npj | clean air

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https://doi.org/10.1038/s44407-026-00085-2

Quantification of life expectancy loss from air pollution in Slovakia

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This study estimates municipality-level life expectancy loss (ΔLE) attributable to PM2.5 and benzo(a) pyrene (B(a)P) air pollution across Slovakia. We conducted a cross-sectional ecological analysis of 2924 municipalities, combining modelled and monitored air pollution data with demographic, healthcare, and mortality records. Exposure was defined using an air quality model for 2020–2024, complemented by long-term monitoring and historical reconstruction dating back to 1990, and categorized into quantiles. Life expectancy at birth was calculated from mortality data for 2008–2024. Associations were assessed using multivariable regression with socioeconomic and healthcare-related covariates. In parallel, WHO and SCHIF exposure response functions were applied within life tables to estimate ΔLE under a counterfactual PM2.5 concentration of 5 μg/m³. The combined IVW estimate indicated a mean national ΔLE of 1.03 years. Municipalities in the highest PM2.5 exposure category showed substantially lower life expectancy (Q20 vs Q1: −1.96 years; 95% CI: −2.51 to −1.41), with a clear monotonic gradient. SCHIF produced a steeper exposure-response gradient, whereas the WHO HRAPIE framework produced higher but more uniform estimates. Results were robust to covariate adjustments. Air pollution effects exceeded those associated with healthcare accessibility indicators and were most pronounced in rural areas dominated by solid-fuel combustion. Results suggest that air pollution explains a substantial, though partial, share of life expectancy differences in Slovakia.

Over the past decade, air pollution has been increasingly recognized as one of the most important environmental determinants of premature mortality and reduced life expectancy worldwide[1–3]. Fine particulate matter smaller than 2.5 μm (PM2.5) accounts for the largest share of attributable health risk, due to increased cardiovascular and respiratory morbidity and mortality[4]. Long-term exposure to PM2.5 has been associated with life expectancy reductions of 1 to 3 years in heavily exposed populations[5]. Most of the world's population remains exposed to air pollution levels exceeding WHO guideline recommendations[2]. Although the new European Union (EU) air-quality standards are stricter than previous EU limits, they remain less stringent than WHO guidelines. Meeting these targets would still require major emission reductions and structural policy interventions[6]. Moreover, several studies[7,8] suggest that achieving WHO guideline concentrations may be unattainable in some regions due to natural PM sources and background concentration levels.

Slovakia, located in Central Europe, belongs to the group of EU countries with persistently elevated PM2.5 exposure and one of the highest average concentrations of B(a)P[3]. Its average exposure indicator rank is the third highest in the EU and has shown only marginal improvement over time[9]. Overall, Slovakia exhibits one of the least favorable combinations of current PM2.5 levels and long-term reduction trends; suggesting that the country risks remaining among the most polluted EU countries in the future[6]. Elevated pollution levels are shaped by both emission structure and terrain morphology, with basin and valley topography limiting atmospheric dispersion[10]. Although emissions from industry, transport, and energy production have declined in recent years, residential combustion has become the dominant source of emissions. Residential combustion accounts for ~74–78% of total national PM2.5 emissions and a slightly higher share of B(a)P emissions, based on official national inventory data reported by the Slovak Hydrometeorological Institute (SHMÚ)[11]. Further details on

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emissions are provided in Supplementary Chapter S4. Among households not connected to district heating systems, up to 80% of households rely primarily on firewood, and approximately two-thirds of households burn inadequately dried fuelwood due to insufficient storage time[12]. Under these conditions, wood combustion generates fine and ultrafine particles[13] enriched with toxic organic compounds, including polycyclic aromatic hydrocarbons (PAHs), with B(a)P widely used as an indicator of PAHs[14–17].

Several frameworks are used to quantify life expectancy impacts of long-term air pollution exposure. The World Health Organization's Health Risks of Air Pollution in Europe (WHO HRAPIE) approach applies a log-linear concentration-response function to PM2.5, with a relative risk of 1.062 per 10 μg/m³, yielding steady-state estimates of life expectancy gains under counterfactual exposure reductions[4,5]. To account for potential non-linearity and low concentration behavior, the saturation concentration hybrid impact function (SCHIF) has been proposed, allowing for exposure cut-offs, producing weaker effects at low concentrations and steeper gradients at higher exposures[18–20]. Both approaches are widely applied but differ in their sensitivity across exposure distributions.

A major limitation of existing methods is the characterization of historical exposure. Systematic PM2.5 monitoring has become widespread in the EU only in recent decades[3]. While air quality models provide spatially continuous exposure fields based on emissions, meteorology, and atmospheric chemistry, substantial methodological advances have occurred only in recent years[8,21]. Model performance depends critically on the quality of emission inventories, which are particularly uncertain for residential heating because information on the spatial distribution, fuel type, and operating conditions of small combustion sources is limited[12,22]. Historical PM2.5 concentrations are commonly reconstructed using hybrid statistical models that integrate satellite and monitoring data[23–25]. Although such models are accurate in data-rich settings (reporting R^2 values roughly between 0.6 and 0.9), uncertainty increases when extrapolating to earlier periods or data-sparse regions[26,27]. Historical reconstructions indicate that PM2.5 concentrations in Central and Eastern Europe during the early 1990s were substantially higher than present-day levels. Available modelling studies and exposure reconstructions suggest typical annual concentrations of ~20 to 35 μg/m³ across the region, with estimates for Slovakia generally below 30 μg/m³ [28,29]. Field measurements from The Central European Study on Air pollution and Respiratory Health (CESAR) campaign conducted in several Central European countries in the mid 1990s reported urban PM2.5 concentrations ranging roughly between 29 and 68 μg/m³, with the lower values observed in Slovakia and the highest in heavily industrialized areas of Poland.

Across the United States and Europe, reductions in long-term PM2.5 exposure have been associated with gains in life expectancy ranging from several months to nearly two years, depending on baseline exposure and analytical approach[30–32]. Global burden assessments suggest average losses of around one year attributable to PM2.5, with substantially higher impacts in high exposure regions[33].

Although PM2.5 is the main exposure metric in most studies, benzo(a)pyrene is frequently correlated with PM2.5 and has shown independent associations with health effects after PM2.5 adjustment in several cohorts, particularly in settings dominated by solid-fuel combustion[34–36]. While the evidence base for B(a)P is smaller and more heterogeneous than for PM2.5, B(a)P is widely recognized as a toxic particle-bound PAH[37] and may contribute to spatial gradients in life expectancy[4,38–42].

It has also been found that air pollution interacts with broader environmental, social, and healthcare determinants of longevity. Large scale studies have demonstrated life expectancy gaps of up to seven years between the most and least socioeconomically advantaged areas[43,44]. Additionally, access to timely healthcare services, including proximity to hospitals and emergency medical response times, also influences population level differences in life expectancy[45]. An extended review of the current state of the art on biomass combustion, health effects, air pollution exposure assessment methods, and historical air pollution projections is provided in the supplementary material, chapter S3.

Most studies linking air pollution to life expectancy rely on short term exposure averages, coarse spatial resolution, and PM2.5 as an air pollution proxy, with limited consideration of historical exposure, lag effects, or alternative concentration response functions, and often omit pollutants such as B(a)P. These limitations may bias local health impact estimates, particularly in settings where PM2.5 and B(a)P are elevated but spatially heterogeneous. In our study, we address these gaps by developing a municipality level, multi predictor life expectancy model that combines long-term exposure reconstruction for PM2.5 and B(a)P with a comparative application of HRAPIE and SCHIF exposure response functions, enabling quantification of pollution life expectancy gaps across municipalities under policy relevant counterfactual scenarios.

