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ASSESSMENT OF 8-OXO-7,8-DIHYDRO-2'-DEOXYGUANOSINE AS A MARKER OF OXIDATIVE DNA DAMAGE IN GASOLINE FILLING STATION ATTENDANTS

RAVICHANDRAN BEERAPPA, DHANANJAYAN VENUGOPAL, SOMNATH SEN, MALA AMBIKAPATHY, and RAJMOHAN HIREHAL RAGHAVENDRA RAO

Regional Occupational Health Center (S), Bangalore, India
Industrial Hygiene & Toxicology Division

Abstract
Objectives: The urinary excretion of 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG) was used as a biomarker of oxidative DNA damage. The urinary 8-oxodG levels in petrol filling station attendants (exposed) at various petrol bunks were estimated as well as in the unexposed (cashier) population. **Materials and Methods:** A total of 100 workers (79 petrol fillers and 21 cashiers) aged from 20 to 41 years participated in the study. An informed consent was taken from each participant. Information on personal habits and health was obtained through a questionnaire. After shifts, urine samples were collected analyzed for 8-oxodG using enzyme-linked immunosorbent assay (ELISA). **Results:** Fifty-three percent of workers were in the 21–30 years age group. The maximum level of 8-oxodG was observed in the age group ≥ 41 years and the minimum in the age group of 31–40 years. The maximum level of 8-oxodG was observed among those workers who had ≥ 21 years of experience. The concentrations of 8-oxodG were significantly higher in petrol fillers than those in cashiers ($p < 0.05$). **Conclusions:** Despite the conflicting results obtained in our study it was shown that 8-oxodG is related to chemical exposure. Further research is needed embracing a bigger number of participants to highlight the correlations between the exposure and the effects.

Key words:
DNA damage, ELISA, Petrol filling attendants, Urinary 8-oxo-7,8-dihydro-2'-deoxyguanosine

INTRODUCTION

Occupational exposure to petroleum derivatives poses genotoxic risk [1,2]. Epidemiological studies showed a clear relationship between the increase in micronucleus frequency and exposure to benzene and benzene metabolites [3,4]. The persons exposed to those substances include service station attendants, drivers of gasoline tank trucks, workers at bulk loading terminals, workers who clean up gasoline spills and leaks, and refinery workers. Petrol (gasoline) is a complex mixture of low molecular mass compounds, mainly paraffinic, naphtenic, olefinic and aromatic, with a carbon number typically within the range of 3–11 and a boiling point in the range of 30–260°C.

Petrol station attendants are chronically exposed to petroleum derivatives primarily through inhalation of the volatile fraction of petrol during vehicle refueling [5]. In urban settings, these workers are exposed to relatively high levels of petrol vapors in their workplace and to low levels of

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Corresponding author: B. Ravichandran, Scientist, Industrial Hygiene & Toxicology Division, Regional Occupational Health Centre (S), Kannamangala Post, Bangalore – 562 110, India (e-mail: ravichandranrohc@gmail.com).

petrol vapors affecting the general population [6]. In addition, exposure to traffic-generated particles increases the burden of oxidative stress occurring due to the particulate matter ($PM_{2.5}$) constituents since the petrol filling stations are always located nearby busy roads [7]. Significant decreases in red blood cell (RBC) counts, hemoglobin values, packed cell volume (PCV), mean corpuscular hemoglobin concentration (MCHC) and white blood cell counts were noticed in petrol filling station attendants exposed to petroleum fumes for a number of years [8].

Previous studies on exposure to volatile organic compounds (VOCs) involving individuals whose occupations are associated with exposure to petrol vapor emissions yielded evidence that these workers were exposed to significantly higher levels of aromatic hydrocarbons. A study conducted in Thailand showed that a stronger exposure to benzene affected service station attendants than representatives of any other studied occupation [9]. Our recent study on health risk assessment of rural and urban population due to indoor/ambient air pollution of Bangalore city showed that petrol filling station attendants had detectable levels of benzene, toluene and m-xylene exposure at their workplace [10].

