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EFFECT OF GARLIC ADMINISTRATION ON RAT HEART MITOCHONDRIAL ENZYMES AFTER NOISE STRESS EXPOSURE

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ABSTRACT

Background: Noise has been recognized as one of the risk factors for cardiovascular diseases. Garlic is one of the oldest medications used in human culture. Garlic is used in herbal medicine for thousands of years and also as one of the main food ingredient. The present study was aimed to investigate the cardioprotective effect of garlic after noise exposure in Wistar albino rats.

Methods: Animals were exposed to 1 day, 15 days and 30 days of noise stress exposure. The myocardial activity was evaluated by estimating the heart mitochondrial enzymes like malate dehydrogenase, isocitrate dehydrogenase, alpha ketoglutarate dehydrogenase, NADH dehydrogenase and cytochrome C oxidase. Garlic was administered to the animals at the dosage of 125 mg/kg b.w.)

Results: Exposure to 1 day, 15 days and 30 days of noise stress has significantly decreased the all the enzymic activities studied when compared with control. Supplementation of garlic (125 mg/kg b.w.) to 1 day noise stress exposed and 15 days noise stress exposed animals could not produce any significant change and it was similar to that of untreated noise exposed animals and markedly showed a decrease in all the enzyme activity from controls. However, in 30 days aqueous garlic homogenate supplementation has significantly increased all the enzyme activities from the 30 days stress exposed group but its enzymic activity still showed a marked decrease from the control animals.

Conclusion: The result indicated that noise exposure affects the mitochondrial performance in the myocardium and that long-term consumption of garlic is beneficial. As garlic is already under human consumption, dietary supplementation of garlic for prolonged period may be beneficial as one could not avoid noise in the modern way of living.

INTRODUCTION: Noise is one of the most widespread sources of environmental stress in the modern living environment [1]. World Health Organization has declared noise to be an international health problem, when it exceeds an intensity of 90 dB [2]. Further, adaptation to noise does not occur even after 30 days of exposure and stress induced oxidative damage to the brain has been reported [3]. Noise has been recognized as one of the risk factors for cardiovascular diseases, because it increases heart rate [4], peripheral vascular resistance [5] and arterial blood pressure [6, 7, 8].

Recent reports on noise indicate that noise affects all the organ systems in the body. Chronic exposure to environmental noise, characterized by loudness and low frequency, increases the risk of cardiovascular diseases, particularly hypertension and ischemic heart disease, as well as gastrointestinal and respiratory diseases and deteriorates the immune system [9, 10, 11, 12, 13].

Further, the epidemiologic studies on the relationships between heart diseases and noise pollution suggest a higher risk of heart attack in persons exposed to elevated levels of traffic noise [14]. It has been predicted that the cardiovascular disease will be the most important cause of mortality by the year 2015.

Mitochondria exists as 'closed spaces' inside the cell in which most of the energy metabolism takes place, hence known as the power house of the cell. Mitochondrial electron transport chain is widely viewed as the main locus inside the cell for generation of reactive oxygen species [15]. Over the past few years, mitochondrion has emerged as the central regulators of cell death in a variety of disease states. Hence, understanding the mitochondrial function is essential in order to define the mechanisms leading to cell death and to design strategies aimed at improving myocardial protection [16].

Since cardiovascular diseases are the leading cause of mortality and morbidity in the modern world [17], it is necessary to understand the role noise stress on heart mitochondrial enzymes and a possible antidote. It has also been proved that antioxidants decrease cellular oxidation, thereby reducing the damaging effects of noise exposure [18].

Garlic (*Allium sativum* Linn.) is used in herbal medicine for thousands of years [19]. The Egyptians cited 3500 years before, the usage of garlic for the treatment of heart diseases, tumors, bites and worm infections [20]. Garlic contains 0.1-0.36% of volatile oil consisting of sulfur-containing compounds: allicin, diallyl disulfide (DADS), diallyl trisulfide (DATS), etc. These volatile compounds are generally considered to be responsible for most of the pharmacological properties of garlic.

The biological effects of additional constituents of intact garlic are due to lectins, prostaglandins, fructan, pectin, adenosine, vitamins B1, B2, B6, C and E, biotin, nicotinic acid, fatty acids, glycolipids, phospholipids and essential amino acids which have been studied for over several decades [21]. There are several reports supporting the pharmacological importance of garlic for hypoglycemic, hypolipidemic, antimicrobial, antitumor, antioxidant and antiatherosclerotic effects [22, 23, 24, 25, 26, 27].

