AEROBIC TRAINING REDUCES THE RISK FACTORS OF CORONARY HEART DISEASES AND ENHANCES THE ANTIOXIDANT STATUS AMONG MIDDLE AGED OBESE MEN

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ABSTRACT

Regular exercise training is recognized as a powerful tool to improve work capacity, endothelial function and the cardiovascular risk profile in obesity. To achieve this purpose the present research was undertaken to investigate that the effect of aerobic training programme performed at different intensities reduces the lipid profiles and enhances the antioxidant status in middle aged obese men. In our study thirty men with coronary heart disease and their age ranges between 35 and 40 years were selected as subjects. They were divided into three groups with 10 members of each. Group I and Group II treated as experimental groups performing aerobic training at low intensity and medium intensity whereas group III remains as control with no practice of aerobic training other than regular activities. Aerobic training programme was conducted for a period of 3 months (3days/week) to the experimental groups. Data were collected and biochemical analysis was done. We observed significant alterations on lipid profiles and enhancement in the antioxidant status in aerobic training groups. Studies also proved that better effect was seen in medium intensity of aerobic training groups than others. Results were statistically analyzed using Anova and DMRT and are significant at p<0.05. Hence the study concludes that aerobic training plays a vital role in reducing the risk factors of cardiac disease by retaining the antioxidant status and shows the importance of health benefits of the today's well being.

Keywords: Antioxidants, Lipid Profile, Aerobic Training, Lipo Proteins, Coronary Heart Disease (CHD), Total Radical Antioxidant Potential (TRAP)

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INTRODUCTION

Obesity has become a major health, social and economical burden of today's world (James et al., 2004). It has now been well established that obesity directly increases cardio metabolic risk by altering the secretion of adipokines and, indirectly, by promoting insulin resistance and its associated metabolic disorders, such as Type-2 diabetes. Moreover, obesity causes additional health problems as it is closely associated with the development and progression of coronary heart disease, certain forms of cancer, respiratory complications (e.g. obstructive sleep apnoea) and osteoarthritis (Kopelman, 2000).

Cardiovascular risk profiling attempts to establish the absence or presence of a number of risk factors that, together with overweight and obesity, contribute to the progression of cardiovascular disease, such as endothelial dysfunction, hypertension, inactivity and poor exercise capacity (Inga E. Schjerve et al.,2008). Both overweight and obesity appear to be associated with low aerobic capacity and impaired endothelial function (Watts et. al, 2004).

Endurance training improves both aerobic capacity (Rognmo et al, 2004; Wisloff et. al., 2007) and endothelial function (Wisloff et. al, 2007; Meyer et. al., 2006), and is now increasingly recommended in the prevention and treatment of overweight and obesity (Haskell et, al., 2007). It has been known that endurance exercise training, decreases cardiovascular risk, but an optimal training programme has not yet been identified. Similarly, criteria for the minimum protective exercise programme against overweight and obesity have not been established. Although the recommended exercise intensity spans the range 40–90% of VO₂max (maximal oxygen uptake), most studies indicate that high-intensity exercise, i.e. toward the upper end of the range, results in larger aerobic and cardiovascular adaptations (Dubach et. al., 1997; Hambrecht, et. al., 2000; Lee et. al., 2003), and many rehabilitation programmes advocate the use of low-to-moderate-intensity exercise.

Exercise remains a key aspect of a healthy lifestyle. However, strenuous physical exercise results in an enhanced uptake of oxygen leading to increased metabolism, which can increase the production of reactive oxygen species (ROS) (Kelle et al., 1998). Cells continuously produce free radicals and ROS as part of metabolic processes. These free radicals are neutralized by an elaborate antioxidant defense system consisting of enzymes such as catalase, superoxide dismutase, glutathione peroxidase, and numerous non-enzymatic antioxidants, including vitamins A, E and C, glutathione, ubiquinone, and flavonoids. Exercise can cause an imbalance between ROS and antioxidants, which is referred to as oxidative stress (Urso and Clarkson, 2003).

Oxidative stress may occur due to an increase in free radical production and/or a decrease in antioxidant defenses (Mustafa Gul et al., 2003). Without the intervention of the cell's antioxidant defense mechanisms, free radical-mediated lipid peroxidation can lead to the loss of the integrity of cell membrane and tissue damage (Maxwell, 1995; Clarkson and Thompson, 2000). The efficiency of the antioxidant defense system depends on the balanced diet.

