



**NEW SMR POWER PLANT
AT THE TEMELÍN SITE**

**ASSESSMENT OF THE EFFECTS OF AIR
POLLUTION AND NOISE EXPOSURE ON
PUBLIC HEALTH**

JUNE 2026

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Assessment of the Effects of Air Pollution and Noise Exposure on Public Health

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INTRODUCTION

The objective of this study is to assess the effects of technological and transport activities during the implementation and operation of the “New SMR Power Plant at the Temelín site” project on public health resulting from exposure to chemical substances in ambient air and to noise.

The project involves the construction of a New SMR Power Plant at the Temelín site, including all associated structures and systems (technological equipment) used for electricity generation and power evacuation and to ensure the safe operation of the nuclear facility.

Based on the results of the supporting air dispersion study [1], the effects of exposure to chemical substances in ambient air are assessed for the following project implementation scenarios:

Scenario 1: 2034 – effects of SMR ETE construction, with the maximum potential cumulative effects arising from the possible concurrent construction of New NPP ETE and SMR ETE (expected duration of up to 1 year)

- No-project scenario 1 = 2034 + initial phase of New NPP ETE preparation
- Project scenario 1 = 2034 + initial phase of New NPP ETE preparation + SMR ETE construction phase

Scenario 2: 2040 – effects of SMR ETE operation, a temporary scenario involving the possible concurrent operation of SMR ETE and the most traffic-intensive stage of New NPP ETE construction (expected duration of up to 1 year)

- No-project scenario 2 = 2040 + main phase of New NPP ETE construction
- Project scenario 2 = 2040 + main phase of New NPP ETE construction + SMR ETE operation

Scenario 3: 2042 – effects of SMR ETE operation, combined operation of New NPP ETE and SMR ETE (permanent scenario)

- No-project scenario 3 = 2042 + New NPP ETE operation
- Project scenario 3 = 2042 + New NPP ETE operation + SMR ETE operation

In accordance with the supporting noise studies, the effects of noise exposure are assessed for the following scenarios:

Future scenario (2034) – traffic volumes in 2034

- Operation of ETE1,2 without SMR ETE operation
- Operation of ETE1,2 with SMR ETE operation

Future scenario (2034) – stationary noise sources

- Operation of ETE1,2 with SMR ETE operation

Target scenario (2042) – traffic volumes in 2042

- Operation of ETE1,2 and New NPP ETE without SMR ETE operation
- Operation of ETE1,2, New NPP ETE, and SMR ETE

Target scenario (2042) – stationary noise sources

- Operation of ETE1,2, New NPP ETE, and SMR ETE

This assessment only considers effects occurring during normal operation – its results cannot be applied to special situations, including accidents.

1. ASSESSMENT METHODOLOGY

The assessment methodology is based on the fundamental Health Risk Assessment procedures developed by the U.S. Environmental Protection Agency (US EPA) and uses the authorisation guidelines of the National Institute of Public Health (hereinafter “NIPH”) for the assessment of health risks from exposure to chemical substances in ambient air, AN 17/15 [2], the assessment of health risks from noise exposure [26], and the scientific literature [4]. The health risk assessment procedure consists of four consecutive steps:

- **Hazard identification** – this involves identifying the factors to be assessed, describing their properties with a focus on their hazards to humans, and determining the conditions under which these hazards may arise.
- **Dose–response assessment** – this step quantitatively assesses the relationship between the level of exposure to a given factor (a substance in ambient air) and the level of risk.
- **Exposure assessment** – this includes a qualitative description of contact between the factor under assessment and the outer boundary of the body and a quantitative description of the intensity of that contact. The objective is to determine the pathways through which, the extent to which, and the quantities in which a specific population is exposed to the chemical substance or other factor under assessment, such as noise.
- **Risk characterisation** – this stage expresses the level of health risk to the exposed population based on knowledge of the hazards posed by the relevant factor and an estimate of the specific level of exposure. It provides a qualitative and quantitative description of the estimated health risk to the population under assessment, i.e. a list of all potential adverse health effects in that population and the probability of their occurrence. All baseline conditions and facts included in the risk assessment procedure must be described, as must all simplifications and uncertainties reflected in the assessment. Risks assessed in this manner must always be regarded as potential, but sufficiently probable for the population in the area of interest.

In accordance with Authorisation Guideline AN 17/15, the assessment is divided into the following sections:

- data for assessing population exposure, including identification of the pollutants under assessment and data for determining background concentrations,
- characteristics of residential areas in the vicinity of the project,
- hazard identification and dose–response assessment,
- exposure assessment and risk characterisation,
- assessment uncertainties,
- conclusion.

2. DATA FOR EXPOSURE ASSESSMENT

The assessment of the effects of air pollution and noise exposure associated with project implementation on public health is based on the air dispersion study [1] and the noise studies [23, 24]. These studies therefore constitute the primary and sole basis for assessing population exposure.

The air dispersion study [1] and, subsequently, this assessment consider the concentrations of the following pollutants: PM₁₀ and PM_{2.5} particulate matter, nitrogen dioxide, benzene, and benzo[a]pyrene. The combined effects of PM_{2.5} and nitrogen dioxide are also assessed. The selection of pollutants thus follows the recommendations of NIPH Authorisation Guideline [2] for assessing the impacts of traffic.

The supporting air dispersion study assesses air pollution using model calculations for three time horizons and also quantifies the contributions of transport and stationary sources to total pollutant concentrations. The overall level of air pollution was assessed using the five-year averages evaluated in the air dispersion study, together with data from automated air quality monitoring (AIM) stations.

A vector layer of existing residential areas was created to quantify the effects of air pollution. The number of residents in individual buildings was derived from cadastral data and the ratio of residents per dwelling in the relevant BSUs within the modelled area.

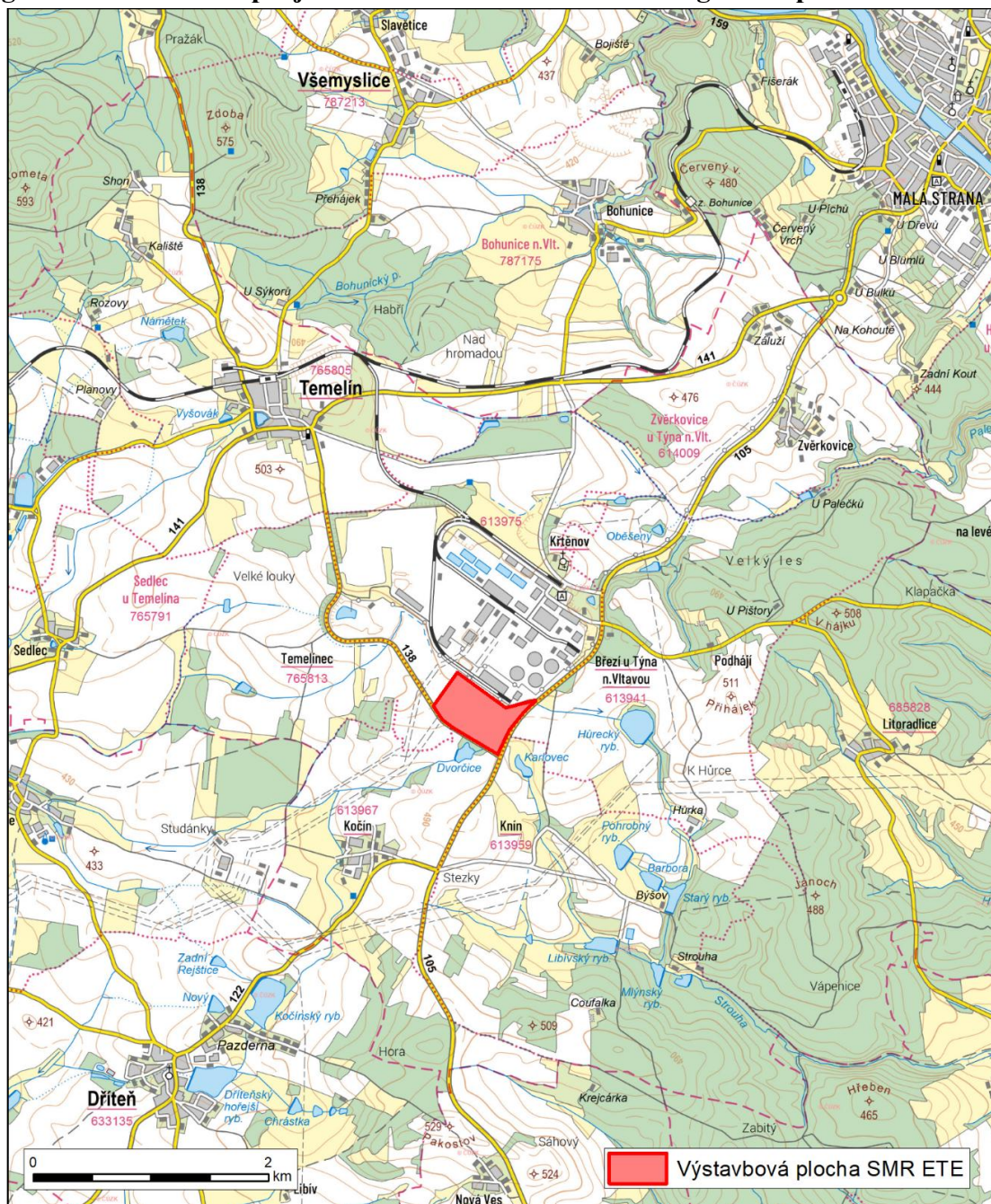
The noise situation is assessed at individual receptor points representing the affected residential areas.

Road traffic and stationary sources associated with the project under assessment were considered as noise sources. Noise is assessed separately for daytime and night-time.

3. CHARACTERISTICS OF RESIDENTIAL AREAS IN THE VICINITY OF THE PROJECT

The project under assessment is located on flat terrain approximately 4 km south-west of Týn nad Vltavou and approximately 20 km north-west of České Budějovice. The nearest residential area is in the municipality of Temelín and its constituent parts of Sedlec, Kočín, and Litoradlice, which are located several kilometres from the project.

Fig. 1. Location of the project in relation to the surrounding development



Výstavbová plocha SMR ETE

SMR ETE construction area

A vector layer of existing residential areas was created for the quantitative assessment of the effects of exposure to chemical substances in ambient air on public health. The number of residents in individual buildings was derived from cadastral data and the ratio of residents per dwelling in the relevant BSUs. The assessment covers an area corresponding to the extent of the model calculations in the supporting air dispersion study [1]. The population within the defined section of the existing surrounding residential areas is 12,650.

Using GIS tools, the population data from the vector layer were overlaid on the pollutant concentration fields for the individual air quality characteristics and aggregated by calculated concentration bands.

For the quantitative assessment of noise exposure, estimated numbers of affected residents were assigned to individual receptor points based on the aforementioned vector layer. These estimates generally cover not only the building represented by the respective point but also other nearby buildings where a similar level of exposure may be assumed.

4. EFFECTS OF AIR POLLUTION ON PUBLIC HEALTH

4.1. Hazard identification and dose–response assessment

4.1.1. Particulate matter

Particulate matter in ambient air is a complex mixture of organic and inorganic substances whose components have diverse chemical and physical properties. It is generated in both outdoor and indoor environments. It is therefore an important factor contributing to adverse health effects.

Particulate matter varies in size, mass, and composition. In general, it can be stated that:

- when solid fuels are burned without particulate removal equipment, particles with an aerodynamic diameter greater than 10 μm predominate in the emissions; when liquid fuels are burned, the proportion of such particles is lower, but still significant. As the efficiency of the particulate removal equipment increases, the proportion of “coarser fractions” decreases significantly, since such equipment is most effective in removing large dust particles.
- Resuspended dust in the vicinity of roads and industrial sites can generally be expected to contain a low proportion of fine particles; however, the proportion of the individual size fractions depends on the composition of the originally deposited particles.
- Fine particles up to 2.5 μm clearly predominate in motor vehicle exhaust emissions (accounting for approximately 90% of the particles); most of the emitted particles are smaller than 1 μm .
- Similarly, the vast majority of aerosols formed secondarily in the atmosphere through the condensation of gaseous substances consist predominantly of fine particles up to 2.5 μm [5].

Different characteristics of particulate matter may be associated with different health effects, depending on particle size, physical characteristics, and chemical composition. Epidemiological indicators of mortality and morbidity are therefore used for general indicative assessment.

In 2021, the World Health Organization (WHO) issued new air quality guidelines [6], which largely supersede the previous guidelines issued in 2005 [5]. According to WHO [6], exposure to particulate matter increases the risk of mortality from the following diseases:

- long-term $\text{PM}_{2.5}$ concentrations – with high certainty for diseases of the circulatory system (particularly ischaemic heart disease) and lung cancer, and with moderate certainty for non-malignant respiratory diseases;
- long-term PM_{10} concentrations – with high certainty for non-malignant respiratory diseases and lung cancer, and with moderate certainty for ischaemic heart disease;

- short-term PM_{10} and $PM_{2.5}$ concentrations – with high certainty for cardiovascular diseases and with moderate certainty for cerebrovascular diseases and non-malignant respiratory diseases.

The effect of long-term particulate matter concentrations on the incidence of cardiovascular disease is generally more consistent for the $PM_{2.5}$ fraction than for PM_{10} . Similarly, a significantly increased risk of stroke has been demonstrated for $PM_{2.5}$, but not for PM_{10} . Further findings indicate a causal relationship between exposure to $PM_{2.5}$ pollution and acute lower respiratory tract infections, chronic obstructive pulmonary disease, type 2 diabetes, and neonatal mortality due to low birth weight and preterm birth. The relationship between particulate matter and the incidence of lung cancer has also been studied extensively, and the risk of death from this type of cancer was found to be significantly associated with both $PM_{2.5}$ and PM_{10} pollution [6]. In 2015, particulate matter was classified by the WHO International Agency for Research on Cancer (IARC) [7] as carcinogenic to humans.

For short-term exposure, WHO reports a 0.65% increase in all-cause mortality for each $10 \mu\text{g}\cdot\text{m}^{-3}$ increase in the 24-hour mean $PM_{2.5}$ concentration. For chronic exposure, an 8% increase in mortality is reported for each $10 \mu\text{g}\cdot\text{m}^{-3}$ increase in the annual mean $PM_{2.5}$ concentration; for PM_{10} , the corresponding increase in mortality is 4% for each $10 \mu\text{g}\cdot\text{m}^{-3}$ increase in the annual mean PM_{10} concentration.