Most studies on garlic during the past 15 years have been primarily in the cardiovascular field and have been mainly related to atherosclerosis, where effects of different garlic preparations have been examined on serum cholesterol, low-density lipoprotein, high-density lipoprotein, triglyceride and oxidative stress [19, 28].

The present study investigates whether exposure to 1 day, 15 days and 30 days of noise exposure deteriorates the heart mitochondrial enzymes if so whether garlic administration could influences the heart mitochondrial enzymes.

To the extent if it occurs, it suggests that garlic homogenate may provide a nutritional benefit for people exposed to noise stress environment.

MATERIALS AND METHODS:

Plant materials: Garlic bulbs were obtained from a commercial source and were identified and the voucher specimen (CAS-BH-15) was deposited at the Center for Advanced Studies, Department of Botany, University of Madras, Guindy campus, Chennai, Tamil Nadu, India.

Preparation of the aqueous Garlic Homogenate: Garlic cloves were peeled, sliced, ground into a paste and then suspended in distilled water and a concentration of 125 mg/kg was administered to the rats[29]. Rats were fed freshly prepared aqueous garlic homogenate within 30 min of its preparation.

Animals: Healthy adult male Wistar albino rats, weighing 150-170 g were used in this study. Animals were maintained in standard conditions with food and water *ad libitum*, under 12 h light/dark cycle (lights on at 07:00 h) at 25±2°C. All animal procedures were approved by the Institutional Animal Ethical Committee (IAEC) (IAEC No. 08/032/07), University of Madras, Chennai, India as well as by the Committee for Purpose of Control and Supervision of Experiments on Animals (CPCSEA), Government of India.

Noise Stress Procedure: Animals were exposed to noise level of 100 dB (4 h/day) by a white noise generator (0-26 kHz) connected to two loudspeakers (15 W) located 30 cm above the animal cage. The background noise level in the room was recorded to be 44±2 dB, due to the ventilation system. The intensity of the sound was measured by a sound level meter (Cygnet systems-D 2023 Serial No. F02199, India) and maintained at 100 dB intensity [30]. To avoid the influence of handling stress on evolution of effects due to noise exposure, control rats were kept in the above described cage during the corresponding period of experiment, without exposing to noise. Animals were exposed to 1 day, 15 days and 30 days of noise stress.

Experimental groups: Animals were divided into eight groups with six rats in each group. Group I: Control animals. Group II: 45 days fresh garlic administered animals. Group III: 1 day noise stress exposed animals. Group IV: 15 days fresh garlic pretreatment and 1 day noise stress exposed animals. Group V: 15 days noise stress exposed animals. Group VI: 15 days fresh garlic pretreatment and 15 days noise stress + simultaneous fresh garlic treatment. Group VII: 30 days noise stress exposed animals. Group VIII: 15 days fresh garlic pretreatment and 30 days noise stress + simultaneous fresh garlic treatment.

Heart Mitochondria Isolation and Enzyme estimation: At the end of experiment animals were euthanized by cervical dislocation and the heart was dissected out and was homogenized in ice-cold medium containing sucrose (0.32 mM), ethylene diamine tetra acetic acid (1 mM) and Tris–HCl (10 mM; pH 7.4) at 4°C using Potter–Elvehjem homogenizer with a motor driven pestle. The homogenates were then centrifuged at 1000*g* for 5 min at 4°C.

The supernatants were transferred to separate tubes and the pellets containing unbroken cells, tissues and nucleus were discarded. The supernatants were then centrifuged at 12,000*g* at 4°C for 10 min. The supernatant was separated and the pellet containing mitochondrial fraction was kept in ice. The mitochondrial pellets were resuspended in 1 ml of Tris-HCl buffer (0.01 M; pH 7.4) and were maintained in ice until use [31].

The mitochondrial enzymes namely malate dehydrogenase (EC 1.1.1.37) [32], isocitrate dehydro genase (EC 1.1.1.42) [33], α-ketoglutarate dehydro genase (EC 1.2.4.2) [34], succinate dehydrogenase (EC 1.3.5.1) [35], NADH dehydrogenase (EC 1.6.5.3) [36] and cytochrome C oxidase (EC 1.9.3.1) [37] were estimated in mitochondrial fraction within 24 h after their isolation from the heart tissues.

