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Human health risk assessment of industry impact in Kikinda industry zone

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ABSTRACT

In industrial zones such as the municipality of Kikinda (presented in this manuscript) it is necessary to monitor environmental pollution and take measures to protect the environment from the effects of harmful polluting and toxic substances (especially from heavy metals). The risk assessment model developed by the US EPA was used to assess the health risks posed by emissions of heavy metals in the air in Kikinda industry zone. It can be concluded that no increased level of risk was identified from heavy metals content in the air from industry systems in the area of the industrial zone in Kikinda, however, it is necessary to apply preventive measures in order to reduce the accumulation of heavy metals in the environment due to activities that are constant in that area.

1. Introduction

Most heavy metals (Cd, Pb, Ni, As, Cr, Hg…) are harmful and dangerous substances which, in addition to polluting the environment, have a very toxic effect in higher concentrations, both on plants and animals and on human health. Therefore, in the last twenty years, public awareness has been developed of the need to monitor environmental pollution and take measures to protect the environment from the effects of harmful substances in general, and especially from heavy metals, because their concentration in soil, water, and air is getting bigger every day. The data indicate that about 150,000 tons of nickel from natural sources and 180,000 tons originating from industrial activities are released into the environment annually, primarily during the emission of fossil fuel combustion, industrial production, and industrial waste rich in nickel (IARC, 1990).

The International Committee on Nickel Carcinogenesis in Man in 1990 suggested that the risks of developing respiratory tumors were associated with exposure to soluble nickel concentrations above 1 mg/m^3, and exposure to less soluble forms at concentrations above 10 mg/m^3. The Committee could not determine with certainty the level at which exposure to nickel becomes substantially hazardous. About 2 % of workers in the nickel industry are exposed to dust particles containing nickel in concentrations from 0.1 to 1 mg/m^3 (Ilić et al., 2007). Exposure to nickel compounds can cause a variety of adverse effects on human health. Nickel allergy in the form of contact dermatitis is the most common reaction.

Although the accumulation of nickel in the body through chronic exposure can lead to pulmonary fibrosis, cardiovascular and kidney disease, the greatest danger is associated with the carcinogenic effects of nickel.

Human exposure to nickel is most often experienced through inhalation and ingestion and is particularly high in metallurgical workers engaged in nickel processing. In developed environments and large cities, atmospheric

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nickel concentrations are associated with dust generated by the combustion of fossil fuels in power plants and automobiles, and can reach 120-170 ng/m^3 compared to 6-17 ng/m^3 in less industrialized areas. (Norseth and Piscator, 1979)

Anthropogenic sources produce about three-quarters of total arsenic emissions into the atmosphere. Significant amounts of arsenic come from the combustion process of fuels (coal and heavy oils), the iron and steel industry, and the production of copper and zinc. The largest natural sources of arsenic are: volcanic activity, fires, mineral decomposition processes, and the activity of microorganisms (in wetlands).

Arsenic mainly occurs in fine fractions of suspended particles (up to 2.5 μm in diameter), which can be transported over long distances and as such can easily penetrate the respiratory system. Almost all forms of arsenic in the air are in the form of particles with an aerodynamic diameter of up to 10 μm.

Previous experimental animal studies and epidemiological studies on the human population have demonstrated the toxic and carcinogenic properties of arsenic. According to the IARC classification, inorganic trivalent arsenic is classified in group I, which means that it has been proven to be carcinogenic to humans, while pentavalent inorganic arsenic and organic arsenic, as well as their compounds are classified as toxic substances. (IARC, 2012) The WHO has estimated that daily intake of water containing 20 mg/L of arsenic for 70 years will increase the likelihood of cancer by 5 % (WHO, 2011).

The degree of toxicity of arsenic depends on the inorganic or organic form and the state of oxidation of arsenic. Prolonged exposure to even low concentrations of arsenic can cause lung cancer and bleeding, cardiovascular, pulmonary, immune, and neurological disorders. The complete mechanism of arsenic action in the body is not yet known. Chronic arsenic poisoning can be the result of the accumulation of arsenic compounds in the body. One of the symptoms of chronic toxicity in humans, resulting from oral exposure to arsenic, are skin lesions. They are characterized by hyperpigmentation, hyperkeratosis, and hypopigmentation.

