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REVIEW ARTICLE

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THE ROLE OF PHYSICAL ACTIVITY AND EXERCISE PATTERN IN TYPE 2 DIABETES

ABSTRACT

Life style changes that include a nutritionally balanced diet and increased physical activity(PA) are effective intervention options for persons with pre-diabetes who want to prevent progression to type 2 diabetes mellitus. Type 2 diabetes mellitus (T2DM) and pre diabetic conditions such as impaired fasting glucose (IFG) and impaired glucose tolerance (IGT) are rapidly increasing in prevalence. There is compelling evidence that T2DM is more likely to develop in individuals who are insufficiently active, exercise training, often in combination with other life style strategies, his beneficial effects on preventing the out of T2DM and improving glycemic control in those with pre-diabetes. In addition, exercise training improves cardiovascular risk profile, body composition and cardiorespiratory fitness, all strongly related to better health outcomes. Increased physical activity may also help to prevent or delay the onset of harmful conditions. This review examines the evidence and possible mechanism of type 2 diabetes by which physical activity produces these benefits, and gives a brief review on physical activity and exercise pattern.

KEY WORDS: Metabolic syndrome, Type 2 Diabetes, Physical activity, Exercise Pattern.

INTRODUCTION

In the healthy functioning human body, pancreas releases the insulin which needed to regulate the metabolism of ingested carbohydrates and fats in the body. Inadequate production of insulin by the pancreas or insufficient action of insulin in the main glucose disposal tissues (skeletal Muscle, liver and adipose tissue) leads to elevations in circulating blood glucose levels and can result in the development of type 2 diabetes mellitus (Thomas et al., 2006). Pre-diabetes is a term used to classify persons with impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or both who have elevated risk of developing type 2 diabetes.

Insulin resistance is often present years before IGT is observed or type 2 diabetes is manifested clinically(Hu et al., 2000) and appears to be precipitated by life style factors, such as positive energy balance and physical inactivity (Tanasescu et al., 2003). The presence of a positive caloric balance resulting from insufficient physical activity and a high energy intake appears to initiate the disease process with the accumulation of excess triglycerides and their associated products into specific body regions, including central (Visceral) adipose tissue, liver and skeletal muscle depots. This adipose accumulation into unsubcutaneous adipocytes, which may affect only genetically predisposed individuals, cause reductions in insulin signaling in the main insulin target tissues (muscle, liver and adipose) and a proinflammatory process within liver and adipose tissue that increases the risk or metabolic dysfunction and cardiovascular disease (ATS Committee on proficiency standards for Clinical Pulmonary Laboratories, 2002). The development of type 2 diabetes starts with a progression from normal glucose intolerance with insulin resistance to IGT caused by deterioration in β–cell function with insulin resistance. The reasons for β–cell failure are unclear, but may be related to lipid and glucose toxicity of the β–cells themselves, because individuals with prediabetes and type 2 diabetes are characterized by defects in both insulin secretion and sensitivity, interventions that enhance β–cell function and counteract insulin resistance are effective in preventing the progression of IGT to type 2 diabetes. Given that excess adiposity strongly drives the underlying pathology of metabolic dysfunction, lifestyle interventions that combine calorie restriction with increased PA are thought to be the best nonpharmacological approach to diabetes prevention. Although diet alone has been shown to be more effective in decreasing body mass. The concurrent reduction in body fat (subcutaneous and visceral) and increase in lean mass that occur using physical activity interventions result in a higher resting metabolic rate (Walker et al., 1999) and are likely more important to health. Sufficient physical activity offers

additional benefits to metabolic function such as increased cardiorespiratory fitness (CRF), which has health benefits beyond those associated with weight loss (Poirien and Despare, 2001).

The highest rate is seen among indians (National Health Survey, 1998; Zimmet et al., 1997; Tan et al., 1992). The lowest physical activity rates were seen among those aged 30 – 49 years, which is also the age range for which the incidence for development of new onset type 2 diabetes begins to raise rapidly. This article highlights the potential mechanism underlying the beneficial effect of regular physical activity and exercise training on type 2 diabetes prevention with prediabetes.

PRE–DIABETES AND METABOLIC SYNDROME

Pre–diabetes refers to a metabolic stage that is intermediate between normal glucose homeostasis and diabetes. These conditions refer to patients who have either impaired fasting glucose (IFG) or impaired glucose tolerance (IGT), (Clinical Practice Guidelines, 2000 and Diabetes care, 2003). Patients with either IGT or IFG have about a 10% risk of developing over all type 2 diabetes over the ensuring 6.5 years (De Vegt et al., 2001). Patients with IFG also have a 40% greater risk of mortality from all causes, even after adjusting for other cardiovascular disease risk factors (Saydah et al., 2001). Many of the risk factors for cardiovascular disease are known to cluster in certain individuals. This clustering has been called the metabolic syndrome (Brotman and Girod, 2002) and consists of hypertension, high-density lipoprotein (HDL), cholesterol levels, high triglyceride levels, high plasma glucose concentrations and abdominal obesity. The clinical criteria for diagnosis of the National cholesterol education programme Adult Treatment Panel (ATP III) although physiological mechanism of the metabolic syndrome has not been fully elucidated a major underlying abnormality is insulin resistance (Ferrannini et al., 1991; DeFronzo, 1991).