The development of oxidative DNA damage coupled with interference in DNA repair processes is likely to play an important role in the development of cancer [11]. Studies showed that urinary 8-oxodG is a good biomarker for risk assessment of various cancers and degenerative diseases [12]. In recent years, 8-oxodG has been widely used in many studies not only as a biomarker for the measurement of endogenous oxidative DNA damage, but also as a risk factor for many diseases including cancer. 8-oxodG is a particular type of Reactive Oxygen Species (ROS)-induced DNA base modification (i.e. C8 hydroxylation of deoxyguanosine). 8-oxodG is premutagenic because it can mispair with adenine instead of cytosine during DNA replication, and if this occurs, a G → T transversion mutation develops. The repair mechanism for DNA after 8-oxodG is incorporated into DNA involves base and nucleotide excision with DNA-specific nucleosides excreted into urine [13]. ROS can induce various kinds of damage to DNA, including oxidized bases which will then be repaired and eliminated in urine. The urinary measurement was used in several studies among subjects with occupational or environmental exposure. Therefore, the measurement of urinary 8-oxodG can be used as an overall estimate of repaired oxidative DNA damage within an individual [14].

The main objective of this study was to investigate the effect of inhalation of petroleum products on the urinary levels of the oxidative injury biomarker, 8-oxodG, in petrol filling station workers. The results were corrected for confounding factors such as age and smoking habits.

MATERIALS AND METHODS

Prior to the study, permission was obtained from each petroleum product company for authorizing the outlets to participate in the study. An informed consent was taken from each participant. Ethical approval was obtained from our center's ethical committee. A total of 100 after-shift urine samples was collected for the estimation of 8-oxodG among petrol filling station attendants from different petrol stations located in Bangalore city. Before sampling, a questionnaire survey was conducted at the petrol filling stations to obtain information on the quantity of petrol sold each day. Based on this data, the volume of petrol sold per month was calculated and the maximum selling petrol filling stations were identified for inclusion in our study. Information about the characteristics of the attendants (age, length of employment, smoking habits, alcohol consumption and food habits, etc.) was collected through a questionnaire. The collected urine samples were centrifuged at 2000 rpm for 10 min and the supernatants were used for the determination of 8-oxodG.

The creatinine level was estimated using a semiauto analyzer (Model RA-50) based on the Jaffe's method. Urinary 8-oxodG levels were determined using a competitive ELISA

immunoassay (Japan Institute for the Control of Ageing, Fukuroi, and Shizuoka, Japan) [15,16]. 50-µl of urine samples and standards were added to precoated 8-oxodG protein conjugate microtiter plates followed by 50 µl of the primary antibody, anti-8-oxodG monoclonal antibody solution. After incubation for 1 h at 37°C, the plates were washed and the enzyme-labeled secondary antibody (100 µl) was applied for 1 h at 37°C. After washing, 100 µl of the chromatic substrate, 3,3',5,5'-tetramethylbenzidene were added to the plate and allowed to react at room temperature for 15 min. The intensity of color produced for each sample was measured at the absorbance of 490 nm. All the values of urinary 8-oxodG levels were subsequently adjusted by urinary creatinine levels.

Statistical analysis

Statistical analyses were performed using SPSS statistical software version 10. Mean, standard error, median and inter-quartile range were determined for all subjects. Due to positively skewed distribution of data, the median test was performed to compare 8-oxodG levels between the groups. Multiple regression analysis was performed after log transformation to evaluate the relationship between the 8-oxodG levels and personal habits of the study subjects. Spearman rank correlation was applied to test the relationship between the 8-oxodG levels and working hours with respect to occupation. The value $p < 0.05$ was considered to mark significant differences. Student't-distribution and Chi^2 were applied to identify the differences in age, experience and personal habits of the study groups.

