

Oral Health Status In Relation To Salivary IgA And Antioxidants Among A Group Of Gasoline Stations Workers

Ahlam T. Mohammed

B.D.S., M.Sc.- Assis. Prof. Department of Pedodontic and Preventive Dentistry. College of Dentistry - University of Baghdad

ABSTRACT

Background: Workers of gasoline stations have the potential to be exposed to hazardous substances that are present in the occupational environment like gasoline vapors which is carcinogenic to the humans. The aim of this study was to evaluate salivary IgA and salivary antioxidants and the relationship of these variables to oral health status among workers in gasoline stations.

Materials and methods: 44 males aged (33-39 year) were enrolled in this study, 23 of them were workers in gasoline station for at least five years and 21 healthy males were control group matching the workers in age. Stimulated saliva was collected and subjected to biochemical analysis for detection of salivary IgA and antioxidants (uric acid, magnesium and zinc ions). Oral health status was evaluated by using decayed, missing and filled surface index (DMFS), plaque index (PII), gingival index (GI), calculus index (Call) and periodontal pocket depth (PPD).

Results: Salivary IgA was lower for the study group compared to the control group with highly significant difference ($P < 0.01$) while uric acid was significantly higher for study group than the control one ($P < 0.05$). Similarly magnesium level was higher among the workers than the control group though the difference was not significant ($P > 0.05$). In contrast the level of salivary zinc was higher for the control group than the study group with no significant difference. Regarding oral health status, DMFS, PII, GI, Call and PPD index were higher for study group compared with control group although the differences were not significant except for Call which was highly significant ($P < 0.01$). In general, not significant correlation were found between salivary variables and the other oral parameters for the two groups.

Conclusions: Gasoline workers stations were associated with decrease salivary IgA and increase salivary antioxidant (except zinc) this may explain the increase in caries experience and periodontal diseases among those workers.

KEYWORDS: Gasoline workers, Oral health, IgA, Antioxidant....

INTRODUCTION

Millions of workers in a variety of occupations were found to be exposed to hazardous substances which can be present in the form of gases, vapors, mist, fumes or particles, so diseases in some of these workers can be attributed to the exposure to these substances.^(1,2) Gasoline is a refined product of petroleum consisting of a complex mixture of chemicals including benzene which is well-known in its carcinogenicity to human.⁽²⁾ Benzene and its metabolites seem to be genotoxic to humans, causing primarily chromosomal aberrations.⁽³⁾ Routes of exposure can take place through inhalation, dermal absorption or ingestion.⁽¹⁾

Salivary IgA is the first line of defense against microbial invasion.⁽⁴⁾ It is present in high concentration in saliva which represent the summation of IgA of salivary glands in addition to serum IgA from gingival fluid.⁽⁵⁾ Similarly antioxidant system is one of the important defense mechanism in saliva. An antioxidant is a molecule capable of inhibiting the oxidation reactions which can produce free radicals. In turn, these radicals can induce damage to the cells.⁽⁶⁾ Antioxidant system includes enzymatic and non-enzymatic antioxidants, uric acid is one of the non-enzymatic antioxidant in human saliva.⁽⁷⁾ It is a relatively powerful scavenging antioxidant of water soluble free radicals.⁽⁸⁾ On the other hand enzymatic antioxidants require a variety of trace elements for their activation like zinc and magnesium.⁽⁹⁾ In addition, it has been found that magnesium

is involved in the antioxidant defense mechanisms of the body⁽¹⁰⁾ and its deficiency was found to induce elevation in the formation of oxygen radicals.⁽¹¹⁾

Extensive studies are needed to evaluate biological damages at different levels and to decrease the risk for serious diseases among workers in gasoline stations. Since no previous Iraqi study concerning this subject, this study was carried out to investigate the oral health condition in relation to salivary IgA and antioxidants among gasoline workers stations.

SUBJECTS, MATERIALS & METHODS

Forty four males aged (32-38) were enrolled in this investigation, study group composed of 23 workers in gasoline stations in Baghdad city for at least five years and control group included 21 males matching the study group by age. All individuals should be non-smokers, with no chronic medical illness and shouldn't take any medications to exclude any factor that may affect the parameters examined in this study. Stimulated salivary samples were collected following assessment of dental plaque under standard condition according to the instructions cited by Tenovuo and Lagerlof.⁽¹²⁾ Salivary samples were taken to the laboratory for biochemical analysis. Immunoglobulin A in saliva was determined by radial immunodiffusion technique of Mancini *et al.*⁽¹³⁾ Salivary antioxidant were determined by using ready kit

(BioMerieux sa, France) for uric acid while salivary magnesium and zinc ions were measured by using atomic absorption spectrophotometer flame (Model Buck 210 VGP USA).

