Effective Dose of Vitamin C Against Passive Smoking

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ABSTRACT

Objective: Ascorbic acid (AA), known as vitamin C, plays an important role in the human body. Therefore, this study aimed to estimate the beneficial effects of two levels of (AA) that can protect or cure the lung against the hazards of passive smoking in male mice.

Materials and Methods: The study was done on forty male albino mice (6 weeks age and average body weight 22 ± 3 g). The mice were assigned into eight equal groups; control group, group 2was exposed to cigarette smoke for 12 weeks, and the other six groups were given low or high doses of vitamin C(0.015and 0.075 mg/g BW)before, during and after exposure to smoke. Doses were given orally via oro-gastric tube.

Results: High doses of vitamin C had a protective effect when taken before and during exposure to smoke. It reduced the negative effect of smoke as indicated by the levels of TBARS and GSH in plasma and lung tissue, and the improvement of studied histological parameters.

Conclusion: Ascorbic acid had no therapeutic effects after lung tissue damage due to exposure to secondhand smoke. Therefore, it is important to protect the lung from the negative effects of smoking by increasing the daily intake of vitamin C doses to 1 gram, from food rich in vitamin C like fresh fruits and vegetables.

Keywords: Vitamin C, Passive smoking, Lung, Biochemical, Histological examinations

INTRODUCTION

Cigarettes carry serious health risks, which are more prevalent than other tobacco products. About half of cigarette smokers die from tobacco-related disease and lose on average 14 years of life *(Doll et al.,2004)* **.** Passive smoking causes many of the same diseases as direct smoking including respiratory diseases and lung cancer. Cigarettes produce an aerosol containing over 4,000 chemical compounds, including nicotine, carbon monoxide, acrolein, and other harmful substances, over 50 of these are carcinogenic *(Seget et al.,2012).*

Passive smoking is a mixture of smoke from the burning end of a cigarette, and the smoke exhaled from the lungs of smokers. It is involuntarily inhaled, lingers in the air hours after cigarettes have been extinguished, and can cause a wide range of adverse health effects, including respiratory infections, asthma and cancer *(ALA, 2010).*

Nonsmokers who are exposed to passive smoking at home or work increase their heart disease risk by 25–30% and their lung cancer risk by 20–30% *(CDCP, 2013).* Sudden infant death syndrome, ear infections, respiratory infections, and asthma attacks can occur in children who are exposed to passive smoking. Scientific evidence shows no level of exposure to second-hand smoke is safe *(CDCP, 2012).*

Dietary antioxidants are an important factor in protecting against the damaging effects of oxidative stress in the airways, a characteristic of respiratory diseases (Wood et al., 2005)**.** Oxidative stress caused by reactive oxygen species (ROS), is generated in the lungs due to various exposures, such as air pollution (Kelly, 2005) **.**Antioxidants vitamins including vitamin C, vitamin E, flavonoids and carotenoids are abundantly present in fruits and vegetables, as well as nuts, vegetable oils, cocoa and green tea.

Some authors dispute the hypothesis that antioxidant vitamins could prevent chronic diseases (NCI, 2007), while others maintain such a possibility is unproved and misguided from the beginning (Huy et al., 2008)**.**

Although certain levels of antioxidant vitamins in the diet are required for good health, there is considerable doubt as to whether antioxidant-rich foods or supplements have anti-disease activity; and if they are actually beneficial, it is unknown which antioxidant(s) are needed from the diet and in what amounts beyond typical dietary intake (Woodside et al., 2005)**.**

It was found that vitamin C reduces the levels of histamine in the blood that increases asthma. Vitamin C in the reduced form, in the lower respiratory tract and airway surface fluids, and has an important role in protecting the lung due to its anti-free radical properties (Yang et al., 1999) (Hemila, 2004) ,and may help to prevent chronic obstructive pulmonary disease (Berthon &Wood, 2015).

Previous studies differed in identifying the effective dose of vitamin C on the respiratory system, and the results are conflicted on the effect of vitamin C as a protector for the respiratory tract especially the lung.

