

Beneficial and Deleterious effects of Wine

Dr. A. Jaganathan

Principal / Secretary, Food Craft Institute, Hoshiarpur, Punjab, India

Abstract: Since ancient times, in various cultures and religions, there has been a strong belief that alcohol offers important health benefits. In recent years, the idea that regular alcohol consumption protects against cardiovascular disease has gained momentum. Individuals who drink moderately reduce their risk of dying from heart disease by approximately 40%. Wine has been part of human culture for 6,000 years, serving dietary and socio religious functions. Its production takes place on every continent, and its chemical composition is profoundly influenced by ecological techniques, the grape cultivar from which it originates, and climatic factors. In addition to ethanol, which in moderate consumption can reduce mortality from coronary heart disease by increasing high-density lipoprotein cholesterol and inhibiting platelet aggregation, wine (especially red wine) contains a range of polyphenols that have desirable biological properties. The antioxidant effects of red wine and of its major polyphenols have been demonstrated in many experimental systems spanning the range from in vitro studies (human low-density lipoprotein, liposomes, macrophages, cultured cells) to investigations in healthy human subjects. Wine is like any other food it should be consumed sensibly and in amounts that are beneficial to health. Overindulgence of any kind does not promote good health.

Keywords: Wine, polyphenols, bacteria, ROS.

I. INTRODUCTION

The important role of wine and their production have had in ancient times as well as in more modern periods of history, particularly in relation to science and technology, deserve wider appreciation. Molecular archaeologists have found evidence of winemaking in northern Iran dating back to 5400 BC. In ancient Egypt, wine that originated in Jordan was buried with King Scorpion in his tomb around 3125 BC. Winemaking and grape growing was certainly well developed by 3000 BC in ancient Egypt, Mesopotamia, and the other areas considered the cradle of Western civilization. At the beginning of recorded history, wines were described, their production portrayed, and their properties critically examined.

By 2500 BC, the Egyptians had evolved hieroglyphics describing various types of wine. As part of the funerary goods in King Tutankhamen's 1339 BC burial, included were wine amphorae stamped with the region of growth, the estate, the vintage and the winemaker's name. Pharaohs and members of the ruling class drank wine regularly, while common people consumed it only during religious festivals and special events.

A. Biblical Times

Alcohol had its initiation under the most auspicious circumstances. Life on earth had just been eliminated by the Great Flood so graphically described in Genesis, Chapter VII, all life except Noah and his binary companions, safe in the Ark constructed according to divine prescription. Following his reprieve, when the flood waters receded and the Ark came safely to rest on dry land, human agriculture commenced with Noah's establishment of a vineyard, according to Genesis, Chapter IX, Verse 20. This was the first documented planting of

any crop by human hand on earth. Unfortunately, Noah did not have the advantage of advice from the Bureau of Alcohol, Drugs, and Firearms and Noah lived after the flood three hundred and fifty years.

Many subsequent passages refer to wine in contexts of varying moral propriety. It was employed by Lot's daughters to intoxicate their father prior to his incestuous seduction during the destruction of Sodom (Genesis Chapter XIX, Verse 33). Jacob, impersonating his older brother Esau in order to secure the "Blessing of the First-Born" from his blind father, offered Isaac wine along with venison prior to the performance of the act (Genesis Chapter XXVII, Verse 25). As for the blessing itself two verses later, it was loaded with metaphysical symbolism and contained the promise of material advantage in only one brief passage: So God give thee . . . plenty of corn and wine. Apparently, no greater possessions had any man in those venerable times. A millennium or so later, Solomon, the wisest of kings, commenced that greatest of love poems, the Song of Songs, with the opening refrain: Let him kiss me with the kisses of his lips, for thy love is sweeter than wine: proof enough that wine is mankind's second-greatest pleasure. Finally, the miracle wrought by Christ during the Wedding Feast at Cana in converting water to wine is all that is needed to sanction its consumption by devout Christians.

II. POST-BIBLICAL TIMES

Wine has an unequivocally recorded history stretching back nearly 6,000 years, with the earliest evidence dating between 5400 and 5000 BC (McGovern et al., 1996). Wine was identified by the presence of the calcium salt of

tartaric acid, which occurs in large amounts only in grapes, and in the resin from the terebinth tree, which was widely used in ancient times as an additive to wine to inhibit the growth of bacteria. Whereas wild grape pips have been found to originate as far back as the eighth millennium, this archaeological discovery marks the earliest scientific record of fermented wine as part of human culture. Some investigators place the discovery of winemaking, or at least its development, in the southern Caucasus. It is also thought that the domestication of the wine grape (*Vitis vinifera*) initially occurred within this area. It is there that the natural distribution of *Vitis vinifera* most closely approaches that of Western viticulture, suggesting spread from the former to the latter (Zohary and Hopf, 1988).

From its postulated origins in the Caucasus, grape growing and winemaking probably travelled southward to Palestine, Syria, Egypt, and Mesopotamia. From this base, wine consumption, and its socio religious connections, distributed winemaking around the Mediterranean. Wine was used for sacramental purposes in Egypt no later than the start of the third millennium BC, although evidence indicates that it was not produced there for general consumption for another 2,000 years. Wines began to take on their modern expressions about the seventeenth century. The widespread use of sulphur in barrel treatment would have greatly increased the likelihood of producing better quality wines and extending their lives.

