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Oral epithelial cells of schoolchildren exposed to air pollution may develop cytotoxic and genotoxic damage

Daños citotóxicos y genotóxicos en células epiteliales orales de escolares expuestos a la contaminación atmosférica

Marco Antonio García Zarate
margarci@cicese.mx
https://orcid.org/0000-0003-3293-3000
Center for Scientific Research and Higher Education of Ensenada, B. C. Applied Physics Department
Ensenada Baja California – México

María Evarista Arellano García
evarista.arellano@uabc.edu.mx
https://orcid.org/0000-0002-0997-6902
Autonomous University of Baja California, Science Faculty
Ensenada Baja California – México

Zayre Ivonne González Acevedo
zgonzale@cicese.edu.mx
https://orcid.org/000-0003-3293-0563
Center for Scientific Research and Higher Education of Ensenada, B. C. Earth Science Division
Ensenada Baja California – México

Olivia Torres Bugarín
olivia.torres@edu.uag.mx
https://orcid.org/0000-0003-4541
Universidad Autónoma de Guadalajara. Unidad Académica de Ciencias de la Salud, Facultad de Medicina
Guadalajara, Jalisco – México

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Abstract

As petrol stations emit pollutants into the atmosphere, their proximity to schools can affect air quality both inside and outside classrooms. School-age children, who spend five to seven hours a day in classrooms and playgrounds during the school year, may be at risk of inhaling potentially harmful gases and particles, which could have a negative impact on their health and academic performance. A biomonitoring study was carried out on 143 children from three schools. Two of the schools were considered to be exposed to volatile organic compounds (VOCs) and were located 50 and 300 metres away from petrol stations. The other school, which was not considered to be exposed, was located 450 metres away. With the exception of MN, the biomarkers of genotoxic damage were generally higher ($p>0.05$) in the school located 50 metres from a petrol station than in the control group, although the results were less clear in the school located 300 metres away. For all biomarkers (KR, CC, PN and KL), the school 50 metres from the source of emissions had higher levels of cytotoxicity ($p <0.05$). This is the first study to link exposure to volatile hydrocarbons in petrol to genotoxic damage in children, and it strongly suggests that urban planning laws should be changed to halt the decline in children's health.

Keywords: volatile organic compounds, micronuclei, nuclear abnormalities, gas stations, air pollution

Resumen

Dado que las gasolineras emiten contaminantes a la atmósfera, su proximidad a las escuelas puede afectar a la calidad del aire tanto dentro como fuera de las aulas. Los niños en edad escolar, que pasan entre cinco y siete horas al día en aulas y patios de recreo durante el curso escolar, pueden correr el riesgo de inhalar gases y partículas potencialmente nocivos, lo que podría repercutir negativamente en su salud y su rendimiento académico. Se realizó un estudio de biomonitorización en 143 niños de tres colegios. Dos de los colegios se consideraron expuestos a compuestos orgánicos volátiles (COV) y estaban situados a 50 y 300 metros de gasolineras. El otro colegio, que no se consideraba expuesto, estaba situado a 450 metros. Con la excepción de MN, los biomarcadores de daño genotóxico fueron en general más elevados (p>0,05) en el colegio situado a 50 metros de una gasolinera que, en el grupo de control, aunque los resultados fueron menos claros en el colegio situado a 300 metros. Para todos los biomarcadores (KR, CC, PN y KL), el colegio situado a 50 metros de la fuente de emisiones presentaba mayores niveles de citotoxicidad (p <0,05). Este es el primer estudio que relaciona la exposición a hidrocarburos volátiles de la gasolina con daños genotóxicos en niños, y sugiere fuertemente la necesidad de modificar las leyes de urbanismo para frenar el deterioro de la salud infantil.

Palabras clave: compuestos orgánicos volátiles, micronúcleos, anomalías nucleares, estaciones de servicio, contaminación atmosférica

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INTRODUCTION

School grounds air quality has received attention from professionals and researchers in the last few years (USEPA, 2011). After their homes, school is the most important place for children's development since they spend more than 800 hours a year in them. In addition, children represent a potentially vulnerable population and specifically susceptible to the exposure of contaminants which may affect their health and performance as students (Neidell, 2004; Stafford, 2015; Annesi-Maesano, 2013; Salthammer, 2016; Daisey, 2003).

