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Genotoxicity of airborne hydrophobic pollutants sampled by semipermeable membrane devices (SPMDs) in Vilnius city

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The genotoxic effects of complex mixtures extracted from semipermeable membrane devices (SPMDs) exposed for 8 weeks during spring, summer, autumn and winter seasons of 2002–2003 in four districts of the Vilnius city (Žirmūnai, Žvėrynas, Senamiestis and Lazdynai) were analyzed. The extracts were assayed for their ability to induce chromosome aberrations and sister chromatid exchanges (SCEs) in human blood lymphocytes *in vitro* and somatic mutations and recombination in *Drosophila melanogaster* wing cells *in vivo.* The test samples did not increase the incidence of chromosome aberrations. However, a significant increase of SCEs was determined in lymphocyte cultures treated with extracts sampled in Žirmūnai during all four seasons and in Žvėrynas during spring and winter seasons. Results of the somatic mutation and recombination test showed that only extracts sampled in Žirmūnai were genotoxic and significantly increased the incidence of somatic mutations. Our results confirm the findings of other authors that SPMDs provide a useful tool to obtain sufficient quantities of airborne hydrophobic pollutants for toxicity and genotoxicity tests, so they can be used for practical air monitoring purposes.

Key words: semipermeable membrane devices, airborne pollutants, chromosome aberrations, sister chromatid exchanges, somatic mutations

INTRODUCTION

Urban air is usually contaminated by different pollutants, their most important sources being combustion processes in industry, household, and vehicle exhaust fumes. The air quality largely depends on the traffic intensity, fuel types, heating systems, industries; it can be modified by seasonal meteorological conditions and photochemical reactions. A wide range of chemicals such as polycyclic aromatic hydrocarbons (PAHs), nitrobenzanthrones, polychlorinated biphenyls, organochlorine compounds, etc. are present in the urban air and may pose a significant hazard to human health. Health risks associated with urban air pollution include respiratory, cardiovascular diseases and an increase in lung cancer incidence [1, 2]. Airborne pollutants such as benzene, benzo[*a*]pyrene are recognized as potential mutagens and carcinogens.

Generally, the risk assessment of airborne pollutants is based on toxicity and genotoxicity data of single compounds and their concentrations estimated by analytical chemistry [3, 4]. However, more than 2800 chemicals have been identified in ambient air. The components of these complex mixtures may interact to produce synergistic, additive or antagonistic effects, and their consequences on biological systems are less known. There are several studies in which extracts of urban air were found to be harmful and induce genotoxicity in animals, plants, mammalian cells or bacteria [5–11]. Only in sparse studies semipermeable membrane devices (SPMDs) were applied for passive air sampling and concentration of airborne organic contaminants [12, 13], though SPMDs are widely used for monitoring of hydrophobic pollutants in aquatic environments [14–18]. The most popular passive sampling configuration is the lipid-containing SPMD developed by Huckins et al. [19]. Generally, SPMDs contain a thin film of neutral lipid, usually triolein, sealed within lay-flat tubing made of low-density polyethylene. This type of polymer (as well as silicone, polypropylene, polyvinyl chloride, etc.) is referred to as nonporous, although transient cavities generally <10Å in diameter form due to random thermal motions of the polymer chains [14, 19]. Because the cross-sectional diameters of the most environmental contaminant molecules are nearly as large as those cavities, only dissolved organic contaminants can diffuse into the membrane and be concentrated in the membrane and triolein [19, 20]. Triolein-SPMDs were designed to mimic the bioconcentration of organic contaminants in fatty tissues of

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organisms, and since their development have been successfully applied for *in situ* monitoring contaminants and concentrating trace organic pollutants for assessing their toxicity [12–18]. Contaminant classes shown to concentrate in triolein containing SPMDs include polycyclic aromatic hydrocarbons, polychlorinated biphenyls, organochlorine compounds. It is worth noting that D. Sabaliūnas et al. [17] were among the first to perform genotoxicity studies of concentrates from SPMDs.

The aim of the present study was to evaluate the genotoxic activities of airborne hydrophobic pollutants sampled by SPMDs in Vilnius during the spring, summer, autumn and winter seasons. Vilnius appears today to be one of the most polluted residential areas in Lithuania, mainly because of its surrounding geography and an extensive increase in traffic, thus four monitoring sites with different traffic intensity were selected to deploy SPMDs for passive sampling of organic contaminants in the air.