Results

Air pollution trends and model

Long-term monitoring data show predominantly steadily declining trends in both PM2.5 and B(a)P concentrations across Slovakia, with annual variability driven mainly by temperature and heating demand. In Slovakia, summer concentrations rise in some stations, while winter concentrations decrease rapidly; therefore, average long-term decreases of 0.54 μg/m³/year for PM2.5 and 0.093 μg/m³/year for B(a)P were used for historical exposure projections. Analysis of monitoring stations further indicates that long-term PM2.5 trends are relatively consistent across regions, with no strong spatial divergence in decline rates. This suggests that differences between municipalities are primarily driven by persistent spatial factors rather than fundamentally different temporal trajectories. Alternative reconstruction scenarios exploring spatially differentiated trends are presented in the sensitivity analysis in supplementary material, chapter S1. Modelled concentration fields reproduced expected spatial gradients in air pollution across Slovakia (see Fig. 1), with higher PM2.5 and B(a)P levels in south-eastern regions, and lower concentrations in southern lowlands. Mean annual concentrations reached 13.6 μg/m³ for PM2.5 and 0.98 ng/m³ for B(a)P, with substantially higher local maximums in some municipalities. Across 2924 municipalities, PM2.5 and B(a)P concentrations were strongly correlated (Pearson's $r = 0.94$). The strong correlation between annual mean PM2.5 and B(a)P concentrations is consistent with the dominant role of residential combustion in Slovakia. It likely reflects the large number of small rural municipalities where wood heating remains common, producing similar spatial emission patterns for both pollutants. However, because PM2.5 fields were used as one predictor in B(a)P interpolation, part of the observed correlation may also reflect shared model structure. Full air quality analysis with model results is provided in supplementary material, chapter S1.

Life expectancy loss through WHO and SCHIF methodologies

Both WHO HRAPIE and SCHIF frameworks showed different types of increase in life expectancy change (ΔLE) with increasing baseline PM2.5 exposure across municipalities, indicating larger potential gains in more polluted municipalities (see Fig. 2). However, the magnitude and distribution of effects differed substantially between methods and historical projections. At concentrations typical of the 2008 exposure baseline, populations lie near the inflection region of the SCHIF exposure response function, where further increases in pollution begin to produce disproportionately larger changes in life expectancy. By contrast, exposure levels representative of the 1990 baseline fall well into the steeper part of the curve, resulting in a markedly stronger ΔLE response. This non-linear behavior, with accelerated gains once PM2.5 equivalent concentrations exceed roughly 20 μg/m³, is not captured by the WHO log-linear framework, which assumes constant proportional effects across exposure ranges. Consequently, SCHIF better distinguishes the health benefits of reductions from historically high pollution levels. Spatially, both methods reproduce larger ΔLE loss in industrial and valley regions with poor dispersion and smaller losses in southern lowlands. However, SCHIF yields steeper contrasts between polluted and cleaner municipalities.

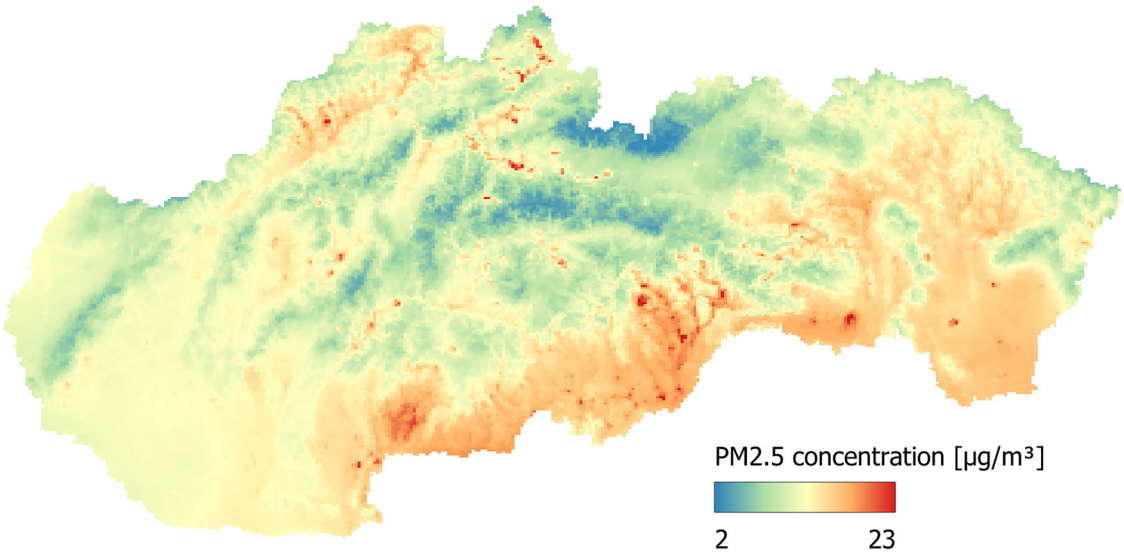

Fig. 1 | Distribution of PM2.5 concentrations in Slovakia in 2024. Spatial distribution of annual mean PM2.5 concentrations in Slovakia for 2024, derived from the national air-quality modelling framework combining monitoring data with regression interpolation (RIO) and IDW-R post-processing. The concentration fields incorporate the effects of emissions, atmospheric dispersion, and meteorological conditions. Annual maps for the period 2020–2024 show only minor differences, mainly reflecting interannual meteorological variability. The multi-year average (2020–2024) was used as the baseline exposure field in the analysis.

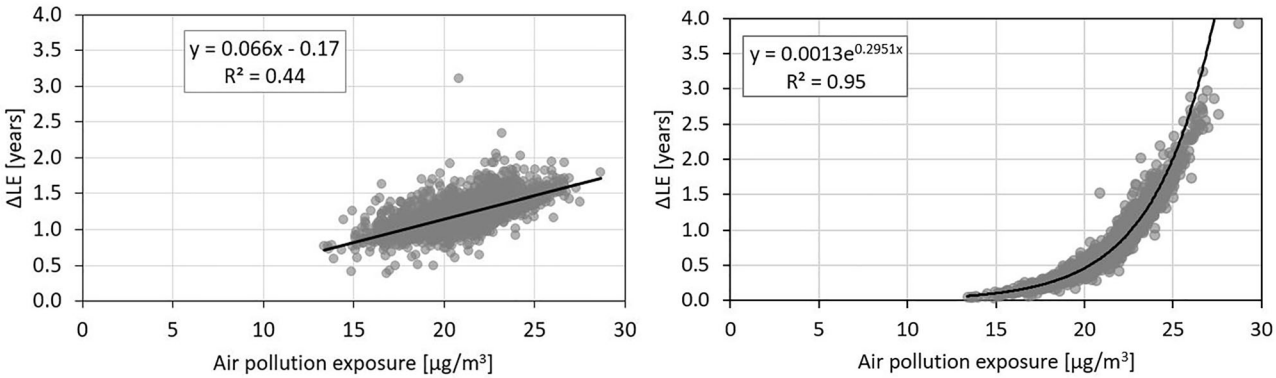

Fig. 2 | Life expectancy change using standard WHO and SCHIF methods. Comparison of life expectancy change (ΔLE) estimated using the a) WHO (left) and b) SCHIF (right) approaches across Slovak municipalities for the 1990–2024 air pollution trajectory. Each point represents an individual municipality. The two methods show systematic differences across the full exposure range, with WHO producing relatively higher and more uniform estimates at lower exposures, while SCHIF yields smaller effects in cleaner areas and a steeper increase in more polluted municipalities. Results for the 2008–2024 period are provided in the Supplementary Information, chapter S1.