Vascular disease of the black foot, which causes gangrene of the lower extremities, has also been detected in humans with chronic arsenic exposure (Sharma and Sohn, 2009). Acute toxicity is associated with the chemical form and oxidative state of arsenic. In adults, the lethal dose of arsenic was estimated at 1-3 mg As/kg (De Zuane, 1997). Characteristics of severe acute toxicity include gastrointestinal distress, vomiting, diarrhea, anuria, convulsions, coma, and death. Lead as a pollutant can be detected in all basic environmental factors and biological systems. Sources of lead contamination are combustion products in metallurgy and chemical industry, industrial wastewater, landfills, and traffic. In atmospheric precipitation, the total lead content ranges from 1-50 μg/dm^3 (Goyer and Clasen, 1995).

From the atmosphere, soil, and water (surface and groundwater), lead is introduced and retained in plants, and further through the food chain and drinking water reaches the human body. In addition to food and drinking water, lead can also be ingested through air contaminated with fossil fuel combustion products (Goyer and Clasen, 1995). The introduced lead is deposited mostly in the bones, then in the liver, kidney, spleen, nervous tissue, and muscles. A blood lead concentration of 60 μg/dL was considered safe during the 1960s. Over time, by studying the toxic effects of lead, the acceptable content in the blood was reduced to 25 μg/dL and then to 10 μg/dL in 1991. Regardless of these changes, subclinical symptoms of lead exposure also occur at a content of less than 10 μg/dL. However, a harmless level of lead in the body has not yet been defined (Ahamed and Siddiqui, 2007).

According to the physic - chemical properties, Pb^{2+} -ion can easily replace Ca^{2+} -ion in calcified tissues (bones and teeth), but also in various soluble complexes of this metal with bioligands in biological fluids and tissues.

Lead in bones contributes to the development of osteoporosis, decreased bone mass, changes in structure, and increased bone resorption in the elderly (Kaličanin et al., 2004).

Daily amount of ingested lead, orally and by inhalation, can be around 0.3 mg. It is partially eliminated from the body by excretion, but it also accumulates, so that about 250 μg/dm^3 can normally be found in the blood. An increase in the levels of this metal in the blood is moderately risky for values (250-490 μg/dm^3), highly risky for (500-690 μg/dm^3), and urgent with more than 700 μg/dm^3 in body fluid (Munoz and Palermo, 2006).

Lead from drinking water is probably more absorbed than lead from food. According to some studies, adults absorb 35 to 50 % of the ingested metal, and the percentage of absorption for children can be higher than 50 %. In addition to age, the absorption of lead is also affected by the general physiological state of the organism (Flora et al., 2006). Death from acute human poisoning can occur with the intake of 25 to 30 g of soluble lead salts. Chronic exposure to this heavy metal leads to mental retardation, psychosis, hyperactivity, weight loss, and muscle weakness and paralysis. Increased presence of this metal is attributed, in some cases, to the appearance of hypertension, cardiac arrhythmias, and malignant changes in the digestive tract, lungs, and kidneys (Counis, 1998).

Volcanic activity is one of the reasons for the occasional increase in the concentration of cadmium in the environment, primarily in the air. Permanent sources of cadmium contamination are related to its application in industry, as an anticorrosive reagent, stabilizer in PVC products and the production of tires, paint pigment, and in the production of Ni-Cd batteries. Although some cadmium - containing products can be recycled, much of the pollution with this metal is the result of inadequate disposal and uncontrolled incineration of cadmium-

containing waste (Jarup, 2003). The biggest source of inhaled cadmium intoxication is smoking. Total global cadmium emissions are estimated at about 7,000 tons per year (Stoeppler, 1991). The maximum permissible values of cadmium for workers are much lower, under German law for example they are 15 μg/dm^3. For comparison, in non-smokers, the average concentration of cadmium in the blood is 0.5 μg/dm^3 (Godt et al., 2006).

Unlike acute, chronic intoxication leads to the development of some diseases such as chronic obstructive pulmonary disease, kidney disease (nephrotoxicity) and bone (arthritis, osteoporosis), anemia, growth disorder, and others (ATSDR, 2012).

