



CBC 2014-2020 South-East Finland - Russia



Green InterTraffic



# **REPORT ON THE HEALTH RISK ASSESSMENT OF HUMAN EXPOSURE TO AMBIENT AIR CHEMICAL EMISSIONS FROM SCANDINAVIA HIGHWAY TRAFFIC**

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## **ACRONYMES AND ABBREVIATIONS**

- MPC<sub>DA</sub>:** Maximum Permissible Concentration (daily average) in the ambient (outdoor) air at populated areas
- MPC<sub>MS</sub>:** Maximum Permissible Concentration (maximum single) in the ambient (outdoor) air at populated areas
- MPC<sub>AA</sub>:** Maximum Permissible Concentration (annual average) in the ambient (outdoor) air at populated areas
- RfC:** Reference (safe) Concentration for Chronic exposures
- ARfC:** Reference (safe) Concentration for short-term Acute exposures
- HQ:** Hazard Quotient
- HI:** Hazard Index for a simultaneous exposure to several substances via the same pathway
- CAS:** Chemical Abstracts Service collects and records main (basic) information on chemical compounds and assigns them with identification numbers
- PM<sub>10</sub>:** fraction of particles with aerodynamic diameter under 10 µm
- PM<sub>2.5</sub>:** fraction of particles with aerodynamic diameter under 2.5 µm

## **TERMS AND DEFINITIONS**

*Risk Analysis* means obtaining information required to prevent negative consequences for human health. It encompasses three components: risk assessment, risk management and risk communication.

*Safety* means a high likelihood of zero hazardous effects of a reviewed chemical substance under certain exposure mode and conditions. In practice, safety means either risk-free or acceptable risk levels.

*Adverse Impact on Humans* means an environmental impact that endangers human life or health or the health of future generations (Article 1 of the Federal Law *on Sanitary and Epidemiological Wellbeing of the Population* adopted March 30, 1999; N 52-ФЗ).

*Adverse Health Effects* are morphological, physiological, growth, developmental or life expectance changes of a human body, population or progeny that manifest through deteriorated functional capacities, decreased ability to compensate for additional stress or increased sensitivity to the impact of other environmental factors.

*Hazard Index (HI)* is a total sum of Hazard Quotients for substances with the same mode of action or a total sum of Hazard Quotients for different pathways of the same chemical substance.

*Hazard Quotient (HQ)* is expressed as a ratio of an exposure to a chemical dose (or concentration) to the safe (reference) exposure level of such chemical.

*Cumulative Risk* is a likelihood of developing an adverse effect if chemical substances of similar action mode simultaneously affect a human body via all possible pathways.

*Health Risk Assessment* is a process to estimate the development probability for and the intensity of adverse human health consequences now or for future generations due to environmental exposure.

*Maximum Permissible Risk* is a top limit of risk acceptance above which extra risk-reduction measures are required.

*Health Risk* is a likelihood of developing a threat to human life/health or to the health of future generations due to the exposure to environmental factors.

## **INTRODUCTION**

*P 2.1.10.1920-04 Guidelines for Risk Assessment of Environmental Chemical Pollutants to Public Health* is the main document to assess health risks for the population exposed to chemical pollutants in the environment. The Guidelines contain standardized requirements, principles, methods and criteria that are used to evaluate health risks associated with the exposure to environmental chemical pollutants. They have incorporated national, international and transnational experience.

Public health risks were assessed within the area directly adjacent to E18 Scandinavia Highway. The paper outlines the public health risk assessment assuming one's permanent presence at the vicinity of the highway and inhaling chemical substances known for their chronic non-cancerogenic effect.

The project has primarily focused on assessing how polluted outdoor air may impact human health and on providing the substance for possibly needed risk reduction measures.

The above objectives were subdivided into the following specific efforts:

- Data from AQT sensors installed in Russia and in Finland were collected and processed; the exposure was assessed;
- Adverse effects to public health associated with the actual burden of pollutants were estimated per sensor locations in Russia and in Finland.

The data collected with AQT sensors between October 2019 and August 2020 were the project's source information.

Key Words: risk assessment, public health, pollutants, cancerogenic risk, hazard identification, non-cancerogenic risk.

## **1. Risk Assessment Methodology. General**

Risk analysis is a step in a systemic approach to political decisions, procedures and practical measures aimed at preventing and reducing human life hazards, morbidity/injuries and property/environmental damage. In Russia, risk analysis makes part of the *Regulations on Industrial Safety*, while internationally it is referred to as *Risk Management*. Risk Analysis or Process Hazard Analysis is defined a systemic use of available information to identify hazards and to assess risk for specific individuals or population groups, property or environment. Risk analysis is focused on determining (identification) of hazards and on risk assessment. Hazard means a source of potential damage or harm or a potentially damaging situation. Risk or Risk Level is a combination of frequency/likelihood and consequences of a certain hazardous event. Risk definition always includes two elements, namely, a hazardous event's frequency and consequences. Once applied, the risk concept allows to manage hazards as measurable categories.

A key document to assess public health risk is *P 2.1.10.1920-04 Guidelines for Risk Assessment of Chemical Environmental Pollutants to Public Health* (approved and enacted on March 5, 2004 by Sanitary Doctor G. G. Onischenko, the First Deputy of the Russian Federation Healthcare Minister). The Guidelines identify the following stages in risk assessment:

- Hazard Identification: to identify potentially noxious factors, to assess the link between a surveyed factor and health impairments; to review the sufficiency and reliability of data on pollution levels at environmental sites with regard to surveyed substances; to prioritize chemical substances for further characterization;
- Assessment of the Dose-Response Relationship: to identify and to quantify the links between human health status and exposure levels;
- Assessment of Chemical Impact on Humans (Exposure): to describe pollution sources as well as pollutant source-to-human routes along with pathways and affected body sites; to determine doses and concentrations that humans were exposed to; to determine exposure levels for the entire population and for subpopulations, therein including supersensitive groups;
- Risk Characterization: to review all collected data, to estimate the risk for population and for population subgroups, to compare these risks with permissible (acceptable) limits, to comparatively assess and to rank various risks as per their statistical, biomedical and social importance; to set healthcare priorities and to identify risks that could be prevented or reduced to an acceptable level.