Statistical Analysis: Data are expressed as mean ± SD. Data comparisons were carried out by one-way analysis of variance (ANOVA) followed by Tukey's multiple comparison fixing the significant level at $p < 0.05$. SPSS 12.0 software was used to analyze the data.

RESULTS: The data from different durations of noise stress exposure and garlic treated groups are shown in **Figure 1 and Table 1**. Exposure to 1 day, 15 days and 30 days of noise stress has significantly decreased the activity of the malate dehydrogenase (df 7, F 28), isocitrate dehydrogenase (df 7, F 24), α-ketoglutarate dehydrogenase (df 7, F 33), succinate dehydrogenase (df 7, F 21), NADH dehydrogenase (electron transport chain complex I enzyme) (df 7, F 28) and cytochrome C oxidase (electron transport chain complex IV enzyme) (df 7, F 48) when compared with control animals as well as control animals treated with garlic. Also 30 days noise stress exposed animals showed a significant decrease in all the enzyme activities studied compared with 1 day and 15 days noise exposed animals.

Supplementation of garlic to 1 day noise stress exposed and 15 days noise stress exposed animals could not produce any significant change and it was similar to that of untreated noise exposed animals and markedly showed a decrease in all the enzyme activity from controls. However, in 30 days aqueous garlic homogenate supplementation has significantly increased the malate dehydrogenase, isocitrate dehydrogenase, α-ketoglutarate dehydrogenase, succinate dehydrogenase, NADH dehydrogenase activity from the 30 days stress exposed group but its enzymic activity still showed a marked decrease from the control animals indicating long-term consumption of garlic may be beneficial.

