Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/jasi

Original Article

Effect of physical exercise on the body morphology of obese females

Ruchira Sethi[a,*], Rashi Singh[b], Vishram Singh[c]

[a] Assistant Professor, Department of Anatomy, Santosh Medical College, Ghaziabad, NCR Delhi, India
[b] Department of Pedodontics and Preventive Dentistry, Santosh Dental College, Ghaziabad, NCR Delhi, India
[c] Professor & Head, Department of Anatomy, Santosh Medical College, Ghaziabad, NCR Delhi, India

ARTICLE INFO

Article history:
Received 21 October 2013
Accepted 7 November 2013
Available online 30 December 2013

Keywords:
Body morphology
Obesity
Female

ABSTRACT

Introduction: The aim of present study is to evaluate the impact of physical exercise on body morphology of obese female as assessed by anthropometric measures of weight, body mass index (BMI) and waist–hip ratio (WHR) along with assessment of blood lipid levels.
Material & methods: Hundred females of 30–40 years of age were selected and grouped in two categories of 50 each; control group and experimental group on the basis of blood lipid levels. Both the groups were subjected to controlled and similar physical exercise regimen for a period of three months. The anthropometric measures and blood lipid levels were assessed pre and post exercise.
Results: In control group (group 1), before exercise the WHR was observed to be from 0.7 to 0.8 while after exercise there was no significant fall in WHR, however there was a significant reduction in weight and BMI. On the other hand, in experimental group (group 2), in 42 females (before exercise) the WHR was >0.8 while in 8 females it ranged from 0.7 to 0.8. While after exercise there was a significant fall in weight, BMI along with WHR in these females. The above changes in experimental group were associated with significant fall in blood lipid levels except HDL.
Conclusion: It was concluded that the measurement of WHR is a significant indicator of regional body fat distribution. It was also concluded that independent of the general BMI levels the higher values of WHR are suggestive of adverse blood lipid levels in an individual. A guided physical exercise schedule even for a short period of 3 months can cause decrease in WHR and reversal of body morphology and reduction in total cholesterol, TGL, LDL and an elevation in HDL levels.

1. Introduction

Obesity in recent times has become an increasingly important health problem worldwide, including the developing countries. In India also almost 30%–65% of adult urban individuals are either overweight or obese.[1] The rising prevalence of obesity in India has not only increased prevalence of obesity related morbidities such as hypertension, metabolic syndromes, dyslipidemia, type 2 diabetes mellitus,

* *Corresponding author.* Tel.: +91 9873621520 (mobile).
E-mail address: ruchirasethi@gmail.com (R. Sethi).
0003-2778/$ – see front matter
http://dx.doi.org/10.1016/j.jasi.2013.11.001

cardiovascular diseases etc but also changed the body morphology of an individual.[2,3]

According to the pattern of fat distribution, the obesity is divided into two types: (a) the *Android type obesity with apple-shaped body morphology* and (b) the *Gynoid type obesity with pear-shaped body morphology*.[4] The Android (or male) type obesity is characterized by central distribution of fat in abdomen whereas the Gynoid (or female) type obesity is characterized by peripheral distribution of fat in the hips and thighs (Fig. 1).

There are enormous evidences suggesting the role of gender specific hormones in defining the type of body morphology (*vide supra*) but the recent changes in life style and consumption of high energy level food has produced profound impact in altering body morphology of the individuals, especially in females.[5,6] Females are relatively less active than males in all age-groups.[7,8] They experience higher incidence of weight related diseases and experience more body image dissatisfaction, low self-esteem and eating disorders at a much higher rate than males. Physical exercises have long been acknowledged as an important part of healthy life style and various scientific evidences have also linked physical exercises to wide range of physical and mental health benefits. Apart from playing an important role in the promotion of health, physical activity contributes to be a significant factor in altering body morphology of an individual.

Studies on the implications of physical activity for disease prevention such as cardiovascular disease, hypertension etc, their management and rehabilitation are enormous[2,3,5,6] but the relationship between physical exercise and altering body morphology have not been adequately understood and addressed and hence this fact needs to be explored further. Therefore the uniqueness of this study lies in the fact that it associates the benefits of physical exercises to WHR and body morphology in the reproductive period of women's life.

