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Uğur GÖĞÜŞ

Participated in "2nd International Congress on Food and Nutrition" held between 24 and 26 October 2007 in Istanbul, TURKEY.

M. Önder YETİŞ
TÜBİTAK MRC
Acting President

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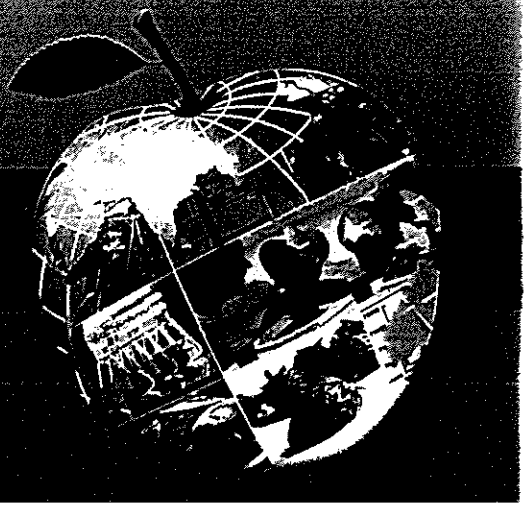
2nd INTERNATIONAL CONGRESS on FOOD and NUTRITION

24 - 26 October 2007

Istanbul, TURKEY

FOOD For FUTURE

PROGRAMME BOOKLET



13:30-15:30	**Session 11: Functional Foods and Nutraceuticals-3** **Chair: Fred Brouns,** Department of Human Biology, University of Maastricht, Belgium **Co-Chair: Ram Reifen,** School of Nutritional Sciences, The Hebrew University of Jerusalem, Israel
13:35-14:05	*Phytopharmaceuticals in cancer chemoprevention* **Debasis Bagchi,** Department of Pharmacy Sciences, Creighton University Medical Centre, USA
14:05-14:25	*Isolation of milk serum proteins from whey powder by gel chromatography and protease treatment and determination of their antioxidant activity* **A. Suha Yalçın,** School of Medicine, Marmara University, Turkey
14:25-14:45	*Copper(II)– and iron(III) – based antioxidant capacity assays for food with enhanced selectivity and sensitivity* **Reşat Apak,** Department of Chemistry, Istanbul University, Turkey
14:45-15:05	*Effect of tea grades on the content of polyphenols, alkaloids and antioxidant activity of Turkish black teas* **Nihal Türkmen,** Department of Food Engineering, Ankara University, Turkey
15:05-15:25	*Bioactive compounds naturally present in olive and virgin olive oil and their physiological effects* **Dilşat Bozdoğan Konuşkan,** Department of Food Engineering, University of Çukurova, Turkey
15:30-16:00	**COFFEE BREAK & POSTER SESSION**
16:00-18:00	**CLOSING CEREMONY & POSTER AWARD WINNERS**

KOCATEPE CONFERENCE ROOM

10:00-10:30	**COFFEE BREAK & POSTER SESSION**
10:30-12:30	**Session 12: Nutrition, Health and Well Being-2** **Chair: Özgen Doğan,** School of Medicine, Columbia University, USA **Co-Chair: Ayşe Baysal,** Emeritus Professor, Turkey
10:35-11:05	*Diet and cancer prevention: Nutrigenomic perspectives* **Yong-Joon Surh,** College of Pharmacy, Seoul National University, South Korea
11:05-11:25	*The innate immune system: Raising the barrier to infection with diet* **Paul Clayton,** Oxford Brookes University, UK
11:25-11:45	*Coffee, a long history of use with recognised protective health benefits, potentially supported by different antioxidant mechanisms* **Christophe Cavin,** Nestlé Research Centre, Switzerland
11:45-12:05	*Alcohol and health: Is it an antioxidant or a poison?* **Uğur Göğüş,** Vocational School of Higher Education, Middle East Technical University, Turkey
12:05-12:25	European food information resource - network (EuroFIR) **Gül Löker,** TÜBİTAK MRC Food Institute, Turkey

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POSTER AWARD JURY

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Juri eleştirilerinden bir tanesi, bu sunumun metninin dosyada bulunmaması şeklindedir ki savunmamda böyle bir şart olmadığını, verilen sertifikanın ve Proceeding kayıtlarında abstract mevcudiyetinin yeterli kanıtlar olduğunu belirtmiştim. Gene de değerlendirmenin bilimsel duyarlılığını artırması ve emeğimin anlaşılmasına yardımcı olacağı düşüncesiyle eski dosyada eksik olan metni ilişiğe ekledim. En derin saygılarımla bilgilerinize sunarım.

