

A COMPARATIVE STUDY OF FAT PARAMETERS IN SEDENTARY AND NON-SEDENTARY SUBJECTS

Jayalakshmi. M.K¹, Ajay K.T², SmileeJohncy.S³, Dhanyakumar.G⁴, Suresh Y. Bondade⁵

¹Assistant Professor, ^{2&3}Associate Professors, ⁴Lecturer,

⁵Professor & Head Department of Physiology, JIM Medical College, Davangere-577004. Karnataka, India.

*Corresponding Author Email: jayalakshimalavar@gmail.com

ABSTRACT

BACKGROUND: Recent studies in adolescents and adults have demonstrated significant relationship between physical inactivity and other adverse health practices, such as consumption of less-healthy foods or increased fat intake. Inactive individuals tend to consume more quantities of dietary fat. These data suggest that inactivity tends to cluster with other health behaviors that have adverse effect on the quantity and location of body fat deposition which results in obesity. A sedentary life style and poor cardio-respiratory fitness not only associated with the metabolic syndrome but could also be considered features of the metabolic syndrome. Many studies have shown an inverse relation between physical activity coronary arterial disease and atherosclerosis. A combination of improper diet, lack of physical activity causes premature coronary heart disease. **METHODS:** 45 sedentary and 55 non-sedentary healthy female subjects were selected randomly from the general population of Davangere city. Weight, Height was taken. BMI was calculated. Fat parameters such as Body Fat Percentage (BF %), Fat Mass (FM), Fat Free Mass (FFM) and Fat Mass Index (FMI) were calculated from the formula. **RESULTS:** Fat parameters such as BF%, FM were increased. FFM decreased in sedentary subjects when compared to non-sedentary subjects. Physical inactivity causes excess fat accumulation which affects anthropometric and cardiovascular parameters. Our study provides a glimpse into the variety of adaptations /alterations in anthropometric and fat parameters that occurs due to sedentary life style, even in the absence of overt disease.

KEY WORDS

Body Fat%, Fat Mass, Fat Free Mass, Sedentary lifestyle.

INTRODUCTION

A sedentary lifestyle has been linked to elevated cholesterol, cardiovascular disease, hypertension, increased blood pressure, obesity, diabetes, increased stress, low endurance, and depression. Sedentary living decreases bone density, which result in increased risk of developing osteoporosis. Obesity is a progressing problem both in developed or developing countries. Relations between increased body fat and high mortality especially with cardiovascular diseases, diabetes mellitus, and hypertension are well known. Several large studies suggest that abdominal obesity is closely related to cardiovascular risk.

The way that fat is distributed also plays an important role. Body fat that accumulates around the waist known as abdominal fat (an 'apple shape') poses a greater health risk than fat carried in the hips and thighs (a 'pear shape'). Men are genetically predisposed to weight gain around their waist, although there are exceptions. By contrast, women's body tends to be more 'pear shape'. Overweight men also tend have more visceral fat, which subsequently increases the risk of heart disease, metabolic syndrome and diabetes.¹

Body Mass Index (BMI) has extremely high specificity but lower sensitivity compared to other tests for estimating fat content of the body. Ideal body weight is defined as a BMI of 18.5 to 24.9 kg/m². Overweight is defined >25 to 29.9 kg/m² and obesity is defined as BMI > 30 kg/m². Abdominal adipose tissue has higher fat cell number, high blood flow, increased receptors for cortisol, testosterone and greater catecholamine induced lipolysis when compared with adipose tissue present elsewhere, which might explain its increase association with diabetes and other metabolic diseases.²

Those who remain or become inactive are usually heavier than those who are physically active.³ Within a permissive environment, the more common genetic factors involved in obesity regulate the distribution of body fat, the metabolic rate with its response to exercise and diet, the control of feeding and food preferences.⁴ Regular exercise improves insulin sensitivity, decreases plasma triglyceride levels and reduces cardiovascular morbidity and mortality.⁵ Energy balance is essentially equivalent to fat balance and since increased fat intake does not stimulate fat oxidation, clearly there is a potential for fat imbalance and hence obesity with high fat intake.⁶ High waist hip ratio (WHR) > 1.0 in men and > 0.85 in women indicates abdominal fat accumulation. Risks of metabolic complications increase in men with waist circumference (WC) > 102 cms and women with WC > 88 cms.⁷

