

INTERACTIVE EFFECTS OF HIGH TEMPERATURE AND AIR POLLUTION IN EUROPE

The report “Interactive Effects of High Temperature and Air Pollution in Europe” was prepared as a milestone by researchers in the EU Project EXHAUSTION*. This report summarises the evidence on the interactive effects of high temperature and various air pollutants on heart- and lung-related deaths and diseases (cardiopulmonary (CPD) mortality and morbidity) across Europe. The study was conducted at the city, small-area (e.g. suburban areas or municipalities), and individual level. The findings highlight the urgency in improving air quality across Europe, and alert stakeholders and policymakers that climate change adaptation measures need to be also taken into account to protect vulnerable subgroups and thereby increase European resilience towards climate change.

[*for more information on the project, visit the [project website](#) and the [preceding report](#)]

Main message

- There is an increased risk for cardiovascular (CVD) and respiratory (RD) disease and death associated with heat exposure in the warm season.
- Two of the most harmful air pollutants were studied, namely fine particulate matter with diameters $\leq 2.5 \mu\text{m}$ (PM_{2.5}) and ozone (O₃).
- A clear interaction between high temperatures and PM_{2.5} was observed, with the highest heat-related effects on mortality and increased rates of hospitalisation (morbidity) on days with high levels of PM_{2.5}.
- We found evidence of interaction between high temperatures and O₃ for CVD and RD deaths, with the highest increase in mortality on high O₃ days. No consistent interaction between high temperatures and O₃ was found for the occurrence of cardiovascular and respiratory diseases (morbidity).



- The interactive effect of heat and air pollution was stronger for respiratory mortality than for cardiovascular (heart-related) causes.

About the Report

This report focuses on one of the many key aspects of the project EXHAUSTION, i.e. quantifying the heat effects and the effect modification by two air pollutants, PM_{2.5} and O₃, on multiple cardiopulmonary outcomes. Data analysis was carried out at three levels: 1: cities; 2: administrative small areas (e.g. municipalities); 3: individual-level data. [For more information, refer to the [preceding report](#)]

Findings

A. European Cities

Across the European cities, CPD mortality increased in association with high summer temperatures, with a higher risk for RD mortality than CVD mortality. A stronger heat effect was observed at elevated levels of PM_{2.5} and O₃ in most locations for both CVD and RD mortality. The interaction between heat and air pollutants (heat effect modification) was stronger for RD than for CVD mortality. The overall heat effect modification by air pollution for Europe at city-level is summarized in Figure 1.

We observed large regional differences in the heat effects as well as for the interactive effects with air pollutants. A significantly higher heat effect with elevated concentrations of air pollutants was observed in countries like Portugal, Spain, the UK, Germany, and Switzerland. In contrast, results from other countries like Norway and Sweden followed inconsistent patterns.