Therefore, the aim of this study was to determine the effective dose of vitamin C on the lung against cigarette

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smoke exposure, and to evaluate its preventive and/or cure effect using some biochemical parameters and histological tests.

MATERIALS AND METHODS

Materials: Ascorbic acid was purchased from El-Gomhoreya Co., Egypt. All reagents used in the study were purchased from Bio-diagnostic Co., Egypt and Sigma Chemical Co., Germany.

Two doses of vitamin C were used and defined as high dose (0.075 mg/g body weight) and low dose (0.015mg/g body weight). Concerning the animal body weight was about 22±3 g.

Experimental animals: Forty male Albino mice aged 6 weeks and weighed (22 ± 3 g) at the beginning of the experiment. They were purchased from the Animal House, Department of Home Economics, Faculty of Agriculture, Alexandria University. The animals received a balanced diet and water *ad libitum* throughout the study. They were maintained under standard housing conditions and housed 5 per cage, and kept for two weeks for acclimatization period before start of the experiment (Childs et al., 2002). The experimental procedure followed the rules of research ethics approved by the Research Ethics Committee, Faculty of Agriculture, Alexandria University. Biochemical analysis were carried out in the Central Laboratory, Institute of Graduate studied and Research, Alexandria University. Histological samples were prepared and examined at Histology Laboratory, Faculty of Medicine, Alexandria University.

The basal diet contents are shown in table (1) and table (2).

Table 1. % Content of basal diet

Diet Ingredient	%
Dextrin	43.65
Casein- Vitamin Free	21
Sucrose	15
RP Mineral Mix #10 (adds 1.29% fiber)	5
Corn Oil	10
Powdered Cellulose	3
RP Vitamin Mix (adds 1.94% sucrose)	2
Choline Chloride	0.2
DL-Methionine	0.15

Protein 18.3%, Fat 22.1%, Carbohydrates 59.6%

Table 2. Minerals and Vitamins mixture composition

Minerals		Vitamins	
Calcium %	0.61	Vitamin A , IU/g	22.1
Phosphorus %	0.57	Vitamin D , IU/g	2.2
Potassium %	0.40	Vitamin E , IU/kg	50.1
Magnesium %	0.07	Vitamin K , ppm	10.4
Sodium%	0.24	Thiamin, ppm	20.7
Chloride%	0.30	Riboflavin, ppm	20.7
Fluorine , ppm	5	Niacin, ppm	90
Iron, ppm	65	Pantothenic acid, ppm	56
Zinc , ppm	27	Folic acid, ppm	4.2
Manganese, ppm	56	Pyridoxine, ppm	16.5
Copper , ppm	15	Biotin, ppm	0.4
Cobalt , ppm	3.2	Vitamin B_{12},mcg/kg	24
Iodine, ppm	0.57	Choline chloride, ppm	1.40
Chromium , ppm	3	Ascorbic Acid, ppm	0.0
Molybdenum, ppm	0.82		
Selenium, ppm	0.30		

Inhalation system was based on the study of (Candon *et al.,1995)* for using two connected glass boxes, one used as a burning box and the other as an exposure box. The animals were exposed one time a day (2 cigarette per exposure period, 5 days a week). Vitamin C solution was freshly prepared and given orally via oro-gastric tube.

Lung damage were assessed using some indicators such as the lung/body relative weight, oxidative stress indices in blood and lung tissues, as well as investigation of lung histology.

Experimental design: Eight groups were studied for 14 weeks, each had 5 mice (Table 3):

(Group1 –Negative control (CONT): served as control kept free from smoke exposure.

(Group 2- Positive control (SMOK): exposed to smoke for 12 weeks without given vitamin C.

(Group 3-HCBS): given high doses of vitamin C for two weeks, then exposed to smoke until the end of the experiment.

(**Group 4–LCBS):** Mice were treated as group 3 but the dose of vitamin C was low.

(Group 5-HCDS): Mice were given high doses of vitamin C during exposure to smoke. **(Group 6-LCDS):** Mice were treated as group 5 but the dose of vitamin C was low.