Over the past several decades, particulate matter pollution levels have declined in developed countries, making it possible to investigate health effects in greater detail even at lower concentration levels. For annual mean $PM_{2.5}$ concentrations, an association between exposure and mortality has been demonstrated even below $10 \mu\text{g}\cdot\text{m}^{-3}$, down to very low exposure levels; moreover, a steeper (supralinear) increase in risk has been demonstrated at lower exposure levels. Adverse health effects were observed even in the lowest percentiles of measured values. For this reason, WHO selected the 5th percentile of the values measured in the underlying studies as the baseline level for determining the guideline values; this corresponds to $4.2\text{--}4.9 \mu\text{g}\cdot\text{m}^{-3}$ for $PM_{2.5}$ and $15.1 \mu\text{g}\cdot\text{m}^{-3}$ for PM_{10} . In addition to evidence of demonstrated health effects, the guideline values for short-term (24-hour) concentrations were also established on the basis of the relationship between 24-hour concentrations and their annual means.

WHO [6] therefore specifies the following guideline values for particulate matter:

- $PM_{2.5}$ – $5 \mu\text{g}\cdot\text{m}^{-3}$ for annual mean concentrations and $15 \mu\text{g}\cdot\text{m}^{-3}$ for 24-hour mean concentrations;
- PM_{10} – $15 \mu\text{g}\cdot\text{m}^{-3}$ for annual mean concentrations and $45 \mu\text{g}\cdot\text{m}^{-3}$ for 24-hour mean concentrations.

WHO also establishes four interim targets for each of the above parameters. The previously applicable guideline values according to [5] – i.e. 10 and $20 \mu\text{g}\cdot\text{m}^{-3}$ for annual

mean PM_{2.5} and PM₁₀ concentrations, respectively, and 25 and 50 µg·m⁻³ for their 24-hour mean concentrations – now correspond to interim target 4. Interim targets are generally established for areas with higher air pollution levels, with the aim of progressively achieving the final guideline values within a realistic timeframe.

The currently applicable air quality limit values in the Czech Republic for PM₁₀ are 40 µg·m⁻³ for the annual mean concentration and 50 µg·m⁻³ for the 24-hour mean concentration (with 35 exceedances permitted per year). For PM_{2.5}, only an annual mean limit value is currently established, at 20 µg·m⁻³.

From 2030, new air quality limit values are expected to apply, amounting to 20 µg·m⁻³ for annual mean PM₁₀ concentrations and 45 µg·m⁻³ for daily mean concentrations (with 18 exceedances permitted per year). For PM_{2.5}, the air quality limit values will be 10 µg·m⁻³ for the annual mean concentration and 25 µg·m⁻³ for the daily mean concentration (with 18 exceedances permitted per year).

To quantify the risk from exposure to particulate matter (and similarly from exposure to nitrogen dioxide, see below), this assessment uses concentration–response functions published by the World Health Organization as part of the *Health Risks of Air Pollution in Europe: HRAPIE-2* project (HRAPIE-2) [8] in 2025. This document updates the previous guidance issued in 2013 [9].

These relationships were derived from comprehensive systematic reviews and meta-analyses of numerous epidemiological studies and health indicator data from global datasets. The individual concentration–response factors are expressed in terms of relative risk (RR). In this context, relative risk expresses the change in the risk of occurrence of a particular health effect in the population for a specified increase in pollutant concentration.

The relationship between concentration and risk is log-linear within the recommended exposure ranges. The attributable fraction method described in Section 4.2 is then used to characterise the risk to the exposed population.

The relationships are divided into three groups:

- Group A – sufficient data are available for reliable quantification of effects;
- Group B+ – the data are subject to greater uncertainty regarding the accuracy of the information used to quantify the effects than Group A data;
- Group B- – data that cannot be recommended for health risk assessment because the strength of the evidence is insufficient.

To ensure the highest possible accuracy of the results, the analyses included Group A relationships, i.e. those for which sufficient data are available for reliable quantification of effects. Group B+ relationships were also included in the analyses. In some cases, “alternative” relationships are provided in addition to the “main” calculation relationships and may be used in specific situations (e.g. where insufficient data are

available to perform the calculation using the preceding relationship). Tables 1 and 2 provide an overview of the relative risk values used in this study; in all cases, these are the “main” RR values. The confidence interval, shown in parentheses, and the central estimate of relative risk are provided in each case.

Table 1. Concentration–response factors – particulate matter – Group A relationships [8]

Air quality characteristic	Health effect	Population segment	RR per increase in concentration of 10 $\mu\text{g}\cdot\text{m}^{-3}$
PM _{2.5} annual mean	adult natural-cause mortality*	≥ 25 years	1.100 (1.060–1.130)
PM _{2.5} annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	1.130 (1.100–1.150)
PM _{2.5} annual mean	adult mortality – cerebrovascular diseases	≥ 25 years	1.150 (1.100–1.190)
PM _{2.5} annual mean	adult mortality – ischaemic heart disease	≥ 25 years	1.140 (1.100–1.190)
PM _{2.5} annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.140 (1.080–1.200)
PM _{2.5} annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	1.140 (1.080–1.200)
PM _{2.5} annual mean	adult mortality – lower respiratory tract infections	≥ 25 years	1.200 (1.100–1.330)
PM _{2.5} annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	1.090 (1.050–1.140)
PM _{2.5} annual mean	morbidity – childhood asthma	0–18 years	1.340 (1.100–1.630)
PM _{2.5} annual mean	morbidity – chronic obstructive pulmonary disease (COPD)	≥ 30 years	1.180 (1.130–1.230)
PM _{2.5} annual mean	morbidity – ischaemic heart disease (myocardial infarction)	≥ 30 years	1.130 (1.050–1.220)
PM _{2.5} annual mean	morbidity – stroke	≥ 30 years	1.160 (1.120–1.200)
PM _{2.5} annual mean	morbidity – high blood pressure	≥ 30 years	1.170 (1.050–1.300)
PM _{2.5} annual mean	morbidity – malignant neoplasm of the bronchus and lung	≥ 30 years	1.160 (1.100–1.230)
PM _{2.5} daily mean**	adult natural-cause mortality*	entire population	1.0068 (1.0059–1.0077)
PM _{2.5} daily mean**	morbidity – respiratory diseases	entire population	1.0090 (1.0026–1.0153)
PM ₁₀ annual mean	adult natural-cause mortality*	≥ 25 years	1.080 (1.050–1.110)
PM ₁₀ annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	1.080 (1.040–1.120)
PM ₁₀ annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.120 (1.080–1.170)
PM ₁₀ annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	1.100 (1.050–1.150)

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

**) Values based on daily means will only be used to determine the effects of short-term exposure during construction works.

Table 2. Concentration–response factors – particulate matter – Group B+ relationships [8]

Air quality characteristic	Health effect	Population segment	RR per increase in concentration of 10 $\mu\text{g}\cdot\text{m}^{-3}$
PM ₁₀ annual mean	adult mortality – ischaemic heart disease	≥ 25 years	1.060 (1.020–1.090)
PM ₁₀ annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	1.220 (1.030–1.440)
PM ₁₀ daily mean*	days of school missed	5–14 years	1.0149 (1.0017–1.0283)
PM _{2.5} annual mean	morbidity – type 2 diabetes	0–18 years	1.100 (1.030–1.180)
PM _{2.5} annual mean	morbidity – dementia	≥ 19 years	1.170 (1.100–1.250)
PM _{2.5} annual mean	morbidity – autism spectrum disorders	0–12 years	1.660 (1.230–2.250)

*) Values based on daily means will only be used to determine the effects of short-term exposure during construction works.

4.1.2. Nitrogen dioxide

Nitrogen dioxide (NO₂) is one of the pollutants most commonly monitored when assessing the effects of combustion sources (i.e. particularly motor vehicle traffic and the heating of buildings) on air quality and public health. These sources predominantly emit nitric oxide (NO), which is gradually oxidised in the air to NO₂; only a small proportion of NO₂ is emitted directly.

When nitrogen dioxide enters the respiratory tract, the most sensitive areas are the trachea and bronchi, followed by the pulmonary alveoli, where type I alveolar epithelial cells are replaced by cells more resistant to oxidation, which also gradually undergo hypertrophy as the NO₂ concentration increases. This reduces the resistance of lung tissue to infection.

According to WHO [6], exposure to nitrogen dioxide increases the risk of mortality from the following diseases:

- long-term NO₂ concentrations – with high certainty for chronic obstructive pulmonary disease, and with moderate certainty for non-malignant respiratory diseases and acute lower respiratory tract infections, including mortality in children;
- short-term (24-hour) NO₂ concentrations – with high certainty for all-cause mortality (excluding injuries) and also for hospital admissions due to asthma.

A meta-analysis conducted by WHO [6] demonstrated a relationship between long-term exposure to NO₂ and all-cause mortality (excluding injuries), as well as

mortality from various causes, even at the lowest exposure levels, with indications of a steeper increase in risk at lower concentrations. As in the case of particulate matter, the baseline level for determining the guideline value was therefore set at the 5th percentile of the values measured in the underlying studies, whose mean was $8.8 \mu\text{g}\cdot\text{m}^{-3}$. Based on the results of this analysis, a guideline value of $10 \mu\text{g}\cdot\text{m}^{-3}$ was established.

The previously applicable guideline value of $40 \mu\text{g}\cdot\text{m}^{-3}$ according to [5] became the first interim target, and two further interim targets of 30 and $20 \mu\text{g}\cdot\text{m}^{-3}$ were established to bridge the gap between this value and the guideline value.

With regard to short-term exposure to NO_2 , WHO states that, for hourly concentrations, the recommendation in the previous guidelines [5], which specifies a guideline concentration of $200 \mu\text{g}\cdot\text{m}^{-3}$, remains applicable. No effects of short-term exposure to NO_2 have been demonstrated below this level, while most studies indicate that health effects occur only at concentrations above $500 \mu\text{g}\cdot\text{m}^{-3}$. Conversely, at higher concentrations, the effects can be regarded as demonstrated.

The current guidelines [6] also address 24-hour NO_2 concentrations in detail, again finding sufficient evidence of an association with all-cause mortality even at very low exposure levels. As in the case of particulate matter, the guideline value for 24-hour NO_2 concentrations was derived taking into account the relationship between 24-hour and annual values and was set at $25 \mu\text{g}\cdot\text{m}^{-3}$.

The currently applicable air quality limit values in the Czech Republic are $40 \mu\text{g}\cdot\text{m}^{-3}$ for annual mean concentrations and $200 \mu\text{g}\cdot\text{m}^{-3}$ for hourly concentrations (with 18 exceedances permitted per year).

From 2030, new air quality limit values are expected to apply: $20 \mu\text{g}\cdot\text{m}^{-3}$ for annual concentrations, $50 \mu\text{g}\cdot\text{m}^{-3}$ for daily concentrations (with 18 exceedances permitted per year), and $200 \mu\text{g}\cdot\text{m}^{-3}$ for hourly concentrations (with 3 exceedances permitted per year).

The HRAPIE-2 project [8] also provides the following relative risk values for the individual effects of long-term exposure to NO_2 . The characteristics of the values and the data source used are described in the preceding section.

Table 3. Concentration–response factors – nitrogen dioxide [8]

Air quality characteristic	Health effect	Population segment	Group	RR per increase in concentration of 10 $\mu\text{g}\cdot\text{m}^{-3}$
NO ₂ annual mean	adult natural-cause mortality*	≥ 25 years	A	1.050 (1.030–1.070)
NO ₂ annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	A	1.050 (1.030–1.070)
NO ₂ annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	A	1.040 (1.020–1.060)
NO ₂ annual mean	adult mortality – lower respiratory tract infections	≥ 25 years	A	1.080 (1.040–1.120)
NO ₂ annual mean	morbidity – childhood asthma	0–18 years	A	1.100 (1.050–1.180)
NO ₂ annual mean	morbidity – asthma in adults	≥ 19 years	A	1.100 (1.010–1.210)
NO ₂ annual mean	morbidity – lower respiratory tract infections in children	0–12 years	A	1.090 (1.030–1.160)
NO ₂ annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	B+	1.05 (1.0300–1.0800)
NO ₂ annual mean	adult mortality – ischaemic heart disease	≥ 25 years	B+	1.05 (1.0300–1.0800)
NO ₂ annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	B+	1.07 (1.04–1.10)
NO ₂ daily mean**	adult natural-cause mortality*	entire population	A	1.0046 (1.0036–1.0057)
NO ₂ daily mean**	morbidity – respiratory diseases	entire population	A	1.0057 (1.0033–1.0082)

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

**) Values based on daily means will only be used to determine the effects of short-term exposure during construction works.

4.1.3. Combined effects of PM_{2.5} and NO₂

To assess the effects of project construction and operation on public health, it is necessary to consider the combined effects of PM_{2.5} and NO₂. In real-world environments, particularly in areas affected by motor vehicle traffic and emissions from combustion processes, residents are not exposed to individual pollutants in isolation.

Quantifying this combined effect is a crucial step in the health risk assessment process. Since PM_{2.5} and NO₂ largely share the same emission sources (e.g. exhaust emissions), their ambient air concentrations are very strongly correlated.

As stated in the preceding sections, NO₂ acts as a strong oxidant in the respiratory tract, reducing the resistance of lung tissue to infection. PM_{2.5} subsequently penetrates

the weakened respiratory system more easily and in greater quantities. This results in higher mortality, particularly from respiratory diseases [10].

According to the HRAPIE-2 project report [8], the following principles must be observed to ensure methodological accuracy of the assessment:

- To prevent double counting of the effects of strongly correlated pollutants, the individual values relating to PM_{2.5} and NO₂ must not simply be added together when calculating the overall health impacts.
- The health impacts of PM_{2.5} and NO₂ must be presented separately.
- Adjusted RR values reflecting the correlation between the two pollutants must be used to determine the cumulative value.

The following table presents the adjusted RR values used to calculate the combined effect of PM_{2.5} and NO₂. For an increase in the PM_{2.5} concentration of 5 µg·m⁻³ and in the NO₂ concentration of 20 µg·m⁻³, the estimated combined health impact is approximately 30.3% lower than the sum of the single-pollutant models.

Table 4. Combined effect of PM_{2.5} and NO₂ – adjusted RR values [8]

Air quality characteristic	Health effect	Population segment	RR	Per concentration increase of
PM _{2.5} annual mean	adult natural-cause mortality*	≥ 25 years	1.0304	5 µg·m ⁻³
NO ₂ annual mean	adult natural-cause mortality*	≥ 25 years	1.0762	20 µg·m ⁻³

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

4.1.4. Benzene

Benzene is released into the atmosphere in emissions from motor vehicle traffic both as a combustion product and as a component of unburned fuel (it is present in motor petrol at approximately 0.5–2.0%, while its proportion in diesel fuel is negligible). Ambient air is the main source of human exposure to benzene. However, significant individual differences resulting from smoking must be taken into account, as smoking may increase exposure severalfold.

