

Environmental pollution and human exposure to polycyclic aromatic hydrocarbons in the east Baltic region

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Environmental contamination by polycyclic aromatic hydrocarbons (PAH) and human exposure were surveyed in the east Baltic region. Polluted and heavily industrialized areas are upper Silesia in Poland, northern Bohemia in the Czech Republic, and the northeast part of Estonia. In Estonia the pollution is in a defined geographic area, where lung cancer incidence is higher than elsewhere. In Silesia, exposure of some 5 million people appears to be elevated; DNA (deoxyribonucleic acid) adduct levels in white blood cells are increased in groups of residents with apparently only environmental exposure. By extrapolation, some 150 annual lung cancer cases could be predicted due to PAH in Silesia. Air levels of benzo[a]pyrene were increased in northern Bohemia, and domestic heating and power plants using brown coal appeared to be major contributors to particulate air pollution in winter. Further studies are needed to assess health risks of PAH exposure in central and eastern Europe.

Key terms extrapolation, cancer risks, DNA adducts, environmental contamination, pollution, unit risks.

Environmental pollution by genotoxic agents is of major societal concern for 2 kinds of human health reasons. An increase in the mutation rate in germ cells may cause an increased incidence of genetic disease in future generations and mutations in somatic cells may lead to an increased cancer incidence in the current generation. The occupational and environmental exposure of people in the eastern and central European countries often exceed that seen in the western countries. This exposure was a consequence of long-term intensive combustion of indigenous fossil fuels in power plants and use as industrial raw materials. Examples of densely populated industrial areas include upper Silesia in southern Poland, where coal is the basis of the industrial activity, northern Bohemia in the Czech Republic, where brown coal is mined and used, and the northeast part of Estonia, where oil shale is extensively used in power stations and petrochemical industries.

There have been alarming mortality data accumulating from central Europe. In many eastern European countries the mortality of middle-aged men has increased for several decades, whereas the development in western Europe has been favorable (1). For example, in Poland the overall mortality, including cancer mortality, has increased dramatically among middle-aged men in the past

30 years (figure 1). In fact, the current expected life span of young Polish men is lower than those living in Latin America and India (1). The reasons for this unfavorable development are likely to be primarily dependent on many life-style and socioeconomic factors. Environmental pollution is probably also a contributing cause, but solid epidemiologic data on its magnitude are either lacking or very fragmentary. However, for the population living in industrial areas, pollution is the main culprit of higher mortality, cancer incidence, birth defects, and immune deficiency in children. A survey among adult Poles revealed that environment was given the highest score (62%) as the main factor influencing human health, while tobacco and genetics were given a much lower weighting of about 26% (1).

Among the many environmental carcinogens known, polycyclic aromatic hydrocarbons (PAH) are of large current and historical concern. PAH represent a major group of organic genotoxic pollutants generated in the combustion and processing of fossil fuels. Hundreds of congeners are chemically known and tested for carcinogenicity and mutagenicity. PAH have been historically measured by surrogate indices such as dust and suspended particulate matter, and these measurements allow a crude estimation of ambient air exposure decades ago. More

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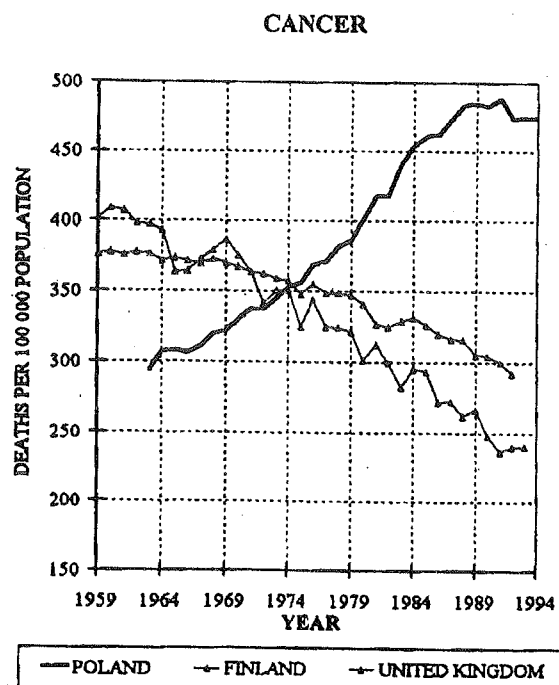


Figure 1. Mortality from cancer among men at 45–64 years of age in Poland, Finland, and the United Kingdom in 1959–1994 (1).