(Group 7-HCAS): Mice were exposed to smoke for 12 weeks, then given high doses of vitamin C only for two weeks after stopping exposure to smoke.

(Group 8-LCAS): Mice were treated as group 7 but the dose of vitamin C was low.

Biochemical tests: Blood samples were collected from abdominal aorta during scarification in tubes containing heparin as anti-coagulant, and then they were placed immediately on ice packs. Plasma was obtained by centrifugation of samples at 4000 RPM (rotation per minute) for 20 minutes, and was separated and stored at -80^{o}C until used for analyses. Stored plasma samples were analyzed for reduced glutathione (GSH) and thiobarbituric acid reactive substances (TBARS) using assay kits from Bio-diagnostic Co. Egypt. Fresh blood samples were immediately sent in ice box to the laboratory for determine its content of vitamin C using commercial kits from Bio-diagnostic Co. Egypt. Lungs were immediately removed, washed, weighted and stored at -80^{o}C until used for analyses. Tissues were minced and homogenized using sucrose solution prepared in concentration (85.57 g sucrose / liter of distilled water), the homogenate was centrifuged at 10,000 xg for 20 min (Youssef, 2012). The resultant supernatant of the tissue was used for measuring (GSH) and (TBARS).

Histological Analysis: The chest was opened and the lungs were washed and weight to calculate their relative weight as mg/g of body weight. The lungs were dissected and fixed in 10% formol saline, and processed to get 6 µm thick paraffin sections. These sections were stained with hematoxylin and eosin stain (H and E) for light microscopic examination (Drury& Wallington, 1980).

Statistical Analysis:

Data were analyzed according to Steel study (Steel& Torrie, 1981). Statistical significance of the difference in values of control and treated animals was calculated by F test with 5% significance level. Then the data were statistically tested by using Dancan's Multiple Range Test (SAS, 1986).

Table 3. The groups' treatments during the experiment period (14 weeks)

Groups	Weeks													
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2	-	-	S	S	S	S	S	S	S	S	S	S	S	S
3	H	H	S	S	S	S	S	S	S	S	S	S	S	S
4	L	L	S	S	S	S	S	S	S	S	S	S	S	S
5	-	-	S+H	S+H	S+H	S+H	S+H	S+H	S+H	S+H	S+H	S+H	S+H	S+H
6	-	-	S+L	S+L	S+L	S+L	S+L	S+L	S+L	S+L	S+L	S+L	S+L	S+L
7	S	S	S	S	S	S	S	S	S	S	S	S	H	H
8	S	S	S	S	S	S	S	S	S	S	S	S	L	L

G= groups S= exposure to Smoke H= high dose L= low dose

At the end of experimental period (14 weeks), all animals were anesthetized by light ether and sacrificed.

RESULTS AND DISCUSSION

Relative Lung / body weight: As shown from Table (4), there were no significant differences in the relative lung/body weight of all studied groups compared to the control except group (SMOK) which exposed to smoke and did not given vitamin C. Exposure to smoke increased the relative lung weight by (205%). Therefore, it can be concluded that treatment with vitamin C prevent the lung from the risk of increasing its relative weight as a sign of illness.

Vitamin C levels in plasma: Table (5) illustrates the effect of exposure to cigarette smoke on levels of plasma vitamin C of the different mice groups. There are significant differences between the different groups except groups 3 and 5. A significant decrease was found in group 2 exposed to smoke (2 SMOK) compared to control by 31.6%.

The vitamin C increased by 453.7% and 242.7%,in the HC and LC groups respectively.

Previous findings suggest that exposure to smoking in groups 3 and 4 has significantly reduced vitamin C levels in plasma compared to its level in groups 3 * and 4 *. Although cessation of smoking in groups 7 and 8, the effects of smoke led to lower levels of vitamin C compared to groups 3 * and 4 *.

Oxidative stress: In the current study, assessment of oxidant status of plasma and homogenates lung tissue revealed significant elevation by of TBARS in group 2 (SMOK) compared to control group. However, administration of vitamin C with low and high doses decreased the level of TBARS compared to the smoke exposure group but the high dose was more effective, which mean that the administration of vitamin C before and during exposure to smoke can protect against oxidative stress damage (Table 6).