RESULTS AND DISCUSSION

The distribution of study subjects according to age and job category in petrol filling stations is given in Table 1. Fifty-three percent, i.e. half of the study subjects, fall in the middle age group. Most of them (79%) worked as petrol filling attendants, whereas 21% worked as cashiers. Seventy-eight percent of the subjects had the length of employment in this profession shorter than ten years. The number of non-smokers (79%) was higher than the number of smokers (21%). The levels of 8-oxodG among the study subjects according to their age groups are given in Table 2. The maximum level

Table 1. Distribution of study subjects according to age and job category

Variable	Study group (n)
Age (years)	
≤ 20	15
21–30	53
31–40	21
≥ 41	11
Job category	
cashier	21
petrol filler	79

Table 2. Levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG) among various study groups according to age

Age group (years)	Study group (n)	8-oxodG (µg/g creatinine)		
		M±SE	median	inter-quartile range
≤ 20	15	4.65±2.00	1.17	3.79
21–30	53	2.97±0.56	1.27	2.83
31–40	21	1.57±0.32	1.04	2.82
≥ 41	11	4.08±1.99	1.53	4.58
Total	100	3.05±0.48	1.27	2.66

M – mean; SE – standard error.
$p > 0.05$ median test.

of 8-oxodG was recorded in the age group ≥ 41 years compared to other age groups. The lowest levels were reported in the age group of 31–40 years (Table 2).

The distributions of the study subjects according to personal habits and the length of employment are given in Table 3 and Table 4. The 8-oxodG levels recorded among the study subjects based on the years of professional experience are not significant ($p > 0.05$). The inhalation exposure depends upon the quantity of petroleum products the workers handle during their work. There was a significant correlation (Spearman rank correlation) observed between working hours per day and the 8-oxodG levels in both groups of workers (Figure 1).

The data on the quantity of petroleum products sold per day was collected, which indirectly revealed the exposure. In accordance with the data from Table 5, higher levels of 8-oxodG were recorded among the workers who handled ≥ 4500 l of petrol per day. The levels of 8-oxodG detected among workers were proportional to the quantity of petrol sold. Since petrol filling station job assignments vary for each individual, the subjects were categorized into two groups (petrol fillers and cashiers) based on

Table 3. Distribution of study subjects according to personal habits

Variables	Study group (n)		p
	cashier (n = 21)	petrol filler (n = 79)	
Age (years), M±SE	31.10±6.62	27.37±8.86	0.067*
Employment (years), M±SE	8.76±6.24	7.30±7.98	0.439*
Alcohol intake			
yes	9	25	
no	12	54	0.335**
Smoking			
yes	6	15	
no	15	64	0.919**
Tobacco chewing			
yes	2	2	
no	19	77	0.193**
Tea/coffee			
yes	21	74	
no	–	5	–
Food habit			
vegetarian	3	3	
non-vegetarian	18	76	0.105**
Physical activity			
yes	5	24	
no	16	55	0.787**

M – mean; SE – standard error.
* t-test.
** Chi^2 test.

Table 4. Levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG) among various study groups according to work experience

Employment (years)	Study group (n)	8-oxodG (μg/g creatinine)		
		M±SE	median	inter-quartile range
≤ 10	78	3.22±0.54	1.28	2.92
11–20	13	1.12±0.36	0.44	2.38
≥ 21	9	4.39±2.38	1.84	3.51
Total	100	3.05±0.48	1.27	2.66

Abbreviations as in Table 3.

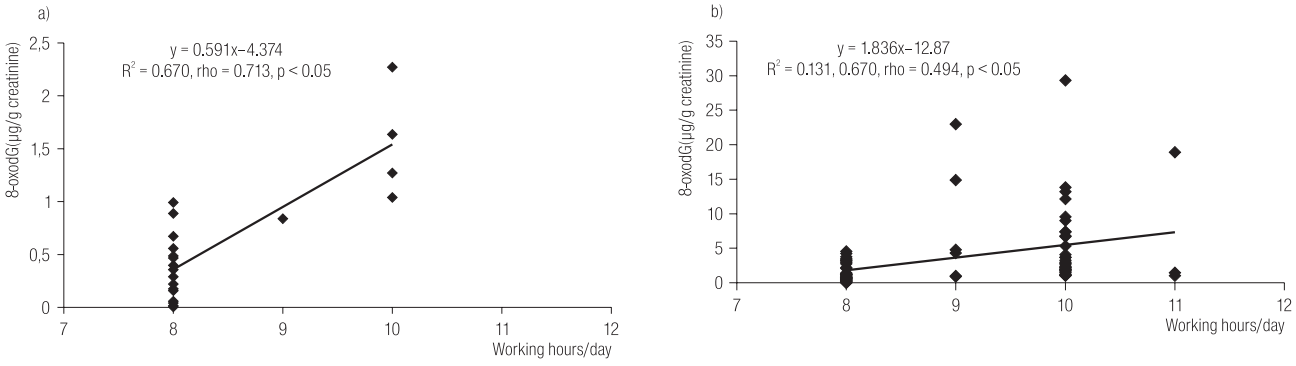

Fig. 1. Correlation between 8-oxo-7,8-dihydro-2'-deoxyguanosine levels and working hours: a) cashier, b) petrol filler

