Studying the relationship between oxidative stress malondialdehyde and ß-carotene in the serum of asthmatic patients in Basrah Governorate-Iraq

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Abstract

The present study was designed to evaluate the relationship between ß-carotene as antioxidant with malondialdehyde (oxidative stress) in the serum of asthmatic patients. In this study, a total number of (50) asthmatic patients (24 males, 26 females) visiting Allergic and Asthma Medical Center in Basrah city were involved and according to age, sex, severity, smoking and family history. From each patient serum blood samples were collected to estimate the malondialdehyde (MDA) and ß-carotene. In addition, (50) healthy subjects were investigated as a control group. We found that a highly significant increase lipid peroxidation measured as MDA ($p < 0.01$) for all asthmatic patients as compared to healthy control.

Also the (MDA) serum level increases significantly ($p < 0.05$) with age and severity, while a highly significant ($p < 0.01$) is with smoker patients and positive family history. The study also revealed a statistically significant decrease ($p < 0.01$) in ß-carotene serum level in all asthmatic patients as compared to healthy control. In regard to age and severity, serum ß-carotene level decreased significantly ($p < 0.05$) while with smoker patients and positive family history a highly significant decrease ($p < 0.01$). There were no significant changes in the (MDA) and ß-carotene levels between male and female patients. Moreover, a negative correlation was also observed between ß-carotene with (MDA) level in the patient group with these variables.

In conclusion, asthmatic patients suffer a high degree of reactive oxygen species (ROS) formation causing considerable oxidative stress indicated by high level oxidant (MDA) and low level of the antioxidant ß-carotene.

Key words: MDA: Malondialdehyde,   ROS: Reactive oxygen species,   RNS: Reactive nitrogen species. LDL: Low density lipoprotein.

Introduction:

Asthma may be regarded as a diffuse, obstructive lung disease with hyper-reactivity of the airways to a variety of stimuli and a high degree of reversibility of the obstructive process, which may occur either spontaneously or as a result of treatment [1]. Asthma is a complex disorder involving biochemical, autonomic, immunologic, infectious, endocrine, and psychological factors in varying degrees in different individuals. In some patients with so called extrinsic or asthma attacks follow exposure to the environmental factor such as dust, pollens, danders and foods, often but not always, such patients have increased concentrations both of total IgE and of specific IgE against the allergen implicated. In other patients with clinically similar asthma, which is seen most often in older adult "Late onset" asthma, has been called intrinsic or non immunologic [2]. Asthma may have its onset at any age. The course and severity of asthma are difficult to predict. The majority of affected individual have only occasional attacks or slight to moderate severity managed with relative case, a minority will develop severe intractable asthma, usually perennial rather than seasonal [3]. Airway hyper reactivity relates to patients but generally is stable over time in the same patient except for temporary fluctuation. An acute decrease in air way irritability is observed following the administration of ß-receptor agonists, theophylline, anticholinergic, and after chronic administration of chromolyn or corticosteroids, systemic or inhaled [4].Several mechanisms operate in cellular damage and death, lipid peroxidation caused by free radicals being one of the most important mechanisms [5]. A free radical is an atom or molecule that has one or more unpaired electrons, they are two types; a reactive oxygen species (ROS), such as super oxide anion radical $(O_2^\cdot)^-$, hydrogen peroxide ($H2O2$), hydroxyl radical ($OH^\cdot$) and singlet oxygen ($O2^1$). And reactive nitrogen species (RNS), such as nitrous oxide ($NO^\cdot$), nitric oxide ($NO2^\cdot$), peroxynitrite ($OONO^\cdot$). Because ROS are so reactive, they can inflict considerable damage on living cells if formed in significant amounts. These damage results primarily form enzyme inactivation, polysaccharide depolimerization, DNA breakage and membrane destruction [6,7].