Table 4. Effect of vitamin C and exposure to smoke on relative lung / body weight

Groups	CONT 1	SMOK 2	HCBS 3	LCBS 4	HCDS 5	LCDS 6	HCAS 7	LCAS 8
Mean	0.7753^{a}	2.301^{b}	0.760^{a}	0.716^{a}	0.779^{a}	0.876^{a}	0.873^{a}	0.748^{a}
S.D	0.028	0.26	0.056	0.045	0.029	0.077	0.027	0.085

Different superscript letters were significantly different, P<0.05

Table 5. Effect of exposure to cigarette smoke on vitamin C levels in plasma

Group	HC 3*	LC 4*	CONT 1	SMOK 2	HCBS 3	LCBS 4	HCDS 5	LCDS 6	HCAS 7	LCAS 8
Vitamin C(µg/L)	743.6^{a}	460.3^{c}	134.3^{h}	91.8^{i}	352.8^{d}	160.5^{g}	356.9^{d}	194.4^{f}	638.2^{b}	258.1^{e}
S.D.	1.70	1.4	2.58	3.5	2.8	0.98^{g}	4.7	2.5	3.6	3.4
% change	+453.7	+242.7	- -----	- 31.6	+162.7	+19.8	+165.7	+44.8	+375.2	+92.2

Different superscript letters were significantly different, P<0.05
HC=High vitamin C dose, LC= Low vitamin C dose.

*3 HC: The highest levels of vit.c in plasma in HCBS group after the first two weeks of taking vit.c.
*4 LC: The highest levels of vit.c in plasma in LCBS group after the first two weeks of taking vit.c.

Table 6. Effect of vitamin C and smoke exposure on TBARS in plasma (nmol/ml) and lung tissue (nmol/g)

Group	CONT 1	SMOK 2	HCBS 3	LCBS 4	HCDS 5	LCDS 6	HCAS 7	LCAS 8
TBARS in plasma								
Mean	1.90^{e}	6.53^{a}	2.26^{d}	3.63^{c}	2.60^{d}	3.76^{c}	3.34^{c}	4.64^{b}
S.D.	0.20	0.69	0.71	0.33	0.64	0.27	0.32	0.69
% change	------	243.7	18.9	91.1	36.8	97.9	75.8	114.2
TBARS in lung								
Mean	0.76^{f}	4.03^{a}	0.86^{f}	1.44^{d}	0.91^{f}	2.02^{c}	1.18^{e}	2.31^{b}
S.D.	0.001	0.14	0.016	0.101	0.033	0.031	0.068	0.049
% change	-------	430.3	13.2	89.5	19.7	165.8	55.3	203.9

Values with different superscript letters were significantly different, P<0.05

Table 7. Effect of vitamin C and smoke exposure on GSH in plasma (μmol/ml) and lung tissue (μmol/g)

Group	CONT 1	SMOK 2	HCBS 3	LCBS 4	HCDS 5	LCDS 6	HCAS 7	LCAS 8
GSH in plasma								
Mean	0.99^{a}	0.23^{f}	0.92^{a}	0.50^{c}	0.74^{b}	0.45^{d}	0.55^{c}	0.35^{e}
S.D.	0.001	0.003	0.027	0.003	0.024	0.004	0.012	0.002
% change	-------	-76.8	-7.07	-49.5	-25.3	-54.5	-44.4	-64.6
GSH in lung								
Mean	6.41^{a}	1.231^{d}	5.08^{a}	3.30^{bcd}	4.19^{ab}	3.04^{bcd}	3.73^{bc}	2.37^{cd}
S.D.	0.023	1.02	0.009	0.024	0.025	0.064	0.25	0.159
% change	-------	-80.8	-20.7	-48.5	-34.6	-52.6	-41.8	-63.0

Values with different superscript letters were significantly different, $P<0.05$

Exposure to cigarette smoke decreased the glutathione (GSH) values both in plasma and lung tissues by 76.8%, and 80.8%, respectively (Table 7).