Multiple enzymatic and non-enzymatic antioxidant defense systems are present in cells to protect the membranes and other cell organelles from the damaging effects of free radical reactions (Evans, 2001). While regular exercise training is associated with numerous health benefits, it can be viewed as an intense physical stressor leading to increased oxidative cellular damage, likely due to enhanced production of ROS (Bloomer et al., 2005). Though many studies have reported that acute aerobic exercise contributes to oxidative stress, especially when performed at high intensity levels (Muaz Belviranli and Hakki Gokbel, 2006). Our present study was carried out to find whether aerobic exercise performing at different intensity reduces lipid profiles and increases antioxidant levels. It also determines a relative effect in depletion of free radicals generation leading to lipid peroxidation and thereby protects the cells by enhancing the antioxidant status.

METHODOLOGY

Selection of Subjects: Thirty men were randomly selected as subjects with coronary heart disease from R.M.M.C&H, Annamalai University and their age ranged between 35 and 40 years. Selected subjects were divided into three groups with ten members in each.

Experimental Design: The primary purpose of the study was to investigate the effect of different intensities of aerobic training on coronary heart disease middle aged obese men. The selected subjects were divided into three groups performing aerobic exercise at different levels.

Group I initiates their work load with 20% of aerobic training ends with 45% (low intensity) for twelve weeks (3days/week)

Group II starts their workload with 35% of aerobic training and ends with 50% (medium intensity) for twelve weeks (3days/week)

Group III served as control who did not participate any special training apart from the regular activities.

Aerobic exercise training: Aerobic exercise training program was conducted in the Department of Physical Education and Sports Science Annamalai University for a period of 3 months (3 days/week). Each exercise session consisted of three components: (i) a 10 min warm-up period consisting of stretching and low-level calisthenics, (ii) a 20 –30 min period of aerobic exercise, and (iii) a 10 min cool-down period also involving low-level calisthenics and walking. Aerobic exercise (for example, fast walking, jogging, and swimming) should be the mainstay of exercise training in patients with CHD. Supervised exercise training programs are also beneficial, especially during the initiation period. They ensure that patients are exercising safely, and permit one to assess progress. Biochemical analysis was done and measured using the appropriate test. All the groups were tested before and after the training period of twelve weeks.

AEROBIC TRAINING REDUCES THE RISK FACTORS

MONTHLY GOALS WEEKLY SCHEDULE								
			Ι	DAY				
WORK OUT	1	2	3	4	5	6	7	
Aerobic	¢		¢		¢			
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Selection of variables:

The following coronary heart disease (CHD) variables were selected as testing variables and they were Lipid peroxidative indices such as TBARS (thiobarbituric acid reactive substances) and hydroperoxides Plasma Lipid profiles such as Cholesterol, Triglycerides, phospholipids and free fatty acids.

Plasma lipoproteins are High density lipoproteins (HDL), Low density lipoproteins (LDL), Very low density lipoproteins (VLDL), and Enzymic antioxidants include Superoxide dismutase (SOD), Catalase (CAT) and Glutathione peroxidase (GPX) and Non-enzymic antioxidants such as Vitamin C, Vitamin E and Reduced Glutathione (GSH).

Biochemical estimations:

Lipid peroxidative indices such as TBARS were estimated by the method of Ohkawa et al., (1979) and Hydro peroxides by Jiang et al., (1992). Estimation of Cholesterol using Allain et al., 1974, triglycerides (Foster and Dunn, 1973), Free fatty acids by (Falholt,1973) and phospholipids (Zilversmit and Davis,1950). Plasma lipoprotein estimated by Boheringer manhim kit. Antioxidants includes enzymic antioxidants such as SOD estimated by the method of Kakkar et al.,(1942), catalase (Sinha, 1972) and GPX (Rotruck etal., 1973). Non enzymic antioxidants include GSH (Ellman, 1959), Vitamin C (Roe and Kuther, 1942), Vitamin E (Baker et al., 1980) were analysed. Biochemical analyses were done accurately in the Department of Biochemistry for the selected variables and the results were reported by the concerned Biochemist to our scholar.