When grouped into exposure quantiles, under SCHIF, the difference in mean ΔLE between the lowest and highest exposure quantiles (Q20-Q1) reached 1.84 years when exposure was defined over the 1990–2024 period, compared with 0.55 years for the 2008–2024 period (see Fig. 4). In contrast, the WHO log-linear approach produced a smaller and stable Q20-Q1 difference of 0.55 years across both exposure periods. Across municipalities, ΔLE estimates were consistently higher when exposure was defined over the longer historical period, reflecting the influence of cumulative exposure. Mean ΔLE increased from 0.17 to 0.72 years under SCHIF and from 0.72 to 1.20 years under WHO when comparing the 2008–2024 and 1990–2024 exposure definitions, respectively. While both frameworks apply mortality adjustments from age 30 years onwards and use identical counterfactual concentrations, their different exposure response formulations lead to markedly different contrasts between low and high exposure areas. Sensitivity analyses indicate that alternative exposure reconstruction scenarios primarily influence the magnitude of estimated health impacts rather than their spatial distribution. Uniform exposure adjustments of ±5% change the national mean life-expectancy loss by approximately ±0.2 years under the SCHIF framework, while preserving the relative ranking of municipalities. In contrast, scenarios that explicitly modify the spatial distribution of exposure lead to somewhat larger changes in municipal rankings, although the overall spatial structure of impacts remains highly correlated with the baseline estimates (see Supplementary material, chapter S1).

Statistical model results

The multivariable regression model explained a substantial proportion of inter municipal variability in life expectancy (LE), with most predictors showing the expected direction and significance (see Fig. 3). Effect estimates are reported as mean differences in LE relative to reference categories, with 95% confidence intervals. Among socioeconomic variables, educational attainment showed the strongest positive association with LE. Municipalities in the highest education quantile (Q5) had 2.78 years longer LE (95% CI: 2.47–3.09) than those in the lowest quantile (Q1), confirming that education remains one of the most powerful structural health determinants in Slovakia. In contrast, the municipal share of Roma residents was negatively associated with LE in a dose-dependent manner, with life expectancy declining progressively from 0.76 years (1–10%) to 3.14 years (91–100%) relative to municipalities without Roma residents. These associations likely

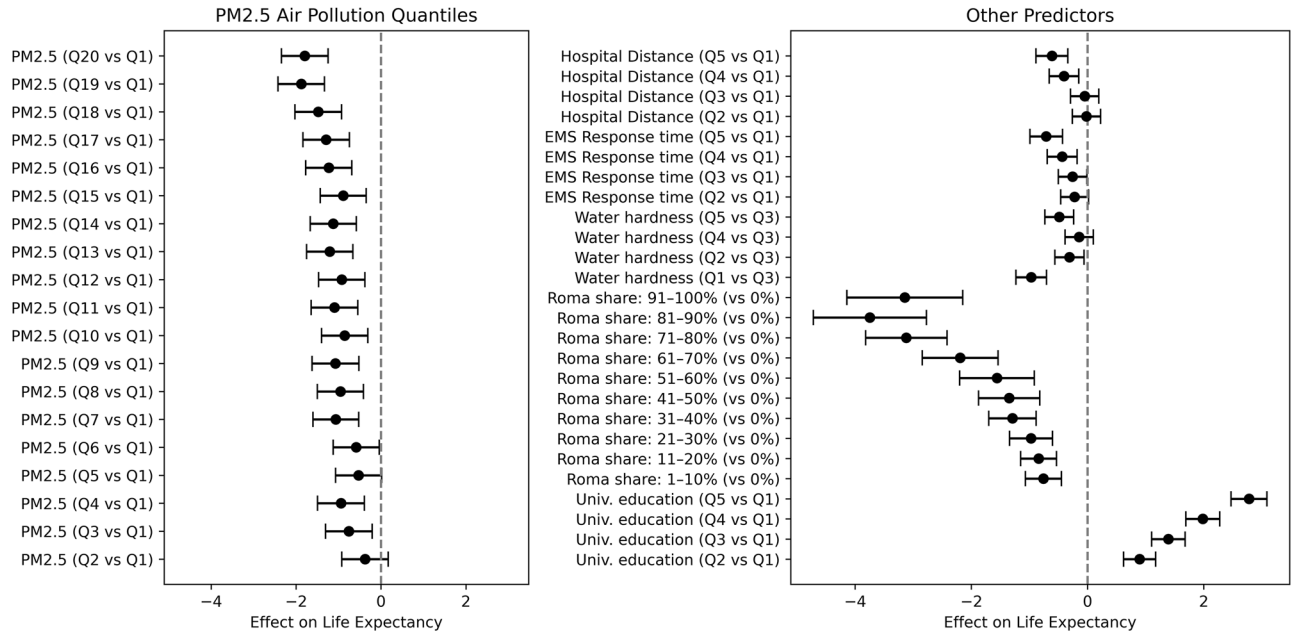

Fig. 3 | Regression model results on life expectancy loss. Results of the regression model showing differences in life expectancy (ΔLE) across municipalities grouped into quantiles (Q1 to Q20) for selected explanatory variables. For PM2.5, Q1 represents the least polluted municipalities and Q20 the most polluted. For hospital distance and emergency medical service (EMS) response time, Q1 corresponds to the shortest distance or fastest response and Q20 to the longest or slowest. For water hardness, Q1 represents the highest mineral content. For socioeconomic variables, Q1 corresponds to the lowest share of university educated population and the lowest share of Roma population.

reflect structural social and environmental disadvantages rather than individual level characteristics. Water hardness (Ca + Mg concentration) also exhibited a consistent association, with softer water linked to lower LE. Municipalities in the softest water category (Q1) had nearly 1-year shorter LE (95% CI: 1.23 to 0.71) compared to those with higher mineral content. Healthcare accessibility indicators showed weaker effects. Longer EMS response times and greater distance to hospitals are associated with modest LE reductions (0.4 to 0.7 years in the highest quantiles). Air pollution variables, represented by B(a)P and PM2.5 exposure quantiles, demonstrated statistically significant, monotonic negative effects on LE. The association was strongest in the upper exposure categories. Compared with the lowest exposure group (Q1), municipalities in the highest B(a)P quantile (Q20) exhibited 1.80 years shorter LE (95% CI: 2.34 to 1.25; $p < 0.001$). A gradual decline was evident from Q7 onwards, suggesting a clear dose-response relationship. Importantly, the magnitude of the air-pollution effect exceeded that of healthcare variables and was comparable to the influence of educational disparities.

Model comparison

Figure 4 displays how, across exposure quantiles, all approaches showed increasing life expectancy loss with rising pollution, but the magnitude and contrast differed between methods. Under the SCHIF framework, life-expectancy loss increased from approximately 0.18 years in the least polluted municipalities (Q1) to approximately 2.0 years in the most polluted municipalities (Q20). Municipality-level regression models yielded comparable estimated decreases at high exposure levels, with life expectancy decreasing by 1.97 years for PM2.5 and 2.20 years for B(a)P in Q20, indicating that both pollutants are associated with losses of roughly two years of life expectancy in the most affected municipalities.

Differences between methods were most pronounced at lower-to-mid-range exposures. In the lower quantiles (Q1–Q5), the regression models estimated greater life expectancy losses (~0.5–1.1 years) than SCHIF (0.18–0.42 years), with B(a)P and PM2.5 results alternating between each other. From mid-range exposures onward (~Q10–Q15), B(a)P estimates increasingly matched or exceeded PM2.5, suggesting a steeper gradient associated with combustion-related pollution. In the highest exposure categories (Q15–Q20), results for B(a)P and PM2.5 are very similar, with maximum estimated life expectancy loss reaching about 2.05 years for PM2.5 and 2.20 years for B(a)P, indicating convergence in overall magnitude but a stronger upper tail response for B(a)P. The WHO HRAPIE model yielded intermediate and comparatively flatter estimates, rising from about 0.95 years in Q1 to 1.50 years in Q20, resulting in a modest Q20–Q1 difference of 0.55 years and a compressed contrast across exposure categories. At higher exposures (~Q10–Q20), SCHIF and both regression models converged more closely, consistently indicating life expectancy losses on the order of two years, while WHO remained systematically lower, suggesting underestimation of the exposure gradient in heavily polluted municipalities.