Risk characterization is the final stage in risk assessment; it is also an element in risk management. This is the stage to combine all outcomes obtained at previous stages. Risk calculation and characterization is done separately for cancerogenic and non-cancerogenic effects.

## 2. Hazard Identification

Hazard identification primarily focuses on selecting priority pollutants in terms of their public health effects. This is the stage to:

- Identify all pollution sources;
- Identify pollutants;
- Determine exposure scenarios and pollutants' pathways into a human body (inhaling, orally, through skin);
- Provide rationale for the chemical substance prioritization;
- Characterize potentially adverse effects of chemical substances;
- Determine adverse effects.

Pollutants are selected mainly based on how they affect human body, their toxic effects and exposure probability.

Any motor road is a pollution source due to exhaust gases and aerosols which trigger many illnesses, therein including pulmonary diseases. Immunocompromised people, children and elderly are especially susceptible to pollution exposure. Reduction of air pollution is a prime objective which drives countries to actively develop new quality standards for fuels and exhaust treatment.

During 2019 and 2020 Green InterTraffic project participants were making an inventory of pollutants. The Finish Meteorological Institute and the Institute of Radar Meteorology were the parties responsible for data collection. Calculations were done for nitrogen dioxide, ozone, carbon oxide, particulate matter PM2.5 and PM10 based on monitoring data registered by Vaisala AQT sensors installed at 4 sites (2 sensors in Russia and 2 sensors in Finland). See Figure 1 for the sensor locations on the map.



Figure 1. Locations of AQT Sensors

All examined substances (particulate matter, nitrogen dioxide, ozone and carbon oxide) are not featured in *The List of Substances, Foods, Household or Natural Factors that Are Cancerogenic to Humans* (Hygienic Standards ГН 1.2.1841-04).

Hazard parameters of non-cancerogenic substances are evaluated using reference levels of acute and chronic effects of chemical compounds included into the preliminary list of studied compounds. Critical organs/systems and effects associated with the determined reference concentrations were also identified. Table 1 presents reference levels of acute (ARfC) and chronic (RfC) exposures as per *P 2.1.10.1920-04 Guidelines for Risk Assessment of Chemical Environmental Pollutants to Public Health*.

Table 1. Non-Cancerogenic Risk Hazards [1]

	Nitrogen Dioxide	PM <sub>2.5</sub>	PM <sub>10</sub>	Carbon Oxide
CAS	10102-44-0	–	–	630-08-0
RfC, mg/m <sup>3</sup>	0.04	0.015	0.05	3
Critical Organs and Systems	Respiratory Organs, Blood System	Respiratory Organs	Respiratory Organs, Cardiovascular System	Blood System, Cardiovascular System, Development, CNS
ARfC, mg/m <sup>3</sup>	0.47	0.065	0.15	23
Critical Organs and Systems	Respiratory Organs	Respiratory Organs, Systemic Impact	Respiratory Organs, Systemic Impact	Cardiovascular System
Class of Hazard	3			4
MPCda	0.04	0.035	0.06	3
MPCms	0.085	0.16	0.3	5

### 3. Choosing the Dose-Response Relationship

Assessment of the dose-response relationship is a major stage in the overall assessment of public health risk associated with the exposure to chemical substances. This is the stage to determine quantitative parameters of the link between an exposure to the studied factor and thereby caused adverse effects.

The assessment allows to quantify the obtained information and to establish links between pollutant concentration (dose) and cases of adverse effects on public health. This stage aligns all available data on risk-free exposure levels, hygienic standards, affected body organs and adverse effects. Hazard parameters of studied substances are reviewed along with the quantified dose-response profile. Non-cancerogenic substances produce no hazardous effects at concentrations below determined thresholds.

The exposure analysis was guided by the following parameters:

- Exposure duration that includes chronic exposure of 30 years for adults;
- Life-long exposure equals to average life expectancy of 70 years;
- Rate of inhalation exposure, i.e., general parameter ( $20 \text{ m}^3/\text{day}$ ).

The assessment of dose-response relationship is based on the data developed through epidemiological studies. A full description of pollutants is provided below and includes hazard indicators of developing non-cancerogenic effects and hazards associated with the examined chemicals. This information was taken from epidemiological studies or clinical research.

The risk of developing non-cancerogenic effects is often described via the following parameters: maximum non-effective dose and minimum dose that produces a threshold effect. These values set reference doses for concentrations of pollutants. Once these doses are exceeded, the likelihood of pathological changes will increase.

In the assessment of effects from the studied pollutants this paper has analyzed the data available on pollutants' safe doses and their effects on public health if inhaled. It has also summarized the data of epidemiological studies and clinical research.

## Substance Properties

### Nitrogen Dioxide

It is a reddish-brown toxic gas (melting point is  $-11.2^\circ\text{C}$ , boiling point is  $-21^\circ\text{C}$ ). Nitrogen dioxide is a paramagnetic, bent molecule. In the environment nitrogen dioxide reacts with atmospheric moisture, forms nitric acid and may significantly corrode metals. Nitrogen dioxide absorbs visible-light spectrum and may reduce visibility; it is also harmful to crops [2].

Nitrogen dioxide primarily forms through burning of organic fuels, use of automobile combustion engines, which is responsible for up to 80% of the gas in the air. In nature nitrogen dioxide is emitted at coal/peat burning and during forest fires.