PHYSICAL ACTIVITY AND OBESITY

It is difficult to overstate the importance of the relationship between lifestyle and the risk of developing type 2 diabetes. A recent study demonstrated that both women and men who have a BMI > 35 kg/m² had a 20-fold increase in their risk of developing diabetes compared to people with a BMI of 18.5-24.9 kg/m (Mokdad et al., 2001 & 2003; Field et al., 2001).

Even after adjustment for BMI, the reduction in diabetes risk remained substantial (17%) (Jeon et al., 2007). In addition, review of six diabetes prevention trials showed that in adults with impaired glucose tolerance or at high risk of cardiovascular disease, increasing moderate physical activity level up to 150 min/wk was associated with lower risk of progression to diabetes independent of weight loss (Gill and coper, 2008). In this study, ≥150 min of moderate aerobic physical activity per week was significantly associated with decreased risk of type 2 diabetes independent of BMI and waist circumference. In the Da Qing Study (Pan et al., 1997) which was designed to investigate the separate effects of physical activity, diet and combined effects of physical activity plus diet on development of diabetes; physical activity independently reduced risk of diabetes incidence. In fact, diabetes risk was reduced by 46% in the physical activity only intervention group without a substantial change in BMI. Approximatly 80 percent of all patients with non-insulin dependent diabetes are obese (National Institute of Health, 1987). The strongest predisposing factors for NIDDM are obesity (West, 1978; Wilson et al., 1981; Karan, 1982 and Marble et al., 1985) and a family history of diabetes (Paffenbarger et al., 1973 and Barrett-Connor, 1989).

Findings from epidemiological studies suggest that regular physical activity prevents unhealthy weight gain and obesity, where as sedentary behaviours such as watching television, working at the computer or playing video games promote obesity (Coakley et al., 1998; Jefferey et al., 2002; Erlichman et al., 2002; Hu et al., 2003a; Saris et al., 2003). Based on data from the nurses 'Health Study' about 30% of new cases of obesity could be prevented by adopting a relatively active lifestyle, including more than 30 min of brisk walking per day and less than 10 h of watching television per week. Epidemiological studies suggest that 45-60 minutes of moderate-intensity physical activity per day may be needed to prevent unhealthy weight gain and obesity (Fogelholm et al., 2000;

Saris et al., 2003). However, there are no RCTs that would have specifically addressed the question of whether physical activity prevents weight gain and fat mass increase over years or whether there is a dose-response relationship between exercise and fat accumulation.

PHYSICAL ACTIVITY AND INSULIN SENSITIVITY

Physical exercise improves insulin sensitivity both acutely and chronically. The acute exercise induced improvement of insulin sensitivity and glucose update of skeletal muscles seems to be related to changes in insulin signalling in response to muscle contraction, such an increased insulin independent translocation of GLUTU glucose transporters to the cell surface (Thompson et al., 2001). The effect is short lived lasting 48 h therefore, to maximize the benefits of physical exercise on insulin sensitivity, exercise should be practical preferable daily. Most exercise training programs that last 3 months or more and are of sufficient intensity and volume have improved insulin sensitivity individually effects on exercise training frequently results in varying degrees of weight loss, and the effect of exercise on insulin sensitivity is stronger if associated with weight reduction. Seperating out the independent effects of exercise training and weight loss on insulin sensitivity can be difficult (Rose, 2003). Even in the absence of weight change, moderate intensity or vigorous endurance or resistance training programs may alter composition of fat distribution over the long term, which can also influence insulin sensitivity.

Although effects on body composition are clearly an important mechanism by which exercise training improves insulin sensitivity over the long term, exercise training also increases GLUTU content in glycogen synthesis activity, mitochandrial enzyme activity and capillary and mitochondrial density in skeletal muscle, improves endothelial function and may alter muscle fibre type, This effect may also contribute to varying degrees to the chronic effect of exercise training on insulin sensitivity. Adipose tissue is a major site for insulin sensitivity, and most obese persons have increased insulin resistance, some degrees of glucose intolerance, or both (Karan et al., 1982; Marble et al., 1985). Physical activity may influence glucose metabolism. Among patients with diabetes, short periods of exercise can lower plasma glucose levels by enhanching the effect of insulin, (Devlin and Horton, 1985; Devlin et al., 1987) and more extended exercise training may improve the action of insulin and glucose tolerance.

