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ORIGINAL ARTICLE **OPEN ACCESS**

The Healing Properties of Dried Figs (*Ficus carica* L.)Against Oxidative Stress Caused By Ethyl Alcohol in Rats

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ABSTRACT
The aim of the study was determined to investigate the role of antioxidants on the lung and muscle tissues against oxidative stress induced by ethanol of dried fig (Ficus carica L.).The experimental animals were designed as Normal Control, 20% Ethanol, 10% FC and 10% FC + 20% Ethanol groups. Antioxidant defense system (ADS) and malondialdehyde (MDA) levels were evaluated on the lungs and muscle tissues after the application of rats, ethyl alcohol and dried fig for 50 days.Lipid peroxidation resulting from ethyl alcohol consumption was found to decrease MDA levels in both tissues with FC-supplemented diet.As a result, oxidative stress caused by ethanol is caused by fluctuations in antioxidant defense system components in the lung and muscle, and dry fig has no clear healing effect against these fluctuations.
Keywords*: Fig, Antioxidant defense system, Malondialdehyde, Rats, Oxidative stress, Ethanol*

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INTRODUCTION

Ever since the plants first appeared on the earth, they have been used in every aspect of life to make life easier for human beings.Ethno-botany has been used for many different purposes from past to present, and many other areas have been used, especially food, shelter, clothing and medicine. [1].One of the most popular uses of plants among populations is to consume them as food and use them for medical purposes. [2]. Not only agricultural societies, but also other societies have used wild plants and used them in a variety of fields, and their nutritional roles and health benefits have been demonstrated in a number of world-wide studies [3,4].

Plants of the genus Ficus are generally used both as an edible food and as a healing plant. *Ficus carica* L., especially , was reported to be better for metabolic disorders than for the other species.*Ficus carica* Moraceae belongs to the family and is called the most popular name "figs." Although F. carica fruits are used as a normal food, polysaccharides and polyphenols in fruits have been used as a daily source of healing[5].Some studies have reported that some compounds in the *F.carica* fruit have antispasmodic, antitumor, anti-inflammatory and antioxidant properties[6,7].However, the antioxidative, antidiabetic and antiobesitic effects of phytochemical components in F. carica cannot be fully understood. The phytochemicals of components such as 6-*O*-acyl-*β*-D-glucosyl-*β*-sitosterol in F.carica were reported as a potential cytotoxic agent and the latex of fig inhibited the proliferation of cancer cells [8,9].Recently, it has been reported that the use of *Ficus carica* in medical treatment of diseases such as anemia, cancer, stroke, diabetes, liver, skin is medically important [10,11].Fig is a fruit with very rich fibrous structure in terms of mineral and vitamin content. [12]. Fig, same to other fruits, is rich in sugar [13] and it has glucose, fructose and organic acid content in particular, but does not contain sodium and fat [12,14,15,16]. Besides, there are many components in the fig which are good for health; anthocyanins, proanthocyanidins, phenolic acids, flavonoids, flavonols, and flavanones [17].

Living things are subjected to oxidation due to various factors until the end of their lives.These oxidation factors are classified as endogenous (peroxides, transition metals etc.) by-products and exogenous (exposure, like UV and other radiations with higher energy and heat) by-products [18,19].Oxidative stress

are directly or indirectly cause hundreds of diseases. [20,21,22].To reduce or even eliminate the effects of reactive oxygen species and other free radicals, living organisms synthesize a variety of substances with strong antioxidant properties [18,23,24]. When the reaction mechanisms of these species are examined, they are similar to polymer stabilizers [22].

The idea first introduced by the food industry side is that the use of natural antioxidants as stabilizers instead of synthetic compounds has prevented the degradation of nutrients and beverages[24]. Antioxidants divided into two main groups, one of which is enzymatic and the other is non-enzymatic antioxidants[25]. Enzymatic antioxidants from antioxidant defense systems catalyze the conversion of direct reactive oxygen species to inactive compounds [26] or provide for the regeneration of non-enzymatic antioxidants[27].These substances can not be consumed in reactions when they are catalysts. Non-enzymatic antioxidants are composed of a large group of substances, which are classified in various forms in themselves. This group can remove the pro-oxidative transition metal ions, clean alkoxy or peroxy radicals or quench singlet oxygen [28]. Antioxidants can be found in the seeds, leaves and fruits of plants, in the shells of vegetables, because they produce high levels of antioxidants as a result of constant exposure to radiation. [29,30].

