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SOMATOTYPE PROFILE OF OBESE AND LEAN WOMEN WITH POLYCYSTIC OVARY SYNDROME: A POPULATION BASED COHORT STUDY

ABSTRACT: Background: Polycystic ovary syndrome (PCOS) is emanating as one of the most commonly occurring endocrine as well as metabolic disorder among women during their reproductive span across the globe. Aim: The present cross-sectional study is an attempt to gauge somatotype profile of obese and lean women having polycystic ovary syndrome. Subjects and Methods: A sample of 150 PCOS women ranging in age from 18 to 35 years was collected from OPD of Department of Obstetrics and Gynaecology, PGIMER, Chandigarh. The subjects were diagnosed as PCOS as per the Rotterdam criteria. Results: The lean PCOS women were taller and significantly lighter than their obese PCOS counterparts. Somatotype profile of obese and lean PCOS women fall in mesomorphic-endomorph (5.7-4.7-0.4) and balanced endomorph (4.2-2.4-2.4) sectors of somatochart respectively, indicating a dominance of endomorphic and mesomorphic component, but less ectomorphic component in obese PCOS women as compared to lean PCOS women at all age groups. One way MANOVA analysis depicted a non-significant shift in component dominance (Wilk's lambda 0.94) among lean PCOS women, while obese PCOS exhibited a significant change in component dominance (Wilk's lambda 0.86). Conclusion: It was observed that endomorphic component was dominant in PCOS women irrespective of their BMI category.*

KEY WORDS: Lean PCOS – Obesity – Somatotype components – Somatochart

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a frequently occurring health problem among women during their reproductive period, with worldwide prevalence ranging from 8%–16% depending upon diagnostic criteria and population under study (Bodzag *et al.* 2016). The clinical presentation of PCOS is heterogeneous in nature and depends upon the presence or absence of characteristics features like hyperandrogenism, hyperinsulinemia, hypersecretion of LH, menstrual irregularity, hirsutism, sterility and

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related complications (Franks 1995, Moran, Teede 2009, Toulis *et al.* 2009). It is responsible for accelerated probability of morbidity in terms of both reproductive as well as non-reproductive events such as increased risk of cardio-metabolic, obstetric, oncology and psychological complication throughout the life span (Palomba *et al.* 2015). Aside from genetic factors (Carey *et al.* 1993, Simpson, 1992), numerous environmental factors including fat and carbohydrate intake, physical activity level, peripubertal stress and/or hormonal exposure, prenatal exposure to androgens are possible contributing factors in the pathogenesis of PCOS (Barry *et al.* 2010).

Obesity is a classic characteristics of polycystic ovary syndrome with approximately 70% of PCOS women showing higher body mass index (>25kg/m^2) (Carmina *et al.* 2006). Earlier studies (Crosignani *et al.* 2003, Hashimoto *et al.* 2003, March *et al.* 2010, Moran, Teede 2009, Penaforte *et al.* 2011, Snijder *et al.* 2004, Toscani *et al.* 2007) also noticed that even lean PCOS women had a tendency towards more android fat distribution. Obesity has been related to irregular function of the hypothalamic- pituitary-ovarian (HPO) axis, thereby leading to the development of PCOS.

Heath and Carter method of somatotyping is one of the most commonly used method to assess the human physique. It is expressed in three components endomorphy, mesomorphy and ectomorphy that empirically signifies different aspects of the body build i.e degree of fatness, musculoskeletal development and the linearity of the body respectively (Carter, Heath 1990). Over the years somatotyping has been used in describing variability of human population (Carter, Heath 1990, Dupertuis 1963), age changes in body morphology (Kalichman, Kobyliansky 2006, Kaur 2017, Singh, Sidhu 1980), growth and physical performance of athletes (Carter *et al.* 1982, Carter 1970). In addition, somatotypes have also been used to evaluate the relationship between body shape and the likelihood of disease occurrence. Previous researchers have recognized that people with higher endomorphic component have an increased risk of developing diabetes mellitus (Yeung *et al.* 2010), hypertension (Badenhorst *et al.* 2003, Herrera *et al.* 2004, William *et al.* 2000), metabolic syndrome (Galic *et al.* 2016, Martinez *et al.* 2012) and cancer (Bertrand 2013). A study performed by Harrison *et al.* (1976) highlighted that some association must exist between physique and endocrine functions as well as metabolism. The findings of Buffa *et al.* (2007) considered somatotyping as a diagnostic tool for monitoring and interpreting morphological changes in the diseases.