PHYSICAL ACTIVITY & GYCOSYLATED HEMOGLOBIN (HbA^{1}C)

Boulez et al. (2001) under took a systematic review and meta-analysis on the effects of structured exercise interventions in clinical trials of $\geq$8 week's duration on HbA1c (A1C) and body mass in people with type 2 diabetes. Post intervention A1C was significantly lower in exercise than control groups (7.65 vs. 8.31%, weighted mean difference -0.66%; P < 0.001). In contrast, post interventions body weight did not differ between the exercise and control groups. Meta-regression confirmed that the beneficial effect of exercise on A1C was independent of any effect on body weight. Therefore, structured exercise programs had a statistically and clinically significant beneficial effect on glycemic control, and this effect was not primarily mediated by weight loss. A subsequent meta-analysis by the same authors, showed that exercise intensity predicted post intervention weighted mean difference in A1C (r =0.91, P = 0.002) to a larger extent than exercise volume (r = - 0.46, P = 0.26). These results provide support for encouraging type 2 diabetic individuals who are already exercising at moderate intensity to consider increasing the intensity of their exercise in order to obtain additional benefits in both aerobic fitness and glycemic control.

Improvement of 1.1% age points in the glycosylated hemoglobin HbA^{1}c, which is clinically very relevant. In a trail of 36 older men and woman, 4 months of strength training added to a weight loss program did not affect the overall amount of weight loss, but fat-free mass increased slightly in the weight loss and strength – training group and decreased somewhat in only the weight loss group (Dunstan et al., 2002). The diet and exercise training group had a 0.8% age point greater decrease in HbA1c. Individuals who have replaced part of endurance training with resistance training have also shown a larger increase in insulin sensitivity as measured by a

glycemic hyper insulinemic clamp than persons in the control group or the endurance training only group (Cuff et al., 2003). The endurance and resistance training group also had larger decreases in weight and abdominal obesity than the control group.

PHYSICAL ACTIVITY AND ENDOTHELIAL FUNCTION

Impaired endothelial function is a common finding among patients with type 2 diabetes (Williams et al., 1996; Caballero et al., 1999). Impaired endothelial vasodilator function in the coronary arteries and micro-vascular may be an important contributor to myocardial ischaemia. Physical exercise has been shown to reduce myocardial perfusion defects in patients with CAD, even in the absence of significant plaque regression and this is believed to be secondary to improved endothelial function. Studies whishes to utilise changes in reactive hyperaemic brachial artery vasodilation as an inside of endothelial health have shown that exercise training restores endothelial fuction among subjects with type 2 diabetes as well as those with the metabolic syndrome (Maiorana et al., 2001; Lavrencic et al., 2000; Hosokawa et al., 2003), measured coronary endothelial fuction dierectly by infusing acetylcholine into the coronary arteries of patients (both diabetic and non-diabeteic) who had suffered a recent myocardial infraction, they also measured artery diameter by contrast angiography. They demonstrated that six months of exercise training improved coronary endothelial response to acetylcholine, independent of type 2 diabetes, smoking status, use of status or calcium channel blockers and lipid lowering. Exercise – induced improvements in endothelial functions are thought to be secondary to the elevated stress that occurs during exercise on vessel walls, which results in upregulation of endothelium – derived nitric oxide, leading to improved smooth muscle relaxation and vasodilation. In healthy individuals, the effect of exercise training on endothelial function has appeared, conflicting on the otherhand, exercise has consistently improved endothelial function, most often measured as flow-mediated dilation of the brachial artery, in individuals with obesity, hypertension, diabetes, hyper cholesterol, CVD and heart failure, all of which are characterized by endothelial dysfuction. Many of these conditions or diseases are part of or are related to the metS, which also associated with endothelial dysfuction. Physical training would also improve endothelial function in individuals.

SEDENTARY LIFE STYLE AND PHYSICAL INACTIVITY

In large, prospective, epidemiological status, sedentary occupations and sedentary activities such as watching television and playing video games are associated with an increased risk of developing obesity and type 2 diabetes (Hu et al., 2001 & 2003). Independent of physical activity in men and women time spent in watching television, at the computer or playing video games also seems to be associated with obesity in children (Janssen and Ross, 2005). Sedentary behaviour, especially TV viewing is positively associated with an increased risk of obesity and Coronary Heart Disease (CHD) risk factors (Jakes et al., 2003). Sedentary life style and physical activity are two distinct classes of behaviour that have different pattern of determinants (Owen et al., 2000), they are likely to have independent effects on total energy expenditure, weight and metabolic variables to date. Number of large population – based studies have assessed simultaneously, the association of TV viewing and physical activity with metabolic syndrome. In addition there is a little information on the extent to which sedentary behaviours and physical activity influence components of the metobolic syndrome other than obesity. Cross sectional study on TV viewing time and physical activity with the presence of metabolic syndrome (as defined by the World Health Organization, 1999).