MATERIALS AND METHODS

Collection of plant material

The fresh fruit of *Ficus carica* L. were collected from its natural habitat in Binatifrom region of Batman, Turkey. The fruits of the plant collected were cleaned, dried under shade at room temperature for three weeks.

Preparation of foods

Ficus carica was powdered in the first stage and then had 10% FC content pellet feed prepared.

Experimental animals

24 Wistar albino female rats weighing 200-300 g were obtained from the Van Yuzuncu Yil University Experimental Animal Research Center.

This study lasted for a total of 50 days. 24 female rats were divided into four groups of six animals each.

Group I (Control): Nothing has been done in this group.
Group II (Ethanol): 20% ethanol application has made.
Group III (10% FC): 10% FC reinforced pellet feed has given.
Group IV (10% FC + 20% ethanol): The rats received 20% ethanol water and 10% FC supplemented feed.

The animals accilimatized at 20 ± 2 ◦C in a 12h daily light/dark cycles. Animals food and tap water were available ad libitium. This study procedures were approved by The Ethic Committee of the Van Yuzuncu Yİl University.

Chemicals

Thiobarbituric acid, butylated hydroxytoluene, trichloroacetic acid, ethylenediaminetetraacetic acid, reduced glutathione, metphosphoric acid, DTNB, Tris, CDNB, GSSG, Nicotinamide adenine dinucleotide phosphate, Potassium dihydrogen phosphate and sodium chloride oftechnical grade used in this study were obtained by Sigma Chemical Company, St. Louis, MO, USA. Reagents for antioxidant enzymes analysis were purchased from Randox Laboratories Ltd.

Preparation of tissues supernatant

At the end of the study, rats were anesthetized with 10% ketamine and sacrificed.The lungs and muscle tissues were removed washed in physiological saline(0.9% NaCl), and then kept at −78 ◦C until the day of the analysis.500 mg of tissue was weighed on a digital scale (Chyo Jl-180) and 5 mL of cold buffer was added. It was homogenized in an ultrasonic homogenizer for 3-5 minutes. The homogenate was centrifuged at 9500 rpm in a refrigerated centrifuge for 30 minutes at + 4 ° C supernatants from lung and muscle tissue were prepared for analysis.

Determination of biochemical parameters

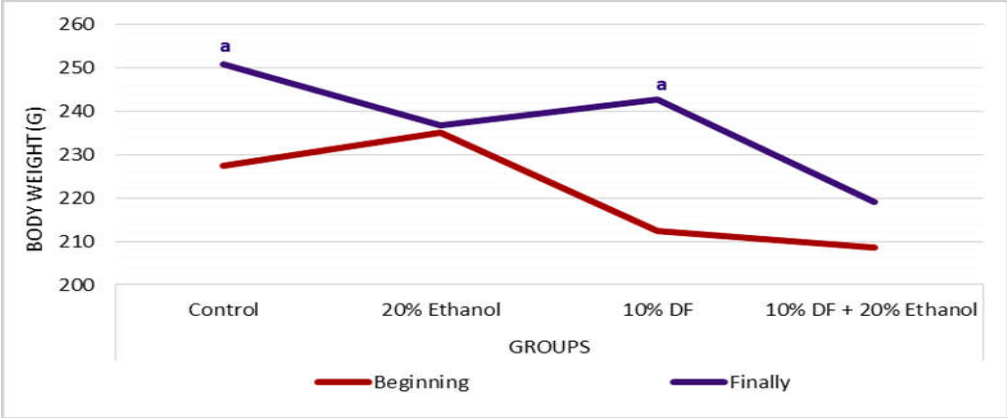
MDA in tissues is measured by entering a colored form with TBA [31]. GSH,reaction of sulfhydryl groups with DTNB in the fluid obtained from GSH using phosphate buffer in tissue supernatants was measured by the formation of the resulting yellow color. [32]. GST was found by following conjugation with glutathione at 340 nm with CDNB [33]. The GR was made according to the method applied by Calberg and Mannevrik [34].GPx catalyzes the reduction of cumene hydroperoxide in the presence of GSH, and the enzyme activity is determined by the absorbance change at 340 nm [35].The tissues SOD activity was measured using the method described by McCord ve Fridovich [36]. The activity of CAT enzyme was determined by spectrophotometric method based on the consumption of H2O2 at 37 ° C at 240 nm [37].