Alcohol and health:Is it an antioxidant or a poison?

Dietary ethanol which is taken nto body by the means of food products is first absorbed in stomach and intestine and then transferred into blood by 80% while 2% is enhaled by lungs and upto 20% accumulates in stomach. The blood ethanol is converted into lactate and lactate is converted into pyruvate. The end product pyruvate is converted into uric acid by gut and excreated out of body by the means of urin. On the other hand, blood ethanol, blood ethanol participates in the structure of acetyl-CoA which is a compound vitgally acts in energy production. Alcohol dehydrogenase, the enzyme in blood and tissues produces NADH, NAD, acetaldehyde (oxygen radicals), new antigens, fibril compounds and some end products formed by lipid oxidation. The reason for the explanation of alcohol metabolism with its initial details is necessary to be able to understand its effects on body since these effects are closely related to the metabolism. As can clearly be understood, ethanol taken into body by the means of food, first accumulates upto 80 % in blood and enters into the structure of acetyl-CoA, the first compound to produce energy in body which means that alcohol gives energy. But its energy potential may cause a great energy loss in the long run. People who consumes alcohol regularly in moderate or plenty amounts always talk about its energy benefit. While ethanol contributes to acetyl-CoA reproductions and energy production, it is constantly converted into acetaldehyde which triggers oxidation and free toxic peroxy radical production because it forms oxygen radicals. The worst side is the formation of fibril compounds which defect tissues especially lungs and liver and new antigens which weak immun system by these free toxic radical compounds.Lactat which is produced by ethanol in blood is converted into pyruvate and uric acid which results in increase of uric acid concentarion in blood increases the risk forthe formation of gout disease while increasing the risk of renal failure because uric acid is one of the worse compounds which can easily calcify the filtering vessels of kidneys.

Uric acid crystalls which accumulates in blood produced by blood ethanol shows its adverse effect not only on kidneys but also on brain that bran is the initial organ which is most adversely affected by dietary ethanol.

The convertion of dietary ethanol into uric acid, antigens, free toxic peroxy radical compounds, oxygen radicals and fibril compounds also exist in the cells of stomach which covers the surface of stomach. Hence, stomach cells are defected, wounded by the effect of these harmful substances while some of them die. The wounded, ,inflammated stomach surface cells which are formed by the effect of ethanol and its decomposition in stomach cells is the main reason for gastrit and ulcer formation due to ethanol consumption.

The brain damage due to alcohol shows its adverse effects first with relieving anxiety followed by slur speech. Coordination in walking is reduced, judgment is impaired and finally uninhibited behaviour is encouraged. Ethanol also triggers the production of diuretic hormon while inhibiting antidiuretic hormon which sometimes may cause strong dehydrtion in body. Note that water is the natural media in each cell for the process of

oxidation and energy production by oxidation. Cells can not produce oxidation and energy production without energy and die as a natural result of dehydration. Briefly, diuretic effect of alcohol will have killed many cells in our body due to energy loss and dehydration.

Two initial and critic questions which are commonly discussed in alcohol issue are 1. what amount is moderate ? and 2. what amount is toxic?. Although many of people uses the terms of 'moderate amounts', none of us knows these moderate amounts.!

Body which weigs 68 Kg (150 pounds) metabolizes 7-14 g alcohol (100-200 mg wine) per hour. Higher amounts cause intoxication. Here below (slight) shows the ethanol (g)/glass and the metabolised ethanol (g) amounts per hour (the second column) with Kcal levels.

According to the data, not only the dietary ethanol (g) amount per glass and metabolized alcohol amount (g) per hour but also the type of alcoholic bevergae and the glass size are the important factors in the evaluation of alcohol toxicity. For example Manhattan, Martini and old-fashioned which are commonly consumed by using champain glass gives more than 14 g alcohol per hour into blood while others are lower than this critic limit.

Another important factor in alcohol toxemia is the residue of ethanol as mg per ml. The table below (slight) shows the mg residue concentration per ml and the symptoms which are observed in those concentrations. It seems very clear that the toxicity and the severity increases with the increase in alcohol residue concentration per ml of blood. For example, 50 ml ethanol residue/ml can be metabolized within 2 or 3 h while no symptoms is observed but 200-250 mg/ml, 300,350 and 500 mg/ml can be metabolized over 6-10 hours and days while increasingthe risk of lethargic behaviour and coma.