The time of exposure to manifest symptoms or illness vary according to various factors such as society, age and economy (Peled, 2011), as well as the type of contaminant such as carbon dioxide (CO2), volatile organic compounds (VOC) and the particles (PM2.5 and PM10). These have been associated with the development of respiratory problems and other adverse side effects in children (Madureira, 2015; Propper, 2015; Zhao, 2016).

The Health Secretariat of Mexico has been working on reducing preventable illness through vaccination of children less than 8 years of age which protects them against 13 different illnesses. Even so, nowadays researchers have shown that the incidence of illness in children with asthma, cancer, leukemia, brain cancer, ADHD, diabetes and obesity (Safer Chemicals, Healthy Families de la Coalition, 2012), have been rising and this is directly linked to the exposure to chemical substances in the environment (Pelallo-Martinez, 2014; Laborde, 2015; Di Renzo, 2015; Calderon-Garcidueñas, 2015; Liu, 2014; Currie, 2013). Therefore, eliminating this exposure to environmental chemical substances in children is still a major objective for public health.

Environmentalists and advocates for vulnerable groups like children, older people and immunocompromised people indicate that the development of public health policies do not take into account children being exposed to chemical substances and federal authorities cannot see the deathly effects the exposure to volatile organic compounds (VOC) can bring. Within the most prominent compounds are benzene, ethylbenzene, toluene and xylene (BTEX) which individually create genotoxic, cancerous, haematotoxic, nephrotoxic, neurotoxic effects or act as endocrine disruptors (ATSDR, 2004) among the children in the urban area.

Children present biological characteristics that increase their vulnerability to the acute and chronic effects of environmental toxins (Wild, 2003; Garry, 2004; Neri, 2006a) that may occur through various means: inhalation, ingestion or dermic absorption, and it include the prenatal stage (Slate, 2011; Peters, 2014; Bailey, 2011; Garlantezec, 2009; Esplugues, 2007; Miligi, 2013). For example, childhood leukemia is the most common type of cancer among children (Howlader et al., 2012); the most common type of sarcoma is acute lymphoblastic leukemia (ALL) which represents 78% of the cases in children; followed by acute myeloid leukemia (AML) which constitutes 16% of leukemia cases in children (Ries et al., 1999).

Various authors have reported a positive association to exposure of BTEX in the mother during pregnancy due to the vulnerability of the fetus to some toxic agents in the environment where chromosomal translocation in the blood has been identified in new born children (Wiemels et al., 1999; Wiemels et al., 2002; Shrestha et al., 2014; Breslow et al., 1993; Selevan et al., 2000). Benzene, a dangerous component found in gasoline, is a genotoxic group 1 human carcinogen (Loomis, 2017; Tunsaringkarn, 2011). Prolonged exposure to low levels of benzene in work environment and the environment (Roma-Torres, 2006), may lead to ruptured in the DNA, micronuclei and chromosomal abnormality (Fracasso, 2010; Angelini, 2011). Therefore, the genotoxic effects of benzene could be associated to DNA damage (Pandey, 2008).

In this work, the cytotoxic and genotoxic damage has been measured through biomonitoring of the oral mucus in groups of children and applying biomarkers through micronuclei (MN). This technique enables the work with different types of cells such as epithelial cells (Lacerda, 2015; Acito 2022; Çelik, 2003; Carere, 1995; Benites, 2006; Hallare, 2009; Fenech M., 1993; Fenech M. et al., 1999; Fenech M., 2000; Zalacain M. et al., 2005, Neri M. et al., 2006b; Fenech M. et al., 2007; Holland N. et al., 2008), to determine the genotoxicity and damage to the DNA (Angelini, 2012; Bagryantseva, 2010; Rekhadevi, 2010) in the population exposed. Aside from the lack of regulation in the matter, the risk evaluation has not been researched because there is a lack of data in the levels of benzene in the rest of the urban population.