Table 1. Benzo[a]pyrene (BaP) in coastal waters of some recreation areas of the Baltic Sea and its seasonal variation (5).

Station	Number of samples	BaP (ng/l)				
		Spring	Summer	Autumn	Winter	Average
Vääna	44	1.6	1.1	0.04	23.5	6.56
Lahepere	132	9.2	3.5	0.04	8.1	5.21
Tallinn	82	2.7	8.3	0.1	2.3	3.35
Pirita	142	9.1	11	0.2	2.3	5.7
Narva	112	1.3	0.2	0.4	0.5	0.6
Pärnu	163	0.7	2.3	1.2	1.0	1.3
Matsalu	78	1.3	0.7	0.4	0.4	0.7

Table 2. Polycyclic aromatic hydrocarbons (PAH) in water, bottom sediments, algae and zooplankton in Lahepere Bay, Baltic Sea (modified from references 5 and 6).

PAH	Water (ng/kg)	Sediment (ng/l)	Algae (ng/kg)	Zooplankton (ng/kg)
Pyrene	35	16000	.	790
Benz[a]anthracene	0	13000	260	700
Chrysene	13	28000	140	1290
Benzo[a]pyrene	22	16500	160	69
Benzo[e]pyrene	15	7900	220	.
Benz[ghi]perylene	0	9300	.	.
Dibenz[a,h]anthracene	0	2500	nd	94
Benz[b]fluoranthene	1	16500	160	69
Benz[k]fluoranthene	1	4300	nd	.

recently, specific PAH, such as benzo[a]pyrene (BaP), have been measured. PAH-containing particles deposit on the ground, are taken up by plants, and, in that way, enter the food chain. In fact, food contributes the largest

source of human exposure to PAH, barring some occupational groups (2, 3). In the present paper we survey some indicators of PAH exposure in the eastern Baltic region, drawing examples from Estonia, Poland, and the Czech Republic. The levels of dust and PAH in the environmental samples and the exposure of humans are reviewed based on published records.

Polycyclic aromatic hydrocarbons in the ecosystem

Environmental monitoring of PAH has been carried out in Estonia using BaP as an indicator. Systematic monitoring of PAH was performed in water, bottom sediments, and water organisms of several bays of the Estonian coastal waters of the Baltic Sea. The main sources of PAH in water are point sources from industrial sewage and related air emissions, as well as mobile sources from automobile exhaust. An example of the point sources is the shale oil industry discharging waste water heavily contaminated by BaP. The content of BaP in waste waters of the oil shale industry range from 1.5–5 µg/l near oil-shale mines, up to 15–160 µg/l at the petrochemical plant in Kohtla-Järve (4). The concentration of BaP in waters of the most popular recreational areas of the Estonian coast was in the nanogram per liter range, and it showed some seasonal variation (table 1) (5). Although the BaP levels in water were low, the compound accumulated in bottom sediments and marine organisms. The sediments contained 10^4 – 10^6 times more BaP than the water samples. The determination of a number of PAH congeners showed that they accumulate in the sediments to a similar proportion as BaP (table 2) (5, 6).

In some surveys the PAH content in different fish species in the Baltic Sea has been determined. Analyzing Baltic herring, caught in the Finnish archipelago, Rainio et al (7) found only 2 (fluoranthene and pyrene) of the total 14 PAH compounds studied, while in pike-perch and burbot additional PAH congeners were detected. Recently Ojaveer & Tanner (8) studied samples of eelpout, *Zoarces viviparus* collected from the Gulf of Finland and the Gulf of Riga, and determined 11 different PAH compounds in fry and in fish tissues. Eleven PAH compounds were all present in edible parts of the fish, while only 6 of them were detected in the fry. In general, preferential accumulation of more water-soluble PAH components was observed. In table 3 the distribution of individual PAH congeners in edible parts of these 2 Baltic species, caught in the Finnish Gulf near Tallinn (flounder) (8) and the Gulf of Riga (eelpout) (8), are cited. Veldre & Bogovski (5, 9) studied BaP content in many fish species to estimate the amount of BaP ingested by an average fish-consuming Estonian. BaP was determined in various organs and tissues of diverse

species of fish, the average concentration being 0.3–3 µg/kg wet weight. It has been calculated that about 25 kg of fish is consumed per person in Estonia per year, of which 80% is caught in the Baltic Sea. Thus an inhabitant of Estonia may ingest up to 70 µg BaP yearly and up to 4.9 mg over a lifetime (70 years).