A lot of oxygenated compounds, particularly aldehyde such as malondialdehyde (MDA), are produced during the attack of free radicals to membrane lipoprotein and polyunsaturated fatty acid, products of lipid peroxidation formed in the primary site reaching the other organs and tissue via the blood stream provoke lipid peroxidation and cause cellular and tissue damage [8]. The Increase of lipid peroxidation could possibly play a role in the complication of cardiovascular disease, chronic pulmonary disease, cataract, cancer [9, 10]. MDA measurement is an indicator of lipid peroxidation and is used as a biomarker of oxidative stress [11]. Oxidative stress occurs when this balance is disrupted by extreme production of reactive oxygen species and / or by insufficient anti oxidative defenses, including superoxide dismutase (SOD), catalase (CAT), vitamins (C,E), ß-carotene, uric acid, glutathion (GSH), and trace elements such as zinc (Zn), selenium (Se), magnesium (Mg), cupper (Cu), iron (Fe), which are Co factor for many biochemical reactions [12,13]. ß-carotene is a member of a class of plant pigment molecules referred to as the carotenoids, it acts as provitamin A. It also performs a function as antioxidants, protecting the body from the potentially damaging effects of various oxidizing agents [14]. ß-carotene can exert antioxidant effect in lipid systems by quenching singlet oxygen, peroxyl radical and inhibit lipid peroxidation.

ß-carotene and other carotenoids offer protection against a variety of serious degenerative diseases especially cancer, cardiovascular and cataract [15].

The present study was undertaken to evaluate the association between MDA (a marker of oxidative stress) with non enzymatic antioxidants ß-carotene with several parameters (age, sex, severity, family history and smoking) in the serum of asthmatic patients in Basrah/Iraq.

Material and Methods:

Samples:

Fifty patients (24 males, 26 females) with asthma clinically diagnosed admitted to Allergic center from the first of October 2009 to the end of March 2010 in Basrah city/Iraq whose age ranged between (15-60) years for males and females, divided into three groups (15-35) years, (35-50) years, (>50) years.

The following information was recorded for asthmatic patients: age, sex, smoking, date of admission, family history of allergy, sign and symptom and severity of the acute attack. Those patients were classified according to the severity into three groups:

1- Mild   2- Moderate   3- Sever

This classification of severity depends on expert panel report; guidelines for the diagnosis and management of asthma, sign and symptoms were used mainly for the classification of severity [16].

Fifty healthy subjects (24 males, 26 females) were investigated as a control group from Public Health Center in Basra city aged between (15-60) years.

Blood samples (5mL) were collected from patients and healthy control by vene puncture using a sterile disposable syringe in plain plastic tube. The blood was centrifuged at 3000 rpm for 10 min; the serum was divided into two equal parts in sample tube, serum malondialdehyde were tested within 24 hours, and the other was frozen at -20°C for estimation of ß-carotene.

Instruments:

1- Spectrophotometer SP 8-100 VV pye Unicom, U.K.

2- Centrifuge, Kokusan, Japan.

3- Vortex stirrer, Gallen Kamp, Germany.

4- Water bath, Gallen Kamp, Germany.

Materials:

The entire chemicals in this study were imported from BDH Co. and Sigma Co.

Assessment of the lipid peroxidation activity:

The assessment of lipid peroxidation process is achieved via the determination of the byproduct; Malondialdehyde was determined by a modified procedure described by Guidet B. and Shah S. V. [17]. In brief; to one mL serum sample (T) add 2 mL of the mixture solution (15 % w/v trichloroacetic acid TCA, 0.375% w/v of thiobarbituric acid TBA, 2% v/v conc. Hydrochloric acid) and mixed thoroughly, then heated for 15 min in boiling water bath. After cooling, the precipitate was removed by centrifugation at 3000 rpm for 10 minutes. The absorbance of red color solution was determined at 535 nm against reagent blank (B) which was containing all the reagents minus the serum.

Calculation:

The concentration of malondialdehyde MDA (μmol/L) =

$$\frac{AT - AB}{1.56 \times 10^5 \text{ (L/mol. Cm)}}$$

AT: The absorbance of the sample serum.
AB: The absorbance of the blank.
1.56×10^5 molar absorptivity of MDA.

Estimation of serum ß-carotenes:

The serum carotene was determined by Kaplan L.A. [18]. Carotene usually binds serum lipoproteins. Ethanol is added to the serum to break these complexes and release carotene. The

carotenoids are extracted into petroleum ether to remove them from interfering substance in serum. Resultant orange-yellow color was read at 450 nm. Briefly, to 1 mL of serum and control were added 2 mL of ethanol drop wise added while vortex mixing to each tube, 2 mL of petroleum ether were added. Each tube was vortex mixed for 3 minutes. Layers were allowed to separate by standing for 5 minutes, the petroleum ether layer was carefully drawn into a cuvette and the absorbance was read at 450 nm against the blank which was containing all the reagent minus serum.

Calculation:

The absorbance can be converted to carotene concentration equivalents using the standard curve.