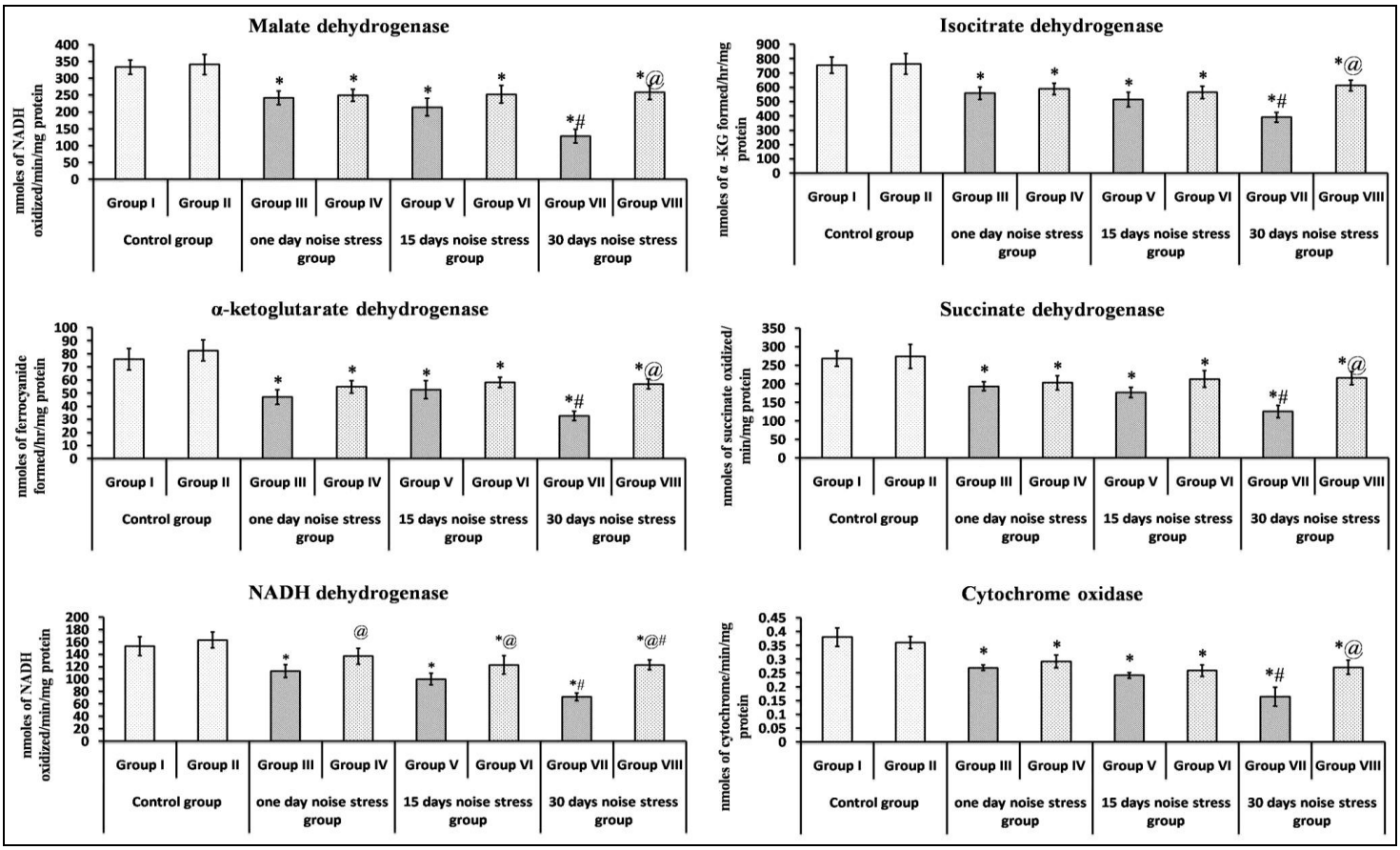

FIGURE 1: EFFECT OF THE GARLIC HOMOGENATE ON HEART MITOCHONDRIAL ENZYMES IN WISTAR ALBINO RATS

Values are Mean ± SD. * indicates significant when compared with group I and Group II, @ indicates significant when compared with its respective stress group, # indicates significant when compared with 1day and 15 noise stress exposure. $p<0.05$ is considered significant.

TABLE 1: EFFECT OF THE GARLIC HOMOGENATE ON HEART MITOCHONDRIAL ENZYMES IN WISTAR ALBINO RATS

Groups	Heart mitochondrial enzymes					
	Malate dehydrogenase	Isocitrate dehydrogenase	α-ketoglutarate dehydrogenase	NADH dehydrogenase	Succinate dehydrogenase	Cytochrome-C-oxidase
Group I	332.77 ± 21.32	753.43 ± 57.47	75.81 ± 8.12	152.93 ± 15.21	267.56 ± 20.75	0.37 ± 0.03
Group II	340.21 ± 30.22	762.43 ± 72.94	82.39 ± 7.98	162.56 ± 12.67	273.79 ± 32.57	0.35 ± 0.02
Group III	241.16 ± 20.20*	557.53 ± 43.36*	46.94 ± 5.54*	112.40 ± 10.40*	192.79 ± 11.77*	0.26 ± 0.01*
Group IV	249.03 ± 18.26*	586.53 ± 41.79*	54.62 ± 4.73*	136.45 ± 12.67@	202.44 ± 19.09*	0.29 ± 0.02*
Group V	213.77 ± 25.88*	512.85 ± 50.04*	52.43 ± 6.90*	99.54 ± 9.29*	175.99 ± 14.38*	0.24 ± 0.01*
Group VI	251.99 ± 26.47*	562.86 ± 43.41*	58.12 ± 3.96*	122.49 ± 14.93*@	212.43 ± 22.41*	0.25 ± 0.02*
Group VII	127.35 ± 20.10*#	388.86 ± 34.29*#	32.42 ± 3.58*#	71.25 ± 6.21*#	124.66 ± 17.13*#	0.16 ± 0.03*#
Group VIII	257.33 ± 20.99*@	611.52 ± 37.95*@	56.83 ± 3.71*@	122.33 ± 7.89*@#	214.95 ± 18.38*@	0.26 ± 0.02*@

Values are Mean ± SD. * indicates significant when compared with group I and Group II, @ indicates significant when compared with its respective stress group, # indicates significant when compared with 1day and 15 noise stress exposure. $p<0.05$ is considered significant. Values are expressed as: MDH—nmoles of NADH oxidized/min/mg protein; ICDH—nmoles of α -KG formed/hr/mg protein; α -KGDH—nmoles of ferrocyanide formed/hr/mg protein; SDH—nmoles of succinate oxidized/ min/mg protein; NADH dehydrogenase—nmoles of NADH oxidized/min/mg protein; Cytochrome-C-oxidase—nmoles of cytochrome/min/mg protein.