At high concentrations (which do not occur in ambient air), benzene has acute irritant and neurotoxic effects. At low doses (which may occur in ambient air), long-term exposure suppresses blood cell production and is also believed to contribute to the initiation of leukaemia. For this reason, both the US EPA and IARC classify benzene as carcinogenic to humans. The World Health Organization specifies a unit cancer risk value for benzene of $UCR = 6 \times 10^{-6} (\mu\text{g}\cdot\text{m}^{-3})^{-1}$. Simple extrapolation can then be used

to determine the level of carcinogenic risk according to the concentration of this substance in ambient air:

Probability of developing leukaemia	Concentration
10^{-5} (1 in 100,000)	$1.6 \mu\text{g}\cdot\text{m}^{-3}$
10^{-6} (1 in 1,000,000)	$0.16 \mu\text{g}\cdot\text{m}^{-3}$

The currently applicable air quality limit value in the Czech Republic for the annual mean concentration is $5 \mu\text{g}\cdot\text{m}^{-3}$, corresponding to a carcinogenic risk of 3×10^{-5} for lifetime exposure. From 2030, new air quality limit values are expected to apply, with the limit value for benzene set at $3.4 \mu\text{g}\cdot\text{m}^{-3}$, corresponding to a carcinogenic risk of 2.04×10^{-5} for lifetime exposure.

4.1.5. Benzo[a]pyrene

The group of polycyclic aromatic hydrocarbons (PAHs) comprises several hundred compounds formed primarily during the incomplete combustion of organic material. Their principal effects on human health are mutagenicity and carcinogenicity, whereas their systemic toxic effects are probably minor (based on animal testing). The mutagenic and carcinogenic effects of many PAHs with higher boiling points are considered to be established, and benzo[a]pyrene is one of the compounds with the highest carcinogenic potency.

According to IARC, benzo[a]pyrene is classified in Group 1 as carcinogenic to humans. Owing to its carcinogenicity, no safe threshold can be established. WHO [5] specifies a guideline value for unit cancer risk for B[a]P of $8.7 \times 10^{-2} (\mu\text{g}\cdot\text{m}^{-3})^{-1}$.

PAHs also generally have non-carcinogenic effects, causing eye and skin irritation, toxic damage to the kidneys and liver, haematotoxicity, immunosuppression, reproductive toxicity, and genotoxicity. For the risk of non-carcinogenic effects from inhalation exposure, the US EPA specifies the reference concentration RfC^{24} of $2 \text{ ng}\cdot\text{m}^{-3}$, derived from a developmental toxicity study in rats using a high uncertainty factor [22].

The currently applicable air quality limit value in the Czech Republic for the annual mean concentration is $1 \text{ ng}\cdot\text{m}^{-3}$, corresponding to a carcinogenic risk of 8.7×10^{-5} for lifetime exposure. From 2030, new air quality limit values are expected to apply, with the limit value for benzo[a]pyrene remaining unchanged.

4.2. Exposure assessment and risk characterisation

The supporting air dispersion study [1] calculates the contributions of the assessed sources to the overall air pollution level. Based on the vector layer of the built-

up area and data from the Cadastre of Real Estate, the corresponding number of residents was assigned to each air pollution band.

The following text quantifies the expected health impacts of this air pollution on the affected population. When assessing the effects of exposure to particulate matter and nitrogen dioxide on the basis of the relative risk values from the HRAPIE-2 project [8], the assessment is performed in accordance with AN 17/15 [2] using the attributable fraction calculation method, which yields the number of persons in the exposed population affected by the relevant health effect. The calculation is described, for example, in the methodology developed by the Charles University Environment Centre for assessing the society-wide impacts of air pollution [3] and in a WHO publication by B. Ostro [11]. For substances with a non-threshold effect, the number of persons affected by the relevant effect is calculated using the following equation:

$$\text{IMP} = \text{EXP} \times \text{AGF} \times \text{RGF} \times \text{BGR} \times [1 - (1/\text{RR})^C],$$

where

- IMP is the expected (attributable) number of cases resulting from air pollution over the given period (e.g. the number of deaths due to exposure to a particular concentration of air pollution, the number of cases of bronchitis, the number of hospital admissions, etc.),
- C is the pollutant concentration in $\mu\text{g}\cdot\text{m}^{-3}$,
- EXP is the exposed population (number of persons),
- AGF is the proportion of the age group to which the effect relates within the total population,
- RGF is the proportion of any at-risk group to which the effect relates (if considered), such as persons with asthma, within the relevant age group of the population,
- BGR is the rate of occurrence of the resulting health outcome in the population (mortality or disease incidence) per individual,
- RR is the relative risk per 1 $\mu\text{g}\cdot\text{m}^{-3}$ increase in concentration.

Furthermore, as shown in Tables 5 and 6, in some cases the input value used in the calculation is the daily (i.e. not annual) mean concentration. The calculation of IMP in these cases is specified in greater detail in Section 4.3. The AGF and BGR values were calculated from data provided by the Czech Statistical Office (CZSO), the Institute of Health Information and Statistics of the Czech Republic (IHIS CR), and the Ministry of Health of the Czech Republic, using the average for 2020–2024 [12, 13, 14, 15, 16]. In view of the limited availability of data, the necessary methodological compromises, and the specific features of the model, the calculation procedure was modified in the following cases:

- For malignant neoplasms of the bronchus and lung, only data for the period 2020–2023 were used in the calculation.

- The IHIS CR data did not allow information on the incidence of different types of diabetes in the paediatric population to be broken down by type. Since type 2 diabetes accounts for only a single-digit percentage of new cases of diabetes mellitus in children, the total number of these cases was multiplied by a factor of 0.05 [17, 18].
- Data on the incidence of dementia were only available for the population aged 50 years and over, whereas the RR values in the HRAPIE-2 project [8] were established on the basis of effects on the population aged 19 years and over. However, the incidence of dementia before the age of 50 is entirely marginal [19]. The data obtained were therefore related to the population aged 19 years and over.
- Data from PAQ Research on the number of school hours missed per pupil in the individual regions of the Czech Republic during 2017–2023 were used as input values for calculating the indicator of days of school missed [20]. The resulting values were subsequently converted into days on the basis of data on the weekly allocation of teaching hours in the Framework Educational Programme for Basic Education issued by the Ministry of Education, Youth and Sports (MEYS) [21].
- When assessing COPD and high blood pressure, following consultation with the IHIS CR, a narrower selection of diagnoses based on ICD-10 codes (J44 and I10) was applied instead of the broader ranges recommended in the HRAPIE-2 methodology [8] (J41–J44 and I10–I11). This approach reflects the official definitions established by the IHIS CR and professional medical societies for reporting data in Czech clinical practice. This reduces the risk of the results being affected by the use of irrelevant data.
- Following consultation with the IHIS CR, the indicator of autism spectrum disorders in children was not included in the health risk assessment. This was due to inconsistencies in the primary reporting of the incidence of this diagnosis in the Czech healthcare system. Furthermore, this indicator falls within Group B+, which is characterised by greater uncertainty regarding the accuracy of the data used to quantify the effects than Group A. Inclusion of these data could lead to significant distortion of the results.

The baseline values used to quantify the individual effects of air pollution are presented in the following table. The data are derived from the statistical data for the South Bohemian Region described above.

Table 5. Input data for quantifying the effects of air pollution – PM [8, 12–16]

Air quality characteristic	Health effect	Population segment	Group	AGF (%)	BGR
PM _{2.5} annual mean	adult natural-cause mortality*	≥ 25 years	A	74.5	0.0138
PM _{2.5} annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	A	74.5	0.0051
PM _{2.5} annual mean	adult mortality – cerebrovascular diseases	≥ 25 years	A	74.5	0.0007
PM _{2.5} annual mean	adult mortality – ischaemic heart disease	≥ 25 years	A	74.5	0.0018
PM _{2.5} annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	A	74.5	0.0013
PM _{2.5} annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	A	74.5	0.0005
PM _{2.5} annual mean	adult mortality – lower respiratory tract infections	≥ 25 years	A	74.5	0.0006
PM _{2.5} annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	A	74.5	0.0007
PM ₁₀ annual mean	adult natural-cause mortality*	≥ 25 years	A	74.5	0.0138
PM ₁₀ annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	A	74.5	0.0051
PM ₁₀ annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	A	74.5	0.0013
PM ₁₀ annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	A	74.5	0.0007
PM ₁₀ annual mean	adult mortality – ischaemic heart disease	≥ 25 years	B+	74.5	0.0018
PM ₁₀ annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	B+	74.5	0.0005
PM _{2.5} annual mean	morbidity – childhood asthma	0–18 years	A	19.9	0.0065
PM _{2.5} annual mean	morbidity – chronic obstructive pulmonary disease (COPD)	≥ 30 years	A	69.2	0.0036
PM _{2.5} annual mean	morbidity – ischaemic heart disease (myocardial infarction)	≥ 30 years	A	69.2	0.0022
PM _{2.5} annual mean	morbidity – stroke	≥ 30 years	A	69.2	0.0029
PM _{2.5} annual mean	morbidity – high blood pressure	≥ 30 years	A	69.2	0.0202
PM _{2.5} annual mean	morbidity – malignant neoplasm of the bronchus and lung	≥ 30 years	A	69.2	0.0009
PM _{2.5} annual mean	morbidity – type 2 diabetes	0–18 years	B+	19.9	0.00005
PM _{2.5} annual mean	morbidity – dementia	≥ 19 years	B+	80.1	0.0045
PM _{2.5} daily mean	adult natural-cause mortality*	entire population	A	100.0	0.0103
PM _{2.5} daily mean	morbidity – diseases of the circulatory system	entire population	A	100.0	0.0223
PM ₁₀ daily mean	days of school missed	5–14 years	B+	10.7	16.8110

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 6. Input data for quantifying the effects of air pollution – NO₂ [8, 12–16]

Air quality characteristic	Health effect	Population segment	Group	AGF (%)	BGR
NO ₂ annual mean	adult natural-cause mortality*	≥ 25 years	A	74.5	0.0138
NO ₂ annual mean	adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	A	74.5	0.0013
NO ₂ annual mean	adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	A	74.5	0.0005
NO ₂ annual mean	adult mortality – lower respiratory tract infections	≥ 25 years	A	74.5	0.0006
NO ₂ annual mean	adult mortality – diseases of the circulatory system	≥ 25 years	B+	74.5	0.0051
NO ₂ annual mean	adult mortality – ischaemic heart disease	≥ 25 years	B+	74.5	0.0018
NO ₂ annual mean	adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	B+	74.5	0.0007
NO ₂ annual mean	morbidity – childhood asthma	0–18 years	A	19.9	0.0065
NO ₂ annual mean	morbidity – asthma in adults	≥ 19 years	A	80.1	0.0046
NO ₂ annual mean	morbidity – lower respiratory tract infections in children	0–12 years	A	13.6	0.0319
NO ₂ daily mean	adult natural-cause mortality*	entire population	A	100.0	0.0103
NO ₂ daily mean	morbidity – respiratory diseases	entire population	A	100.0	0.0133

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

For benzene and benzo[a]pyrene, the assessment is performed in a similar manner, except that the AGF, RGF, and BGR values are equal to one (the effect always relates to the entire affected population), and the resulting impact is quantified as the number of residents per new case of the relevant effect.

The following table presents the range of five-year average concentration values based on CHMI data for residential areas within the area covered by the model calculations. The values were obtained from 78 grid squares.

Table 7. Range of background concentrations based on five-year averages from CHMI data (2020–2024)

	IH _r PM ₁₀	IH _r PM _{2.5}	IH _r NO ₂	IH _r BZN	IH _r B[a]P
	μg.m ⁻³				ng.m ⁻³
Minimum	11.3	7.9	4.7	0.5	0.2
Mean	12.8	9.0	5.8	0.6	0.3
Maximum	15.3	10.8	11.3	0.8	0.6

IH_r: annual mean concentrations

For the quantitative assessment of the effects of air pollution in the following section of the study, the highest value from the five-year averages was used for each pollutant; the assessment is therefore conservative.

4.2.1. Particulate matter

Elevated concentrations of particulate matter in ambient air are generally associated with respiratory diseases, lung cancer, cardiovascular diseases and, in the case of the PM_{2.5} fraction, stroke.

For chronic exposure, WHO [6] specifies a guideline value for annual mean concentration of 5 $\mu\text{g}\cdot\text{m}^{-3}$ for PM_{2.5} and 15 $\mu\text{g}\cdot\text{m}^{-3}$ for PM₁₀.

Tables 8–10 present the numbers of residents in the individual PM_{2.5} concentration bands, while Tables 11–13 present the numbers of residents in the individual bands of PM_{2.5} concentration change.

Tables 14–16 present the numbers of residents in the individual PM₁₀ concentration bands, while Tables 17–19 present the numbers of residents in the individual bands of PM₁₀ concentration change.

As can be seen from the assessment of the overall air pollution situation, PM_{2.5} concentrations throughout the modelled area exceed the WHO guideline value (reaching interim targets 3 and 4 in residential areas). For PM₁₀, concentrations are below the WHO guideline value across most of the modelled area; slightly higher concentrations were only recorded locally (at the level of interim target 4).

As stated above, for the purpose of determining the number of residents in the individual concentration bands and subsequently quantifying the effects of air pollution, the highest recorded five-year average value in the assessed area was applied uniformly throughout the modelled area. From this perspective, the overall air pollution levels in most of the modelled area may be considered overestimated; however, the change in the incidence of effects related to air pollution exposure due to the project corresponds to the actual changes in pollutant concentrations according to the results of the model calculations.