North Americans are relatively latecomers to viticulture. Franciscan missionaries planted the first large-scale vineyards in California only 200 years ago, and these had to be re-established after the repeal of Prohibition. A by-product of the Californian vineyards in the nineteenth century threatened the extinction of European vines. The plant louse *Phylloxera vitifoliae*, carried to Europe on California rootstocks, caused a pandemic devastating some 1 million hectares (2.5 million acres) of vineyards in France alone. Ultimately, the tide was turned when Europe's vineyards were replanted entirely with *Phylloxera*-resistant rootstocks (*Vitis labrusca*) native to the eastern United States, onto which vines of the European wine grapes, *Vitis vinifera*, were grafted.

A. Wine the most consumed drink

Red wine is a commonly consumed beverage worldwide which is rich in antioxidants (Jamroz and Beltowski, 2001). Observational and intervention studies suggest a beneficial association between moderate alcohol consumption and systemic inflammation (Sierksma et al., 2002). Epidemiological data indicates that a moderate intake of red wine is associated with a reduced incidence of CVD whereas a higher intake is associated with an increased risk of cancer and CVD (Di Castelnuovo et al., 2002). The protection that is observed as a result of a moderate red wine consumption may partly be explained by the intake of red wine polyphenols. Polyphenols are efficient antioxidant compounds and is especially abundant in red wine (Waterhouse, 2002). Red wine polyphenols (tannins, flavonoids, catechins, anthocyanins and phenolic acids) have been implicated in several

biological processes, such as free-radical scavenging and inflammatory modulation, metal chelating and enzyme modulation, (Rahman et al., 2006) as well as reduction of the susceptibility of low-density lipoproteins (LDL) to oxidation both in vitro (Bertelli et al., 1996) and in vivo (Nigdikar et al., 1998). Red wine polyphenols may also be involved in non-antioxidant processes as inhibition of platelet aggregation, vasorelaxing activity and modulation of lipid metabolism (Demrow et al., 1995).

Table 1: Consumption of wine in 2011 compared between 2009 and 2010.

Countries	Difference in volume of wine between 2009 2010
US	+0.9 Mhl
Italy	-6.3 Mhl
French	+1.0 Mhl
Spanish	-0.2 Mhl
British	-0.4 Mhl
Portuguese	-0.15 Mhl
China	+1.15 Mhl

B. Chemical composition of grapes and wine

More than 500 compounds have been recognized in wine thus far, of which 160 are esters. The concentrations of the majority range between 10-1 and 10-6 mg/L. At these levels the individual compounds play very little or no role in the human organoleptic (taste) perception, but collectively they may be very significant (Amerine and Roessler, 1982). The taste and mouth-feel sensations are due primarily to the few compounds that occur individually at concentrations > 100 mg/L. These include water, ethanol, organic acids, sugars, and glycerol. Unlike the taste and mouth-feel sensations, odour has a much lower olfactory threshold. It is measured as olfactory potential, which represents the number of molecule-grams per litre of air at threshold concentration. Chemical composition of wine is shown in Table 3.

Table 2 Estimates of Typical Gross Composition (% weight) of Wines

Component	Table wines		Dessert wines	
	White	Red	White	Red
Water	87	87	76	74
Ethanol	10	10	14	14
Other volatiles	0.04	0.04	0.05	0.05
Extract	2.6	2.7	10.1	12.2
Sugars	0.05	0.05	8	10
Pectins	0.3	0.3	0.25	0.25
Glycerol	1.1	1.1	0.9	0.9
Acids	0.7	0.6	0.5	0.5
Ash	0.2	0.2	0.2	0.2
Phenols	0.01	0.2	0.01	0.1
Amino acids	0.25	0.25	0.2	0.2
Fats, terpenoids	0.01	0.02	0.01	0.02
Vitamins, etc.	0.01	0.01	0.01	0.01

C. Constituents of wine

Wine phenolic compounds include flavonoids (Fig. 1) and nonflavonoids (Fig. 2). Flavonoids are polyphenols consisting of anthocyanins. Nonflavonoids include hydroxycinnamic acid, benzoic acid, tannins and stilbenes. Catechin (and derivatives) on its own has also been reported to demonstrate protective effects against CVD. These include a protective effect against tamoxifen-induced oxidative damage (Tabassum et al., 2007); increased alpha-tocopherol concentration in blood plasma, liver, and lungs (Frank et al., 2003); reduced plasma lipid peroxides resulting in protection against atherosclerosis (Miura et al., 2001); prevention of endothelial dysfunction in Otsuka Long-Evans Tokushima fatty (OLETF) rats at the prediabetic stage (Ihm et al., 2009); and modulation of cytokine expression and thus prevention of low-grade inflammation (Terra et al., 2009).

D. Proanthocyanidins

Proanthocyanidins and condensed tannins are complex flavonoid polymers naturally present in cereals, legumes, and fruits. They are mainly formed by the condensation of flavanol units to generate oligomers (proanthocyanidins) and polymers (condensed tannins). Their levels in wine depend on pressing techniques and grape varieties. Typically they range from 5mg/L in white wines to 1 g/L or even higher levels in old red wines (Santos-Buelga and Scalbert, 2000). They are associated with a change in wine quality such as a modification of the hue and a decrease in astringency. Condensed tannins should be degraded in monomeric phenols, absorbed and metabolized. Numerous studies indicate that proanthocyanidins and condensed tannins might prevent both cancers and cardiovascular diseases (Aldini et al., 2003). Some reports demonstrate that its biological abilities to scavenge the reactive oxygen species (ROS) are associated to the degree of polyphenol oligomerization. Some of these polyphenols might have specific structures that exhibit neuroprotective effects by interacting with putative neuron specific receptors (Narita et al., 2011). Takahashi et al. have shown that procyanidin oligomers from grape seed exhibit higher growth promoting activity than the monomers toward mouse hair epithelial cells in vitro and in vivo, these results indicating that the specific effect might be correlated with their structure (Takahashi et al., 1999). Other research on rat brain suggests that grape seed extract enriched in proanthocyanidins might protect against pathology age-related oxidative brain damage (Deshane et al., 2004).