ß-carotene standard:

a- prepare a stock standard solution (1000 mg/L) of ß-carotene.

b- Working standard (20 µg/mL). Add 2 mL of stock standard, and the volume was completed with petroleum ether to 100 mL. From this solution, makes different concentrations of working standard of 0.5 to 5 µg/mL.

c-The absorbance was read at 450 nm against petroleum ether as a blank then was plotted versus concentration as in Fig.1 .

Statistical Analysis:

The results were expressed as mean ± SD. The data were analyzed statistically by one-way analysis (ANOVA), while the correlation between the data was tested statistically by simple linear test by using computer SPSS (14) program.

P-value of less than 0.05 was considered as statistically significant, P-value less than 0.01 as highly significant.
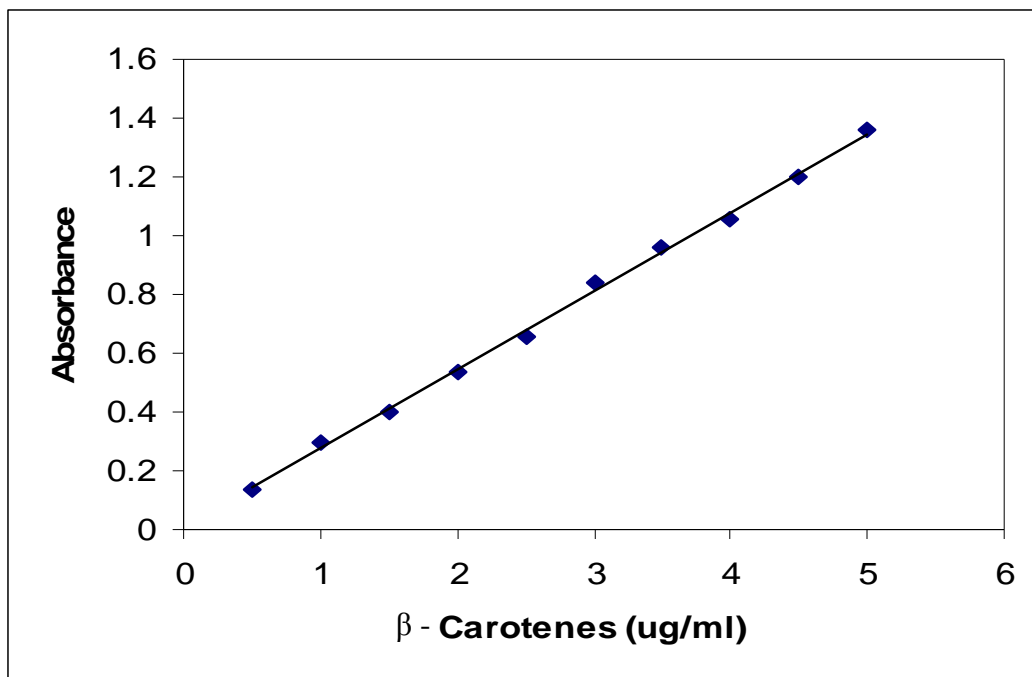

Figure 1: Carotene standard curve

Result and Discussion:

Asthma is a chronic inflammatory disease of the respiratory tract of unknown etiology. According to the world health organization, asthma is now a serious public health problem with over 100 million sufferers worldwide, death from this condition has reached over 180,000 annually [19, 20].

Asthma remains the most common chronic illness of childhood, it is regarded as the fifth cause of death in children [21].

The present study revealed a highly significant elevation of malondialdehyde (MDA) among all asthmatic patients studied compared to healthy control (p< 0.01). In addition there was a significant increase (p< 0.05) in the (MDA) serum level with increasing age in both males and females patients, but statistically there were no significant differences between both sexes as shown in (Table1, Fig.2). These imply that patients during acute asthmatic attack are exposed to considerable degree of lipid peroxidation. This finding is consistent with observations of others [22, 23].

MDA is a marker of lipid peroxidation, it has a strong correlation with atopic asthma suggesting that oxidative stress occurs simultaneously on lipid peroxidation. Oxidative stress can have many effects on airway function, including airway smooth muscle contraction, induction of airway hyper responsiveness, mucus hyper secretion, epithelial shedding and vascular exudation. Furthermore, ROS can induce cytokin and chemokin, the production through the induction of oxidative stress-sensitive transcription on nuclear factor in bronchial epithelial cells involves the uptake of oxygen and subsequent release of ROS into surrounding cells [24].