Normal blood cadmium levels in adults are less than 1 μg/dm^3. Although cadmium spreads through the blood throughout the body, the largest accumulation (50 to 60 % of the body load of cadmium) is in the kidneys and liver. The load of cadmium, especially in the kidneys, mostly increases linearly with age, up to 50-60 years of age, after which the level of cadmium in the kidneys remains constant or very little decreases (Webb, 1979). Highly toxic effect of cadmium is the result of its interactions with the necessary micro and macro bioelements, especially with iron, calcium, copper, and zinc (Brzoska and Moniuszko-Jakoniuk, 1997). Cadmium poisoning can be acute or chronic. Acute poisoning occurs by inhalation of vapors or particles of cadmium salts (oxides, chlorides, sulfides, sulfates, carbonates, and acetates). Symptoms of acute cadmium exposure include nausea, vomiting, loss of smell, taste and appetite, and abdominal pain (Wentz, 2000).

Chronic poisoning can occur after prolonged exposure to cadmium by inhalation or oral administration, and systematic exposure to cadmium leads to increased calcium excretion, which poses an increased risk of kidney stones and bone damage (Godt et al., 2006). Emissions of toxic trace elements can come from a variety of sources in urban areas, including vehicle emissions, industry and other activities (Harrison et al., 1981). Industry is considered to be one of the most important anthropogenic sources of emissions of trace elements into the air (Niragu and Pacyna, 1988) and is considered an activity with an extremely adverse impact on the environment. Huge amounts of dust with elevated levels of trace elements are released into the air during industrial activities (Csavina et al., 2012).

One of them is the industrial zone in Kikinda. Despite this, limited attention has been paid to industrial zones. Therefore, it is necessary that the risks to the environment and human health in industrial zones are assessed in a timely manner and, accordingly, appropriate protection measures are planned and taken.

Numerous health risk studies related to heavy metals in the air have been done in densely trafficked areas, mining areas, and industrial complexes (Ferreira-Baptista and De Miguel, 2005; Zheng et al., 2010a; Zheng et al., 2010b; Csavina et al., 2012).

One of the industrial giants whose influence is also considered from the health aspect of the population is RTB Bor in Serbia (Mrazovac Kurilić et al., 2019; Mrazovac Kurilić et al., 2020). Based on the results from Bor, the alarming situation in Kikinda cannot be expected, but the emphasis can certainly be placed on the cumulative effect and its reduction.

The aim of this paper is to assess the current state of air quality and impact on health, i.e. health risk assessment in the industrial zone of Kikinda for timely preventive steps to reduce harmful effects.

2. Materials and methods

2.1. Study area

Kikinda is a municipalty located in northeastern part of Serbia (and Vojvodina), on the border of Romania (Figure 1 a) and b).

a)

b)

Figure 1. a) Kikinda on the map of Serbia b) industrial zone of Kikinda - map

Data from the Kikinda industrial zone were used to analyze the risks to the environment and human health.

Kikinda is an industrially developed area and it occupies an important place in the economic development of the Republic of Serbia. Industry in the municipality of Kikinda is a priority area and is based largely on the available raw material base, production capacity, and quality human resources. Industrial production is very diverse, and the available production capacities are mainly of a processing character with a high degree of product finalization. There are several IPPC and SEVESO plants operating in the Kikinda industrial zone, which are very important and important from the aspect of environmental protection, because an integrated permit is obtained for the operation of such plants. The following plants belong to the IPPC and SEVESO groups:

Foundry "Kikinda" metal industry; foundry "Le Belier Kikinda" production of aluminum castings for the automotive industry; building materials industry - "Toza Marković"; "MSK" Kikinda, methanol - vinegar complex; petroleum and chemical industry - NIS "North Banat"; "Bell Chemicals" oil and lubricant refinery; "Blik Produkt"; food industry - "Banini", "Prima Produkt", „Kikindski mlin", "Mokrin imlek"; "G-Graphix", "Grindex", "Agroseme" and fodder factory. Plants belonging to IPPC and SEVESO plants have a major impact on the environment.

2.2. Sampling and analysis

Measurements were made during 2019, from January to December. The used monitoring station was a part of the local network of automatic monitoring of ambient air quality (part of sepa.gov.rs) in AP Vojvodina, which was managed by the Provincial Secretariat for Urbanism and Environmental Protection. Measurements were performed daily based on a 24 h sample.