Lake Peipsi is one of the largest lakes in Europe. It plays an important role in the economy of Estonia as a health resort, fishery waterbody, and potential source of drinking water for northeast Estonia. Because of the importance, systematic data on the pollution of Lake Peipsi by BaP have been collected during the last 20 years. Recently, the data on pollution by BaP and congeners in Lake Peipsi and its tributaries in the 1980s have been summarized (10). The contamination varied typically in different seasons and years, due to both pollution sources and meteorological conditions. The BaP content in the lake varied from <0.1 to 237 ng/l (ie, in the aforementioned range of the contamination of the Baltic waters). The concentration of BaP in bottom sediments varied from 16 to 580 µg/kg in the lake and from 40 to 4000 µg/kg in its tributaries, levels similar to or higher than those in the Baltic region (table 2). Among the pollution sources precipitation from the atmosphere, sewage, and waste water discharge, and spillage from boats are important.

A comparative study on the accumulation of PAH in pine needles in different sampling sites in Sweden and Estonia has been carried out (11). The 2 sampling sites in Estonia were near the oil shale petrochemical plant in Kohtla-Järve and 50 km southeast, near a highway. The Swedish sites were in different parts of the northern Stockholm area. The results are cited in table 4. The overall PAH concentration in the Estonian samples was comparatively low, because of the low traffic intensity

Table 3. Distribution of individual polycyclic aromatic hydrocarbons (PAH) in edible parts of 2 species of Baltic fish caught in the Finnish Gulf (flounder) and the Gulf of Riga (eelpout) (Modified from references 8 and 9).

PAH	Flounder (µg/kg)	Eelpout (µg/kg)
Fluoranthene	146.5	190.2
Phenanthrene	92	69.7
Anthracene	37	31.2
Pyrene	205	51.9
Benzo[a]anthracene	65	22.9
Chrysene	53.9	-
Benzo[a]pyrene	26	5.8
Benzo[e]pyrene	82.5	-
Benzoperylene	29.5	20.8
Dibenzo[a,c]anthracene	126	-
Dibenzo[a,h]anthracene	286	31
Dibenzo[a,i]anthracene	64	-

Table 4. Concentration of polycyclic aromatic hydrocarbons (PAH) in scots pine needles collected from 4 sites in Sweden and 2 sites in Estonia (11).

Compound	Concentration (ng/l dry weight)					
	Märsta ^a	Arlanda ^b	Rosersberg ^c	Frescati ^d	Estonia 1 ^e	Estonia 2 ^f
2-Methylfluorene	0	0	0	0	0	0
Dibenzothiophene	0.9	12	0	2.5	0.1	12
Phenanthrene	350	1500	1	240	0	1000
Anthracene	20	70	0	7.5	0	37
2-Methylanthracene	250	330	18	53	21	95
2-Methylphenanthrene	18	0	0	0	32	0
1-Methylphenanthrene	160	150	11	26	27	53
Fluoranthene	1400	400	170	230	0	160
Pyrene	1700	280	100	220	41	150
Retene	230	50	130	13	9	26
2-Methylpyrene	180	20	12	29	2.9	9.8
1-Methylpyrene	140	16	9	24	4.1	7.7
Benzo[ghi]fluoranthene	650	60	50	130	7.6	15
Cyclopenta[cd]pyrene	46	5.2	5.5	14	1.1	2
Benzo[a]anthracene	180	19	23	61	4.7	8.4
Chrysene/Triphenylene	520	68	110	210	31	51
Benzo[b&k]fluoranthene	63	20	41	69	8.7	6.3
Benzo[e]pyrene	71	14	26	45	5.6	5.3
Benzo[a]pyrene	29	7.6	7.9	7.4	1.3	3.2
Perylene	0	0	0	0	0	0
Indeno[1,2,3-cd]fluoranthene	0	0	0	0	0	0
Indeno[1,2,3-cd]pyrene	0	0	3	6.9	0	0
Dibenzo[a,b]anthracene	0	0	0	0	0	0
Benzo[ghi]perylene	44	0	13	23	0	2.9
Coronene	26	0	0	17	2.4	0
Total	6100	3000	730	1400	200	1600