MATERIALS AND METHODS

Semipermeable membrane devices, study locations and sample processing

Semipermeable membrane devices were prepared in a way described earlier by D. Sabaliūnas et al. [15]. Briefly, 4.5 × 28 cm membrane devices were made from lay-flat polyethylene tubing (membrane thickness 75–80 μm, from Carl Roth GmbH & Co., Germany). The segments were extracted in cyclohexane (>99.5% purity, Riedel-de Haen, Germany) for 48 h, filled with 0.5 ml triolein (95% purity, from Sigma, USA) to form a thin film and thermosealed. The membrane area to lipid volume ratio was maintained at about 500 cm^2/ml.

Four districts of Vilnius, Žvėrynas, Žirmūnai, Senamiestis and Lazdynai were chosen to deploy SPMDs for passive sampling of organic air contaminants. The main sources of air pollution in Žirmūnai are heavy traffic and industry, in Senamiestis and Žvėrynas it is traffic, which here is less intensive as compared to that in Žirmūnai. The place of deployment of SPMDs in Lazdynai was considered as a control site. SPMDs were deployed during spring, summer, autumn and winter seasons of 2002–2003. SPMDs, six for each study site, were placed in metal cages designed to protect the SPMDs from rain, direct sunlight, wind and mechanical damage. The cages were fastened on the roofs of municipal air monitoring stations at about 2.5 m above the soil. Six control SPMDs were maintained in a heat-sealed jars and processed at the end of exposure as a SPMD sample.

After 8 weeks of exposure, the SPMDs were retrieved and immediately transported to the laboratory for sample processing. SPMDs were first rinsed under running tap water followed by a rinse with distilled water to remove surface fouled residues, and dried with clean paper. Afterwards all six SPMDs from each site were transferred into glass jars with screw-type lids. Hexane (>99% purity, from Riedel-de Haen, Germany) was added (120 ml hexane per 1 ml of SPMD triolein), and membranes were dialyzed for 48 h at 18 °C in the dark. Subsequently, SPMDs were removed from the jars, dialysates evaporated and the residues were dissolved in dimethyl sulphoxide (DMSO, 99.5% purity, Sigma) at a ratio of 2.5 ml DMSO per 1 ml of SPMD triolein.

Cytogenetic procedures

Whole peripheral blood from a healthy volunteer was grown in HEPES-buffered RPMI 1640 medium supplemented with 12% heat-inactivated newborn calf serum, 7.8 μg/ml phytohemagglutinin P, 50 μg/ml gentamycin, 10 μg/ml 5-bromo-2'-deoxyuridine. All reagents used for the cell culture were purchased from Sigma, USA. Cell cultures were incubated for 72 h at 37 °C. Treatment with SPMD extracts was carried out for the last 24 h of culture incubation. In addition to a blank control (untreated cultures), a solvent control was performed for any slight direct toxicity caused by the DMSO carrier. All tests were conducted using 0.5% (v/v) DMSO as the highest concentration in the culture. Two parallel cultures were used for each experimental point. The cultures were exposed to colchicine at a final concentration of 0.6 μg/ml for the last 3 h of incubation. The cells were harvested, hypotonically swollen in 0.075 M KCl (25 min) and fixed in methanol : acetic acid (3 : 1) fixative with three changes. Air-dried slides were differentially stained by fluorescence plus Giemsa technique [21]. Chromosome aberration analysis was performed in no less than 100 first-division metaphases per culture and sister chromatid exchanges – in 50 second-division metaphases. No less than 200 hundred cells were scored for the cell replicative kinetics determined by means of replication index RI (RI = $[M_1 + 2M_2 + 3M_3] / N$, where M_1, M_2, M_3 are the numbers of cells that had undergone one, two or three cycles of replication, and N is the total number of cells scored).

The wing spot test

The somatic mutation and recombination test (SMART) was essentially performed as described by Graf et al. [22]. For this assay, the following cross of *Drosophila melanogaster* flies was used: *ORR (1); ORR (2); flr^3/In (3LR) TM3, Bd^S* virgin females were crossed with *mwh* males (flies were kindly provided by Dr. H. Frei, Zurich, Switzerland). The first strain is characterized by constitutively high cytochrome P-450 activity. The markers *mwh* (multiple wing hairs) and *flr^3* (misshapen, flare-like hairs) are recessive wing-hair mutations located on the third chromosome at 0.3 and 38.8, respectively.