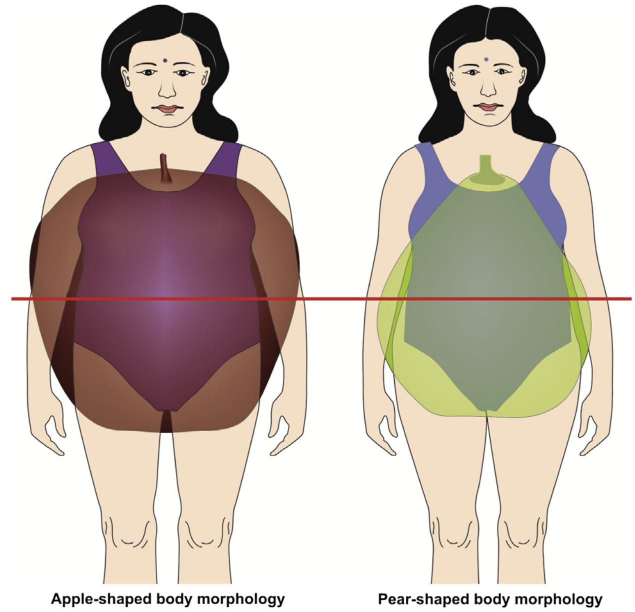

Fig. 1 – Body morphology.

2. Materials & methods

To conduct this study, 100 women were enrolled for a period of 3 months intervention. *Inclusion criteria*: Females of age group between years 30 and 40 with BMI between 26 and 39 kg/m^2, with normal hormonal assay and absence of contraindications to any form of physical exercise. *Exclusion criteria*: Subjects suffering from type II diabetes mellitus, hypertension, taking lipid-lowering drugs or medications that affected body weight and pregnant females were excluded from the study.

The diet pattern of subjects was assessed by food frequency questionnaire, with respect to daily calorie intake and was re-assessed during the intervention at intervals of 15 days.[9,10] All subjects underwent a comprehensive medical examination and routine blood tests, before subjecting them for physical exercise regimen. The subjects underwent a regular physical activity schedule that included 40 min of active cardio-workout with 15 min of strength training for a period of five days a week. This regime was strictly monitored by us and continued for a total duration of three months.

The measurements of height, weight, waist and hip circumferences were taken to calculate Body mass index (BMI) & waist–hip ratio (WHR). The serum lipid profile was done by autoanalyzer in the laboratory. All the parameters were assessed at baseline and after 12 weeks of regular programmed physical exercises. Anthropometric measurements (*vide supra*) were taken in a uniform manner with subjects wearing light clothing. Weight and height were obtained using calibrated balance scales, and BMI (Quetelet index) (kg/m^2) was calculated as a measure of weight relative to height.[11] Waist circumference was measured at the level of umbilicus, and hip circumference was measured at the widest part of the hips. The total blood cholesterol, triglycerides, LDL-cholesterol and HDL-cholesterol were measured by automated procedures and high blood cholesterol was defined as total serum cholesterol >240 mg/dL (6.21 mM), low HDL-C was defined as <45 mg/dL (1.16 mM).[12–14]

The 100 women selected were grouped into two groups:

Group 1, included 50 women with normal lipid levels, *(control group)*

Group 2, included 50 women with abnormal lipid levels, *(experimental group)*

In both the groups the anthropometric measurements were used to calculate the BMI and WHR.

3. Results

It was observed that in group 1 all 50 females had normal WHR (0.7–0.8), while in group 2, 42 females had WHR more than 0.8 whereas 8 females had WHR less than 0.8. After 3 months of guided physical exercise schedule, the anthropometric parameters and blood lipid levels were reassessed and recorded in both the groups .The changes in various anthropometric indices and blood lipid levels for the control group (group 1) are depicted in Table 1 and for the experimental group (group 2) are depicted in Table 2.

Table 1 – Changing parameters in control group.

Group 1 ($n = 50$)			
Parameter	Pre-test	Post-test	p-value
Weight (kg)	71.16 ± 4.29	67.66 ± 4.24	<0.05
BMI	32.88 ± 3.55	30.76 ± 3.49	<0.05
WHR	0.76 ± 0.03	0.73 ± 0.23	>0.05
T.CHOL (mg/dl)	162.92 ± 17.16	157.16 ± 14.67	<0.05
TGL (mg/dl)	125.12 ± 10.22	118.48 ± 6.23	<0.05
LDL (mg/dl)	116.8 ± 3.94	109.88 ± 4.54	<0.05
HDL (mg/dl)	50.24 ± 3.57	50.32 ± 2.88	>0.05

$p < 0.05$ is significant.

