Free Radicals and Antioxidants in Cigarette Smokers

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Abstract

To study the effect of smoking on the antioxidant parameters, young male smokers(n=15) and non smokers(n=15) aged 19 to 25 years. Our study underway to determine the antioxidants such as vitamin $E(\alpha$ -Tocopherol), through its ability to limit production of free radicals and help prevent or delay the development of diseases by the cigarettes smoking and the antioxidants which works together(co-antioxidants), and recycle each other or work in concert; therefore, the total and reduce vitamin C(ascorbic acid) were determine, we found the levels of these vitamins in smokers less than non smokers. In conclusion, according to the results of the present study, we explain how much smoking lead to release of free radicals and cause depletion of antioxidants, finally causing oxidative damage.

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Introduction.

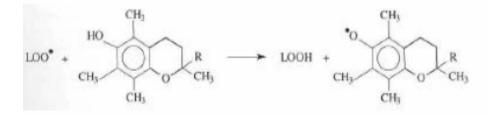
Cigarette smoking is associated with increased free radical generation and cause inhalation of various oxidant and prooxidant compounds, and results in the formation of more than a billion oxy-radicals in each puff, therefore, smoking is associated with multiple complication diseases^(1,2).

A free radical is any chemical species capable of independent existence and possessing one or more unpaired electron, an paired electron being one that alone in an orbital. Radicals, often denoted by the insertion of the superscript dot(.), are generally less stable than non radicals, although their reactivities vary. Free radicals of importance in living organisms include hydroxyl(OH), superoxide(O₂⁻), and peroxyl (LOO⁻), peroxynitrite(ONOO⁻), hydrogen peroxide(H₂O₂), single oxygen ${}^{1}\Delta g$ and ozone(O₃), are not free radical reactions in living organisms⁽³⁾.

The term reactive oxygen species(ROS) is often used to include both the radical and non radical species. Oxidative stress is the term referring to the imbalance between the generation of reactive oxygen species and the activity of the antioxidant defenses ⁽⁴⁾. Antioxidants help to reduce the damage in the body caused by toxic chemicals and pollutants such as Cigarette smoking.

There are many types of antioxidants; they can be classified by their mechanism of action. Preventative antioxidants include peroxide decomposers, while chain breaking antioxidants intercept chain carrying radicals. Many chain breaking antioxidants donate a hydrogen atom to the chain carrying radical thereby stopping the oxidation process, this results in an antioxidant radical. However, this radical is much less reactive than the chain carrying radical but even this much more domesticated radical must be removed ⁽⁵⁾.

 α -Tocopherol (TOH) is a topical donor antioxidant in this study; it protects against lipid peroxidation, which is a chain reaction, its breaks the propagation by donation a hydrogen atom to the chain carrying peroxyl radical, LOO', thereby stopping the oxidation process⁽⁶⁾, the tocopheroxyl radical (TO') can be repaired by additional donor compound that more reducing than TOH such as ascorbate (AscH⁻)⁽⁷⁾.



The reaction of AscH⁻ with TO naturally results information of Asc radical, this radical is in turn removed by enzymatic systems; thus AscH⁻ is recycled, ascorbate, an excellent reducing agent undergoes two consecutive, reversible, one electron oxidation

 $AscH^- + X$ $\rightarrow Asc^+ + ZH$

The ascorbate radical formed in these reactions is relatively stable and does not react with O_2 to form dangerous peroxyl radicals and appears to produce very low levels of $O_2^{-(10,11)}$.

Methods and Material.

A group 30 healthy, male subjects volunteers for the study. The volunteers were classified as smokers(those who had smoked at least twenty cigarette a day for at least one month; n=15), and non

processes forming the Asc radical as an intermediate. These properties make ascorbate a superior biological, donor antioxidant⁽⁸⁾. It can repair many free radicals that are produce during oxidative stress, such as OH, LOO and TO⁽⁹⁾.

smokers(those never have smoked; n=15).We aimed to examine their effects on antioxidants parameters in the same subject group. The blood samples were collected from smokers and non smokers, after clotting, serum was separated by centrifugation, the analytical determinations described below were either preformed immediately, or serum was stored at -20° C and used within 72 hours.

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Vitamin E was measurement using the methods described by Toro et al.⁽¹²⁾, α – tocopherol react with α, α' - dipyridyl to produce a complex, which has λ max in 520nm,Vitamin E concentration was express as mg/L. Vitamin C concentration were evaluated in serum by using chemically analytical, briefly, ascorbic acid in the protein free supernatant is oxidized by Cu⁺² ions to dehydroscobic acid and diketogulonic acid. These ketones react with 2,4- dinitrophenyl hydrazine in H₂SO₄ to form the phenyl hydrazone products, which absorb at 520nm⁽¹³⁾.