The impact of inducing the high dose of vitamin C on the GSH values was more effective than

the low dose either before, during , or after smoke exposure.

As a result, it can be suggested that exposure to passive cigarette smoke cause oxidative stress and vitamin C may prevent the body cell from damaged.

The results of the present study are in agreement with those of *(Passamai et al.,2010)*, the oxidative stress biomarkers were measured in individuals exposed to passive smoke before and after supplementation with vitamins C (500 mg) for six months and compared to a control group, they concluded that vitamin C supplementation was effective in decreasing markers of lipid and protein damage and improved both enzymatic and non-enzymatic antioxidant defenses.

Reduced glutathione (GSH) is one of the most abundant intracellular thiols, and aids in protection of cells from the lethal effects of toxic and carcinogenic compounds as well as a wide variety of drugs *(Townsend et al., 2003)(Go& jones, 2010)***.** GSH can function as an antioxidant in the ingest free radicals, can maintain ascorbate in a reduced and functional form *(Sheweita& Tilmisany, 2003)*. Therefore, GSH depletion may promote tumor development through a mechanism that involves cytotoxicity and other different ways (Ortega et al., 2011) **.**

Glutathione is present in two forms, one is called reduced form (GSH) and the other form is the oxidized form (GSSG). Many toxic compounds may eventually lead to glutathione oxidation (GSSG) which changed into its reduced form by glutathione reductase (GR) activity (Petrulea et al., 2012) **.**

These results agreed with those of *(Ghoneim et al., 2015)* who found higher levels of antioxidants in healthy plasma compared with patients with chronic obstructive pulmonary disease.

Histological results

Group 1 (Control): Light micrograph of control group mice section of lung Figure (1), showing a part of respiratory bronchiole (RB) with single columnar lining epithelium (SC). Alveoli (A) appear patent with thin wall linked by type I squamous epithelium (P_1) and type II pneumocystis (P_2). Thin inter-alveolar septum was noticed (↑). Some small blood vessels (BV) reveal congestion with RBCs.

Group 2 (SMOK): Figure (2) shows congested thick walled blood vessels (Bv), cellular infiltration I, and stratification collapsed respiratory bronchiole (Rb). Alveoli are patent with thick wall and some shows secretions with their lumen ↑.

Group 3 (HCBS): Photomicrograph of group(CBSH) mice lung showing mild peri-bronchiole, cellular infiltration, and patent alveoli with thin IAS (↑) as shown in (Fig 3).

Group 4 (LCBS): Lung section shows patent alveoli with areas of thick IAS (↑) extravasations of RBCs (R) congested this walled blood vessel (BV) (Fig 4)**.**

Group 5 (HCDS): Photomicrograph of mice lung section shows patent alveoli (A), some showing thick inter-alveolar septum (↑), dust and dark particles (D) were striking in this photo (Fig. 5).

Group 6 (LCDS): Section of mice lung reveals collapsed alveoli (A) lined by irregular wall, thick inter-alveolar Septum (↑), some alveoli are completely obliterated with massive intercellular infiltration (I) (Fig 6).