Eggs from the crosses were collected during 10-h periods. 72-h-old larvae were exposed to extracts

by adding 1 ml of the test solution (diluted with distilled water) to the surface of the medium. The larvae were fed on this medium until pupation, thus, the exposure duration was approximately 48 h. Progeny were raised on the Instant Drosophila Medium at 25 °C. Trans-heterozygous (*mwh / flr³*) flies were collected and stored in a 70% ethanol solution. The wings were removed and mounted in Faure's solution on slides. Both surfaces of wings were examined at 400× magnification for the presence of mutant spots. The type and number of spots as well as their sizes were recorded. The spots were grouped into two main categories: single spots showing either the multiple wing hairs (*mwh*) or the flare (*flr³*) phenotype, and twin spots showing adjacent *mwh* and *flr³*clones. No less than 40 wings were analysed per each experimental point.

Statistical analysis

Statistical analyses were performed using InStat V2.02 (GraphPad Software, CA, USA) statistical package. Statistical tests were chosen according to the nature of the data analysed. χ^2-test with Yate's correction was used to estimate the induction of chromosome aberrations and somatic mutations. A one-way analysis of variance (ANOVA) and the Student's two-sided t test was used for the evaluation of SCE occurrence and z test for RI analysis [23]. $P < 0.05$ was considered as the level of significance.

RESULTS AND DISCUSSION

Neither of the tested SPMD extracts induced a statistically significant increase of chromosome aberrations in human peripheral blood lymphocytes as compared to SPMD controls (Table 1). However, a statistically significant increase of SCEs was observed after treatment with the highest doses of extracts sampled in Žirmūnai during all four seasons and in Žvėrynas during spring and winter seasons (Table 2). Inhibition of cell replicative kinetics was evident only after treatment with the summer extracts from Žirmūnai and autumn extracts from Žvėrynas. SPMD extracts sampled in winter were slightly more genotoxic than those sampled during other seasons, however, ANOVA did not reveal any significant variations between the samples ($P = 0.2633$).

The results of somatic mutation and recombination analysis in *Drosophila melanogaster* wing cells are presented in Table 3. Only extracts sampled in Žirmūnai induced a statistically significant increase of somatic mutations in *D. melanogaster.* Also in this case, SPMD extracts sampled in winter were slightly more genotoxic than those sampled in spring and autumn. Summer samples did not induce a statistically significant increase of somatic mutations.

Our results are in good agreement with the results obtained earlier by other researchers. Several papers deal with genotoxicity testing of air pollution in different European cities – Prague, Teplice (Czech Republic), Sofia (Bulgaria), Caserta, Parma (Italy). Studies were made using different endpoints – sister chromatid exchange and comet assays in human leukocytes, somatic mutations in *Drosophila melanogaster,* gene mutations in *Salmonella typhimurium,* gene conversion and reversions in *Saccharomyces cerevisiae* [5, 7, 8, 11, 12]. In all of these studies the genotoxicity of urban air was demonstrated, and pol-

Table 1. **Induction of chromosome aberrations in human lymphocytes by SPMD extracts sampled during spring, summer, autumn and winter seasons of 2002–2003 in Vilnius**