E. Anthocyanins

Anthocyanins act as guard systems in plants and protect them from UV damage. They form complex molecules with other phenolic molecules and strongly contribute to the colour and the aging of wine (De Freitas et al., 2004). The aglycone ring of these flavonoids is called anthocyanidin. However, no conjugated anthocyanidins are ever found in grapes or wine, except in trace quantities. In wine there are five anthocyanidins: malvidin, cyanidin, delphinidin, peonidin, and petunidin. Malvidin is

the most abundant anthocyanidin in red wines (Waterhouse, 2002).

Among the wine flavonoids, anthocyanins constitute one of the higher potent antioxidants correlated to their capacity to delocalize electrons and form resonating structures (Rivero-Perez et al., 2008). Anthocyanins present numerous health benefits such as anti-carcinogenic, anti-inflammatory, or anti-diabetic effects (Toufektsian et al., 2008). Anthocyanins also possess beneficial neuroprotective abilities. Some of them have the ability to cross the blood-brain barrier and diffuse through the central nervous system (Milbury and Kalt, 2010). Anthocyanins have neuroprotective benefits in reducing age-associated oxidative stress and improving cognitive brain function (Shih et al., 2010). They induce significant neuroprotective effects against oxidative stress, DNA fragmentation and lipid peroxidation in mouse brain (Di Giacomo et al., 2007). Thus, it appears that the antioxidant and anti-inflammatory effects of anthocyanins contribute to its neuroprotective effect.

III. NON-FLAVONOIDS

A. Phenolic Acids

The benzoic acids are a minor component in wines. Whereas the hydroxycinnamates are the most important class of non-flavonoid phenols in grape vine and the major class of phenolics in white wine. The three important ones in wine are coumaric acid, caffeic acid, and ferulic acid. Amount of total hydroxycinnamates in wine are typically about 60 mg/L in reds and 130 mg/L in whites (Waterhouse, 2002). Hydroxycinnamates have an antioxidant activity by scavenging free radicals (Maurya and Devasagayam, 2010). Their strong antioxidant properties help to explain their beneficial role on health and in reducing disease risk.

Hydroxycinnamates and other phenolic acids have received less attention. It has been shown that p-coumaric acid, hydroxycinnamates caffeic acid, and a Champagne wine extract rich in these compounds have neuroprotective effects against injury induced by 5- S-cysteinyl-dopamine in vitro. Caffeic acid has been reported to have neuroprotective effects against A β -induced neurotoxicity in vitro and to inhibit peroxy-nitrite-induced neuronal injury (Vauzour et al., 2010). Ferulic acid has been showed to protect primary neuronal cell cultures against hydroxyl- and peroxy-radical-mediated oxidative damage (Sultana, 2012).

Caffeic and ferulic acids may play a role in the body's defense against carcinogenesis by inhibiting the formation of N-nitroso compounds. Caffeic and ferulic acids were reported to react with nitrite in vitro and to inhibit nitrosamine formation in vivo. In simulated gastric fluid, caffeic acid and ferulic acid reacted rapidly and completely with an equimolar quantity of sodium nitrite. Caffeic acid was more effective than ferulic acid in both

the *in vitro* (reaction with nitrite) and *in vivo* (inhibition of hepatotoxicity) systems (Kuenzig et al., 1984).

Gentisic acid, due to its antioxidant activity and its ability to inactivate hydroxyl free radicals, might also prevent, as has been suggested for aspirin, the development of some forms of cancer (Betts et al., 1985). Gallic acid, caffeic acid, and other phenolic compounds inhibited aflatoxin B1 (AFB1)-induced mutagenesis in a *Salmonella typhimurium* strain TA 98 in a suspension assay in the presence of rat liver microsomes (San and Chan, 1987). The inhibitory effect was observed only when the phenolic compound and the mutagen were administered concurrently and in a dose dependent manner.

B. Hydrolyzable Tannins

Tannins are water-soluble polyphenols. One of the major properties of these molecules is their capacity to precipitate proteins such as gelatin from solution (Adrian et al., 1996). In wine, hydrolyzable tannins arise during maturation and ageing of wines in oak barrels. Castalagin and vescalagine are the main representative compounds of ellagic tannins. Their levels are about 100 mg/L in aged white wines, while red wine levels are about 250 mg/L after aging in oak barrels for two or more years (Quinn and Singleton, 1985). They are mainly ellagic acid and gallic acid ester derivatives with glucose or other sugars. Due to the presence of the ester linkage, they are described as being hydrolyzable. Hydrolyzable tannins are not present in *Vitis vinifera* but are present in other fruits such as muscadine grapes and raspberries (Landete, 2011). These polyphenols are excellent antioxidants and natural preservatives, also helping give the wine structure and texture. However, recent research on tannins has focused on their potential to impact positively on human health. Tannins have demonstrated a host of potent biological activities, anti-peroxidation properties, inhibition of mutagenicity of carcinogens and tumor promotion, specific anti-tumor abilities in relation with tannin structures, anti-bacterial activity, and anti-viral activity (Okuda, 2005). *In vivo*, ellagitannins are mainly transformed into ellagic acid and its metabolites. In fact, they could be the agent responsible for the effects of dietary ellagitannins observed *in vivo* (Quideau et al., 2011).