DISCUSSION: The most likely metabolic source of reactive oxygen species (ROS) is the mitochondrion where 'leakage' of superoxide occurs [38] in proportion to the rate of oxygen consumption [39]. Increased metabolic activity with intensified reactive oxygen species production during noise exposures were also reported [40]. In the present study the suppression of mitochondrial tricarboxylic acid cycle enzymes (malate dehydrogenase, isocitrate dehydrogenase, α-ketoglutarate dehydrogenase, succinate dehydrogenase) and electron transport chain Complex I (NADH dehydrogenase) and Complex IV enzymes (cytochrome C oxidase) was observed. The decrease in these enzymes clearly indicates a reduction in the glucose metabolism and possibly a reduction in oxygen supply.

Increase in the heart rate and blood pressure has been observed in using grinder unit with 90 dB noise level [41]. Hence in our study, 100 dB used was definitely a stressor and might increase the autonomic response. Noise influences blood pressure and heart rate by its direct effect on arterial wall tension and by the activation of adrenergic system [42]. It has been postulated that persons exposed to environmental noise for prolonged periods, might develop increased blood pressure and thus an increased risk for cardiovascular diseases [43].

It is a known fact that stress activates the sympathetic nervous system which in turn increases the heart rate and it is also essential to remember that only at the expense of diastolic duration the heart rate increases. However, the myocardium receives blood supply only during diastolic phase and diastolic time is an important determinant of myocardial oxygen supply [44]. It is now clear that during stress there will be a reduction in blood supply thereby a decrease in the oxygen level.

A sufficiently prolonged reduction in coronary blood flow can result in severe damage to the myocardium; this causes cellular injury and eventually leads to cell death due to apoptosis and/or necrosis [45]. Again the fall in these enzyme activities is well correlated by few reports. The increase in energy demands [46] and the decrease in blood flow induce an energy imbalance by the Ca^{2+} overload [47]. This is accompanied by disruption of the mitochondria [48], with inactivation of tricarboxylic acid cycle enzymes and an altered mitochondrial respiration [49]. Hypoxia in mammalian cells is often correlated with a reduction in cytochrome levels and mitochondrial enzyme activities [50]. A significant increase in systolic blood pressure, diastolic blood pressure and heart rate in individuals working in lock factories have also been reported [41]. Moreover, it has been reported that both acute and subchronic noise stimulations lead to ultrastructural modifications in atrial myocardial mitochondria [51].

Particularly in heart muscle, extraction ratio for oxygen is high. Hence, the decrease in oxygen level may inhibit these enzymes. Most studied and reported health-promoting effect of garlic is cardioprotective [52, 53, 54, 55]. Several flavonoids were found to possess a vasodilatory effect [56, 57, 58, 59]. Since garlic contains flavonoids [60], the above effect may be mediated by garlic. It is also well established that Allium species has been found to be antihypertensive [61]. Although garlic appears to hold promise in reducing parameters associated with cardiovascular disease, more in-depth investigations are required. S-allylcysteine is a sulfur containing amino acid derived from garlic and has been reported to have antioxidant property [62].

Pretreatment with S-allylcysteine for a period of 45 days significantly increased the activities of mitochondrial and respiratory chain enzymes and decreased the activities of lysosomal enzymes in isoprotereno induced cardiac toxicity in rat [63]. In this study, restoration of mitochondrial enzymes towards normal by garlic homogenate treatment was observed only in 30 days noise stress exposed treated animals but it still showed a marked decrease from control indicating that the noise-induced factors are multi-facet and the dose of garlic used may not be adequate to overcome these changes induced by noise stress.

CONCLUSION: From the above results, it is clear that noise affects the mitochondrial performance in the myocardium and that long-term consumption of garlic is beneficial. Moreover, control animals fed with garlic did not show any alteration in the enzymes, its usage is recommended. As it is under human consumption, based on the current study reports dietary supplementation of garlic for prolonged period may be beneficial as one could not avoid noise in the modern way of living.