Physical inactivity is associated with a greater risk of cardiovascular disease (CVD) (Wannamethee et al., 1998) and type 2 diabetes (Helmrich et al., 1991). Recognition of the importance of physical activity has increased in the face of epidemics of sedentary life style and obesity associated metabolic syndrome. The metabolic syndrome provides a unifying aetiological frame work for the development of CVD, hypertension, dyslipidaemia and glucose intolerance. While epidemiological studies shown that physical activity and cardiovascular fitness reduce the syndrome, in many studies, the lack of standared definition of the metabolic syndrome and its components has hampered efforts to interprit relationships with physical activity (Laaksonen et al., 2002; Warehan et al., 1998; Rennie et al., 2003; Gustat et al., 2002; Lakka et al., 2003).

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PHYSICAL ACTIVITY AND BLOOD PRESSURE

Hypertension is commonly seen in persons with type 2 diabetes. In a population-based survey in Singapore conducted by Hughes et al. (1998), the prevalence of hypertension among men was found to be 43% in those with type 2 diabetes, compared to 21% without the condition. Among women, the prevalence of hypertension was 57% versus 24%, respectively. According to the sixth report of the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (The sixth report of the Joint National Committee on prevention, 1997). Diabetes increases cardiovascular disease risk at any stage of hypertension. In the United Kingdom Prospective Diabetes Study (UKPDS) trials, blood pressure reduction resulted in a significant benefit on cardiovascular disease risk reduction over and above that achieved by tight glycemic control (Tight blood pressure control and risk of macrovascular and microvascular complications, 1998). The role of exercise in the management of patients with hypertension is well documented (Stewart, 2002). Although there are no randomised clinical trials examining the efficacy of exercise to reduce blood pressure in persons with type 2 diabetes, it is likely that exercise training would produce benefits in such patients.

Systolic blood pressure increases during aerobic exercise in retation to the intensity of effort, but blood pressure levels drop below resting levels in the 2-4 hours or more following an exercise session of at least 20 min. (Kenney and Seals 1993; Thompson et al., 2001). In individuals without hypertension, systolic blood pressure decreases after a single bout of exercise by 8-10 mmHg and diastolic blood pressure decreases by 3-5 mmHg, but the decrease can be up to 2-fold greater in patients with hypertension. Even relatively low-intensity exercise, corresponding to 40% of maximal oxygen consumption (*VO2max*) acutely lowers blood pressure. Meta-analysis decreases systolic and diastolic blood pressure by 3.8 mmHg and 2.6 mmHg in adults with normal or elevated blood pressure (Whelton et al., 2002). The drecrease in blood pressure was similar in trials in which no weight loss occurred, and also in both lean and obese individuals. The benefit of exercise in individuals with hypertension was slightly more than in those without hypertension. A recent review restricted to Randomized Control Trails RCTs in persons with mildly to moderately elevated blood pressure published since 1998 found reductions in systolic blood pressure of 5.0 mmHg with exercise training, improvements in body composition, insulin sensitivity, endothelial dysfunction and autonomic balance that may mediate the decrease in blood pressure occurring as a result of regular aerobic exercise.

A study demonstrated that a history of NIDDM independently of other known risk factors, including obesity. An earlier study of the same alumni of the University of Pennsylvania showed that higher levels of blood pressure in college were associated with an increased risk of adult-onset diabetes. In addition, the higher rate of NIDDM among men with a parental history of diabetes is consistent with the results of previous studies (Khan et al., 1971; Beaty et al., 1982).

PHYSICAL ACTIVITY AND INFLAMMATION

A single study of strenuous physical exercise elicits an acute inflammatory response characterized by the increased release of proinflammatory cytokines, with leukocytosis and increased plasma concentrations of C-reactive protein (CRP) (Vider et al. 2011; Kasapis and Thompson, 2005). This proinflammatory response to acute exercise is also accompanied by an acute increase in oxidative stress (Sen and Packer, 1996). The acute increase in oxidative stress and inflammation may elicit the responses in cellular protection that decrease inflammation.

Most cross-sectional studies have found lower plasma concentrations of inflammatory markers in physically active or fit individuals than in inactive or unfit persons (Church et al., 2002; Lakka et al., 2003). Data from prospective cohort studies are lacking. In some uncontrolled or non-randomized exercise interventions, regular exercise has decreased CRP concentrations in individuals at a moderate to high risk for CVD (Tisi et al., 1997; Mattusch et al., 2000; Lakka et al., 2005). In another study, exercise training decreased the pro-inflammatory activity of blood mononuclear cells, but CRP concentrations did not decrease (Smith et al., 1999).