C. Deleterious role of wine

Wine bottle labels contain a warning about their content of sulfites (sulfur dioxide in its various stages of dissociation and binding) because about 5% of people with asthma can suffer from a severe potentially deadly adverse reaction. This translates into about 1 in 600 people affected by sulfites. There is no evidence however, that sulfites contribute to a "wine headache," which is likely due to dehydration or other allergens. The aforementioned effect on blood vessel dilation due to phenolic materials may explain the more common complaints about headaches from red wines which on average also contain more alcohol. It is a good idea to drink a glass of water with

each glass of wine consumed. Partly due to international enological practices agreements, there is no general difference between global wine-producing regions and the sulfite (or other additive) levels found in wines. Optional wine fining agents such as egg white protein are only minimally retained in wine after the treatment but residues could trigger allergic reactions in some of the most sensitive individuals.

However, alcohol is not totally benign, especially at higher doses (Gronbaek 2009). It has been reported to elevate heart rate and blood pressure 8 to 10 h after drinking red wine (375 mL; 39 grams of alcohol) and beer (1125 mL; 41 grams alcohol) (Mukamal and Rimm 2008). A consumption of high doses of alcohol has also been linked to the cause of some cancers (Petti 2009). Lachenmeier et al. (2009) reported that acetaldehyde may be a contributing factor in the carcinogenicity of alcohol. However, as acetaldehyde is found in an enormous range of foods, including fresh fruits and vegetables and practically every food subjected to fermentation, this report awaits further scrutiny as to the true risks associated with acetaldehyde consumption.

A considerable body of evidence supports the hypothesis that habitual consumption of large amounts of alcohol has a variety of deleterious effects on the kidney (Cecchin and Marchi, 1996). Thus, consumption of more than two standard drinks per day (24 g ethanol/day) was associated with an increased risk of kidney failure in the general population (Parekh and Klag, 2001).

D. Beneficial role of wine

Wine was considered somewhat less dangerous because it was fairly dilute and consumed with meals, but most public health specialists did not credit wine or any other such beverage with desirable effects. Ethanol is readily metabolized by the body, providing about 435 kJ (104 kcal) of energy from a glass of dry table wine, and any alcoholic beverage can have favorable effects on Type II diabetes, when properly consumed.

Only recently have the government and other health agencies bowed to the preponderance of clinical, experimental, epidemiological, and historical evidence that moderate consumption of wine is not only not detrimental, but is beneficial. The proven benefit is in lowered incidence of cardiovascular complications in wine consumers (Cooper et al., 2004). A flurry of recent studies further suggests that wine, particularly red table wine, has an additional favorable effect over other alcoholic beverages. This is attributed to the antioxidant, free-radical chain-breaking effect of the wine's natural phenolic compounds (Jamroz and Beltowski, 2001). The ability of phytochemicals in wine to reduce platelet aggregation and prevent oxidation of blood cholesterol and its subsequent deposit on artery walls seem to be the major effects. In combination with their ability to moderate the dilation of blood vessels, their presence in both red and white (though

to a lesser degree based on their limited extraction during the white wine making process) wines may explain the so-called French Paradox.

This epidemiological phenomenon shows that the population of France and other Mediterranean countries have a higher life expectancy and much lower rate of heart attack and stroke than the U.S. population despite a higher intake of foods rich in cholesterol and saturated fats such as whole-milk cheeses or liver. The 1995 Dietary Guidelines for Americans acknowledges that moderate drinking of alcoholic beverages with meals is associated with a lower risk for coronary heart disease, the leading cause of death in this country. Moderation is defined very conservatively as one drink per day with a meal for a woman and two for a man (150 mL or 5 oz of table wine per drink). Wine consumption has been proven beneficial to a number of vision problems, such as macular degeneration, cataract and glaucoma. Grape phenols' role as an anticancer agent appears relevant in regard to prostate cancer in men. Much of the epidemiological wine and health research however is inconclusive, as much as Cogan commented in his 1584 Haven of Health, "Drink wine and have the gout, drink none and have it, too!"

E. Cardioprotective effect

Coronary heart disease (CHD) is one of the major causes of death worldwide. Epidemiologic and human intervention studies have shown the inverse relationship between the consumption of plant-based diets and deaths attributed to heart disease. Most dietitians and nutritionists around the world are recommending an increase in the consumption of plant foods for the prevention of CHD. Certain foods are well known for their ability to protect human health from CHD. Grape is the most well known among them and has been used in medicinal science from the time immemorial. Ayurveda, one of the ancient medicinal books of Hindus, described "darakhasava" (fermented juice of red grapes) as a cardio tonic (Paul et al., 1999). Grape juice or red wine was also described as a "gift of god" in The Bible.

There is now almost universal acceptance among the scientific community that alcohol, when consumed in moderation, is associated with a lower incidence of cardiovascular disease and generally better health outcomes (Lang and Melzer, 2009). Some controversy still exists over whether red wine has superior protective effects than other alcoholic beverages (Cordova et al., 2005).

Numerous epidemiological studies have demonstrated an association between moderate alcohol consumption and reduced risk of cardiovascular disease. Thun et al. (1997) examined the effect of alcohol consumption on mortality among U.S. adults. Of 4,90,000 men and women WHO reported their alcohol and tobacco use, 46000 died during 9 years of follow-up. The death rates from cardiovascular disease were 30% to 40% lower among men and women

reporting at least 1 drink (assumed average 12 g of alcohol) daily compared with nondrinkers. The overall death rates were lowest among men and women reporting approximately 1 drink daily (Thun et al., 1997). Criqui and Ringel (1994) reached comparable conclusions, but further noted that wine was more strongly correlated with lower rates of coronary heart disease (CHD) than either beer or spirits. Red wine can increase high-density lipoproteins (HDL), popularly known as "good cholesterol." HDL is required for transport of cholesterol from the arteries and various other parts of the body back to the liver for metabolism and/or excretion.