Statistical analysis:

The data were collected before and after the different intensities of aerobic training programme on both the experimental and control group. Biochemical variables were assessed before and AEROBIC TRAINING REDUCES THE RISK FACTORS

after 3 months of aerobic training. The data were analyzed using ANOVA and the group means were compared by Duncan's Multiple Range Test (DMRT). There differences was considered to be significant when p<0.05. Results showed that aerobic training modifies the selected coronary heart risk factors in experimental groups than control. Better improvement was seen in medium intensity of training.

RESULTS:

Table. 1 Changes in the levels of TBARs and hydroperoxides in control and exercise groups

Groups	TBARS(mM)	Hydroperoxides (×10 ⁻⁵ mM)
CHD Low Intensity Work Load	1.10±0.1ª	6.5±0.2 ^a
CHD Medium Intensity Work Load	0.95±0.02 ^b	4.5±0.3 ^b
Control	2.1±.0.1°	8.4±0.3c

Data represents mean± SD from 10 subjects in each group. Values not sharing a common superscript letter (a,b,c) differ significantly at p≤0.05 (Duncan's multiple range test) Group comparison: Group 3 with all, Group2 with 1 and 3.

Table: 2

Changes in the levels of cholesterol, triglycerides, phospholipids and free fatty acids in control and exercise groups

Groups	Cholesterol mg/dl	Triglycerides mg/dl	Phospholipids mg/dl	Freefattyacids mg/dl
CHD Low	263.25 ± 11.5^{a}	192 ± 11.4^{a}	190.2±11.2 ^a	9.2±1.3 ^a
Intensity				
Work Load				
CHD Medium	211.12 ± 17.2 ^b	174.11 ± 18.2 ^b	184.3±14.3 ^b	8.3 ±0.8 ^b
Intensity				
Work Load				
Control	281.12 ± 18.1c	218.9 ± 24.0°	195.7±22.8°	10.7 ±1.2 ^c

7

Data represents mean± SD from 10 subjects in each group.

Values not sharing a common superscript letter (a,b,c) differ significantly at p≤0.05 (Duncan's multiple range test) Group comparison: Group 3 with all, Group2 with 3 and 1.

Table: 3 Changes in the levels of Plasma Lipoprotein in control and exercise groups

Groups	HDL mg/dl	LDL mg/dl	VLDL mg/dl
CHD (Low intensity)	42.3 ± 5.2^{a}	169.2 ± 11.3^{a}	37.65 ± 2.9^{a}
CHD (Medium intensity)	48.2 ± 3.8^{b}	155.5 ± 12.6 ^b	30.8 ± 4.6^{b}
Control	$37.6 \pm 4.6^{\circ}$	$196.3 \pm 21.6^{\circ}$	$43.9 \pm 5.3^{\circ}$

Data represents mean± SD from 10 subjects in each group. Values not sharing a common superscript letter (a,b,c) differ significantly at p≤0.05 (Duncan's multiple range test) Group comparison: Group 3 with all, Group 2 with 3 and 1.

Table.4 Changes in the levels of enzymic antioxidant status in control and exercise group

Groups	SOD (U ^A /mg Hb)	CATALASE (U ^A /mg Hb)	GPX (U ^A /mg Hb)
CHD (Low intensity)	0.93±0.002ª	8.7±0.07ª	55.11±0.4ª
CHD (Medium intensity)	1.36±0.03 ^b	9.8±0.2 ^b	60.33±0.1 ^b
Control	1.7±0.02 ^c	11.8±1.3 ^c	62.11±0.4 ^c

Data represents mean± SD from 10 subjects in each group.

Values not sharing a common superscript letter (a,b,c) differ significantly at p≤0.05 (Duncan's multiple range test)

Group comparison: Group three with all, Group2 with3 and 1.

