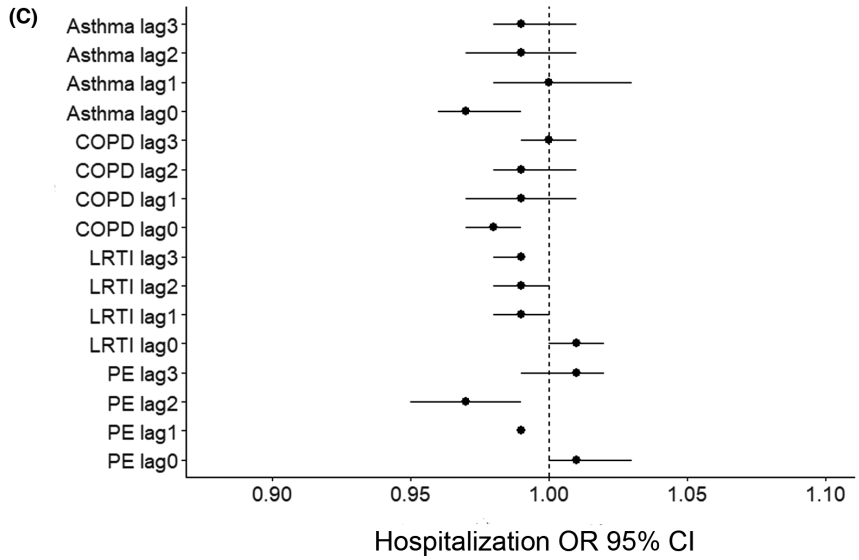

FIGURE 2 Effects of maximum temperatures on respiratory indices at a lag time of 0-3 days. Results of generalized additive Poisson models for predicting number of deaths (A), emergency room (ER) visits (B), and hospitalization (C) due to asthma, chronic obstructive pulmonary disease (COPD), lower respiratory tract infections (LRTI), and pulmonary emboli (PE)

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CONFLICT OF INTEREST

The authors declare that they have no competing interests.

Mustafa Boğan[1,2]
Seval Kul[3]
Behçet Al[1]
Mehmet Murat Oktay[1,4]
Müge Akpinar Elçi[5]
Kent E. Pinkerton[6]
Hasan Bayram[7,8]

[1]*Department of Emergency Medicine, University of Gaziantep, Gaziantep, Turkey*
[2]*Department of Emergency Medicine, Düzce University, Düzce, Turkey*
[3]*Department of Biostatistics, University of Gaziantep, Gaziantep, Turkey*
[4]*Vocational High School, Hasan Kalyoncu University, Gaziantep, Turkey*
[5]*School of Public, Health University of Nevada, Reno, Nevada, USA*
[6]*Center for Health and the Environment, University of California, Davis, California, USA*
[7]*Department of Chest Diseases, University of Gaziantep, Gaziantep, Turkey*
[8]*Koc University Research Center for Translational Medicine (KUTTAM), Istanbul, Turkey*

Correspondence
Hasan Bayram, Koc University Research Center for Translational Medicine (KUTTAM), Koc University Hospital, Davutpasa cad. No: 4, Topkapi, Zeytinburnu 34010, Istanbul, Turkey.
Email: habayram@ku.edu.tr

ORCID

Hasan Bayram https://orcid.org/0000-0002-5236-766X

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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Chronic rhinosinusitis endotypes associate with distinct local cytokine milieus that shape the distribution of innate lymphoid cells

To the Editor,

Chronic rhinosinusitis (CRS) is a heterogeneous group of chronic inflammatory diseases of the nasal/paranasal cavities.[1] It is frequently classified according to the presence or absence of nasal polyps (NP) (CRSwNP and CRSsNP, respectively). Depending on eosinophil numbers, CRSwNP is further classified as eosinophilic (ECRS) or noneosinophilic (NECRS).[2] To date, it is unclear how innate lymphoid cells (ILCs) and the nasal epithelium-derived cytokines contribute to these CRS endotypes.

Therefore, we first characterized the ILC subsets in the uncinate tissues (UTs) from control, CRSsNP, and CRSwNP patients and NPs from CRSwNP patients (Table S1). Flow-cytometric analyses