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REFERENCES:

1. Wallenius MA: The interaction of noise stress and personal project stress on subjective health. Journal of Environmental Psychology 2004; 24:167-177.
2. Ramsey JM: Modern stress and disease process. Basic physiology. Addison-Wesley, California, 1982.
3. Samson JD, Wiktorek SA, Politanski P, Rajkowska E, Pawlaczyk LM, Dudarewicz A, Sha SH, Schacht J and Sliwinska KM: Noise induced time dependent changes in oxidative stress in the mouse cochlea and attenuation by d-methionine. Neuroscience 2008; 152:146-150.
4. Linden W, Franckish J and McEachern HM: The effect of noise interference type of cognitive stressors and order of task on cardiovascular activity. International Journal of Psychophysiology 1985; 3:67-74.
5. Britan D, Carlson D, Leschiner S and Gavish M: Ovarian steroids and stress produce changes in peripheral benzodiazepine receptor density. European Journal of Pharmacology 1998; 361:235-242.
6. Cohen S, Evans GW, Krantz DS and Stokols D: Physiological, motivational and cognitive effects of aircraft noise on children: moving from the laboratory to the field. American Psychologist 1980; 35:231-243.
7. Talbott EO, Findlay RC, Kuller LH, Lenkner LA, Matthews KA, Day RD and Ishii EK: Noise-induced hearing loss: a possible marker for high blood pressure in older noise-exposed populations. Journal of Occupational Medicine 1990; 32:690-697.
8. Altura BM, Altura BT, Gebrewold A, Ising H and Gunther T: Noise-induced hypertension and magnesium in rats: relationship to microcirculation and calcium. Journal of Applied Physiology 1992; 72:194-202.
9. Hessel PA and Sluis-Cremer GK: Occupational noise exposure and blood pressure: longitudinal and cross-sectional observations in a group of underground miners. Archives of Environmental Health 1994; 49:128-134.
10. Castro AP, Aguas AP, Grande NR, Monteiro E and Castelo BNA: Increase in $CD8^+$ and $CD4^+$ T lymphocytes in patients with vibroacoustic disease. Aviation Space and Environmental Medicine 1999; 70:A141-A144.
11. Van Campen LE, Murphy WJ, Franks JR, Mathias PI and Toraason MA: Oxidative DNA damage is associated with intense noise exposure in the rat. Hearing Research 2002; 164:29-38.
12. Van Kempen E, Kruize H, Boshuizen H, Ameling CB, Staatsen BA and de Hollander AE: The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environmental Health Perspectives 2002; 110:307-308.
13. Yamashita D, Jiang HY, Schacht J and Miller JM: Delayed production of free radicals following noise exposure. Brain Research 2004; 1019:201-209.
14. Babisch W, Beule B, Schust M, Kersten N and Ising H: Traffic noise and risk of myocardial infarction. Epidemiology 2005; 16:33-40.
15. Shoal RS and Brink UT: Mitochondrial production of pro-oxidants and cellular senescence. Mutation Research 1992; 275:295-304.
16. Suleiman MS, Halestrap AP and Griffiths EJ: Mitochondria: a target for myocardial protection. Pharmacology & Therapeutics 2001; 89:29-46.
17. Jialal I and Devaraj S: Low-density lipoprotein oxidation, antioxidants, and atherosclerosis: a clinical biochemistry perspective. Clinical Chemistry 1996; 42:498-506.