Nitrogen dioxide irritates eyes, skin and respiratory tract. In lungs the gas may provoke pulmonary edema. It affects respiratory organs; a prolonged contact induces bronchitis and asthma. High level exposure may be fatal. The effects can be deferred. Having a strong oxidizing potential, the substance reacts vigorously with combustible materials and reducers. Nitrogen dioxide reaction with ozone may result in forming of nitric acid.

In lungs nitrogen dioxide (toxic gas) reacts with moisture and forms nitric and nitrous acids which react with tissue alkalis to form nitrates and nitrites. While nitrates have no tangible effect on human body, nitrites may provoke the following changes: blood pressure drop or central nervous system depression. Children with chronic respiratory diseases and patients with bronchial asthma are especially sensitive to nitrogen dioxide (0.19 mg/m<sup>3</sup> concentration initiates a bronchial spasm).

MPCda rise by 30 µg/m<sup>3</sup> leads to a 20% increase in respiratory disease incidence among children under 12 y.o. MPCda rise by 10 µg/m<sup>3</sup> makes the duration of asthmatic attacks 6,5% longer. P 2.1.10.1920-04 *Guidelines for Risk Assessment of Environmental Chemical Pollutants to Public Health* recognize the following organs and systems as critical at chronic nitrogen dioxide exposure: respiratory organs and blood; acute exposure affects respiratory organs.

## Ozone

Under normal conditions ozone is a pale blue poisonous gas with a distinctly pungent (somewhat metallic) smell. The gas boiling and melting points are, respectively, - 111.8°C and - 197.2°C. It is a powerful oxidant far more reactive than dioxygen. The reaction product is, most often, oxygen. Free radicals of oxygen are produced in many reactions involving ozone thus making it a highly toxic gas. Ozone is generated by photochemical reactions with nitrogen oxides, hydrocarbons and other substances. Ozone plays a positive role at the top atmospheric layers where it reduces sun radiation. However, the gas is harmful to human health at the earth surface. Significant concentrations of the gas cause dryness of the mouth, coughing, eye irritation, difficult breathing and dyspnea. Ozone exposure increases risk of cardiovascular diseases. Ozone reaction with cholesterol produces insoluble compounds and subsequently leads to atherosclerosis.

Sanitary and hygienic standards put ozone into the top (1<sup>st</sup>) class of hazards. It has an expressed irritating and general toxic impact. Ozone hazardous potential is higher than that of chlorine. Even in small quantities it already can irritate mucous surfaces, exacerbate respiratory tract diseases, provoke headaches, nausea, cough and respiratory dysfunction. Prolonged exposure results in chronic pulmonary diseases and weakened immune system. European researchers proved that

good health status is preserved until ozone concentration stays under  $100 \mu\text{g}/\text{m}^3$ . Concentration increased to  $120 \mu\text{g}/\text{m}^3$  brings the mortality 0,5% up. For instance, some USA studies demonstrated that every third American is hypersensitive to even small ambient ozone concentrations [3].

Ozone concentrations mostly rise on hot days. Adults who spend much time outdoors are most sensitive to ozone exposure as well as children, ozone-sensitive individuals, elderly and persons with chronic respiratory and cardiovascular diseases [4].

### **PM2.5, PM10 Fine Dust or Aerosols**

PM10 are particulate aerosols of  $10 \mu\text{m}$  or less in diameter, while PM2.5 are solids of  $2.5 \mu\text{m}$  or less in diameter.

Aerosols include soot particles, heavy metal compounds, pollen, bacteria, soil particles, tire/stud/asphalt wear particles, etc.

At no-wind conditions, aerosol in-the-air concentration increases. High contents of dust and soot particles absorb more radiation thus increasing the surface air temperature. Population exposed to heavy aerosol pollution develops a significantly higher incidence of respiratory and eye diseases, cardiovascular system malfunction and a variety of allergic reactions. Aerosols have chronic implications which means that continued chronic exposure will provoke negative physiological developments in the body. [5,6,7] The majority of world's population suffers because of that exposure. The smaller is the diameter, the deeper can particles penetrate into lungs. Particles of over  $10 \mu\text{m}$  will settle down already in the nose and throat. Particles under  $5 \mu\text{m}$  will go into larynx and main bronchi and will attain lungs. Fines particles under  $1 \mu\text{m}$  in diameter will reach alveolae and enter into the blood system. Small exposures to PM2.5 are dangerous for their deferred effects as the accumulated particles may later lead to serious health issues.

Particulate matter broadly and negatively impacts human's respiratory system and blood circulation. Aerosols with absorbed toxic substances in general adversely affect public health and depress body functions. Particulate matter most commonly affects children, elderly and persons with cardiovascular diseases or asthma. Research shows that aerosol concentrations of PM2.5 at  $20 \mu\text{g}/\text{m}^3$  or PM10 at  $30 \mu\text{g}/\text{m}^3$  promote the incidence rate of bronchitis and reduce lung function.

### **Carbon Oxide**

It is a colourless and tasteless gas which remains in the atmosphere for about 2 months on average. In nature carbon oxide balance is regulated by further oxidation to  $\text{CO}_2$  in the stratosphere or via absorption by earth fungi, microorganisms or by higher/inferior plants.

## Toxic Effect on Humans

Chiefly, carbon oxide acts through forming carboxyhemoglobin which reduces oxygen transport to body tissues. CO is highly focused in action. High carbon oxide content causes various degrees of poisoning that is most dangerous for women and children. Acute poisoning manifests itself through general symptoms like headache, dizziness and fatigue. Carbon oxide usually does not irritate respiratory tract. Chronic poisoning manifests through asthenia, encephalopathy, respiratory and cardiovascular disorders; and carboxyhemoglobin concentration in blood is above the norm.

The nervous system responds to the impact with noises in one's head and headaches, dizziness, sense of carbon-monoxide poisoning, increased tiredness, weaker memory, reduced attention, apathy, irritancy and loss of physical coordination. It affects hematopoietic system as inhaled carbon oxide vapors raise hemoglobin or, to the contrary, may onset hypochromic anemia; hemogram is shifted to the left with the elevated level of carboxyhemoglobin [8].

