Full Length Research Paper

# The effects of exercise program based on OMNI scale on metabolic syndrome criteria and C-reactive protein in males

# Serkan İBİŞ

Department of Physical Education and Sports, Niğde University, Niğde, Turkey. E-mail: serkanibis@nigde.edu.tr

### Accepted 29 February, 2012

With its multiple components, metabolic syndrome is a serious problem that adversely affects the quality of life. Exercise programs have proven to be quite effective alternatives to drug treatments with regard to preventive medicine. We investigated the effects of personal exercises that do not require a professional using physiological and biochemical parameters and OMNI scale. Adult males performed aerobic exercise with an intensity ranging between easy and somewhat hard on OMNI scale for 12 weeks. Physiological parameters, including weight, height, waist/hip ratio, body mass index (BMI), visceral fat rating (VFR)%, resting heart rate (RHR), blood pressure and biochemical parameters including serum fasting blood glucose (FG), triglyceride (TG), total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and C-reactive protein (C-RP) levels before and after exercise were recorded. There were significant differences in all physiological parameters (P<0.01) except diastolic pressure. Biochemical parameters showed similar significant changes (P<0.01). Exercise programs serve not only as alternatives to treatment with medications but also as part of preventive medicine. The results of this study suggest that the most significant handicaps of such programs, namely the need for a professional instructor or trainer and the perceived exertion issue, can be overcome by using the OMNI scale. Performing the most comfortable personal exercise program for him/herself will enable the person to get rid of the MetS components and improve the quality of life.

Key words: OMNI scale, exercise, metabolic syndrome, C- reactive protein (C-RP).

# INTRODUCTION

Induced by insulin resistance, metabolic syndrome (MetS) is a fatal endocrinopathy that can be accompanied by systemic disorders such as abdominal obesity, glucose intolerance, increase in serum lipid levels, hypertension and coronary artery disease (Alberti et al., 2006). World Health Organization criteria for the diagnosis of MetS require one of impaired fasting blood glucose, diabetes or impaired glucose tolerance and two of the following conditions: hypertension, hyperlipidemia, central obesity and micro albuminemia (Akram et al., 2002). About a third of the adult population in the world has MetS (Ford et al., 2002). Age and sedentary lifestyle are associated with the increase in the number of people

with MetS and mortality rate among them. This, in turn, made MetS a bigger community health problem. Cardiovascular mortality among patients with and without MetS is 12 and 2.2%, respectively (Grundy et al., 2004).

Besides changes in lifestyle, there is not a single medication that can treat MetS alone. The most appropriate approach, alternative to medical treatment, would be a total change in lifestyle with the help of healthy nutrition, cessation of smoking, loss of weight and regular exercise (Pyorala et al., 2000).

It has been shown that chronic adaptation to exercise, especially regularly performed, long term, medium intensity aerobic exercise, lowered risk factors of

Parameter	Females	Males
Abdominal obesity (Waist length)	>88 cm	>102 cm
Triglyceride	≥150 mg/dL	≥150 mg/dl
HDL	<50 mg/dL	<40 mg/dl
Fasting blood glucose	≥100 mg/dL	≥100 mg/dl
Blood pressure	≥130/85 mmHg	≥130/85 mmHg

 Table 1. Criteria of national cholesterol education ATP III. American Heart Association

 Treatment Panel III, 2002).

coronary artery disease such as total cholesterol, LDL-C, and triglyceride while increasing high-density lipoproteins. In the meantime, researchers also emphasized that exercise reduced high blood pressure and obesity related diseases (Linda et al., 2000). Regular physical activity reduces the risks of heart attack, ischemic stroke, early cardiovascular disorders and mortality. It is also known that exercise decreases the mortality risk due to coronary heart disease (Timo et al., 2005; Gunter et al., 1996). Regular, well-planned exercise of no less than 45 to 60 min in duration is required for the prevention of weight gain.

In related studies the exercises' frequency and intensity was determined in terms of the percentage of the heart rate. However, tolerance to the intensity of the exercise varies between individuals. Exercise programs prepared based on resting heart rate or the Karvonen method (Timo et al., 2005; Arnt et al., 2008) could be perceived as high intensity by some individuals while others perceive it as low-intensity. Individual differences, especially genotype, motivation and ability to exercise may affect the tolerance to intensity (Janine et al., 2006).

The aim of the present study was to examine the effects on metabolic syndrome components and CRP level of a 12 week walking/running exercise, which started with 40 to 60% of resting heart rate and the intensity is adjusted between easy and somewhat hard on OMNI scale.