the nature of their job. Significant variations in 8-oxodG levels were observed ($p < 0.05$) between the petrol fillers (1.86 μg/g creatinine) and the cashiers (0.47 μg/g creatinine) (Table 6). A similar study conducted on other occupations showed higher 8-oxodG levels in taxi drivers than in the general population of men [17]. Another study on urinary 8-oxodG as a biomarker of oxidative DNA damage in workers exposed to fine particulates was reported by Kim et al. [18].

Since the lifestyle of a person plays a role in the excretion of 8-oxodG, the obtained data was categorized based on the various habits of the workers. In the present study, the

Table 5. Levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG) among subjects according to the quantity of petrol sold per day

Quantity of petrol (l)	Study group (n)	8-oxodG (μg/g creatinine)		
		M±SE	median	inter-quartile range
≤ 1 500	37	3.42±0.92	1.14	1.89
1 501–3 000	51	2.75±0.57	1.27	2.91
3 001–4 500	6	4.52±2.97	1.42	1.70
≥ 4 501	6	1.90±0.59	1.77	1.02
Total	100	3.05±0.48	1.27	2.66

Abbreviations as in Table 3.

Table 6. Levels of 8-oxo-7,8-dihydro-2'-deoxyguanosine 8-oxodG) among various study groups according to habits and occupation

Parameters	Study group (n)	8-oxodG (μg/g creatinine)			p (median test)
		M±SE	median	inter-quartile range	
Occupation					
cashier	21	0.61±0.13	0.47	0.77	0.001
petrol filler	79	3.70±0.58	1.86	2.84	
Smoking					
yes	21	2.77±1.06	1.30	2.71	0.806
no	79	3.13±0.54	1.24	2.67	
Alcohol habit					
yes	34	2.23±1.01	0.56	2.48	0.673
no	66	3.48±0.66	1.50	2.72	
Tobacco chewing					
yes	4	11.01±6.87	7.24	25.11	1.000
no	96	2.72±0.40	1.27	2.61	
Tea/coffee					
yes	95	3.11±0.51	1.27	2.67	0.646
no	5	2.48±0.82	2.68	3.19	
Vegetarian					
yes	6	5.59±4.76	0.75	8.64	0.674
no	94	2.89±0.42	1.28	2.62	
Physical activity					
yes	29	2.52±0.68	1.24	2.65	0.826
no	71	3.27±0.62	1.41	2.66	

Abbreviations as in Table 3.

smokers (1.30 μg/g creatinine) and non-smokers (1.24 μg/g creatinine) did not show any variations in their 8-oxodG levels (Table 6). However, it was reported that the smoking habit and other personnel characteristics increased the excretion of 8-oxodG in urine [19,20]. Various studies on exposure to a variety of carcinogens, including PAHs and asbestos, and their relationship with the smoking status and elimination of urinary 8-oxodG did not show any correlation [14,21,22]. Whereas a study by Nilsson et al. [23] failed to show the effect of smoking on the urinary excretion of 8-oxodG, studies by Kim et al. [18] reported a significant difference in the 8-oxodG concentration between smokers and non-smokers. Smoking has been consistently identified as a confounder for 8-oxodG, but various occupational studies did not reveal higher levels of 8-oxodG [24] in smokers as indicated by the present study.

A study by Besarati Nia et al. [25] reported that the level of 8-oxodG was consistently increased among smokers. Heavy exposure to air pollution in occupational settings in terms of diesel exhaust, polycyclic aromatic hydrocarbons and benzene was associated with increased 8-oxodG excretion, whereas non-occupational exposure to ambient air pollution was not significantly lower in smokers as compared with non-smokers [26,23].

