Section: Biochemistry

Research

IMPACT OF PASSIVE SMOKING ON OXIDATIVE STRESS AND ANTIOXIDANT LEVELS: A COMPARATIVE STUDY

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Received : *10/12/2022*
Received in revised form : *05/01/2023*
Accepted : *19/01/2023*

Keywords:
Passive smoking, Second hand smoke, Oxidative stress, antioxidants, Smoke-free policy.

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DOI: 10.47009/jamp.2023.5.1.207

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (1); 995-1000

Abstract

Background: Passive smokers are equally exposed to the harmful effects of tobacco when they inhale the components of cigarette smoke that active smokers blow out into the environment. The objective of this study was to evaluate oxidative stress and antioxidant status in individuals who are exposed to passive smoking compared to those who are not exposed. **Materials and Methods:** Study was done on 50 individuals of both sexes exposed to passive smoking between 18-60 years and 50 individuals not exposed to passive smoking. 5ml of fasting venous sample was collected and the plasma separated was analysed for Malondialdehyde (MDA) by Thiobarbituric acid method, Glutathione (GSH) by Ellman's method, Vitamin C by method described by Roe and Kuether and Fasting plasma Glucose (FPG) was estimated by Glucose oxidase peroxidase (GOD–POD) method. **Result:** Two tailed t- test for significance was done to assess the significance in age and the parameters among the two groups. There were significant difference between the two groups with respect to Vitamin C (p value <0.0001), GSH (p value <0.0001) and MDA levels (p value <0.0001), but no significant difference in age (p value 0.910) and FBG levels (p value 0.595). Vitamin C and GSH had a negative correlation (p<0.0001) whereas MDA showed a positive correlation (p<0.0001) with the the duration of passive smoke exposure in years. (HOPS). **Conclusion:** The results consolidated the fact that there is an increased oxidative stress and decreased plasma antioxidant levels when exposed to passive smoking. Overwhelming evidence on the harmful effects of passive smoking must prompt change in attitude of smokers to quit smoking as more often than not the people who are exposed to second-hand smoke are their family members and friends.

INTRODUCTION

Oxidative stress is characterized by a situation where the production of reactive oxygen species (ROS) surpasses our capacity to defend against them, leading to an elevation in oxidative damage to the biomolecules.[1]

Environmental tobacco smoke (second-hand tobacco smoke - SHS) is an extended, diluted blend of mainstream smoke, which includes the smoke exhaled by smokers, and side-stream smoke, which consists of smoke released from the burning tip of a cigarette. The act of inhaling environmental tobacco smoke is commonly referred to as 'passive smoking' or 'involuntary smoking.' Laboratory-based research has identified over 4,000 compounds in mainstream smoke, with many of them being recognized or suspected human carcinogens. (NRC, 1986).[2]

The components of cigarette smoke blown out by active smokers into the environment are inhaled by passive smokers, thus exposing them equally to the harmful effects of tobacco

Passive smokers inhale the components of cigarette smoke exhaled by active smokers, subjecting them to the same detrimental consequences of tobacco exposure.[3]

The 2004 SCOTH report suggests that non-smokers exposed to SHS face a 25% higher risk of developing heart disease.[4]

The International Agency for Research on Cancer (IARC) and the World Health Organization (WHO) have categorized SHS as a confirmed (class A) human carcinogen.[5]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

India holds the position of being the world's second-largest consumer and producer of tobacco.[6,7]

The Government of India enacted 'Cigarettes and Other Tobacco Products (Prohibition of Advertisement and Regulation of Trade and Commerce, Production, Supply and Distribution) Act, 2003 (COTPA)', to enhance public health, there is a need to ban the use of all tobacco products, including cigarettes, in public spaces, as well as to regulate the advertising and oversee the trade, commerce, production, supply, and distribution of cigarettes and other tobacco items within the country.[8]

In October 2008, smoke free rules in COPTA were enhanced, redefining 'public places' to include all workplaces also.[9]

Hence this work was done to evaluate oxidative stress and antioxidant status in person's exposed to passive smoking compared to individual not exposed to passive smoking. Fasting blood glucose levels was also assessed.