#### MATERIALS AND METHODS

#### Study population and design

This study was carried out on 24 males with MetS who fulfilled at least 3 of the five criteria published in the National Cholesterol Education ATP III (Table 1), (American Heart Association Treatment Panel III, 2002). The control and exercise groups were randomized. Four subjects of the control group were excluded from study due to various reasons, rendering the control group comprising 20 male subjects. Mean ages of the exercise and control groups were 42.08±3.08 and 41.37±2.66 years, respectively. Mean height of the exercise group was 172.83±5.42 cm while that of the control group was 172.38±8.08 cm. The volunteers participating in the study were informed about this study and they signed informed consent form. Every stage of the study was carried out according to Helsinki Degleration. Ethics approval was obtained from the Nigde University Ethics Board. In order to keeps the exercise program to be the only factor affecting the volunteers, diet and daily routines were not altered throughout the study. None of

the subjects used any medication or consumed cigarette throughout the study. Health of the subjects was supervised by physicians during the study.

#### Measurements

Weight, height and waist measurements were taken by calibrated tools in the Physiology Laboratory of Nigde University School of Physical Education and Sports. Weight was measured while the subjects were bare foot, wearing t-shirt and shorts using a scale (holtain limited) sensitive to 0.01 kg. Height was measured using a metal meter sensitive to 0.01 cm while the subjects were bare-foot, standing straight, with the heels and the head in normal anatomical position. Waist and hip measurements were taken using inelastic Gulick measuring tape. While the subjects were standing, with upper limbs hanging freely, waist measurements were taken after a normal exhalation on a horizontal plane passing through the superior-most point of the iliac bone using inelastic Gulick measuring tape. Hip region was measured at the level of maximal extension of the buttocks. All measurements were made in the morning, while the subjects were hungry (Grundy et al., 2005).

Body mass index of the subjects was calculated by the formula [(weight (kg)/height<sup>2</sup> (m<sup>2</sup>)]. Visceral fat rating (VFR) was determined using bioelectric impedance analyzer (BIA, Tanita Body-Fat Analyzer model TBF 300). Resting heart rate (RHR) was measured in the morning from the carotid artery while the subjects lay in the supine position. Following a 5 min resting, systolic and diastolic arterial blood pressures were measured in mmHg by the same researcher over the left arm, using a calibrated aneroid sphygmomanometer and stethoscope, while the subjects were in sitting position.

#### Laboratory procedures

Blood samples were taken 24 h before the onset of the exercise program and 48 h after the end of the 12 week exercise program. After a 12 h fasting, 5 cc of blood was drawn from the antecubital vein between 8 to 10 am and collected in tubes. Samples were centrifuged at 3000 rpm for 15 min. The supernatant was transferred to an eppendorf tube and store at -70°C. F asting blood glucose (FG), total cholesterol (TC), triglyceride, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) levels of the exercise and control groups were measured by Roche Sismex 2000 XLl kit and analyzer. C- reactive protein (C-RP) was measured using Olympus AU-600 Autoanalyzer and relevant test kits. Blood samples were taken by a registered nurse under the supervision of an Internist in Nigde State Hospital.

#### **Exercise protocol**

The exercise group, comprised of 12 males with MetS, performed

Table 2. Physical characteristics of the subject
--

Variable	Exercise group	Control group	Z	Р
Age (years)	42.08±3.08	41.37±2.66	-1.290	0.197
Height (cm)	172.83±5.42	172.38±8.08	-0.348	0.728
Weight (kg)	96.41±13.35	94.93±10.54	-0.270	0.787

There were no statistically significant differences between the subjects in terms of physical characteristics (P>0.05).

walking/ running exercise with loading intensity of 60%, three times performed the exercise for 60 min and in subsequent weeks, as endurance and adaptation increased, the duration gradually increased to 120 min between 7 to 12 weeks. Subjects started the exercise program with Training Heart Rate Range (THRR), calculated as THRR=220-age x 60, set between 106 to 110 bpm. The intensity of exercise was increased by increasing the THRR 5% every two weeks, taking care not to exceed 70% of the maximal heart rate. As the exercise program progressed, heart rate increased to a level of 140 to 150 bpm (American College of Sports Medicine Position Stand, 2004). Increases in loading intensity were determined according to OMNI scale two weeks after the start of the exercise program. The level of exertion neither fell below easy nor exceeded somewhat hard (Utter et al., 2004).

#### Applied exercise

In the exercise group, subjects performed 5 min of walking, 5 min of warming up and 5 min of stretching before each training. Main training phase comprised of walking/running, followed by 5 to 10 minutes of stretching. To determine the intensity of training, heart rate was continuously monitored by telemetry. In the first two weeks, subjects walked at a normal pace three times a week for 3 to 4 km. In weeks 3 and 4, pace was increased and subjects walked for 5 to 6 km. In weeks 5 and 6, duration of walking and, therefore, distance was increased. Subjects walked for 1.5 to 2 h and covered a 7 to 8 km distance. Between weeks 7 and 12, in addition to walking, subjects participated in group jogging. At the end of 12 weeks, measurements were repeated and compared to the first measurements.

#### Statistical analysis

Statistical analyses were carried out using SPSS (version 16.0) statistical package. Means and standard deviations of the parameters were calculated. Wilcoxon and Mann-Whitney U tests were used to examine the relationships between parameters in dependent and independent groups, respectively. A p-value less than 0.05 or 0.01 was considered statistically significant.