Caries experience was assessed by the application of decayed, missing and filled surface index⁽¹⁴⁾ (D1-2 MFS). Plaque index⁽¹⁵⁾ (PII) and calculus index⁽¹⁶⁾ (Call) were used for recording oral cleanliness. Periodontal status evaluated by using gingival index⁽¹⁷⁾ (GI) and periodontal pocket depth⁽¹⁸⁾ (PPD). Data analysis was conducted through the application of the SPSS (version 16). Student's-test and Pearson's correlation coefficient was applied. The confidence limit was accepted at 95% ($P < 0.05$).

RESULTS

Table 1 revealed that the salivary IgA was lower among study group compared with control group with highly significant difference ($P < 0.01$). On the other hand uric acid showed higher value among study group with highly significant difference ($P < 0.01$). Although salivary magnesium was higher among study group but the difference was not significant ($P > 0.05$) also no significant difference was found regarding salivary zinc ($P > 0.05$) in spite it was higher among control group. Table 2 illustrates that DMFS, D, M and F values were higher among the study group but the differences were statistically not significant ($P > 0.05$). Results also showed that PII, GI, Call and PPD were higher among the study group with no significant differences ($P > 0.05$) except for Call which was highly significant difference ($P < 0.01$) (Table 3).

As shown in Table 4 no significant correlations were recorded between caries experience with salivary variables in various directions for the two groups. Similarly no significant correlations were found between salivary variables and severity of dental caries in various directions except for IgA and D2 among the study group which was highly significant correlation (Table 5). Regarding oral cleanliness and periodontal health, Table 6 demonstrates no significant correlations between PII, GI, CAL and PPD with salivary variables in various directions for the study and control group.

DISCUSSION

Exposure to toxic agents can result from natural and environmental factors, occupational environment or industrial accidents.⁽¹⁹⁾ Gasoline station workers have increasing the risk for developing serious diseases due to the possible long-term effects of the petroleum derivatives on their health.⁽²⁰⁾

Saliva is a unique fluid, as it is the first biological medium confronted by external materials that are taken into the body as parts of food, drink or inhaled volatile ingredients⁽²¹⁾ also saliva used to study the influence of environmental factors on immunological parameters.⁽²²⁾ In the present study salivary IgA was lower among study group than the control group with highly significant difference. This could be attributed to that salivary IgA is often decreased after chemical exposure.^(22,23) This finding may explain the low defense of a given individual against external intruders.⁽²⁴⁾ In addition decreased concentration of secretory IgA is a potential indicator of increased disease risk.⁽²⁵⁾

Concerning antioxidant, uric acid was significantly higher among study group than control group also magnesium level was higher among the study group though the difference was not significant. This may be related to the fact that the body raises the level of its antioxidant system to combat the oxidative damage,⁽²⁶⁾ as the functions of antioxidant are to prevent the generation of free radicals and to inactivate them after generation.⁽²⁷⁾ Level of salivary zinc ion was lower among the study group but statistically the difference was not significant. Zinc regarded as one of the main healing minerals, it is essential for the activity of over 300 enzymes in the body. In addition it forms part of the enzyme (carbonic anhydrase) and functions as antioxidant.⁽²⁸⁾ Previous studies have shown that constituent of the gasoline fumes could be carcinogenic^(2,29) by producing free radicals which reacts with trace elements and in turn affects the total antioxidant status of the individual. In addition it was found that decrease in plasma levels of zinc probably as a result of interference in their metabolic pathway of the exposed groups,⁽³⁰⁾ this may explain the low level of zinc among the study group.