During the respiratory burst, the inflammatory cells have released high concentration of $O_2^\cdot$, $OH^\cdot$, HOCl and H_2O_2 that may leak into surrounding cells resulting in increased quantities of free radicals in airway tissues. Furthermore, the inflammatory cell of asthmatics has an increased capability to generate free radicals compared to controls, which further contributes to high concentrations of ROS. Excess reactive nitrogen species (RNS) may be also produced by asthmatics. Cytokines may stimulate increased production of nitrosyl ($NO^\cdot$) radical which reacts with O_2 to form peroxynitrite, a cytotoxic species that has many damaging effects, including lipid peroxidation. $NO^\cdot$ can also be converted to nitrite, which can oxidize proteins. Thus, the excess quantities of ROS and RNS that are produced by asthmatics may overcome the host antioxidant defenses and cause oxidative stress [24,25].

Asthma severity is related to the extent of lipid peroxidation, with a positive association between MDA concentration and disease severity (p< 0.05). A similar trend was reported [26]. On the other hand, there was a highly significant difference in MDA level between asthmatic smokers and non smokers (p< 0.01) as indicated in (Table1, Fig.2). Cigarette smoke contains high amount of free radicals such as superoxide and nitric oxide which are found in cigarette, gas phase can react chemically to form highly reactive free radical peroxynitrite. In addition superoxide can react with hydrogen peroxide to form the more active hydroxyl free radical [27]. Many researchers have reported increased levels of superoxide anion from circulating neutrophils and increased lipid peroxidation products in the plasma of smokers, supporting the concept of systematic oxidative stress in these individuals [28].

Also family history seems to influence the level of MDA as compared to positive and negative family history of asthmatic patients, there was a highly significant increase (p< 0.01) in the (MDA) serum level as shown in (Table1, Fig.2). Some genetic variants may only cause asthma when they are combined with specific environmental exposures, the genetic trait, CD14 single nucleotide polymorphism, and exposure to endotoxin (a bacterial product).

Researchers have found that the risk for asthma changes based on a person's genotype at CD14 and level of endotoxin exposure [29, 30]. Asthma itself may cause physiological changes in serum antioxidants burden associated with disease [31, 32]. Numerous disturbance of anti-oxidant defense mechanisms have been described in asthma, the epithelial lining fluid of the lung contains high concentration of anti-oxidants which provide a first line defense against inhaled and endogenously oxidant [33].

Anti-oxidant status may affect asthmatic risk by influencing the development of immune phenotype [33].

The present study has thrown a light on the level of serum ß-carotene as antioxidant and lipid peroxidation as oxidant in patients with bronchial asthma.

This study revealed a highly significant decrease (p< 0.01) in ß-carotene serum level in all asthmatic patients during acute attack as compared to healthy control. These significant changes increase with age, severity from mild to moderate than severe (p< 0.05). as indicated in (Table 2, Fig. 3). Also there were no significant differences between males and females patients.

Furthermore, there were highly significant differences (p< 0.01) in ß-carotene level between the groups of patients depending on family history as illustrated in (Table 2, Fig.3). These significant changes are due to genetic and endotoxin exposure [29,30].

In inflammation increased generation of reactive oxygen species, ß-carotene acts to quench ROS in order to counter effect and render them harmless [34].

However, continuous ROS quenching results in ß-carotene depletion and a reduction in these oxygen radicals' scavengers lead to oxygen radical airway inflammation that might promote access of allergen to submucosal T lymphocytes is an essential element in allergen recognition [35].

This is supported by the strong negative correlation between MDA and ß-carotene that depended on age, severity as shown in (Table 3, Fig. 4, 5).

Moreover, the significant negative correlation between MDA and ß-carotene is stronger with smoker patients than in non-smokers (p< 0.01) as shown in (Table 3). These results are in agreement with the earlier studies. They found that both ascorbic acid and ß-carotene concentration were lower in the smokers than did non-smokers [36,37].

Antioxidants are of particular importance to smokers because of scavenge of free radicals, which are found in large quantities in tobacco smoke [38]. Also studies have reported that an increased intake of natural sources of antioxidants from fruit and vegetables had a protective effect of the susceptibility of LDL to oxidation, which offers protection against oxidative diseases [39, 40].

Also there is strong significant negative correlation between (MDA) and ß-carotene in patients with positive family history as shown in (Table 3).