In group 1 ($n = 50$) there was a significant reduction in weight and BMI without any significant fall in WHR i.e. the body morphology remained the same (pear type body morphology). On the other hand in group 2 ($n = 42$) there was a significant fall in weight, BMI along with WHR suggesting an exercise induced alteration of body morphology from an apple to pear type body morphology. This change in body morphology in category 2 women was associated with significant fall in all forms of blood lipid levels except HDL (where the changes were minimal). On the other hand, in 8 females of experimental group with WHR between 0.7 and 0.8, there occurred changes in all parameters but in WHR, suggesting no affect on the body morphology.

From the above observations it is concluded that, obese females with Android type body morphology (WHR more than 0.8) undergo rapid and significant alteration in their body morphology, where as those with Gynoid type body morphology (WHR less than 0.8) though may have a significant reduction in weight and BMI but the WHR remains the same, i.e. the body morphology remains the same.

4. Discussion

Obesity is now the major health problem all over the world, to the extent that it is beginning to replace health hazards of undernutrition and infectious diseases. The mechanism of obesity though not properly understood but is supposed to be caused by multiple factors. Environmental factors, life style preferences, and cultural environment play pivotal roles in the rising prevalence of obesity worldwide.[15–18] In general, overweight and obesity are assumed to be the results of an increase in caloric and fat intake. Exercises have been widely perceived to be beneficial for weight loss in normal and other obese individuals with chronic diseases, but the role of physical exercises in altering body morphology in otherwise normal individuals is less understood and addressed. The present study defined the beneficial effects of exercise in female body morphology of a defined age group who were subjected to a set pattern of exercise schedule. The graded classification of overweight and obesity using BMI values, proposed by WHO committee in 1995 provided valuable information about increasing body fatness but failed to localize the area of fat deposition or define the pattern of body morphology of an overweight individual.[19,20] Hence, classification based on fat distribution and body morphology, suggested by Vague was taken into consideration for the present study.[4] As per this classification, females usually have fat deposition in gluteofemoral region giving them a *GYNOID or PEAR* shaped body morphology; whereas in males the deposition of fat is minimal in these regions and more around abdomen, giving them an *ANDROID or APPLE* shaped body morphology.[21,22] This fat distribution in humans is regulated by sex hormones as evidenced by the extensive work done by Björntorp and Rubuffe-Scrive.[23–25] Their work demonstrated the ways by which sex hormones affect specific regional adiposity and regulate utilization and accumulation of fat. According to their work, the estrogen inhibits fat deposition in the abdominal region and stimulates fat deposition in the gluteofemoral region more than in other body regions while testosterone stimulates fat deposition in the abdominal region and inhibits deposition in the gluteofemoral region. The recent development of new techniques for measurements of adipocyte morphology and metabolism has also made possible studies, that strongly suggest that human adipose tissue is not evenly distributed in the body instead there are sex dependent differences in deposition of adipocytes in different regions.[26] Apart from the role of hormones, the increasing tendency towards sedentary life-style and excessive intake of high-energy food are becoming major cause of abdominal obesity, and is considered an important factor for altering body type of females, as evident in the present study. The selection of subjects in the present study eliminates the hormonal role as a causative factor of obesity since all subjects included were females with normal hormone assay.

Body mass index (BMI) is widely used as a measure of overweight and obesity, but underestimates the prevalence of both these conditions, defined as an excess of body fat. Body mass index classification of obesity lacks subjects with increased cardiometabolic risk factors related to increased adiposity hence to ascertain the degree of fat distribution, the measurement of waist circumference and hip circumference are taken to calculate WHR.[27] The WHR is a stable and highly reliable measure and is significantly correlated with fat distribution measures using computed tomography scanning.[28,29] The 42 subjects in category 2 of our experimental group presented with a high WHR, suggesting physical inactivity as a causative factor for altering body morphology.

Abdominal obesity is also emerging as one of the important factor behind the increase in cardiometabolic risks in the general population. A study done by Jean-Pierre Despres on

Table 2 – Changing parameters in experimental group.