Statistical analysis

Mean and standard deviation (X ± SD), according to standard methods using Minitab packet program; The difference between the group averages was calculated using the One Way ANOVA-Tukey test and level atp ≤ 0.05.were considered statistically significant.

RESULTS

The body weight obtained from the rats during the experiment is given in Figure 1.



a: Significantly different from the beginning.

Figure 1.Weight gain in rats during the experiment (Mean).

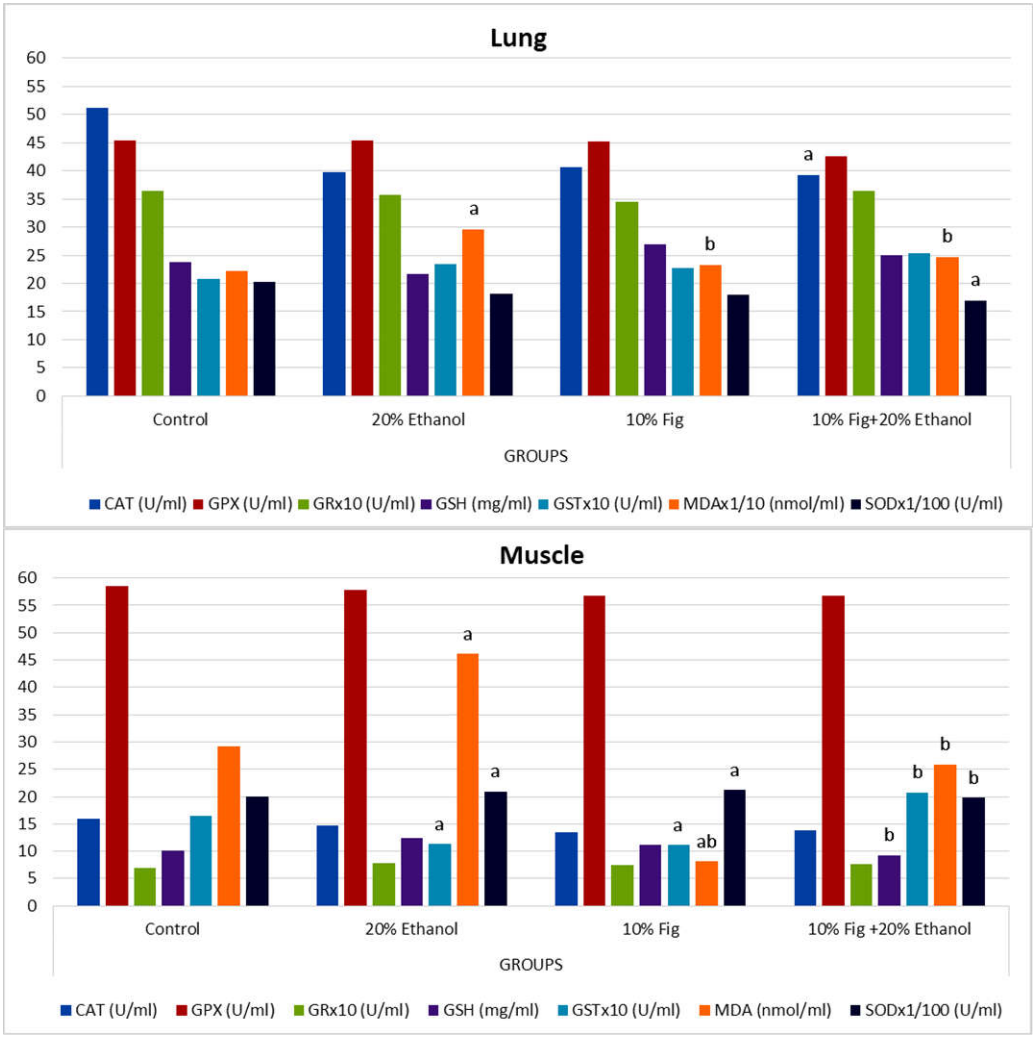
Following the exposure of experimental groups, the effects of ethanol and the FC-supplemented diet on lung and muscle index and antioxidative constituents were evaluated as ADS constituents and MDA content of lung and muscle tissues samples from control and treated rats. Although Table 1 shows that there is a significant increase in the MDA content of the ethanol group relative to the control group, the tissue MDA content in the 10% FC + 20% ethanol group ($p \leq 0.05$) are significantly reduced compared to the 20% ethanol group ($p \leq 0.05$).That is, it can be said that fig is good against lipid peroxidation which is caused by oxidative stress.Ethanol caused fluctuations in the components of the antioxidant defense system at a level of the oxidative stress condition in rats (Table 1). When we looked at the levels of CAT and SOD in lung tissue, it was determined that10% FC + 20% Ethanol group was statistically significant decrease compared to control. Muscle tissue, it was found that there was a statistically significant decrease with respect to CAT level 20% Ethanol and 10% FC group and there was a statistically significant increase in 10% FC + 20% Ethanol group compared to. Furthermore, when we compared the rats with the control and the 20% ethanol group, there was a fluctuation in the comparisons of GPx, GST, SOD and ADS components.In other words, since ethanol may be the source of oxidative stress in rats, it may cause certain fluctuations in antioxidant defense systems, but a full improvement effect can have not determined even though the dried fig supplemented could have been a positive effect on. (Table 1 and Figure 2).