In regard to dental caries, the current study demonstrated a higher caries experience for the study group than the control one although the differences were not significant. This may be due to the lower level of salivary IgA among study group. On the other hand no significant correlation were recorded between salivary IgA and caries experience except with DS which was significant negative correlation among the study group only, this may explain the higher caries experience among them as salivary IgA consider as the first line of defense against microbial invasion⁽³¹⁾ and increased its level in saliva can enhance the elimination of *Streptococcus mutans* from oral cavity and interfere with its cariogenicity,⁽³²⁾ this also may be the cause of highly significant correlation between IgA and D2 among the study group. In addition no significant correlations were recorded between an-

tioxidants (uric acid, magnesium and zinc) and caries experience in both groups, the negative correlations between magnesium with DMFS and DS may be attributed to that magnesium level was inversely associated with caries experience.⁽³³⁾

The results also showed higher PII, GI and PPD for study than control group in spite that the differences were not significant. This may be attributed to the lower level of salivary IgA among the study group also no significant correlation were recorded between salivary IgA and PII, GI, Cal and PPD in both groups. Salivary IgA has antibacterial properties that can protect against oral infections⁽³⁴⁾ and this may explain the negative correlation between IgA and gingival index. Furthermore, It was documented that different salivary antioxidant provide protection against radicals-induced damage of periodontal tissues.⁽³⁵⁾ This may explain the non significant differences for

PII, GI, and PPD between study and control group although they were higher among the study group. While other studies found that the level of salivary uric acid⁽³⁶⁾ and magnesium⁽³⁷⁾ were decrease in patients with severe periodontitis. On the other hand, calculus accumulation was higher among study group with highly significant difference, this may give another explanation for the higher plaque, gingival and periodontal pocket depth indices among gasoline stations workers because dental calculus plays a role in periodontal disease pathogenesis since it is a mineralized dental plaque and it acts as a retentive factor for dental plaque.⁽³⁸⁾

Results of this study showed that gasoline workers stations belong to a risk group for public agencies concerned with environmental quality and public health to applied educational and preventive programs concerning those populations.

(Table 1) Salivary variables among study and control group

Variable (mg/dl)	Study group	Control group	Statistics	
	Mean ±SD	Mean ±SD	t-value	P
IgA	37.01 1.05	39.09 2.30	3.79	0.00**
Uric acid	2.17 0.90	1.03 0.58	4.91	0.02*
Magnesium	1.45 0.80	0.55 0.28	4.86	0.12
Zinc	11.56 4.21	15.17 3.55	-3.05	0.31

* Significant($P < 0.05$) ** Highly significant($P < 0.01$) *d.f.* = 42

(Table 2) Caries -experience among study and control group

Caries experience	Study group	Control group	Statistics	
	Mean ±SD	Mean ±SD	t-value	P
D	10.21 5.12	5.14 4.46	3.48	0.95
M	15.00 13.56	8.28 7.43	2.00	0.06
F	6.26 10.77	6.23 7.68	0.00	0.24
DMFS	31.47 18.37	20.38 13.01	2.29	0.11

d.f. = 42

(Table 3) PII, GI, CalI and PPD among study and control group

Variable (mg/dl)	Study group	Control group	Statistics	
	Mean ±SD	Mean ±SD	t-value	P
PII	1.36 0.50	0.86 0.44	3.40	0.32
GI	0.95 0.51	0.64 0.36	2.31	0.11
CalI	0.53 0.51	0.14 0.16	3.23	0.00**
PPD	1.86 0.71	1.49 0.33	2.15	0.10

** Highly significant($P < 0.01$) *d.f.* = 42

(Table 4) Correlation coefficient between salivary variables and caries experience

Variable	Study group				Control group			
	DMFS		DS		DMFS		DS	
	r	P	r	P	r	P	r	P
IgA	-0.10	0.64	-0.41	0.04*	0.23	0.31	-0.14	0.53
Uric acid	0.05	0.82	0.24	0.28	0.19	0.64	0.17	0.47
Magnesium	-0.25	0.26	-0.08	0.71	-0.14	0.54	0.00	1.00
Zinc	-0.22	0.31	0.16	0.60	0.13	0.56	0.01	0.97

* Significant(P<0.05)

(Table 5) Correlation coefficients between salivary variables and severity of dental caries

Groups	Dental caries	IgA		Uric acid		Magnesium		Zinc	
		r	P	r	P	r	P	r	P
Study group	D1	0.07	0.73	0.04	0.86	-0.11	0.61	0.20	0.36
	D2	-0.51	0.01**	0.31	0.15	-0.16	0.46	-0.06	0.78
	D3	-0.33	0.11	0.23	0.29	-0.12	0.58	0.02	0.89
	D4	0.06	0.75	-0.15	0.47	0.23	0.29	0.06	0.78
Control group	D1	-0.19	0.39	0.00	0.98	0.07	0.76	-0.19	0.39
	D2	-0.01	0.94	0.11	0.63	-0.09	0.70	0.02	0.99
	D3	-0.23	0.30	0.01	0.95	0.04	0.86	0.16	0.46
	D4	0.02	0.92	0.47	0.31	-0.00	0.98	0.20	0.38