Table 8. Numbers of residents in I_{Hr} PM_{2.5} concentration bands (Scenario 1 – 2034)

I _{Hr} PM _{2.5} concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 10.85	< 2.17	11,159	5,197
10.85–10.90	2.17–2.18	1,447	5,413
10.90–10.95	2.18–2.19	44	718
10.95–11.00	2.19–2.20	0	834
11.00–11.10	2.20–2.22	0	348
11.10–11.20	2.22–2.24	0	131
11.20–11.40	2.24–2.28	0	7
> 11.40	> 2.28	0	2
Total		12,650	12,650

* The background concentration was assumed to be 10.8 µg·m⁻³

Table 9. Numbers of residents in I_{Hr} PM_{2.5} concentration bands (Scenario 2 – 2040)

I _{Hr} PM _{2.5} concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 10.85	< 2.17	7,846	7,834
10.85–10.90	2.17–2.18	3,356	3,364
10.90–10.95	2.18–2.19	771	773
10.95–11.00	2.19–2.20	643	645
11.00–11.05	2.20–2.21	22	22
> 11.05	> 2.21	12	12
Total		12,650	12,650

* The background concentration was assumed to be 10.8 µg·m⁻³

Table 10. Numbers of residents in I_{Hr} PM_{2.5} concentration bands (Scenario 3 – 2042)

I _{Hr} PM _{2.5} concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 10.81	< 2.162	6,806	6,206
10.81–10.82	2.162–2.164	5,370	5,910
10.82–10.83	2.164–2.166	434	485
> 10.83	> 2.166	40	49
Total		12,650	12,650

* The background concentration was assumed to be 10.8 µg·m⁻³

Table 11. Numbers of residents in bands of IH_r $PM_{2.5}$ concentration change (Scenario 1 – 2034)

Change in IH_r $PM_{2.5}$ ($\mu g \cdot m^{-3}$)	Difference
< 0.10	11,881
0.10–0.20	550
0.20–0.30	190
0.30–0.40	27
0.40–0.60	0
> 0.60	2

Table 12. Numbers of residents in bands of IH_r $PM_{2.5}$ concentration change (Scenario 2 – 2040)

Change in IH_r $PM_{2.5}$ ($\mu g \cdot m^{-3}$)	Difference
< 0.001	12,474
0.001–0.002	159
0.002–0.003	14
> 0.003	3

Table 13. Numbers of residents in bands of IH_r $PM_{2.5}$ concentration change (Scenario 3 – 2042)

Change in IH_r $PM_{2.5}$ ($\mu g \cdot m^{-3}$)	Difference
< 0.001	9,513
0.001–0.002	2,906
0.002–0.003	180
0.003–0.004	34
> 0.004	17

Table 14. Numbers of residents in IH_r PM_{10} concentration bands (Scenario 1 – 2034)

IH_r PM_{10} concentration band ($\mu g \cdot m^{-3}$)	Ratio to the guideline value	Baseline scenario	Project scenario
< 15.5	< 1.033	11,137	1,781
15.5–15.7	1.033–1.047	1,452	7,363
15.7–15.9	1.047–1.060	61	1,833
15.9–16.1	1.060–1.073	0	803
16.1–16.3	1.073–1.087	0	277
16.3–16.8	1.087–1.120	0	414
16.8–17.3	1.120–1.153	0	172
17.3–19.3	1.153–1.287	0	5
> 19.3	> 1.287	0	2
Total		12,650	12,650

* The background concentration was assumed to be $15.3 \mu g \cdot m^{-3}$

Table 15. Numbers of residents in IH_r PM_{10} concentration bands (Scenario 2 – 2040)

IH_r PM_{10} concentration band ($\mu g \cdot m^{-3}$)	Ratio to the guideline value	Baseline scenario	Project scenario
< 15.50	< 1.03	7,930	7,923
15.50–15.70	1.03–1.05	3,292	3,297
15.70–15.90	1.05–1.06	746	747
15.90–16.10	1.06–1.07	639	640
16.10–16.30	1.07–1.09	31	31
> 16.30	> 1.09	12	12
Total		12,650	12,650

* The background concentration was assumed to be $15.3 \mu g \cdot m^{-3}$

Table 16. Numbers of residents in IH_r PM_{10} concentration bands (Scenario 3 – 2042)

IH_r PM_{10} concentration band ($\mu g \cdot m^{-3}$)	Ratio to the guideline value	Baseline scenario	Project scenario
< 15.35	< 1.023	8,571	7,357
15.35–15.40	1.023–1.027	3,892	5,034
15.40–15.45	1.027–1.030	158	228
15.45–15.50	1.030–1.033	29	31
> 15.50	> 1.033	0	0
Total		12,650	12,650

* The background concentration was assumed to be $15.3 \mu g \cdot m^{-3}$

Table 17. Numbers of residents in bands of IH_r PM_{10} concentration change (Scenario 1 – 2034)

Change in IH_r PM_{10} ($\mu g \cdot m^{-3}$)	Difference
< 0.20	5,992
0.20–0.50	5,862
0.50–1.00	462
1.00–1.50	280
1.50–2.00	52
2.00–4.00	0
> 4.00	2

Table 18. Numbers of residents in bands of IH_r PM_{10} concentration change (Scenario 2 – 2040)

Change in IH_r PM_{10} ($\mu g \cdot m^{-3}$)	Difference
< 0.005	12,650

Table 19. Numbers of residents in bands of IH_r PM_{10} concentration change (Scenario 3 – 2042)

Change in IH_r PM_{10} ($\mu g \cdot m^{-3}$)	Difference
< 0.000	53
0.000–0.005	11,770
0.005–0.010	794
0.010–0.015	30
> 0.015	3

Tables 20–25 present the quantification of the incidence of the health effects defined by the HRAPIE-2 project [8] (see Tables 1 and 2), calculated using the method described in the introduction to Section 4.2. When interpreting the results, it should be borne in mind that the values for the individual scenarios only represent the effect of air pollution itself, not the total incidence of the health effect concerned.

Table 20. Assessment of changes in the level of health risk in residential areas due to changes in PM_{2.5} pollution (Scenario 1 – 2034)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	12.7640	12.8097	0.0457
adult mortality – diseases of the circulatory system	≥ 25 years	5.9814	6.0024	0.0210
adult mortality – cerebrovascular diseases	≥ 25 years	0.9116	0.9147	0.0031
adult mortality – ischaemic heart disease	≥ 25 years	2.3052	2.3132	0.0080
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.5764	1.5819	0.0055
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.5684	0.5704	0.0020
adult mortality – lower respiratory tract infections	≥ 25 years	1.0816	1.0853	0.0037
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.5556	0.5576	0.0020
morbidity – childhood asthma	0–18 years	4.4275	4.4416	0.0141
morbidity – chronic obstructive pulmonary disease (COPD)	≥ 30 years	5.1033	5.1208	0.0175
morbidity – ischaemic heart disease (myocardial infarction)	≥ 30 years	2.3355	2.3437	0.0082
morbidity – stroke	≥ 30 years	3.8112	3.8244	0.0132
morbidity – high blood pressure	≥ 30 years	27.6132	27.7085	0.0953
morbidity – malignant neoplasm of the bronchus and lung	≥ 30 years	1.1784	1.1825	0.0041
morbidity – type 2 diabetes	0–18 years	0.0115	0.0116	0.0001
morbidity – dementia	≥ 19 years	7.0620	7.0864	0.0244

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 21. Assessment of changes in the level of health risk in residential areas due to changes in PM_{2.5} pollution (Scenario 2 – 2040)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality [*]	≥ 25 years	12.78807	12.78815	0.00008
adult mortality – diseases of the circulatory system	≥ 25 years	5.99248	5.99252	0.00004
adult mortality – cerebrovascular diseases	≥ 25 years	0.91324	0.91324	0.00001
adult mortality – ischaemic heart disease	≥ 25 years	2.30941	2.30943	0.00001
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.57933	1.57934	0.00001
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.56945	0.56945	0.00000
adult mortality – lower respiratory tract infections	≥ 25 years	1.08351	1.08352	0.00001
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.55669	0.55669	0.00000
morbidity – childhood asthma	0–18 years	4.43494	4.43496	0.00002
morbidity – chronic obstructive pulmonary disease (COPD)	≥ 30 years	5.11251	5.11254	0.00003
morbidity – ischaemic heart disease (myocardial infarction)	≥ 30 years	2.33980	2.33981	0.00001
morbidity – stroke	≥ 30 years	3.81818	3.81820	0.00002
morbidity – high blood pressure	≥ 30 years	27.66344	27.66361	0.00017
morbidity – malignant neoplasm of the bronchus and lung	≥ 30 years	1.18054	1.18055	0.00001
morbidity – type 2 diabetes	0–18 years	0.01155	0.01155	0.00000
morbidity – dementia	≥ 19 years	7.07482	7.07487	0.00004

^{*}) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 22. Assessment of changes in the level of health risk in residential areas due to changes in PM_{2.5} pollution (Scenario 3 – 2042)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	12.74049	12.74108	0.00059
adult mortality – diseases of the circulatory system	≥ 25 years	5.97051	5.97079	0.00027
adult mortality – cerebrovascular diseases	≥ 25 years	0.90992	0.90996	0.00004
adult mortality – ischaemic heart disease	≥ 25 years	2.30099	2.30109	0.00010
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.57357	1.57364	0.00007
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.56737	0.56740	0.00003
adult mortality – lower respiratory tract infections	≥ 25 years	1.07967	1.07972	0.00005
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.55461	0.55463	0.00003
morbidity – childhood asthma	0–18 years	4.42017	4.42035	0.00018
morbidity – chronic obstructive pulmonary disease (COPD)	≥ 30 years	5.09422	5.09444	0.00023
morbidity – ischaemic heart disease (myocardial infarction)	≥ 30 years	2.33122	2.33133	0.00011
morbidity – stroke	≥ 30 years	3.80439	3.80456	0.00017
morbidity – high blood pressure	≥ 30 years	27.56397	27.56521	0.00124
morbidity – malignant neoplasm of the bronchus and lung	≥ 30 years	1.17628	1.17633	0.00005
morbidity – type 2 diabetes	0–18 years	0.01151	0.01151	0.00000
morbidity – dementia	≥ 19 years	7.04939	7.04970	0.00032

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 23. Assessment of changes in the level of health risk in residential areas due to changes in PM₁₀ pollution (Scenario 1 – 2034)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	14.5670	14.8038	0.2368
adult mortality – diseases of the circulatory system	≥ 25 years	5.4000	5.4878	0.0878
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.9109	1.9411	0.0301
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.8524	0.8661	0.0136
adult mortality – ischaemic heart disease	≥ 25 years	1.4976	1.5223	0.0247
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	1.1348	1.1515	0.0167

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 24. Assessment of changes in the level of health risk in residential areas due to changes in PM₁₀ pollution (Scenario 2 – 2040)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	14.64158	14.64172	0.00014
adult mortality – diseases of the circulatory system	≥ 25 years	5.42765	5.42770	0.00005
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.92042	1.92044	0.00002
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.85674	0.85675	0.00001
adult mortality – ischaemic heart disease	≥ 25 years	1.50541	1.50543	0.00001
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	1.14005	1.14006	0.00001

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 25. Assessment of changes in the level of health risk in residential areas due to changes in PM₁₀ pollution (Scenario 3 – 2042)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	14.4932	14.4978	0.0045
adult mortality – diseases of the circulatory system	≥ 25 years	5.3727	5.3743	0.0017
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	1.9015	1.9021	0.0006
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.8482	0.8484	0.0003
adult mortality – ischaemic heart disease	≥ 25 years	1.4899	1.4904	0.0005
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	1.1296	1.1299	0.0003

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

As shown in the tables above, the assessed project is expected to result in a slight increase in health risk from exposure to both PM_{2.5} and PM₁₀.

In Scenario 1 (2034, expected duration of up to 1 year), exposure to PM_{2.5} pollution was calculated to result in an increase in adult natural-cause mortality of the order of hundredths of a new case and an increase in the individual morbidity categories of less than one tenth of a case across the entire affected population. Exposure to PM₁₀ pollution was calculated to result in an increase in natural-cause mortality of the order of tenths of a new case across the entire affected population. The highest increase in PM_{2.5} pollution in residential areas was calculated at up to 0.7 µg·m⁻³ (affecting a few residents), while the highest increase in PM₁₀ pollution was calculated at up to 2 µg·m⁻³ (affecting no more than a few dozen residents); a value of approximately 4 µg·m⁻³ was only recorded in a very localised area (affecting one residential building). Even in the residential area most affected by the project, the increases in natural-cause mortality and the individual morbidity categories were several orders of magnitude below one case in the affected population.

In Scenario 2 (2040, expected duration of up to 1 year), exposure to PM_{2.5} pollution was calculated to result in an increase in adult natural-cause mortality of the order of hundred-thousandths of a new case and an increase in the individual morbidity categories also of the order of hundred-thousandths, and definitely no more than approximately one ten-thousandth of a case across the entire affected population. Exposure to PM₁₀ pollution was calculated to result in an increase in natural-cause mortality of the order of ten-thousandths of a new case across the entire affected population. The highest increase in PM_{2.5} pollution in residential areas was calculated at approximately 0.003 µg·m⁻³ (affecting a few to a few dozen residents), while the highest increase in PM₁₀ pollution was calculated at up to 0.005 µg·m⁻³. Even in the residential area most affected by the project, the increases in natural-cause mortality and the individual morbidity categories were several orders of magnitude below one case in the affected population.

In Scenario 3 (2042, permanent scenario), exposure to PM_{2.5} pollution was calculated to result in an increase in adult natural-cause mortality of the order of ten-thousandths of a new case and an increase in the individual morbidity categories also of the order of ten-thousandths of a case across the entire affected population. Exposure to PM₁₀ pollution was calculated to result in an increase in natural-cause mortality of the order of thousandths of a new case across the entire affected population. The highest increase in PM_{2.5} pollution in residential areas was calculated at up to 0.005 µg·m⁻³ (affecting a few dozen residents), while the highest increase in PM₁₀ pollution was calculated at up to 0.020 µg·m⁻³ (affecting a few dozen residents). Even in the residential area most affected by the project, the increases in natural-cause mortality and the

individual morbidity categories were several orders of magnitude below one case in the affected population.

Therefore, even in the residential areas most affected by the increase, the assessed project will result in changes in health risk that are insignificant in terms of adverse health effects and will be outweighed by other factors, such as lifestyle (e.g. smoking) or exposure to other sources of pollution.

4.2.2. Nitrogen dioxide

The most commonly reported chronic effects of NO₂ are structural changes in the lungs and increased susceptibility to bacterial and viral infections.

For chronic exposure, WHO [6] specifies a guideline value of 10 µg·m⁻³ for the annual mean nitrogen dioxide concentration.

Tables 26–28 present the numbers of residents in the individual nitrogen dioxide concentration bands, while Tables 29–31 present the numbers of residents in bands of concentration change.

As can be seen from the assessment of the overall air pollution situation, concentrations in most of the modelled area are below the WHO guideline value; slightly higher concentrations were recorded only locally (at the level of interim target 3).