Group 2 ($n = 42$)			
Parameter	Pre-test	Post-test	p-value
Weight (kg)	80.92 ± 6.82	72.20 ± 5.91	<0.05
BMI	32.64 ± 2.90	30.36 ± 2.68	<0.05
WHR	0.85 ± 0.03	0.80 ± 0.03	<0.05
T.CHOL (mg/dl)	226.68 ± 11.06	196.04 ± 13.60	<0.05
TGL (mg/dl)	202.84 ± 12.93	130.28 ± 16.99	<0.05
LDL (mg/dl)	134.7 ± 7.46	111.28 ± 6.81	<0.05
HDL (mg/dl)	35.44 ± 2.48	51.20 ± 2.04	>0.05

$p < 0.05$ is significant.

the measurement of waist circumference readily identified the presence of increased cardiometabolic risks associated with abdominal obesity, on the other hand, the study of anthropometric measurements on patients with evidence of cardiovascular disease often display abdominal obesity.[30–32] Various other studies have also identified abdominal obesity as a predictor of adverse metabolic or cardiovascular outcomes independent of body mass index.[33] These studies are of the view that abdominal obesity (android type) is considered a more significant health hazard as compared to peripheral obesity (pear type). Thus, females who otherwise manifest peripheral obesity but acquire abdominal obesity, as an outcome of sedentary life style, will be more exposed to hazards of overweight and obesity. For many years, exercise has been considered as one of the keystone in managing obesity. The results of present study depict an obvious change in body morphology of female individuals associated with other beneficial outcomes of lowering of lipid levels. The measurements of WHR related significantly with the regional body fat distribution; a women with WHR of 0.7–0.8 had a Gynoid type body morphology whereas a women with WHR more than 0.8 had an Android type of body morphology. Also, independent of the general BMI levels the higher values of WHR were observed to be associated with adverse blood lipid levels.