^a Märsta, adjacent (5 m) to a major road (approximately 10 000 cars/day). ^b Arlanda, approximately 200 m from the southern end of the north-south runaway at Arlanda International Airport. ^c Rosersberg, background area more than 2 km away from the nearest major road or industry. ^d Frescati, adjacent to road E 20 to Norrtälje. ^e Estonia 1, 50 km southeast from Kohtla-Järve. ^f Estonia 2, 3 km northeast of the oil shale processing plant.

as compared with the Swedish roadside sites. The PAH profile of the Estonian samples was balanced towards heavier PAH than in the Swedish samples, which may have been due to engine and fuel characteristics of the Estonian vehicles. Noteworthy is also the high proportion of phenanthrene (60%) in 1 of the Estonian samples, probably due to emissions from the oil shale processing.

Human exposure to polycyclic aromatic hydrocarbons

In the 1990s biomonitoring studies were started in the central and eastern European countries, including Poland, the Czech Republic, and Estonia. Biological monitoring gives an opportunity to assess the uptake of chemicals in

the body of exposed persons and to determine biologically effective doses of such chemicals.

Air concentrations and absorbed doses

The exposure of cockery workers to PAH at an Estonian oil shale processing plant was assessed using occupational hygiene and biomonitoring measurements (12). In the cockery, the pyrene concentration of the filter samples was 0.01 to 69.6 $\mu\text{g}/\text{m}^3$, and the BaP concentration was 0.02 to 39.6 $\mu\text{g}/\text{m}^3$, the mean values being 8.1 $\mu\text{g}/\text{m}^3$ and 5.7 $\mu\text{g}/\text{m}^3$, respectively. Eighteen percent of the sampled workers were exposed to BaP concentrations of over 10 $\mu\text{g}/\text{m}^3$ during the workshift. According to a skin-wipe sample analysis, skin contamination was also obvious. The range of the BaP concentration in the samples collected after the shift was between 0.1 to 9 ng/cm^2 with a mean of 1.2 ng/cm^2 . When urinary 1-hydroxypyrene as a marker of the internal dose of exposed workers was measured (range 0.2–69.5 nmol/mmol creatinine, mean 6.0 nmol/mmol creatinine), a correlation between airborne pyrene, as well as BaP and urinary 1-hydroxypyrene, was established (r -value 0.56 and 0.63, respectively).

A series of environmental studies has been carried out in Poland. It was initiated several years ago in response to alarming reports on the environmental pollution in Silesia, a heavily industrialized area. The air concentration of BaP, measured over 3 years, from the polluted area (Ruda Slaska), is shown in figure 2. The peaks in the winter were 60 ng/m^3 in 1988, and they had decreased to 20 ng/m^3 in 1990 (13). The difference in the air concentration between summer and winter is marked. In western European and North American cities the concentration of BaP is currently around 1 ng/m^3 .

PAH concentrations were measured in air samples in Poland also in 1992–1993 and correlated to urinary 1-hydroxypyrene levels. In the polluted area, Gliwice, the air concentrations of BP were 41 and 9 ng/m^3 in February and September, respectively (14). At the coke

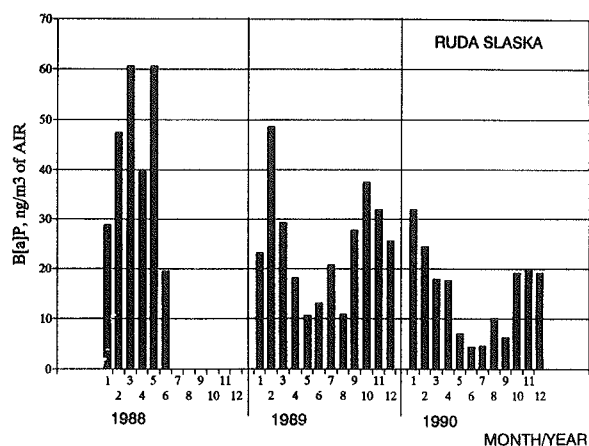


Figure 2. Air concentrations of benzo[a]pyrene (BaP) in Silesia between 1988 and 1990. Note the variation between summer and winter (13).