Table 1. Detection of MDA and ADS components in rat tissue of experimental groups (Mean ± SD).

		GROUPS			
Parameters	Tissues	Control	20% Ethanol	10% Fig	10% Fig +20% Ethanol
CAT (U/ml)	Lung	51,18±7,63	39,83±7,32	40,67±6,38	39,27±7,87[a]
	Muscle	15,91±5,40	14,70±4,11	13,40±2,64	13,87±2,13
GPX (U/ml)	Lung	45,43±2,93	45,35±2,17	45,17±1,86	42,53±3,68
	Muscle	58,54±0,51	57,84±1,37	56,80±2,31	56,77±1,61
GR (U/ml)	Lung	3,65±0,57	3,58±0,35	3,45±0,34	3,64±0,14
	Muscle	0,69±0,08	0,78±0,06	0,75±0,04	0,77±0,03
GSH (mg/ml)	Lung	23,78±3,69	21,75±2,56	26,91±5,64	25,09±3,88
	Muscle	10,10±0,84	12,42±1,69	11,14±2,58	9,31±0,61[b]
GST (U/ml)	Lung	2,08±0,46	2,34±0,32	2,27±0,23	2,54±0,43
	Muscle	1,65±0,25	1,13±0,22[a]	1,11±0,14[a]	2,07±0,49[b]
MDA (nmol/ml)	Lung	222,14±30,43	295,85±5,92[a]	232,83±29,07[b]	246,75±35,49[b]
	Muscle	29,23±5,38	46,07±6,59[a]	8,10±2,89[ab]	25,91±11,74[b]
SOD (U/ml)	Lung	2019,27±128,75	1809,77±138,25	1805,53±121,22	1695,19±285,77[a]
	Muscle	2000,56±48,91	2087,72±54,28[a]	2118,19±49,16[a]	1977,47±51,71[b]

a: The difference according to the control group is statistically significant ($p<0.05$).

b: The difference according to the %20 ethanol group is statistically significant ($p<0.05$).



a: The difference according to the control group is statistically significant ($p<0.05$).
b: The difference according to the %20 ethanol group is statistically significant ($p<0.05$).

Figure 2. Detection of MDA and antioxidant enzyme activityin experimental group(Mean ± SD).