A- Enzymes required for 50% inhibition of NBT reduction/min/mg Hb

Table: 5

Changes in the levels of non-enzymic antioxidant status in control and exercise groups

Groups	Vitamin E (mg/dl)	Vitamin C (mg/dl)	Glutathione (mg/dl)
CHD Low Intensity	1.18 ± 0.14^{a}	1.32 ± 0.15^{a}	42.3±2.8 ^a
CHD medium Intensity	1.25 ± 0.16^{b}	1.66 ±0.11 ^b	48.2±1.6 ^b
Control	$0.86 \pm 0.07^{\circ}$	$0.83 \pm 0.06^{\circ}$	39.7±3.5°

Data represents mean± SD from 10 subjects in each group. Values not sharing a common superscript letter (a,b,c) differ significantly at p≤0.05 (Duncan's multiple range test) Group comparison: Group 3 with all, Group 2 with 3 and 1.

The levels of TBARS and hydroperoxides were found to be increased in control groups of coronary heart patients. Aerobic training at different intensity lowers the levels of TBARS and hydroperoxides in CHD patients. Medium intensity training exercises shows better effect in reducing the lipid peroxides levels than other groups. (Table.1)

Table 2 shows the levels of cholesterol, triglycerides, free fatty acids and phospholipids in control and exercise groups. The levels of lipid profiles in experimental groups were decreased by performing the aerobic exercise at different intensities. Medium intensity exercise shows significant changes than control and low intensity groups.

Table 3 shows the levels of HDL, LDL and VLDL in control and aerobic training groups. The increased levels of lipoprotein in control group were seen than aerobic training groups. Exercise training for three months shows predominant changes in aerobic training groups when compared to control. We also found significant increase in HDL and decrease in LDL and VLDL levels in work performing at medium level of CHD patients.

SOD, CAT and GPX were significantly decreased in control group of CHD patients. The activities of enzymic antioxidants were significantly elevated in low and medium intensity CHD group. Regular practice of exercise enhances the antioxidant status and it was also found that significant effect was seen only in medium intensity exercise group than other. (Table.4)

Non-enzymic antioxidants such as GSH, vitamin-C and vitamin-E levels were decreased in control group of CHD patients. Hence three months training retained the levels of GSH, vitamin-C and vitamin-E in experimental training groups than control. Much better improvement in the non-enzymic antioxidant status was noticed in performing the aerobic exercise of medium intensity work load. (Table.5)

DISCUSSIONS

Physical activity is widely accepted to be a protective factor against the development of atherosclerotic disease (Berlin and Colditz, 1990). Thiobarbituric reactive substances (TBARS) and hydroperoxides, (HP) a marker of in vivo lipid peroxidation were increased in control group. Therefore, the improved antioxidant status found in the aerobic training groups could efficiently counter balance the oxidative stress produced by exercise.

Exercise has been shown to favorably alter lipids and lipoprotein concentrations, especially to raise plasma HDL-C and lower triglyceride concentration, blood pressure and inflammation and improve insulin sensitivity (Thompson et al., 2001). However our present study shows that different intensity of aerobic training groups reduces the concentration of triglyceride, cholesterol, phospholipids and free fatty acids and thereby promotes the HDL cholesterol. This may be due to the medium intensity of aerobic training though low level training slightly moderates the lipid levels, compared to control, medium intensity of aerobic exercise works better and proved to be efficient in reducing lipid levels thereby enhances the antioxidant status.

Most studies reported that exercise generally lowers plasma triglycerides (TG) concentrations when TG are moderately to markedly elevated, and high intensity exercise is more effective than low intensity exercise (Gyntelberg etal.,1977;Kraus etal.,2002). In our study we found that medium intensity of aerobic training groups are very effective when compared to low level intensity. This study was supported by the recent statements from the Surgeon general (US Department of Health and Human Services,1996), the National Institute of Health Consensus Development Panel on Physical Activity and Cardiovascular Health (NIH, 1996) and the centers for disease control and prevention and the American College of Sports Medicine (Pate etal.,1995) recommend that every adult should accumulate at least

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30 minutes of moderate intensity of physical activity on most preferably all days of the week to prevent CHD and other chronic disease. Recent intervals also suggested that moderate intensity physical activity may have benefits on coronary risk factors than vigorous activities of high intensity (Howard et. al., 2009).

Among the modifications in the lipoprotein profile assigned to regular exercise, the most generally diffused one could be a moderate increase in HDL-C levels (Nakamura et. al., 1983), a wellknown anti atherogenic factor. In this study, different levels of aerobic training exhibited increase in HDL-C concentration in comparison with the control subjects. But better effect was seen in subjects performing aerobic exercises at medium intensity work load.