Table 7. Levels 8-oxo-7,8-dihydro-2'-deoxyguanosine (8-oxodG) among various sub-groups of subjects according to occupation and habits

Parameters	Study group (n)	8-oxodG (μg/g creatinine)			p (median test)
		M±SE	median	inter-quartile range	
Smoking					
cashier					0.361
yes	6	0.81±0.31	0.58	0.86	
no	15	0.53±0.13	0.36	0.93	
petrol filler					0.957
yes	15	3.56±1.44	2.76	2.37	
no	64	3.73±0.64	1.86	3.24	
Alcohol intake					
cashier					1.000
yes	9	0.63±0.25	0.47	1.03	
no	12	0.60±0.13	0.48	0.71	
petrol filler					0.373
yes	25	2.80±0.73	1.41	2.86	
no	54	4.12±0.78	2.11	3.04	
Vegetarian					
cashier					1.000
yes	3	0.52±0.28	0.47	0.06	
no	18	0.63±0.14	0.44	0.74	
petrol filler					0.982
yes	3	10.66±9.34	2.21	0.44	
no	76	3.43±0.50	1.86	2.79	

Abbreviations as in Table 3.

In the recent years, many youngsters have taken up a habit of chewing smokeless tobacco in our country for unknown reasons. Hence, we also looked into the effect of chewing this smokeless tobacco among the examined workers. As a result, elevated levels of 8-oxodG in chewers were noted (7.24 μg/g creatinine) compared to non-chewers (1.27 μg/g creatinine) (Table 6). However, the difference was not statistically significant. The present study did not find any influence on the levels of 8-oxodG exerted by lifestyle habits (Table 7).

Although the 8-oxodG levels differed between the categories, the differences were not significant. Nevertheless, large numbers of samples are needed to ascertain a better conclusion based on the personnel characteristics. To investigate the effect of possible personal habits of study subjects on the urinary 8-oxodG levels, multiple regression analysis was performed (Table 8). It was observed that in the workers involved in petrol filling working hours of exposure had influence on the excretion levels of 8-oxodG ($p < 0.05$). However, the personal habits of the study subjects did not influence the levels of 8-oxodG. Although the levels were not significantly different, a marginal variation was observed in all categories.

Table 8. Multiple regression analysis of 8-oxo-7,8-dihydro-2'-deoxyguanosine

Independent variables	Regression coefficient	Standard error	p
Cashier vs. petrol filler	0.832	0.163	0.001
Age (years)	0.011	0.013	0.385
Employment (years)	–0.012	0.014	0.403
Alcohol habit (yes vs. no)	0.209	0.135	0.127
Smoker (yes vs. no)	–0.083	0.159	0.604
Physical activity (yes vs. no)	0.097	0.141	0.495
Tobacco chewing (yes vs. no)	–0.565	0.332	0.093
Tea/coffee (yes vs. no)	0.075	0.304	0.807
Food habits (vegetarian vs. non-vegetarian)	–0.121	0.281	0.669
Working hours	0.363	0.060	0.001

Nilsson et al. [23] reported that dietary factors can probably also influence the excretion of 8-oxodG. Kasai et al. [27] indicated that low meat intake (less often than once/week) induces an increase of 8-oxodG. The attributing factor of low consumption of meat may be associated with the serious modulation of other dietary factors, which might induce oxidative stress. Another possibility was that meat components may be required to scavenge oxygen radicals or to repair DNA damage efficiently. Kasai et al. [27] also studied the effects of exercise, working conditions, meat intake, body mass index, smoking habit and other lifestyle factors on 8-oxodG excretion. Our present study did not confirm the statistical significance reported by various other researchers, but a detailed study is required focusing on lifestyle habits and their influence on the 8-oxodG excretion among exposed workers.

CONCLUSION

Petrol evaporates more readily in hot than cold countries. In India, unlike in the western countries, employing attendants operating petrol pumps is more popular than self-service. These workers are prone to develop occupational diseases since they do not wear personal protective masks and do not follow personal hygiene rules at the workplace. Under prevailing conditions, a mixture of components present in the petrol can be absorbed through inhalation. The results were clued-up to the workers as well as the owners of the petrol pump stations.