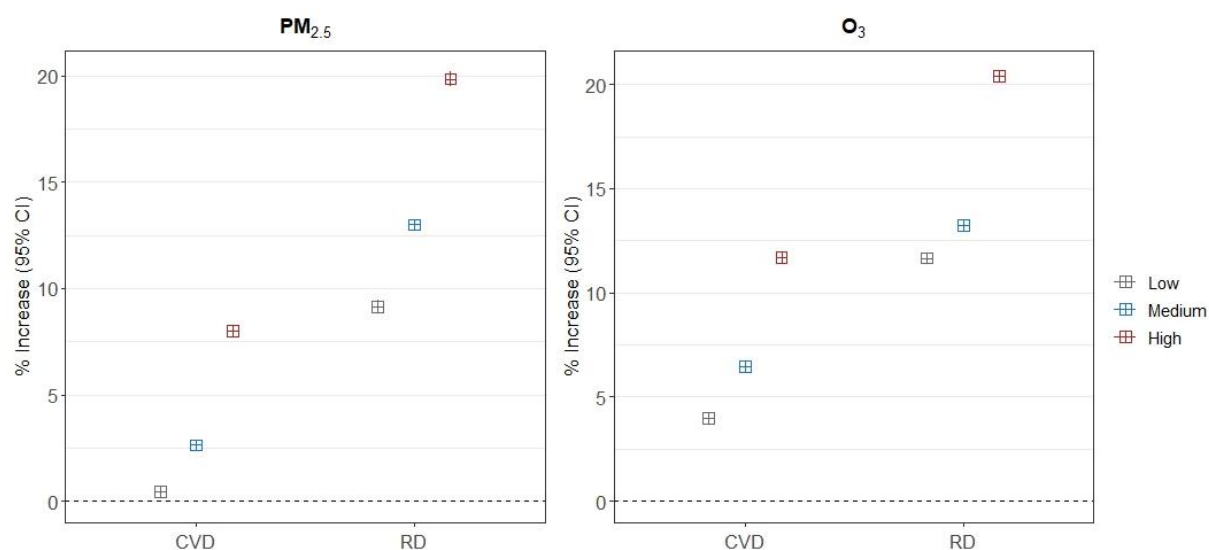


Figure 1: City-level overall effect estimates of heat stratified by the air pollutants PM_{2.5} and O₃ for Europe.

Estimates are presented as the % change in cause-specific mortality with the corresponding 95% confidence intervals for an increase in the mean temperature from the 75th to the 99th percentile of the location-specific temperature distribution by low, medium, and high levels of air pollutants represented by the 5th, 50th, and 95th percentile of city-specific respective pollutant distribution

B. Small-area level

Throughout the small areas in the five countries or regions (Norway, England and Wales, Germany, Italy, and Greece), the meta-analyses showed that the heat effects on natural-cause (or all-cause), CPD, CVD, and RD mortality were stronger at elevated levels of PM_{2.5}^a. We observed a stronger interaction between heat and PM_{2.5} for RD than for CVD mortality. Moreover, there was a suggestion of effect modification by O₃ in the relationship between heat and mortality, with the highest estimates at high levels of O₃. The overall heat effect modification by air pollution for CVD and RD mortality is presented in Figure 2.

We also investigated the heat effects on hospitalization in Germany and Italy. In both countries, we observed increases in heat-related natural-cause and RD morbidity, but no or negative associations with CVD morbidity. Furthermore, for natural-cause and RD morbidity in Germany, the heat effects were strongest at high PM_{2.5} levels and low O₃ levels. However, we detected no heat effect modification by air pollutants in Italy.

^a[particulate matter with diameters ≤10 µm (PM₁₀) for Greece as PM_{2.5} data was not available]

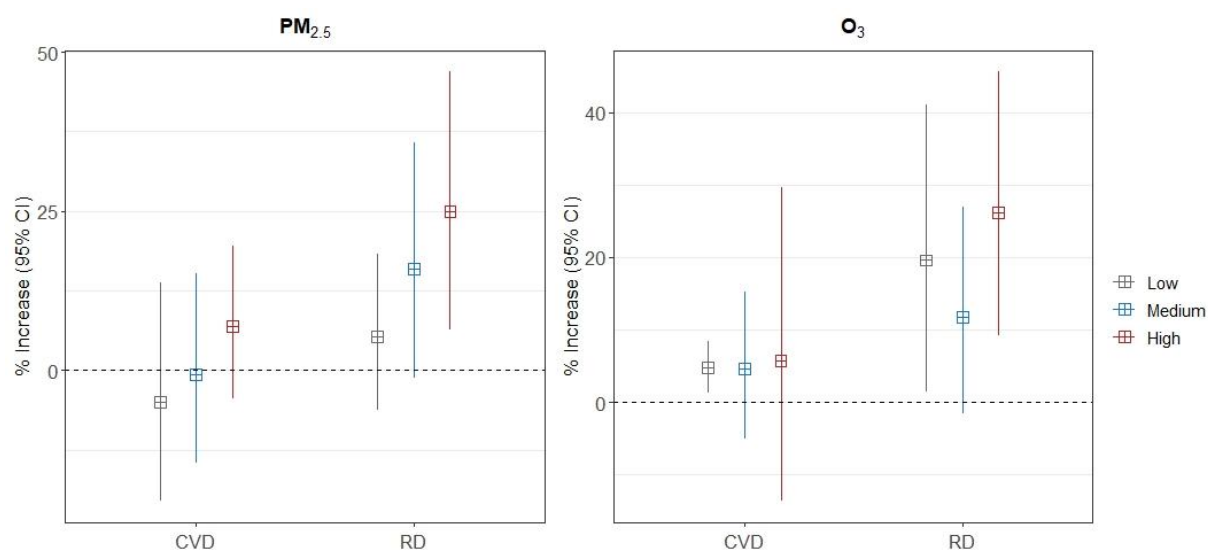


Figure 2: Small area-level overall effect estimates of heat stratified by the air pollutants PM_{2.5} and O₃.