5. Conclusion

From this study, we conclude that a guided physical exercise schedule for a period of 3 months cause decrease in WHR and reversal of body morphology from apple to pear shaped. This was seen to be associated with significant reduction in total cholesterol, TGL, LDL and an elevation in HDL levels. The physical activity alters body morphology, directly or indirectly by preventing overweight and abdominal obesity. In the recent times, there is growing awareness of gender specific health issues hence there is need to have a planned physical activity programme specifically for female gender. From this analysis, future research strategies and physical activity schedules may be planned and implemented to improve the health and well-being of women.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. Misra A, Khurana L. Obesity and the metabolic syndrome in developing countries. *J Clin Endocrinol Metab*. 2008;93(11 suppl 1):S9–S30.
2. Gupta R, Gupta VP, Sarna M, et al. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. *Indian Heart J*. 2002;54(1):59–66.
3. Gupta R, Misra A. Type 2 diabetes in India: regional disparities. *Br J Diabetes Vasc Dis*. 2007;7:12–16.
4. Vague J. *La Differentiacion Sexuelle Humaine Ses Incidences en Pathologie*. Paris: Masson Editeur; 1953:386–396.
5. Evans PJ, Hoffman RG, Kalkhoff RK, Kissebah AH. Relationship of androgenic activity to body fat topography, fat cell morphology and metabolic aberrations in premenopausal women. *J Clin Endocrinol Metab*. 1983;57:304–310.
6. Hartz AJ, Rupley DC, Rimm AA. The association of girth measurements with disease in 32,856 women. *Am J Epidemiol*. 1984;119:71–80.
7. Monteiro CA, Conde WL, Matsudo SM, Matsudo VR, Bonsenor IM, Lotufo PA. A descriptive epidemiology of leisure-time physical activity in Brazil, 1996–1997. *Rev Panam Salud Publica*. 2003;14:246–254.
8. Burton NW, Turrell G. Occupation, hours worked, and leisure-time physical activity. *Prev Med*. 2000;31:673–681.
9. Feunekes GI, van Staveren WA, De Vries JH, et al. Relative and biomarker-based validity of a food-frequency questionnaire estimating intake of fats and cholesterol. *Am J Clin Nutr*. 1993;58:489–496.
10. Feunekes IJ, van Staveren WA, Graveland F, De Vos J, Burema J. Reproducibility of a semiquantitative food frequency questionnaire to assess the intake of fats and cholesterol in The Netherlands. *Int J Food Sci Nutr*. 1995;46:117–123.
11. Keys A, Fidanza F, Karvonen MJ, et al. Indices of relative weight and obesity. *J Chronic Dis*. 1972;25:329–343.
12. Block WD, Jarrett Jr KJ, Levine JB. Use of a single color reagent to improve the automated determination of serum total cholesterol. In: Skeggs Jr LT, ed. *Automation in Analytical Chemistry*. New York: Mediad; 1966:345–347.
13. Kessler G, Lederer H. Fluorometric measurement of triglycerides. In: Skeggs Jr LT, ed. *Automation in Analytical Chemistry*. New York: Mediad; 1966:341–344.
14. *Manual of Laboratory Operation, Lipid Research Clinics Program*. vol. 1. Bethesda, MD: National Institutes of Health; 1974. Department of Health Education and Welfare Publication No. (NIH) 75-628.
15. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science*. 1998;280:1371–1374.
16. Goodrick GK, Poston WS, Foreyt JP. Methods for voluntary weight loss and control: update 1996. *Nutrition*. 1996;12:672–676.
17. Eckel RH, Krauss RM. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. AHA Nutrition Committee. *Circulation*. 1998;97:2099–2100.
18. Grundy SM. Multifactorial causation of obesity: implications for prevention. *Am J Clin Nutr*. 1998;67:563S–572S.
19. World Health Organization Expert Committee. *Physical Status: the Use and Interpretation of Anthropometry*. WHO Tech Rep, Ser No. 854. Geneva: World Health Organization; 1995.
20. World Health Organization. *Obesity: Prevention and Managing the Global Epidemic*. Geneva: World Health Organization; 1997.
21. Krotkiewski M, Blohme B, Lindholm N, Björntorp P. The effects of adrenal corticosteroids on regional adipocyte size in man. *J Clin Endocrinol Metab*. 1976;42:91–97.
22. Krotkiewski M, Björntorp P. The effects of estrogen treatment of carcinoma of the prostate on regional adipocyte size. *J Endocrinol Invest*. 1978;1:365–366.
23. Björntorp P. Fat cell distribution and metabolism. In: Wurtman RJ, Wurtman JJ, eds. *Human Obesity*. New York: New York Academy of Sciences; 1987:66–72.
24. Björntorp P. Adipose tissue distribution and function. *Int J Obes*. 1991;15:67–81.
25. Refubbe-Scrieve M. Metabolic differences in fat deposits. In: Bouchard C, Johnston FE, eds. *Fat Distribution During Growth and Later Health Outcomes*. New York: Alan R. Liss; 1988:163–173.

26. Krotkiewski M, Sjostrom L, Björntorp P, Smith U. Regional adipose tissue cellularity in relation to metabolism in young and middle-aged women. *Metab Clin Exp*. 1975;24:703–710.
27. Gomez-Ambrosi J, Silvia C, Galofré JC, et al. Body mass index classification misses subjects with increased cardiometabolic risk factors related to elevated adiposity. *Int J Obes (Lond)*. 2012 Feb;36(2):286–294.
28. Despres JP, Prudhomme D, Pouliot M, Tremblay A, Bushard C. Estimation of deep abdominal adipose tissue accumulation from simple anthropometric measurements in men. *Am J Clin Nutr*. 1991;54:471–477.
29. Poirier P, Lemieux I, Mauriege P, et al. Impact of waist circumference on the relationship between blood pressure and insulin: the Quebec Health Survey. *Hypertension*. 2005;45:363–367.
30. Despres Jean-Pierre. Abdominal obesity: the most prevalent cause of the metabolic syndrome and related cardiometabolic risk. *Eur Heart J Suppl*. 2006;8(suppl B):B4–B12.
31. Gorter PM, Olijhoek JK, van der Graaf Y, et al. Prevalence of the metabolic syndrome in patients with coronary heart disease, cerebrovascular disease, peripheral arterial disease or abdominal aortic aneurysm. *Atherosclerosis*. 2004;173:363–369.
32. Sonmez K, Akcakoyun M, Akcay A, et al. Which method should be used to determine the obesity, in patients with coronary artery disease? (body mass index, waist circumference or waist-hip ratio). *Int J Obes*. 2003;27:341–346.
33. Yusuf S, Hawken S, Ounpuu S, et al, INTERHEART Study Investigators. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: a case–control study. *Lancet*. 2005;366:1640–1649.