In this study, the relationship between air pollution and (chronic) human exposure to the contaminants has been analyzed, focusing on the schools in which children remain from 5 to 7 hours daily (except for weekends and holidays). The main intention is to evaluate the measure in which the exposure of people to these contaminants seems to become affected while they remain in the area. A Geographic Information System (GIS) has been used in order to investigate the relationship between environmental factors and the damage incidences in micronuclei. The risk was estimated using the data on BTEX distribution for each gas station (GS) in levels of AGEB and there was biomonitoring of 143 children of school age in areas exposed and not exposed to GS. Lastly, there was a correlation between determining the vulnerable areas and the genotoxic damage to define the risk. With this research, it is expected to provide technical information to aid public policies that protect children living in the areas potentially exposed to gas stations.

METHODOLOGY

Two procedures were performed to carry out this study. At the beginning, it was necessary to estimate the amount of volatile organic compounds (VOC) by emissions from service stations in each school in the urban area of Ensenada, Baja California, Mexico, using a geographic information system (GIS) with QGIS software 3.34.5 On the other hand, to determine the biomarkers of cytotoxicity and genotoxicity of school children, we followed the procedures of the oral micronucleus cytome assay (Fenech, 2007); finally a nonparametric statistical analysis was applied to compare the cyto-genotoxic biomarkers of children in school who are or are not exposed to VOC.

VOC in schools : To estimate the amount of volatile organic compounds (VOC) in each school, it was necessary to build a database that was based on the annual sales reports of each service station for the years 2010, 2011 and 2012, provided by the Mexican company that distributes fuels in Mexico (Petróleos Mexicanos, PEMEX), and information on housing, population and urban equipment contained in the units known as the Basic Geo-statistical Area (AGEB) compiled from the Statistics and Geography National Institute (Instituto Nacional de Estadística y Geografia, INEGI) website. With these two data sources it was possible to feed a geographic information system (GIS) with the software QGIS 3.34.5.

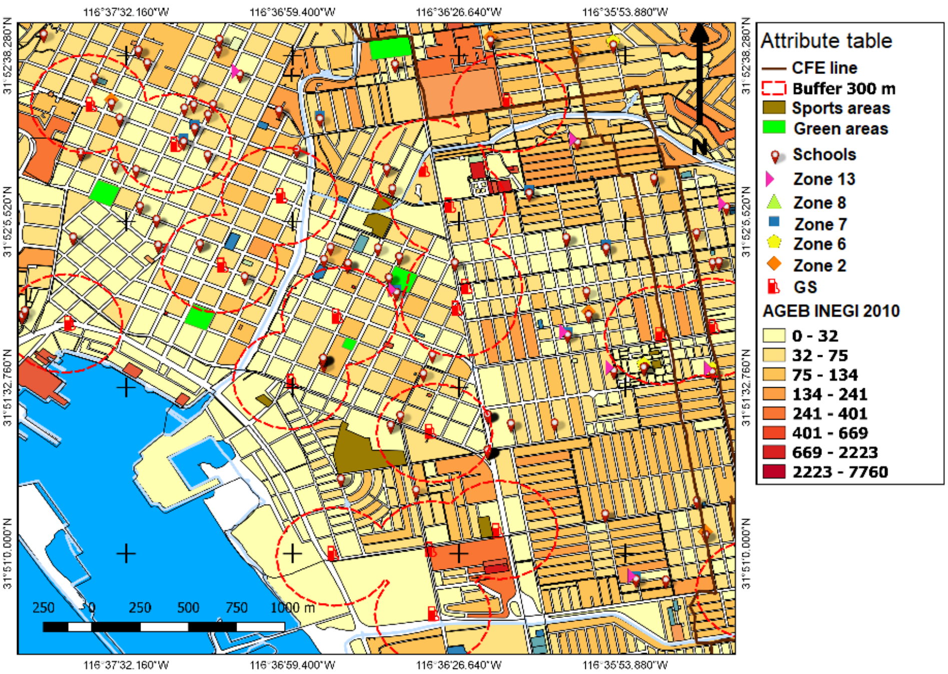
The emissions from gas stations were measured using the factor of emission (FE), which rises to 44.50x10-5 kg L-1 day-1, by Garcia-Zarate et al. (2015), which is the average quantity of volatile organic compounds (VOC) that each gas station throws into the atmosphere in Baja California as a result of the loading and unloading processes (Radian International 1996). This number was estimated based on the chapter AP-42 published in the Environmental Protection Agency (EPA; method 18) of the United States (US EPA, 1995, 1995ª; EPA, 1973, 2003). By other hand, the method used to determine the VOC from area sources by the Secretariat of Labor and Social Benefits (Secretaria del Trabajo y Prevision Social or STPS), NOM-010-STPS-1999. This estimator was important because it allowed for the risk zones by gas station proximity to be determined according to the dispersion model resulting from the annual sales volume and the weather conditions in the area of study.