F. Neuroprotective effect

Wine related phenolic compounds exhibit a positive effect on nerve cells (Assuncao et al., 2007). The mechanism proposed as explaining the effect on wine polyphenolic compounds on health can be principally summarized as scavenging intracellular ROS and inhibition of LDL oxidation (Jang and Surh, 2003). In recent years, studies on the activity of wine polyphenols have been extended to animal models of CNS disorders and injury (Chan et al., 2008). These effects are principally associated to their strong antioxidant capacities, since they can act as free-radical scavengers and hydrogen or electron, to preventing DNA damage and lipid peroxidation (Giovannelli et al., 2000). Antioxidant polyphenols protect cell constituents from oxidative alteration and thus limit the risk of developing degenerative disorders induced by oxidative stress, such as in ischemia, Parkinson's disease or Alzheimer's disease. For example, an increasing number of reports have shown that acute chronic treatment of resveratrol exhibits neuroprotective effects against colchicine and nitropropionic acid (Kumar et al., 2007) or motor impairment as well as hippocampal neuron loss (Zhang et al., 2008). These properties are mainly associated to the antioxidant activity of resveratrol. Resveratrol decreases the oxidative damages, in reducing the levels of malondialdehyde, lipid peroxidation, xanthine oxidase, and nitric oxide, and in increasing the depleted glutathione levels and succinate dehydrogenase activity in rat brain (Ates et al., 2007).

G. Nephroprotective effect

ROS play a key role in the pathophysiological processes of renal diseases. The cellular damage is mediated by an alteration in the antioxidant status, which increases the concentration of ROS in the stationary state (oxidative stress). The abundance of polyunsaturated fatty acids (PUFA) makes the kidney an organ particularly vulnerable to ROS attack (Kubo et al., 1997). The involvement of ROS is supported by two lines of experimental evidence; namely, (i) detection of products of oxidant injury in renal tissue or urine, and (ii) experimental demonstration of a protective effect of metabolic inhibitors of ROS (Ishikawa et al., 1994).

Oxidative stress mediates a wide range of renal impairments, from acute renal failure, rhabdomyolysis,

obstructive nephropathy, hyperlipidemia, and glomerular damage to chronic renal failure and hemodialysis (Ramon and Gonzalo, 2002). Therefore, interventions favoring the scavenging and/or depuration of ROS (dietary and pharmacological antioxidants), should attenuate or prevent the oxidative stress, thereby mitigating against the subsequent renal damage. Polyphenols are a group of naturally occurring antioxidant substances found in vegetables, fruits or tea, and are particularly abundant in red wine (McDonald et al., 1998).

H. Anti-diabetic effect

The effect of alcohol in moderation on reducing the incidence of diabetes, (Baliunas et al., 2009) a strong risk factor for CVD, may be a mediating mechanism. Moderate alcohol consumption (<60 g/d in men and <50 g/d in women) was inversely associated with diabetes risk. In healthy women, an inverse association between moderate alcohol intake and lower diabetes risk was most apparent in those who reported wine or beer drinking compared to women who reported liquor intake (Wannamethee et al., 2003). A salient feature of alcohol consumption is the increase in HDL cholesterol (HDL-C) and apolipoprotein (Apo) A-I concentrations. HDL-C and ApoA-I positively affect insulin secretion and pancreatic β -cell survival, thereby enhancing insulin sensitivity (IS) (von-Eckardstein and Sibling, 2011). Since insulin resistance increases the risk of both CVD and diabetes, moderate alcohol consumption could possibly decrease these risks by improving IS. However, clinical trials assessing the short-term effects of moderate consumption of different alcoholic beverages on IS are few and the results are contradictory, as some studies have shown a positive effect while most have reported no benefit (Kim et al., 2009).

IV. CONCLUSION

Many phenolic compounds present in food and vegetables demonstrate potent and desirable biological activities. The most universal property relates to their function as antioxidants, manifested by their ability to trap free radicals and inhibit their enzymatic generation, and to block the oxidation of cellular and extracellular components such as membranes and LDL. More selectively, certain of these polyphenols can prevent or diminish aggregation of platelets and their synthesis of pro-aggregatory eicosanoids such as thromboxane A₂, as well as synthesis by leukocytes of pro-inflammatory leukotrienes. However, some polyphenols are able to promote the synthesis of prostacyclin and nitric oxide and in this way may play a role in optimizing blood flow through the arterial system. Several, especially quercetin, have anticancer potential despite their mutagenic capabilities, and others may have lipid lowering properties, although such effects appear to be relatively weak. An intake of two glasses of red wine per day will provide ~40% of the total antioxidant polyphenols present in a healthy diet, as well as a number such as resveratrol

that are virtually absent from commonly consumed fruit and vegetables. Therefore moderate consumption of wine is beneficial to health.