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BENEFICIAL EFFECTS OF PHYSICAL ACTIVITY

Physical inactivity and poor physical fitness have been associated with increased mortality among persons with established type 2 diabetes (Wei et al., 2000). A study demonstrated that physical activity was associated with a reduced risk of both cardiovascular and total mortality among men with type 2 diabetes, (Tanasescu et al., 2003). Beneficial physical activities associated with decreased risk of type 2 diabetes have not been clarified completely. In addition, there is controversy that whether physical activity can prevent diabetes independent of BMI and body fatness. Finally, the minimal physical activity level to preclude development of type 2 diabetes based on each individual's susceptibility to become diabetic is a critical issue, which needs further investigation.

CONCLUSION

Diabetes may be prevented by increasing overall activity. Decreased physical activity associated with obesity, pre-diabetes and metabolic syndrome, obesity, diabetes, endothelial dysfunction and inflammation etc. Prolonged TV viewing is significantly associated with an increased prevelance of the metabolic syndrome and several of its components. Despite the preponderance of evidence of the benefits for physical activity and exercise, there is still lack of participation among patients who are at risk or who have type 2 diabetes. The reasons for the under-participation in regular exercise include patient's lack of knowledge about the benefits of exercise, lack of motivation and a lack of clear recommendations from health care professionals. Clinician should observe the need to encourage such patients to exercise regularly as an essential part of management of their condition, specific instructions should be given to patients rather than general advice, which doesn't increase compliance.

More studies should clarify particularly what type, intensity and duration of physical activity can be optimal for decreased risk of obese NIDDM patients or Type 2 Diabetes.

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REFERENCES

1. Albright A, Franz M, Horns by G, et al. American College of Sports Medicine position stand, Exercise and type 2 diabetes. Med. Sci. Sports Exerc. 2000;32(7):1345-1360.
2. American Diabetes Association. Diagnosis and classification of diabetes mellitus, Diabetes care, 2009; 32(suppl 1):s62-s67.
3. Barrett-Connor E. Epidemiology, obesity and non-insulin-dependent diabetes mellitus. Epidemiol Rew. 1989;11:172-81.
4. Beaty TH, Neel JV, Fajans SS. Identifying risk factors for diabetes in first degree relatives of non-insulin dependent diabetic patients. Am. J. Epidemiol., 1982;115:380-97.
5. Blair SN. Evidence for success of exercise in weight loss and control. Ann. Intern. Med. 1993;119(7pt 2):702-706.
6. Bluher M. Adipose tissue dysfunction in obesity. Exp. Clin. Endocrinol. Diabetes, 2009;117(6):241-250.
7. Boulez NG, Haddad E, Kenny GP, Wells GA and Sigal RJ. Effect of exercise onglycemic control and body mass in type 2 diadetes mellitus: a meta-analysis of controlled clinical trials. JAMA, 2001;286:1218-1227.
8. Boulez NG, Kenny GP, Haddad E, Wells GA and Sigal RJ. Meta-analysis of the effect of structured exercise training on cardiorespiratory fitness in type 2 diabetes mellitus. Daibetologia, 2003;46:1071-1081.
9. Brotman DJ, Girod JP. The metabolic syndrome: a tug-of-war with no winner. Cleve. Clin. J. Med., 2002; 69:990-4.