As stated above, for the purpose of determining the number of residents in the individual concentration bands and subsequently quantifying the effects of air pollution, the highest recorded five-year average value in the assessed area was applied uniformly throughout the modelled area. From this perspective, the overall air pollution levels in most of the modelled area may be considered overestimated; however, the change in the incidence of effects related to air pollution exposure due to the project corresponds to the actual changes in pollutant concentrations according to the results of the model calculations.

Table 26. Numbers of residents in IH_r NO₂ concentration bands (Scenario 1 – 2034)

IH _r NO ₂ concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 11.30	< 1.130	12,442	50
11.30–11.32	1.130–1.132	208	11,135
11.32–11.34	1.132–1.134	0	1,214
11.34–11.36	1.134–1.136	0	242
11.36–11.38	1.136–1.138	0	7
> 11.38	> 1.138	0	2
Total		12,650	12,650

* The background concentration was assumed to be 11.3 µg·m⁻³

Table 27. Numbers of residents in IH_r NO₂ concentration bands (Scenario 2 – 2040)

IH _r NO ₂ concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 11.290	< 1.1290	52	46
11.290–11.295	1.1290–1.1295	525	471
11.295–11.300	1.1295–1.1300	6,917	5,383
11.300–11.305	1.1300–1.1305	4,530	5,440
11.305–11.310	1.1305–1.1310	624	1,173
> 11.310	> 1.1310	2	137
Total		12,650	12,650

* The background concentration was assumed to be 11.3 µg·m⁻³

Table 28. Numbers of residents in IH_r NO₂ concentration bands (Scenario 3 – 2042)

IH _r NO ₂ concentration band (µg·m ⁻³)	Ratio to the guideline value	Baseline scenario	Project scenario
< 11.290	< 1.1290	2,757	1,651
11.290–11.295	1.1290–1.1295	5,299	5,674
11.295–11.300	1.1295–1.1300	4,579	5,035
11.300–11.305	1.1300–1.1305	9	283
11.305–11.310	1.1305–1.1310	2	1
11.310–11.315	1.1310–1.1315	2	3
> 11.315	> 1.1315	2	3
Total		12,650	12,650

* The background concentration was assumed to be 11.3 µg·m⁻³

Table 29. Numbers of residents in bands of IH_r NO₂ concentration change (Scenario 1 – 2034)

Change in IH _r NO ₂ (µg·m ⁻³)	Difference
< 0.005	26
0.005–0.010	376
0.010–0.015	5,322
0.015–0.020	4,610
0.020–0.040	2,000
0.040–0.060	281
0.060–0.080	33
> 0.080	2

Table 30. Numbers of residents in bands of IH_r NO₂ concentration change (Scenario 2 – 2040)

Change in IH _r NO ₂ (µg·m ⁻³)	Difference
< 0.002	11,969

0.002–0.004	599
> 0.004	82

Table 31. Numbers of residents in bands of IH_r NO_2 concentration change (Scenario 3 – 2042)

Change in IH_r NO_2 ($\mu g \cdot m^{-3}$)	Difference
< 0.002	11,152
0.002–0.004	1,410
> 0.004	88

Tables 32–34 present the quantification of the incidence of the health effects defined by the HRAPIE-2 project [8] (see Table 2), calculated using the method described in the introduction to Section 4.2. When interpreting the results, it should be borne in mind that the values for the individual scenarios only represent the effect of air pollution itself, not the total incidence of the health effect concerned.

Table 32. Assessment of changes in the level of health risk in residential areas due to changes in NO_2 pollution (Scenario 1 – 2034)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	6.9748	6.9882	0.0134
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	0.6386	0.6398	0.0012
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.1861	0.1864	0.0004
adult mortality – lower respiratory tract infections	≥ 25 years	0.5023	0.5033	0.0010
adult mortality – diseases of the circulatory system	≥ 25 years	2.5856	2.5905	0.0050
adult mortality – ischaemic heart disease	≥ 25 years	0.9338	0.9356	0.0018
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.4585	0.4594	0.0009
morbidity – childhood asthma	0–18 years	1.6627	1.6658	0.0031
morbidity – asthma in adults	≥ 19 years	4.7655	4.7744	0.0089
morbidity – lower respiratory tract infections in children	0–12 years	5.0957	5.1053	0.0096

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 33. Assessment of changes in the level of health risk in residential areas due to changes in NO₂ pollution (Scenario 2 – 2040)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	6.9803	6.9809	0.0006
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	0.6391	0.6391	0.0001
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.1862	0.1863	0.0000
adult mortality – lower respiratory tract infections	≥ 25 years	0.5027	0.5027	0.0000
adult mortality – diseases of the circulatory system	≥ 25 years	2.5876	2.5878	0.0002
adult mortality – ischaemic heart disease	≥ 25 years	0.9345	0.9346	0.0001
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.4589	0.4589	0.0000
morbidity – childhood asthma	0–18 years	1.6640	1.6641	0.0001
morbidity – asthma in adults	≥ 19 years	4.7692	4.7695	0.0004
morbidity – lower respiratory tract infections in children	0–12 years	5.0996	5.1001	0.0004

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

Table 34. Assessment of changes in the level of health risk in residential areas due to changes in NO₂ pollution (Scenario 3 – 2042)

Health effect	Population segment	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	6.9766	6.9771	0.0005
adult mortality – respiratory diseases, excluding neoplasms	≥ 25 years	0.6387	0.6388	0.0000
adult mortality – chronic obstructive pulmonary disease (COPD)	≥ 25 years	0.1861	0.1861	0.0000
adult mortality – lower respiratory tract infections	≥ 25 years	0.5024	0.5025	0.0000
adult mortality – diseases of the circulatory system	≥ 25 years	2.5862	2.5864	0.0002
adult mortality – ischaemic heart disease	≥ 25 years	0.9340	0.9341	0.0001
adult mortality – malignant neoplasm of the bronchus and lung	≥ 25 years	0.4587	0.4587	0.0000
morbidity – childhood asthma	0–18 years	1.6631	1.6632	0.0001
morbidity – asthma in adults	≥ 19 years	4.7666	4.7670	0.0003
morbidity – lower respiratory tract infections in children	0–12 years	5.0969	5.0973	0.0004

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

As shown in the tables above, the assessed project is expected to result in a slight increase in health risk from exposure to nitrogen dioxide.

In Scenario 1 (2034, expected duration of up to 1 year), exposure to nitrogen dioxide pollution was calculated to result in an increase in adult natural-cause mortality of the order of hundredths of a new case and an increase in the individual morbidity categories of the order of thousandths of a case across the entire affected population.

The highest increase in nitrogen dioxide pollution in residential areas was calculated to be below $0.1 \mu\text{g}\cdot\text{m}^{-3}$ (affecting a few residents). Even in the residential area most affected by the project, the increases in natural-cause mortality and the individual morbidity categories were several orders of magnitude below one case in the affected population.

In Scenario 2 (2040, expected duration of up to 1 year), exposure to nitrogen dioxide pollution was calculated to result in an increase in adult natural-cause mortality of the order of ten-thousandths of a new case and an increase in the individual morbidity categories also of the order of ten-thousandths of a case across the entire affected population. The highest increase in nitrogen dioxide pollution in residential areas was calculated at approximately $0.005 \mu\text{g}\cdot\text{m}^{-3}$ (affecting dozens of residents). Even in the residential area most affected by the project, the increases in natural-cause mortality and the individual morbidity categories were several orders of magnitude below one case in the affected population.

In Scenario 3 (2042, permanent scenario), exposure to nitrogen dioxide pollution was calculated to result in an increase in adult natural-cause mortality of the order of ten-thousandths of a new case and an increase in the individual morbidity categories also of the order of ten-thousandths of a case across the entire affected population. The highest increase in nitrogen dioxide pollution in residential areas was calculated at approximately $0.005 \mu\text{g}\cdot\text{m}^{-3}$ (affecting dozens of residents). Even in the residential area most affected by the project, the increases in natural-cause mortality and the individual morbidity categories were several orders of magnitude below one case in the affected population.

Therefore, even in the residential area most affected by the increase, the assessed project will result in changes in health risk that are insignificant in terms of adverse health effects and will be outweighed by other factors or exposure to other sources of pollution.

For the assessment of acute exposure to NO_2 , the WHO guideline value of $200 \mu\text{g}\cdot\text{m}^{-3}$ for hourly concentrations may be used as a safe threshold below which no health risk arises.

According to the results of the air dispersion study, concentrations exceeding the guideline value are not expected anywhere in the area of interest, including under the project scenario. Therefore, health effects resulting from acute exposure to nitrogen dioxide are not expected in any part of the area of interest.

4.2.3. Combined effects of $\text{PM}_{2.5}$ and nitrogen dioxide

As already stated in Section 4.1.3, $\text{PM}_{2.5}$ and NO_2 largely share the same emission sources, and people are not exposed to them in isolation. To ensure the methodological

accuracy of the calculation, adjusted RR values reflecting the correlation between the two pollutants must therefore be used to determine the cumulative effect, thereby limiting the risk of double counting their impacts. Such double counting would occur if the results of the single-pollutant models were simply added together.

The RR value for the combined effects of PM_{2.5} and NO₂ depends to a large extent on the concentrations of these pollutants in ambient air, as expressed by the following equation:

$$RR_{\text{comb}} = (RR_{\text{PM}_{2.5}}^{C_{\text{PM}_{2.5}}}) \times (RR_{\text{NO}_2}^{C_{\text{NO}_2}})$$

where

- RR_{comb} is the relative risk for the combined effects of PM_{2.5} and NO₂ at concentration C,
- $RR_{\text{PM}_{2.5}}$ is the adjusted RR value for PM_{2.5} reflecting the combined effects of PM_{2.5} and NO₂ for a 1 µg·m⁻³ increase in concentration,
- RR_{NO_2} is the adjusted RR value for NO₂ reflecting the combined effects of PM_{2.5} and NO₂ for a 1 µg·m⁻³ increase in concentration,
- C is the concentration of the relevant pollutant in µg·m⁻³.

Tables 35–37 present the numbers of residents in the individual relative risk bands for the combined effects of PM_{2.5} and NO₂ at concentration C.

As stated above, for the purpose of determining the number of residents in the individual relative risk bands and subsequently quantifying the effects of air pollution, the highest recorded value from the five-year averages in the assessed area was applied uniformly throughout the modelled area. From this perspective, the overall relative risk values in most of the modelled area may be considered to be overestimated; however, the change in the incidence of effects related to air pollution exposure due to the project corresponds to the actual changes in pollutant concentrations according to the results of the model calculations.

Table 35. Numbers of residents in RR_{comb} bands (combined effects of PM_{2.5} and NO₂) (Scenario 1 – 2034)

RR_{comb} band*	Baseline scenario	Project scenario
< 1.113	12,650	11,085
1.113–1.114	0	1,365
1.114–1.115	0	192
1.115–1.116	0	6
> 1.116	0	2
Total	12,650	12,650

*) This refers to the relative risk for the combined effects of PM_{2.5} and NO₂ at concentration C (see Section 4.2.3).

Table 36. Numbers of residents in RR_{comb} bands (combined effects of $PM_{2.5}$ and NO_2) (Scenario 2 – 2040)

RR_{comb} band*	Baseline scenario	Project scenario
< 1.1125	9,715	9,708
1.1125–1.1130	2,190	2,199
1.1130–1.1135	733	731
> 1.1135	12	12
Total	12,650	12,650

*) This refers to the relative risk for the combined effects of $PM_{2.5}$ and NO_2 at concentration C (see Section 4.2.3).

Table 37. Numbers of residents in RR_{comb} bands (combined effects of $PM_{2.5}$ and NO_2) (Scenario 3 – 2042)

RR_{comb} band*	Baseline scenario	Project scenario
< 1.1122	12,590	12,581
1.1122–1.1124	41	54
1.1124–1.1126	14	12
1.1126–1.1128	5	0
> 1.1128	0	3
Total	12,650	12,650

*) This refers to the relative risk for the combined effects of $PM_{2.5}$ and NO_2 at concentration C (see Section 4.2.3).

Table 38 presents a quantification of the incidence of the health effect defined by the HRAPIE-2 project [8] (see Table 3), calculated using the method described in the introduction to Section 4.2. When interpreting the results, it should be borne in mind that the values for the individual scenarios only represent the effect of air pollution itself, not the total incidence of the health effect concerned.

Table 38. Assessment of changes in the level of health risk in residential areas due to the combined effects of $PM_{2.5}$ and NO_2 – Scenarios 1–3 (2034, 2040, and 2042)

Health effect	Population segment	Scenario (Year)	Baseline scenario	Project scenario	Difference
adult natural-cause mortality*	≥ 25 years	1 (2034)	13.15992	13.17465	0.01474
adult natural-cause mortality*	≥ 25 years	2 (2040)	13.14897	13.14899	0.00002
adult natural-cause mortality*	≥ 25 years	3 (2042)	13.11798	13.11799	0.00001

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

As shown in the table above, in Scenario 1 (2034, expected duration of up to 1 year), an increase in natural-cause mortality of approximately one hundredth of a case was calculated across the entire affected population; in Scenarios 2 and 3 (2040 and 2042, expected duration of 1 year and permanent scenario, respectively), the calculated increase in natural-cause mortality was of the order of hundred-thousandths of a case across the entire affected population.

4.2.4. Benzene

Benzene is a known human carcinogen. For this assessment, the WHO unit risk value of $6 \times 10^{-6} (\mu\text{g}\cdot\text{m}^{-3})^{-1}$ was used. This value means that a benzene concentration of $1 \mu\text{g}\cdot\text{m}^{-3}$ increases the risk of leukaemia by 6 cases per million people (for lifetime exposure over a period of 70 years). There is therefore no safe threshold. European and Czech legislation takes these facts into account, and a long-term (annual) limit value representing an acceptable (reference) threshold for carcinogenic risk has been adopted to protect public health. Based on the available supporting information and in accordance with information provided by the National Institute of Public Health, the highest acceptable risk values should be considered to be of the order of 10^{-6} .

As can be seen from the assessment of the overall air pollution situation, annual mean concentrations in the modelled area range from 0.5 to $0.8 \mu\text{g}\cdot\text{m}^{-3}$. This corresponds to a carcinogenic risk level of $3.0\text{--}4.8 \times 10^{-6}$. These values are therefore at the threshold of acceptable risk.

Tables 39–41 present the numbers of residents in the individual benzene concentration bands, while Tables 42–44 present the numbers of residents in bands of concentration change. Table 45 then shows the increase in the number of cases of leukaemia due to chronic benzene exposure across the entire affected population.