DISCUSSION

Excessive ethanol application to animals is designed to determine physiological changes and blood alcohol levels comparable to human binge drinking[38,39,40].In experimental models, both acute and chronic ethanol treatments were applied to determine various aspects of oxidative stress induced by ethanol. The consequence of ethanol metabolism is increased ROS sources, such as xanthine oxidase,cytochrome p450 and NADPH oxidase; these reactions can lead to lipid peroxidation and can be demonstrated by MDA prediction. [41,42].

Studies have shown that consumption of ethanol leads to free radical formation and increases oxidative stress and lipid peroxidation [31,43-48, 49]. The increase in oxidative stress due to ethanol consumption is thought to be caused by ethanol metabolism [40]. After depletion of some xenobiotics, lipid peroxidation is elevated and then dismutation can be converted to reactive OH after the production of superoxide, which again produces singlet oxygen and H_2O_2. Radicals such as oxygen and OH have the potential to initiate free radical chain reactions of lipid peroxidation.The presence of OH radicals in the tissues can initiate lipid peroxidation [53], and MDA is a major oxidation product of polyunsaturated fatty acids. Morever, increased levels of MDA in the tissue are considered to be indicative of lipid peroxidation[50]. In our study, we observed that there was a significant increase in MDA level due to ethanol consumption but there was no significant increase or decrease in SOD, GR, GPx, GST and GSH levels.However, in the FC-consuming groups, it was observed that the MDA level was lowered but not the expected positive effect on other parameters (Table 1). The effects on the dry figs MDA and ADS parameters are not fully understood at this time. Likewise, increased oxidative stress due to ethanol

consumption may have led to changes in the activities of antioxidant defense systems and MDA levels in tissues.

Figs are rich in antioxidant substances [12,13,51,52,53] only contribute both to make or become better human health and to the property of the fruit, because they are highly affected by the sensory properties such as color, flavor and bitterness and shrinkage [58, 59]. The health characteristics of the figs have led to several investigations on polyphenol content and antioxidant activity [12,17,51,53,54, 55, 56] and their functional properties.When the total antioxidant activity (TAA) of fig was found to be in both the skin and the meat, it was found that it contributed to the total antioxidant activity such as flavonoids and vitamins in fruit and vegetables because it contained hydrophilic (H-TAA) and lipophilic compounds[57].

As shown in Table 1, this study has shown that FC may have a therapeutic role on lipid peroxidation in rats. This is our observation that, as a result of in vivo FC supplementation, the MDA concentration in tissues was different from the concentration of the ethanol-exposed group. According to the results of the study we conducted, the concentration of MDA in the tissues of the rats in the ethanol treated group increased statistically, but MDA components in the FC-reinforced group decreased statistically compared to the ethanol group. At present, the effects of Ethanol and FC reinforcement on rats are not fully understood. However, increased ROS factor due to ethonol consumption may have resulted in increased MDA concentration in tissues [58, 59].Therefore, consumption of foods that can help the antioxidant defense system to cope with parameters that increase oxidative stress such as ethanol may accelerate the adaptation of organisms. As a result, since ethanol may be the source of oxidative stress in rats, it may cause certain fluctuations in antioxidant defense systems, but a full improvement effect can have not determined even though the dried fig supplemented could have been a positive effect on.

CONFLICTS OF INTEREST STATEMENT

The authors have no conflict of interest to declare within this article.