10. Caballero AE, Arora S, Saouaf R, Lim SC, Smakowski P, Park JY, et al. Microvascular and macrovascular reactivity is reduced in subjects at risk for type 2 diabetes. Diabetes, 1999; 48:1856-62.
11. Church TS, Cheng YJ, Earnest CP, Barlow CE, Gibbons LW, Priest EL and Blair SN. Exercise capacity and body composition as predictors of mortality among men with diabetes. Diabetes Care, 2004;27:83-88.
12. Coakley, E.H., Rimm, E.B., Colditz, G., Kawachi, I. and Willet W. Predictors of weight change in men: results from the Health Professionals Follow-up Study. Int. J. Obes. Relat. Metab. Disord., 1998;22:89-96.
13. Cuff DJ, Meneilly GS, Martin A, lanaszewski A, Tildesley HD and Frohlich JJ. Effective exercise modality to reduce insulin resistance in women with type 2 diabetes. Diabetes Care, 2003; 26:2977-2982.
14. Mokdad AH, Ford ES, Bowman BA and Dietz Defronzo RA. Banting Lecture. From the thiumvirate to the ominous octet: a new paradigm for the treatment of type 2 diabetes mellitus. Diabetes, 2009; 58(4):773-795.
15. Devlin JT, Hirshman M, Horton ED and Horton ES. Enhanced peripheral and splanchnic insulin sensitivity in NIDDM men after a single out of exercise. Diabetes, 1987; 36:434-9
16. Devlin JT and Horton ES. Effects of prior high-intensity exercise on glucose metabolism in normal and insulin-resistant men. Diabetes, 1985; 34:973-9.
17. Erlichman, J., Kerbey, A.L., and James, W.P. Physical activity and its impact on health outcomes. Paper 2. Prevention of unhealthy weight gain and obesity physical activity: an analysis of the evidence. Obes. Rev, 2002; 3:273-287.
18. Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III). JAMA, 2001; 285:2486-97.
19. Ferrannini E, Haffiner SM, Mitchell BD and Stern MP. Hyperinsulinaemia: the key feature of a cardiovascular and metabolic syndrome. Diabetologia, 1991; 34:416-22.
20. Geffken DF, Cushman M, Burke GL, Polak JF, Sakkinen PA and Tracy RP. Association between physical activity and markers of inflammation in a healthy elderly population. Am. J. Epidemiol., 2001; 153:242-50.
21. Gill JM, Cooper AR. Physical activity and prevention of type 2 diabetes mellitus. Sports Med., 2008; 389100:807-24.
22. Gillies CL., Abrams KR, Lambert PC, et al. Pharmacological and life-style interventions to prevent or delay type 2 diabetes in people with impaired glucose tolerance: systematic review and meta-analysis, BMJ, 2007; 334(7588):299.
23. Gustat J, Srinivasan SR, Elkasabany A and Berenson GS. Relation of self-rated measures of physical activity to multiple risk factors of insulin resistance syndrome in young adults: the Bogalusa Heart Study. J. Clin. Epidemiol., 2002; 55:997–1006.
24. Helmrich SP, Ragland DR, Leung RW and Paffenbarger RS. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. New Engl. J. Med., 1991; 325:147-152.
25. Hosokawa S, Hiasa Y, Takahashi T and Itoh S. Effect of regular exercise on coronary endothelial function in patients with recent myocardial infarction. Circ. J., 2003; 67:221-4.
26. Hu FB, Leitzmann MF, Stampfer MJ, Colditz GA, Willett WC and Rimm EB. Physical activity and television watching in relation to risk for type 2 diabetes mellitus in men. Arch. Intern. Med., 2001; 161:1542–1548.
27. Hu FB, Li TY, Colditz GA, Willett WC and Manson. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. J. Am. Med. Assoc., 2003; 289:1785–1791.
28. Hu FB, Li TY, Colditz GA, Willett WC. and Manson JE. Television watching and other sedentary behaviors in relation to risk of obesity and type2 diabetes mellitus in women, JAMA, 2003a; 289: 1785-179.
29. Hughes K, Choo M, Kuperan P, Ong CN and Aw TC. Cardiovascular risk factors in non-insulin-dependent diabetics compared to non-diabetic controls: a population-based survey among Asians in Singapore. Atherosclerosis, 1998; 136:25-31.
30. Jakes RW, Day NE, Khaw KT et al. Television viewing and low participation in vigorous recreation are independently associated with obesity and markers of cardiovascular disease risk: EPIC-Norfolk population-based study. Eur. J. Clin. Nutr., 2003; 57:1089–1096.
31. Jeffery RW, McGuire MT and French SA. Prevalence and correlates of large weight loss: Int. J. Obes. Relat. Metab. Disord., 2002; 26:969-972.