Estimates are presented as the % change in cause-specific mortality with the corresponding 95% confidence intervals for an increase in the mean temperature from the 75th to the 99th percentile of the location-specific temperature distribution by low, medium, and high levels of air pollutants represented by the 5th, 50th, and 95th percentile of location-specific respective pollutant distribution

C. Cohort

In a third step, we also investigated individual data from study participants of European cohort studies. A cohort study is a type of epidemiological study in which a group of people with a common characteristic is followed over time to find how many reach a certain health outcome of interest (e.g. disease, death, or, more generally, a change in health status). When pooling the heat effects on mortality across cohorts [cohorts with mortality data: KORA (Germany), RoLS (Italy), and CONOR (Norway)], we observed stronger heat effects on natural-cause, CPD, CVD, and RD mortality on days with higher PM_{2.5}. The most significant effect modification was found for RD mortality. No consistent effect modification was found by O₃: the heat effects on natural-cause mortality were stronger at the medium and high levels of O₃, whereas the effects on CPD, CVD, and RD mortality were strongest at low O₃ levels. The overall heat effect modification by air pollution for CVD and RD mortality across cohorts is presented in Figure 3. The cohort-specific results showed the most consistent and significant heat effect modification by air pollution in the RoLS (Italy) cohort.

Besides mortality, we also examined heat effects on coronary morbidity (heart diseases) in the SWEDEHEART (Sweden) and KORA (Germany) cohorts. In the SWEDEHEART cohort, we unexpectedly observed stronger protective effects of heat on coronary morbidity with elevated levels of O₃. However,



the heat effects were not modified by the level of $PM_{2.5}$. In the KORA cohort, we found no heat effect modification by $PM_{2.5}$ or O_3 .

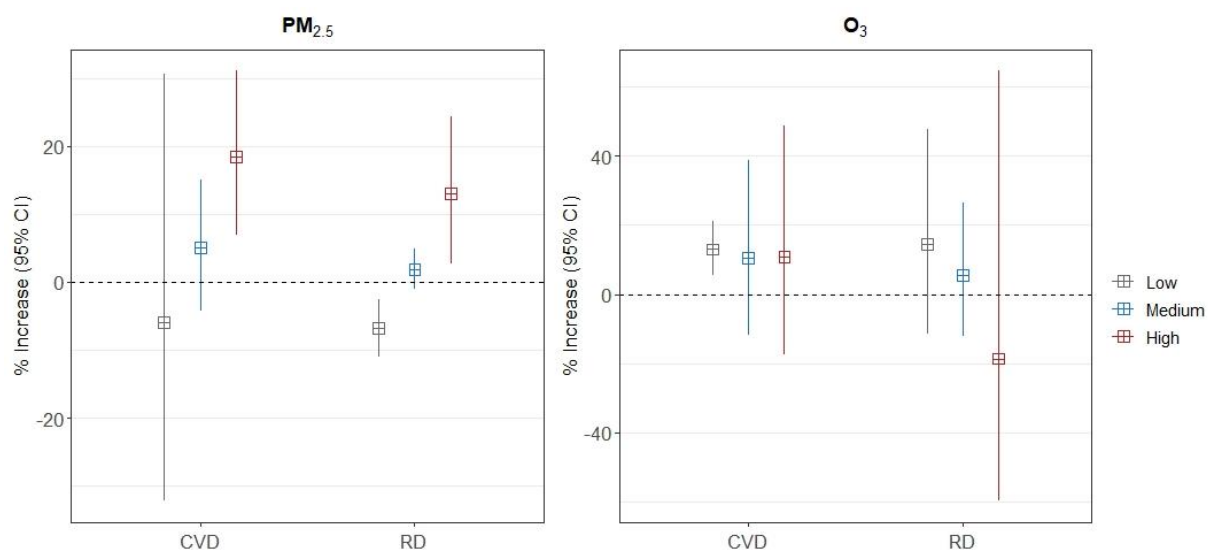


Figure 3: Cohort-level overall effect estimates of heat stratified by the air pollutants $PM_{2.5}$ and O_3 .

Estimates are presented as the % change in cause-specific mortality with the corresponding 95% confidence intervals for an increase in the mean temperature from the 75th to the 99th percentile of the cohort-specific temperature distribution by low, medium, and high levels of air pollutants represented by the 5th, 50th, and 95th percentile of cohort-specific respective pollutant distribution