The affected cardiovascular system then develops arrhythmia, tachycardia, ectopic heartbeat, unstable pulse and blood pressure with a hypotonic trend (however, hypertonic disease may sometimes develop) and stenocardia. Persons with coronary vessels disease, cerebrovascular and periphery vascular system malfunctions, anemia, pulmonary disease and under high physical strain are in the risk group for carbon oxide effects. In concentrations of 9-16 mg/m<sup>3</sup> carbon oxide may increase myocardial infarction mortality. P 2.1.10.1920-04 *Guidelines for Risk Assessment of Environmental Chemical Pollutants to Public Health* recognize the following organs and systems as critical under chronic carbon oxide exposure: blood, cardiovascular system, development, CNS; while acute exposure affects cardiovascular system and development.

Epidemiologic research had produced indicators to assess risks across a wide range of negative changes in public health. Effects of monitored substances are listed in Table 2.

**Table 2. Concentration-to-Response Relations Obtained via Epidemiologic Research [1]**

Compound	Effect
Nitrogen Dioxide	Increased incidence of upper respiratory tract symptoms Longer exacerbations of upper respiratory tract diseases Increased incidence of lower respiratory tract diseases
PM2.5; PM10	Total mortality Cardiovascular mortality Respiratory tract disease mortality Number of children and adolescents with bronchitis (under 18 y.o.) Incidence of upper respiratory tract symptoms Incidence of lower respiratory tract symptoms Coughing incidence (man/days) Health care requests due to respiratory diseases Incidence of bronchial asthma exacerbations
Ozone	Asthma attacks Slight reduction of daily activities Hospitalization due to respiratory illnesses
Carbon Oxide	Change in carboxyhemoglobin percentage in blood Rate of hospitalization and/or health care requests due to heart diseases (for seniors of 65 y.o. and older) Changed frequency of angina attacks for nonsmoking patients at age 35-37 y.o.; the percent reduction in the interval duration between attacks

#### **4. Exposure Assessment**

Exposure means a bodily contact with a chemical, physical or biological agent. Exposure value means the agent amount - either measured or estimated - within a specific volume of ambient medium that contacts border organs (lungs, gastrointestinal tract, skin) of human body within a certain time. Exposures were assessed at 4 posts. The chemical impact scenario was direct, where the surveyed population is exposed to chemical agents via direct human-to-ambient air contact; the air is the impacting medium. The pollutant pathway is via inhaling the ambient air. The assessment of risk to public health under the scenario where toxic agents have multi-media pathways to human bodies was recognized as unsuitable because the surveyed area has no sources of water supply or foodstuffs manufacturing sites. Having reviewed the possible routes of human exposure to hazardous substances, we may conclude that a public health risk related to operations of Scandinavia

highway and on the adjacent territories will be primarily determined by chemical substances inhaled from the outside air.

## 5. Risk Characterization

Risk characterization is a final stage of risk assessment and includes some initial elements of risk management. This stage integrates information collected during identification of hazards, evaluates the exposure-response relationship, describes risks associated with the impact of factors (singularly or combined), determines the likelihood and severity of adverse effects for human health. Risk values are calculated separately for cancerogenic and non-cancerogenic effects.

Risk for development of non-cancerogenic effects can be profiled either by comparing actual vs safe exposure levels (hazard index/quotient) or by using the values in the “concentration-to-response” relationship (Table 2) obtained through epidemiological surveys. The risk is estimated for specific substances with the hazard quotient calculated using the equation below:

$$HQ = AC/RfC \text{ where} \quad (1)$$

HQ - hazard quotient;

AC - average concentration, mg/m<sup>3</sup>;

RfC - reference (safe) concentration, mg/m<sup>3</sup>.

The risk for development of non-cancerogenic effects of combined and complex exposure to chemical substances is characterized by calculating the hazard index (HI). The following HI equation is used to determine the simultaneous exposure to several substances via the same pathway (for instance, inhaling or oral):

$$HI = \sum HQ_i, \text{ where} \quad (2)$$

$HQ_i$  – hazard indices for specific components in the mix of affecting substances.

## 6. Risk Calculation

Data review outcomes for Scandinavia highway are presented below. The data were collected via AQT sensors manufactured by Vaisala.