As stated above, for the purpose of determining the number of residents in the individual concentration bands and subsequently quantifying the effects of air pollution, the highest recorded five-year average value in the assessed area was applied uniformly throughout the modelled area. From this perspective, the overall air pollution levels in most of the modelled area may be considered overestimated; however, the change in the incidence of effects related to air pollution exposure due to the project corresponds to the actual changes in pollutant concentrations according to the results of the model calculations.

Table 39. Numbers of residents in I_{Hr} benzene concentration bands (Scenario 1 – 2034)

Air pollution concentration band I _{Hr} benzene (µg·m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.801	< 4.806	12,647	11,996
0.801–0.802	4.806–4.812	3	519
0.802–0.803	4.812–4.818	0	90
0.803–0.804	4.818–4.824	0	38
> 0.804	> 4.824	0	7
Total		12,650	12,650

Table 40. Numbers of residents in IH_r benzene concentration bands (Scenario 2 – 2040)

Air pollution concentration band IH _r benzene (μg·m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.801	< 4.806	11,444	11,196
0.801–0.802	4.806–4.812	982	1,049
0.802–0.803	4.812–4.818	224	347
> 0.803	> 4.818	0	58
Total		12,650	12,650

Table 41. Numbers of residents in IH_r benzene concentration bands (Scenario 3 – 2042)

Air pollution concentration band IH _r benzene (μg·m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.801	< 4.806	12,648	12,647
> 0.801	> 4.806	2	3
Total		12,650	12,650

Table 42. Numbers of residents in bands of IH_r benzene concentration change (Scenario 1 – 2034)

Change in IH _r benzene (μg·m ⁻³)	Difference
< 0.001	11,966
0.001–0.002	570
0.002–0.003	76
> 0.003	38

Table 43. Numbers of residents in bands of IH_r benzene concentration change (Scenario 2 – 2040)

Change in IH _r benzene (μg·m ⁻³)	Difference
< 0.001	12,650

Table 44. Numbers of residents in bands of IH_r benzene concentration change (Scenario 3 – 2042)

Change in IH _r benzene (μg·m ⁻³)	Difference
< 0.001	12,650

Table 45. Increase in the level of health risk from benzene exposure (increase in the number of cases of leukaemia in the affected population)

Health effect	Scenario (Year)	Baseline scenario	Project scenario	Difference
Incidence of leukaemia	1 (2034)	0.00086797	0.00086804	0.00000007
Incidence of leukaemia	2 (2040)	0.00086809	0.00086814	0.00000004
Incidence of leukaemia	3 (2042)	0.06075796	0.06075797	0.00000001

The assessed project will result in a very slight increase in the level of health risk from chronic benzene exposure. The increase in the number of cases of leukaemia is of the order of hundred-millionths of a new case across the entire affected population.

In Scenario 1 (2034, expected duration of up to 1 year), the highest increase in residential areas was calculated at up to $0.004 \mu\text{g}\cdot\text{m}^{-3}$ and affects several dozen residents. This value corresponds to an increase in the risk of health effects from chronic benzene exposure of no more than 2.40×10^{-8} (1 case in more than 41.6 million residents). In view of the number of residents exposed to the highest increase and the duration of Scenario 1 (1 year), it can be concluded that the calculated changes in health risk are entirely insignificant in terms of adverse health effects.

4.2.5. Benzo[a]pyrene

For the assessment of risk from B[a]P exposure, the WHO unit risk value for lifetime exposure of $8.7 \times 10^{-5} (\text{ng}\cdot\text{m}^{-3})^{-1}$ was used. This value means that a benzo[a]pyrene concentration of $1 \text{ ng}\cdot\text{m}^{-3}$ increases the risk of cancer by 8.7 cases per 100,000 people (for lifetime exposure – over a period of 70 years). The highest acceptable risk is again considered to be of the order of 10^{-6} .

As can be seen from the assessment of the overall air pollution situation, annual mean concentrations in the modelled area range from 0.2 to $0.6 \text{ ng}\cdot\text{m}^{-3}$. This corresponds to a carcinogenic risk level of $17.4\text{--}52.2 \times 10^{-6}$. These values are already above the acceptable risk threshold. An acceptable risk level of the order of 10^{-6} would be achieved at a concentration of $0.1 \text{ ng}\cdot\text{m}^{-3}$ or lower; however, this value is exceeded at all monitoring stations in the Czech Republic.

Tables 46–48 present the numbers of residents in the individual benzo[a]pyrene concentration bands, while Tables 49–51 present the numbers of residents in bands of concentration change. Table 52 then shows the increase in the number of cancer cases due to chronic benzo[a]pyrene exposure across the entire affected population.

As stated above, for the purpose of determining the number of residents in the individual concentration bands and subsequently quantifying the effects of air pollution, the highest recorded five-year average value in the assessed area was applied uniformly throughout the modelled area. From this perspective, the overall air pollution levels in most of the modelled area may be considered overestimated; however, the change in the incidence of effects related to air pollution exposure due to the project corresponds to the actual changes in pollutant concentrations according to the results of the model calculations.

Table 46. Numbers of residents in IH_r B[a]P concentration bands (Scenario 1 – 2034)

Air pollution concentration band IH _r B[a]P (ng.m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.600	< 52.20	0	0
0.600–0.602	52.20–52.37	12,374	10,596
0.602–0.604	52.37–52.55	276	1,913
0.604–0.606	52.55–52.72	0	106
0.606–0.608	52.72–52.90	0	30
0.608–0.610	52.90–53.07	0	5
> 0.610	> 53.07	0	0
Total		12,650	12,650

Table 47. Numbers of residents in IH_r B[a]P concentration bands (Scenario 2 – 2040)

Air pollution concentration band IH _r B[a]P (ng.m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.600	< 52.20	0	0
0.600–0.602	52.20–52.37	10,058	9,966
0.602–0.604	52.37–52.55	2,400	2,475
0.604–0.606	52.55–52.72	190	206
0.606–0.608	52.72–52.90	2	3
> 0.608	> 52.90	0	0
Total		12,650	12,650

Table 48. Numbers of residents in I_{Hr} B[a]P concentration bands (Scenario 3 – 2042)

Air pollution concentration band I _{Hr} B[a]P (ng.m ⁻³)	Carcinogenic risk level (×10 ⁻⁶)	Baseline scenario	Project scenario
< 0.6000	< 52.20	0	0
0.6000–0.6005	52.20–52.24	8,032	6,606
0.6005–0.6010	52.24–52.29	3,848	4,888
0.6010–0.6015	52.29–52.33	757	1,014
0.6015–0.6020	52.33–52.37	13	142
> 0.6020	> 52.37	0	0
Total		12,650	12,650

Table 49. Numbers of residents in bands of I_{Hr} B[a]P concentration change (Scenario 1 – 2034)

Change in I _{Hr} B[a]P (ng.m ⁻³)	Difference
< 0.0002	11,997
0.0002–0.0004	475
0.0004–0.0006	144
> 0.0006	34

Table 50. Numbers of residents in bands of I_{Hr} B[a]P concentration change (Scenario 2 – 2040)

Change in I _{Hr} B[a]P (ng.m ⁻³)	Difference
< 0.0001	11,703
0.0001–0.0002	786
0.0002–0.0003	147
> 0.0003	14

Table 51. Numbers of residents in bands of I_{Hr} B[a]P concentration change (Scenario 3 – 2042)

Change in I _{Hr} B[a]P (ng.m ⁻³)	Difference
< 0.0001	10,947
0.0001–0.0002	1,486
0.0002–0.0003	191
> 0.0003	26

Table 52. Increase in the level of health risk from benzo[a]pyrene exposure (increase in the number of cancer cases in the affected population)

Health effect	Scenario (Year)	Baseline scenario	Project scenario	Difference
Incidence of cancer	1 (2034)	0.00944969	0.00945456	0.00000487
Incidence of cancer	2 (2040)	0.00945593	0.00945621	0.00000027

Incidence of cancer	3 (2042)	0.66084008	0.66092451	0.00008443
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The assessed project will result in a very slight increase in the level of health risk due to chronic exposure to benzo[a]pyrene. The increase in the number of cancer cases ranges from ten-millionths to hundred-thousandths of a new case across the entire affected population.

The highest increase in residential areas was calculated at up to 0.0005 ng.m^{-3} (affecting dozens of residents). This value corresponds to an increase in the risk of health effects from chronic exposure to benzo[a]pyrene of no more than 4.35×10^{-7} (1 case per almost 2.3 million residents). In view of the number of residents affected by the highest increase, it can be concluded that the calculated changes in health risk are entirely insignificant in terms of adverse health effects.

4.3. Exposure assessment and risk characterisation – project construction

Effects on residents must also be expected during construction work. Air pollution will be generated both by the construction activities at the construction site itself and by the associated motor vehicle traffic.

The supporting air dispersion study [1] calculates the contributions of construction work to the overall air pollution level at the individual receptor points. The number of affected residents was subsequently assigned to each point.

The method used to quantify the expected health impacts of these changes on the affected population is similar to that used for long-term effects (see Section 4.2). The only difference is the conversion of the expected annual number of cases in a given location, which is independent of the air pollution concentration, to a daily value and the use of daily rather than annual concentrations. This involves an assessment of the effects of exposure to particulate matter and nitrogen dioxide based on the relative risk values from the HRAPIE-2 project [8], in accordance with AN 17/15 [2]. The calculation is described, for example, in the methodology developed by the Charles University Environment Centre for assessing the society-wide impacts of air pollution [3] and in a WHO publication by B. Ostro [11]. The output is the number of persons in the exposed population affected by the relevant effect in one day, calculated using the following equation:

$$\text{IMP} = [(\text{EXP} \times \text{AGF} \times \text{RGF}) \times \text{BGR}] / 365 \times [1 - (1/\text{RR})^C],$$

where

- IMP is the expected (attributable) number of cases resulting from air pollution over the given period (e.g. the number of deaths due to exposure to a particular concentration of air pollution, the number of cases of bronchitis, the number of hospital admissions, etc.),
- C is the pollutant concentration in $\mu\text{g}\cdot\text{m}^{-3}$,
- EXP is the exposed population (number of persons),
- AGF is the proportion of the age group to which the effect relates within the total population,
- RGF is the proportion of any at-risk group to which the effect relates (if considered), such as persons with asthma, within the relevant age group of the population,
- BGR is the rate of occurrence of the resulting health outcome in the population (mortality or disease incidence) per individual,
- RR is the relative risk per 1 $\mu\text{g}\cdot\text{m}^{-3}$ increase in concentration.

Table 53. Input data for quantifying the effects of air pollution [8, 12–16]

Air quality characteristic	Health effect	Population segment	Group	AGF (%)	BGR
PM _{2.5} daily mean	adult natural-cause mortality*	entire population	A	100.0	0.0103
PM _{2.5} daily mean	morbidity – diseases of the circulatory system	entire population	A	100.0	0.0223
PM ₁₀ daily mean	restricted activity days (RADs) – days of school missed	5–14 years	B+	10.7	16.8110
NO ₂ daily mean	adult natural-cause mortality*	entire population	A	100.0	0.0103
NO ₂ daily mean	morbidity – respiratory diseases	entire population	A	100.0	0.0133

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

In the supporting air dispersion study, maximum daily PM₁₀ concentrations and maximum hourly NO₂ concentrations were calculated at the individual receptor points representing residential areas. Their highest values were calculated in Temelín, both for buildings along the road and (in the case of the effects of New NPP ETE construction) for buildings on the outskirts of Temelín, and were as follows (effects of New NPP ETE construction / effects of SMR ETE construction):

- Maximum daily PM₁₀ concentration: <600.0 µg.m⁻³ / 168.5 µg.m⁻³
- Maximum hourly NO₂ concentration: < 7.00 µg.m⁻³ / 6.72 µg.m⁻³

A PM_{2.5}/PM₁₀ fraction ratio of 30% was assumed to determine daily PM_{2.5} concentrations. For daily NO₂ concentrations, the ratio of daily to hourly concentrations was assumed to be 50%.

Table 54 presents the assumed input air pollution values used for the subsequent quantification of the individual effects. It should be noted that the increases in air pollution levels determined in this manner may be considered the maximum possible values. The assessment of increases in annual mean concentrations during construction in 2034 (Section 4.2) shows that these levels, if reached at all, would result from a combination of specific conditions (particularly dry days and a constant airflow direction throughout the day), and it may reasonably be expected that such conditions will occur only rarely.

For short-term exposure, WHO [6] specifies guideline values for daily mean concentrations of 15 µg.m⁻³ for PM_{2.5}, 45 µg.m⁻³ for PM₁₀, and 25 µg.m⁻³ for NO₂.

Table 54. IH_d PM_{2.5}, IH_d PM₁₀, and IH_d NO₂ air pollution data points

Air quality characteristic	Number of affected residents	Air pollution level – background (µg.m ⁻³)	Air pollution level – contribution (µg.m ⁻³)	Air pollution level – contribution (µg.m ⁻³)
			New NPP ETE construction	SMR ETE construction
PM _{2.5} daily mean	100	7.6	< 180	50.6
PM ₁₀ daily mean	100	10.8	< 600	168.5
NO ₂ daily mean	100	11.3	3.5	3.4

Table 55 presents a quantification of the incidence of the health effects defined by the HRAPIE-2 project [8] (see Tables 1–3), calculated using the method described in the introduction to Section 4.3. When interpreting the results, it should be borne in mind that the values for the individual scenarios only represent the effect of air pollution itself, not the total incidence of the health effect concerned.

The effects are quantified for one day with the daily concentration values specified above. The frequency of occurrence of these concentrations cannot be determined by modelling, as it will depend primarily on the prevailing meteorological conditions.

Table 55. Assessment of changes in the level of health risk in residential areas due to changes in PM_{2.5} and NO₂ pollution levels

Air quality characteristic	Health effect	Population segment	Baseline scenario	New NPP ETE construction		SMR ETE construction	
				Project scenario	Difference	Project scenario	Difference
PM _{2.5} daily mean	adult natural-cause mortality*	entire population	0.000015	0.000349	0.000334	0.000109	0.000094
PM _{2.5} daily mean	morbidity – diseases of the circulatory system	entire population	0.000041	0.000983	0.000942	0.000306	0.000265
PM ₁₀ daily mean	restricted activity days (RADs) – days of school missed	5–14 years	0.014660	0.729814	0.715153	0.215499	0.200839
NO ₂ daily mean	adult natural-cause mortality*	entire population	0.000015	0.000024	0.000009	0.000019	0.000004
NO ₂ daily mean	morbidity – respiratory diseases	entire population	0.000023	0.000030	0.000007	0.000030	0.000007

*) This refers to all mortality except mortality from external causes; according to the International Classification of Diseases (ICD-10), this therefore covers causes of death coded A00–R99.