#### Findings

When the pre- and post-exercise measurements were compared in the exercise group, significant differences were found in weight, waist, BMI, VFR, RHR, systolic pressure, FG, TC, triglyceride, HDL-C, LDL-C, and C-RP (P<0.05) while that in diastolic pressure was not statistically significant (P>0.05).

When the pre- and post-exercise parameters were compared between the exercise and control groups, significant differences in RHR, FG, triglyceride, HDL-C, C-RP (P<0.05) was found. On the other hand, weight, waist, BMI, VFR, systolic and diastolic

pressures, TC, and LDL-C were not statistically different (P>0.05).

## DISCUSSION

MetS represents a group of metabolic disorders (Alberti and Zimmet, 1998). It is an important cause of morbidity that affects more and more people around the world (Meigs, 2002). Besides environmental factors such as sedentary lifestyle and changes in nutritional habits, some genetic factors play roles in MetS becoming more prevalent (Janssen et al., 2002; Speakman, 2004).

In the present study, we determined the intensity of exercise based on the hypotheses that tolerance to exercise showed personal variations and that variations in perception of intensity, genetics, motivation and training ability (Jannie et al., 2006) affected the tolerance to exercise. At the beginning of the exercise program, the intensity was adjusted with respect to 60% of the resting heart rate and during the course of the program adjustments were made between easy and somewhat hard on OMNI scale. Therefore, while the duration and intensity of exercise was determined according to RHR at the beginning of the program, with adaptation to exercise, it was determined based on OMNI Scale taking into account the perceived exertion. Following exercise, subjects in the exercise group experienced 5.67, 8.68, 6.25, 4.55 and 15.89% reductions in body weight, waist measurement, waist/hip ratio, BMI and VFR, respectively. These decreases in males of the exercise group were significant (P<0.002, Table 2). Comparison of the postexercise parameters between exercise and control groups yielded a 6.67% difference in waist-hip ratio (P<0.05, Table 3) while differences in other parameters were not statistically significant (P > 0.05; Table 4). Significant reductions in body weight, waist-hip ratio and BMI following exercise programs have been reported in numerous studies (Leslie et al., 1995; Sang et al., 2003; Barbara et al., 2009; David et al., 2002). Amano et al. (2001) in their study gave a 12 week aerobic exercise program to obese males and females whose mean age was 41.6 years. The authors reported significant reduction in BMI when the subjects exercised for 30 min, three times a week (Amano et al., 2001). Maison et al. (2001) argued that visceral obesity played a key role in the development of MetS (Patrick et al., 2001). The findings of our study are consistent with the literature.

Variable	Exercise groups	Mean ± SD	Min.	Max.	Z	Р	
$M_{a}$ whet $(l_{a}a)$	Pre-exercise	96.41±13.35	79	126	2.050	0.000**	
vveight (kg)	Post-exercise	90.94±12.49	77.60	121.50	-3.059	0.002**	
Waist (cm)	Pre-exercise	106.58±8.25	97	123	-3 074	0 002**	
	Post-exercise	97.33±6.97	87	113	0.01		
		0.06+0.021	0.00	101			
Waist-hip ratio	Pre-exercise	$0.96\pm0.031$	0.90	101	-3.064	0.002**	
	F USI-EXEICISE	0.90±0.039	0.03	0.90			
2	Pre-exercise	32.28±4.69	26.15	41.72		0.004*	
BMI (kg/m²)	Post-exercise	30.81±4.33	25.69	40.23	-2.845		
	Pre-exercise	12.08±3.31	8	17	2 1 1 1	0.02*	
VFK (%)	Post-exercise	10.16±3.01	7	16	-3.114	0.02	
RHR heat/min	Pre-exercise	72.91±1.97	69	76	-2.822	0.005*	
	Post-exercise	69.91±1.67	67	73	-		
		120.0 52	110	120			
Systolic (mmHg)	Pie-exercise Doct oversion	120±9.00 112+0 07	100	130	-1.994	0.46*	
	F USI-EXEICISE	115±0.07	100	150			
Diastolic (mmHg)	Pre-exercise	78.33±3.89	70	80		0.180	
	Post-exercise	75.83±5.14	70	80	-1.342		
	Pre-exercise	111.33±7.19	106	127	-3.050	0.002**	
r G (mg/di)	Post-exercise	91.25±5.70	80	98	-3.039	0.002	
TC(mg/dl)	Pre-exercise	225.92±49.12	159	295	-2.589	0.010*	
	Post-exercise	207±42.70	144	284			
	Pre-exercise	166 58+24 06	116	197			
Triglyceride (mg/dl)	Post-exercise	140.50±27.79	114	195	-2.432	0.015*	
HDL-C (mg/dl)	Pre-exercise	39.91±2.23	35	42	0.004	0.003**	
	Post-exercise	45.83±4.32	40	54	-2.984		
LDL-C (mg/dl)	Pre-exercise	128.92±33.74	79	181	-3 062	0 002**	
	Post-exercise	113±29.06	58	147	0.002	0.002	
		4.04.0.04	4 50	0.50			
C-RP (mg/dl)	Pre-exercise	1.91±0.34	1.50	2.50	-3.065	0.002**	
,	FUSI-exercise	1.01±0.19	0.70	1.30			

 Table 3. Comparisons of MetS components and C-RP measurements before and after exercise.