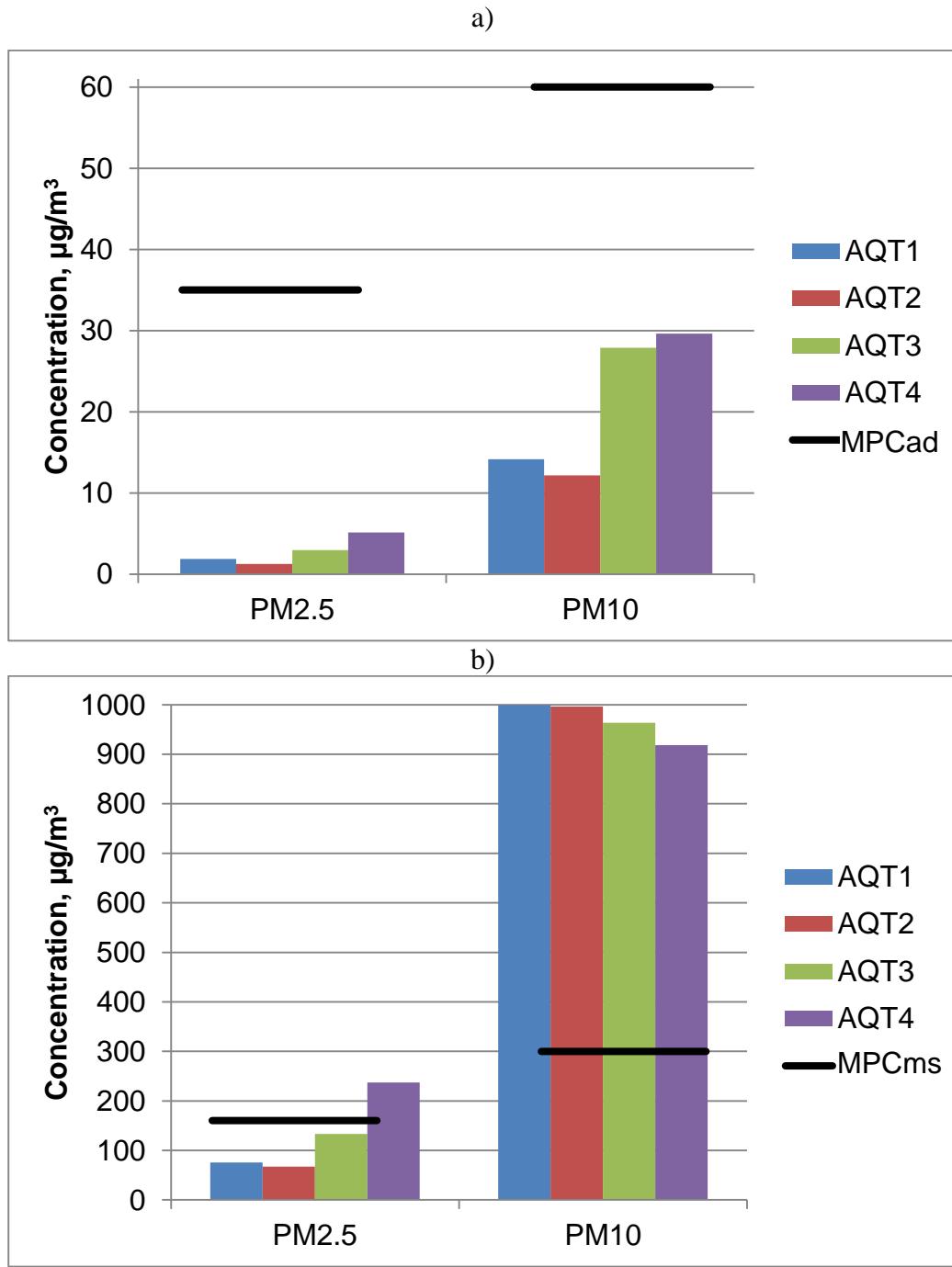


Figure 2. Average (a) and maximum (b) concentration values for suspended particulate matter during the 2019-2020 monitoring compared to adjusted values of daily average (MPCda) and maximum single (MPCms) Maximum Permissible Concentrations

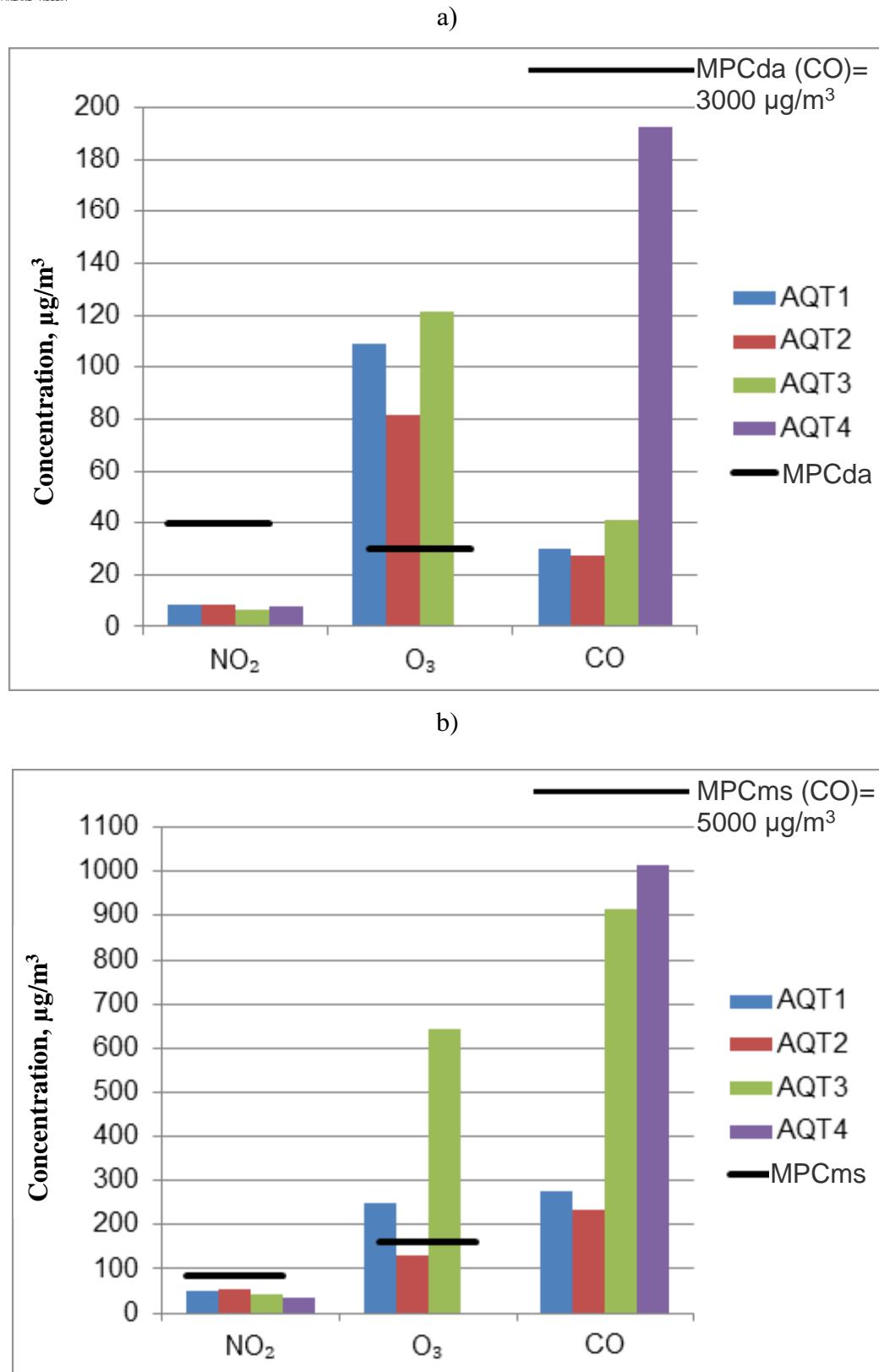


Figure 3. Average (a) and maximum (b) concentration values for gases during the 2019-2020 monitoring compared to adjusted values of daily average (MPCda) and maximum single (MPCms) Maximum Permissible Concentrations.