With regards to LDL-C and VLDL levels, we were able to find variation in aerobic groups when compared to control subjects. We found significant decrease in LDL and VLDL in medium intensity aerobic groups than other. Studies also reported that related to physical activity. It seems that athletes generally show similar or slightly decreased LDL-C concentrations in comparison with sedentary people, whereas a greater reduction is only observed after repeated intense training (Cullinane et. al., 1982).

In the present study resulted that generation of free radicals have been suppressed due to the enhanced level of antioxidants. The study shows increased lipid peroxidation in control group when compared to experimental treated groups. Aerobic training performed at different levels reduces the formation of peroxidation products by increasing the antioxidant levels.

The production of oxygen free radicals is increased during exercise as a result of increases in mitochondrial oxygen consumption and electron transport flux, inducing lipid peroxidation and a series of adaptive responses (Kanter, 1994).

Plasma levels of nonenzymatic antioxidants can be modified by exercise. Consistent with a previous work (Sen ,1993), a high

training status has a favourable influence on plasma TRAP levels; TRAP mainly accounts for the aqueous antioxidant compounds, which include glutathione, ascorbic acid, uric acid and bilirubin (Evelson et. al.,2001)

In the present study, we found a significant increase in the total radical antioxidant potential (TRAP) values of aerobic training groups as compared to samples obtained from non trained individuals of control. But the medium intensity of training shows better effect than other groups. This difference could be assigned to the increment in ascorbic acid levels found in aerobic training groups, Accordingly, different studies (Brites et. al., 1999; Sen, 1993; Dekkers et. al., 1996; Maxwell et. al., 1993) have reported higher levels of ascorbic acid in circulation because of exercise.

Several reports (Jenkins, 1993; Sjodin et. al.,1990) have suggested that the activity of the antioxidant enzymes are increased in skeletal muscles of trained individuals. This response seems to depend on the duration and intensity of exercise. In the present study, we evaluated the activity of superoxide dismutase, catalase and glutathioneperoxidase in plasma samples obtained from aerobic training groups of coronary disease patients and control CHD patients. Trained individuals showed a significantly increased activity of superoxide dismutase, catalase and GPX as compared to sedentary individuals.

Alpha-tocopherol is considered the most important lipid soluble antioxidant, with several other functions as the maintainer of membrane fluidity and stability. This compound directly scavenges peroxyl radicals, which are generated in the lipid moiety of membranes and lipoproteins (Diplock ,1983). Plasma levels of atocopherol were significantly increased in the aerobic training CHD as compared to the control values. This increase in plasma atocopherol levels could attenuate exercise-induced lipid peroxidation by scavenging free radicals and thus protecting the muscle from being damaged during exercise. Moreover, a-

tocopherol has been shown to exert several atheroprotective actions by reducing platelet adhesion and aggregation, decreasing the expression of adhesion molecules that can cause neutrophils to stick to the endothelium, inhibiting K-dependent clotting factors, stimulating nitric oxide production, impairing cell proliferation, and stabilizing the atherosclerotic plaque (Pryor, 2000).

Thus in our study medium intensity of aerobic training plays a vital role in decreasing the blood lipids and lipoprotein levels and retains the antioxidant activity.

CONCLUSION

The study concluded that performing different intensity of aerobic training ameliorates the coronary risk factors in middle aged coronary heart disease obese men by reducing the lipid levels and enhancing the antioxidant status. It was also found that medium intensity of aerobic exercise was very effective in reducing the lipid levels and promotes the antioxidant activity. Thus our present study also assures medium intensity training better safer than vigorous exercises in CHD patients.

REFERENCES

- Allain CC, Poon L S, Chan C S, Richmond W and Fu P C, Enzymatic determination of serum cholesterol, *Clin Chem*, 1974, 20: 470.
- Baker H., Frank O., De Angelis B., Feingold S. Plasma tocopherol in man at various times after ingesting free or acetylated tocopherol. Nutr. Rep. Int. 1980, 21: 531–536.
- Bloomer RJ, Goldfarb AH, Wideman L, McKenzie MJ, Consitt LA. Effects of acute aerobic and anaerobic exercise on blood markers of oxidative stress. J Strength Cond Res 2005;19(2):276-85.

