Supplementary Material

1. TM5-FASST and health impact assessment methodology

The TM5-FASST tool is based on source-receptor (SR) coefficients derived and validated with the full chemistry transport model TM5 (Krol et al., 2005).

TM5-FASST considers the following pollutants: \( \text{O}_3 \), \( \text{SO}_2 \), \( \text{NO}_x \), VOCs, \( \text{NH}_3 \), \( \text{CH}_4 \) and particulate matter. The latter includes primary \( \text{PM}_{2.5} \) and its components black carbon (BC), organic carbon (OC), sea salt (SS), mineral dust (DUST), and secondary components (sulphate, nitrate, and ammonium). TM5-FASST splits global emissions in 56 regions (Van Dingenen, 2018) while the concentrations and impacts are computed on a \( 1° \times 1° \) gridded global domain which is further downscaled to \( 0.125° \times 0.125° \) for population exposure estimation.

Population weighted annual mean \( \text{PM}_{2.5} \) at 35\% relative humidity and seasonal daily maximum 8h average \( \text{O}_3 \) concentration metric (SDMA8h) are the exposure metrics used to compute health impacts in line with epidemiological studies (Jerrett et al., 2009; Krewski et al., 2009; Pope III et al., 2002). Mortality associated with \( \text{PM}_{2.5} \) is calculated, using the integrated exposure-response model (IER) adopted in the Global Burden of Disease (GBD2017) assessment (Stanaway et al., 2018), as the number of annual premature mortalities from six causes of death: chronic obstructive pulmonary disease (COPD), lung cancer (LC), lower respiratory airway infections (LRI), type 2 diabetes mellitus (DM), ischemic heart disease (IHD), and stroke.

Cause-specific excess mortalities are calculated at grid cell level using a population-attributable fraction approach (Murray et al., 2003):

\[
\Delta \text{Mort} = m_0 \cdot AF \cdot POP
\]

\[
AF = \frac{(RR-1)}{RR}
\]  

(1)  

(2)

where \( m_0 \) is the baseline mortality rate (deaths per capita) for the exposed population POP, \( AF \) is the fraction of total mortalities attributable to air pollution, and \( RR \) is the relative risk of death attributable to a change in P.W. mean pollutant concentration. For \( \text{PM}_{2.5} \) exposure, \( RR \) is derived from the IER functions (Burnett et al., 2014):

\[
RR_{PM_{2.5}} = 1 + \alpha \left( 1 - \exp \left[ -\gamma \left( PM_{2.5} - zcf \right)^\delta \right] \right) \quad \text{for} \ PM_{2.5} > zcf
\]

\[
RR_{PM_{2.5}} = 1 \quad \text{for} \ PM_{2.5} \leq zcf
\]  

(3)

where \( \alpha, \gamma, \delta \) are parameters provided in the abovementioned references and \( zcf \) is the counterfactual concentration, i.e. a theoretical minimum exposure level below which there is no excess risk. \( \alpha, \gamma, \delta \), and \( zcf \) were obtained from fittings to the median and 95 percentile exposure response curves of 1000
sampled RR’s in the exposure range 1 – 600 µg/m³. Our fittings reproduce the IER functions applied in the Global Burden of Disease 2017 assessment (Stanaway et al., 2018).

Mortality attributable to ozone exposure is based on the log-linear exposure-response function following the GBD approach, using the SDMA8h indicator with a RR of 1.06/10 ppb for COPD and a zero-risk threshold (zcf) of 29.1 ppb (Van Dingenen et al., 2018; Belis et al., 2022).

Bibliography


Krewski, D., Jerrett, M., Burnett, R. T., Ma, R., Hughes, E., and Shi, Y.: Extended Follow-Up and Spatial Analysis of the American Cancer Society Study Linking Particulate Air Pollution and Mortality, Health Effects Institute, Boston, USA, 2009.


2. **Source apportionment of O$_3$ exposure in selected Eclipse v 6b scenarios.**

Figure S1 Apportionment of O$_3$ exposure to its sources in the CLE scenario in 2020 and 2050.

Figure S2 Apportionment of O$_3$ exposure to its sources in the MFR BASE and MFR SDS scenarios in 2025 and 2050.