For technical reasons, ozone concentrations were unavailable for AQT4 sensor. Average annual ozone concentrations at AQT1, AQT2 and AQT3 observation posts were 2.7 to 4 times higher than the average daily MPC. Other measured substances had their average annual concentrations within the MPCda limits. The average annual concentration of carbon oxide registered by AQT4 was between 5 to 7-fold higher the values at other observation posts. Short-term surges in PM10 concentrations were detected all along the highway with the values more than 3-fold exceeding the maximum single MPC. For PM2.5 the 1.5-fold exceedance of maximum single MPC was detected only at the location of AQT4 sensor. At AQT1 (in Finland) and AQT3 (in Russia) short-term ozone surges, respectively, 1.6 and 4-fold above the maximum single MPC were detected.

Table 3. Number of Surges above the Maximum Single (MPCms) and Daily Average (MPCda) MPCs during the 2019-2020 Monitoring

Substance	MPC Type	AQT1	AQT2	AQT3	AQT4
NO <sub>2</sub>	MPCms	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	MPCda	0 (0%)	0 (0%)	0 (0%)	0 (0%)
PM2.5	MPCms	0 (0%)	0 (0%)	0 (0%)	8 (0.15%)
	MPCda	0 (0%)	2 (0.8%)	0 (0%)	3 (1.23%)
PM10	MPCms	47 (0.76%)	34 (0.57%)	94 (1.47%)	72 (1.33%)
	MPCda	13 (5.06%)	9 (3.61%)	28 (9.93%)	40 (16.39%)
O <sub>3</sub>	MPCms	0 (0%)	0 (0%)	12 (0.19%)	—
	MPCda	99 (39.29%)	89 (36.33%)	160 (54.61%)	—
CO	MPCms	0 (0%)	0 (0%)	0 (0%)	0 (0%)
	MPCda	0 (0%)	0 (0%)	0 (0%)	0 (0%)

During 2019 and 2020 the most frequent surges above MPCda were registered for ozone at AQT1, AQT2 and AQT3 with the respectful values of 39.29%, 36.33% and 54.61%. Along Scandinavia highway PM10 demonstrated the highest number of MPCms exceedances with the values of 4.34% in Finland and 13.16 % in Russia.

The estimated Scandinavia highway contribution to the non-cancerogenic risk is detailed in Tables 4 and 5.

Table 4. Non-Cancerogenic Risk of Background Concentrations and Non-Cancerogenic Risk Contributions by Scandinavia Highway Pollutants during the 2019-2020 Monitoring

Substance		NO <sub>2</sub>	PM2.5	PM10	O <sub>3</sub>	CO
Average concentration, $\mu\text{g}/\text{m}^3$	AQT1	8.4	1.9	14.2	108.8	30.2
	AQT2	8.5	1.3	12.2	81.7	27.3
	AQT3	6.5	3.0	27.9	121.4	41.1
	AQT4	7.5	5.1	29.6	—	192.5
Background Concentration, $\mu\text{g}/\text{m}^3$		3.7	—	9	19.7	17.5
RfC, $\mu\text{g}/\text{m}^3$		40	15	50	30	3000
HQ	Background	0.09	—	0.18	0.66	0.01
	AQT1	0.21	0.13	0.28	3.63	0.010
	AQT2	0.21	0.08	0.24	2.72	0.009
	AQT3	0.16	0.20	0.56	4.05	0.014
	AQT4	0.19	0.34	0.59	—	0.064
Highway HQ within the background HQ, %	AQT1	55.8	—	36.4	81.9	42.0
	AQT2	56.6	—	26.1	75.9	35.9
	AQT3	43.2	—	67.7	83.8	57.5
	AQT4	51.0	—	69.6	—	90.9

Table 5. Non-Cancerogenic Risk Values of Background Concentrations and Non-Cancerogenic Risk Inputs by Acute Exposure to Scandinavia Highway Pollutants during the 2019-2020 Monitoring

Substance		NO <sub>2</sub>	PM2.5	PM10	O <sub>3</sub>	CO
Maximum Concentration, $\mu\text{g}/\text{m}^3$	AQT1	49.3	75.4	999.3	250.0	275.0
	AQT2	55.1	67.1	996.4	131.5	232.1
	AQT3	41.8	133.4	963.4	642.0	913.6
	AQT4	34.2	237.3	918.3	—	1014.8
Background Concentration, $\mu\text{g}/\text{m}^3$		3.7	—	9	19.7	17.5
ARfC, $\mu\text{g}/\text{m}^3$		470	65	150	180	23000
HQ	Background	0.01	—	0.06	0.11	0.00076
	AQT1	0.10	1.16	6.66	1.39	0.012
	AQT2	0.12	1.03	6.64	0.73	0.010
	AQT3	0.09	2.05	6.42	3.57	0.040
	AQT4	0.07	3.65	6.12	—	0.044
The highway HQ within the background HQ, %	AQT1	92.5	—	99.1	92.1	93.6
	AQT2	93.3	—	99.1	85.0	92.5
	AQT3	91.2	—	99.1	96.9	98.1
	AQT4	89.2	—	99.0	—	98.3

For all substances (except ozone) the non-cancerogenic risk of chronic exposure was below 1. Among the rest monitored substances, PM10s came with the highest values between 0.24 (AQT2 in Finland) and 0.59 (AQT4 in Russia), while carbon oxide demonstrated the lowest values (below 0.1). Average values of non-cancerogenic risk of chronic exposure to individual substances in Russia and Finland were as follows:

- 0.21 (in Finland) and 0.175 (in Russia) for nitrogen dioxide;
- 0.105 (in Finland) and 0.27 (in Russia) for PM2.5;
- 0.26 (in Finland) and 0.575 (in Russia) for PM10;
- 3.175 (in Finland) and 4.05 (in Russia) for ozone;
- 0.0095 (in Finland) and 0.039 (in Russia) for carbon oxide.