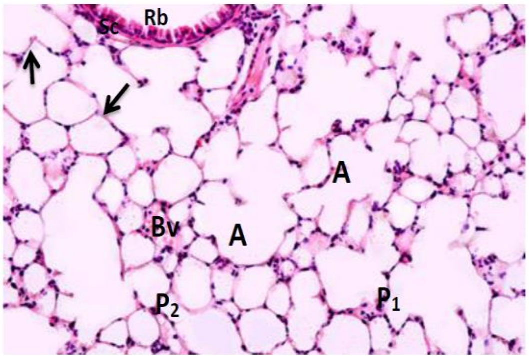

Fig.1. Light micrograph of control group mice section of lung

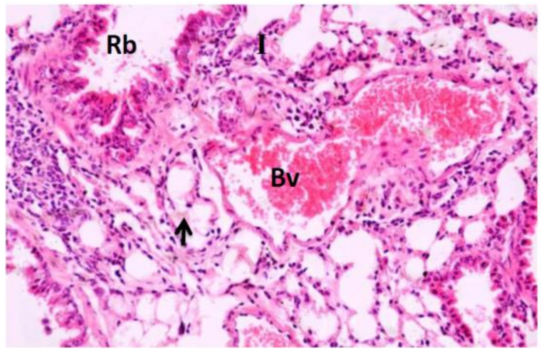

Fig.2. Light micrograph of SMOK group mice section of lung

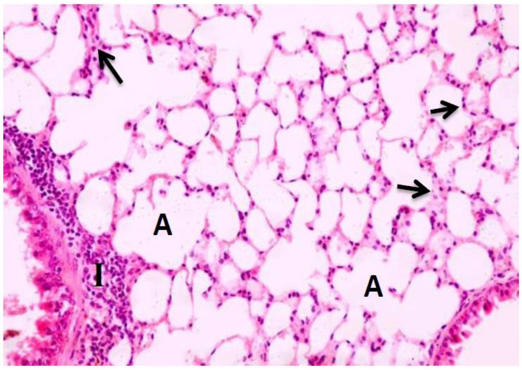

Fig.3. Light micrograph of HCBS group mice section of lung

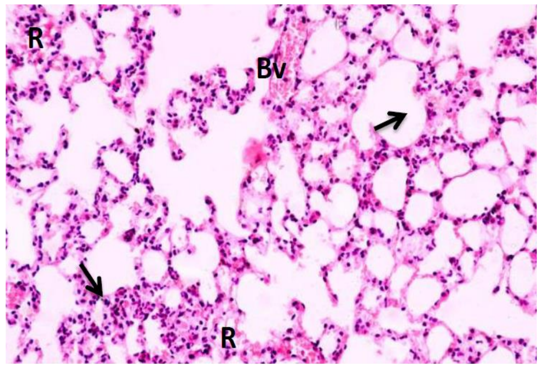

Fig.4. Light micrograph of LCBS group mice section of lung

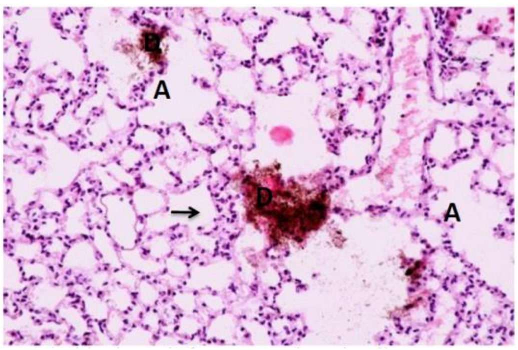

Fig.5. Light micrograph of HCDS group mice section of lung

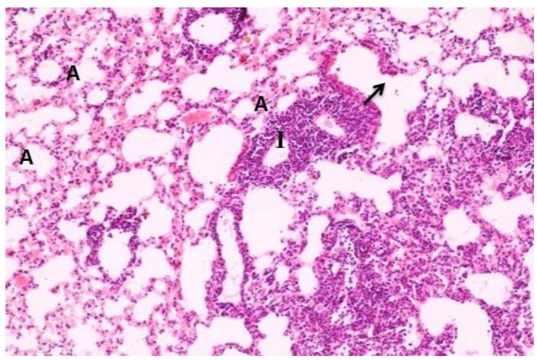

Fig.6. Light micrograph of LCDS group mice section of lung

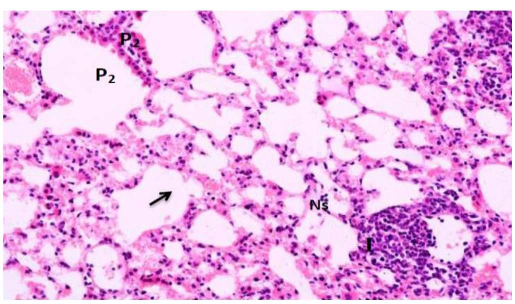

Fig.7. Light micrograph of HCAS group mice section of lung

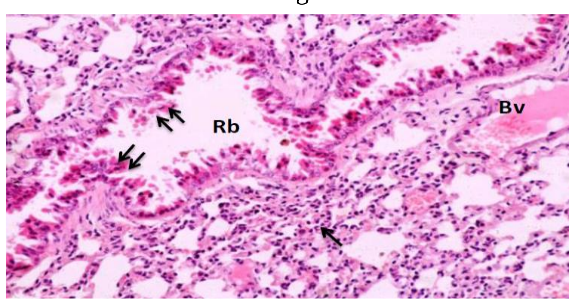

Fig.8. Light micrograph of LCAS group mice section of lung

Group 7 (HCAS): Lung tissues show patent alveoli (A) , while other are collapsed. Thick inter-alveolar septum (↑) with cellular infiltration (I) esp. Neutrophil (Ns), RBCs extravasation between alveoli. Increased numbers of Pheunocytes II (P_2) were noticed in the upper hyper-inflated alveolus (Fig 7).

Group 8 (LCAS): Lung section illustrats massive collapse of most of alveoli, thick IAS (↑), congested blood vessel (BV), stratification of respiratory bronchiole (Rb) with exfoliation of some cells in its lumen (↑↑) (Fig 8).

In this study, most bronchioles showed partial shedding of the mucous lining and the appearance of cellular residue inside. These cloud changes are due to the direct toxic effect of smoke on the bronchioles lining the mucosa. These results were consistent with those previously reported after cigarette smoking in terminal bronchioles, where incomplete areas of epithelial desquamation and accumulation of cellular debris within the cavity were detected (Dye& Adler, 1994).

The inflammatory cellular infiltration that was discovered in this study can be considered a lung defense mechanism against the toxic effect of air pollutants. Infiltration cells help the rapid and rapid removal of foreign particles such as tissue debris and red cells, paving the way for regeneration. The current data was more consistent with the view that cellular defensive reaction occurs primarily against pathogenic bacteria or irritating chemicals (Junqueira&Carneiro, 2003).