Another important factor which affects the toxicity of alcohol is the time period and the daily amount of alcohol consumption. 15 year alcohol consumption as approximately 80 g/day has been claimed as a reason to form cirrhossis (the defects on liver cells and the loss of these cells followed by an inflammation o toxicity). In fact, the dietary ethanol amounts do not need to be higher to be able to cause toxic effects. Even 20 and 40 g daily alcohol consumptions may cause cirrhosis in women and men, respectively.

The toxicity of liver by ethanol first decrease the concentration of thiamin coenzyme, an enzyme which regulates the production of thiamin. Thiamin, an important member of B complex, enters into the structure of enzymes which are responsible for nerve cell and blood cell proliferations. Especially, nerve cells decrease in number and become susceptible to the defects caused by free toxic radicals and other harmful compounds followed by thiamin deficiency. Retinol, a vitamin A substance, is mobilized from liver bound to retinol binding protein (RBP). But, the damage in liver by ethanol directly decreases the concentration of retinol binding protein which inturn decreases vitamin A concentration and may cause a vitamin A deficiency. The vitamin is vitgally important in boosting immun system and also for visual health. Cornea, the layer which covers the surface of eye, is covered by a layer made by a solid particle (keratin) in vitamin A

deficiency, the thickness of the layer increases which may not be treated. The disorder is called as Xeroftalmia. Briefly, ethanol toxemia may also cause to visual health problems like cataract and Xeroftalmia by causing retinol and vitamin A deficiency. Another important disorder due to ethanol toxicity is Pellegra. Ethanol decreases the absorption level of niacin and may cause niasin deficiency. Pellegra which is caused by niacin deficiency is an important disease since blood and muscle cells decrease and energy loss may appear.

The liver damage in ethanol toxicity shows its one of adverse effects on bones and muscles since the defected liver tissue can not convert the precursor of vitamin D, cole and ergocalciferol, into vitamin D. Thus, vitamin D deficiency is inevitable in Ethanol toxicity. Vitamin D is an essential vitamin which enhances Ca absorption through bowel mucosa. Anyone who aims Ca supplementation by the means of tablets shoud pay attention that Ca should have been exist in the tablet as combined with vitamin D. Always Ca and vitamin D combined tablets as in one formula should have been preferred since Ca bioavalibility is the highest as combined with vitamin D. Ca can not be bound to bone surface and enter into muscle cells in the case of vitamin D deficiency. In conclusion, ethanol causes vitamin D deficiency and vitamin D deficiency causes Ca deficiency, osteoporosis (density loss in bone) and osteomalacia (density loss in both bones and muscles) and fibromyalgia (a chronic condition characterized by widespread pain in muscles, ligaments, tendons as well as fatigue and multiple tender points-places on body where slight pressure causes pain.

In the case of regular alcohol consumption, not only vitamin D, members of vitamin B complex, vitamin A but also vitamin C (ascorbic acid) like many others can not be absorbed through bowel mucosa. Instead, there is another energy source, ethanol, which can easily be absorbed instead of vitamines and minerals. Hence, alcohol is always absorbed first, in front of vitamin C and vitamin C deficiency forms. It should be noted that the damage in kliver caused by ethanol is another important contributor to viaminm C deficiency since this important vitamin is produced in liver. Vitamin C is a strong, the most versatile vitain. It increases seratonin (relaxing hormon) and may protect against stress and depression, manic depression, anxiety,. It increases dopamin, the hormon responsible for the bone and muscle coordination) and may protect against Parkinson and Alzheimer. The vitamin decreases cholesterol and bad cholesterol while increasing good cholesterol (HdL), dilutes blood and increases the thickness of the antioxidant protective barrier around the cell membranes. Briefly the vitamin is a strong antiaging compound and an essential vitamin to improve cardiovascukar health. Vitamin C also reduces TNF (Tumor Necrosis Factor) which significantly decreases the risk for tumor formation. Vitamin C also enters into the enzymes which are responsible for dental restoration, collogen (the connective tissue) and hydroxyprolin (the precursor of collogen). Finally, it increases the production of melathonin, the hormon and neurotransmitter which is tresponsible for sleeping process. Therefore, the vitamin is an essential for the protection against Parkinson, Alzheimer, depression, cardiovascular diseases and disorders like stroke and hypertension, anxiety, insomnia, bone and muscle disorders, immun and dental disorders. This important vitamin is also depleted by the effect of ethanol and one

or some of the mentioned diseases and disorders may form due to dietary ethanol consumption.