The adipose tissue is not simply a passive storehouse for fat but an endocrine organ that is capable of synthesizing and releasing into the blood stream an important variety of peptides and non-peptides compounds that may play a role in cardiovascular homeostasis. Adipose tissue is a significant source of Tumor Necrosis Factor-alpha (TNF- α), Interleukin-6, Plasminogen activator inhibitor - 1, Leptin, Angiotensinogen and Insulin like growth factor-1 (IGF-1).⁸

Excess of adipose tissue augments cardiac output, stroke volume, left ventricular filling pressure and expands intravascular volume. There is increased prevalence of high blood pressure associated with obesity results from a discrepancy between raised cardiac output and a relatively normal arterial capacity.⁹ Fat-free mass, fat mass, age and sex account

for about 80% of the variance in BMR was related to a familial factor, suggesting that BMR is, at least partly genetically determined.¹⁰ The WHR is more significant than total degree of obesity and associated with hypertension, dyslipidemia, insulin resistance and increased coronary artery disease mortality.¹¹

The effect of different patterns of body fat distribution on mortality confirms earlier clinical observations that an abdominal rather than gluteal distribution of fat increases susceptibility to health hazards including cardiovascular diseases and diabetes mellitus. Therefore distribution fat should be analyzed in its own right; independent of overall obesity by clinically obtaining WHR.¹² Individuals who are relatively inactive are more likely to gain weight than those who frequently engage in physical activity. The relative risk of gaining 5 kg or more during a 5 years follow-up study among inactive Finns was 1.6 in women and 1.9 in men.¹³

A 10% increase in body weight is associated with decline in parasympathetic tone accompanied by a rise in Mean heart rate and conversely, heart rate declines during weight reduction.¹⁴ Body fat and sympathetic nerve activity in healthy subjects were studied and it was found that resting rate of sympathetic nerve discharge to skeletal muscle was directly correlated with BMI and percent of body fat. In addition to body fat, muscle sympathetic nerve activity was correlated with age, plasma insulin concentration, and plasma lactate concentration. Together these four co-variants accounted for 58 % of the variance of the muscle sympathetic activity. It was concluded that in healthy humans, body fat is a major determinant of the resting rate of muscle sympathetic discharge. Overweight associated with sympathetic activation could represent one potential mechanism contributing to the increased incidence of cardiovascular complications in overweight subjects.¹⁵ Not only the age but also BMI, BF% and WHR influence serum lipids, apoproteins and fasting blood glucose in Thai women evidenced by the higher correlation coefficients.¹⁶

Systolic BP increased by 6 mmHg and diastolic BP by 4 mmHg for a 10% gain in body fat. Weight loss of 1 kg produced a 20% decrease in both systolic and diastolic blood pressure in hypertensive patients even when the sodium intake was kept constant. The most

characteristic lipid disorder in obesity was elevated total cholesterol and triglycerides, high LDL cholesterol and low HDL cholesterol. For every 1 kg of weight loss, there was corresponding reduction by about 1% in total cholesterol and LDL, a rise by 1% in HDL and a reduction by 3% in triglycerides.¹⁷ There is evident linking increased physical activity to a more favorable fat distribution (a lower proportion of visceral fat at a given BMI).¹⁸

Low level of physical activity is associated with an increased risk of weight gain and obesity.¹⁹ Lower physical activity was associated with higher subcutaneous adipose tissue and visceral adipose tissue volume in both the sex.²⁰ Higher fatness in sedentary subjects than in sportspersons, though BMI had insignificant variation.²¹ Central obesity significantly and independently contributes to cardiovascular outcome.²² Body mass index (BMI) and waist circumference are strongly correlate with total body adipose tissue but waist circumference is a better predictor of Intra-Abdominal Adipose Tissue (IAAT) than BMI.²³