REFERENCES

- [1] Adrian, J.C., Baxter, N.J., Lilley, T.H., Haslam, E., McDonald, C.J. and Williamson, M.P. 1996. Tannin interactions with a full-length human salivary proline-rich protein display a stronger affinity than with single proline-rich repeats. *FEBS Letters*. 382(3): 289-292.
- [2] Aldini, G., Carini, M., Piccoli, A., Rossoni, G. and Facino, R. M. 2003. Procyanidins from grape seeds protect endothelial cells from peroxynitrite damage and enhance endothelium-dependent relaxation in human artery: new evidences for cardio-protection. *Life Sci*. 73(22): 2883-2898.
- [3] American Institute for Cancer Research/World Cancer Research Fund. Food, nutrition and the prevention of cancer: a global perspective. Washington, DC: American Institute for Cancer Research. 1997.
- [4] Amerine, M.A. and Roessler, E.B. 1982. Composition of wines. In *Wines, Their Sensory Evaluation*, MA Amerine, ed. Freeman, New York, 67-80.
- [5] Amira-Guebailia, H., Valls, J. and Richard T. 2009. Centrifugal partition chromatography followed by HPLC for the isolation of cis- ϵ -viniferin, a resveratrol dimer newly extracted from a red Algerian wine. *Food Chem*. 113(1): 320-324.
- [6] Ansari, M.A., Abdul, H.M., Joshi, G., Opii, W.O. and Butterfield, D.A. 2009. Protective effect of quercetin in primary neurons against $A\beta(1-42)$: relevance to Alzheimer's disease. *J. Nutri. Biochem*. 20(4): 269-275.
- [7] Assuncao, M., Santos-Marques, M.J. and DeFreitas, V. 2007. Red wine antioxidants protect hippocampal neurons against ethanol-induced damage: a biochemical, morphological and behavioral study. *Neurosci*. 146(4):1581-1592.
- [8] Ates, O., Cayli, S. and Altinoz, E. 2007. Neuroprotection by resveratrol against traumatic brain injury in rats. *Mol. Cell. Biochem*. 294(1-2): 137-144.
- [9] Baliunas, D.O., Taylor, B.J., Irving, H., Roerecke, M., Patra, J. and Mohapatra, S. 2009. Alcohol as a risk factor for type 2 diabetes: a systematic review and meta-analysis. *Diab. Care*. 32: 2123-2132.
- [10] Bertelli, A., Migliori, M. and Bertelli, A.A. 2002. Effect of some white wine phenols in preventing inflammatory cytokine release. *Drugs Exp. Clin. Res*. 28: 11-5.
- [11] Bertelli, A.A., Giovannini, L., Stradi, R., Bertelli, A. and Tillement, J.P. 1996. Plasma, urine and tissue levels of trans- and cis-resveratrol (3,4',5-trihydroxystilbene) after short-term or prolonged administration of red wine to rats. *Int. J. Tissue React*. 18(2-3): 67-71.
- [12] Betts, W.H., Whitehouse, M.W. and Cleland, T.J. 1985. Vernon-Roberts B: In vitro antioxidant properties of potential biotransformation products of salicylate, sulphasalazine, and amidopyrine. *Free Rad. Biol. Med*. 1: 273-280.
- [13] Blanco-Colio, L.M., Valderrama, M. and Alvarez-Sala, L.A. 2000. Red wine intake prevents nuclear factor-kappaB activation in peripheral blood mononuclear cells of healthy volunteers during postprandial lipemia. *Circulation*. 102: 1020-1026.
- [14] Cecchin, E. and DeMarchi, S. 1996. Alcohol misuse and renal damage. *Addict. Biol*. 1: 7-17.
- [15] Chan, S.L., Tabellion, A., Bagrel, D., Perrin-Sarrado, C., Capdeville-Atkinson, C. and Atkinson, J. 2008. Impact of chronic treatment with red wine polyphenols (RWP) on cerebral arterioles in the spontaneous hypertensive rat. *J. Cardiovas. Pharmacol*. 51(3): 304-310.
- [16] Cooper, K.A., Chopra, M. and Thurnham, D.I. 2004. Wine polyphenols and promotion of cardiac health. *Nutr. Res. Rev*. 17: 111-129.
- [17] Cordova, A.C., Jackson, L.M., Berke-Schlessel, D.W. and Sumpio, B.E. 2005. The cardiovascular protective effect of red wine. *J. Am. Coll. Surgeons*. 200: 428-439.
- [18] Criqui, M.H. and Ringel, B.L. 1994. Does diet or alcohol explain the French paradox. *Lancet*. 344: 1719-1723.