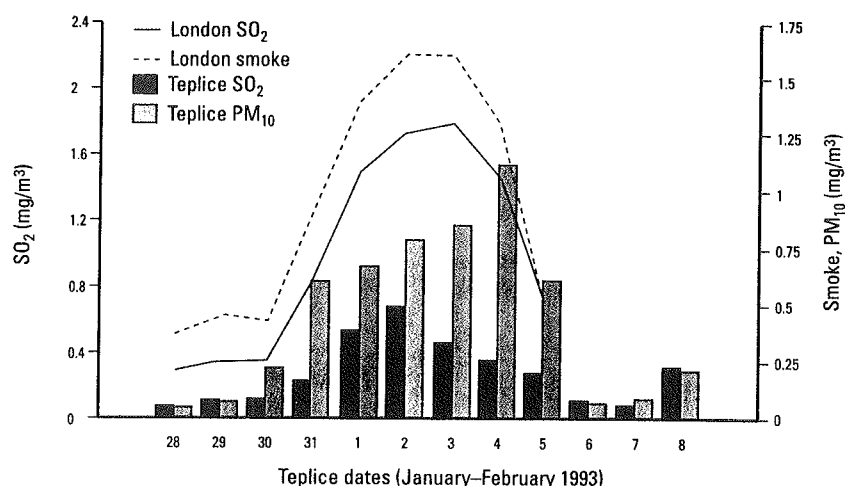


Figure 3. Airborne particulate material (PM_{10} = particles $<10 \mu\text{m}$) and sulfur dioxide (SO_2) in Teplice during 1993, compared with the concentrations in London during the pollution episode 1952 (15).

works, the main polluters in the industrialized area, the levels were 10–40 times higher. Differences were also observed in the 1-hydroxypyrene levels. In the Gliwice samples the winter levels exceeded those of the summer 2-fold. Coke workers had about 10 times higher levels but no difference between winter and summer.

A research program has been carried out on the environmental and health effects in the brown coal mining area of the Czech Republic. Air concentrations of pollutants, DNA (deoxyribonucleic acid) adducts, DNA strand breaks, and many health parameters have been measured in the polluted area (Teplice) and in the reference area (Prachatice). The main source of pollution was the burning of coal for heat and power generation, resulting in marked seasonal variation with maximal pollution in the winter (15). Because of the high sulfur content of the brown coal, emissions contain large amounts of sulfur dioxide. On the average, the air concentrations of PAH and metals were 2–3 times, and of sulfur dioxide some 5 times, higher in Teplice than in the reference area. The Teplice area was hit by severe periods of pollution during winter inversions. The measurements carried out in the course of 1 inversion episode, February 1993, were recorded (figure 3). The levels of sulfur dioxide and black smoke (blackness of the filter used for the collection of particles) were compared in Teplice in 1993 with those in a London pollution episode, December 1952, which caused deaths of hundreds of people. The levels of pollution during the Teplice inversion were remarkably high but still lower than during the London episode. There has been no detectable increase in mortality in Teplice resulting from the episode.

Adducts of deoxyribonucleic acid in humans

Most chemical carcinogens cause DNA adducts, which in turn lead to mutations. The detection of increased levels of DNA adducts in groups of humans has been considered an indicator of cancer risk in such a population (16, 17). Aromatic DNA adducts, including those caused by exposure to PAH, were determined in leukocytes of 3 Polish groups, environmentally exposed Silesians, occupationally exposed Silesians (positive referents), and unexposed residents from an unpolluted eastern Polish town. The first study in this series showed an elevated level of DNA adducts, by postlabeling and immunoassay, in the total white blood cells of the environmentally exposed Silesian residents (18). This finding was followed by reports on seasonal differences in adduct levels, which matched the air concentrations of PAH (19). The effects were mainly seen in DNA of the long-lived lymphocytes, while granulocytes showed no clear effect. Sampling in the summer and winter allowed a rough estimation of the half-life of aromatic adducts in lymphocytes (1–2 months) (13) (figure 4). Cytogenetic damage was also seen in the Silesian population (20).