The chronic exposure contribution by Scandinavia highway pollutants to the overall non-cancerogenic risk looks as follows:

- for nitrogen dioxide – 56.2% (Finland) and 47.1% (Russia);
- for PM10 – 31.25% (Finland) and 68.65% (Russia);
- for ozone – 78.9% (Finland) and 83.8% (Russia);
- for carbon oxide – 38.9% (Finland) and 74.2% (Russia).

Both in Finland and in Russia the non-cancerogenic risk of acute exposure to nitrogen dioxide did not exceed 0.1 and to carbon oxide was below 0.1. Non-cancerogenic risk of PM2.5 averaged for 2 monitoring posts in Finland came as 1.1, and in Russia, as 2.85. The suspended PM10 particulates demonstrated the highest risk level of 6.27 in Russia and 6.65 in Finland.

Thus, the highway's contribution into the total non-cancerogenic risk of acute exposure to pollutants looks as follows:

- for nitrogen dioxide: 92.9% (Finland) and 90.2% (Russia);
- for PM10: 99.1% (Finland) and 99.05% (Russia);
- for ozone: 88.55% (Finland) and 96.9% (Russia);
- for carbon oxide: 93.05% (Finland) and 98.2% (Russia).

The data were used to calculate hazard index (HI) values of chronic exposure for the entire monitoring duration (Table 6).

**Table 6. Calculated Hazard Indices (HI) of Chronic Exposure during the 2019-2020 Monitoring**

Sensor	HQ					HI		
	NO <sub>2</sub>	PM2.5	PM10	O <sub>3</sub> *	CO	Total	Respiratory Organs	Cardiovascular System
AQT1	0.21	0.13	0.28	3.63	0.010	0.63	0.62	0.42
AQT2	0.21	0.08	0.24	2.72	0.009	0.55	0.54	0.34
AQT3	0.16	0.20	0.56	4.05	0.014	0.93	0.92	0.77
AQT4	0.19	0.34	0.59	–	0.064	1.19	1.12	1.00

Remark: Hazard quotient for ozone was not included into the hazard index calculation due to a plausible overstatement in the received data.

Comparative non-cancerogenic risk (HI) indices were calculated for all substances except ozone. Calculations under the equation (2) resulted in hazardous indices of chronic exposure between 0.59 (in the Republic of Finland) and 1.06 (in the Russian Federation). The highest HI indices were detected at the location of AQT4 sensor with the input into the total HI per substances as follows: PM2.5 – 0.34, PM10 – 0.59, nitrogen dioxide – 0.19, carbon oxide – 0.06.

Chronic exposure risk at monitoring posts in Finland (AQT1 and AQT2) and in Russia (AQT3) was acceptable (HI<1). The acceptable (minimum) risk is associated with the background incidence of diseases among population. Therefore, the risk does not require any extra measures and just needs to be periodically monitored [1]. At the location of AQT4 sensor the chronic risk of non-cancerogenic hazard was somewhat above 1. This is a low risk grade associated with an increased trend in the background incidence of diseases. Such levels must be continuously monitored. At some instances the risk levels may trigger additional risk-reduction measures.

A risk of non-cancerogenic effects at acute exposure was assessed for nitrogen dioxide, carbon oxide, PM2.5 and PM10. Inhalation was identified as the primary pathway for chemicals into the human body, while outdoor air was determined the medium under analysis. The assessed risk of non-cancerogenic effects at acute exposure demonstrated the minimum risk (HQ below 0.1) of nitrogen dioxide and carbon oxide exposure, except for the Finnish territory where the nitrogen dioxide exposure risk was graded as low (HQ between 0.1 and 1). The assessment of chronic and acute non-cancerogenic risk has identified particulate matter (PM) as the prime pollutant among highway emissions. The PM2.5 exposure risk was moderate (HQ between 1 and 5). The highest

impact is associated with PM10 where the risk is graded as high (HQ between 5 and 10). High risk levels are linked with the statistically significant increase in the background disease incidence among the population. The risk requires development and systematic implementation of curative measures at the populated areas. These risk-reduction actions must be underpinned by a deeper assessment of various aspects in the challenge at hand.

Average annual concentrations of NO<sub>2</sub> and PM2.5 across all monitoring locations in the Russian Federation and in the Republic of Finland were within the values recommended by the WHO. Average annual concentrations of PM10 in Finland meet the WHO guideline values. In Russia average annual concentrations of PM10 are marginally above the guideline value which – in the long run – will stipulate for the mortality risk at 3% higher vs the risk associated the WHO recommended level [9,10].

## CONCLUSIONS

From October 2019 through July 2020 average values of pollutants registered at two measuring posts in Russia and at two measuring posts in Finland were as follows:

NO<sub>2</sub>: 8.5 µg/m<sup>3</sup> in the Republic of Finland and 7 µg/m<sup>3</sup> in the Russian Federation;

PM<sub>2.5</sub>: 1.6 µg/m<sup>3</sup> in the Republic of Finland and 4.1 µg/m<sup>3</sup> in the Russian Federation;

PM<sub>10</sub>: 13.2 µg/m<sup>3</sup> in the Republic of Finland and 28.8 µg/m<sup>3</sup> in the Russian Federation;

CO: 95.3 µg/m<sup>3</sup> in the Republic of Finland and 112.7 µg/m<sup>3</sup> in the Russian Federation.