\*P<0.05, \*\*P<0.01.

Ajlan and Mehdi (2005) reported an inverse relation between BMI and exercise (Al-Ajlan et al., 2005).

Reductions in weight, waist size, waist/hip ratio and BMI following exercise program are indicators of benefits of exercise on these parameters. The risk of CVD increases when BMI is over 30 kg/m<sup>2</sup> and decreases when it is under 20 kg/m<sup>2</sup> (Zhengming et al., 2006).

Reductions in these parameters can be explained by increased utilization of fat for the basal metabolism during the process of moderate intensity exercise or increased utilization of fat as a source of energy during aerobic exercise (Jacops et al., 2000).

In the present study, a 4.11% decrease in RHR and 5.8% decrease in systolic pressure was found after the

Table 4. Comparison of the exercise and control groups with respect to pre-exercise and post-exercise MetS components and CRP measurements.

Variable	Groups	Pre-exercise (Mean±SD)	Z	Р	Post-exercise (Mean±SD)	Z	Р
Weight (kg)	Exercise group Control group	96.41±13.35 94.93±10.54	-270	0.787	90.94±12.49 95.50±10.78	-1.389	0.181
Waist (cm)	Exercise group Control group	106.58±8.25 94.93±10.54	-928	0.384	97.33±6.97 110.38±13.04	-1.819	0.069
Waist-hip ratio	Exercise group Control group	0.96±0.031 0.95±0.062	-349	0.727	0.90±0.039 0.96±0.052	-2.404	0.016*
BMI (kg/m <sup>2</sup> )	Exercise group Control group	32.28±4.69 31.86±4.24	-0.077	0.939	30.81±4.33 32.23±3.99	-0.772	0.440
VFR%	Exercise group Control group	12.08±3.31 11.85±3.44	-0.117	0.910	10.16±3.01 12.37±3.70	-1.755	0.82
RHR beat/min	Exercise group Control group	72.91±1.97 73.37±1.50	-0.395	0.693	69.91±1.67 74.25±2.12	-3.269	0.001*
Systolic (mmHg)	Exercise group Control group	120±9.53 116.25±11.87	-797	0.473	113±8.87 118.12±11.31	-0.788	0.473
Diastolic (mmHg)	Exercise group Control group	78.33±3.89 76.25±5.17	-1.027	0.473	75.83±5.14 77.50±5.34	-0.182	0.910
FG (mg/dl)	Exercise group Control group	111.33±7.19 107.88±5.13	-1.279	0.208	91.25±5.70 108.38±5.37	-3.709	0.000*
TC (mg/dl)	Exercise group Control group	225.92±49.12 225.13±44.49	-0.116	0.910	207±42.70 225.13±44.84	-1.004	0.343
Triglyceride (mg/dl)	Exercise group Control group	166.58±24.06 166.62±20.99	-0.270	0.792	140.50±27.79 167.50±20.24	-2.085	0.039*
HDL-C (mg/dl)	Exercise group Control group	39.91±2.23 37.87±5.19	-1.949	0.57	45.83±4.32 37.25±4.13	-3.095	0.001*
LDL-C (mg/dl)	Exercise group Control group	128.92±33.74 140.88±38.12	-0.733	0.473	113±29.06 142.12±37.86	-1.466	0.157
C-RP (mg/dl)	Exercise group Control group	1.91±0.34 1.85±0.34	-0.505	0.624	1.01±0.19 1.88±0.30	-3.726	0.000*

\*P<0.05, \*\*P<0.01. (RHR beat/min 0.001\*\*, FG (mg/dl) 0.000\*\*, HDL-C (mg/dl) 0.000\*\* and C-RP (mg/dl) 0.000\*\*).

exercise program, both of which were statistically significant (P<0.05, Table 3). Diastolic pressure before and after exercise did not change significantly (P>0.05, Table 3). Comparisons of the pre- and post-exercise RHR between exercise and control groups revealed a

6.21% difference in RHR (P<0.05, Table 4) while differences in systolic and diastolic pressures were not statistically significant (P>0.05, Table 4).