Our results revealed that the level of urinary 8-oxodG is associated with occupation. The differences between the results of the present study and those obtained in other investigations might be explained by variations in the sample size, sample composition, and methods of urinary 8-oxodG measurement. Inclusion of a greater number of petrol filling station attendants might highlight the association of 8-oxodG excretion and their exposure to air pollutants. Despite the conflicting results obtained in our study it was shown that 8-oxodG is associated with chemical exposure. Further research is needed to demonstrate the correlation between the exposure and effects.

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REFERENCES

1. Oesch F, Fuchs J, Vaupel J, Hengstler JG. DNA single strand break analysis in mononuclear blood cells of petrol pump attendants. Int Arch Occup Environ Health. 1995;67:35–9, http://dx.doi.org/10.1007/BF00383130.
2. Bukvic N, Bavaro P, Elia G, Cassano F, Fanelli M, Guanti G. Sister chromatid exchange (SCE) and micronucleus (MN) frequencies in lymphocytes of gasoline station attendants. Mutat Res. 1998;415:25–33, http://dx.doi.org/10.1016/S1383-5718(98)00045-X.
3. Tompa A, Major J, Jakab MG. Monitoring of benzene exposed workers for genotoxic effects of benzene improved working conditions related decrease in the frequencies of chromosomal aberrations in peripheral blood lymphocytes. Mutat Res. 1994;304:164–5, http://dx.doi.org/10.1016/0027-5107(94)90207-0.
4. Turkel B, Egeli U. Analysis of chromosomal aberrations in shoe workers exposed long term to benzene Occupant. Environ Med. 1994;51:50–3, http://dx.doi.org/10.1136/oem.51.1.50.
5. Celik A, Cavas T, Ergene-Gozukara S. Cytogenetic biomonitoring in petrol stations attendants: micronucleus test in exfoliated buccal cells. Mutagenesis. 2003;18:417–21, http://dx.doi.org/10.1093/mutage/geg022.
6. Pitarque M, Carbonell E, Lapena N, Marsa M, Valbuena A, Creus A, et al. SCE analysis in peripheral blood lymphocytes of a group of petrol filling station attendants. Mutat Res. 1997;390:153–9, http://dx.doi.org/10.1016/S0165-1218(97)00011-6.
7. Wei Y, Han IK, Shao M, Hu M, Zhang OJ, Tang X. PM2.5 constituents and oxidative DNA damage in humans. Environ Sci Technol. 2009;43:4757–62, http://dx.doi.org/10.1021/es803337c.
8. Okoro AM, Ani EJ, Ibu JO, Akpogomeh A. Effect of Petroleum products inhalation on some hematological indices of fuel attendants in Calabar metropolis, Nigeria. Niger J Physiol Sci. 2006;21:71–5.
9. Navasumrit P, Chanvaivit S, Intarasunanont P. Environmental and occupational exposure to benzene in Thailand. Chem Biol Interact. 2005;153–4:75–83, http://dx.doi.org/10.1016/j.cbi.2005.03.010.
10. National Institute of Occupational Health. Health risk assessment of rural and urban population due to indoor/ambient air pollution. Ahmadabad: NIOH, ICMR; 2006. p. 1–115.
11. Cerutti PA. Oxy-radicals and cancer. Lancet. 1994;344:862–3, http://dx.doi.org/10.1016/S0140-6736(94)92832-0.
12. Valavanidis A, Vlachogianni T, Fiotakis C. 8-hydroxy-2'-deoxyguanosine (8-OHdG): A critical biomarker of oxidative stress and carcinogenesis. J Environ Sci Health C Environ Carcinog Ecotoxicol Rev. 2009;27:120–39, http://dx.doi.org/10.1080/10590500902885684.
13. Pilger A, Germadnik D, Schaffer A. 8-Hydroxydeoxyguanosine in leukocyte DNA and urine of quartz-exposed workers and patients with silicosis. Int Arch Occup Environ Health. 2000;73:305–10, http://dx.doi.org/10.1007/s004200000117.
14. Toraason M, Hayden C, Marlow D. DNA strand breaks, oxidative damage, and 1-OH pyrene in roofers with coal-tar pitch dust and/or asphalt fume exposure. Int Arch Occup Environ Health. 2001;74:396–404, http://dx.doi.org/10.1007/s004200100238.
15. Leinonen J, Lehtimaki T, Toyokuni S. New biomarker evidence of oxidative DNA damage in patients with non-insulin-dependent diabetes mellitus. FEBS Lett. 1997;417:150–2, http://dx.doi.org/10.1016/S0014-5793(97)01273-8.
16. Jikimoto T, Nishikubo Y, Koshiba M. Thioredoxin as a biomarker for oxidative stress in patients with rheumatoid arthritis. Mol Immunol. 2002;38(10):765–72, http://dx.doi.org/10.1016/S0161-5890(01)00113-4.
17. Chuang C-Y, Lee C-C, Chang Y-K, Sung F-C. Oxidative DNA damage estimated by urinary 8-hydroxydeoxyguanosine: influence of taxi driving, smoking and areca chewing. Chemosphere. 2003;52:1163–71, http://dx.doi.org/10.1016/S0045-6535(03)00307-2.
18. Kim JY, Mukherjee S, Ngo L, Christiani DC. Urinary 8-oxo-7,8-dihydro-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates. Environ Health Perspect. 2004;112:666-71, http://dx.doi.org/10.1289/ehp.6827.