Aims and Objectives

1. To measure Plasma Malondialdehyde, Glutathione, Vitamin C and Fasting plasma Glucose in passive smokers and individuals with no exposure to passive smoking.
2. To compare the levels of Plasma Malondialdehyde, Glutathione, Vitamin C and Fasting plasma Glucose in passive smokers and individuals with no exposure to passive smoking.

MATERIALS AND METHODS

This study was conducted in the Department of Biochemistry, Saveetha Medical College and Hospital outpatient department. Study was done on 50 individuals of both sexes exposed to passive smoking between 18-60 years and 50 individuals not exposed to passive smoking. Total of 100 samples were collected from patients coming for master health check-up.

Inclusion Criteria

Group exposed to passive smoking

1. 18-60 years age group.
2. Both genders.
3. Exposure to passive smoking at home/workplace for a minimum of 10 years.

Group not exposed to passive smoking

1. 18-60 years age group.
2. Both genders.
3. No exposure to passive smoking

Exclusion Criteria

1. Age group less than 18 years and more than 60 years.
2. Any comorbid illness like diabetes, hypertension, etc.
3. Person suffering from acute infections.
4. Pregnant women
5. Past and present history of smoking, alcohol intake and paan usage.
6. Person's with history of COPD, allergic bronchitis, Koch's.

After taking written and informed consent, detailed history regarding exposure to environmental tobacco smoking, duration of exposure, any current medical illness and family history was taken.

Under aseptic precautions, venous blood samples were collected from antecubital vein 5 ml in overnight fasting state.

The fasting sample was transferred into heparinized vacutainers. The heparinized tube samples were centrifuged immediately and the plasma was separated. The plasma was used immediately for the estimation of the following parameters:

Plasma Malondialdehyde was measured by Thiobarbituric acid reactive substances (TBARS) method.[10]

Plasma Reduced Glutathione (GSH) was measured by Ellman's method.[11]

Plasma Vitamin C was measured using method described by Roe, J. H., and Kuether, C. A.[12]

Fasting Plasma Glucose by Glucose Oxidase Peroxidase method.[13]

Statistical Analysis

The data collected was entered into a MS-EXCEL sheet and the data was expressed in mean, Standard deviation (SD) and standard error of mean. The two groups were compared using two tailed t-test for significance. A p value of < 0.05 was regarded as significant.

RESULTS

Table 1 depicts the mean, standard deviation and standard error of the ages. The parameters estimated between the group exposed to passive smoking and group not exposed to passive smoking was calculated using group statistics.

Two tailed t-test for significance was done to assess the significance in age and the parameters among the two groups. There were significant difference between the two groups with respect to Vitamin C (p value <0.0001), GSH (p value <0.0001) and MDA levels (p value <0.0001), but no significant difference in age (p value < 0.910) and FPG levels. [Table 2]

Table 3 shows a linear correlation regression analysis which was performed with the parameters measured against number of years of exposure to passive smoking (HOPS) in the group exposed to passive smoking and age in case of group not exposed to passive smoking. There was a significant correlation between Vitamin C, GSH and MDA with the HOPS. Vitamin C and GSH had a negative correlation (p<0.0001) whereas MDA had a positive correlation (p<0.0001) with HOPS. There was no significant association between FBS and HOPS. [Table 2]

There was no significant age or sex difference between both the passive smoking group and control group. There was increased plasma malondialdehyde levels (p<0.0001) in passive smoking group when compared to the group not exposed to passive

ISSN (O): 2687-5365; ISSN (P): 2753-6556

smoking. The vitamin C and reduced glutathione levels were significantly lower (p value <0.0001) in passive smoking group when compared to control group. [Table 3]

Table 1: Group Statistics

	Group	N	Mean	Std. Deviation	Std. Error Mean
Age	Not exposed to PS	50	32.5400	9.64938	1.36463
	Exposed to PS	50	32.7400	7.88646	1.11531
Vit C	Not exposed to PS	50	1.1640	.31736	.04488
	Exposed to PS	50	.3560	.15140	.02141
GSH	Not exposed to PS	50	5.4680	1.13721	.16083
	Exposed to PS	50	1.8546	.48716	.06890
MDA	Not exposed to PS	50	1.8380	.55545	.07855
	Exposed to PS	50	3.7720	.87878	.12428
FBS	Not exposed to PS	50	81.5000	10.19654	1.44201
	Exposed to PS	50	82.5200	8.88071	1.25592