- [19] De Freitas, V., Sousa, C., Silva, A.M.S., Santos-Buelga, C. and Mateus, N. 2004. Synthesis of a new catechin-pyrylium derived pigment. *Tetrahedron Letters*. 45(51): 9349-9352.
- [20] Demrow, H.S., Slane, P.R. and Folts, J.D. 1995. Administration of wine and grape juice inhibits in vivo platelet activity and thrombosis in stenosed canine coronary arteries. *Circulation*. 91(4): 1182-1188.
- [21] Deshane, J., Chaves, L., Sarikonda, K.V. 2004. Proteomics analysis of rat brain protein modulations by grape seed extract. *J. Agri. Food Chem*. 52(26): 7872-7883.
- [22] Di Castelnuovo, A., Rotondo, S., Iacoviello, L., Donati, M.B. and de Gaetano, G. 2002. Meta-analysis of wine and beer consumption in relation to vascular risk. *Circulation*. 105(24): 2836-2844.
- [23] Di Giacomo, C., Acquaviva, R. and Piva, A. 2007. Protective effect of cyanidin 3-O- β -D-glucoside on ochratoxin A-mediated damage in the rat. *British J. Nutr.* 98(5): 937-943.
- [24] A.S.Syed Navaz & Dr.G.M. Kadhar Nawaz "Layer Orient Time Domain Density Estimation Technique Based Channel Assignment in Tree Structure Wireless Sensor Networks for Fast Data Collection" June - 2016, *International Journal of Engineering and Technology*, Vol No - 8, Issue No - 3, pp.-1506-1512.
- [25] A.S.Syed Navaz, P.Jayalakshmi, N.Asha. "Optimization of Real-Time Video Over 3G Wireless Networks" September - 2015, *International Journal of Applied Engineering Research*, Vol No - 10, Issue No - 18, pp. 39724 - 39730.
- [26] Estruch, R., Sacanella, E. and Badia, E. 2004. Different effects of red wine and gin consumption on inflammatory biomarkers of atherosclerosis: A prospective randomized crossover trial. *Effects of wine on inflammatory markers. Atherosclerosis*. 175: 117-23.
- [27] Faria, A., Pestana, D. and Teixeira, D. 2011. Insights into the putative catechin and epicatechin transport across bloodbrain barrier. *Food Function*. 2(1): 39-44.
- [28] Fernandez-Mar, M.I., Mateos, R., Garcia-Parrilla, M.C., Puertas, B. and Cantos-Villar, E. 2012. Bioactive compounds in wine: resveratrol, hydroxytyrosol and melatonin: a review. *Food Chem*. 130(4): 797-813.
- [29] Frank, J., Lundh, T., Parker, R.S., Swanson, J.E., Vessby, B. and Kamal-Eldin, A. 2003. Dietary (+)-catechin and BHT markedly increase alpha-tocopherol concentrations in rats by a tocopherol-omega-hydroxylase-independent mechanism. *J. Nutr.* 133: 3195-3199.
- [30] P.Nazni & A.Jaganathan, STUDY ON MICROBIAL ANALYSIS OF STREET-VENDED FOOD SAMPLES SOLD IN SALEM DISTRICT, *International Journal of Research in Biological Sciences*, Vol -4, Issue - 3, August 2014. Pp. 75-78.
- [31] P.Nazni & A.Jaganathan, STANDARDIZATION AND PROXIMATE ANALYSIS OF STREET FOODS SOLD IN SALEM DISTRICT, *International Journal of Agricultural and Food Science*, Vol -4, Issue - 3, August 2014. Pp. 94-99.
- [32] Dr. A.Jaganathan, Human Nutrition to Prevent from Diet-Related Chronic Diseases, *International Journal of Scientific Research*, Vol -5, Issue - 8, August 2016. Pp. 1410-1413.
- [33] Dr. A.Jaganathan, Production of Rice Flour and Peanut Paste in Yellow Cake, *International Journal of Scientific Research*, Vol -5, Issue - 9, September 2016. Pp. 597-600.
- [34] Fuhrman, B. and Aviram, M. 2001. Flavonoids protect LDL from oxidation and attenuate atherosclerosis. *Curr. Opin. Lipidol*. 12 :41-48.
- [35] Giovannelli, L., Testa, G., DeFilippo, C., Cheyner, V., Clifford, M.N. and Dolara, P. 2000. Effect of complex polyphenols and tannins from red wine on DNA oxidative damage of rat colon mucosa in vivo. *Eur. J. Nutr.* 39(5): 207-212.
- [36] Gronbaek, M. 2009. The positive and negative health effects of alcohol- and the public health implications. *J. Int. Med*. 265: 407-420.
- [37] Ho, J.H. and Chang, Y.L. 2004. Protective effects of quercetin and vitamin C against oxidative stress-induced neurodegeneration. *J. Agri. Food Chem*. 52(25): 7514-7517.
- [38] Hosokawa, N., Hosokawa, Y. and Sakai, T. 1990. Inhibitory effect of quercetin on the synthesis of a possible cell-cycle-related 17 kDa protein in human colon cancer cells. *Int. J. Cancer* 45:1119-1124.
- [39] A.S.Syed Navaz & A.S.Syed Fiaz, "Load Balancing in P2P Networks using Random Walk Algorithm" March - 2015, *International Journal of Science and Research*, Vol No - 4, Issue No - 3, pp.2062-2066.
- [40] A.S.Syed Navaz & Dr.G.M. Kadhar Nawaz "Flow Based Layer Selection Algorithm for Data Collection in Tree Structure Wireless Sensor Networks" March - 2016, *International Journal of Applied Engineering Research*, Vol No - 11, Issue No - 5, pp.-3359-3363.
- [41] A.S.Syed Navaz & R.Barathiraja "Security Aspects of Mobile IP" *Journal of Nano Science and Nano Technology (International)*, February 2014, Vol- 2, Issue - 3, pp - 237-240.
- [42] A.S.Syed Navaz, S.Gopalakrishnan & R.Meena "Anomaly Detections in Internet Using Empirical Measures" February 2013, *International Journal of Innovative Technology and Exploring Engineering*, Vol 2 - Issue 3. pp.58-61.
- [43] A.S.Syed Navaz, H.Iyyappa Narayanan & R.Vinoth." Security Protocol Review Method Analyzer (SPRMAN)" August - 2013, *International Journal of Advanced Studies in Computers, Science and Engineering*, Vol No - 2, Issue No - 4, pp. 53-58.
- [44] A.S.Syed Navaz, M.Ravi & T.Prabhu, "Preventing Disclosure of Sensitive Knowledge by Hiding Inference" February 2013, *International Journal of Computer Applications*, Vol 63 - No 1. pp. 32-38.
- [45] Ihm, S.H., Lee, J.O., Kim, S.J., Seung, K.B., Schini-Kerth, V.B., Chang, K. and Oak, M.H. 2009. Catechin prevents endothelial dysfunction in the prediabetic stage of oledt rats by reducing vascular NADPH oxidase activity and expression. *Atherosclerosis*. 206: 47-53.
- [46] Ishikawa, I., Kiyama, S. and Yoshioka, T. 1994. Renal antioxidant enzymes: their regulation and function. *Kidney Int*. 45: 1-9.
- [47] Jamroz, A. and Beltowski, J. 2001. Antioxidant capacity of selected wines. *Med. Sci. Monit*. 7(6): 1198-1202.
- [48] Jang, J.H. and Surh, Y.J. 2003. Protective effect of resveratrol on β -amyloid-induced oxidative PC12 cell death. *Free Rad. Biol. Med*. 34(8): 1100-1110.
- [49] Kim, S.H., Abbasi, F., Lamendola, C. and Reaven, G.M. 2009. Effect of moderate alcoholic beverage consumption on insulin sensitivity in insulin-resistant, non-diabetic individuals. *Met*. 58: 387-392.
- [50] Kubo, K., Saito, M., Tadocoro, T. and Maekawa, A. 1997. Changes in susceptibility of tissues to lipid peroxidation after ingestion of various levels of docosahexanoic acid and vitamin E. *Br. J. Nutr.* 78: 655-669.
- [51] Kuenzig, W., Chan, J. and Norkus, E. 1984. Caffeic acid and ferulic acid as blockers of nitrosamine formation. *Carcinogenesis*. 5: 309-313.
- [52] Kumar, A., Naidu, P.S., Seghal, N. and Padi, S.S.V. 2007. Neuroprotective effects of resveratrol against intracerebroventricular colchicine-induced cognitive impairment and oxidative stress in rats. *Pharmacol*. 79(1): 17-26.
- [53] Lachenmeier, D.W., Kanteres, F. and Rehm, J. 2009. Carcinogenicity of acetaldehyde in alcoholic beverages: risk assessment outside ethanol metabolism. *Addiction*. 104: 533-550.
- [54] Landete, J.M. 2011. Ellagitannins, ellagic acid and their derived metabolites: a review about source, metabolism, functions and health. *Food Res Inter*. 44(5): 1150-1160.
- [55] Lang, I.A. and Melzer, D. 2009. Moderate alcohol consumption in later life: time for a trial? *J. Am. Geriatr. Soc*. 57: 1110-1112.
- [56] Makela, P., Gmel, G., Grittner, U., Kuendig, H., Kuntsche, S. and Bloomfield, K. 2006. Drinking patterns and their gender differences in Europe. *Alcohol Suppl*. 41(1): 118-119.
- [57] Makris, D.P., Kallithraka, S. and Kefalas, P. 2006. Flavonols in grapes, grape products and wines: burden, profile and influential parameters. *J. Food Comp. Anal*. 19(5): 396-404.
- [58] Manach, C., Morand, C. and Texier, O. 1995. Quercetin metabolites in plasma of rats fed diets containing rutin or quercetin. *J. Nutr*. 125: 1911-1922.
- [59] Mandel, S., Amit, T., Reznichenko, L., Weinreb, O., and Youdim, M.B.H. 2006. Green tea catechins as brain-permeable, natural iron chelators-antioxidants for the treatment of neurodegenerative disorders. *Mol. Nutr. Food Res*. 50(2): 229-234.
- [60] Marques-Vidal, P., Cambou, J.P., Nicaud, V., Luc, G., Evans, A., Arveiler, D., Bingham, A. and Cambien, F. 1995. Cardiovascular risk-factors and alcohol-consumption in France and Northern-Ireland. *Atherosclerosis*. 115: 225-232.