The nature of the DNA adducts detected by postlabeling has been studied in more detail by comparing nuclease P1, butanol extraction, and immunoaffinity purification of the adducts. The adduct recovery was approximately equal by the P1 and butanol techniques, a result suggesting that the adducts are of the PAH type. For immunoaffinity chromatography an antibody raised against BaP diol epoxide-DNA was used. Only about 25% of the adducts were bound by the antibody, indicating that most of the adducts in DNA are not closely related to BaP. However, in a winter with high air pollution the relative binding by the immunoaffinity column was higher than in the summer (21). In a high-performance liquid chromatography (HPLC) analysis using a flow-through radioactivity detector, typical seasonal adduct peaks were noted, and they were particularly prominent in lymphocyte DNA collected in the winter. They eluted in the area of PAH-DNA adducts, a finding that gave additional support to the assumption that the adducts were PAH-like (22).

The concentration of BaP in the winter air samples from Teplice and Prachatice were 8 and 4.7 ng/m³, respectively (15). These can be compared to the winter air concentrations from Silesia (20–60 ng/m³) and the mean exposure of the population in Sweden (<1 ng/m³) (23), showing that the pollution levels are relatively high in the Czech localities. The source apportioning indicated domestic heating and power plants as major contributors to particulate pollution in Teplice in the winter; for Prachatice no source apportioning was presented. There was a significant correlation between white blood cell aromatic DNA adducts and exposure to PAH, but no data were given on the DNA adduct levels between the polluted and the reference areas (15).

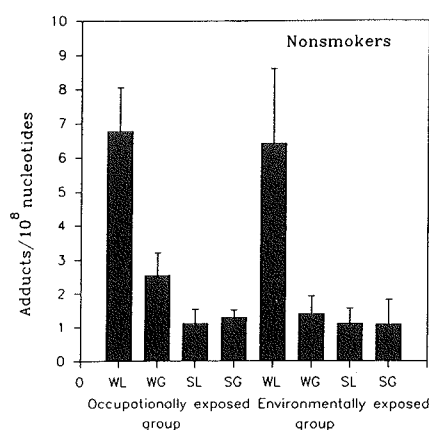


Figure 4. Aromatic adduct levels of the nonsmoking population in Silesia, occupationally or environmentally exposed to air pollution (13). Blood cells were separated. (WL = winter lymphocytes, WG = winter granulocytes, SL = summer lymphocytes, SG = summer granulocytes)

Cancer risks and risk assessment

Epidemiologic evidence on environmental pollution and cancer has been difficult to establish for a number of reasons, including some related to biology and logistics (2). As to biology, the risks inflicted by air pollution may be weak as compared with prominent risks caused by smoking. Furthermore, the risk caused by air pollution may interact with that caused by active and passive smoking and occupational exposure. As to logistic reasons, relatively few countries have an infrastructure commensurate with reliable epidemiologic observation. Another reason is that comparisons are usually made to some kind of average population rather than to a pristine unexposed population, and therefore the observable risk ratios are reduced. Thus, with a few exceptions, discussed in this volume by Jedrychowski, the effects of environmental pollution on cancer incidence remain to be established in central and eastern Europe.

Estonia has had a population-based cancer register covering all Estonia since 1968, with an estimated reporting rate of 95–98% (24). The risk of lung cancer, based on the Estonian Cancer Registry, is highest in the northeastern industrial towns of Kohtla-Järve and Narva, and in the capital Tallinn (figure 5). Kohtla-Järve is the center of oil shale mining and related industries; the risk of lung cancer is high there both for men and for women. However, the ethnic and cultural differences in the Estonian population reduce the value of ecological comparisons in establishing environmental cancer risks.

In addition to direct epidemiologic studies, cancer risk estimation is possible by extrapolating known exposure levels of carcinogens and their unit risks. For example, the inhalation of PAH from ambient air at a BaP level of 1 ng/m³ is estimated to convey a lifetime risk of lung cancer of 1×10^{-4} (2, 3). Unit risks have been used to calculate the number of annual lung cancers induced in

Sweden at an estimated BaP level of 0.7 ng/m³, resulting in 1 cancer/10⁶ inhabitants (2). On the assumption that, in Silesia, the lifetime exposure to PAH has been 20 ng/m³, the resulting annual number of lung cancers induced would be 30 cancers/10⁶ inhabitants or 150 lung cancer cases per the Silesian population of 5 million. If the total lung cancer incidence is 50/10⁵, which should be a fair approximation for Silesia, then the annual number of lung cancers would be 2500 in the whole population.