32. Jeon CY, Lokken RP, Hu FB, van Dam RM. Physical activity of moderate intensity and risk of type 2 diabetes: a systematic review. Diabetes Care, 2007; 30(3): 744-52.
33. Kahn HA, Herman JB, Medalie JH, Neufeld HN, Riss E and Goldbout U. Factors related to diabetes incidence: a multivariate analysis of two years observation on 10,000 men: the Israel Ischemic Heart Disease Study. J. Chronic. Dis. 1971; 23:617-29.
34. Karan JH. Obesity and diabetes in humans. In: Brodoff BN, Bleicher SJ, eds. Diabetes mellitus and obesity. Baltimore: Williams & Wilkins, 1982; 294-300.
35. Kasapis C. and Thompson PD. The effects of physical activity on serum C-reactive protein and inflammatory markers: a systematic review. J. Am. Coll. Cardiol., 2005; 45:1563-1569.
36. Kenney MJ, and Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertention, 1993; 22:653-664.
37. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 Diabetes with lifestyle intervention or metformin. N. Eng. J. Med., 2002; 346:393-403.
38. Kopp HP, Kopp CW, Festa A, Krzyzanowska K, Kriwanek S, Minar E, et al. Impact of Weight Loss on Inflammatory Proteins and Their Association With the Insulin Resistance Syndrome in Morbidly Obese Patients. Arterioscler. Thromb. Vasc. Biol., 2003.
39. Kronenberg F, Pereira MA, Schmitz MK et al. Influence of leisure time physical activity and television watching on atherosclerosis risk factors in the NHLBI Family Heart Study. Atherosclerosis, 2000; 153:433–443.
40. Laaksonen DE, Lakka HM, Salonen JT, Niskanen LK, Rauramaa R and Lakka TA. Low levels of leisure-time physical activity and cardiorespiratory fitness predict development of the metabolic syndrome. Diabetes Care, 2002; 25:1612–1618.
41. Lakka TA, Laaksonen DE, Lakka HM et al. Sedentary lifestyle, poor cardio respiratory fitness, and the metabolic syndrome. Med. Sci. Sports Exerc., 2003; 35:1279–1286.
42. Lavrencic A, Salobir BG, Keber I. Physical training improves flow-mediated dilation in patients with the polymetabolic syndrome. Arterioscler. Thromb. Vasc. Biol., 2000; 20:551-5.
43. Lyon CJ, Law RE, Hsueh WA. Minireview: adiposity, inflammation, and atherogenesis. Endocrinology, 2003; 144:2195-200.
44. Maiorana A, O'Driscoll G, Cheetham C, Dembo L, Stanton K, Goodman C, et al. The effect of combined aerobic and resistance exercise training on vascular function in type 2 diabetes. J. Am. Coll. Cardiol., 2001; 38:860-6.
45. Marble A, Krall LP, Bradley RF, Christlieb AR, Soeldner JS, eds. Joslin's diabetes mellitus. 12th ed, Philadelphia: Lea & Febiger, 1985.
46. Mattusch F, Dufaux B, Heine O, Mertens I, Rost R. Reduction of the plasma concentration of C-reactive protein following nine months of endurance training. Int. J. Sports Med., 2000; 21:21-4.
47. National Health Survey. Ministry of Health, Singapore. 1998.
48. National Institutes of Health, Consensus Development Conference on Diet and Exercise in Non-Insulin-Dependent Diabetes Mellitus. Diabetes Care, 1987; 10:639-44.
49. Owen N, Leslie E, Salmon J and Fotheringham MJ. Environmental determinants of physical activity and sedentary behavior. Exerc. Sport Sci. Rev., 2000; 28:153–158.
50. Paffenbarger RS Jr. and Wing AL. Chronic disease in former college students, XII. Early precursors of adult-onset diabetes mellitus. Am. J. Epidemiol., 1973; 97:314-23.
51. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX. Effects of diet and exercise in preventing NIDDM in people with impaired glucose. The Da Qing IGT and Diabetes Study. Diabetes Care, 1997; 20:537-44.
52. Poirier P and Despres JP. Exercise in weight management of obesity. Cardiol. Clin., 2001; 19(3):459-470.
53. Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. JAMA, 2001; 286:327-34.