Dietary ethanol interrupts the turnover of glutathion, the antioxidant protective layer which protects it against many harmful compounds such as cholesterol, salt, sugar, fat, bad cholesterol and homocytein by surrounding their membranes. Glutathion decrease shows its effect on body as aging. For this reason, ethanol taken into body by the means of foods has got an aging effect in the long run rather than antiaging.

Dietary ethanol is converted into lactate, pyruvate and then acetaldehyde and acetate. Finally, acetate is converted into fatty acids to build up trigliserids (the comlex fatty acids). No matter how dietary ethanol in low or moderate intakes cause blood thinning and cholesterol-bad cholesterol lowering effect, on the other hand, it slowly increases the serum trigliseride concentration which inturn is the initial cause of liver, heart, kidney and pancreas fattening.

One of the hazardous effect of dietary ethanol is on kidneys. Some media claims that the blood thinning and cholesterol and bad cholesterol reducing effect make some protection of kidneys which may be correct. But, acetate and acetaldhyde which accumulate in blood and exceed standard levels are directly converted into uric acid. Uric acid increase is an initial factor in the formation and progress of renal failure since uric acid crystalls can easily accumulate on the walls of filtering membranes of kidneys.

The toxicity of dietary alcohol changes according to sex that the thickness and surface area of gastric mucosa, a barrier for preventing alcohol entrance, is more than in women which is the initial reason for the higher tolerance of men to alcohol. For this reason, the equal amount of dietary alcohol may cause more toxic and harmful effects on female body.

The damage caused by dietary ethanol on Fat Binding Proteins (FBP) in the cytosol of hepatocyctes (liver cells) increases the free fat globules in liver tissue since FABPs decomposes and metabolize complex fat globules. The free fat globules transmitted by FABPs into liver tissue causes a great damage in liver.

Alcohol is also a great contributes in the formation of malnutrition. 1 g of carbohydrate or 1 g of protein gives 4 Kcal energy while the same amount of fat or oil gives 9 Kcal, the enery amount of more than 2 times of the equal amount of carbohydrate or protein. This excess energy coming from fats and oils sometimes may not be excreated out and accumulate in body as in fat form. When the energy status of dietary ethanol is concerned, 1 g of dietary ethanol gives 7 Kcal of energy potential which is very close to the energy potential of fats or oils. People who consumes alcohol regularly, no matter how they are in low or moderate amounts, are used for supplying energy and bodies of such people accept alcohol as the initial and the easiest energy source. Because dietary ethanol gives a high rate of energy easily, proteins, carbohydrates, fats and oils, vitamines which are very close to fats in structure are not properly absorbed through bowel mucosa.

As previously mentioned above, ethanol is converted into acetaldehyde and toxic radicals which are strong inflammatory agents are produced from these toxic radicals. This process shows itself in a different manner in liver tissue that microsomal ethanol oxidative system in liver synthesis collogen by using these toxic radicals. Hence, collogen starts to accumulate in liver among liver cells which results in liver failure. The mentioned disorder is called as 'liver fibrosis'. The disease not only defects the liver cells but also mitochondria, the energy producing organelles in these cells. The result is inevitably the energy depletion and fatigue. Liver damage caused by dietary ethanol is an important reason for the energy depletion and chronic fatigue which is often complained by regular alcohol users. The picture below (slight) shows the tissue filled with fibrilles which is very poor in function. The appearance of abnormal liver tissue clearly shows the important toxic effect of dietary alcohol.

Dietary alcohol also activates phospholipase C which stimulates inositol triphosphate 3. Inositol triphosphate 3 inhibits gamma-aminobutyric acid (GABA). Under normal conditions, gamma aminobutyric acid (GABA) ables Ca release from endoplasmic reticulum which significantly decreases serum Ca concentration. The decrease in Ca concentration increases the speed for the formation of osteoporosis and osteomalacia. As clearly can be understood from these explanations, dietary ethanol shows its adverse and harmful effect on bones and muscles two ways 1. It competes with Ca in foods and absorbed instead of Ca and 2. It inhibits Ca release from ER and decreases Ca concentration in blood.