The values attained from the concentrations of normalized benzene, toluene, ethylbenzene and xylene (BTEX) came as a result of the dispersion model, and using maps, the polygons with different risk levels to the population and schools were gathered for each station within the urban area.

Similarly, data from the school's potentially exposed and not exposed population, healthy children of schooling age, enrolled in the levels of elementary and middle school in Ensenada, Baja California, was incorporated. Figure 1 shows the location of the GS and schools, as well as the Unit of Support Services or Regular Education (Unidades de Servicios de Apoyo a la Educacion Regular USAER-SEP) that helps schools and students in need in Ensenada, Baja California. The selection of the groups of children to make the biomonitoring of genotoxicity and cytotoxicity is based on this model.

Figure 1

Location of schools and USAER-SEP centers within a radius of 300m from the gas stations in the urban area of Ensenada, Baja California



Biomonitoring of cytotoxicity and genotoxicity

In the second part of this study there was a biomonitoring of genotoxic damage in 143 children of schooling age corresponding to three groups: the first two are considered exposed and the third as a control group. The following criteria were considered to make the selection: approval from the children and consent from their parents to include their children in the study. There was a micronuclei (MN) assay on oral epithelial cells (Thomas, 2009; Bonassi, 2011) frequently used in evaluations of people exposed to organic volatile compounds and another cito-genotoxic agents (Patlolla B. et al., 2005).

Briefly, each child rinsed their mouth twice using drinking water, there were two samples taken of the exfoliated buccal cells by gently scraping the inside of the cheek, a extend was made in slide and dry at air, fixated in 80% ethanol and staining with eosin and fast green. 2000 epithelial cells were read in the smears with optical microscope at 100X registering the different biomarkers of genotoxic and cytotoxic damage: micronuclei (MN), nuclear gems or broken eggs (NL), basal cells (BC), binucleated cells (BN), condensate chromatin (CC), karyorrhexis (KR), karyolysis (KL) and Pyknosis (PN) (Torres-

Bugarin, 2013, 2007; Holland et al., 2008; Heddle et al., 1991; Violante, 2012; Grover, 2012; Lacerda, 2015; Ceretti,, 2014; Feretti, 2014; Martins et al. 2009; Majer, 2001).

The amount of nuclear abnormalities was coded into an Excel© sheet. To guarantee homogeneity in the variances and making the comparisons by confidence intervals viable, there was an arcsine transformation of concepts and methodologies given by Bartlett (1936), Anscombe (1948) and *Zar* (*1984*) for the variables distributed according to the Poisson distribution, based on the following expression:

$$\hat{x} = \sqrt{(n + \frac{1}{2})} * aseno \sqrt{\frac{X + \frac{3}{8}}{n + \frac{3}{4}}}$$

$$where:$$

$$\hat{x} = estimator\ of\ the\ transformed\ variable$$

$$n = 2000\ counted\ cells$$

$$X = original\ variable, number\ of\ counted\ anomalies$$

The transformed data was analyzed using the Graph-Pad-Prism© v7 program and a Kruskal-Wallis test with Bonferroni post hoc multiple comparison test was performed to compare the groups of children being studied.

