Air quality and related health impact in the UNECE region: source attribution and scenario analysis

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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: O_3, SO_2, NO_X, VOCs, NH_3, CH_4 and particulate matter. The latter includes primary $PM_{2.5}$ 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° x 1° gridded global domain which is further downscaled to 0.125° x 0.125° for population exposure estimation.

Population weighted annual mean $PM_{2.5}$ at 35% relative humidity and seasonal daily maximum 8h average 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 $PM_{2.5}$ 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):

$$\Delta\, Mort = m_0 . AF . POP \qquad (1)$$

$$AF = \frac{(RR-1)}{RR} \qquad (2)$$

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 $PM_{2.5}$ exposure, *RR* is derived from the IER functions (Burnett et al., 2014):

$$RR_{PM2.5} = 1 + \alpha\left\{1 - \exp\left[-\gamma(PM2.5 - zcf)^{\delta}\right]\right\} \; for\; PM2.5 > zcf \qquad (3)$$

$$RR_{PM2.5} = 1 \qquad for\; PM2.5 \leq zcf$$

where α, γ and δ 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. α, γ, δ, 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).

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2. Source apportionment of O_3 exposure in selected Eclipse v 6b scenarios.

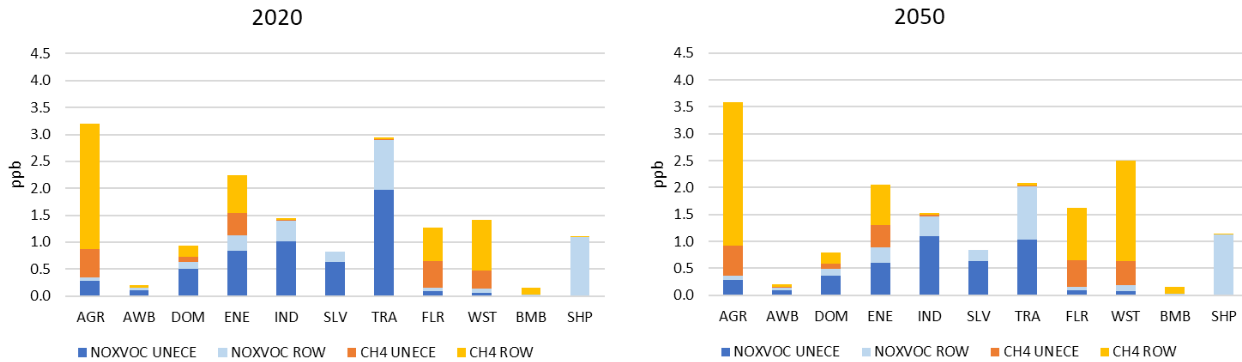

Figure S1 Apportionment of O_3 exposure to its sources in the CLE scenario in 2020 and 2050.

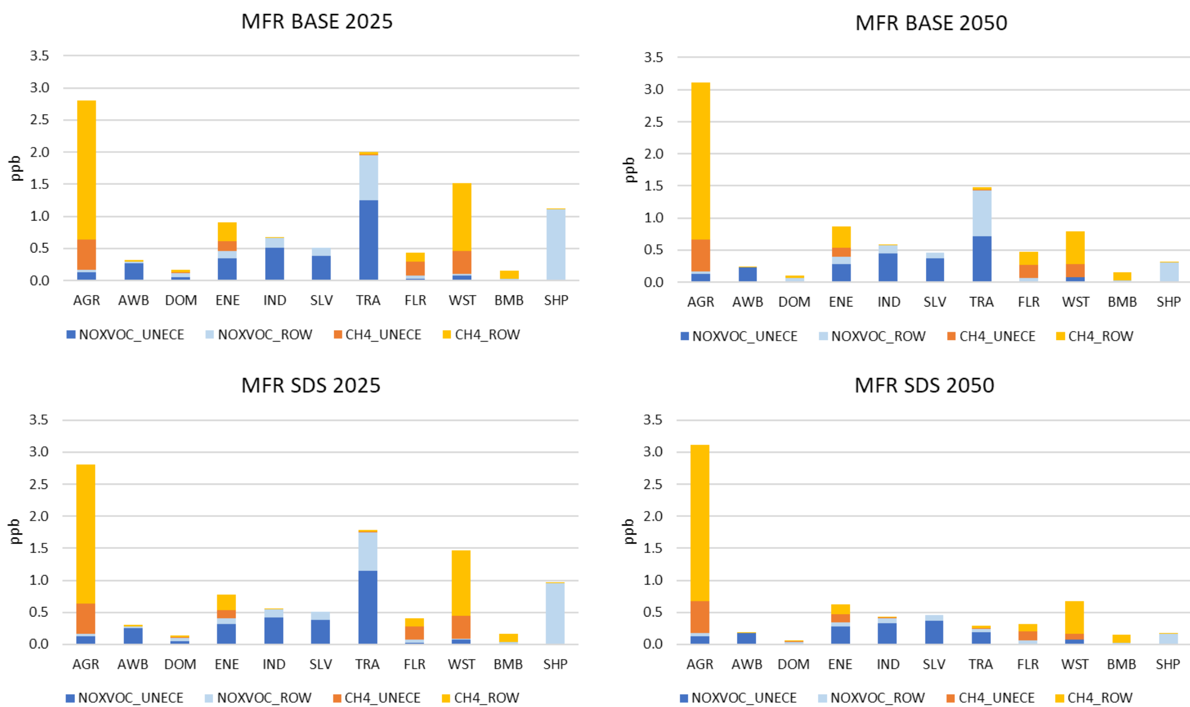

Figure S2 Apportionment of O_3 exposure to its sources in the MFR BASE and MFR SDS scenarios in 2025 and 2050.