Till now only one study has been carried out on lean PCOS women from Vilnius and its surrounding districts from Lithuania to find out the relationship between body morphology and PCOS (Zabuliene *et al.* 2013). To best of our knowledge, this is the first study from India to explore the somatotypes profile of lean and obese women with PCOS. In view of paucity of literature on the physique of PCOS women the present study investigated the somatotype profile of obese and lean PCOS women from Chandigarh Capital region.

MATERIAL AND METHODS

The current cross-sectional study is based on a sample of 150 women ranging in age from 18 to 35 years, who were suffering from polycystic ovary syndrome. The data for the present investigation were gathered from OPD of Department of Obstetrics and Gynecology, Postgraduate Institute of Medical research and Education (PGIMER), Chandigarh. All the diagnosed patients of PCOS were from Chandigarh Capital region (North India). This area is also known as Greater Chandigarh, which incorporates the union territory city of Chandigarh, and its adjoining cities of Mohali, Zirakpur, Kharar, Kurali (in Punjab) and Panchkula (in Haryana).The economy of the greater Chandigarh is interdependent and is continuously inhibited, although these regions fall under different states of North India.

The prevalence of the PCOS was assumed to be 3.7% in North India (Gill *et al.* 2012). The sample size was assessed employing the following formula with 95% of confidence interval and 5% probability of type 1 error; $n = Z\alpha 2 \times p \times q/d2 = (1.96)2 \times 3.7 \times 0.96/(0.05)2$

Where, $Z\alpha=1.96$ for 95% level of confidence; α denotes the risk of type 1 error; p=assumed prevalence; $q = 1 - p$; and d signifies the error of estimate. The calculated sample size was 50 by using the aforementioned formula.

Polycystic ovary syndrome among patients were diagnosed following the protocol of the Rotterdam criteria (2003). According to this criteria, a total of two out of the following three symptoms were required for PCOS diagnosis: oligo-and/or anovulation (defined by the presence of oligomenorrhea or amenorrhea); clinical and/or biochemical signs of hyperandrogenism (defined by presence of hirsutism (Ferriman-Gallwey score ≥6), acne or alopecia, and/or elevated androgen

levels); and polycystic ovaries by gynecological ultrasound. Any subject with congenital adrenal hyperplasia, cushing's syndrome, androgen-secreting tumors, known hypothyroidism on treatment and on any medication related to endocrinal parameters were not included in the study. The menstrual history of the participants concerning their frequency and duration of menstrual cycle was also noted. Menstruation was considered impaired, if periods were less than 25 days or more than 35 days and less than 9 episodes in a year (Azziz *et al.* 2009).

Written consent was taken from the patients prior to the initiation of the data collection. Ethical approval was taken from institutional ethical committee of Panjab University, Chandigarh (PUIEC/2018/109/A/09/01) and Postgraduate Institute of Medical Research and Education (PGIMER), Chandigarh (INT/IEC/2018/000450). The PCOS women were divided into obese and lean groups on the basis of their body mass index (BMI) following the revised cut off values of BMI for Asians (WHO 2004). Lean group included underweight and normal weight women with BMI ≤24.99kg/m^2 and obese group embodied overweight and obese with BMI >25kg/m^2. According to WHO (2004) classification of BMI (kg/m^2) adult underweight (<18.50), normal (18.50–24.99), overweight (≥25.00) and obesity (≥30.00) categories were adapted. Out of the total sample 69.33% PCOS women were obese, while 30.66% were found to be lean (*Table 1*). From the date of birth age has been converted to decimal age following decimal age calendar of Tanner *et al.* (1966). All the participants from both categories were divided into three age groups with an interval of five years except for last age group, which is of seven years duration as depicted in *Table 1*.