In a study that examined the effects of regular exercise on cardiovascular risk factors, researchers found that resting heart rate of people who exercised was lower than resting heart rate of people who did not (Mahanonda et al., 2000). Kerrie et al. (2001) noted a significant decrease in systolic but not in diastolic blood pressure after a 12 week walking exercise. Authors did not find significant changes in blood pressures in the control group (Kerrie et al., 2001). Janssen et al. (2001) reported a systolic pressure of 125 mmHg and a diastolic pressure of 77 mmHg. In another study, heart patients performed circuit training which resulted in a significant decrease in systolic blood pressure after exercise (Green et al., 2001). Fagard (2001) observed decreases in blood pressures following training three to five times a week, lasting 30 to 60 min per session, at an intensity of about 40 to 50% of net maximal exercise performance (Fagard, 2001). Oksüz (2004) reported 4 to 8 mmHg decrease in systolic blood pressure in people who exercised regularly. The author argued that walking 3 to 4 times a week for 30 to 45 min, at 60 to 70% of target heart rate was more effective than jogging. The decrease in RHR in response to aerobic exercise and the significant decrease in systolic blood pressure as opposed to insignificant decrease in diastolic pressure could be attributed to physiological adaptation to exercise (Williams et al., 2007) and, alleviation of arterial stiffness, total systemic arterial compliance and, consequently, decrease in blood pressure (Tanaka et al., 2000).

In the exercise group, pre- and post-exercise FG levels were  $111.33\pm7.19$  and  $91.25\pm5.70$  mg/dl, respectively. This 11.13% decrease was statistically significant (*P*<0.002, Table 3). Even though pre-exercise FG level was not significantly different between the exercise and control groups (*P*>0.05, Table 4), the 18.77% post-exercise difference between exercise and control groups was statistically very significant (*P*<0.00, Table 4).

Normand et al. (2005) carried out a study in which 280 males conducted a 20 week endurance exercise, whereby the subjects started the exercise with 55% of maxVO2 and ended with 75% of MaxVO2 after gradual increases and noted significant change in FG. In a 6 week treadmill exercise study (2005) on 20 subjects with a mean age of 52, Nayak et al. (2005) reported a 39.4% decrease. The authors argued that aerobic exercise was a means to regulated blood glucose level, attributing this to endurance exercise, facilitating glucose uptake and working muscle being more sensitive to insulin than muscle at rest.

As defined in the AHA ATP III, one of the MetS criteria, FG should exceed 110. In order to gain a metabolic benefit from exercise and have an impact on insulin resistance, the intensity of the exercise programs should be over 60% (Ronald et al., 2006). Researchers have emphasized that FG is decreased significantly and sensitivity to insulin was increased after structured, regular, long-term exercise (lan et al., 2007).

In the exercise group, there were 8.37, 15.65 and 12.34% decreases and 15.65% increase in Total-C,

triglyceride, LDL-C and HDL-C levels, respectively, between before and after exercise, with all changes being statistically significant (P<0.05, Table 3). When the post-exercise values were compared between exercise and control groups, changes in the triglyceride (19.22%) and HDL-C (18.72%) levels in the exercise group were statistically significant (P<0.05, Table 4).

The study of Fröhlich et al. (2000) showed that Total-C, triglyceride, LDL-C levels were elevated and HDL-C level was lowered in MetS. In a study by Linda et al. (2000), a 16 week aerobic, resistance or combined training given to three groups showed a decrease in triglyceride and increase in HDL-C levels in the aerobic training group. The authors reported improvements in lipoprotein and lipid profiles and body composition of the subjects in the aerobic training group at the end of the program while there were no changes in the resistance group (Linda et al., 2000). In another study, Arthur et al. (2000) investigated the effects of a 20-week bicycle ergometer exercise on plasma lipid levels on a total of 675 males and females, and reported decreases in Total-C and triglyceride levels, increase in HDL-C level after the exercise and a negative correlation between HDL-C increase and body fat in males. Our results are consistent with their findings.

The study of Couillard et al. (2001) on 200 males revealed that regular endurance exercise was useful in elevating HDL-C while decreasing triglyceride levels and excessive abdominal fat. They argued that the increase in HDL-C with endurance exercises was as a result of the decrease in body weight or body fat. In another study on 444 subjects with a mean age of 58±9 years, significant decreases in the levels of Total-C, triglyceride and LDL-C and a significant increase in HDL-C were reported following a 3-week cardiac rehabilitation program 3. Significant and beneficial, relationships have been reported between regular, moderate physical activity and plasma lipids, leading to the conclusion that physical activity could prevent coronary heart disease (Lakusic et al., 2004). Kim et al. (2001) studied the effects of frequency and intensity of exercise on lipid levels in male coronary heart disease patients demonstrated that increase in the number of exercise sessions being attended caused an increase in HDL-C, which, in turn, improved LDL-C and Total-C ratios. It has been argued that performing exercise more frequently was more effective on serum lipid levels than on the intensity of training (Kim et al., 2001) and that the level of physical activity strongly correlated with plasma HDL-C level (Al-Ajlan et al., 2005). It has been reported that regular physical activity of aerobic nature had positive effects on lipid metabolism and lipid profile, resulting in lowered Total-C, LDL-C and triglyceride levels and elevated HDL-C level (Lakusic et al., 2004; Kim et al., 2001). In the present study, significant decreases in serum lipid levels observed in the experiment group (P<0.05, Table 3) suggested an improvement in CVD risk, which is in line with the

literature findings (Scott et al., 2002).