Sampling of suspended particles of PM_{10} fraction in ambient air in automatic station in Kikinda was performed by a reference sampler of suspended particles of the manufacturer DIGITEL, model LVS DPA 14, Switzerland. Determination of mass concentration was performed by gravimetric method in accordance with the standard SRPS EN: 12341:2015. Determination of lead, cadmium, nickel, and arsenic was performed by an automatic absorption spectrophotometer in accordance with the standard SRPS EN: 19402:2008, (Inductively coupled plasma mass spectrometry ICP-MS).

2.3. Human health risk assessment model

The risk assessment model developed by the US EPA was used to assess the health risks posed by emissions of trace elements in the air in Kikinda. The research was performed for two groups of people: adults and children, and the following exposure categories were used: adults and children 1) inhalation through the mouth and nose; 2) swallowing through the mouth; and 3) skin contact through exposed skin.

According to the Human Health Assessment Manual (Part A) (US EPA, 1989) and the Dermal Risk Assessment Supplementary Guidelines (Part E) (US EPA, 2004), the hazard ratio (dimensionless, HQ) was used to assess non-cancerous risks of trace element emissions in air (equations 1-3), where RfD was the appropriate reference dose.

HQ < 1 means that there are no adverse health effects, while HQ > 1 indicates that adverse health effects are likely to occur due to the action of a particular element.

$$HQ_{ing} = \frac{(C_{95\%UCL} \cdot IngR \cdot EF \cdot ED \cdot CF)}{(BW \cdot AT \cdot RfD_{ing})} \quad (1)$$

$$HQ_{inh} = \frac{(C_{95\%UCL} \cdot InhR \cdot EF \cdot ED)}{(BW \cdot AT \cdot PEF \cdot RfD_{inh})} \quad (2)$$

$$HQ_{dermal} = \frac{(C_{95\%UCL} \cdot SA \cdot SL \cdot ABS \cdot EF \cdot ED \cdot CF)}{(BW \cdot AT \cdot RfD_{dermal})} \quad (3)$$

The hazard index (dimensionless, HI) is equal to the sum of the HQs and is used to assess the overall potential non-carcinogenic risk of different pollutants through the three types of exposure described above. HI < 1 indicates that there is no significant risk of non-carcinogenic effects. If HI > 1, then there is likely to be a non-carcinogenic effect on the health of residents of a particular category (US EPA, 1989).

According to the classification made by the International Agency for Research on Cancer (IARC), As, Cd, and Ni have been characterized as elements of increased carcinogenic risk (IARC, 2014). The lifespan of carcinogenic risks can be estimated by equation 4 where SF is the appropriate slope factor.

$$R = \left(\frac{(C_{95\%UCL} \cdot EF \cdot SF_{inhal})}{(AT \cdot PEF)} \cdot \frac{InhRchild \cdot Edchild}{(Bwchild)} + \frac{(InhRadult \cdot Edadult)}{Bwadult} \right) \quad (4)$$

Any carcinogenic risk in the range of 10^{-6}-10^{-4} (dimensionless) is considered acceptable (US EPA, 1989). All exposure factors for these models are shown in the Table 1. RfD and SF values of all examined elements (Ferreira-Baptista and De Miguel, 2005; Zheng et al., 2010a; Zheng et al., 2010b) are shown in Table 2.

Table 1
Exposure factors and their values for the human health risk assessment model (US EPA, 2004)

Factor	Meaning	Values for adults	Values for children	Unit of measure
BW	Average body mass	70	15	kg
IngR	Ingestion frequency	100	200	mg/day
InhR	Inhalation frequency	20	7.6	m^3/day
PEF	Particle emission factor	$1.36 \cdot 10^9$	$1.36 \cdot 10^9$	m^3/kg
SA	Surface parts of the skin that touch air particles	5700	2800	cm^{-2}
SL	Skin adhesion factor	0.07	0.2	mg/cm^2day
EF	Exposure frequency	180	180	day/year
ED	Duration of exposure	24	6	Year
ET	Exposure time	24	24	Hour/day
AT (non-carcinogenic risk)	Average time	ED · 365	ED · 365	day
AT (carcinogenic risk)	Average time	70 · 365	70 · 365	day
ABS	Dermal absorption factor	0.03 (As); 0.001 (other)	0.03 (As); 0.001 (other)	-
CF	Conversion factor	$1 \cdot 10^{-6}$	$1 \cdot 10^{-6}$	kg/mg