Sampling point	Dose, µl	Frequency of aberrant cells, % ± S.E.M.			
		Spring	Summer	Autumn	Winter
Blank	0	2.5 ± 1.1	3.0 ± 1.7	1.5 ± 0.9	3.0 ± 1.7
DMSO	50	3.5 ± 1.3	1.0 ± 0.9	2.5 ± 1.1	5.0 ± 2.2
SPMD control	5	3.5 ± 1.3	2.0 ± 1.4	3.5 ± 1.3	5.0 ± 2.2
	25	6.0 ± 1.7	4.0 ± 1.9	4.0 ± 1.4	4.0 ± 1.9
	50	2.5 ± 1.1	4.0 ± 1.9	2.0 ± 1.0	4.0 ± 1.9
Lazdynai	5	3.0 ± 1.2	3.0 ± 1.7	1.5 ± 0.9	2.0 ± 1.4
	25	6.0 ± 1.7	3.0 ± 1.7	2.5 ± 1.1	3.0 ± 1.7
	50	3.5 ± 1.3	4.0 ± 1.9	3.5 ± 1.3	5.0 ± 2.2
Žvėrynas	5	5.0 ± 1.5	2.0 ± 1.4	3.0 ± 1.2	5.0 ± 2.2
	25	4.0 ± 1.4	4.0 ± 1.9	4.5 ± 1.5	5.0 ± 2.2
	50	4.5 ± 1.5	6.0 ± 2.4	3.5 ± 1.3	4.0 ± 1.9
Senamiestis	5	2.5 ± 1.1	1.0 ± 0.9	3.0 ± 1.2	4.0 ± 1.9
	25	3.0 ± 1.2	5.0 ± 2.2	3.0 ± 1.2	5.0 ± 2.2
	50	1.5 ± 0.9	3.0 ± 1.7	1.5 ± 0.9	4.0 ± 1.9
Žirmūnai	5	2.5 ± 1.1	4.0 ± 1.9	2.5 ± 1.1	3.0 ± 1.7
	25	5.0 ± 1.5	3.0 ± 1.7	5.5 ± 1.6	4.0 ± 1.9
	50	5.0 ± 1.5	6.0 ± 2.4	3.0 ± 1.2	6.0 ± 2.4

Table 2. **Effects of SPMD extracts on the frequency of sister chromatid exchanges (SCEs) and replicative index (RI) values in human lymphocyte cultures**

Sampling point	Dose, µl	Spring		Summer		Autumn		Winter	
		SCE/cell ±	RI ±	SCE/cell ±	RI ±	SCE/cell ±	RI ±	SCE/cell ±	RI ±
		S.E.M.	S.E.M.	S.E.M	S.E.M.	S.E.M.	S.E.M.	S.E.M.	S.E.M.
Blank	0	10.30 ± 0.58	2.53 ± 0.05	9.55 ± 0.49	2.53 ± 0.05	9.98 ± 0.32	2.55 ± 0.06	9.15 ± 0.25	2.54 ± 0.06
DMSO	50	9.67 ± 0.42	2.59 ± 0.05	9.68 ± 0.38	2.49 ± 0.03	9.57 ± 0.49	2.54 ± 0.05	9.42 ± 0.54	2.54 ± 0.06
SPMD	5	9.74 ± 0.41	2.61 ± 0.05	9.66 ± 0.65	2.56 ± 0.04	8.91 ± 0.46	2.50 ± 0.05	9.63 ± 0.21	2.45 ± 0.04
control	25	9.51 ± 0.56	2.56 ± 0.05	9.67 ± 0.75	2.48 ± 0.06	9.46 ± 0.59	2.45 ± 0.04	9.88 ± 0.56	2.49 ± 0.07
	50	9.62 ± 0.51	2.52 ± 0.05	10.02 ± 0.61	2.56 ± 0.05	9.89 ± 0.51	2.58 ± 0.05	10.02 ± 0.58	2.54 ± 0.04
Lazdynai	5	9.39 ± 0.50	2.65 ± 0.05	9.69 ± 0.57	2.54 ± 0.03	9.66 ± 0.25	2.55 ± 0.04	9.89 ± 0.24	2.55 ± 0.06
	25	10.76 ± 0.44	2.56 ± 0.05	9.99 ± 0.29	2.42 ± 0.06	9.99 ± 0.36	2.52 ± 0.06	9.93 ± 0.55	2.55 ± 0.05
	50	10.51 ± 0.60	2.50 ± 0.06	10.03 ± 0.56	2.48 ± 0.03	10.52 ± 0.35	2.55 ± 0.04	10.32 ± 0.56	2.44 ± 0.06
Žvėrynas	5	10.6 ± 0.48	2.65 ± 0.04	9.56 ± 0.42	2.49 ± 0.07	9.54 ± 0.79	2.56 ± 0.05	9.87 ± 0.54	2.46 ± 0.07
	25	10.23 ± 0.65	2.65 ± 0.04	9.83 ± 0.65	2.54 ± 0.05	9.76± 0.39	2.50 ± 0.05	10.33 ± 0.24	2.54 ± 0.04
	50	11.35 ± 0.56*	2.63 ± 0.05	10.22 ± 0.35	2.56 ± 0.06	11.25 ± 0.52	2.45 ± 0.06*	12.02 ± 0.59*	2.45 ± 0.05
Senamiestis	5	9.35 ± 0.41	2.58 ± 0.04	9.76 ± 0.61	2.56 ± 0.07	9.25 ± 0.35	2.59 ± 0.05	9.78 ± 0.45	2.54 ± 0.04
	25	10.24 ± 0.56	2.48 ± 0.05	9.78 ± 0.54	2.48 ± 0.06	10.16 ± 0.36	2.59 ± 0.04	9.87 ± 0.57	2.44 ± 0.06
	50	10.50 ± 0.48	2.60 ± 0.03	9.99 ± 0.62	2.54 ± 0.04	10.22 ± 0.56	2.57 ± 0.04	10.22 ± 0.97	2.51 ± 0.07
Žirmūnai	5	9.57 ± 0.42	2.51 ± 0.04	9.64 ± 0.35	2.51 ± 0.05	9.89 ± 0.39	2.55 ± 0.05	10.12 ± 0.45	2.48 ± 0.03
	25	9.84 ± 0.37	2.48 ± 0.05	9.68 ± 0.43	2.56 ± 0.03	9.96 ± 0.47	2.52 ± 0.05	10.19 ± 0.56	2.54 ± 0.07
	50	12.13 ± 0.51*	2.39 ± 0.06	11.54 ± 0.45*	2.49 ± 0.06*	11.99 ± 0.41*	2.60 ± 0.04	12.98 ± 0.68*	2.56 ± 0.05