In a study, C-RP decreased from  $1.91\pm0.34$  to  $1.01\pm0.19$  mg/dl following a 12 week exercise program. This corresponded to a statistically significant 47.12% decrease (*P*<0.05, Table 3). Comparison of the after-exercise values of the exercise and control groups showed 86.14% difference in C-RP in favor of the exercise group (*P*<0.001, Table 4).

Donges et al. (2010) carried out a study on 102 sedentary subjects and investigated the effects of resistance or aerobic exercise training on Interleukin-6, C-RP and body composition. They found that C-RP level in the aerobic exercise group was 32.8% lower. Another study reported high CRP levels in subjects with MetS (Ridker et al., 2001). C-reactive protein is a major acute phase reactant that has been identified a long time ago. Serum concentration of C-RP increases in the presence of inflammation, infection and tissue damage. As an acute phase reactant, C-RP facilitates removal of damaged cells by binding to phosphocholine (Gabay et al., 1999). C-RP adds to the predictive capacity of cardiovascular risk factors (Pearson et al., 2003). Numerous studies reported associations between high C-RP and low physical activity levels (Nicklas et al., 2005; Fischer et al., 2005). Kasapis and Thompson (2005) and Fallon et al. (2001) reported that chronic physical activity lowered C-RP significantly. Long-term, regular physical exercise also result in a decrease in C-RP as physical activity suppresses low grade inflammation (Keller et al., 2004). Low grade inflammation and long-term regular exercise protect people against the syndrome (Pedersen et al., 2003). In another study, Ford (2002) argued that physical activity reduced inflammation and had a critical impact on preventing the development of CVD. Despite certain constraints of the study such as small sample size, the results are consistent with previous findings such that it demonstrates the benefits of physical activity on known adverse effects of infection and obesity on chronic systemic inflammation in healthy individuals.

By implementing a 12 week long walking/running exercise program and adjusting the loading intensity between easy-somewhat hard, significant changes in the MetS components and C-RP levels were achieved in the subjects (P<0.05). Following the exercise program, comparison of the exercise and control groups showed significant differences in the MetS components and CRP in favor of the exercise group (P<0.05). By using OMNI scale, we excluded changes outside of these values and achieved a standard exercise.

To date, different and effective drugs are being used for the control of each of the components in people with MetS. However, persuading patients to exercise and getting into a habit of exercising regularly is a more potent and effective way. Moreover, patients need to be educated with regard to habit of exercising. One of the secondary protective measures in patients with MetS is personalized, regular and structured exercise. In various studies (Al-Ajlan et al., 2005; Fagard, 2001; Normand et al., 2005; Ridker et al., 2001), loading intensity was based on certain criteria. In the present study, loading intensity of the walking/running program was assessed by OMNI scale. We noted that the beneficial effects observed in MetS components and C-RP during classical practice was comparable to those when OMNI Scale was used.

In conclusion, by using OMNI scale to determine the loading intensity, people can plan and perform an exercise program without the need for a trainer or a gym by adjusting the intensity of exercise based on perceived exertion. This, in turn, will help get rid of the adverse effects of MetS and reduce inflammation that leads to development and progression of CVD.

#### REFERENCES

- Akram DS, Astrup AV, Atinmo T, Bossin JL, Bray GA, Caroll KK (2000). Obezity preventing and managing the global epidemic, Geneva: Who Organization, pp. 4-6.
- Al-Ajlan AR, Mehdi SR (2005). Effects and a dose response relationship of physical activity to high density lipoprotein cholesterol and body mass index among Saudis, Saud. Med. J., 26(7): 1107-1111.
- Alberti KG, Zimmet PJ, Shaw J (2006). Metabolic syndrome a new world-wide definition, Diabet. Med., 23(5): 469-480
- Alberti KG, Zimmet PZ (1998). Definition, diagnosis and classification of diabetes and its complications part 1: Diagnosis and classification of diabetes provisional report of a WHO consultation, Diabet. Med., 15: 539-553.
- Amano M, Kanda TU, Maritani T (2001). Exercise training and autonomic nervous system activity in obese individuals, Med. Sci. Sport. Exerc., 33(8): 1287-1291.
- American College of Sports Medicine Position Stand (2004). Exercise and hypertension, Med. Sci. Sport. Exerc., 36(3): 533-552.
- American Heart Association Treatment Panel (2002). Evaluation and treatment of high blood cholesterol in adults, Circulation, 106: 3143-421.
- Arnt ET, Oivind R, Tomas S, Anja B, Per MH, Jan PL, Stig AS, Ulrik W, Sang JL, Qusay Y, Al S, Sonia MN, Erik S, Ole JK (2008). Aerobic interval training versus continuous moderate exercise as a treatment for the metabolic syndrome, Circulation, 118: 346-354.
- Arthur S, Leon TR, Stephen M, Jean PD, Jean BJ, Gagnon DC, Rao JS, Skinner JH, Wilmore CB (2000). Blood lipid response to 20 weeks supervised exercise in a large biracial population: the heritage family study, Metabolism, 49(4): 513-520.
- Barbara JN, Xuewen W, Tongjian Y, Mary FL, Jamehl D, Linda E, Michael JB, Leon L, Jeffrey C (2009). Effect of exercise intensity on abdominal fat loss during calorie restriction in overweight and obese postmenopausal women, Am. J. Clin. Nutr., 89(4): 1043-1052.
- Couillard C, Despres PJ, Lamarche B, Bergeron J, Gagnon J (2001). Effects of endurance exercise training on plasma hdl cholesterol levels depend on levels of triglycerides evidence from men of the health risk factors, exercise training and genetics family study, Arteiosc. Thromb. Vasc. Biol., 21: 1226-1232.
- David E, Laaksonen HL, Leo K, Niskanen GA, Kaplan JT, Salonen TA (2002). Metabolic syndrome and development of diabetes mellitus: application and validation of recently suggested definitions of the metabolic syndrome in a prospective cohort study, Am. J. Epidemiol., 156(11): 1070-1077.
- Donges CE, Duffield R, Drinkwater EJ (2010). Effects of resistance or aerobic exercise training on interleukin-6, c-reactive protein, and Body composition. Med. Sci. Sport. Exerc. 42(2): 304-313.
- Fagard RH (2001). Exercise characteristics and the blood pressure response to dynamic physical training, Med. Sci. Sport. Exerc., 33(3):484-492.