- Brites FD, Evelson PA, García Christiansen M, Nicol MF, Basílico MJ, Wikinski RW et al. Soccer players under regulartraining show oxidative stress but an improved plasmaantioxidant status. Clin Sci 1999; 96:381-5.
- Berlin JA, Colditz GA. A meta-analysis of physical activity in he prevention of coronary heart disease. Am J Epidemiol 1990; 132:612-28.
- Cullinane EM, Siconolfi S, Saritelli A, Thompson PD. Acute decrease in serum triglycerides with exercise: is there threshold for an exercise effect? Metabolism 1982;31:844-7.
- Clarkson PM, Thompson HS, Antioxidants: what role do they play in physical activity and health? Am. J. Clin. Nutr. 2000, 72: 637S-646S.
- Dubach, P., Myers, J., Dziekan, G. et al. Effect of exercise training on myocardial remodeling in patients with reduced left ventricular function after myocardial infarction: application of magnetic resonance imaging. Circulation 1997, 95: 2060–2067
- Dekkers JC, van Doornen LJP, Kemper HCG. The role of antioxidant vitamins and enzymes in the prevention of exercise-induced muscle damage. Sports Med 1996, 21: 213-38.
- Diplock A. The role of vitamin E in biological membranes. In: Biology of Vitamin E. London: Pitman Books;1983.pp. 45-55.
- Evelson P, Travacio M, Repetto M, Escobar M, Llesuy S,Lissi E. Evaluation of total reactive antioxidant potential (TRAP)of tissue homogenates and their cytosols. Arch Biochem Biophys 2001, 2:261-6.
- Evans J.E. Vitamin E, vitamin C, and Exercise Am J Clin Nutr (suppl) 2001, 72: 647S-652S.

- Falholt, K lund B and Falholt W. An easy colorimetric micromethod for routine determination of free fatty acids in plasma, Clin Chim Acta, 1973, 46:105
- Foster L B and Dunn R T, Stable reagents for determination of serum triglycerides by a colorimetric hantzsch condensation method. *Clin Chem*, 1973, 19:338
- Gyatelberg F, Brennan R, Holloszy JO, Schonfeld G, Rennie MJ, Weidman SW. Plasma triglyceride lowering by exercise despite increased food intake in patients with type IV hyperlipoproteinemia. J Clin Nutri 1977, 30: 716-720.
- Haskell, W. L., Lee, I. M., Pate, R. R. et al. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Med. Sci. Sports Exercise 2007, 39: 1423–1434
- Hambrecht, R., Gielen, S., Linke, A. et al. Effects of exercise training on left ventricular function and peripheral resistance in patients with chronic heart failure: a randomized trial. JAMA, J. Am. Med. Assoc. 2000, 283: 3095–3101
- Jenkins RR. Exercise, oxidative stress, and antioxidants: a review. Int J Sport Nutr 1993; 3: 356-75.
- Jiang Z.Y., Hunt J.V., Wolff S.P. Ferrous ion oxidation in thepresence of xylenol orange for detection of lipid hydroperoxides in low density lipoprotein. *Anal. Biochem.* 1992, 202: 384–389.
- James, P. T., Rigby, N. and Leach, R. International Obesity Task Force. The obesity epidemic, metabolicsyndrome and future prevention strategies. Eur. J.Cardiovasc. Prev. Rehabil. 2001, 11: 3–8
- Kraus WE, Houmard JA, Duscha BD, Knetzger KJ, Wharton MB, Mecatney JS, Bales CW, Henes S, Samsa GP, Otvos JD, Kulkarani KR, Slentz CA. Effects of the amount and

intensity of exercise on plasma lipoproteins. N Eng J Med 2002, 347:1483-1492.