** Highly significant(P<0.01)

(Table 6) Correlation coefficient between salivary variables and PII, GI, CaII and PPD

Groups	Variable	IgA		Uric acid		Magnesium		Zinc	
		r	P	r	P	r	P	r	P
Study group	PII	0.00	0.97	-0.40	0.6	0.23	0.29	0.17	0.43
	GI	-0.01	0.95	0.06	0.78	0.16	0.46	0.18	0.40
	CaII	-0.03	-0.89	-0.29	0.17	0.07	0.76	0.15	0.49
	PPD	0.07	0.74	0.08	0.72	-0.12	0.58	0.10	0.65
Control group	PII	0.10	0.64	0.17	0.48	-0.11	0.63	-0.02	0.92
	GI	0.17	0.44	-0.25	0.27	-0.29	0.21	-0.23	0.31
	CaII	-0.03	0.88	-0.03	0.90	-0.02	0.91	0.12	0.58
	PPD	0.13	0.55	-0.44	0.40	-0.56	0.80	0.05	0.88

ACKNOWLEDGEMENT

I wish to express my sincere gratefulness to Prof. Sulafa El-Samarrai for her suggestion, cooperation and advice who made this work possible. Also my deep thanks to Dr. Ban S. Diab and Dr. Ammar Fouad for their help in performing this study.

REFERENCES

1. Keshava N and Ong TM. Occupational exposure to genotoxic agents. *Mutat. Res.*1999; 437: 175-194.
2. Carere A, Antocchia A, Crebelli R, Degrossi F. Genetic effects of petroleum fuels: cytogenetic monitoring of gasoline station attendants. *Mutat. Res.* 1995;332:17-26.
3. Majer BJ, Laky B, Knasmüller S and Kassie F. Use of the micronucleus assay with exfoliated epithelial cells as a biomarker for monitoring individuals at elevated risk of genetic damage and in chemoprevention trials. *Mutat. Res.* 2001;489:147-72.