Antioxidant ß-carotene could play an important role in gene-environment interaction in complex lung disease such us asthma.

Conclusion:

The presence of the relationship between oxidative stress malondialdehyde (MDA) and ß-carotene as antioxidant in the blood of asthmatic patients was observed in the present study.

Asthmatic patients during acute attack have increased lipid peroxidation level as detected by the increase of serum MDA which was an important factor in increasing the production of free radical that developed bronchoconstriction.

As a result of continuous production of ROS the antioxidant ß-carotene level was significantly lowered especially in severe asthmatic attack, increasing age, and with smoking. There were no significant statistical differences in the mean serum level of MDA and ß-carotene between male and female patients. Also the family history of patients affected the MDA serum level and ß-carotene level.

Table 1   Serum level of malondialdehyde (µ mol/ L) in asthmatic patients classified according to (Age, Sex, Severity, Smoking, Family history) and healthy control

Variable		Asthmatic patients (50)		Control (50)		P-value
		No	Mean ± SD	No	Mean ± SD	
Age/year	15-35	15	7.2620 ± 0.1704*	15	4.6293 ± 0.1919	P<0.01
	35-50	19	7.8869 ± 0.0963*	19	4.7923 ± 0.2352	
	>50	16	8.4283 ± 0.1726*	16	4.9067 ± 0.1348	
sex	male	24	7.7916 ± 0.5592*	24	4.7887 ± 0.2377	P<0.01
	female	26	7.8314 ± 0.565*	26	4.7524 ± 0.254	
Severity	mild	15	7.2930 ± 0.3686*	50	4.805 ± 0.2378	P<0.01
	moderate	19	7.8942 ± 0.1055*			
	sever	16	8.3340 ± 0.2360*			
Smoking	positive	27	8.2091 ± 0.2617**	50	4.7923 ± 0.2352	P<0.01
	negative	23	7.2912 ± 0.2751			
Family history	positive	30	8.1062 ± 0.4390**	50	4.9068 ± 0.1348	P<0.01
	negative	20	7.3250 ± 0.3600			

* The mean difference is significant at the 0.05 level
** The mean difference is significant at the 0.01 level
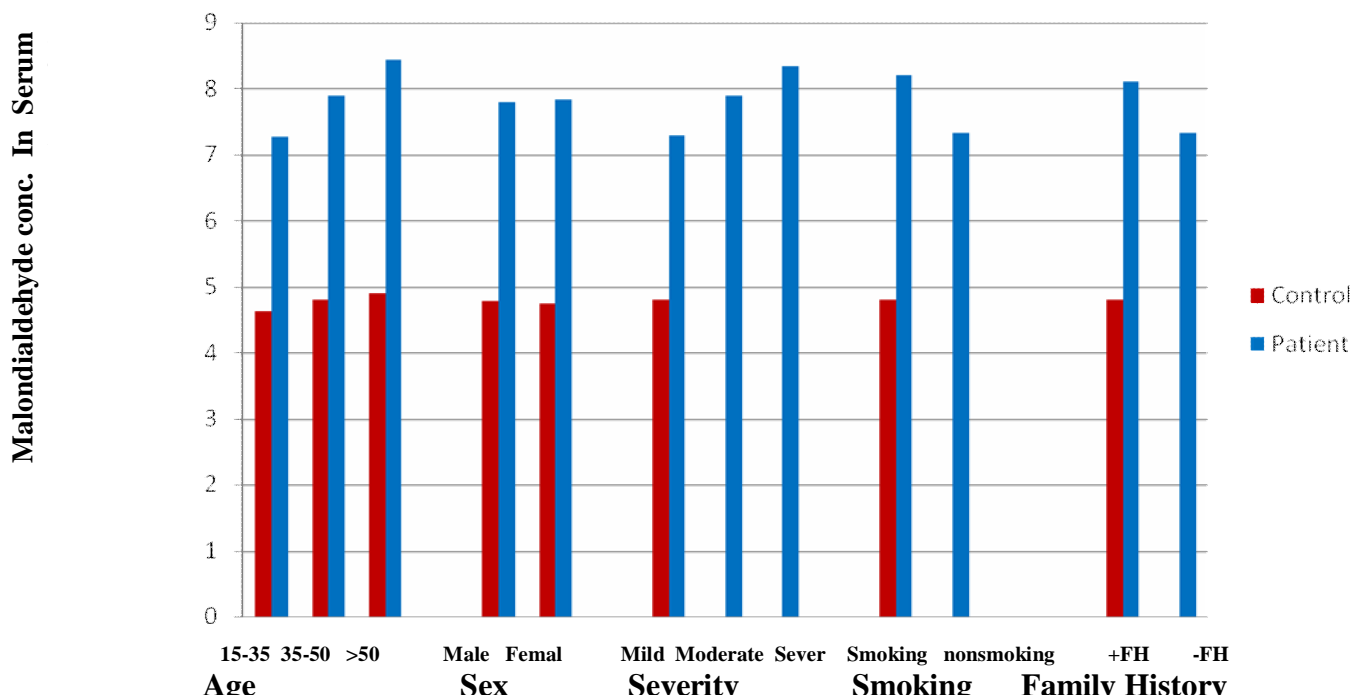
Table 2   Serum level of ß-carotene (µg/mL) in asthmatic patients classified according to (Age, Sex, Severity, Smoking, Family history) and healthy control