METHODOLOGY

The study was undertaken to analyze the differences in certain fat parameters in healthy sedentary and non-sedentary subjects in the age group of 25 - 55 years. 45 sedentary subjects and 55 healthy non-sedentary female subjects were selected from the general population of Davangere city randomly. A simple two compartment model that divides the body into fat and fat free mass may be obtained using age and gender specific regression equations that incorporate the BMI of the individual, one such equation is that of Deurenberg, which is used in the practice.²⁴

The factors that may affect fat and fat-free mass includes the following: Gender, Age, Race, Food intake, Physical activity/ athletic training, Drugs/stimulants and disease.²⁵

The methodology adopted to determine the body fat percentage in the present study was based on BMI by using Deurenberg's equation.

The exclusion criteria in this study were
 Subjects suffering from endocrinal disorders

Hypertensive individuals

Subjects with renovascular and cardiovascular diseases.

Pregnant and lactating women.

All the subjects gave consent after explaining the procedure of the non-invasive technique to them. A brief personal history, childhood obesity, detailed history of exercise and a clinical examination of all the systems were done to exclude medical problems and to prevent confounding of results.

Weight and Height was taken, Body Mass Index (BMI) was derived by Quetlet's index from body weight (kg) / Height (m²) which were used for the calculation of fat parameters (Table.1)

Body Fat Percentage (BF %) was calculated by using the formula: $BF\% = 1.2 \times BMI + 0.23 \times Age - 10.8 \times Sex - 5$. (Where, Male = 1 and Female = 0).

Fat Mass (FM) was calculated in the following way

$FM = \text{Weight} \times BF\%$ and expressed in kgs.

Fat Free Mass (FFM) was calculated and expressed in kilograms (kg) by using the formula $FFM = \text{Weight} - \text{Fat mass}$.

Fat Mass Index (FMI) was calculated from Fat mass in (kg) / Height in (m²).²⁶

STATISTICAL ANALYSIS

The results were given as Mean ± Standard Deviation and range values. Comparisons were made between sedentary and non-sedentary subjects. A p-value of 0.05 or less was considered as statistical significance.

RESULTS

There was statically significant increase in BF % & FM (kg) in sedentary subjects when compared to non-sedentary subjects. There was slight increase in Mean FMI (kg/m²) in sedentary subjects when compared to non-sedentary subjects which was statistically not significant. There was slight decrease in FFM (kg) in sedentary subjects when compared to non-sedentary subjects which was statistically not significant (Table. 1 and 2)

DISCUSSION

In our study BMI was increased in sedentary subjects. Recent studies in adolescents and adults have demonstrated significant relationship between

physical inactivity and other adverse health practices, such as consumption of less-healthy foods or increased fat intake. Inactive individuals tend to consume more quantities of dietary fat. These data suggest that inactivity tends to cluster with other health behaviors that have adverse effect on the quantity and location of body fat deposition which results in obesity.²⁷ It is hypothesized that excess catecholamine triggers various adverse processes which, if persist, can lead or aggravate hypertension and insulin resistance. Visceral fat but not peripheral fat mass was correlated with atherogenic effect.²⁸

The mean BF% was increased, the methodology adopted to determine the body fat percentage in the present study was based on BMI by using Deurenberg's equation whereas, most of the reports on body fat percentage were based on the use of techniques like bioelectrical impedance, hydrodensitometry, X-ray absorptiometry and skin fold thickness.

In our study there was increased FM (kg), FMI (kg/m²) and decreased FFM (kg) in sedentary subjects when compared to non-sedentary subjects.

CONCLUSION

The conclusions of our study are:

Body fat percentage and fat mass were increased significantly in sedentary subjects, there was slightly increased in FMI (kg/m²) in sedentary though statistically not significant.

There was decreased Mean FFM (kg) in sedentary male subjects though statistically not significant.