4. McGhee JR, Mesteck J, Dertzbaugh MT, Eldridge JH, Hirasawa M and Kiyono H. The mucosal immune system: from fundamental concepts to vaccine development. *J Vaccine*. 1992;10:75-88.
5. Camiling E, Gahnberg L, Krasse B. The relationship between IgA and antibodies to streptococcus mutans antigens in human saliva and breast milk and the numbers of endogenous oral streptococcus mutans. *Arch Oral Biol*. 1987;32:21-25.
6. Sies H. Oxidative stress: oxidants and antioxidants. *Exp Physiol*. 1997;82(2):291-5.
7. Karatas F, Kara H, Servi S, Tug T, Erulas FA, Koca M. Investigation of antioxidant vitamins (A,E,C) and lipid peroxidation levels in rats injected N-(1,3-benzothiazol-2-yl)-N-(4,5-dihydro-1H-imidazol-2-YI) amine. *Molecules*. 2005;10:922-8.
8. Ames BN, Cathcart R, Schwiers E, Hochstein P. Uric acid provides an antioxidant-and radical-caused ageing and cancer: a hypothesis. *Proc Natl Acad Sci USA*. 1981;78:6858-62.
9. Knight JA. The biochemistry of aging. *Adv Clin Chem*. 2000;35:1-62.
10. Lalle SB, Singh B, Gulatin K, Seth SD. Role of nutrition in toxic injury. *Ind J Exp Biol*. 1999;37(2):109-16.
11. Chakraborti S, Chakraborti T, Mandal A, Sudipda T, Ghosh. Protective role of magnesium in cardiovascular disease. *Mol Cell Bio*. 2002;1-2:163-78.
12. Tenovuo J and Lagerlof F. Saliva In: Thylstrup A and Fejerskov O eds. *Textbook of clinical cariology*. 2nd ed. Munksgaard. Copenhagen. 1996.
13. Mancini G, Carbonara AO, Heremans JF: Immunochemical quantitation of antigens by single radial immunodiffusion. *J Immunochemistry*. 1965;2:235-39.
14. Manjie F, Fejerskov O, Baelum V. Pattern of dental caries in an adult rural population. *Caries Res*. 1989;23:55-62.
15. Silness J and Loe H. Periodontal disease in pregnancy II. *Acta Odontol Scand*. 1964;24:747-59.
16. Ramfjord SP. Indices for prevalence and incidence of periodontal disease. *J Periodontol*. 1959;30:51-9.
17. Loe H and Silness J. Periodontal disease in pregnancy I. *Acta Odontol Scand*. 1963;21:533-51.
18. Carranza FA. Classification of disease of the periodontium. In Carranza F and Newman M ed. *Clinical periodontology*. 8th ed. WB Saunders. USA. 1996.
19. Anderson D. Factors contributing to biomarker responses in exposed workers. *Mutat. Res*. 1999;428:197-202.
20. Benites CI, Amado L, Vianna P and Martino-Roth G. Micronucleus test on gas station attendants. *Genet. Mol. Res*. 2006;5(1):45-54.
21. Klein I, Nagler RM, Toffler R, VanDer V, Reznick A. Effect of cigarette smoke on oral peroxidase activity in human saliva: role of hydrogen cyanide. *Free Radical Bio Med* 2003;35:1448-52.
22. Ewers U, Stiller-Winkler R and Idel H. Serum immunoglobulin, complement C3, and salivary IgA levels in lead workers. *Environmental Research*. 1982;29(2):351-7.
23. Lange A, Smolike R, Zotoski W. Serum immunoglobulin levels in workers exposed to benzene, toluene and xylene. *Int Arch Arbeits Med*. 1973;31:37-44.
24. Pranjić N, Mujagić H, Pavlović S. Inhalation of gasoline and damage to health in workers at gas stations. *Medical archives*. 2003;57(1):17-20.
25. Pacque P, Booth C and Dwyer D. Salivary Immunoglobulin A (sIgA) as a Biomarker of Immune Suppression Following the Combat Fitness Assessment. DSTO, Australia 2002.
26. Dean V, Scully-Simon C. Salivary antioxidants and periodontal disease status. *Proceeding of the nutrition society*. 2002;61:137-43.
27. Yorbik O, Sayal A, Akay C, Akbiyik DI and Sohmen T. Investigation of antioxidant enzymes in children with autistic disorder. *Prostaglandins Leukot Essent. Fatty Acids*. 2002;67:341-3.
28. Prasad A S. Zinc: an overview. *Nutrition*. 1995;11(1):93-9.
29. Halliwell B, Gutteridge JC. The definition and measurement of antioxidants in biological systems. *Free Radic Biol Med*. 1995;18:125-6.
30. Okuonghae PO, Aberare LO, Mukoro N, Osazuwa F, Dirisu JO, Ogbuzulu J, Omoregie R, Igbinuwen M. Total antioxidant status of zinc, manganese, copper and selenium levels in rats exposed to premium motor spirit fumes. *North Am J Med Sci*. 2011;3:4-7.
31. Proctor GB and Carpenter GH. Chewing stimulates secretion of human salivary secretory immunoglobulin A. *J Dent Res*. 2001;80:909-13.
32. Marcotte H and Lavoie M. Oral microbial ecology and the role of salivary immunoglobulin A. *J Microbiol Mol Biol Rev*. 1998;62(1):71-109.
33. Suddick R, Hyde R and Feller R. Salivary water and electrolytes and oral health. In Menaker L. *Biological basis of dental caries*. 1st ed. Harper and Row Publishers Inc. New York 1980.
34. Lingstrum P, Moynihan P. Nutrition, saliva and oral health. *Nutrition*. 2003;19:567-9.
35. Enwonwu CO. Interface of malnutrition and periodontal diseases. *Am J Clin Nutr*. 1995;61(1):430-6.
36. Sculley DV and Langley-Evans SC. Periodontal disease is associated with lower antioxidant capacity in whole saliva and evidence of increased protein oxidation. *Clin Sci*. 2003;105:167-72.
37. Zaichk V and Bagirov S. The chemical element content of mixed saliva in periodontal disease. *Stomatologilia Mosk*. 1994;73:8-11.
38. Haake S K. Etiology of periodontal disease. In Newman M, Taki H and Carranza F. *Carranza's clinical periodontology*. 9th ed. Saunderson Elsevier 2002.