There is pulmonary congestion with the thickness and expansion of the pulmonary vessels, the thickening of the barrier between the vesicles by a marked infiltration of a single nucleus cell including macrophages with blood leaking into the lumen of some of the pulmonary vesicles. Camouflage of most air sacs was detected, then the adjacent parts were stretched and compensated with the alveolar wall destroyed with the increased deposition of collagen fibers in the interstitial, around the bronchi and pulmonary vessels.

The pulmonary vascular congestion observed in rats exposed to tobacco smoke may be from un filtered cigarettes due to the toxic effects of smoke. Smoking may affect blood vessels by releasing vasodilators into the bloodstream. Stagnant blood in dilated capillaries will cause hypoxia in the lung tissue resulting in greater pulmonary congestion (Gilman et al.,1981).

Interstitial bleeding as well as within the alveoli in these mice can be explained by increased vascular permeability. Vascular permeability was a result of the release of polypeptide mediators from smoke-exposed cells (Shoji et al., 1995). In addition, cigarette smoke has impaired endothelial function in smokers due to increased oxidative stress and enhanced formation of free radicals derived from oxygen (Motoyama et al., 1997).

Toxicity is observed directly on the capillary wall leading to ischemia, followed by vasodilation and blood escaping from its dead wall to the barrier between the vesicles and the lumen of the alveoli. This was similar to the result that long-term exposure to nitrous oxides as an air pollutant showed significant lung congestion (Nakai et al., 1999). Moreover, lung congestion was usually associated with a decrease in gas exchange resulting in an expansion of the alveolar space in the air (Carpy et al., 2000).

Disruption and injury to the blood vessel wall may be explained by the relative hypoxia caused by exposure to carbon monoxide (CO) (Holley et al., 1999).