* $P < 0.05$ as compared to respective SPMD controls.

lutant extracts sampled in winter were generally more genotoxic. In some cases the genotoxicity of winter air samples was six to 10-fold higher as compared with the summer air samples [7, 11]. Though vehicle exhaust fumes are usually considered to be the main source of pollutants, residential heating is often proposed as an additional emission source of pollutants and a possible cause of increased genotoxicity in winter. The amount of airborne particle matters in winter is usually higher. PAHs and their derivatives are among the main pollution markers from sources of combustion, and they contribute most to air genotoxicity [7, 10]. In our study, SPMD extracts sampled in two districts of Vilnius (Žirmūnai and Žvėrynas) were genotoxic, and winter samples were slightly more genotoxic than samples obtained during other seasons. Besides, winter and spring SPMD extracts sampled in Žirmūnai induced slightly more SCEs as compared to the respective season samples from Žvėrynas (difference is not statistically significant, $P > 0.05$). The site of SPMDs deployment in Žirmūnai is characterized by an extremely heavy traffic. The second major source of pollution in this district is industrial processes. In Žvėrynas the traffic density is lower, but coal and wood are frequently used for residential heating, and this may pose an additional source of pollution and contribute to the pool of genotoxic compounds. In summer and autumn a higher ambient temperature and more frequent rains may tend to clean the atmosphere and reduce the amount of contaminants.

The results of this investigation demonstrate the genotoxicity of the airborne contaminants sampled by SPMDs in Vilnius in high traffic areas. Similar results were obtained by M. Isidori [12] who used SPMDs as air samplers in Caserta, South Italy. The SPMDs were shown to concentrate trace contaminants effectively, and significant mutagenic effects of SPMD extracts were observed at all monitoring si-

Table 3. **Effects of SPMD extracts in the *Drosophila melanogaster* wing spot test**