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REFERENCES

1. Abbasi A M, Khan MA, Khan N, ShahMH (2013) Ethnobotanical survey of medicinally important wild edible fruits species used by tribal communities of Lesser Himalayas–Pakistan. *Journal of Ethnopharmacology.* 148, 528–536.
2. Andreas CH, Paraskeva d, Della A, Giusti ME, De Pasquale C, Lenzarini C, et al. (2008) Wild and semi domesticated food plant consumption in seven circum-Mediterranean areas. *International Journal of Food Science and Nutrition.* 59, 383–414.
3. Pardo-de-Santayana M, Tardío J, Blanco E, Carvalho A, Lastra J, San Miguel E, et al. (2007) Traditional knowledge of wild edible plants used in the northwest of the Iberian Peninsula (Spain and Portugal): A comparative study. *Journal of Ethnobiology and Ethnomedicine*.7, 3-27.
4. Kirschweng B, Tátraaljai D, Földes E, Pukánszky B(2017) Natural antioxidants as stabilizers for polymers. *Polymer Degradation and Stability*.*145*, 25-40.
5. Tian J, Zhang Y, Yang X, Rui K, Tang X, Ma J, Chen J, Xu H, Lu L, Wang S(2014) *Ficus carica* polysaccharides promote the maturation and function of dendritic cells. *International Journal of Molecular Science.* 15,12469-79.
6. Solomon A, Golubowicz S, Yablowicz Z, Grossman S, Bergman M, Gottlieb HE, Altman A, Kerem Z, Flaishman MA(2006) Antioxidant activities and anthocyanin content of fresh fruits of common fig (*Ficus carica* L.). *Journal of Agricultural and Food Chemistry.*54, 7717-23.
7. Yang XM, Yu W, Ou ZP, Ma HL, Liu WM, Ji XL (2009) Antioxidant and immunity activity of water extract and crude polysaccharide from *Ficus carica* L. fruit. *Plant Foods Human Nutrition.*64,167-73.
8. Lazreg Aref H, Gaaliche B, Fekih A, Mars M, Aouni M, Pierre Chaumon J, Said K (2011) In vitro cytotoxic and antiviral activities of *Ficus carica* latex extracts. *Natural Product Researh.*25,310-19.
9. Rubnov S, Kashman R, Rabinowiz M, Schlesinger M, Mechoulam R (2001) Suppressors of cancer cell proliferation from fig (Ficus carica) resin: isolation and structure elucidation. *Journ of Natural Product.* 64, 993-996.
10. Badgujar SB, Patel VV, Bandivdekar AH, Mahajan RT (2014) Traditional uses phytochemistry and pharmacology of Ficus carica: a review. *Pharmaceutical Biology*52,1487-1503.
11. Mopuri R, Ganjayi M, Meriga B, Koorbanally NA, IslamM S (2017) The effects of *Ficus carica* on the activity of enzymes related to metabolic syndrome. *Journal of Food and Drug Analysis*.26(1),201-210
12. Veberic R, Colaric M, Stampar F (2008) Phenolic acids and flavonoids of fig fruit (*Ficus carica* L.) in the northern Mediterranean region. *Food Chemistry*. 106, 153–157.
13. Crisosto CH, Ferguson L, Bremer V (2011) Fig (*Ficus carica* L.). In: Yahia, E.E. (Ed.), Posharvest Biology and Technology of Tropical and Subtropical Fruits. Cambridge, UK, pp. 134–158.
14. Crisosto CH, Bremer V, Ferguson L, Crisosto GM (2010) Evaluating quality attributes of four fresh fig (*Ficus carica* L.) cultivars harvested at two maturity stages. *Hortscience.* 45, 707–710.