To summarize results across methods, we derived a combined estimate using inverse-variance weighted fixed-effects fusion (IVW) of WHO, SCHIF, and both regression models (see Fig. 5). The combined estimates lay between SCHIF and the regression model results and closely matching the regression-based estimates at higher exposure levels. A Bayesian hierarchical combination produced similar central estimates but greater dispersion at lower exposures and was therefore used only as a sensitivity analysis. The results of IVW fusion applied to each municipality can be found in the supplementary material (Fig. S6).

Discussion

This study provides, to our knowledge, the first municipality-level assessment of life expectancy differences attributable to long-term air pollution exposure in Slovakia, integrating exposure response functions with empirical mortality and socioeconomic data. Across multiple approaches, air pollution emerges as one of the strongest determinants of spatial differences in life expectancy, with losses approaching 1.5–2 years in the most polluted municipalities, comparable in magnitude to educational disparities and exceeding effects associated with healthcare accessibility. The health burden is unevenly distributed across the exposure gradient. Life expectancy losses rise steeply with pollution levels and are concentrated in municipalities with high use of residential solid-fuel combustion and poor dispersion[10,22]. Differences between modelling approaches are largest

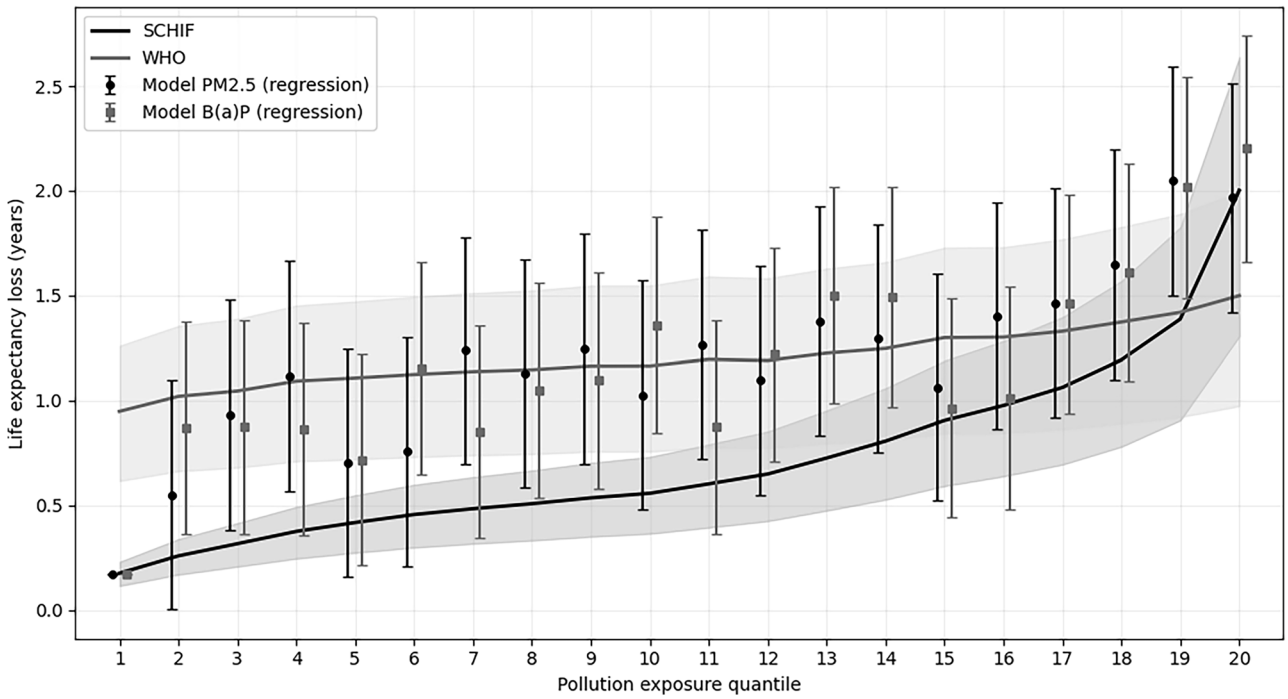

Fig. 4 | Comparison of all methods used to quantify life expectancy loss due to air pollution. Comparison of life expectancy change (ΔLE) estimated using WHO, SCHIF, and regression-based approaches across municipalities grouped into 20 exposure quantiles (Q1–Q20). Solid lines represent the WHO (light grey) and SCHIF (black) estimates, with shaded areas indicating their respective confidence intervals. Points show regression-based estimates for PM2.5 and B(a)P (average exposure for each quantiles are Q1 = 16.79, Q2 = 18.09, Q3 = 18.77, Q4 = 19.27, Q5 = 19.63, Q6 = 19.93, Q7 = 20.12, Q8 = 20.28, Q9 = 20.43, Q10 = 20.59, Q11 = 20.80, Q12 = 21.13, Q13 = 21.47, Q14 = 21.85, Q15 = 22.17, Q16 = 22.50, Q17 = 22.78, Q18 = 23.17, Q19 = 23.72, Q20 = 25.27). The figure highlights systematic differences between methods, with SCHIF and regression results showing a steeper increase in ΔLE at higher exposure levels, while the WHO approach produces a more uniform response across the exposure range.

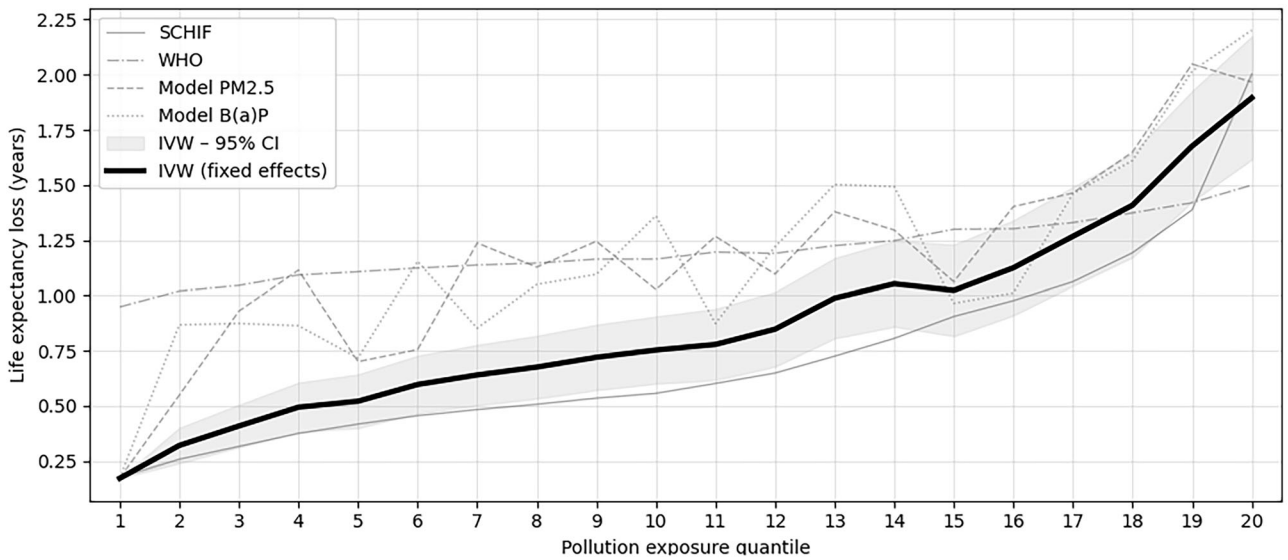

Fig. 5 | Inverse-variance weighted fusion of life expectancy results. Results of inverse-variance weighted (IVW) fusion of WHO, SCHIF, and regression-based estimates of life expectancy loss (ΔLE) associated with PM2.5 exposure. The black line represents the combined IVW estimate, with the shaded grey area indicating its confidence interval. Dashed lines show the mean estimates from individual approaches (WHO, SCHIF, and regression model). The IVW curve provides a consolidated estimate across methods, capturing the central tendency of different exposure–response approaches while accounting for their uncertainty, with particularly strong agreement across methods at higher exposure levels.

at lower exposures but converge in highly polluted areas, where all methods consistently indicate substantial life expectancy deficits, underscoring the robustness of these findings in the most affected communities. By combining regression-based estimates with WHO and SCHIF frameworks, the analysis highlights the importance of accounting for historical exposure trajectories and lagged mortality responses in regions undergoing rapid emission transitions. The results indicate that present day air quality alone does not fully capture long-term health consequences in Central and Eastern Europe, where major pollution reductions occurred only after the 1990s.