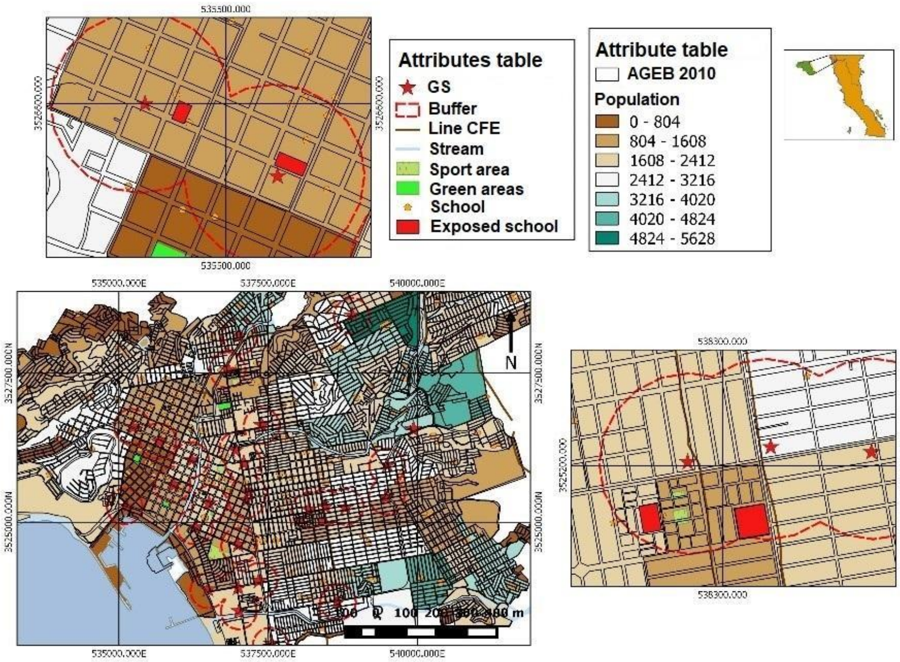
RESULTS

The children at the "Justo Sierra" school (Escuela Primaria Urbana Estatal "Justo Sierra"), located 50 m from a gas station, formed the first group with 48 children exposed (n = 48), 18 children and 30 girls with an average age of 10.17 ± 0.66. Group two was "Sec. 9" (Escuela Secundaria Urbana Estatal Numero 9) at 300 m from the service station with 55 children exposed, 31 boys and 24 girls with an average age of 13.96 ± 0.43. A third group consisting of 40 children was the control group (not exposed) located 425 meters from the nearest service station with 23 children and 17 girls, with an average age of 10.50 ± 0.43.

Following the suggestions of different authors (Kales, 1997; Horton, 2003; Terres, 2010; Silva 2009; Santos, 2013; Karakitsios, 2007; Correa, 2012; Hystad, 2011; Beltrán, 1996; Zagal, 1996; CENAPRED, 2001; Arcos, 2007; Rivera, 2006), the type of pollutant, the type of exposure (point source or diffuse) and the location of schools within a 300 m radius of each petrol station were used to determine the level of exposure in the geographical scenarios of this study. It was found that many petrol stations in cities are located close to residential buildings or educational institutions. The locations of the schools with the highest emissions from petrol stations in relation to the emission factor are shown in Figure 2.

Figure 2

Location of schools in sampling



The increase in nuclear abnormalities could be due to regular exposure to gasoline vapours (BTEX) from the GS, which contain genotoxic compounds and alter the structural make-up of all cells in the bone marrow (Bukvic, 1998). They are also leukaemogenic and anaplastic, which has shown that there is a significant difference in the rate of micronuclei and nuclear abnormalities between the population exposed to BTEX in the environment from GS (Table I) and the unexposed group. In Justo Sierra, the percentage of students of both sexes exposed to the contaminant (F/M) was 30/18 (65%/27%), while in the unexposed group it was 17/23 (43%/57%).

Table 1

The mean frequency of micronuclei per 2000 cells of the buccal mucosa in the groups studied

					MN / 2000			
Group	**Men**	**Women**	**n**	**Distance to GS (m)**	**min**	**max**	**cases**	**%**
Justo sierra	18	30	48	50	0	7	20	42%
Sec. No. 9	31	24	55	300	0	3	23	42%
UNEXPOSED POPULATION	23	17	40	425	0	1	3	8%

Exposure-induced damage to genetic material may lead to genotoxic or cytotoxic events in epithelial cells where the primary chromosomal abnormalities required for classification have been observed. According to the exposure to genotoxic and cytotoxic damage leading to cell death, such as necrosis and apoptosis, Table II shows the mean and standard error for each cellular abnormality. (Filipo, 2018; Ceretti, 2014).