Though our study is by no means exhaustive, it does provides glimpse into the variety of adaptations /alterations in anthropometric and cardiovascular structure and function that causes adipose tissue accumulation, even in the absence of overt disease. Hormonal assay and lipid profile estimation along with fat parameters would have given a better understanding about sedentary life style and its consequences. We need to evaluate the strategies and efficacy of physical activity in various diseases. The benefits of regular physical activity are numerous, people who exercise live longer and healthier.

Table.1: Anthropometric parameters in sedentary and non-sedentary subjects

GROUP	BMI (Kg/m ²)		WHR		MAC (cms)		
	N	Range	Mean	Range	Mean	Range	Mean
Sedentary	45	18.6-33.3	26.3 ± 3.3	0.84-0.99	0.93 ± 0.03	21 – 23	27.9 ± 2.2
Non-sedentary	55	14.2-38.7	23.3 ± 2.1	0.82– 1.05	0.91± 0.03	22 – 29	26.1 ± 1.3
Mean difference		3.0		0.02		1.8	
Significance	t	5.72		2.59		5.25	
	P	< 0.001, HS		< 0.05, S		< 0.01, S	

All values expressed as Mean ± SD.

Analysis for all parameters done by unpaired 't' test.

HS-Highly significant, S- Significant, NS- Not significant.

BMI- Body Mass Index, WHR- Waist Hip Ratio, MA Range Mean ± SD

MAC- Mid Arm Circumference

Table 2: COMPARISON OF FAT PARAMETERS BETWEEN SEDENTARY AND NON-SEDENTARY SUBJECTS

Group	BF%		FM (Kg)		FFM(Kg)		FMI (Kg/m ²)		
	N	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD	Range	Mean ± SD
Sedentary	45	23.1- 45.3	35.3 ± 5.1	10.2- 35.8	22.6 ± 5.8	33.9 – 46.8	39.9 ± 4.5	4.3- 22.8	12.1 ± 4.1
Non-sedentary	55	21.7-42.4	31.0 ± 4.2	10.0- 32.2	18.0 ± 3.8	29.5- 57.8	40.5 ± 3.5	4.2- 18.0	11.1 ± 4.6
Mean difference			4.3		4.6		0.7		1.0
Significance t			4.75		4.94		0.90		1.19
p			< 0.001, HS		< 0.001, HS		0.37, NS		0.24, NS

All values expressed as Mean ± SD.

Analysis for all parameters done by unpaired 't' test.

HS-Highly significant, S- Significant, NS- Not significant.

BF% - Body Fat Percentage, FM – Fat Mass, FFM – Fat Free Mass, FMI – Fat Mass Index

ACKNOWLEDGEMENTS

Authors are grateful to the Principal J.J.M.Medical College, Davangere for his support and encouragement, to the statistician and to all the volunteers who participated in this study.