As shown in the overview above, each day on which the highest calculated daily

values occur will result in an increase in natural-cause mortality at most of the order of ten-thousandths of a new case and an increase in circulatory system morbidity of approximately one thousandth of a new case. For restricted activity days expressed as days of school missed, an increase of just under one case was calculated (assuming the concurrent construction of both projects, i.e. New NPP ETE and SMR ETE). It follows from the above that, although no significant increase in morbidity (in terms of the specified diagnoses) or mortality in the affected population is expected, in the case of restricted activity days, more frequent occurrence of the highest calculated values may realistically result in an increase in days of school missed, which also reflects morbidity to a certain extent (e.g. milder respiratory symptoms).

This demonstrates the need (particularly on dry days) to comply strictly with the measures specified in the supporting air dispersion study, thereby minimising dust from the construction site and from the arrival and departure routes used by construction traffic and thus minimising the occurrence of the highest daily values.

4.4. Uncertainties in the assessment

When interpreting the results of the assessment of effects on the population, account must be taken of the uncertainties inherent in the assessment given the current state of knowledge. These uncertainties concern the following areas:

- forecasts of traffic volumes during project operation in the South Bohemian Region,
- forecasts of fuel consumption and fuel mix at stationary sources, including local heating,
- the location of new technological emission sources in the area, the possible decommissioning of existing sources beyond the assumptions of spatial planning, and changes in technology at major stationary sources,
- the composition of the vehicle fleet, particularly in relation to the use of alternative fuels, and the associated uncertainty in calculating the quantity and composition of emissions,
- the effect of events of society-wide significance on overall patterns of passenger and freight transport in the area of interest (pandemic or political situation),
- the effects of air pollution sources for which no established assessment methodology currently exists,
- the effects of traffic management measures to regulate traffic (other than measures already included in the traffic model),
- determination of concentrations using air dispersion modelling,
- the exposure scenario for the population living in the surrounding area, the time residents spend away from home, and their presence in the outdoor environment,
- the influence of occupational exposure, lifestyle (particularly smoking), and migration on individual risk,

- the available information on the relationship between pollutant concentration levels and their health effects, in particular taking into account the greater uncertainty associated with quantifying the risks of carcinogenic pollutants (benzene and benzo[a]pyrene),
- the establishment of reference concentrations and guideline values for pollutants.

Despite these uncertainties, the data may be considered sufficiently reliable for drawing conclusions concerning the effect on the overall level of health risk.

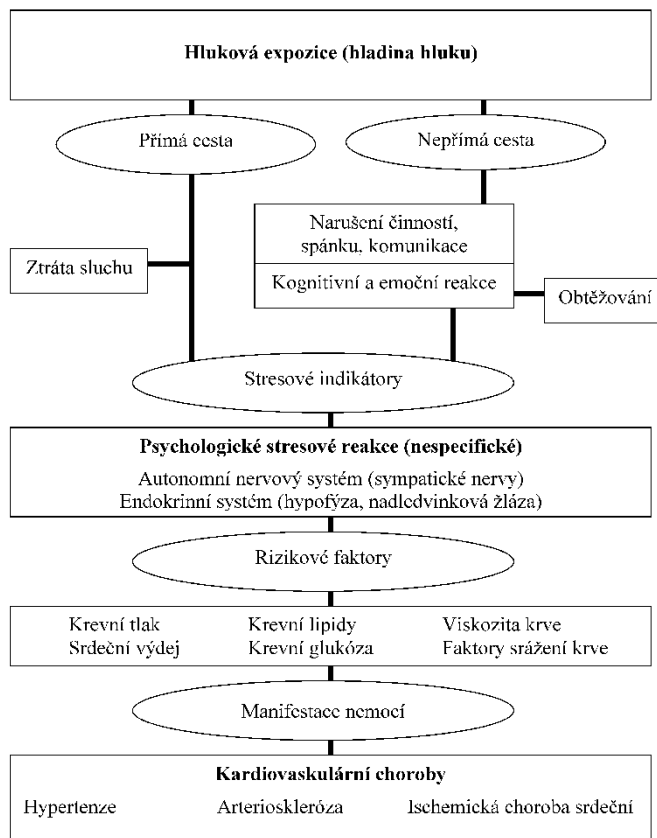
5. EFFECTS OF NOISE ON PUBLIC HEALTH

5.1. Hazard identification and dose–response assessment

Adverse effects of noise on human health are generally defined as morphological or functional changes in the body that lead to impairment of its functions, a reduction in its compensatory capacity in response to stress, or increased susceptibility to other adverse environmental effects. With a certain degree of simplification, the effects of noise on human health may be divided into specific effects, manifested as impaired functioning of the auditory system, and non-specific (non-auditory) effects, involving effects on the functions of various systems of the body.

Normal exposure to traffic noise results primarily in systemic (non-specific) effects involving the functions of various systems of the body, often mediated by a stress response, sleep disturbance, and effects on higher nervous functions. Chronic stress caused by noise may contribute to triggering or accelerating the progression of diseases with multifactorial causes. A simplified causal diagram of the effects of noise on health according to [25], following the chain “noise exposure – physiological (stress) response of the body – biological response and development of disease”, is shown in Figure 2.

Fig. 2. Diagram of the effects of noise



Source: Babisch 2002 in [25]

Hluková expozice (hladina hluku)	Noise exposure (noise level)
Přímá cesta	Direct pathway
Nepřímá cesta	Indirect pathway
Ztráta sluchu	Hearing loss
Narušení činností, spánku, komunikace	Disturbance of activities, sleep, and communication
Kognitivní a emoční reakce	Cognitive and emotional responses
Obtěžování	Annoyance
Stresové indikátory	Stress indicators
Psychologické stresové reakce (nespecifické)	Psychological stress responses (non-specific)
Autonomní nervový systém (sympatické nervy)	Autonomic nervous system (sympathetic nerves)
Endokrinní systém (hypofýza, nadledvinková žláza)	Endocrine system (pituitary gland, adrenal gland)
Rizikové faktory	Risk factors
Krevní tlak	Blood pressure
Srdeční výdej	Cardiac output
Krevní lipidy	Blood lipids
Krevní glukóza	Blood glucose
Viskozita krve	Blood viscosity
Faktory srážení krve	Blood coagulation factors
Manifestace nemocí	Manifestation of diseases
Kardiovaskulární choroby	Cardiovascular diseases
Hypertenze	Hypertension
Arterioskleróza	Arteriosclerosis
Ischemická choroba srdeční	Ischaemic heart disease

According to the current WHO guidelines [32], serious effects of noise considered to be supported by sufficient evidence include annoyance, sleep disturbance, cardiovascular disease, cognitive impairment, and hearing impairment. The following overview provides brief characteristics of these effects according to NIPH [33]:

- **Noise annoyance** is the most general human response to noise exposure. Annoyance refers to a psychological state arising from the involuntary perception of effects towards which an individual has a negative attitude and to which they respond with feelings of aversion, irritation, and, in some cases, even psychosomatic disorders; for simplicity, the term annoyance also covers other negative emotions associated with noise (anger, dissatisfaction, anxiety, and agitation). Annoyance is significantly influenced by the individual characteristics of the recipient; from the perspective of the individual, it is therefore considered a non-threshold effect, meaning that even the quietest sounds may annoy a sensitive person.
- **Adverse effects of noise on sleep** are demonstrably manifested by changes in physiological responses (changes in heart rate, signs of awakening on an EEG, changes in the duration of sleep stages, increased movement during sleep, difficulty falling asleep, awakening, and reduced sleep duration); sufficient evidence also exists for subjectively perceived sleep disturbance, environmental insomnia, and increased use of sleeping medication. The health consequences of sleep disturbance caused by night-time noise include changes in stress hormone levels, cardiovascular disease, mental disorders, obesity, reduced life expectancy, an increased incidence of occupational accidents, and psychological and social consequences (drowsiness and fatigue, irritability, reduced performance, impaired cognitive abilities, and disruption of social contacts).
- The **effects of noise on the cardiovascular system** have been demonstrated in numerous epidemiological studies. The recognised mechanism is the body's stress response, in which a sound signal is subconsciously assessed as alarming, triggering a stress response associated with activation of the autonomic nervous system and the release of stress hormones, which leads to a temporary increase in blood pressure and heart rate and to vasoconstriction. Following long-term exposure, permanent effects such as hypertension and ischaemic heart disease may develop in susceptible individuals. Other possible mechanisms by which noise affects the cardiovascular system include magnesium depletion (with magnesium being released from the body as a result of repeated nerve impulses) or long-term sleep deprivation and its consequences. According to current WHO data, the increased risk of ischaemic heart disease due to road traffic noise is considered established; by contrast, the quality of evidence for the previously reported risk of hypertension is now considered low, while the findings for stroke are inconsistent.
- Noise-related **impairment of cognitive abilities** includes impaired speech comprehension, impaired attention, and reduced working memory capacity. This results in reduced performance, poorer task performance, errors at work, and potentially accidents and injuries. Noise may also seriously interfere with speech communication or mask other signals containing important information. Impaired speech communication has a number of demonstrated adverse consequences for behaviour and relationships, leading to irritability, uncertainty, reduced work performance, and feelings of dissatisfaction. An association between aircraft noise and impaired reading ability, speech comprehension, and test performance among schoolchildren has been confirmed in field studies. For road and rail traffic noise, the results are inconsistent and the quality of the evidence is insufficient.

- **Damage to the auditory system** essentially involves two mechanisms. Extremely high sound pressure levels may cause acoustic trauma involving injury to the eardrum, auditory ossicles, or membranous labyrinth, resulting in permanent hearing impairment. Long-term to lifelong exposure of the auditory system to noise results in hearing impairment, initially involving temporary and subsequently permanent functional and morphological changes to the sensory and nerve cells of the organ of Corti in the inner ear. These disorders initially manifest as a temporary increase in the hearing threshold; with continued noise exposure, permanent hearing impairment develops after a certain latency period. Hearing impairment has been sufficiently demonstrated for occupational noise exposure, depending on the equivalent sound pressure level and the number of years of exposure; however, it also occurs with noise in non-occupational environments, for example in connection with noise from leisure activities. Hearing loss is generally the result of combined exposure to noise from various sources, i.e. occupational, environmental, and leisure noise.

Effects for which the quality of evidence is lower (or the existence of a relationship with noise exposure is uncertain) include an increased risk of diabetes and obesity and effects on pregnancy, foetal development, and mental health [32].

The effects of noise are considered non-threshold (i.e. no safe limit can be established below which the effect no longer occurs); in practice, however, certain threshold values are used above which the relationship between the effect and noise exposure is considered significant. WHO [32] specifies the following recommended exposure values for road traffic noise:

- average value, expressed by the day-evening-night noise indicator (L_{dvn}) – 53 dB
- night-time noise (L_n) – 45 dB

For the quantitative assessment of the effects of road traffic noise in the study area, the procedures specified in NIPH Authorisation Guideline [26], based on Annex III to Commission Directive (EU) 2020/367, were used. The assessment covers the following effects of noise:

- high annoyance
- high sleep disturbance
- ischaemic heart disease (IHD)

For high annoyance and high sleep disturbance, the so-called absolute risk is determined, expressed as the proportion of persons experiencing the relevant effect within the total number of exposed residents at the respective receptor point or within the respective noise exposure band. The calculation equations are as follows:

$$AR_{\text{HA, road}} = (78.927 - 3.1162 \times L_{\text{dvn}} + 0.0342 \times L_{\text{dvn}}^2) / 100$$

$$AR_{\text{HSD, road}} = (19.4321 - 0.9336 \times L_n + 0.0126 \times L_n^2) / 100$$

where:

$AR_{\text{HA, road}}$ = absolute risk of high noise annoyance due to road traffic noise

$AR_{HSD, road}$ = absolute risk of high sleep disturbance due to road traffic noise

L_{dvn} = day-evening-night noise indicator

L_n = night-time noise

The risk of developing ischaemic heart disease (IHD) in relation to noise is quantitatively expressed as a relative risk comparing the risk in the noise-exposed population with the risk in the population not exposed to noise. For quantification, a procedure based on determining the so-called population attributable fraction is used; this may comprise both exposed and unexposed persons, and exposed persons may be exposed to the risk factor to varying degrees. Individual population segments (expressed as a proportion of the total population in the study area) are assigned a level of exposure to road traffic noise (L_{dvn}). The relative risk of developing IHD is then determined for each segment using the following equations:

$$RR_{IHD, road} = 1.007733L_{dvn} - 53 \quad \dots \text{ for } L_{dvn} > 53 \text{ dB}$$

$$RR_{IHD, road} = 1 \quad \dots \text{ for } L_{dvn} \leq 53 \text{ dB}$$

where:

$RR_{IHD, road}$ = relative risk of developing IHD in the population exposed to noise at the given L_{dvn}

The proportion of the population within the study area is also determined for each population segment. The absolute annual number of IHD cases estimated to result from road traffic noise in the study area is then determined using the following equation:

$$N = \sum_j (p_j \times (RR_j - 1)) / (\sum_j (p_j \times (RR_j - 1)) + 1) \times I \times P$$

where:

p_j = proportion of the population in the respective segment

RR_j = relative risk of developing IHD within the respective population segment

I = incidence of IHD in the unaffected population; a value of 9.275 per 1,000 persons per year is used in accordance with Authorisation Guideline [26]

P = number of residents in the study area

For noise from stationary sources of the type occurring in the area, WHO does not specify recommended exposure values.

For an indicative assessment of the annoyance and disturbance effects of stationary sources, the procedure according to [30] was used, based on converting noise from different sources to an equivalent road traffic noise level:

$$L_{r,i} = L_i + 3$$

where:

L_i = noise level from stationary sources

$L_{r,i}$ = derived road traffic noise level

The equations for high annoyance and high sleep disturbance due to road traffic noise are then applied. This assessment is purely indicative, as these cannot be described as health effects, but rather as annoyance effects.

The health risk assessment methodology currently provides no tool for assessing the combined (synergistic) effects of noise from different source categories; the assessment is therefore performed separately for each source category (road traffic, rail traffic, and stationary sources).

5.2. Exposure assessment and risk characterisation

Table 56 presents the calculated road traffic noise exposure levels for 2034, for the daytime and night-time periods in the baseline scenario (operation of ETE1,2 without SMR ETE operation) and the project scenario (operation of ETE1,2 with SMR ETE operation), according to the noise study [23].