Table 2

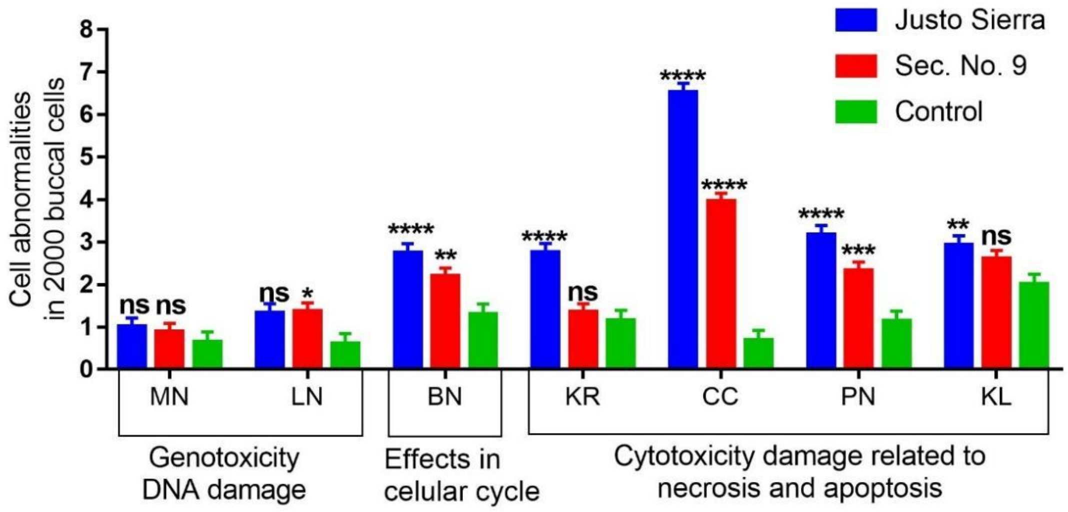
Basic statistics for transformed variables by each one nuclear anomalies for three of children groups

Type of damage	Biomarker	School	Mean	Standard error	Inferior Confidential Limit (95%)	Superior Confidential Limit (95%)	N
Genotoxicity	Micronuclei (MN)	Justo Sierra	1	0.22	0.57	1.43	48
		Sec. 9	0.89	0.2	0.49	1.29	55
		Control	0.65	0.24	0.19	1.12	40
	Lobulated Nucleus (LN)	Justo Sierra	1.33	0.22	0.9	1.76	48
		Sec. 9	1.37	0.2	0.97	1.77	55
		Control	0.61	0.24	0.14	1.08	40
Effects in cellular cycles	Binucleated (BN)	Justo Sierra	2.74	0.22	2.31	3.17	48
		Sec. 9	2.19	0.2	1.79	2.58	55
		Control	1.3	0.24	0.83	1.77	40
Cytotoxicity: Necrosis and Apoptosis	Karyorrhexis (KR)	Justo Sierra	2.75	0.22	2.32	3.17	48
		Sec. 9	1.35	0.2	0.95	1.74	55
		Control	1.16	0.24	0.69	1.63	40
		Justo Sierra	6.52	0.22	6.09	6.95	48
	Condensed Chromatin (CC)	Sec. 9	3.95	0.2	3.55	4.35	55
		Control	0.68	0.24	0.22	1.15	40
	Pyknosis (PN)	Justo Sierra	3.17	0.22	2.74	3.6	48
		Sec. 9	2.33	0.2	1.93	2.73	55
		Control	1.14	0.24	0.67	1.61	40
	Karyolysis (KL)	Justo Sierra	2.93	0.22	2.51	3.36	48
		Sec. 9	2.6	0.2	2.2	3	55
		Control	2	0.24	1.54	2.47	40

The frequency of micronuclei and nuclear abnormalities in the control group is shown in graphic 1, and the exposed groups - including the Justo Sierra and Sec. No. 9 boys - show that those exposed to BTEX have significantly more DNA damage than the control group.

Graphic 1

Biomarkers of genotoxic and cytotoxic damage in 2000 cells in children of the "Justo Sierra" and "Sec. No. 9" groups and control



The vertical lines express the standard error P ± 0.95. The asterisks and "ns" indicate significance level with the Bonferroni multiple comparisons test against the control group.