- [61] Maurya, D.K. and Devasagayam, T.P.A. 2010. Antioxidant and prooxidant nature of hydroxycinnamic acid derivatives ferulic and caffeic acids. *Food Chem. Toxicol.* 48(12): 3369-3373.
- [62] McDonald, M.S., Hughes, M., Burns, J., Lean, M.E.J., Matthews, D. and Crozier, A. 1998. Survey of the free and conjugated myricetin and quercetin content of red wines of different geographical origins. *J. Agric. Food Chem.* 46: 368-375.
- [63] McGovern, P., Glusker, D. and Exner, L. 1996. Neolithic resinated wine. *Nature.* 381: 480-481.
- [64] Milbury, P.E. and Kalt, W. 2010. Xenobiotic metabolism and berry flavonoid transport across the blood? brain barrier. *J. Agri. Food Chem.* 58(7): 3950-3956.
- [65] Miura, Y., Chiba, T., Tomita, I., Koizumi, H., Miura, S., Umegaki, K., Hara, Y., Ikeda, M. and Tomita, T. 2001. Tea catechins prevent the development of atherosclerosis in apoprotein e-deficient mice. *J. Nutr.* 131: 27-32.
- [66] Mukamal, K.J. and Rimm, E.B. 2008. Alcohol consumption: risks and benefits. *Curr. Atheroscler. Rep.* 10: 536-543.
- [67] Narita, K., Hisamoto, M., Okuda, T. and Takeda, S. 2011. Differential neuroprotective activity of two different grape seed extracts. *PLoS. One.* 6(1): 145-175.

BIOGRAPHY



Dr. A. Jaganathan received the B.Sc in HCM at Madras University, M.Sc HM at Annamalai University, M.A TM in Madurai Kamaraj University and his Ph.D in Periyar University. He has completed NET 2 times in Tourism Administration and Management & Home Science. He is

having more than 20 years experience. Currently he is working as Principal / Secretary in Food Craft Institute, Hoshiarpur, Punjab, India. His Areas of interest are Food Science & hotel management.