Table 2: Independent Samples Test

		t-test for Equality of Means			
		T	Df	Sig. (2-tailed)	Mean Difference
Age	Equal variances assumed	-0.113	98	0.91	-0.2
	Equal variances not assumed	-0.113	94.265	0.91	-0.2
Vit C	Equal variances assumed	16.249	98	0	0.808
	Equal variances not assumed	16.249	70.205	0	0.808
GSH	Equal variances assumed	20.653	98	0	3.6134
	Equal variances not assumed	20.653	66.398	0	3.6134
MDA	Equal variances assumed	-13.154	98	0	-1.934
	Equal variances not assumed	-13.154	82.763	0	-1.934
FPG	Equal variances assumed	-0.533	98	0.595	-1.02
	Equal variances not assumed	-0.533	96.187	0.595	-1.02

Table 3: HOPS versus measured parameters in group exposed to passive smoking

Parameter	R	R^2	P
HOPS- Vitamin C	-0.730	0.533	0.0001
HOPS- GSH	-0.638	0.407	0.0001
HOPS- MDA	+0.612	0.375	0.0001
HOPS- FBS	+0.103	0.011	0.477

DISCUSSION

Passive smoking otherwise called as second hand smoking, itself is a health hazard as identified by the WHO14. The spectrum of complications due to passive smoking ranges from cardiovascular diseases, pulmonary diseases, impaired glucose tolerance, increased risk of developing malignancy, etc15. Many epidemiological studies have proved that passive smoking is a strong risk factor for the development of atherosclerosis leading to coronary heart disease and stroke.[16,17,18,19] This is why the Indian government has recognized it as a significant risk factor for non-communicable diseases. To address its prevention, strict regulations have been enacted to prohibit tobacco smoking in public places.[9].

In view of the above, the present study was undertaken to see the effect of passive smoking exposure in Tirupati region. Oxidative stress was measured by plasma malondialdehyde and antioxidant status was determined by estimating plasma reduced glutathione and vitamin C levels and glucose tolerance was assessed by measuring fasting plasma blood glucose levels in individuals exposed to passive smoking versus non-smoking control group.

Side-stream smoke contains substantially higher quantities of cytotoxic substances, heavy metals, poisonous gases, and radioactive elements compared to those present in mainstream cigarette smoke.[20,21] The cytotoxic substances are polycyclic aromatic hydrocarbons, nicotine and nitrosamines. Heavy metals like nickel, chromium, cadmium, etc. Poisonous gases like CO and N2O.[22,23]

Currently there are measures of the dose absorbed of environmental tobacco smoke in a population using hair nicotinine.[24] Exposure to environmental tobacco smoke can be assessed by questionnaires, air monitoring, modelling of concentrations, or biological markers.[25,26,27,28] Indirect Reporting of second-hand tobacco smoke exposure by answering a questionnaire was used in the current study to assess the exposure to passive smoking.[29] In a comprehensive collaborative study spanning multiple countries, the most effective measure of exposure to husbands' smoking was the number of cigarettes smoked per day, while workplace exposure was more significantly associated with the duration of passive smoking exposure.[30]

In 2002, the International Agency for Research on Cancer (IARC) conducted a comprehensive review of the evidence regarding second-hand smoking and its association with cancer. They concluded that exposure to the smoke of others increases the risk of

ISSN (O): 2687-5365; ISSN (P): 2753-6556

lung cancer by 20-30% and coronary heart disease by 25-35% in non-smokers.[31]

Oxidative stress may be described as a condition in which oxidation surpasses the body's antioxidant systems due to an imbalance between them. This not only leads to harmful occurrences like lipid peroxidation and oxidative DNA damage but also triggers physiological adaptation processes and the regulation of intracellular signal transduction.[32]