Table 2
Reference dose (RfD) and slope factor (SF) values for the health risk assessment model

	Ing RfD	Inhal RfD	Dermal RfD	Oral SF	Inhal SF	Dermal SF
Pb	3.50E-03	3.52E-03	5.25E-04	-	-	-
Cd	1.00E-03	1.00E-03	1.00E-05	-	6.30E+00	-
Ni	2.00E-02	2.06E-02	5.40E-03	-	8.40E-01	-
As	3.00E-04	3.01E-04	1.23E-04	1.5E+01	1.51E+01	3.66E+01

2.4. Statistical analysis

$C_{95\%UCL}$ (upper exposure concentrations, mg kg^{-1}) is considered a "reasonable maximum exposure" (US EPA, 1989; US EPA, 1992; Zheng et al., 2010a; Zheng et al., 2010b; Hu et al., 2011), which is the upper limit of the 95 % confidence interval for the mean. A 95 % confidence limit (UCL) was calculated using the adjusted central limit theorem (CLT) (Singh et al., 1997; US EPA, 2002). Although the approach was developed for normally distributed large data sets, the theorem does not say how many samples are sufficient for normality. $C_{95\%UCL}$ was calculated using the equation 5:

$$C_{95\%UCL} = \overline{X} + \left[z_\alpha + \frac{\beta}{6\sqrt{n}} (1 + 2 \cdot z_\alpha^2) \right] \cdot \frac{S.D.}{\sqrt{n}} \quad (5)$$

where: $\overline{X}$ = arithmetic mean; S.D. = standard deviation; β = skewness; α is the probability of type I error (false positive result) and its value is 0.05; for $Z\alpha$ = (1−α) quantile of standard normal distribution.

For a confidence level of 95 %, $Z\alpha$ = 1,645; n = number of samples.

All statistical analyzes were performed in the software package Statistica 8.

3. Results

Heavy metals concentrations were detected during 2019, as a result of monitoring under the jurisdiction of the state monitoring network. Measurements in the industrial zone of Kikinda showed the presence of heavy metals (Ni, As, Pb, and Cd). All concentration values below the detection limit were calculated as the detection limit.

In Table 4 are shown values of non-carcinogenic and carcinogenic risk of analyzed elements.

Table 3
Basic statistics of measured parameters - Kikinda (2019) (www.sepa.gov.rs)

mg/kg	Pb	Cd	Ni	As
MIN	0	0	3.15E-06	2.31E-07
MAX	4.69E-05	1.54E-06	1.43E-05	6.92E-06
MEAN	6.05E-06	2.42E-07	3.47E-06	8.17E-07
SD	5.73E-06	1.73E-07	1.46E-06	6.84E-07
SKEW (β)	4.109609	3.507945	5.713115	4.624892
$C_{95\%}$	**7.61E-06**	**2.854E-07**	**3.953E-06**	**10.163E-07**

Table 4
Calculated HQ, HI, and R values for each trace element measured in air in Kikinda (2019)

	HQ ingestion		HQ inhalation		HQ dermal		HI	
	A	**Ch**	**A**	**Ch**	**A**	**Ch**	**A**	**Ch**
Pb	1.532E-09	1.430E-08	2.240E-13	3.971E-13	4.073E-11	2.667E-10	1.57E-09	1.46E-08
Cd	2.011E-10	1.877E-09	2.957E-14	5.243E-14	2.810E-11	5.251E-10	2.29E-10	2.40E-09
Ni	1.392E-10	1.300E-09	1.988E-14	3.525E-14	2.057E-12	1.347E-11	1.41E-10	1.31E-09
As	2.387E-09	2.227E-08	3.498E-13	6.202E-13	2.322E-11	1.520E-10	2.41E-09	2.24E-08

*A - adults; Ch - children

Table 5
Calculated R values for each trace element measured in air in Kikinda (2019)

	Ring	Rinh	Rder	Ri
Cd	-	921.81E-16	-	921.81E-16
Ni	-	170.24E-15	-	170.24E-15
As	7,815.55E-16	7,867.65E-16	19,069.94E-16	3.48E-12

4. Discussion

Linear correlation coefficients between analyzed heavy metals showed low interdependence between metals, lower than 0.75, (except between As and Pb), which implied anthropogenic origin of these elements in atmosphere (Table 6).