54. Rennie KL, McCarthy N, Yazdgerdi S, Marmot M and Brunner E. Association of the metabolic syndrome with both vigorous and moderate physical activity. Int. J. Epidemiol., 2003; 32:600–606.
55. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care, 2003; 26 Suppl 1:S5-20. 14.
56. de Vegt F, Dekker JM, Jager A, Hienkens E, Kostense PJ, Stehouwer CD, et al. Relation of impaired fasting and postload glucose with incident type 2 diabetes in a Dutch population: The Hoorn Study. JAMA, 2001; 285:2109-13.
57. Ross, R. Does exercise without weight loss improve insulin sensitivity? Diabetes Care, 2003; 26:944-945.
58. Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L, et al. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1 st Stock Conference and consensus statement. Obes. Rev., 2003; 4:101-114.
59. Saydah SH, Loria CM, Eberhardt MS and Brancati FL. Subelinical states of glucose intolerance and risk of death in the U.S. Diabetes Care 2001; 24:447-53.
60. Schneider SH, Amorosa LF, Khachadurian AK, Ruderman NB. Studies on the mechanism of improved glucose control during regular exercise in type 2 (non-insulin-dependent) diabetes. Diabetologia, 1984; 26:355-60.
61. Sen and Packer L. Antioxidant and redox regulation of gene transcription. FASEB J., 1996; 10:709-720.
62. Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C and White RD. Physical activity/exercise and type 2 diabetes: a consensus statement from the American Diabetes association. Diabetes care. 2006; 29(6):1433-1438.
63. Smith JK, Dykes R, Douglas JE, Krishnaswamy G and Berk S. Long-term exercise and atherogenic activity of blood mononuclear cells in persons at risk of developing ischemic heart disease. JAMA, 1999; 281:1722-1727.
64. Stewart KJ. Exercise training and the cardiovascular consequences of type 2 diabetes and hypertension: plausible mechanisms for improving cardiovascular health. JAMA, 2002; 288:1622-31.
65. Stiegler P and Cunliffe A. The role of diet and exercise for the maintenance of fat-free mass and resting metabolic rate during weight loss. Sports Med., 2006; 36(3):239-262.
66. Tamakoshi K, Yatsuya H, Kondo T, Hori Y, Ishikawa M, Zhang H, et al. The metabolic syndrome is associated with elevated circulating C-reactive protein in healthy reference range, a systemic low-grade inflammatory state. Int. J. Obes. Relat. Metab. Disord., 2003; 27:443-449.
67. Tan CE, Emmanuel SC, Tan BY and Jacob E. Prevalence of diabetes and ethnic differences in cardiovascular risk factors. The 1992 Singapore National Health Survey. Diabetes Care, 1999; 22:241-247.
68. Tanasescu M, Leitzmann MF, Rimm EB and Hu FB. Physical activity in relation to cardiovascular disease and total mortality among men with Type 2 diabetes. Circulation, 2003; 107:2435-2439.
69. The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch. Intern. Med., 1997; 157:2413-46.
70. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Moyna N. and Pescatello L. The acute versus the chronic response to exercise. Med. Sci. Sports exerc., 2001; 33 Suppl 6: S438-S445 discussions S452-453.
71. Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes: UKPDS 38. UK Prospective Diabetes Study Group. Br. Med. J., 1998; 317:703-13.
72. Tisi PV, Hulse M, Chulakadabba A, Gosling P. and Shearman CP. Exercise traing for intermittent claudication: does it adversely affect biochemical markers of the exercise-induced inflammatory response? Eur. J. Vasc. Endovac. Surg., 1997; 14:344-350.
73. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P et al. Finnish Diabetes Prevention Study Group: Prevention of type 2 daibetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N. Engl. J. Med., 2001; 344:1343-50.
74. Vider J, Laalsonen DE, Kilk A, Atalay M, Lehtmaa J, Zilmer M, et al. Physical exercise induces activation of NF kappaB in human peripheral blood lympocytes. Antioxid. Redox Signal., 2001; 3:1131-1137.

75. Wannamethee SG, Shaper AG, Walker M, Ebrahim S, Life style and 15-year survival free of heart attack, stroke, and diabetes in middle-aged British men. Arch. Intern. Med., 1998; 158:2433–2440.
76. Wareham NJ, Hennings SJ, Byrne CD, Hales CN, Prentice AM and Day NE. A quantitative analysis of the relationship between habitual energy expenditure, fitness and the metabolic cardiovascular syndrome. Br. J. Nutr., 1998; 80:235–241.
77. Wei M, Gibbons LW, Kampert JB, Nichaman MZ and Blair SN. Low cardiorespiratory fitness and physical inactivity as predictors of mortality in men with type 2 diabetes. Ann. Intern. Med., 2000; 132:605-11.
78. West KM. Epidemiology of diabetes and its vascular complications. New York: Elsever, 1978.
79. Whelton SP, Chin A, Xin X, He J: Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. Ann. Intern. Med., 2002; 136:493-503.
80. Williams SB, Cusco JA, Roddy MA, Johnstone MT and Creager MA. Impaired nitric oxide-mediated vasodilation in patients with non-insulin dependent diabetes mellitus. J. Am. Coll. Cardiol., 1996; 27:567-74.
81. Wilson PW, McGee DL, Kannel WB. Obesity, very low density lipoproteins and glucose intolerance over fourteen years: the Framingham Study. Am. J. Epidemiol., 1981; 114:697-704.
82. World Health Organization, Definition. Diagnosis and classification of diabetes mellitus and its complications. World Health Organization, 1999, Geneva.
83. Yudkin JS, Stehouwer CD, Emeis JJ and Coppack SW. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? Arterioscler. Thromb. Vasc. Biol., 1999; 19:972-8.
84. Zimmet PZ, McCarty DJ and de Courten MP. The global epidemiology of non-insulin-dependent diabetes mellitus and the metabolic syndrome. J. Diabetes Complications, 1997; 11:60-8.

V. Chinnari Harika[1], B. Babitha[2] and Ch. Manmohan[3]

[1,2] Department of Foods & Nutritional Sciences, Acharya Nagarjuna University, Nagarjunanagar-522510, Guntur, Andhra Pradesh, India.

[3]Govt. General Hospital, Guntur-522002, Andhra Pradesh, India.

E-mail: vcharika@yahoo.com