Concluding remarks

The extensive use of indigenous coal, brown coal, and oil shale in heating, energy production, and industry caused heavy environmental pollution in certain areas of eastern and central Europe. In these heavily polluted areas the physical signs of damage to landscape and buildings are readily apparent, whereas the effects on human health remain conjecture, probably forever. Upper Silesia, an old coal-producing industrial area, is one of these regions, and northern Bohemia, a brown coal mining area, is another. In Silesia the ambient soot has colored the buildings black. In Bohemia the high sulfur content of brown coal has caused massive releases of sulfur dioxide, which in turn has led to vast deforestation of the Bohemian woods. The shale production in northeastern Estonia affects a smaller geographic area, but, because of the large amounts of side rock produced, the landscape is reshaped with numerous waste hills around Kohtla-Järve.

There are a few analytical epidemiologic studies connecting air pollution to an increased risk of respiratory cancers in eastern and central Europe, particularly considering the reasonably high level of epidemiologic

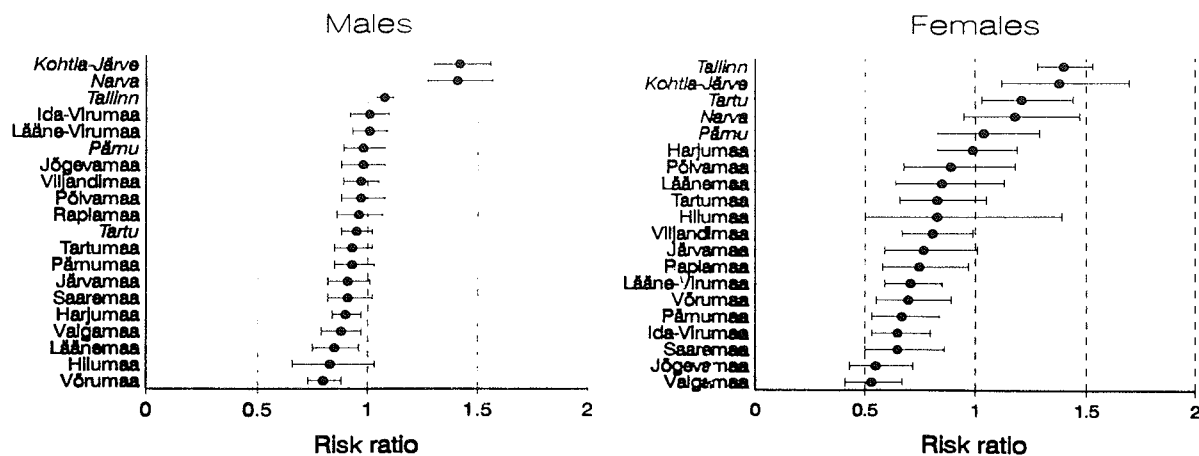


Figure 5. Ratio of age standardized lung cancer incidence for men and women in Estonian counties (ending with -maa) and towns (Kotla-Järve, Narva, Tallinn, Pärnu and Tartu), 1968–1992 (24).

expertise and infrastructure. The studies on the environmental levels of PAH and aromatic DNA adducts in humans support the notion of pollution-related risks. Yet, in the past, there has been a vigorous restructuring of industries, particularly of outdated facilities, in almost all parts of eastern and central Europe, and this restructuring has led to decreased production and, hence, pollution. Air pollution levels in Poland give evidence of improvements, which are likely to speed up with economic growth. This change implies that the nascent time of initiating epidemiologic studies of interest to the world at large is growing short. The lack of western support for comprehensive studies is regrettable, and the chances may be dwindling in years to come.