The statistical regression model developed in this study incorporates a broader set of determinants than air pollution alone and therefore captures a more complex structure of life expectancy (LE) variation across Slovak municipalities. Whereas WHO and SCHIF rely solely on exposure contrasts and assume equilibrium mortality responses, the regression integrates empirical health outcomes from 2008 to 2024 together with socioeconomic, demographic, and environmental covariates implicitly embedded in the long-term mortality data. This allows the model to reflect both historical exposure patterns and the lagged recovery of population health following the large reductions in pollution that occurred after the 1990s in Slovakia[46,47]. This finding indicates that, even some municipalities currently located in the lowest pollution quantiles often exhibit higher ΔLE in the regression model than predicted by SCHIF or WHO, because their present-day mortality still carries the imprint of past, substantially higher exposure levels[19]. In this sense, the regression model captures a time integrated, lag sensitive response rather than the steady state contrasts assumed in standard exposure response functions. These differences are most visible in the lower half of the exposure distribution (Q1-Q10), where SCHIF predicts very small impacts and WHO produces nearly constant relative changes, while the regression values remain appreciably higher. This suggests that part of the observed LE gradient in cleaner areas is influenced not only by current ambient air quality but also by residual effects of historical pollution, as well as by local determinants not represented in exposure only frameworks, as discussed in ref. 40. Indoor air quality, heating technologies, housing insulation, ventilation patterns, the prevalence of solid fuel use in colder regions, socioeconomic disparities, healthcare accessibility, and demographic structure all modulate underlying health risks[16]. These factors are indirectly absorbed into the regression coefficients but cannot be disentangled explicitly within WHO or SCHIF approaches.

When the four approaches were synthesized into a combined estimate (inverse-variance weighted fusion), the resulting curve closely followed the regression and SCHIF trajectories from approximately Q15 upward, indicating strong agreement in the high exposure domain where the mortality burden is primarily driven by chronic inhalation of combustion derived particles[20]. The Bayesian hierarchical fusion, although more uncertain at low exposures, converged toward the same pattern in higher quantiles. This reinforces the conclusion that despite methodological differences, all approaches identify a consistent and robust increase in ΔLE at the upper end of the pollution distribution (approximately from 22 $\mu g/m^3$). In contrast, the divergence at low exposures highlights the limitations of steady state models and the importance of incorporating temporal dynamics and contextual factors when interpreting health impacts in historically transitioning regions[48,49]. A methodological caveat is that the four input methods are not statistically independent, they rely on overlapping exposure fields and partially shared epidemiological evidence[34]. The inverse-variance weighted fusion therefore likely underestimates the true combined uncertainty, because between method correlations are not explicitly modelled.

Overall, the regression model complements WHO and SCHIF by providing a data driven perspective that integrates real world heterogeneity, historical exposure trajectories, and lagged mortality responses. At the same time, its behavior in low exposure areas illustrates that air pollution is only one component of a broader constellation of environmental and social determinants shaping regional differences in life expectancy. The regression model should not be interpreted as providing individual-level causal estimates. Higher pollution levels, particularly in areas dominated by solid-fuel combustion, are likely to co-occur with broader socioeconomic disadvantage that may not be fully captured by the included covariates, and residual confounding cannot be excluded. However, the exposure-response relationship is consistently monotonic across the exposure distribution and remains robust after adjustment for key structural determinants, including educational attainment and the spatial distribution of marginalized Roma communities. These variables capture major axes of social inequality in Slovakia and explain a substantial share of variation in life expectancy. Within this adjusted framework, the persistence and coherence of the air pollution gradient support its interpretation as a meaningful contributor to population health differences, while acknowledging the limitations inherent to ecological analysis. The remaining covariates included in the regression model showed associations with life expectancy that were consistent in direction and magnitude with previously reported evidence. Unmeasured or imperfectly measured confounders, such as dietary and physical activity patterns, smoking prevalence, alcohol consumption, occupational exposures, and detailed healthcare quality may still bias the estimated associations, particularly in the lower exposure quantiles where the air pollution signal is weaker[19]. Collinearity between socioeconomic indicators and pollution metrics further complicate causal attribution and suggests that our regression results should be viewed as integrated "signatures" of place based disadvantage rather than isolated pollution effects[50]. The IVW synthesis provides a pragmatic way to combine estimates derived from different frameworks. While these approaches share common exposure fields and life tables, they rely on different exposure response functions and therefore represent alternative interpretations of the health impacts of PM2.5 exposure. By incorporating the uncertainty ranges of the individual estimates, IVW offers a transparent method for deriving a consolidated estimate suitable for policy-oriented applications. This is particularly important in applications where a single estimate is required for spatially resolved analyses, such as economic assessments of air pollution impacts at the municipal level.

The WHO HRAPIE function assumes a log-linear exposure response relationship, resulting in a constant relative risk per unit of concentration across the entire range of pollution levels. While epidemiologically conservative, this formulation produces artefacts in settings with substantial spatial heterogeneity[4]. In our analysis, the WHO model generated comparatively high ΔLE values even in the lowest exposure quantiles, implying unrealistically large benefits in municipalities that are already relatively clean. Because the log-linear function scales proportionally, backward shifts in historical exposure preserve relative differences between areas. Therefore, the Q20-Q1 difference remains nearly unchanged regardless of the baseline level in the WHO model. This behavior contrasts with empirical evidence showing diminishing marginal risks at low concentrations and tends to flatten the true spatial gradient of life expectancy differences[40,51]. Consequently, the WHO approach is more suitable for national level burden of disease assessments than for fine scale or historical reconstructions such as those performed here[19]. The SCHIF approach, in contrast, applies a shape constrained nonlinear function that increases slowly at low exposures and steeply in the upper range. This yields very small ΔLE estimates in the cleanest areas but a rapid rise in health impact among municipalities with higher pollution, patterns that closely match our regression-based results, particularly above approximately the 15th exposure quantile. SCHIF therefore captures the nonlinear amplification of chronic mortality risk in polluted environments more realistically than the WHO function, which is confirmed also in many studies[19,33,52,53]. Its main limitation lies in the sensitivity to the assumed inflection region; small shifts in the curve's low exposure domain can alter national ΔLE estimates, particularly in countries like Slovakia, where a sizeable share of the population resides near the lower middle portion of the curve. SCHIF may also understate long-term effects of persistent low-level exposure, which continues to generate cardiovascular and inflammatory burden even below the apparent threshold region[34,52].

Together, the two methodologies thus define a useful uncertainty envelope. WHO provides a relatively high estimate in low exposure settings but with a compressed Q20-Q1 gradient of about half a year, whereas SCHIF yields smaller ΔLE at the clean end but a much steeper rise toward high exposures that aligns better with the regression results. The convergence of SCHIF and the regression curves in upper quantiles, combined with the comparatively flat WHO profile, indicates that nonlinear functions are more appropriate for capturing the steep life expectancy deficit in the most polluted municipalities[40]. A further limitation common to both WHO and SCHIF is that their exposure response functions are derived predominantly from Western European and North American cohorts, where the particle mixture is more strongly dominated by traffic and secondary aerosols than by residential solid-fuel combustion[51]. Extrapolating these relationships to

Slovak conditions, therefore, assumes that equal PM2.5 increments have comparable toxicity across different source mixtures, an assumption that may not fully hold in regions with a high contribution of coal and biomass burning. Moreover, both methods operate on area level average concentrations and do not account for within municipality exposure gradients or indoor-outdoor differences, which are likely to be substantial in settings with heterogeneous heating technologies and building characteristics[20,21].