15. Genna A, De Vecchi P, Maestrelli A, Bruno M (2008) Quality of 'Dottato' dried figs grown in the Cosenza region, Italy. A sensory and physical–chemical approach. Acta Hortic.*International Society for Horticultural Science.* 798, 319–323.
16. Veberic R, Mikulic-Petkovsek M (2016) Phytochemical composition of common fig (*Ficus carica* L.) cultivars. In: Simmonds, Preddy (Eds.), Nutritional Composition of Fruit Cultivars. American Press, UK, pp. 235–255.
17. Pereira C, López-Corrales M, Serradilla MJ, del Carmen Villalobos, Ruiz-Moyano S, Martín A (2017) Influence of ripening stage on bioactive compounds and antioxidant activity in nine fig (*Ficus carica* L.) varieties grown in Extremadura, Spain. *Journal of Food Composition and Analysis. 64*, 203-212.
18. [Brewer MS (2011) Natural Antioxidants: sources, compounds, mechanisms of action, and potential applications, *Comprehensive Reviews in Food Science and Food Safety*. 10 (4), 221-247.
19. Riley PA (1994) Free Radicals in Biology: oxidative stress and the effects of ionizing radiation.*International Journal of Radiation Biology*. 65 (1), 27-33.
20. Gutteridge JMC, Halliwell B (1993) Invited review free radicals in disease processes: a compilation of cause and consequence.*Free Radical Research Communication.* 19 (3), 141-158.
21. Halliwell B, Gutteridge JMC, Cross CE(1992) Free radicals, antioxidants, and human disease: where are we now? *Journal of Laboratory and Clinical Medicine.* 119 (6), 598-620.
22. Pisoschi AM, Pop A(2015) The role of antioxidants in the chemistry of oxidative stress: a review.*European Journal of Medicinal Chemistry.* 97, 55-74.
23. Agati G, Matteini P, Goti A, Tattini M (2007) Chloroplast-located flavonoids can scavenge singlet oxygen.*New Phytologist.* 174 (1), 77-89.
24. Milda E,(2015) EmbuscadoSpices and herbs: natural sources of antioxidants e a mini review.*Journal of Functional Foods.*18, 811-819.
25. Rahman K (2007) Studies on free radicals, antioxidants, and co-factors.*Clincal Interventions in Aging.*2 (2), 219-236.
26. Mates JM, Perez-Gomez C (1999) I. Nunez de Castro, Antioxidant enzymes and human diseases.*Clinical Biochemistry.* 32 (8), 595-603.
27. Gamble PE, Burke JJ (1984) Effect of water stress on the chloroplast antioxidant system.*Plant Physiology.*76, 615-621.
28. Martysiak- Zurowska D, Wenta W (2012) A comparison of abts and DPPH methods for assessing the total antioxidant capacity of human milk.*Acta Scientiarum Polonorum Technologia Alimentaria*. 11 (1), 83-89.
29. Majer P, Neugart S, Krumbein A, Schreiner M, Hideg E (2014) Singlet oxygen scavenging by leaf flavonoids contributes to sunlight acclimation in Tilia platyphyllos.*Environmenta and Experimental Botany.* 100, 1-9.
30. Kirschweng B, Tátraaljai D, Földes E, Pukánszky B (2017) Natural antioxidants as stabilizers for polymers. *Polymer Degradation and Stability*. *145*, 25-40.
31. Jain SK, McVie R, Duett J, Herbst JJ (1989)Erythrocyte membrane lipid peroxidation and glycosylated hemoglobin in diabetes. *Diabetes.*38, 1539–1543.
32. Beutler E, Dubon O, Kelly BM (1963) Improved method for the determination of blood glutathione.*Journal of Laboratory and Clinical Medicine*. 61, 882–888.
33. Mannervik B, Guthenberg C (1981) Glutathione transferase (human placenta). *Methods Enzymol*. 77, 231–235.
34. Carlberg I, Mannervik B (1975)Purification and characterization of the flavoenzyme glutathione reductase from rat liver. *Journal of Biological Chemistry.* 250, 5475–5480.
35. Paglia DE, Valentine WN (1967)Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *Journal of Laboratory and Clinical Medicine.*70, 158.
36. McCord JM, Fridovich I (1969)Superoxide Dismutase An Enzymıc Functıon For Erythrocupreın (Hemocupreın).*Journal of Biological Chemistry.*244, 6049–6053.
37. Aebi H (1974) Methods of Enzymatic Analysis, in: H.U. Bergemeyer (Ed.).*Academic Press, New York*.
38. Kanan M, Wang L, Kang YJ (2004) Myocardial oxidative stress and toxicity induced by acute ethanol exposure in mice.*Experimental Biology and Medicine*. 229, 553–559.
39. Zhou Z, Sun X, Kang YJ (2002) Metallothionein protection against alcoholic liver injury through inhibition of oxidative stres. Experimental Biology and Medicine. 227, 214–222.
40. Zhou Z, Wang L, Song Z, Lambert JC, McClain CJ, Kang YJ (2003) A critical involment of oxidative stress in acute alcohol induced hepatic TNF-production. American Journal of Pathology. 163, 1137–1146.
41. Lucas DL, Brown RA, Wassef M, Giles TD (2005)Alcohol and the cardiovascular system.*Journalof the American Collage of Cardiology.* 45, 1916–1924.
42. Hassoun EA, Stohs SJ (1995)Comparative studies on oxidative stress as a mechanism for the fetotoxic of TCDD, endrin and lindane in C57BL/6J and DBA/2J mice. *Teratology.* 51, 186–192
43. Aykac G, Uysal M, Yalcin AS, Kocak-Toker N, Sivas A, Oz H (1985)The effect of chronic ethanol ingestion on hepatic lipid peroxide, glutathione, glutathione peroxidase and glutathione transferase in rats*Toxicology.* 36, 71–76.
44. Sonde V, D'souza A, Tarapore R, Pereira LL, Khare MP, Sinkar P, Krishnan S, Rao CV (2000)Simultaneous administration of diethylphthalate and ethyl alcohol and its toxicity in male Sprague-Dawley rats. *Toxicology.*147, 23–31.
45. Yurt B, Celik I (2011)Hepatoprotective effect and antioxidant role of sun, sulphited-dried apricot (Prunus armeniaca L.) and its kernel against ethanol-induced oxidative stress in rats. Food and Chemical Toxicology. 49, 508–513.