REFERENCES

- Matsuzawa Y, Shimomura I, Nakamura T, Keno Y, Tokunaga K. Pathophysiology and pathogenesis of visceral fat obesity. *Diabetes Res ClinPract* 1994; 24: S111-6.
- Thomas CS, Krishnaswami S. Distribution of body mass index in Indian patients with coronary artery disease. *Indian heart J* 1995; 47: 134-7.
- Kopelman PG, Ian D, William C, Dietz H. *Clinical obesity in adults and children*. 2nded. BlackWell Publications; 2005; 269-80.
- Vohl MC, Sleddek R, Robitaille J, Gurd S, Marceau P, Richard D, et al. A survey of genes differentially expressed in subcutaneous and visceral adipose tissue in men. *Obes Res* 2004; 12:1217-22.
- Koplan JP, Dietz WH. Caloric imbalance and public health policy. *JAMA* 1999; 282: 1579-81.
- Zurlo F, Lillioja S, Knowler WC. Low ratio of fat and carbohydrate oxidation as a predictor of weight gain: study of 24-hr RQ. *Am J Physiol* 1990; 259:650-7.
- World Health Organization. WHO: Tech Rep Ser 916; 2003.
- Hotamisligil GS, Arner P, Caro JF, Atkinson RL, Spiegelman BM. Increased adipose tissue expression of tumor necrosis factor-alpha in human obesity and insulin resistance. *J Clin Invest* 1995; 95:2409-15.
- Messerili FH. Cardiovascular effects of obesity and hypertension. *The Lancet* 1982; 1165-1168.
- Bogardus C, Lillioja S, Ravussin E. Familial dependence of the resting metabolic rate. *N Engl J Med* 1986; 315:96-100.
- Depress J P. Regional distribution of body fat, plasma lipoproteins and cardiovascular disease. *Atherosclerosis* 1990; 10: 497-511.
- Tanphaichitr V, Kulapongse S, Pakpeankitana R, Leelahagul P, Tamwiwat C, Loclaya S. Prevalence of obesity and its associated risk in urban Thais. Oomuray, Tarui S, Shimazu T, ed. *Progress in obesity research* 1990: 649453.
- Rissanen AM, Heliovaaram M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. *Eur J Clin Nut*1991; 45:419-430.
- Hirsch J, Leibel RL, Mackintosh R, Aguirre A. Heart rate variability as a measure of autonomic function during weight change in humans. *Am J Physiology* 1991; 261:1418-23.
- Scherrer U, Randin D, Tappy L, Volleweider P, Jequier E, Nicod P. Body fat and sympathetic nerve activity in healthy subjects. *Circulation* 1994; 89:2634-2640.
- Leelahagul P, Soipet S, Acharyont P, Pakpeankitvatana R, Tanphaichitr V. Influence of body composition on risk factors for coronary heart disease in Thai women. *Pacific J ClinNutr* 1995; 4:79-80.
- Jung R. Obesity as a disease. *British Medical Bulletin* 1997; 53(2):307-21.

18. Siedell JC. Environmental influences on regional fat distribution. *International Journal of obesity* 1999; 15 (2):31-35.
19. Jobb SA, Moore MS. Contribution of a sedentary lifestyle and inactivity to the etiology of overweight and obesity: current evidence and research issues. *Med sci Sports Exerc* 1999; 11:S534-41.
20. Komiya H, Mori Y, Yokose T, Tajima N. Smoking as a risk factor for visceral fat accumulation in Japanese men. *Tohoku J ExpMed* 2006; 208:123-32.
21. AmithBandyopadhyay. Anthropometry and body composition in soccer and volleyball players in West Bengal, India. *J PhysiolAnthropol* 2007; 66: 384-94.
22. Dhaliwal SS, Wellborn TA. Central obesity and multivariable cardiovascular risk as assessed by the Framingham prediction scores. *Am J Cardiol* 2009;103 (10); 1403-7.
23. Klein S, Allison DB, Heymsfield SB, Kelly DE, Leibel RL, Nonas C, et al. Waist circumference and cardiovascular risk. *Am J ClinNutr* 2007; 85: 1187-202.
24. Deurenberg P, Wstrasted JA, Seidll J. Body mass index as a measure of body fatness;Age and sex specific prediction formulas” *Br J Nutr* 1991;65(2):105-114.
25. Shetty PS, James WPT. “Body Mass Index, A measure of chronic energy deficiency in adults” In *FAO and Food and Nutrition, FAO, ROME* 1994; 56.
26. Vanitallie TB, Yang MU, Heymsfield SB, Funk RC, Boileau RA. “Height normalized indices of the body’s fat free mass and potentially useful indicator of nutritional status”. *Am J ClinNutr* 1990; 52:953-59.
27. The role of lifestyle in health: epidemiology and consequences of inactivity. *Proceedings of the Nutritional Society.* 1996; 55:829-40.
28. Heinz R, Bernhard M. Abdominal fat and sympathetic over activity. *Herz* 2003; 28:668-73.



***Corresponding Author:**

Dr. Jayalakshmi MK

Assistant Professor,
Department of Physiology.

JJM Medical College,
Davangere -577004.

Karnataka, India.

E-mail- iavalakshmimalavar@gmail.com