Across different settings, reductions in PM2.5 have repeatedly been linked with measurable gains in life expectancy, typically on the order of 0.04–0.08 years per 1 μg/m³ decrease in long-term exposure, as shown for example in US cohorts and global burden assessments[4,5,54]. In our Slovak municipal level analysis, both the WHO and SCHIF applications fall into this plausible range, a mean ΔLE of about 1.36 years (WHO, 1990 baseline) and 0.70 years (SCHIF, 1990 baseline) for exposure reductions roughly in the order of 10 to 20 μg/m³ implies effect sizes very close to 0.04–0.08 years per 1 μg/m³. When the regression model is transformed into an approximate linear slope, it yields an LE gain of about 0.08 years per 1 μg/m³. That is at the upper end of the range reported by published studies, which is consistent with the historically high pollution levels experienced in many Slovak municipalities[19]. At the same time, our comparison of PM2.5 and B(a)P based regressions confirms that bulk particle mass is a robust but could be somewhat conservative proxy for health impacts. Both PM2.5 and B(a)P models predict similar losses of roughly 1.8 to 2.0 years in the highest exposure quantiles, with overlapping confidence intervals, yet the B(a)P curve is slightly steeper and more nonlinear, especially in mid exposure quantiles, in high quantile both B(a)P and PM2.5 results are very similar, indicating a common source of pollution[55]. This pattern aligns with evidence that PAH enriched particles retain independent associations with mortality and cardiopulmonary outcomes even after adjustment for PM2.5, suggesting that using PM2.5 alone may underestimate the true benefit of interventions targeting solid fuels combustion sources in some areas[4]. In our context, the strong correlation between B(a)P and PM2.5 implies that most of the risk is captured by PM2.5 fields, but the B(a)P results indicate that part of the remaining uncertainty, and likely a fraction of the residual LE gap is linked to this more toxic PAH fraction.

From a measurement perspective, B(a)P poses additional challenges. Monitoring networks are sparser, time series are shorter, and analytical detection limits are higher and more variable than for PM2.5[7]. As a result, our B(a)P based regression is fitted on a more restricted and potentially noisier subset of the exposure space, which may contribute to the wider confidence intervals and the more nonlinear pattern at high quantiles. Furthermore, neither the PM2.5 nor B(a)P based models explicitly adjust for pollutants such as NO2 or O3, nor for particle composition beyond a single PAH tracer[56]. In practice, any LE effect attributed to PM2.5 or B(a)P likely reflects a broader mixture of combustion-related pollutants, and the partitioning of risk between these components remains uncertain. Finally, the magnitude of life expectancy differences attributable to air pollution in our study, ~0.5–2 years across municipalities is comparable to major behavioral risk factors such as unhealthy diet, physical inactivity, smoking or harmful alcohol use, and clearly exceeds the impact of any other single environmental exposure[57]. Targeted air quality policies, particularly those addressing residential combustion, should therefore be a central pillar of public health strategy, alongside measures targeting lifestyle, behavioral and socioeconomic risk factors. Air pollution represents a complex mixture of pollutants, and the focus on PM2.5 and B(a)P does not capture the full spectrum of potential exposures. However, in the Slovak context, these pollutants are closely linked to residential combustion and represent the dominant exposure pathways. Other pollutants, such as NOx, may play an important role at the local scale, particularly in traffic influenced by urban environments. However, given the resolution and structure of national scale air quality modelling, these localized effects cannot be consistently captured at the municipality-level across the entire country.

Our reconstruction of historical exposure relied on a linear backward extrapolation of monitored concentrations to 1990. It is notable that even though the overall trend decreases, some stations report long-term increases of PM concentrations in summer, which can be attributed to cyclical climate change and resulting dry periods creating dry and dusty periods[28,58]. The assumption of linear extrapolation is methodologically common[25,59,60], but historically imperfect, as the emission trajectory of Slovakia (in supplementary material, chapter S4), and of most Central and Eastern European countries was strongly nonlinear around the political and economic transition after fall of socialism in 1989[61]. During the early 1990s, several major industrial sources rapidly declined or shut down, pollutant abatement technologies were progressively introduced, and residential heating shifted from coal to natural gas in large parts of the country[11]. These structural changes produced a rapid decrease in primary particulate and PAH emissions that far exceeded the gradual decline observed after the year 2000[62]. It is therefore plausible that ambient concentrations of PM2.5 and B(a)P declined more steeply between 1990 and 2000 than assumed in the linear extrapolation based solely on recent monitoring data.

The reconstruction of historical exposure trajectories represents an important source of uncertainty. In the absence of spatially resolved monitoring data prior to 2010, the study relies on a linear extrapolation of observed trends. Although the exact year-to-year evolution of concentrations may have differed locally, several lines of evidence support the plausibility of this approximation. First, independent exposure reconstructions suggest that PM2.5 concentrations in Slovakia during the early 1990s were typically below 30 μg/m³ (28,29), consistent with the baseline reconstruction used in this study. Second, sensitivity analyses indicate that alternative nonlinear scenarios frequently produce unrealistically high concentrations exceeding 35 μg/m³ on average. Finally, monitoring data show no statistically significant regional differences in long-term decline rates, suggesting that applying a single national trend represents a reasonable simplification of historical dynamics. The historical exposure reconstruction should be interpreted as an approximation of long-term exposure trends rather than an exact reconstruction of annual concentrations in individual past years, including year-specific meteorological effects.

The dynamics of historical air pollution change could also be spatially heterogeneous. Industrial restructuring and heating modernization progressed more rapidly in urban centers than in rural, solid fuel dependent regions[61,63]. As a result, historical exposure patterns likely differed slightly from the present ones[64]. Cities that today fall into the lower pollution quantiles may have experienced some of the highest exposures around 1990, whereas rural municipalities, now among the most polluted may have improved more slowly[50,65]. This temporal inversion could help explain why, in our regression model, lower pollution quantiles (dominated by today's urban or near urban areas) sometimes show greater ΔLE than expected from SCHIF. Their long-term cumulative exposure could be underestimated by the linear back projection. This spatial contrast is reflected in models in current patterns, with higher PM2.5 and B(a)P concentrations persisting in south-eastern regions dominated by residential solid fuel heating and weaker atmospheric dispersion, while lower levels prevail in southern lowlands with more effective ventilation. The close covariation of PM2.5 and B(a)P supports a shared origin in residential solid-fuel combustion, consistent with European receptor studies attributing the majority of B(a)P emissions to this source[66].

Collectively, these considerations highlight that health impact assessments based solely on present day exposure distributions omit an essential component of long-term cumulative risk[19]. Incorporating the nonlinear emission transitions of the post communist period is therefore crucial for accurate life expectancy attribution in Central and Eastern Europe. Linear extrapolation likely underestimates both true historical concentrations and the corresponding ΔLE gradient, particularly in the nonlinear region of the SCHIF curve, where modest changes in concentration produce disproportionately large mortality effects[4]. In addition, our exposure reconstruction treats population as spatially static, without explicitly modelling migration between high and low pollution municipalities over the life course. This may blur true individual exposure histories and contribute to uncertainty in the attribution of post 1990 LE gains to air-quality improvements. COVID

19 lockdowns affected short term pollution levels but had only a limited influence on annual mean PM2.5.