Table 56. Noise exposure levels [dB] – road traffic (2034)

Receptor point	Storey	Daytime		Night-time	
		Baseline scenario	Project scenario	Baseline scenario	Project scenario
KOC 1	1	53.4	53.5	45.8	45.8
	2	54.1	54.2	46.5	46.5
TEM 1	1	62.0	62.0	54.0	54.0
	2	62.3	62.3	54.4	54.4
TEM 2	1	59.1	59.2	51.3	51.4
	2	62.4	62.4	54.6	54.7
TEM 3	1	63.7	63.7	56.2	56.3
	2	64.3	64.4	56.9	56.9
TEM 4	1	64.5	64.5	57.0	57.1
	2	64.9	64.9	57.4	57.4
TEM 5	1	63.4	63.4	55.8	55.8
	2	64.8	64.8	57.1	57.2
TEM 6	1	56.1	56.1	47.9	47.9
	2	57.4	57.5	49.2	49.3
TEM 7	1	59.0	59.0	50.8	50.8
	2	60.3	60.4	52.1	52.1
TYN 1	1	61.8	61.9	53.8	54.0
	2	63.5	63.6	55.5	55.7
TYN 2	1	56.5	56.6	48.3	48.6
	2	57.3	57.4	49.2	49.5
	3	58.1	58.2	50.0	50.3
	4	58.8	58.9	50.7	51.0
	5	59.5	59.5	51.3	51.6
	6	60.1	60.2	52.0	52.3
TYN 3	1	58.4	58.5	50.2	50.6

Receptor point	Storey	Daytime		Night-time	
		Baseline scenario	Project scenario	Baseline scenario	Project scenario
	2	59.5	59.6	51.3	51.7
	3	60.6	60.7	52.3	52.7
	4	61.5	61.6	53.3	53.7
	5	62.0	62.1	53.8	54.2
	6	62.2	62.3	54.0	54.4
TYN 4	1	62.5	62.6	54.3	54.5
	2	65.4	65.5	57.3	57.4
TYN 5	1	57.9	57.9	50.7	50.7
	2	62.9	62.9	55.6	55.6
TYN 6	1	62.9	62.9	55.6	55.7
	2	64.1	64.1	56.8	56.8
ZVER 1	1	58.1	58.3	50.6	50.8
	2	59.0	59.2	51.5	51.7
ZVER 2	1	61.9	62.0	54.4	54.6
	2	63.2	63.4	55.7	55.9

Table 57 presents the calculated road traffic noise exposure levels for 2042, for the daytime and night-time periods in the baseline scenario (operation of ETE1,2 and ETE3,4 without SMR ETE operation) and the project scenario (operation of ETE1,2, ETE3,4, and SMR ETE), according to the noise study [23].

Table 57. Noise exposure levels [dB] – road traffic (2042)

Receptor point	Storey	Daytime		Night-time	
		Baseline scenario	Project scenario	Baseline scenario	Project scenario
KOC 1	1	53.6	53.7	46.0	46.1
	2	54.3	54.4	46.7	46.8
TEM 1	1	62.0	62.0	54.0	54.0
	2	62.3	62.3	54.4	54.4
TEM 2	1	59.2	59.2	51.4	51.4
	2	62.5	62.5	54.7	54.7
TEM 3	1	63.9	63.9	56.3	56.3
	2	64.6	64.6	57.0	57.0
TEM 4	1	64.8	64.8	57.1	57.2
	2	65.1	65.1	57.5	57.5
TEM 5	1	63.7	63.7	55.9	55.9
	2	65.1	65.1	57.2	57.2
TEM 6	1	56.2	56.6	48.3	48.3
	2	57.5	58.0	49.6	49.6
TEM 7	1	59.1	59.5	51.1	51.1
	2	60.4	60.9	52.5	52.5
TYN 1	1	62.1	62.2	54.2	54.6
	2	63.8	63.9	55.9	56.3
TYN 2	1	56.7	56.8	48.9	49.1
	2	57.6	57.6	49.7	49.9
	3	58.3	58.4	50.5	50.7
	4	59.1	59.2	51.2	51.4
	5	59.7	59.8	51.9	52.1
	6	60.4	60.4	52.5	52.7
TYN 3	1	58.7	58.8	50.8	50.9
	2	59.8	59.8	51.9	52.0
	3	60.8	60.9	52.9	53.0
	4	61.8	61.9	53.9	54.0
	5	62.3	62.3	54.4	54.5
	6	62.5	62.5	54.6	54.7
TYN 4	1	62.8	62.8	54.7	54.8
	2	65.7	65.8	57.6	57.7
TYN 5	1	58.1	58.2	51.0	50.9
	2	63.1	63.1	55.9	55.8
TYN 6	1	63.1	63.3	55.8	55.8
	2	64.3	64.4	57.0	57.0
ZVER 1	1	58.4	58.6	51.2	51.4
	2	59.3	59.5	52.1	52.3
ZVER 2	1	62.2	62.3	55.0	55.2
	2	63.5	63.7	56.4	56.5

Table 58 presents the calculated noise exposure levels from stationary sources for the assessment years 2034 (ETE1,2 + SMR) and 2042 (ETE1,2, ETE3,4 + SMR), according to the noise study [24]. The assessment was performed for the peripheral parts of the built-up areas of the surrounding settlements.

Table 58. Noise exposure levels [dB] – stationary noise sources

Receptor point	Location	Height	Daytime and night-time	
			2034	2042
MB01	Edge of Litoradlice	3 m	30.2	30.7
		6 m	30.6	31.2
MB02	Near the former farmstead beyond the former village of Knín	3 m	32.0	33.2
		6 m	32.6	33.9
MB03	North-eastern edge of Kočín	3 m	37.8	39.6
		6 m	38.3	39.9
MB04	North-eastern edge of Kočín	3 m	38.1	39.7
		6 m	38.3	39.7
MB05	South-western edge of Kočín	3 m	36.2	36.9
		6 m	37.3	37.8
MB06	Edge of Malešice	3 m	30.8	33.3
		6 m	31.5	33.9
MB07	Edge of Sedlec	3 m	27.7	32.4
		6 m	28.4	32.8
MB08	Edge of Temelín	3 m	30.8	37.4
		6 m	31.1	37.5

Based on the results of the noise study presented above, the proportion of residents in noise exposure bands above the recommended exposure values, the numbers of residents experiencing high noise annoyance and high sleep disturbance, and the incidence of IHD attributable to road traffic noise were quantified. Although the calculation is subject to relatively significant uncertainty, as it does not take into account differences in the sound insulation of building envelopes, the actual presence of persons at their places of residence, or differences in individual sensitivity to noise, it may nevertheless be considered sufficient for assessing the effects of motor vehicle traffic in the modelled area.

Table 59. Proportion of residents exposed above the recommended exposure values specified in the WHO guidelines for road traffic

	Without the project	With the project	Change
2034			
Average day-evening-night noise (%)	100	100	0
Night-time noise (%)	100	100	0
2042			
Average day-evening-night noise (%)	100	100	0
Night-time noise (%)	100	100	0

Table 60. Total values for high annoyance, high sleep disturbance, and the incidence of IHD due to road traffic noise (for a total of 350 affected residents)

	Without the project	With the project	Change
2034			
High annoyance	64	65	1
High sleep disturbance	19	19	0
Incidence of IHD	0.2358	0.2381	0.0023
2042			
High annoyance	65	66	1
High sleep disturbance	19	19	0
Incidence of IHD	0.2421	0.2443	0.0022

As shown by the assessment, even under the no-project scenario, the proportion of residents in the surrounding residential areas falling within bands above the WHO guideline values for average day-evening-night noise and night-time noise will be 100%. The project will not result in any change.

Under the baseline scenario, the number of residents experiencing high annoyance due to road traffic in the existing surrounding residential areas will be approximately 65 in both future scenarios, while the number of residents experiencing high sleep disturbance will be 19. The project was calculated to result in an increase of one case of high annoyance, while no change was recorded for sleep disturbance.

With regard to cardiovascular risk, the incidence of IHD under the baseline scenario was calculated to be of the order of tenths of a case across the entire affected population. The project was calculated to result in an increase of up to approximately 0.0022 cases (an increase in incidence of one case approximately every 454 years). This is therefore an entirely negligible change.

Overall, it can therefore be concluded that the project will result in a very slight increase in the level of health risk. In the case of IHD incidence, the change is several orders of magnitude below the threshold of detectability.

The indicative assessment of the effects of stationary sources shows that their operation may only cause annoyance or sleep disturbance in the Kočín area, involving 2–3% of local residents; this represents, at most, isolated cases.

5.3. Uncertainties in the assessment

When interpreting the results of the assessment of effects on the population, account must be taken of the uncertainties inherent in the assessment given the current state of knowledge. These uncertainties concern the following areas:

- determination of motor vehicle traffic volumes for the period following the commissioning of the project
- the effect of events of society-wide significance on overall patterns of passenger and freight transport within the city (pandemic or political situation)
- the effect of traffic management measures to regulate traffic
- the exposure scenario for the population living in the surrounding area, the time residents spend away from home, and their presence in the outdoor environment
- differences in the airborne sound insulation of building envelopes
- effects on individual risk, particularly due to varying degrees of susceptibility and sensitivity among exposed persons
- the available information on the relationship between noise exposure and its health effects. Particularly in the case of cardiovascular diseases, it should be noted that the quantitative relationships used have not yet been conclusively established and are applied as a precautionary measure.

Despite these uncertainties, the health risk data may be considered sufficiently reliable for drawing overall conclusions regarding the effects of the assessed project on the overall level of health risk.

C O N C L U S I O N

The objective of this study was to assess the effects of technological and transport activities during the implementation and operation of the “New SMR Power Plant at the Temelín site” project on public health resulting from exposure to chemical substances in ambient air and to noise.

The project involves the construction of a New SMR Power Plant at the Temelín site, including all associated structures and systems (technological equipment) used for electricity generation and power evacuation and to ensure the safe operation of the nuclear facility.

Air pollution

The assessment of the effects of air pollution on public health covered ambient concentrations of PM_{2.5} and PM₁₀, nitrogen dioxide, the combined effects of PM_{2.5} and nitrogen dioxide, benzene, and benzo[a]pyrene.

Of the pollutants listed above, an increased risk from chronic exposure to PM_{2.5}, nitrogen dioxide, and benzo[a]pyrene must be expected under the baseline scenario throughout the modelled area, and also from PM₁₀ and nitrogen dioxide in part of the modelled area. However, a similar situation is typical of most settlements in the Czech Republic. Short-term nitrogen dioxide concentrations will remain below the WHO guideline values [6] throughout the modelled area. Benzene pollution levels are expected to be at the threshold of an acceptable level of risk.

The assessment of long-term exposure indicates that the project may result in a slight increase in the level of health risk. For particulate matter, the calculated increase in adult natural-cause mortality was at most of the order of tenths of a new case across the entire affected population (and only in Scenario 1, lasting no longer than 1 year). In Scenario 2 and permanent Scenario 3, the calculated increase in natural-cause mortality was at most of the order of thousandths of a new case across the entire affected population. For nitrogen dioxide concentrations, the calculated increase in adult natural-cause mortality was at most of the order of hundredths of a new case across the entire affected population (and only in Scenario 1, lasting no longer than 1 year). In Scenario 2 and permanent Scenario 3, the calculated increase in natural-cause mortality was at most of the order of ten-thousandths of a new case across the entire affected population. The assessment of the combined effects of PM_{2.5} and NO₂ shows that the increase in natural-cause mortality is of the order of hundredths of a case in Scenario 1 (lasting no longer than 1 year) and of the order of hundred-thousandths of a case in Scenarios 2 and 3. Short-term nitrogen dioxide concentrations will not exceed the WHO guideline values even with the effects of the project taken into account. For annual mean benzene and

benzo[a]pyrene concentrations, the project was not found to cause any changes significant in terms of adverse health effects. The increase in health risk is several orders of magnitude below one new case of leukaemia or cancer.

The assessment of short-term exposure during construction indicates that the project, particularly in conjunction with the construction of New NPP ETE, may result in an increased incidence of diseases of the circulatory system during construction works, with a calculated increase of approximately one thousandth of a case for each day on which the highest calculated values occur. The increase in mortality was calculated to be no more than of the order of ten-thousandths of a new case for each day on which the highest calculated values occur. The increase in the number of days of school missed was calculated to be just under one case for each day on which the highest calculated values occur. If these values occur repeatedly, certain health effects may therefore arise, as represented by an increase in the number of days of school missed.

Overall, it can be concluded that the project will result in changes in the level of health risk that will have no discernible effect. During construction, however – if adverse meteorological and emission conditions coincide – certain health effects could potentially occur. For this reason, measures to reduce particulate matter pollution during construction must be strictly observed, particularly during periods of dry weather, thereby minimising the occurrence of the highest calculated daily concentrations.

Noise exposure

Under the baseline scenario, the number of residents experiencing high annoyance due to road traffic in the existing surrounding residential areas will be approximately 65 in both future scenarios, while the number of residents experiencing high sleep disturbance will be 19. The project was calculated to result in an increase of one case of high annoyance, while no change was recorded for sleep disturbance.

With regard to cardiovascular risk, the incidence of IHD under the baseline scenario was calculated to be of the order of tenths of a case across the entire affected population. The project was calculated to result in an increase of up to approximately 0.0022 cases (an increase in incidence of one case approximately every 435 years). This is therefore an entirely negligible change.

The indicative assessment of the effects of stationary sources shows that their operation may only cause annoyance or sleep disturbance in the Kočín area, and only in isolated cases.

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LIST OF ABBREVIATIONS

CUEC	Charles University Environment Centre
CHMI	Czech Hydrometeorological Institute
CR	Czech Republic
CZSO	Czech Statistical Office
EEG	Electroencephalography
ETE	Temelín Nuclear Power Plant
EU	European Union
COPD	Chronic obstructive pulmonary disease
IARC	International Agency for Research on Cancer
IHD	Ischaemic heart disease
ICD	International Classification of Diseases
MEYS	Ministry of Education, Youth and Sports
New NPP ETE	New Nuclear Power Plant at the Temelín site
NO	Nitric oxide
NO ₂	Nitrogen dioxide
PAH	Polycyclic aromatic hydrocarbons
PM ₁₀	Particulate matter with an aerodynamic diameter of 10 µm or less
PM _{2.5}	Particulate matter with an aerodynamic diameter of 2.5 µm or less
RR	Relative risk
SMR	Small modular reactor
SMR ETE	New SMR Power Plant at the Temelín site
NIPH	National Institute of Public Health
US EPA	U.S. Environmental Protection Agency
IHIS CR	Institute of Health Information and Statistics of the Czech Republic
WHO	World Health Organization