19. Lagorio S, Tagesson C, Forastiere F, Lavarone I, Axelson O, Carere A. Exposure to benzene and urinary concentrations of 8-hydroxydeoxyguanosine, a biological marker of oxidative damage of DNA. Occup Environ Med. 1994;51:739–43, http://dx.doi.org/10.1136/oem.51.11.739.
20. Toraason M. 8-hydroxyguanosine as a biomarker of workplace exposure. Biomarkers. 1999;4:3–26, http://dx.doi.org/10.1080/135475099230967.
21. Tagesson C, Chabiuk D, Axelson O, Baranski B, Palus J, Wyszynska K. Increased urinary excretion of the oxidative DNA adduct, 8-Hydroxydeoxyguanosine as a possible early indicator of occupational cancer hazards in the asbestos, rubber and azo-dye industries. Pol J Occup Med Environ Health. 1993;6:357–68.
22. Hong Y-C, Park H-S, Ha E-H. Influence of genetic susceptibility on the urinary excretion of 8-hydroxydeoxyguanosine of firefighters. Occup Environ Med. 2000;57:370–5, http://dx.doi.org/10.1136/oem.57.6.370.
23. Nilsson R, Nordlinder R, Moen BE, Ovrebo S, Bleie K, Skorve AH, et al. Increased urinary excretion of 8-hydroxy deoxyguanasine in engine room personnel exposed to polycyclic aromatic hydrocarbons. Occup Environ Med. 2004;61:692–6, http://dx.doi.org/10.1136/oem.2003.007435.
24. Pilger A, Rüdiger HW. 8-oxo-7,8-dihydro-2'-deoxyguanosine as a marker of oxidative DNA damage related to occupational and environmental exposures. Int Arch Occup Environ Health. 2006;80:1–15, http://dx.doi.org/10.1007/s00420-006-0106-7.
25. Besarati Nia A, Van Schooten FJ, Schilderman PAEL, De Kok TMCM, Haenen GR, Van Herwijnen MHM, et al. A Multi-biomarker approach to study the effects of smoking on oxidative DNA damage and repair and antioxidative defense mechanisms. Carcinogenesis. 2001;21:395–401.
26. Nilsson RI, Nordlinder RG, Tagesson C, Walles S, Jarvholm BG. Genotoxic effects in workers exposed to low levels of benzene from gasoline. Am J Ind Med. 1996;30:317–24, http://dx.doi.org/10.1002/(SICI)1097-0274(199609)30:3<317::AID-AJIM10>3.0.CO;2-Z.
27. Kasai H, Iwamoto-Tanaka N, Miyamoto T, Kawanomi K, Kawanami S, Kido R, et al. Life style and urinary 8-hydroxydeoxyguanosine, a marker of oxidative DNA damage: effects of exercise, working conditions, meat intake, body mass index, and smoking. Jpn J Cancer Res. 2001;92: 9–15, http://dx.doi.org/10.1111/j.1349-7006.2001.tb01041.x.