Dietary ethanol is detoxified in two ways; by the effect of an enzyme called alcohol dehydrogenase of by another detox enzyme system in liver, P450 cytochrome.

Another organ which is adversely affected by dietary ethanol is stomach. The cells which covers the surface of both stomach and intestines are decomposed by the effect of alcohol. Cells are surrounded with a cell membrane and one layer in the structure of cell membrane is lipoprotein, half protein and half lipid. Alcohol melts lipid. Lipid decomposition in lipoprotein structure of the membrannes of cells is the main cause of wounds of stomach cells. The wounded cells which cover the surface of stomach can not accumulate gastric acid and release it. The increase in gastric acid secretion increases the solubility of iron which results in increase of iron absorption by bowel mucosa. Briefly, dietary ethanol may cause iron toxicity by increasing iron absorption. Note that iron toxicity is one of the wort toxicities since it can contribute to tumor formation.

One argument about dietary ethanol besides its amount and addiction is the target group. Although there are some arguments about dietary alcohol, its amounts for addiction, what seems clear is that the dietary ethanol toxicity can not be predicted since many factors play important role in the toxicity. One clear example is the sex difference which was also mentioned above. The protective stomach-alcohol barrier (like brain-blood barrier) is more in men than in women which is the main reason for the greater tolerance of men to alcohol than women. Similarly, the same protective layer, which slows and decreases the absorption of dietary ethanol through the mucosa of bowel is not enough thick in children and adolescents which also makes children and adolescents other susceptible target group

to dietary ethanol toxicity. Acetomnophen, CCl4, cocain, pentobarbital, corticosteroids, antiacids, parasetamol, niton and alcohol itself with some others are the initial drug substances and harmful compounds defect and decrease the thickness of the barrier. Therefore, persons who take medications, cocain and nikotin addicted persons are the other target groups which show more susceptibility to dietary ethanol toxicity and less tolerance to drunkness.

Another risk about dietary ethanol toxicity is the probability of stroke. In spite of many scientific claims that show the cholesterol-bad cholesterol reducing and blood thinning effect of alcohol, there is another possibility which can not be disregarded. Assume that there is already a clot which blocks and closes the diameter and cavity of blood vessel. Followed by blood dilution by the effect of alcohol, the possibility for clot fragmentation may increase and the fragments separated from clot may move and close the diameter of an important brain or coronary (the vessels which feed and supply blood for heart) vessels (emboly) which may result in stroke or cardiac spasm or ventricular or artrial fibrillosis. Briefly, blood diluting effect may show its benefciail effect on persons who follows cholesterol and bad cholesterol low or free diets but others may face with stroke or life threatining cardiovascular diseases and disoredrs in case of cholesterol, bad cholesterol, trans fat, saturated fat, salt and sugar rich diets as combined with alcohol consumption sinmce the mentioned harmful compounds are the structural compounds for clot formation. Alcohol may decompose and cause fragmentation of the clot which may result in stroke and life threatining cardiovascular diseases and disorders.

Here below is the list of some hazards of alcohol (slight). Alcohol may be an origin in the formation of diabet, hepatit, ulcer, gastrit, osteoporosis, osteomalacia, cardiac spasm, Alzheimer, Parkinson, trombose (clot formation which closes the cavity of blood vessel), emboly, atrial and ventricular fibrillosis.

Regular dietary ethanol alcohol intake may increase some types of cancer especially oesopageal cancers. The gastric acid increase followed by gastrit and ulcer due to alcohol degenerative effect shows its first harmful effect on the surface of oesophagus. Alcohol residues which reach to eosophageal tears due to retching and vomitting which is called as Mallory-Weiss syndrome. Another risk for eosophagus is the risk for the formation of episodic ruptured eosophageal varices.

Acetaldehyde which forms due to dietary alcohol oxidation in blood triggers the oxidation of especially B complex vitamines such as folate, folic acid and B12. These vitamines enter into the structures of the enzymes responsible for blood cell proliferation. Therefore, the deficiency of B12, folate and folic acid due to alcohol is an important factor which cause anemia.