Table 6
Correlation between measured heavy metals

	Pb	Cd	Ni
Cd	0.509	1	
Ni	0.135	0.071	1
As	0.859	0.277	0.128

Based on quantitative values, it can be concluded that no increased level of risk was identified by technical-technological systems in the area of the industrial zone Kikinda.

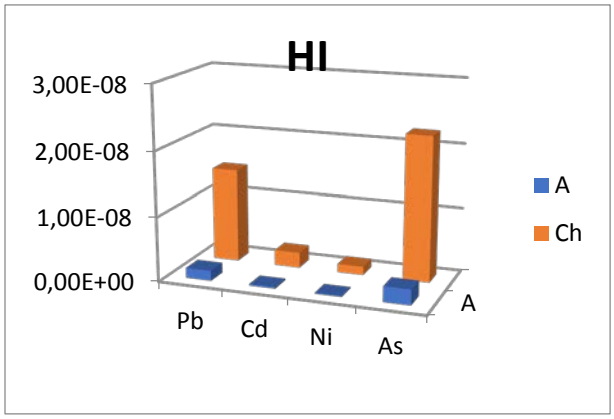

Figure 2. Graphical representation of HI values for each analyzed element, for adults (A) and children (Ch)

The Figure 2 shows a significantly higher value of HQ in children, which implied a significantly higher non-carcinogenic impact of harmful elements on the younger population. Also, the most significant influence of As and Pb was noticeable.

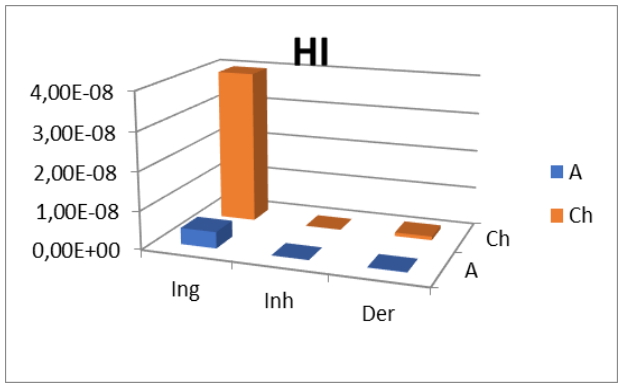

Figure 3. Graphical representation of HI values depending on the way of intake, for adults and children

Based on the graphic presentation, the dominance of oral intake of harmful elements by the intensity of action can be observed, also with a far more pronounced effect on the younger population.

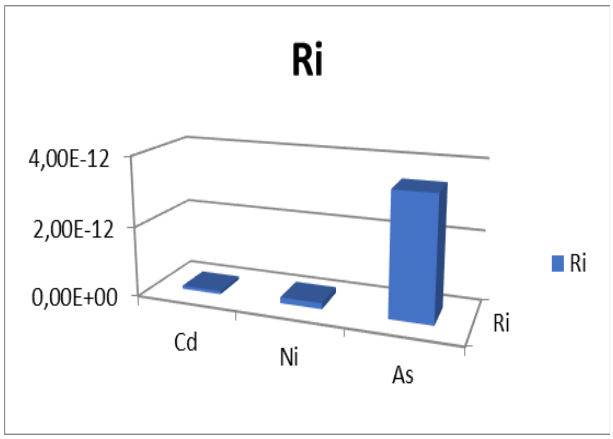

Figure 4. Graphical representation of R values for each analyzed element

In the case of carcinogenic risk, As can be pointed out as the biggest influence. The most significant was the dermal effect, in case of carcinogenic risk.

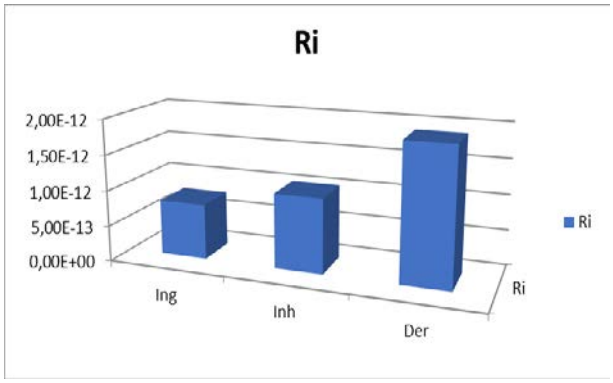

Figure 5. Graphical display of HQ values depending on the way of intake

Compared to other localities that were mainly developed industrial and mining centers (Luanda - Angola, mega city Nanjing - China, Bor - Serbia...) for which the human health risk was assessed, it can be said that the situation in the industrial zone of Kikinda is far safer and not in an area of increased health risk.