The increased thickness of the walls between the follicles observed in the experimental mice of this study can be explained by the presence of excess inflammatory cells, capillary congestion, increased interstitial connective tissue and the associated alveolar collapse. These results coincided with those who linked the thickness of the alveolar septum with a modification in the blood vessels leading to infiltration and inflammatory edema (Hora et al., 2003)

The current study showed the destruction of some alveolar walls and foci of collapsed vesicles with the subsequent expansion of adjacent alveolar spaces and the formation of large irregular areas (maternal changes). These changes were previously described by researchers who transmitted this result to lung tissue injury by oxidative mechanisms created by oxidants in cigarette smoke extracts as well as those released by specific inflammatory cells, especially alveolar macrophages and neutrophils (Czekaj et al., 2002).

Important role was played by macrophages in the cause of smoke emphysema. The elastic enzymes released by macrophages have damaging effects on the airway wall. Exposure to chronic smoke increases the production of metallo-proteinases (MP) by macrophage. These are proteolytic enzymes and their enhanced release may be responsible for the destruction of lung tissue (Barnes et al., 2003) (Stewart& Voelkel, 2008).

CONCLUSION

The results indicated the following:

1. Exposure to smoke led to doubling of the relative weight of the lung.
2. Exposure to smoke led to decrease the vitamin C levels in plasma.

3.Plasma and lung TBARS increased by exposure to smoke.
4.Plasma and lung GSH decreased by exposure to smoke.
5.Vitamin C high dose intake showed protective effect against the negative impacts of smoke.
6.Vitamin C high dose intake showed a relative improvement in the histology of the lung.

SIGNIFICANCE STATMENT

This study indicated that vitamin C increases the ability to reduce the risk of passive smoking, especially high doses of vitamin C (1 g / day for human) had a protective effect on respiratory health, it helps to reduce the prevalence of chest diseases caused by air pollution. The recommended dose in the study is able to protect the lung from the harmful effects of smoking. Whereas, this dose of vitamin C (1 g per day) is unable to repair damaged lung cells. So, this study may help researchers to estimate another dose of vitamin C as cured dose to repair this damage.

RECOMMENDATIONS

1)The importance of having (3 servings/ day) of fresh vegetables at least, especially sweet peppers, leafy vegetables and tomatoes as rich sources of vitamin C. For example, eating three servings or three cups of chopped sweet pepper daily give approximately 500 mg of vitamin C.
2)The importance of intake (3 servings/ day) of fresh fruits at least, especially, guava, kiwi, strawberries and citrus fruits. For example, eating three servings of guava a day will give approximately 684 mg of vitamin C.
3)Avoiding exposure to all sources of air pollution especially passive smoking.

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الملخص العربي

الجرعة الفعالة من فيتامين ج لمواجهة التدخين السلبي

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نظرا للدور الهام الذي يلعبه فيتامين ج في جسم الانسان والذي يحمي الجسم من التأثيرات الضارة للشوارد الحرة وخاصة التي تتكون بفعل التدخين السلبي فقد استهدفت الدراسة معرفة الجرعة الفعالة من فيتامين ج والتي قد يكون لها دور في حماية الرئة من التأثيرات الضارة للتدخين السلبي، وذلك من خلال اختبار تأثير جرعتين محددتين من فيتامين ج على فئران التجارب (0.015 – 0.075 ملجم \ جم من وزن الحيوان) قبل وأثناء وبعد التعرض للتدخين السلبي والتي تعادل 200 ملجم و 1000ملجم من فيتامين ج على التوالي يوميا للإنسان.

وقد كان للجرعة الكبيرة منه تأثيرات وقائية، حيث أنها قللت من التأثيرات السلبية للدخان وفقا للاختبارات الكيميائية والنسيجية المذكورة في الدراسة، ولكن لم يكن لفيتامين ج تأثير علاجي يذكر بعد تعرض نسيج الرئة للتلف بفعل الدخان.

ولهذا كان من الضروري حماية الرئة من التأثيرات الضارة للتدخين السلبي بزيادة الجرعة اليومية للإنسان لتصل الى 1000ملجم، وذلك من خلال زيادة المتناول من الأغذية الغنية بفيتامين ج مثل الخضروات والفاكهة بالحصص المقررة في توصيات الدراسة.