Average annual pollutant concentrations were within maximum permittable concentrations.

At AQT4 the average annual concentration of carbon oxide was 5 to 7-fold higher than those of AQT 1, 2 and 3; however, it was still much below the MPCda. Short-term significant increases (vs annual data) were registered for PM concentrations maximizing at 237.27 µg/m<sup>3</sup> (PM<sub>2.5</sub>) and 999.31 µg/m<sup>3</sup> (PM<sub>10</sub>). These values 1.5-fold exceeded the MPCms for PM<sub>2.5</sub> and 3.3-fold for PM<sub>10</sub>.

The highest number of exceeded MPCms was registered only for PM<sub>10</sub>: at AQT3 (Russia) and at AQT1 (Finland). Rare instances of exceeded MPCms and MPCda for PM<sub>2.5</sub> were registered only at AQT4. The least polluted territory in Finland was at AQT2 and in Russia at AQT3.

Average annual concentrations of NO<sub>2</sub> and PM<sub>2.5</sub> across all observation sites in Russia and in Finland stayed under the WHO recommended limits. Average annual concentrations of PM<sub>10</sub> in Finland meet the WHO guideline value while in Russia PM<sub>10</sub> average annual concentrations are marginally above the guideline value which – in the long run –stipulates for the mortality risk at 3% higher vs the risk associated the WHO recommended level.

Inhaling was determined as the prime pathway of body exposure to chemical substances. Outdoor air was the analyzed medium. Reference inhaling concentrations were used to evaluate emissions according to comparative non-cancerogenic hazard of each compound. P 2.1.10.1920-04 *Guidelines for Risk Assessment of Environmental Chemical Pollutants to Public Health* provided reference concentrations and hazard parameters of developing non-cancerogenic effects of chronic inhaling as well as critical organs and systems.

The estimated non-cancerogenic health risk at chronic exposure to all reviewed substances was below 1 (ozone was not included into the calculation). This risk is characterized as low or permissible. The highest values were determined for PM<sub>10</sub> with the risks (HQ) of 0.28 in Finland (AQT1) and 0.59 in Russia (AQT4). The least values were determined for carbon oxide (HQ under 0.1) in Russia and in Finland. It corresponds to the minimum (permissible) risk associated with the background disease incidence among population. The risk requires no extra measures and just needs to be periodically monitored.

Non-cancerogenic risk of acute exposure to PM<sub>2.5</sub> was 1.1 in Finland and 2.85 in Russia. The risk is graded as moderate. While permissible for professional groups, the risk is not permissible

for general population. Such risk requires developing and implementing systemic improvement measures across populated areas. The highest risk levels were determined for PM10: 6.42 in Russia (AQT3) and 6.65 in Finland (AQT1). Graded as high, the risk level is associated with statistically significant increase in the background disease incidence among population.

Scandinavia highway's chronic pollutant effect makes the following contribution to the overall non-cancerogenic risk: nitrogen dioxide – 56.2% (Finland) and 47.1% (Russia); PM10 – 31.25% (Finland) and 68.65% (Russia); carbon oxide – 38.9% (Finland) and 74.2% (Russia). Highway's contribution into the total non-cancerogenic public health risk at acute exposures is as follows: nitrogen dioxide – 92.9% (Finland) and 90.2% (Russia); PM10 – 99.1% (Finland) and 99.05% (Russia); carbon oxide – 93.05% (Finland) and 98.2% (Russia).

Hazard Index (HI) was calculated for critical body organs and affected body systems. It has used hazard quotient values determined for separate components in the mixture of affecting chemicals. The HI allows to assess the risk of developing non-cancerogenic effects at a combined monodirectional exposure of specific body organs and systems to the studied chemicals.

Nitrogen dioxide and particulate matter affect respiratory organs; carbon oxide affects the CNS; and two compounds - carbon oxide and particulate matter – affect cardiovascular system.

The determined comparative non-cancerogenic hazard indices (HI) at chronic exposure of body organs have the following range: respiratory organs from 0.54 (AQT2 in Finland) to 1.12 (AQT4 in Russia), cardiovascular system from 0.43 (AQT2 in Finland) to 1 (AQT4 in Russia).

HI values below 1 are graded as minimal risk (acceptable). Chronic exposures to studied pollutants at sites of AQT 1, 2 and 3 are associated with the background incidence of diseases and perceived by the population as negligible and not different from everyday risk. As such, the risk does not require any additional risk-reduction measures and shall be monitored from time to time in order to keep the air pollution at the low level.

The AQT4 site was graded as low risk that associates with an increased trend in the background disease incidence. The risk is ranked as conditionally acceptable (permissible). Such level requires continuous monitoring and sometimes additional risk-reduction measures. Most hygienic standards of international organizations are specifically set for this health risk level.

The assessed risk of non-cancerogenic effects at acute exposure has demonstrated that the impact level of nitrogen dioxide and carbon oxide can be characterized as minimum (HQ under 0.1), except for the Finnish territory where the nitrogen dioxide impact level was graded as low (HQ from 0.1 to 1). The assessment of chronic and acute non-cancerogenic risk has identified particulate matter (PM) as the prime risk-generating pollutant among highway emissions. The PM2.5 exposure risk was moderate (HQ between 1 and 5). The highest impact is associated with PM10 where the risk was graded as high (HQ between 5 and 10). High risk level is linked with the statistically significant increase in the background disease incidence among population. The risk requires curative measures developed and systematically implemented at permanently populated areas. The

specific planned risk-reduction activities must be underpinned by a deeper assessment of various aspects in the challenge at hand.

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