Analysis of exfoliated cells from buccal mucus reveals evidence of other nuclear changes, including binucleated cells, karyorrhexis and karyolysis. According to Carlin et al (2010), the presence of binucleated cells is thought to be a sign of cytotoxicity, DNA damage and cell death. In addition, chromosomal abnormalities caused by DNA breakage are also thought to be caused by karyorrhexis and karyolysis, which can result in both minor and major structural changes or be removed by the mechanism of cellular repair through cell death, which is planned as an indicator of apoptosis (Tolbert, 1992). Only that children are more susceptible than adults has been suggested by previous research (Lakhanisky, 1993; Laurent, 1993; Klemans, 1995).

Apoptotic cells differ from necrotic cells in that the latter undergo "passive" cell death, including a rupture of the plasma membrane that releases intracellular material and triggers an inflammatory response (Wyllie, 1980). On the other hand, apoptosis, also known as physiological death, is an active process involving protein synthesis and characterised by cytoplasmic and nuclear condensation (pyknosis). The final stage of internucleosomal fragmentation and DNA rupture occurs in the cytoplasm, where there is nuclear chromatin condensation, nuclear disintegration, reduced damage, cytoplasmic and organelle compaction, and a bubble-like membrane (Wyllie, 1984). On the other hand, an increase in apoptosis could be a sign of genotoxic damage (Tolbert, 1992). It has been said that apoptosis occurs by ionizing radiation, as well as, chemical compounds that attach to the DNA or by genetic deregulation. It can also act as a recognition mechanism eliminating the cells with genetic damage. In this way, the excess of apoptosis could indicate genotoxic damage (Tolbert, 1992). Aneuploidies (both germinal and somatic) are associated with significant genetic changes linked to spontaneous miscarriage, mental retardation and cancer, although rupture can lead to cell death (Hagmar, 1994).

CONCLUSIONS

The unplanned growth of urban areas has led to the inappropriate use of land for various economic activities, which becomes problematic over time. The use of GIS can help predict the impact on educational centres. Based on the distance from the GS, the vulnerability of schools is strongly related to their location.

This is the first study carried out on a group of children using the MN test, from which we can say that the genotoxic effect is caused by exposure to petrol emissions (BTEX). The test for micronuclei in buccal epithelial cells is one of the most sensitive methods used to measure the rate of DNA damage in the human population, as it is relatively easy to determine damage in micronuclei compared to other methods, such as chromosomal aberrations. It can be used not only to identify groups at risk of developing cancer, but also to identify individuals who are susceptible to developing cancer.

The results indicate that people exposed to the chemicals of the GS are groups at risk, taking into account that they are strong mutagens and that the emission of these products has genotoxic and cytotoxic properties, as demonstrated by Hadnagy (1988, 1989) in buccal cells. It was found that the frequency of micronuclei and nuclear abnormalities, genotoxic damage and health consequences increased with exposure, in correlation with the increasing number of petrol stations in urban areas.

The levels of apoptosis observed in this study are significantly higher in the exposed group, as observed by Revazova et al. (2001). All the cytotoxic and mutagenic changes observed in this document are related to the strength of petrol and its components, especially benzene.

The methodology used in the GIS is not difficult to apply and the results can be considered for prevention, territorial planning and minimisation of risks due to the presence of GS within a radius of 300 m around schools.

These factors could contribute to a chronic exposure at low concentrations, posing a threat to the health, productivity and efficiency of children in neighbouring schools, considered a public health issue and detectable with genotoxicity tests.

The public health sector can play a major role in promoting the prevention of exposure to air pollution. Improving air quality should be considered as an important part of public policy making in various economic sectors. The effects on health are highly unequal: air pollution combines with other aspects to create a disproportionate morbidity burden for low-income groups. All this should be taken into account when designing and implementing long-term policies and programmes to reduce air pollution and improve overall health. The results suggest the need for environmental policies to reduce the burden of GHS, which results from neglecting the vulnerability of children when setting limits for exposure to pollutants in urban areas. It is known that among the most serious diseases are genetic diseases caused by genotoxic effects on somatic cells, such as cancer. It is therefore very important to publicise these studies in order to encourage the public authorities to inform themselves of the possible health risks when authorising filling stations and to find solutions that could help to maintain the environmental balance in urban areas.

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