- Fallon KE, Fallon SK, Boston T (2001). The acute phase response and exercise court and field sports, Br. J. Sport. Med., 35: 170-173.
- Fischer CP, Berntsen A, Perstrup LB, Eskildsen P, Pedersen BK (2005). Plasma levels of interleukin-6 and C-reactive protein are associated with physical inactivity independent of obesity, Scand. J. Med. Sci. Sport., 17: 580-587.
- Ford ES (2002), Does Exercise Reduce Inflammation? Physical activity and C-reactive protein among us adults, Epidemiology, 13: 5, 561-568.
- Ford ES, Giles WH, Dietz WH (2002). Prevalence of the metabolic syndrome among us adults: Findings from the third national health and nutrition examination survey, J. Jama., 287: 356-359.
- Fröhlich M, Imhof A, Berg G, Hutchinson WL, Pepys MB, Boeing H, Muche R, Brenner H, Koenig W (2000). Association between Creactive protein and features of the metabolic syndrome. Diabet. Care., 23(12): 1835-1839.
- Gabay C, Kushner I (1999). Acute-phase proteins and other systemic responses to inflammation, N. Engl. J. Med., 340: 448-454.
- Green DJ, Watts K, Maiorana AJ, Driscoll JG (2001). A Comparison of ambulatory oxygen consuption during Circucit training and aerobic exercise in patients with chronic heart failure, J. Cardio. Rehabil., 21(3): 167-174.
- Grundy SM, Brewer BH, Cleeman JI, Smith SC, Lenfant C (2004). Definition of metabolic syndrome, Circulation, 109: 433-438.
- Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA (2005). Diagnosis and management of the metabolic syndrome. Circulation, 112: 2735-2752.
- Gunter EW, Lewis BG, Koncikowski SM (1996). Laboratory procedures used for the third national health and nutrition examination survey 1988-1994. Centers for Disease Control and Prevention, National Center for Enviroment, J. Healt., pp. 245.
- Ian G, Chairperson DA, Knut BG, Gudrun B, Gunilla B, Renata C (2007). European guidelines on cardiovascular disease prevention in clinical practice: executive summary, Eur. Heart J., 28: 2375-2414.
- Jacops KA, Paul DR, Sherman WM (2000). Fat Metabolism. In Garrett WE, Kırkendal DT. (Eds). Philadelphia, Lippincott Williams and Wilkins, Exerc. Sport Sci., pp 12.
- Janine H, Dorret I, Boomsma JM, Vink, BK, Nicholas GM, Axel S, Kristen OK, Richard JR, Urho MK, Jaakko K, Jenifer RH, Nancy LP, Janice H, Tim DS, Eco JCG (2006). Genetic influences on exercise participation in 37.051 twin pairs from seven countries, Plos one., 1(1): 22-29
- Janssen I, Katmarzyk PT, Ross R (2002). Body mass index, waist circumference and health risk: evidence in support of current national institutes of health guidelines, Arch. Int. Med., 14: 2074-2079.
- Kasapis C, Thompson PD (2005). The effects of physical activity on serum C-reactive protein and inflammatory markers, J. Am. Coll. Cardiol., 45(10): 1563-1569.
- Keller C, Keller P, Giralt M, Hidalgo J, Pedersen BK (2004). Exercise normalises overexpression of Tnf-alpha in knockout mice, Biochem. Biophys. Res. Com., 321(1): 179-182.
- Kerrie LM, Degarmo R, Langley J, Mcmahon C, Howley, ET, Bassett, DR, Thompson LD (2001). Increasing daily walking lowers blood pressure in postmenopausal women, Med. Sci. Sport Exerc., 33(11): 1825-1831.
- Kim JR, Oberman AL, Fletcher GF (2001). Effect of exercise intensty and frequency on lipid levels in men with coronary heart disease training level comparison Trial, Am. J. Cardio. 87: 942- 946.
- Lakusic N, Mahovic D, Ramqaj T, Cerovec D, Grbavac Z, Babic T (2004). The effect of 3-weeks stationary cardiac rehabilitation on plasma lipids level in 444 patients with coronary heart disease, Coll. Antro., 28(2): 623-629.
- Leslie IK, Eugene R B, Eric GC, Ellen MR, John DS (1995). Effects of weight loss aerobic exercise training on risk factors for coronary disease in healthy obese middle-aged and older men, Jama., 274(24): 1915-1921.
- Linda ML, Serge PD, Joseph A, Jodi MK, Sara AC, Joseph R (2000). Lipit and lipoprotein profiles, cardiovascular fitness, body composition and diet during and after resistance, aerobic and combination training in young woman, Eur. J. Appl. Phys., 82: 451-458.

