CHAPTER I

INTRODUCTION

Researches indicate that regular physical activity reduces risk of coronary heart disease (CHD), diabetes, colon cancer, hypertension, and obesity. (Fletcher GF et al,1996) Physical activity is identified as any form of body movement that has a significant metabolic demand. Thus physical activities include training for and participation in athletic competitions, the performance of strenuous occupations, doing household chores, and non-sporting leisure activities that involve physical effort (Kent M,1998). It also promotes psychological well-being; builds and maintains healthy bones , muscles, and joints. Physical activity is also an important part of the self-care regimen of many chronic conditions.

However, physical inactivity is a significant risk factor for cardiovascular disease. It ranks similarly to cigarette smoking, high blood pressure, and elevated cholesterol. It is estimated that approximately 35 % of coronary heart disease mortality were due to physical inactivity.

Physical inactivity is a term used to identify people who do not perform the recommended level of regular physical activity. Moreover, the people who have no reported activity or any physical activity done for less than 20 minutes or less than three times per week, It was called the sedentary lifestyle.

Up to date, the American Heart Association recommends 30-60 minutes of aerobic exercise three to four times per week to promote cardiovascular fitness. . (Fletcher GF et al,1996) In 1996 the report of the Surgeon General on Physical Activity and Health recommend the minimum level of physical activity required to achieve health benefits was a daily expenditure of 150 kilo-calories in moderate or vigorous activities. This recommend is consistent with guidelines established by the centers for disease control and prevention, and American College of Sports Medicine. It also is consistent with the 1996 consensus statement from the National Institutes of Health, recommending adults to accumulate at least 30 minutes of moderate activity most days of the week. Moderate activities include pleasure walking ,climbing stairs, gardening, yard work,

moderate- to-heavy housework, dancing and home exercise. These are well known and used to instruct the patients, the family and the public widely.(Fletcher GF et al, 1996)

Although physical exercise was known to have many beneficial effects, there was also many evidences that free radical production increases during exercise (Witt et al, 1992) and that oxidative damage occurred in the muscles, liver, blood and perhaps other tissues.

There are at least two mechanisms by which reactive oxygen species (ROS) may be produced during exercise. The first is via an electron leak, probably at the quinone-cytochrome level, of the mitochondrial electron transport chain, which produces superoxide radical. The second possible mechanism is that of ischemic reperfusion. At the cessation of exercise, these regions then undergo reoxygention may lead to the well-known burst of ROS. ROS can damage practically every component of the cell including proteins, nucleic acids and lipids. It becomes apparent that for many human disease, formation of reactive oxygen species is sedentary to the primary disease process. The roles of reactive oxygen species in pathology are proposed as follows : 1. Haemochromatosis : the iron- induced lipid peroxidation causes an increase in collagen gene transcription, and then the lipid peroxidation may play an important role in the development of cirrhosis

2. Carcinogenesis : DNA strands can be altered or broken by reactive oxygen species.

3. Ischaemic reperfusion damage : There is considerable evidence that injury to ischemic tissue occurs almost exclusively during the reperfusion phase and that the damage is caused by the large flux of superoxide radicals which are generated when oxygen is reintroduced to the ischaemic tissue. Such damage is clearly relevant in patients presenting with acute cerebrovascular accidents or myocardial infarction .

4. Inflammation and immune injuries : All kinds of tissue damage ,mechanical, physical or chemical trauma, are followed by an inflammatory response. Reactive oxygen species may perpetuate tissue damage.

5. Neurological disorder. : It suggests that in several neurological disease , for example Parkinson' s disease, there is iron accumulation secondary to the initial toxic lesion.

6. Atherosclerosis : The pathogenesis of atherosclerosis is unclear, but it is hypothesized that it is initiated by damage to the vascular endothelium. Endothelial cells are known to be sensitive to damage by reactive oxygen species and lipid hydroperoxides. (Marshall WJ and Bangert SK,1995)

In addition, the intensity and duration of exercise also influence the appearance of lipid peroxidation products , a decrease in plasma lipid peroxidation occurred with exercise at 40% and 70 % of VO_{2max} , whereas an increase was observed at 100% VO_{2max} (Lowin R. ,1987).

Wetzstein CJ., et al ,1998 studied the acute effect of exercise on the susceptibility of low density lipoprotein to oxidation. The results showed that acute exercise for 30 minutes, intensity 55% and 70 % for exerciser and sedentary groups increased thirty percent of plasma myeloperoxidase (MPO), the heme enzyme which used to detect lipid peroxidation (Carr AC et al,2000), when comparing between pre and post exercise.

Therefore, the information about the effects of exercise on lipid peroxidation are still controversial and somewhat limited. Different methods to detect lipid peroxidation used in previous studies have led to inconsistent reports and problems in confirming the relationship between lipid and exercise. There is no one biomarker that is best at assessing lipid peroxidation, especially during exercise. For example, conjugated dienes appear at the onset of lipid peroxidation and seem to be linked to several steps of lipid peroxide degradation. (Corongiu F et al,1983) In fact, only 30-55% of lipid peroxidation is actually detected by diene measurement. (Corongiu F et al,1983 ,Frankel E.N.1987,and Greenen DL,1988) Another biomarker of lipid peroxidation, lipid hydroperoxide, is formed when hydrogenperoxides are reduced by the peroxidase activity of hemoglobin. Because hemoglobin has broad substrate specificity, including nonperoxides, the sensitivity of this method is low in unpure system.(Maridonneau I,et al,1983)Nevertheless, serum lipid hydroperoxides were found to increase 96 % above rest immediately following exhaustive exercise .(Cutler RG.and Alessio HM, 1990)This measure of lipid peroxidation increased in a similar manner to TBARS (thiobarbituric acid – reactive substrances) as a result of exhaustive exercise. However, when exercising at intensities below maximum effort, an increase in lipid hydroperoxides was

not always evident. For example Ji L., et al,1988 ,Viinikka L.,et al. ,1984 and Salminen A. and Vihko V.,1983 did not find significant increases in lipid hydroperoxides as a results of moderate intensity exercise. However, conjugated diene measurement had been chosen for this study because it is fast and simple to perform and can be applied to clinical purposes. (Ahotupa M et al., 1996)

By the way, the recommendation of exercise for preventing coronary artery disease must concern and precaution about the harmful from exercise induced oxidative stress. Sometimes in untrained people with sedentary lifestyle who don't know about their health such as their vital organ impairment or their asympomatic heart diseases . The acute exercise may induce oxidative stress enough to lead to angina pectoralis or myocardial infarction. Hence, this study was thus performed to investigate the effect of acute and moderate exercise on low density lipoprotein diene conjugation in sedentary Thais. The objective of this study was investigated the number of oxidative stress which was detected from the level of low density lipoprotein diene conjugation caused by acute moderate exercise. Furthermore, this data can be fundamental information to assessment the rising risk from oxidative stress for middle age people.