Reactive oxygen species can be generated through processes such as covalent bond cleavage, electron addition to a molecule, or the removal of hydrogen by other radicals. These species are typically highly reactive and often function as electrophilic agents or oxidizing agents. Reactive oxygen species have the potential to induce oxidative stress in our bodies, either directly or indirectly.[33]

The most damaging effect of these Reactive oxygen species is, they trigger the initiation of lipid peroxidation. The cell membrane comprised of polyunsaturated fatty acids being a primary focal point for the attack by reactive oxygen species, resulting in damage to the cell membrane.[34]

According to the report from the Scientific Committee on Tobacco and Health, individuals who are not smokers but are exposed to second-hand smoke face a 25% higher risk of developing heart disease.[35]

The endogenous antioxidant defence in our body comprises three distinct systems: antioxidant enzymes such as catalase and superoxide dismutase (SOD), metal-binding proteins like ferritin, and small molecules with low molecular weight, such as vitamin C and glutathione.[36]

Vitamin C acts as a powerful reducing agent in many oxidation-reduction reactions by transferring of protons. It has an important role in wound healing, nutrient metabolism (iron, protein and fat), immune function and during the biosynthesis of some neurotransmitters. Vitamin C is one of the strongest determinant of plasma total antioxidant defence.[37]

Not only does ascorbic acid possess anti-oxidative properties on its own, but it also contributes to the anti-oxidative status of a cell by reducing both, vitamin E, a major membrane-associated antioxidant ,and/or glutathione, the major cellular antioxidant.[38,39]

Jacob et al. proposed that glutathione and vitamin C collaborate synergistically in neutralizing free radicals and that they mutually conserve each other.[40]

Even minimal exposure to environmental tobacco smoke in children (<3.2 µmol/L on average) can lead to decreased levels of ascorbate, a significant blood antioxidant.[41] 2011 study found a significant reduction in vitamin C levels ($p<0.05$) among passive smokers when compared to the control group, which aligns with the findings of the present study.[42]

In a study conducted in Berkeley and Oakland, California, plasma malondialdehyde and levels of ascorbate were measured and compared with respect to active, passive and non- smoking population, and analysed by multivariable model. In their study, plasma ascorbic acid had a strong inverse relation ($p < 0.0014$) with the level of malondialdehyde in passive smokers. The plasma malondialdehyde levels were significantly higher in the active and passive smoking group when compared to the non-smoking group ($p<0.0001$).[43]

In a study done in 2011, the levels of MDA and glutathione peroxidase levels were higher in smokers. MDA levels were lower in active smokers than passive smokers. The levels of vitamin C were similar in all groups.[44]

In a study done in 2005, Plasma thiobarbituric acid reactive substances (nmol/ml) in non-smokers was 2.092±0.184 and 8.975±1.49 in passive smokers ($p<0.005$), plasma Vitamin-C (mg/dl) in non-smokers- 0.728+0.043 and passive smokers - 0.632+0.064 ($p<0.005$) and erythrocyte GSH-Px (U/g Hb) in non-smokers 60±7.21 and 55.8±6.18 in passive smokers ($p<0.005$) which is in concordance with the current study.[45]

Cigarette smoke exposure in school children showed reduced glutathione (mg/dl) was 125 ±6 in children not exposed to passive smoking and 116 ± 5 in children exposed to passive smoking where the p value was not significant46. In the current study, there was decrease in, reduced glutathione levels in passive smoking group when compared to the control group.

CONCLUSION

The results consolidated the fact that there occurs increased oxidative stress and the plasma antioxidant levels decrease when exposed to passive smoking. Few studies report impaired glucose tolerance in individuals exposed to passive smoking.[47,48] However in the current study, no significant difference in fasting blood glucose level was observed between the passive smoking group and the group not exposed to passive smoking. This may have been due to confounding factors like diet, lifestyle changes and genetic predisposition to develop glucose intolerance.

Overwhelming evidence on the harmful effects of passive smoking must prompt smokers to quit smoking as more often than not the people who are exposed to second-hand smoke are their family members and friends.

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ISSN (O): 2687-5365; ISSN (P): 2753-6556