Acknowledgments

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References

1. Zatonski W, Boyle P. Commentary-health transformations in Poland after 1988. *J Epid Biostat* 1996;1:183—97.
2. Hemminki K, Pershagen G. Cancer risk of environmental air pollution: epidemiological evidence. *Environ Health Perspect* 1994;102 suppl 4:187—92.
3. Törnqvist M, Ehrenberg L. On cancer risk estimation of urban air pollution. *Environ Health Perspect* 1994;102 Suppl 4:173—81.
4. Veldre IA, Itra AR, Paalme LP. Levels of benzo(a)pyrene in oil shale industry wastes, some bodies of water in the Estonian S.S.R and in water organisms. *Environ Health Perspect* 1979;30:211—6.
5. Veldre IA, Bogovski PA. Polynuclear aromatic hydrocarbons in Estonian coastal waters. *Proc Estonian Acad Sci Ecol* 1993;3:149—57.
6. Veldre IA, Itra AR, Kuk H, Paalme LP. Algae as indicators of seawater pollution by polycyclic arenes. *Proc Acad Sci Estonian SSR Biol* 1984;33:67—71.
7. Rainio K, Linko RR, Ruotsila L. Polycyclic aromatic hydrocarbons in mussel and fish from the Finnish Archipelago Sea. *Bull Environ Contam Toxicol* 1986;37:337—43.
8. Ojaveer H, Tanner R. Levels of polycyclic aromatic hydrocarbons in eelpout, *Zoarces viviparus*, in Estonian marine waters. *Proc Estonian Acad Sci Ecol* 1996;6:136—43.
9. Bogovski S, Sergejyev B, Veldre I, Karlova S. Correlation of liver microsomal cytochrome p-450 activity with levels of PAH in some tissues of flounders from two differently polluted marine areas of Estonia: diseases and parasite of flounder in the Baltic Sea. *PMB Publ* 1994;15:105—7.
10. Trapido M, Veldre I. On polynuclear aromatic hydrocarbon contamination levels in the ecosystem of Lake Peipsi in the 1970s—1980s. *Hydrobiologia* 1996;338:185—90.
11. Kylin H. Airborne lipophilic pollutants in pine needles. Stockholm: Environmental Chemistry, Stockholm University, 1994.
12. Kuljukka T, Vaararinta R, Veidebaum T, et al. Exposure to PAH compounds among cockery workers in the shale oil industry. *Environ Health Perspect* 1996;104 suppl 3:539—41.
13. Grzybowska E, Hemminki K, Szeliga J, Chorazy M. Seasonal variation of aromatic adducts in human lymphocytes and granulocytes. *Carcinogenesis* 1993;14:2523—6.
14. Övrebö S, Fjeldstad PE, Gzybowska E, Kure EH, Chorazy M, Haugen A. Biological monitoring of polycyclic aromatic hydrocarbon exposure in a highly polluted area of Poland. *Environ Health Perspect* 1995;103:838—43.
15. Sram RJ, Benes I, Binkova B, Dejmek J, Horstman D, Kotesovec F, et al. Teplce Program - the impact of air pollution on human health. *Environ Health Perspect* 1996;104:699—714.
16. Hemminki K. DNA adducts and mutations in occupational and environmental biomonitoring. *Environ Health Perspect* 1997;105 suppl 4:823—7.
17. Poirier M. DNA adducts as exposure biomarkers and indicators of cancer risk. *Environ Health Perspect* 1997;105 suppl 4:907—12.
18. Hemminki K, Grzybowska E, Chorazy M, Twardowska-Sauch, Sroczynski JW, Putman KL, et al. DNA adducts in humans environmentally exposed to aromatic compounds in an industrialized area of Poland. *Carcinogenesis* 1990;7:1229—31.
19. Grzybowska E, Hemminki K, Chorazy M. Seasonal variations in levels of DNA adducts and X-spots in the human populations living in different parts of Poland. *Environ Health Perspect* 1993;99:77—81.
20. Perera FP, Hemminki K, Grzybowska E, Motykiewicz G, Michalska J, Santella RM, et al. Molecular damage from environmental pollution in Poland. *Nature* 1992;360:256—8.
21. Widlak P, Grzybowska E, Hemminki K, Santella R, Chorazy M. ³²P-Postlabelling of bulky human DNA adducts enriched by different methods including immunoaffinity chromatography. *Chem Biol Interact* 1996;99:99—107.
22. Möller L, Grzybowska E, Zeisig M, Cimander B, Hemminki K, Chorazy M. Seasonal variation of DNA adduct pattern in human lymphocytes analyzed by ³²P-HPLC. *Carcinogenesis* 1996;17:61—6.
23. Boström C-E, Almen J, Steen B, Westerholm. Human exposure to urban air pollution. *Environ Health Perspect* 1994;102 suppl 104:39—47.
24. Thomson H, Rahu M, Aareleid T, Gornoi K. Cancer in Estonia 1968—1992. Tallinn: Institute of Experimental and Clinical Medicine, 1996.