18. Quirk WS, Shivapriya BG, Schwimmer CL and Seidman MD: Lipid per oxidation inhibitor attenuates noise-induced temporary threshold shifts. Hearing Research 1994; 74:217-220.
19. Ramaa CS, Shirode AR, Mundada AS and Kadam VJ: Nutraceuticals - an emerging era in the treatment and prevention of cardiovascular diseases. Current Pharmaceutical Biotechnology 2006; 7:15-23.
20. Rahman K: Historical perspectives on garlic and cardiovascular disease. Journal of Nutrition 2001; 131:977S-979S.
21. Fenwick GR and Hanley AB: The genus Allium. Part 2. Crit. Rev. Food Science 1985; 22:273-377.
22. Agarwal K: Therapeutic actions of garlic constituents. Medicinal Research Reviews 1996; 16:111-124.
23. Alder R, Lookinland S, Berry JA and Williams M: A systematic review of the effectiveness of garlic as an antihyperlipidemic agent. J Journal of the American Academy of Nurse Practitioners 2003; 15:120-129.
24. Banerjee SK and Maulik SK: Effect of garlic on cardiovascular disorders: A review. Nutrition Journal 2002;1 :4.
25. Brace LD: Cardiovascular benefits of garlic (Allium sativum L.). Journal of Cardiovascular Nursing 2002; 16:33-49.
26. Elkayam A, Mirelman D, Peleg E, Wilchek M, Miron T, Rabinkov A, Herman MO and Rosenthal T: The effects of allicin on weight in fructose induced hyperinsulinemic, hyperlipidemic, hypertensive rats. American Journal of Hypertension 2003; 16:1053-1056.
27. Orekhov AN, Tertov VV, Sobenin IA and Pivovarova EM: Direct anti-atherosclerosis related effects of garlic. Annals of Medicine 1995; 27:63-65.
28. Zhang XH, Lowe D, Giles P, Fell S, Connock MJ and Maslin DJ: Gender may affect the action of garlic oil on plasma cholesterol and glucose levels of normal subjects. Journal of Nutrition 2001; 131:1471-1478.
29. Banerjee SK, Sood S, Dinda AK, Das TK and Maulik SK: Chronic oral administration of raw garlic protects against isoproterenol-induced myocardial necrosis in rat. Comparative Biochemistry and Physiology – Part C 2003; 136:377-386.
30. Archana R and Namasivayam A: Effect of Ocimum sanctum on noise induced changes in neutrophil functions. Journal of Ethnopharmacology 2000; 73:81-85.
31. Johnson D and Lardy H: Isolation of liver or kidney mitochondria, In R. W. Estabrook and M. E. Pullman (ed.), Methods in Enzymology, vol. 10. Academic Press Inc., New York. 1967.
32. Mehler AH, Konberg A, Criscolin S and Ochon S: The enzymatic mechanism of oxidation reductions between malate or isocitrate or pyruvate. Journal of Biological Chemistry 1948; 174:961-977.
33. Bell LJ and Baron DN: A colorimetric method for determination of isocitric dehydrogenase. Clinica Chimica Acta 1960; 5:740-747.
34. Reed LJ and Mukherjee RB: α-Ketoglutarate dehydrogenase complex from Escherichia coli. Methods in Enzymology 1969; 13:55-61.
35. Slater ECC and Bonner WD: The effect of fluoride on succinic oxidase system. Biochemical Journal 1952; 52:185-196.
36. Minakami S, Ringler RL and Singer SP: Studies on the respiratory chain linked dihydrodiphosphopyridine nucleotide dehydrogenase, assay of the enzyme in particulate and in soluble preparations. Journal of Biological Chemistry 1962; 237:569-576.