Dietary ethanol may dilute blood and decrease cholesterol and bad cholesterol as mentioned above which are all proven by scientific researches. But, the other side of the relation between heart and alcohol is different. Superoxide dismutase, catalase, gulutathione reductase, vitamin E and ascorbic acid (vitamin C) are the enzymes and vitamines in antioxidant activity and protects cardiac tissue (heart) against oxidation and

its free toxic peroxy radical compounds which are inflammatory. Dietary ethanol and its end product acetaldehyde oxidize the mentioned vitamines and minerals. The protection of heart by these important antioxidants become weaker. In conclusion, in spite of cholesterol and bad cholesterol reducing and blood thinning effect, the low or moderate intakes of dietary ethanol has to be reconsiled with the possible counteracting influence of prooxidative effect of alcohol on cardiovascular system.

Ca intake in sufficient amounts by the means of Ca rich foods like milk and dairy products, fish meat, soy and soy products, dark green leafy vegetables is a must for the protection against osteoporosis and osteomalacia. Many of us knows the importance and necessity of Ca in bone and muscle health but the importance of vitamin D, which should be combined with Ca is not commonly known. Vitamin D is the main vitamin that enhances Ca absorption from gut mucosa and increases Ca binding to bone and muscle while paratyhroid hormon is the main hormon in similar effect. Unfortunately, dietary ethanol depletes Ca and vitamin D concentrations by decreasing their absorption levels since it is absorbed prior to them. No matter how dietary ethanol improves heart and blood circulation, it is an antagonist which depletes Ca, vitamin D and parathyroid hormon concentrations and increases the risk of osteoporosis and osteomalacia due to Ca, vitamin D and parathyroid deficiency.

The main argument about the status of dietary ethanol in healthy living and life style is mainly because of the antioxidant substances which are naturally produced during the production of alcoholic beverages. Resveratrol, the antioxidant in grape skin and seeds, an antioxidant which is 20 times more than vitamin C and 50 times more than vitamin E in antioxidant capacity. Phenols and polyphenols, the compounds which are not only the antioxidants but also they are the substances which can decompose nitrite, a strong carcinogen, into diazophenols and decrease the risk for cancer types in stomach and bowel origin. Phenols and polyphenols like resveratrol are isoflavones which thin blood, increase the diameter size of blood vessels, decrease cholesterol and bad cholesterol and homocystein (the initial substances which are responsible for the formation of hypertension since they can easilu accumulate on the walls of blood vessels) and finally increase seratonin (the relaxing hormon). They are exist in rich amounts in rice, grape, corn, briefly in the materail which produce ethanol via to fermentation, yeast and bacterial activity. Finally, flavon and flavonoids in these raw materials and in the end product (alcoholic beverage) are some other strong antioxidants which may prevent body against cardiovascular diseases and disorders and cancer with their similar effects. Flavon and flavonoids decompose heterocyclic amines which are strong carcinogenes. These substances especially increase the risk of cancers in stomach and bowel origin like nitrit and nitrate in processed foods. In conclusion, dietary ethanol may have got some important beneficial effects on body with its mentioned antioxidants, while it may cause harmful effects on heart, liver, brain and pancreas. Some authorities and media advise to use dietary alcohol in moderate amounts due to its rich antioxidant capacity. The problem appears at this point. Because the 'moderate amount', whatever its is, differs and changes alcohol intake, alcohol, accumulation level as mg per ml of blood and the thickness of stomach-alcohol mucosa layer. It is impossible to determine each of these criteria during alcohol consumption. One last and important question which is still waiting for to be

highlighted is 'what amount of dietary alcohol' causes addiction since the amounts here to be investigated may change due to the thichkness of stomach-alcohol mucosa barrier, the accumulative level of dietary alcohol, sex, age, any disease or disorders in stomach and bowel, any other diseases and disorders like diabet, renal failure, liver failure or arthrit which can easily increase the harmful potential of dietary alcohol on body even in moderate amounts. As a result, a food is supposed to increase the overall quality of body. A product in nutrition and consumption which can cause harmful effects on some organs while showing some beneficial effects on some others should be taken out of the list.

Finally, the last important question is the maximum amount which can be tolerated and does not jump to ethanol metabolism and only forms antioxidant activity. This question is also waiting to be highilgted and still unclear. In conclusion, there is no need to take an antioxidant rich product but also very harmful product on some organs into the body. Instead, prefer grape especially purple grape and consume plenty amounts of this fruit. You can easily supply a rich content of resveratrol, quercetin, flavon and flavonoids without any risk.