Sampling	Dose, µl	Spring		Summer		Autumn		Winter	
		Wings with spots, % ± S.E.M.	Spots per wing	Wings with spots, % ± S.E.M.	Spots per wing	Wings with spots, % ± S.E.M.	Spots per wing	Wings with spots, % ± S.E.M.	Spots per wing
Blank	0	18.7 ± 4.4	0.20	15.0 ± 3.9	0.15	15.0 ± 3.9	0.15	16.2 ± 4.1	0.18
DMSO	30	16.2 ± 4.1	0.17	20.0 ± 4.5	0.20	16.2 ± 4.1	0.18	15.0 ± 3.9	0.15
	40	15.0 ± 3.9	0.15	17.5 ± 4.2	0.18	20.0 ± 4.5	0.23	20.0 ± 4.5	0.20
	60	17.5 ± 4.2	0.19	18.7 ± 4.4	0.20	15.0 ± 3.9	0.15	17.5 ± 4.2	0.20
SPMD	30	16.2 ± 4.1	0.17	12.5 ± 3.7	0.15	18.7 ± 4.4	0.20	18.7 ± 4.4	0.20
control	40	15.0 ± 3.9	0.15	17.5 ± 4.2	0.18	17.5 ± 4.2	0.18	21.2 ± 4.6	0.23
	60	17.5 ± 4.2	0.18	20.0 ± 4.5	0.20	17.5 ± 4.2	0.17	17.5 ± 4.2	0.18
Lazdynai	30	21.2 ± 4.6	0.23	22.5 ± 4.7	0.25	18.7 ± 4.4	0.20	17.5 ± 4.2	0.18
	40	20.0 ± 4.5	0.20	12.5 ± 3.7	0.13	22.5 ± 4.7	0.23	20.0 ± 4.5	0.20
	60	22.5 ± 4.7	0.24	21.2 ± 4.6	0.25	23.7 ± 4.7	0.28	20.0 ± 4.5	0.20
Žvėrynas	30	21.2 ± 4.6	0.23	13.7 ± 3.8	0.15	15.0 ± 3.9	0.15	16.2 ± 4.1	0.18
	40	22.5 ± 4.7	0.23	23.7 ± 4.7	0.25	20.0 ± 4.5	0.20	15.0 ± 3.9	0.18
	60	26.2 ± 4.9	0.30	20.0 ± 4.5	0.20	22.5 ± 4.7	0.30	21.2 ± 4.6	0.25
Senamiestis	30	21.2 ± 4.6	0.22	15.0 ± 3.9	0.15	22.5 ± 4.7	0.23	22.5 ± 4.7	0.23
	40	17.5 ± 4.2	0.18	17.5 ± 4.2	0.18	15.0 ± 3.9	0.15	18.7 ± 4.4	0.20
	60	21.2 ± 4.6	0.25	25.0 ± 4.8	0.25	23.7 ± 4.7	0.25	20.0 ± 4.5	0.20
Žirmūnai	30	26.2 ± 4.9	0.28	16.2 ± 4.1	0.18	17.5 ± 4.2	0.18	25.0 ± 4.8	0.25
	40	22.5 ± 4.7	0.23	12.5 ± 3.7	0.13	22.5 ± 4.7	0.23	23.7 ± 4.7	0.25
	60	31.2 ± 5.2*	0.35	20.0 ± 4.5	0.25	32.5 ± 5.2*	0.35	33.7 ± 5.3*	0.40

*P < 0.05 as compared to respective SPMD controls.

tes. Earlier, D. Sabaliūnas et al. [17] demonstrated the mutagenicity of triolein-SPMDs extracts deployed in an aquatic environment (polluted water sources of Lithuania). All these results confirm that the use of triolein-SPMDs for the preconcentration of certain trace contaminants in combination with the subsequent assay of the extracts by standard genotoxicity tests provides a useful approach for practical air monitoring purposes.

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VILNIAUS MIESTE EKSPONUOTUOSE MEMBRANINIUOSE KAUPIKLIUOSE SUKAUPTŲ HIDROFOBINIŲ ORO TERŠALŲ GENOTOKSIŠKUMO TYRIMAI

S a n t r a u k a

Tirtas membraniniuose kaupikliuose sukauptų hidrofobinių oro teršalų genotoksiškumas chromosomų aberacijų ir seserinių chromatidžių mainų (SCM) žmogaus limfocituose *in vitro*, taip pat somatinių mutacijų bei rekombinacijų (SMART) *Drosophila melanogaster* sparnų ląstelėse *in vivo* analizės metodais. Membraniniai kaupikliai buvo eksponuoti keturiuose Vilniaus miesto rajonuose (Žirmūnuose, Žvėryne, Senamiestyje ir Lazdynuose) 2002–2003 metų pavasarį, vasarą, rudenį ir žiemą (ekspozicijos laikas – 8 savaitės). Genotoksiniu aktyvumu pasižymėjo dviejuose Vilniaus miesto rajonuose – Žirmūnuose ir Žvėryne sukaupti oro teršalai. Žvėryne sukaupti teršalai indukavo patikimą SCM kiekio padidėjimą žmogaus limfocituose, Žirmūnuose – SCM limfocituose ir somatines mutacijas drozofilos sparnų ląstelėse. Mūsų gauti rezultatai patvirtina kitų autorių duomenis, kad membraniniai kaupikliai sukoncentruoja hidrofobinių teršalų kiekius, kurių pakanka jų genotoksiškumui įvertinti, ir gali būti panaudoti praktiniams aplinkos monitoringo tikslams.