- Kanter M.M. Free radicals exercise and antioxidant supplementation. Int J Sport Nutr 1994, 4:205-20.
- Kopelman, P. G. Obesity as a medical problem. Nature 2000, 404: 635–643
- Kakkar P., Das B., Viswanathan P.N. A modified spectrophotometric assay of superoxide dismutase. *Ind. J. Biochem. Biophys.* 1984, 21: 130–132.
- Kelle M, Diken H, Sermet A, Atmaca M, Kocyigit Y. Changes in blood antioxidants status and lipid peroxidation following distance running. Tr. J. Med. Sci. 1998, 28: 643-647.
- Lee, I. M., Sesso, H. D., Oguma, Y. and Paffenbarger, Jr, R. S. Relative intensity of physical activity and risk of coronary heart disease. Circulation 2003, 107: 1110–1116
- Muaz Belviranlı, Hakkı Gökbel Acute Exercise Induced Oxidative Stress And Antioxidant Changes-A Review Eur J Gen Med 2006; 3(3): 126-131
- Maxwell SRJ, Jakeman P, Thomason H. Changes in plasma antioxidant status during eccentric exercise and the effect of vitamin supplementation. Free Rad Res Comm 1993, 19:191-201.
- Maxwell SRJ. Prospects for the use of antioxidant therapies. Drugs. 1995, 49: 345-361.
- Mustafa Gül, Mustafa Atalay, and Osmo Hänninen Endurance Training and Glutathione-Dependent Antioxidant Defense Mechanism in Heart of the Diabetic Rats. Journal of Sports Science and Medicine 2003, 2: 52-61.
- Nakamura N, Uzawa H, Haeda H, Inomoto T. Physical fitness,its contribution to serum high density lipoprotein. Atherosclerosis1983; 48:173-81.

- NIH Consensus Development Panel on physical Activity and cardiovascular health. Physical activity and cardiovascular health. JAMA 1996, 276:241-246.
- US Department of Health and Human Services. Physical Activity and health: A report of the Surgeon General, Atlanta GA: US Department of Health and human services, Centres for disease control and prevention, National centre for chronic Disease prevention and health promotion, 1996.
- Ohkawa H., Onishi N., Yagi K. Assay of lipid peroxidation in animal tissue by thiobarbituric acid reaction. *Anal. Biochem* 1979, 95: 351–358.
- Pryor WA. Vitamin E and heart disease: basic science to clinical intervention trials. Free Radic Biol Med 2000, 28:141-64.
- PateRR, Pratt M, Blair SN et. al., Physical activity and public health, a recommendation from the centres for Disease control and prevention and the American college of sports medicine. JAMA 1995, 273: 402-406.
- Rotruck J.T., Pope A.L., Ganther H.E., Swanson A.B. Selenium:biochemical roles as a component of glutathione peroxidase. Science 1973, 179: 588–590.
- Rognmo, O., Hetland, E., Helgerud, J., Hoff, J. and Slordahl, S. A. High intensity aerobic interval exercise is superior to moderate intensity exercise for increasing aerobic capacity in patients with coronary artery disease. Eur. J. Cardiovasc. Prev. Rehabil. 2004, 11: 216–222
- Roe J.M., Kuether C.A. Detection of ascorbic acid in whole blood, and urine through the 2,4-DNPH derivative of dehydroascorbic acid. J. Biol. Chem. 1942, 147: 399–407.

- Sjodin B, Hellsten-Westing Y, Apple FS. Biochemical mechanisms for oxygen free radical formation during exercise. Sports Med 1990;10:236-54.
- Sen C. Oxidants and antioxidants in exercise. J Appl Physiol 1993;79:675-83.
- Sinha A.K. Colorimetric assay of catalase. Anal. Biochem. 1972, 47: 389–394.
- Thompson PD, Crouse Sf, Good paster B, Kelley D, Moyna N, castello L. The acute versus the chronic response to exercise. Med Sci. Sports Exercise 2001, 33: S438-S445: Discussion S452- S453.
- Urso ML, Clarkson PM. Oxidative stress, exercise, and antioxidant supplementation. Toxicology 2003;189(1-2):41-54
- Watts, K., Beye, P., Siafarikas, A. et al. Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents. J. Am. Coll. Cardiol. 2004, 43, 1823–1827.
- Wisloff, U., Stoylen, A., Loennechen, J. P. et al. Superior cardiovascular effect of aerobic interval training versus moderate continuous training in heart failurepatients: a randomized study. Circulation 2007, 115, 3086–3094.
- Zilversmit D B and Davis A. K. Microdetermination of plasma phospholipids by tricholroacetic acid precipitation. J Lab clin Med, 1950, 35: 155