A correlation study between the 2,4dinitrophenyl hydrazine method (DNPH) and 2,6-dichlorophenolindophenol method (DCIP) showed good agreement⁽¹⁴⁾, as below:

DNPH = 1.088(DCIP) + 0.12

Statistical analysis.

All results are expressed as mean \pm SD(standard deviation), comparison between smokers and non smokers was performed by the Student's t- test. Pearson's correlations were used to determine relationship between parameters studied taken P \leq 0.05 as the lowest limited of significant.

Results and Discussion.

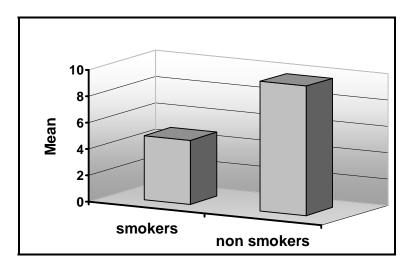
Vitamin E concentrations were significantly lower in smokers compared to that found in non smokers(P< 0.01) Fig 1, while the values of total and showed reduced ascorbic acid а significant decreased in smokers compared to that in non smokers(P< 0.05) Fig.2, 3 receptively, Table 1 shown The levels of each of vitamin E and C(total and reduced) in mg/L.

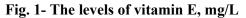
Type of Vitamin in mg/L	Non smokers (n=15)	Smokers (n=15)	Р
Vit. E	12.4 ± 3.7	4.8 ± 1.8	0.000 S
Vit. C(total)	10.2 ± 2.5	7.7 ± 2.1	0.00 S
Vit. C(reduced)	9.9 ± 2.5	7.5 ± 2.1	0.00 S

Table 1- The levels of each of vitamin E and C(total and reduced) in mg/L.

Results are expressed as mean ± SD. Abbreviations: Vit. E, Vitamin E; Vit. C, Vitamin C; S, significant.

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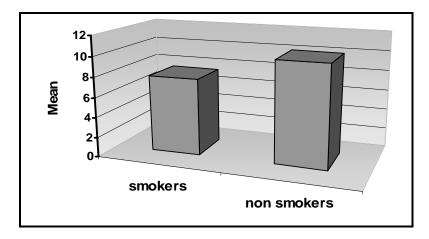
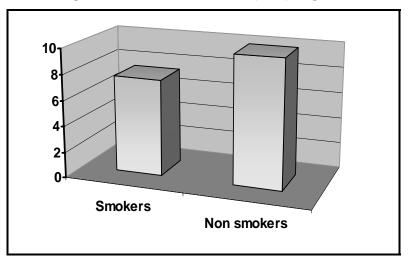
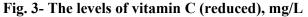


Fig. 2- The levels of vitamin C(total), mg/L





The smoke of cigarettes reduces the activity of antioxidant system, this may

lead to imbalance between oxidantantioxidant in favor of the prooxidant

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leading to potential damage and dominant risk factor of many diseases⁽¹⁵⁾. Tobacco smoke as a source of exposure to carbon monoxide(CO), to give high levels of hemoglobin carbon monoxide, which were found to be associated with the prevalence of coronary heart disease⁽¹⁶⁾. Evidence for increased oxidation in smokers compared with non smokers were also provided by the presence of increased prooxidation products in blood⁽¹⁷⁾.

Low levels of vitamin C and E have been associated with a cigarette smoking due to gas phase of cigarette smoke which is a complex oxidizing milieu processing an array of free radical species including peroxy radicals (LOO'), and $(O_2^{\bullet^-})$. which has been shown to deplete lipid phase^(18, 19).

The difference in antioxidant levels of smokers and non smokers can be due to the different in age, lifestyle, type of cigarettes and nutrition.

Smoker serum levels of vitamin E were correlated with vitamin C values (Fig. 4), which suggests consumption of the vitamin E as a radical scavenger, and recycle it by the vitamin C. Accordingly, in this report we investigated the oxidant damage due to the oxidative stress that occur in subjects that are under oxidative stress cigarette smoking.

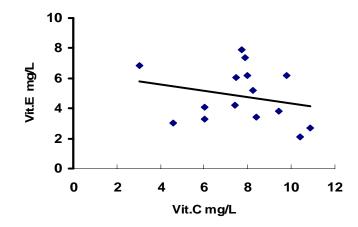


Fig. 4- Correlation between the levels of vitamin C(total) and vitamin E

Conclusion.

According to the results of the present study, we explain how much smoking lead to release of free radicals and cause depletion of antioxidants, and investigated that the oxidant damage due to the oxidative stress which occur in subjects that are under oxidative stress cigarette smoking.

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