Variable		Asthmatic patients (50)		Control (50)		P-value
		No	Mean ± SD	No	Mean ± SD	
Age/year	15-35	15	1.4233 ± 0.5538*	15	2.1867 ± 0.1368	P<0.01
	35-50	19	1.1846 ± 0.5938*	19	2.0115 ± 0.1529	
	>50	16	0.8792 ± 0.4255*	16	2.0375 ± 0.1583	
sex	male	24	1.2105 ± 0.6810	24	2.0974 ± 0.1939	P<0.01
	female	26	1.2262 ± 0.6069	26	2.0690 ± 0.1847	
Severity	mild	15	1.6600 ± 0.4060*	50	2.0840 ± 0.5500	P<0.01
	moderate	19	1.2875 ± 0.5780*			
	sever	16	0.9250 ± 0.4469*			
Smoking	positive	27	0.9000 ± 3522**	50	2.0840 ± 0.5500	P<0.01
	negative	23	1.2559 ± 0.4863			
Family history	positive	30	0.9327 ± 0.3712**	50	2.0840 ± 0.5500	P<0.01
	negative	20	1.4500 ± 0.4476			

* The mean difference is significant at the 0.05 level
** The mean difference is significant at the 0.01 level
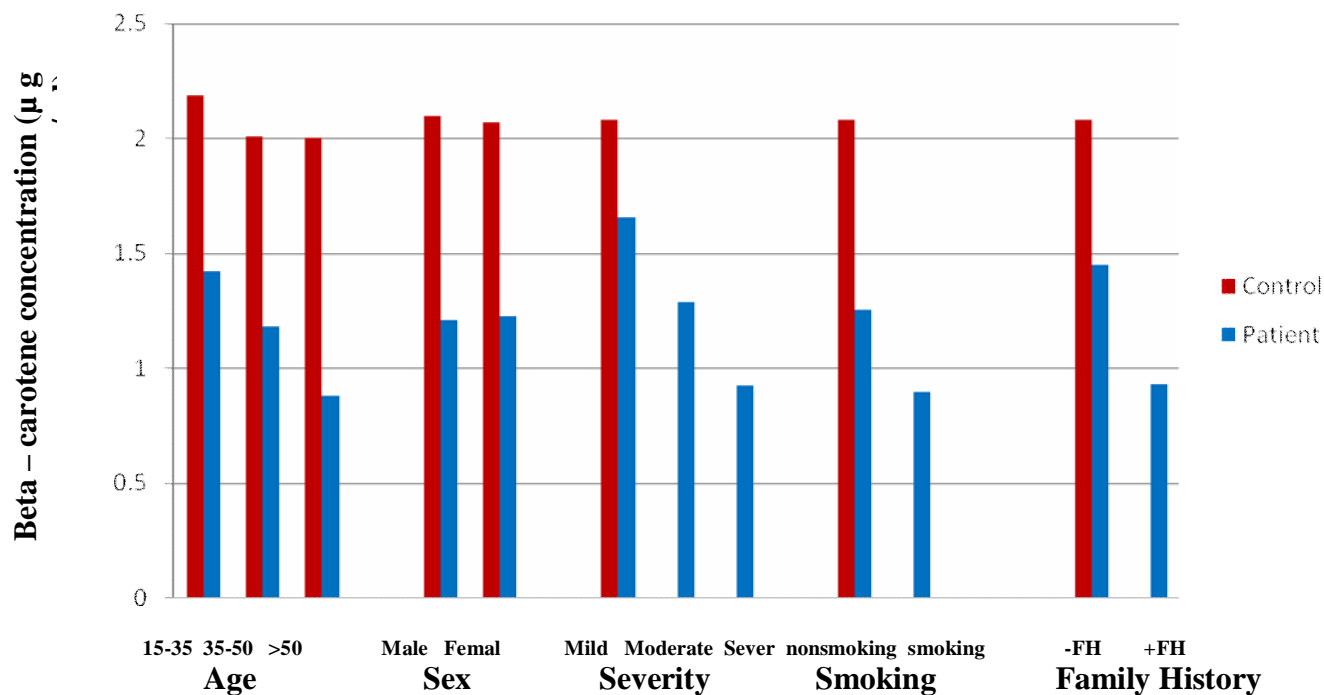
Figure 3 . Serum Beta – carotene concentration (µ g /ml) in asthmatic patients and healthy control classified according to (Age , Sex , Severity , Smoking and Family history).