37. Pearl W, Caocaranoj BW and Zeiyach: Micro determination of Cytochrome oxidase in rat tissues by the oxidation of n-phenyl-p-phenylenediamine or ascorbic acid. Journal of Hisochemistry & Cytochemistry 1963; 11:102-107.
38. Nohl H and Hegner D: Do mitochondria produce oxygen radicals in vivo? European Journal of Biochemistry 1978; 82:563-567.
39. Boveris A and Chance B: The mitochondrial production of hydrogen peroxide. General properties and effect of hyperbaric oxygen. Biochemical Journal 1973; 134:707-716.
40. Samson J, Sheela Devi R and Ravindran R: Oxidative stress in brain and antioxidant activity of Ocimum sanctum in noise exposure. Neurotoxicology 2007; 28:679-685.
41. Sangeeta S, Berendra Y, Hashmi SF and Md Muzammil: Effects of workplace noise on blood pressure and heart rate. Biomedical Research 2009; 20:122-126.
42. Andren L, Hanson L, Bjorkman M and Jonsson A: Noise as a contributing factor in the development of elevated arterial pressure. Acta Medica Scandinavica 1980; 207:493-498.
43. Rylander R: Physiological aspects of noise-induced stress and annoyance. Journal of Sound and Vibration 2004; 277:471-478.
44. Boudoulas H, Rittgers SE, Lewis RP, Leier CV and Weissler AM: Changes in diastolic time with various pharmacologic events. Circulation 1979; 60:164-169.
45. Cuong DV, Kim N, Joo H, Youm JB, Chung JY, Lee Y, Park WS, Kim E, Park YS and Han J: Subunit composition of ATP sensitive potassium channels in mitochondria of rat hearts. Mitochondrion 2005; 5:121-133.
46. Prabhu SD, Chandrasekar B, Murray DR and Freeman GL: β-Adrenergic blockade in developing heart failure: effects on myocardial inflammatory cytokines, nitric oxide, and remodeling. Circulation 2000; 101:2103-2109.
47. Sanchez VC, Hernandez-Munoz R, Barrera FL, Yanez L, Vidrio S, Suarez J, Cota-Garza MD, Fraustro AA and Cruz D : Sequentiel changes of energy metabolism and mitochondrial function in myocardial infarction induced by isoproterenol in rats: a long-term and integrative study. Canadian Journal of Physiology and Pharmacology 1997; 75:1300-1311.
48. Xia T, Jiang C, Li L, Wu C, Chen Q and Liu S: A study on permeability transition pore opening and cytochrome c release from mitochondria, induced by caspase-3 in vitro. FEBS Letters 2002; 510:62-66.
49. Prabhu S, Jainu M, Sabitha KE and Shyamala Devi CS: Effect of mangiferin on mitochondrial energy production in experimentally induced myocardial infracted rats. Vascular Pharmacology 2006; 44:519-525.
50. St-Pierre J, Glenn J, Tattersall and Robert GB: Metabolic depression and enhanced O_2 affinity of mitochondria in hypoxic hypometabolism. American Journal of Physiology - Regulatory, Integrative and Comparative Physiology 2000; 279:R1205-R1214.
51. Paparelli A, Pellegrini A, Lenzi P, Gesi M and Soldani P: Ultrastructural changes in atrial tissue of young and aged rats submitted to acute noise stress. Journal of Submicroscopic Cytology and Pathology 1995; 27:137-142.
52. Banerjee SK, Maulik M, Mancahanda SC, Dinda AK, Gupta SK and Maulik SK: Dose-dependent induction of endogenous antioxidants in rat heart by chronic administration of garlic. Life Science 2002; 70:1509-1518.
53. Durak I, Ozturk HS, Olcay E, Can B and Kavutcu M: Effects of garlic extract on oxidant/antioxidant status and atherosclerotic plaque formation in rabbit aorta. Nutrition Metabolism and Cardiovascular Diseases 2002; 12:141-147.
54. Williams MJA, Sutherland WHF, McCormick MP, Yeoman DJ and de Jong SA: Aged garlic extract improves endothelial function in men with coronary artery disease. Phytotherapy Research 2005; 19: 314-319.
55. Zahid AM, Hussain ME and Fahim M: Antiatherosclerotic effects of dietary supplementations of garlic and turmeric: restoration of endothelial function in rats. Life Science 2005; 77:837-857.
56. Havsteen BH: The biochemistry and medical significance of the flavonoids. Pharmacology & therapeutics 2002; 96:67-202.
57. Middleton E, Kandaswami C and Theoharides TC: The effects of plant flavonoids on mammalian cells: implications for inflammation, heart disease and cancer. Pharmacological Reviews 2000; 52:673-751.
58. Kim HP, Son KH, Chang HW and Kang SS: Anti-inflammatory plant flavonoids and cellular action mechanisms. Journal of Pharmaceutical Sciences 2004; 96:229-245.
59. Kim SH, Kang KW, Kim KW and Kim ND: Procyanidins in crataegus extract evoke endothelium-dependent vaso-relaxation in rat aorta. Life Science 2000; 67:121-131.
60. Bozin B, Mimica-Dukic N, Samojlik I, Goran A and Igic R: Phenolics as antioxidants in garlic (Allium sativum L., Alliaceae). Food Chemistry 2008; 111:925-929.
61. Srinivasan V: A new antihypertensive agent. Lancet 1969; 294:800.
62. Imai J, Ide S, Moriguchi T, Matsuura H and Itakura Y: Antioxidant and radical scavenging effects of aged garlic extract and its constituents. Planta Medica 1994; 60:417-420.
63. Padmanabhan M and Stanely MP: S-allylcysteine ameliorates isoproterenol-induced cardiac toxicity in rats by stabilizing cardiac mitochondrial and lysosomal enzymes. Life Science 2007; 80:972-978.

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