Several notable limitations need to be addressed. Residential solid-fuel combustion is among the most uncertain emission sectors in Central and Eastern Europe, as national inventories are based on incomplete information on household heating technologies, fuel quality, and user behavior. Local practices, including waste burning, are rarely documented and may contribute to remaining spatial inconsistencies, particularly in rural areas. Reconstruction of historical exposure is further limited by the scarcity of monitoring data before the late 1990s. As a result, the linear back extrapolation of PM2.5 and B(a)P concentrations could be biased by several μg/m^3, especially during the period of rapid economic and political change after 1989, however historical studies provide evidence that air pollution used in this study is well aligned. While sensitivity analyses indicate that our main conclusions are robust, more detailed historical emission inventories and chemically consistent analyses would substantially improve future assessments. Both the WHO HRAPIE and SCHIF exposure response functions are based largely on evidence from Western Europe and North America. Applying these relationships to Slovak conditions assumes broadly comparable particle toxicity, baseline health risks, and population susceptibility that are problematic. Differences in pollution mixtures, demographic structure, and healthcare access may influence the true shape and slope of the exposure response relationship. The regression analysis relies on administrative mortality data, which introduces additional uncertainty in small municipalities with limited populations. Individual municipal estimates should therefore be interpreted with caution, with greater confidence placed on aggregated patterns and gradients. Exposure misclassification is unavoidable. Because PM2.5 concentrations were used as one of the predictor variables in the B(a)P interpolation framework, part of the observed PM2.5 and B(a)P spatial correlation may reflect shared model structure in addition to genuine emission patterns. This approach may reduce sensitivity to municipalities with atypical pollutant composition, where the B(a)P fraction is relatively high despite only moderate PM2.5 concentrations, or vice versa. However, independent monitoring data still show strong covariation between measured annual mean PM2.5 and B(a)P concentrations, supporting that much of the observed spatial overlap reflects real emission patterns dominated by residential combustion. Also, air quality models smooth fine scale spatial variability, and available monitoring data cannot capture microenvironmental or indoor-outdoor exposure differences. Indoor air quality may also contribute to total exposure, particularly in households using solid fuels for heating. While cooking-related emissions from open combustion sources are relatively limited due to widespread gasification, indoor exposure may still vary with socioeconomic conditions and heating practices. Due to the lack of high-resolution data on indoor air quality and household energy use, this aspect could not be explicitly quantified and represents a limitation of the study. In addition, the use of single pollutant models limits attribution within complex combustion-related mixtures, while concurrent changes in healthcare, demographics, and behavior cannot be fully separated from air quality effects. Nevertheless, the consistency of results across mechanistic, statistical, and combined modelling approaches, particularly at higher exposure levels, supports the robustness of the main findings.

This study estimates municipality-level life-expectancy loss associated with long-term PM2.5 and B(a)P exposure in Slovakia, integrating air quality models, long-term monitoring, life table analysis, multivariable regression, and explicit fusion of multiple health impact frameworks. Across all approaches, life expectancy loss increases monotonically with exposure. Depending on the method, the mean national ΔLE associated with current pollution ranges from 0.72 years (SCHIF, 1990 baseline) to 1.20 years (WHO), while regression estimates imply ~1.17 years nationally and an average slope of 0.08 years per 1 μg/m^3, consistent with international evidence. At higher exposure levels (~Q15–Q20), except WHO, all methods converge, indicating a robust life expectancy deficit of about 1.8–2.2 years in the most polluted municipalities. Combining WHO, SCHIF, and regression based estimates through inverse-variance weighted fusion yields a smooth, coherent dose-response curve with relatively narrow uncertainty, representing a stable summary of health burden at high exposures. In cleaner municipalities, results are more divergent, WHO predicts comparatively large losses, SCHIF very small ones, and the regression model suggests modest but non negligible deficits, likely reflecting historical exposure and contextual factors. PM2.5 and B(a)P based regression results converge in the highest exposure quantiles, indicating a common pollution source, while modest differences at intermediate exposures suggest that PAH-related toxicity may account for part of the residual life-expectancy gradient. Overall, ambient air pollution explains an important, but partial, share of regional life expectancy differences in Slovakia. Effective health-gain strategies should therefore prioritize reductions in residential PM2.5 and PAH emissions while being integrated with broader social and healthcare policies. The fused exposure response curve provides a practical tool for translating heterogeneous evidence into policy relevant estimates of life expectancy gains across the exposure gradient. The results presented in this study should be interpreted as associational rather than causal, reflecting spatial relationships between long-term exposure and life expectancy across municipalities. Future research should aim to strengthen causal inference by integrating higher-resolution exposure data, individual-level health information, and quasi-experimental study designs.

Methods

Air quality exposure

Air pollution exposure was defined at the municipality-level using spatially averaged concentrations of PM2.5 and B(a)P derived from national air quality modelling calibrated to monitoring observations. B(a)P was used as a proxy indicator for polycyclic aromatic hydrocarbons (PAHs), which are not systematically monitored at high spatial resolution. For recent years (2020–2024), annual mean concentrations of PM2.5 and B(a)P were estimated using an air quality transport model which combines observation based regression interpolation techniques with chemical transport model information. In this framework, the model RIO and the IDW-R post-processing procedure are used to derive final concentration fields, while outputs from the chemical transport model CMAQ (v5.3) are incorporated as one of several spatial predictor fields describing regional pollutant distributions. This modelling approach reflects current best practice in European air quality assessment[22]. As air quality monitoring stations are not available in every municipality, modelled concentrations provide the most consistent approach for capturing fine spatial differences in exposure at the national level[21,67]. For B(a)P mapping, PM2.5 concentration fields were used as one of the predictor variables in interpolation, meaning that part of the observed PM2.5 and B(a)P spatial correlation may reflect shared model structure in addition to common emission sources. To represent population exposure within municipalities, residential address data were used to spatially aggregate modelled concentrations around inhabited areas. Pollution concentrations were assigned to each municipality using a spatial overlay between the modelled concentration field and a 400 m residential buffer around inhabited areas within each municipality. Sensitivity analyses using alternative buffer sizes (200–2000 m) were carried out and yielded consistent results.

Historical air pollution trends were characterized using validated monitoring data from the national air quality network and the EEA Air Quality Data Service[3]. Continuous PM2.5 measurements have been available in Slovakia since 2010, while B(a)P monitoring began in 2017. Earlier air-quality information was limited to sparse monitoring observations and model-based reconstructions. According to SHMÚ, remote-sensing data are not sufficiently reliable for reconstructing historical air pollution in Slovakia. Annual mean concentrations were calculated from raw one hourly averaged data for each station, and long-term linear trends were estimated to characterize average rates of change. This approach is consistent with previous studies reporting predominantly linear long-term behavior with annual variability driven mainly by meteorological conditions[63,68–71].

To align exposure with the temporal coverage of available health data, historical exposure trajectories were approximated by projecting linear

trend derived from monitoring backward from recent modelled concentrations to earlier periods (up to 2008 and 1990). Linear approach reflects limitations in historical reconstruction prior to widespread monitoring[23,26] and the absence of evidence for rapid, non-linear changes in air pollution during the 1990s[59,72], when many political, industrial and socio-economic changes happened in Slovakia after fall of socialism in 1989. Importantly, the regression statistical model in this study estimates between municipality differences in exposure rather than absolute historical levels. Therefore, results are robust to assumptions about the precise shape of long-term trends. Accordingly, the main exposure inputs to the statistical analysis were municipality-level average PM2.5 and B(a)P concentrations derived from air quality models for the period 2020–2024. The robustness of this linear backwards assumption was evaluated using several alternative exposure reconstruction scenarios with details on model configuration, post processing, and validation together with detailed description of monitoring stations data and analysis are provided in the supplementary material, chapter S2.