By comparing with other results from authors from different parts of the world, it can be concluded that similar findings and results have been reached.

In Wanshan district of Guizhou province in China, oral ingestion was the main pathway affecting the carcinogenic risk and hazard quotient (HQ) of heavy metals in air. The total carcinogenic risk of twenty-five sampling points was between 1.219×10^{-6} and 3.446×10^{-4}, and the total - HQ was between 0.37 and 43.56. (Wu et al., 2020)

In Tehran, based on the study of Zarandi et al., 2018, the rank order of exposure pathways based on health risk was ingestion > inhalation > dermal contact. Moreover, the significant health risks for Tehran residents due to heavy metals bound $PM_{2.5}$ (HQ > 1; carcinogenic risk > 1.00E−06) were noted based on the health risk assessment. Carcinogenic risk of PAHs bound $PM_{2.5}$ was 4.16E−07 that demonstrated that there was no considerable risk (< 1.00E−06).

Also, in Tehran, The average risk of carcinogenic at urban stations in the spring for As, Cd, and Cr was 2.25×10^{-9}, 2.09×10^{-12}, and 2.05×10^{-11} (Anoushiravan et al., 2018).

In Delhi, India, hazard quotient (HQ) values indicated that ingestion was the major pathway of road dust heavy metal exposure to human beings. Hazard index values showed that there was no probable non - carcinogenic risk of the heavy metals present in the road dust of the area. Children were found vulnerable to the risks of road dust metals. (Roy et al, 2019)

The purpose of this assessment is to prevent population from unrestrained development of the industry and the danger of a cumulative effect. In that sense, it is necessary to think about long-term monitoring of the health risk trend of heavy metals in order to be able to take adequate protection measures.

5. Conclusion

Monitoring of the air quality in the industrial zone in Kikinda showed the presence of heavy metals. During the analysis of the presence of heavy metals in the atmosphere in the industrial zone of Kikinda, no increased risk to the environment and human health from the presence of nickel, cadmium, arsenic, and lead compounds was noticed. As the most significant impact the impact of arsenic can be singled out, in the case of carcinogenic and non-carcinogenic risk, as well as in the case of adults and children. The most significant way of intake of heavy metals into the body for non-carcinogenic risk was ingestion, while for carcinogenic risk the effect through the skin was dominant. The greatest danger was the ability of heavy metals to accumulate in the human body.

How to prevent environment pollution and human health deterioration from heavy metals?

- Application of process technologies with low emissions, especially in new plants;
- Waste gas treatment (secondary measures of reduction) by means of filters, scrubbers, adsorbers, etc.;
- Modification or preparation of raw materials, fuels, or others materials for production (e.g. use of raw materials with low heavy metal content);
- Best management practices, such as good maintenance by space, preventive plant maintenance programs, or primary measures, such as closing units that produce dust; and
- Appropriate environmental management measures for the use and disposal of certain products that contain Cd, Pb, and / or Hg.

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Procena rizika po ljudsko zdravlje od uticaja industrije u industrijskoj zoni Kikinde

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I Z V O D

U industrijskim zonama kao što je opština Kikinda (predstavljena u ovom radu) neophodno je pratiti zagađenje životne sredine i preduzeti mere za zaštitu životne sredine od uticaja štetnih zagađivača i toksičnih supstanci (posebno od teških metala). Model za procenu rizika koji je razvila Agencija za zaštitu životne sredine Sjedinjenih Država je korišćen za procenu zdravstvenih rizika koje predstavljaju emisije teških metala u vazduh u industrijskoj zoni Kikinde. Može se zaključiti da nije utvrđen povećani nivo rizika od sadržaja teških metala u vazduhu koji potiče iz industrijskih sistema na području industrijske zone u Kikindi. Međutim, neophodno je primeniti preventivne mere kako bi se smanjila akumulacija teških metala u životnoj sredini koja nastaje zbog aktivnosti koje se na tom području odvijaju konstantno.