Table 3: Correlation Coefficient (r) between oxidant (MDA) and antioxidant ß-carotene in asthmatic patients

Variables	Correlation Coefficient (r)	P – Values sig.
Age	- 0.370*	0.019
Sex	- 0.316*	0.047
Severity	- 0.403**	0.010
Smoking	- 0.411**	0.008
Family history	- 0.252*	0.016

** Correlation is significant at the 0.01 level (2-tailed).
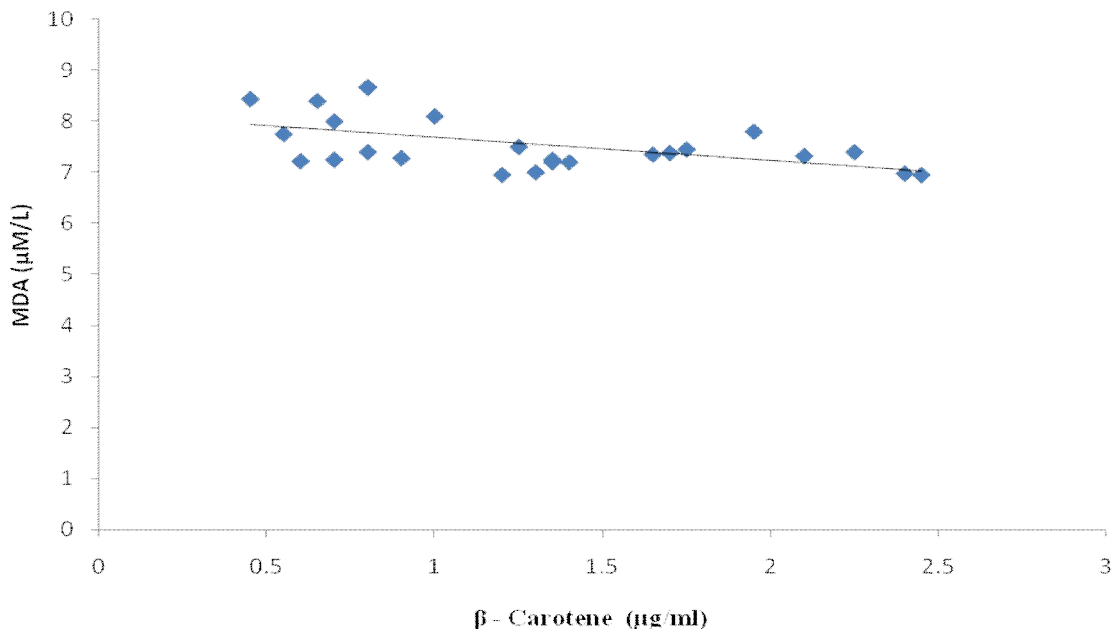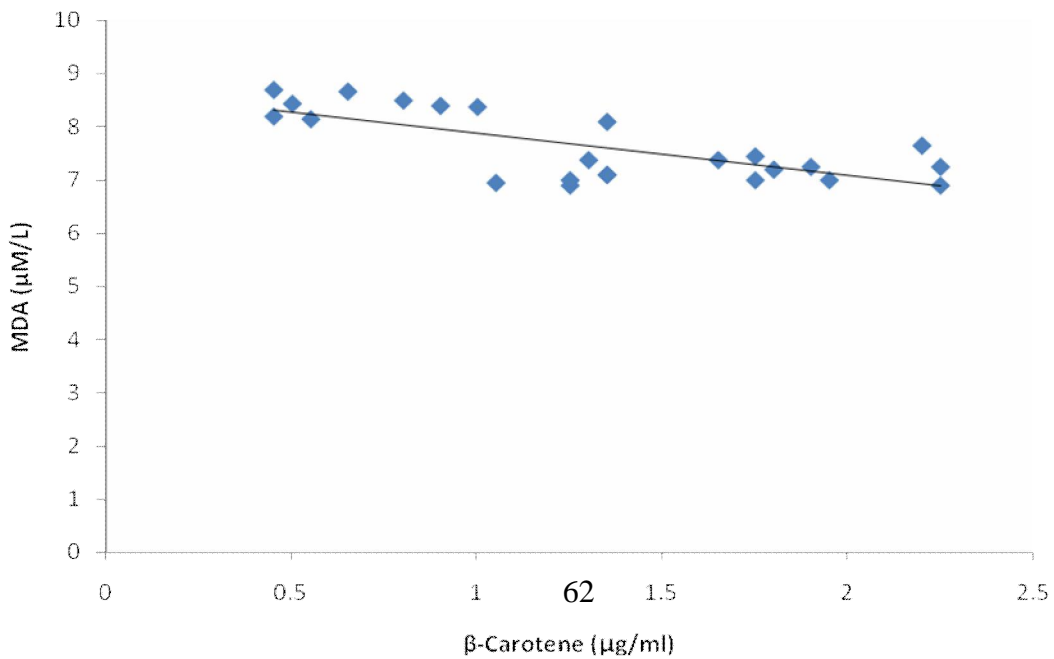
* Correlation is significant at the 0.05 level (2-tailed).