Statistical model—inputs and functionality

The statistical model was constructed to quantify the contribution of air pollution to life expectancy while accounting for a wide range of socioeconomic and healthcare related factors as covariates. Covariate selection was guided by the aim to capture major structural determinants of spatial variation in life expectancy in Slovakia. Educational attainment was included as a primary indicator of socioeconomic status due to its strong and consistent association with health outcomes. The proportion of the Roma population was incorporated as a contextual variable reflecting a well-documented dimension of structural disadvantage not fully captured by conventional socioeconomic indicators. Roma populations in Central and Eastern Europe experience substantially poorer health outcomes and shorter life expectancy compared with the majority population, largely driven lack of employment options and de facto segregation, reduced access to healthcare, and adverse living conditions[73,74]. Healthcare accessibility variables were included based on ongoing analyses within the Ministry of Health, which indicate that treatment adherence and continuity of care are influenced by access barriers, and prior evidence showing that longer travel time to hospital is associated with worse chronic disease outcomes. Median income was considered but excluded from the final specification, as its effect was not significant after adjustment for other covariates, while education remained a more stable and explanatory predictor. Exposure to PM2.5 and B(a)P was represented using 20 exposure quantiles (Q1-Q20), with the lowest quantile serving as the reference category. This approach allows the exposure response relationship to vary flexibly across the exposure distribution, without imposing linearity, and accommodates the possibility of threshold effects or non-linear amplification at higher concentrations, as suggested by toxicological evidence for fine particulate matter and polycyclic aromatic hydrocarbons. Additional pollutants (e.g., NOx and SO2) were explored in preliminary analyses but did not show a robust relationship with life expectancy at the municipality level. Therefore, the analysis focused on PM2.5 and B(a)P, which represent the dominant exposure pathways in Slovakia. All covariates were harmonized at the municipality-level and included in the model as categorical variables to reduce sensitivity to extreme values and to avoid assumptions about functional form. The proportion of the Roma population was grouped into 10 percentage point categories, while educational attainment, drinking water hardness, emergency medical service (EMS) response time, and distance to the nearest hospital were classified into quantiles. This categorization mirrors the exposure quantile structure and allows each determinant to contribute independently and potentially non-linearly to differences in life expectancy. Model parameters were estimated using ordinary least squares. Alternative model specifications using continuous predictors or spline terms were explored and produced highly consistent results. Multicollinearity was assessed using variance inflation factors, which remained well below conventional thresholds for all variables. Under this specification, the model estimates the expected difference in life expectancy at birth associated with belonging to each PM2.5 or B(a)P exposure quantile after adjustment for the full set of covariates. The regression, therefore, captures long-term differences between municipalities in pollution exposure, rather than temporal trends, which is consistent with the study's focus on cross-sectional disparities in life expectancy attributable to spatially patterned air pollution. All analyses were conducted in Python (v3.10) using the statsmodels package (v0.14.2). Sensitivity analyses confirmed that results were robust to alternative exposure coding and to the exclusion of municipalities with small populations.

All input variables were assembled at the municipality level. Life expectancy at birth was derived from complete life tables constructed from official population and mortality data available from 2008 onwards, provided by the Statistical Office of the Slovak Republic (details in supplementary material, chapter S2). Educational attainment was obtained from the same source. Average EMS response times were calculated from event level data supplied by the EMS Command and Control Centre of the Slovak Republic, and travel time to the nearest hospital was derived from municipality to hospital travel matrices compiled by the Institute of Healthcare Analyses at the Ministry of Health. Estimates of the Roma population were taken from the Slovak Atlas of Roma Communities, while drinking water hardness was obtained from the Water Research Institute. Low concentrations of calcium and magnesium in drinking water have previously been associated with increased cardiovascular and oncological mortality in Slovakia[75]. This is supported by physiological evidence from a regional study in the Krupina district, where higher water hardness was associated with greater arterial elasticity, consistent with a potential cardiovascular benefit[76]. Because water hardness is strongly determined by underlying geology and correlates with rurality and infrastructure, it may also reflect broader environmental and socioeconomic gradients. Its inclusion, therefore, helps reduce residual confounding in the estimation of air pollution effects, while we recognize that it may partly act as a proxy for unmeasured contextual factors rather than a direct causal determinant. On this basis, water hardness was included in the model as a concentration of calcium and magnesium and turned into quintile categorical covariate. Meteorological conditions were not included as separate covariates in the life-expectancy model because the exposure variable was defined directly from ambient concentration fields and historical reconstruction, which already incorporate the combined effects of emissions, dispersion and meteorological variability.

Model comparison

To translate long-term PM2.5 exposure into changes in life expectancy (ΔLE), we applied two established health-impact frameworks: the WHO HRAPIE log-linear approach[4] and the non-linear saturation concentration hybrid impact function (SCHIF)[18–20]. For both HRAPIE and SCHIF applications, concentrations entered the exposure response functions were defined as municipality-level average exposures over two periods: 2008–2024 and 1990–2024. In both approaches, changes in PM2.5 exposure were converted into age specific mortality adjustments from age 30 years onwards and propagated through period life tables to estimate ΔLE under counterfactual exposure scenarios. HRAPIE assumes a log-linear exposure response relationship without an explicit minimum risk threshold and is commonly interpreted as a steady state estimate of life expectancy gains under reduced current pollution levels[5,20]. SCHIF allows for non-linearity and low concentration cut offs, achieving weak effects at low concentrations and steeper gradients at higher exposure levels[19]. Coefficients of SCHIF function were taken from[19]. For comparability reason, the counterfactual concentration was set to 5 μg/m³ in both frameworks. We did not apply outcome specific inception or cessation lags. ΔLE estimates, therefore, represent steady-state differences between baseline mortality and mortality under a counterfactual PM2.5 concentration of 5 μg/m³, rather than retrospective attributions of historical exposure. Uncertainty was propagated using reported confidence intervals for the respective exposure response parameters. Full details and equations are provided in the supplementary material, chapter S2. To synthesize results from multiple epidemiological frameworks, we applied inverse-variance weighting (IVW). This method

assigns greater weight to estimates with smaller statistical uncertainty, allowing results derived from different risk modelling approaches to be combined while accounting for differences in their confidence intervals. Because the estimates share common exposure fields and demographic inputs, their uncertainties are likely to be partially correlated. Consequently, the IVW estimate should be interpreted primarily as a synthesis of central estimates rather than a fully independent uncertainty propagation.

Data availability

Aggregated municipality level data used in this study are derived from publicly available sources or from institutional databases subject to data protection and use agreements. Due to legal and ethical restrictions, individual level mortality and healthcare data cannot be shared publicly.

Code availability

Code used for data processing and analysis is available from the corresponding author upon reasonable request.

Received: 30 January 2026; Accepted: 15 May 2026;
Published online: 16 June 2026

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Acknowledgements

The authors thank the Slovak Hydrometeorological Institute and the Statistical Office of the Slovak Republic for providing air quality and mortality

data, and the Institute for Healthcare Analyses for access to healthcare accessibility indicators. Institutional and project based support that contributed to personnel time is gratefully acknowledged. This publication was supported by the project "Innovative Technologies for Smart Low Emission Mobilities", funded as project No. CZ.02.01.01/00/23_020/0008528 by Programme Johannes Amos Comenius, call Intersectoral cooperation. During the preparation of this work, the authors used ChatGPT (OpenAI) in order to assist with shortening selected sections of the text, improving readability, and restructuring the manuscript. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Author contributions

Imrich Berta and Jakub Linda contributed equally to this work and share first authorship. They conceived the study, curated and integrated datasets, interpreted the results, created visualizations, and drafted the manuscript. Jakub Linda implemented the WHO HRAPIE and SCHIF life table methods and performed the corresponding life expectancy calculations. Imrich Berta developed and implemented the multivariable regression model. Dušan Štefánik was responsible for air quality modelling and exposure assessment. Veronika Rybanská and Michaela Mikulová contributed to data collection, data curation, and interpretation of results. Alexandra Bražinová contributed to study supervision, interpretation of findings, and critical revision of the manuscript. Imrich Berta and Jakub Linda directly accessed, verified, and validated the underlying data and model outputs. All authors reviewed and approved the final version of the manuscript.

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s44407-026-00085-2.

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