- Mahanonda N, Bhuripanyo K, Leowattana W (2000). Regular exercise and cardiovascular risk factors, J. Med. Assoc., 83(2): 153-158.
- Meigs JB (2002). Epidemiology of the metabolic syndrome, Am. J. Manag. Care., 8: 283-292.
- Nayak S, Arun Maiya A, Hande M (2005). Influence of aerobic treadmil exercise on blood glucose homeostasis in noninsulin dependent diabetes mellitus patients, Indian J. Clin. Biochem., 20(1): 47-51.
- Nicklas BJ, You T, Pahor M (2005). Behavioural treatments for chronic systemic inflammation, C. Maj., 172: 1199-1209.
- Normand G, Boul ES, John W, Timo A, Angelo T, Richard NB, Tuomo R, Arthur S, James S, Skinner JH, Wilmore DC, Claude B (2005). Effects of exercise training on glucose homeostasis, Diabet. Care., 28(1): 108-114.
- Patrick M, Christopher DB, Nicholas H, Nicholas ED, Nicholas JW (2001). Do different dimensions of the metabolic syndrome change together over time? Diabet. Care., 24(10): 1758-1763.
- Pearson TA, Mensah GA, Alexander RW (2003). Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the centers for disease control and prevention and the american heart association, Circ., 107: 499-511.
- Pedersen BK, Steensberg A, Fischer C, Keller C, Keller P, Plomgaard P, Febbraio M, Saltin B (2003). Searching for the exercise factor is IL-6 a candidate, J. Musc. Res. Cell. Metabol. 24: 113-119.
- Pyorala M, Miettinen H, Halonen P, Laakso M, Pyorala K (2000). Insulin resistance syndrome predicts the risk of coronary heart disease and stroke in healthy middle-aged men: the 22-year follow-up results of the helsinki policemen study, Arteriosc. Thromb. Vasc. Biol., 20: 538-544.
- Ridker PM, Stampfer MJ, Rifai N (2001). Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein and standard cholesterol screening as predictors of peripheral arterial disease. Jama., 285(19): 2481-2485.
- Ronald JS, Glen PK, David HW, Carmen CS, Russell DW (2006). Physical activity in then treatment of type 2 diabetes. Diabet. Care., 29(6): 1433-1438.
- Sang KP, Jae HP, Yoo CK, Mi SY, Chang SK (2003). The effect of longterm aerobic exercise on maximal oxygen consumption, left ventricular function and serum lipids in elderly women, J. Physiol. Anthro. App. Hum. Sci., 22 (1):11-17.
- Scott M, Grundy MD, Diane SD, Luther TC, Richard SC, Margo A (2002). Detection evaluation and treatment of high blood cholesterol in adult final report, Circulation, 106: 3143-3421.
- Speakman JR (2004). The integrated roles of environment and genetics, J. Nutr., 134(8): 20905-21055.
- Tanaka H, Dinenno FA, Monahan KD, Clevenger CM, Desouza CA, Seals DR (2000). Aging habitual exercise and dynamic arterial compliance, Circulation, 102(11): 1270-1275.
- Timo A. Lakka HM, Tuomo R, Arthur S. Leon DC, Rao JS, Skinner J, Wilmore H, Claude B (2005). Effect of exercise training on plasma levels of C-reactive protein in healthy adults: the heritage family study, Eur. Heart. J., 26: 2018-2025.
- Utter A, Robertson RJ, Green JM, Suminski RR, Mcanulty SR, Nieman DC (2004). Validation of the adult Omni scale of perceived exertion for walking running exercise, Med. Sci. Sport Exerc.., 36: 1776-1780.
- Williams MA, William LH, Philip AA, Ezra AA, Bittner V, Franklin BA, Gulanick M, Laing ST, Stewart KJ (2007). Scientific statement from the American heart association resistance exercise in individuals with and without cardiovascular disease, Circulation, 116: 572-584.
- Zhengming C, Gonghuan Y, Maigeng Z, Margaret S, Alison O, Jieming M (2006). Body mass index and mortality from ischaemic heart disease in a lean population, Intl. J. Epid., 35(1): 141-150.