Figure 4 . Correlation between malondialdehyd (MDA) (µmol/L) and β- carotene(µg/ml) in asthmatic patients according to Age.

Figure 5 . Correlation between malondialdehyd (MDA) (µmol/L) and β- carotene(µg/ml) in asthmatic patients according to severity.

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دراسة علاقة إجهاد التأكسد المالونداي الديهايد مع بيتا – كاروتين في مصل دم مرضى الربو في محافظة ألبصره – العراق

ساهره غريب صياح
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الخلاصة:

صممت الدراسة على أساس تقييم العلاقة بين بيتا– كاروتين كمضاد للاكسدة مع المالونداي الديهايد (فرط الاكسدة) لمصل دم مرضى الربو، حيث تضمن البحث دراسة (50) حالة مرضية (24 ذكور, 26 إناث) حيث تم جمع العينات من مركز الحساسية والربو في محافظة ألبصره – العراق.

تم تقسيم المرضى على وفق مجموعة متغيرات تشمل العمر,الجنس, شدة المرض, التدخين, والتاريخ العائلي. مقارنة مع (50) حالة للأصحاء (24 ذكور , 26 إناث) كمجموعة سيطرة.

أظهرت نتائج الدراسة ارتفاعا معنويا في بيروكسيد الشحوم (فرط الأكسدة) المقاس كمالونداي الديهايد في مصل دم مرضى الربو ($p < 0.01$) مقارنة مع الاصحاء.

كذلك ارتفع مستواه معنويا ($p < 0.05$) بتقدم العمر وزيادة شدة المرض. بينما مع المرضى المدخنين والذين لديهم تاريخ عائلي موجب يكون ($p < 0.01$) .

كما أظهرت النتائج انخفاضا معنويا في مستوى بيتا – كاروتين كمضاد للأكسدة ($p < 0.01$) في مصل دم مرضى الربو مقارنة مع الأصحاء. ويزداد هذا الانخفاض معنوي ($p < 0.05$) بتقدم العمر وشدة المرض, بينما مع المرضى المدخنين والذين لديهم تاريخ عائلي ($p < 0.01$). علاوة على ذلك لوحظ ارتباط سالب بين مستوى بيتا – كاروتين مع مستوى المالونداي الديهايد في مجموعة المرضى مع هذه المتغيرات.

نستنتج من خلال نتائج هذه الدراسة بان عند المرضى المصابين بالربو درجة أعلى من تكوين الأنواع النشطة للأوكسجين والتــي تسبب إجهادا مؤكسدا وزيادة في بيروكسيد الشحوم مصحوبا بتناقص في مضادات الأكسدة مثل بيتا – كاروتين.