46. Dogan A, Celik I (2012)Hepatoprotective and antioxidant activities of grapeseeds against ethanol induced oxidative stress in rats.*British journal of Nutrition.*107, 45–51.
47. Turan A, Celik I (2016) Antioxidant and hepatoprotective properties of dried fig against oxidative stress and hepatotoxicity in rats. *International journal of biological macromolecules*. *91*, 554-559.
48. Bati B, Celik I, Dogan A (2015) Determination of hepatoprotective and antioxidant role of walnuts against ethanol-induced oxidative stress in rats. *Cell biochemistry and biophysics. 71*(2), 1191-1198.
49. Halliwell B (1996) Oxidative stress, nutrition and health. Experimental strategies for optimization of nutritional antioxidant intake in humans. *Free Radical Research*. 25, 57–74.
50. Freeman BA, Crapo JD (1981) Hyperoxia increases oxygen radical production in rat lung and lung mitochondria. *Journal Biological Chemistry.* 256, 10986–10992.
51. Solomon A, Golubowicz S, Yablowicz Z, Grossman S, Bergman M, Gottlieb HE, Altman A, Kerem Z, Flaishman MA (2006) Antioxidant activities and anthocyanin content of fresh fruits of common fig (*Ficus carica* L.). *Journalof Agriculture and Food Chemistry*. 54, 7717–7723.
52. Tanwar B, Andallu B, Modgil R (2014) Influence of processing on physicochemical, nutritional and phytochemical composition of *Ficus carica* L. (fig) products. *Agriculture* Research Communication Centre. 33 (1), 37–43
53. Vallejo F, Marín JG, Tomás-Barberán FA (2012) Phenolic compound content of fresh and dried figs (*Ficus carica* L.). *Food Chemistry.* 130, 485–492.
54. Lee SK, Kader AA (2000) Preharvest and postharvest factors influencing vitamin C content of horticultural crops. *Postharvest Biology and Technology*. 20, 207–220.
55. Piga A, Del Caro A, Milella G, Pinna I, Vacca V, Schirru S, (2008) HPLC analysis of polyphenols in peel and pulp of fresh figs. *Acta Horticulturae.* 798, 301–306.
56. Dueñas M, Pérez-Alonso JJ, Santos-Buelga C, Escribano-Bailón T (2008) Anthocyanin composition in fig (*Ficus carica* L.). *Journal of Food Composition Analysis.* 21, 107–115.
57. Valero D, Serrano M (2010) Fruit ripening. Postharvest Biology and Technology for Preserving Fruit Quality. *CRC Press*, Boca Raton, FL,
58. Smith GJ, Litwack G (1980)Roles of ligandin and the glutathione S-transferases in binding steroid metabolites, carcinogens and other compounds Rev. *Biochem. Toxicol.* 2, 1–47.
59. Vinson JA, Zubik L, Bose P, Samman N, Proch J(2005)Dried Fruits: Excellent *in Vitro* and *in Vivo*Antioxidants.*Journal of American Collage of Nutrition*. 24, 44–50.

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