Somatotypes of each PCOS women were determined employing Heath-Carter anthropometric somatotype method (Carter, Heath 1990). It is represented in three numeral rating i.e endomorphy, mesomorphy and ectomorphy respectively. The ten measurements, viz. height, weight, biepicondylar breadths of humerus and femur, mid upper arm and calf circumferences, and four skinfolds (triceps, subscapular, supraspinale and median calf) were measured as per the standard protocol given by Weiner, Lourie (1969). Height (cm) was taken nearest to 0.1cm with anthropometer and weight (kg) was measured in upright position to the nearest 0.1 kg with a weighing machine, humerus and femur breadth was gauged with sliding caliper and mid upper arm as well as calf circumferences were taken with Freeman's steel tape. All the four skinfolds measurements were taken with Holtain skinfold caliper (UK made).

TABLE 1: Distribution of obese and lean PCOS women according to their age group

Age group (in years)	Obese PCOS women N (%)	Lean PCOS women N (%)
18–22	25 (16.66%)	18 (12%)
23–27	50 (33.33%)	21(14%)
28–35	29 (19.33%)	7 (4.66%)
TOTAL	104 (69.33%)	46 (30.66)

Endomorphy, mesomorphy and ectomorphy were calculated with help of following equations (Carter, 1983): Endomorphy = - 0.7182 + 0.1451 (X) - 0.00068 (X 2) + 0.0000014 (X 3) where X = (sum of triceps, subscapular and supraspinale skinfolds) multiplied by (170.18/height in cm).

Mesomorphy= 0.858 × humerus breadth + 0.601 × femur breadth + 0.188 × corrected arm girth + 0.161 × corrected calf girth – height 0.131 + 4.5.

Three different equations are used to calculate ectomorphy according to the height-weight ratio: If HWR is greater than or equal to 40.75 then

Ectomorphy= 0.732 HWR - 28.58, If HWR is less than 40.75 but greater than 38.25 then

Ectomorphy= 0.463 HWR - 17.63, If HWR is equal to or less than 38.25 then ectomorphy is equal to 0.1.

The individual and mean somatotype were plotted on the two dimensional somatochart using X and Y coordinate on a superimposed grid system and somatotype altitudinal distance as well as somatotype altitudinal mean were computed as per Carter *et al.* (1983).

Statistical Analysis: Using statistical package for the social sciences (SPSS) version 19.0 (SPSS Inc.), baseline anthropometric and somatotype data were presented by descriptive statistics. Quantitative data were expressed as mean +SD, whereas qualitative data were presented as percentage. The student's t-test was used to determine the differences in various variables of obese and lean subjects. The level of significance for all analysis was set at $p < 0.05$ and a confidence interval of 95% was considered for all the tests. One way ANOVA analysis was used to evaluate difference in age groups in all the somatotype components of both the lean and obese PCOS women. One way MANOVA analysis was performed to gauge the age difference in

somatotype of obese and lean PCOS women with Wilk's lambda employed as test statistics.

RESULTS

Descriptive statistics of mean height, weight, somatotype components and SAM among obese and lean PCOS subjects by age is presented in *Table 2*. Lean PCOS women were taller (157.72 cm vs 156.02 cm, p >0.05) and significantly lighter (51.87 kg vs 70.30 kg p <0.01) than their obese PCOS counterparts. The obese PCOS women demonstrated higher mean values for endomorphic and mesomorphic components, but lower mean value for ectomorphic component as compared to lean PCOS women at all age levels and t-values exhibited statistically significant (p <0.01) differences for all the three components of somatotypes at all age levels in both the groups of present study. Endomorphic component of both the groups revealed an age related increment in their mean values, while ectomorphic component showed an inverse trend with advancing age.

A fluctuating trend was noted for mesomorphic components in both the lean and obese groups. Results of one way analysis of variance (ANOVA) showed non-significant age differences for height (F = 1.28), weight (F = 1.27), endomorphy (F = 0.91), mesomorphy (F = 0.46) and ectomorphy (F = 0.62) of lean PCOS women, while obese women demonstrated significant age differences for weight (F = 4.56*), endomorphy (F = 3.01*), mesomorphy (F = 6.52*) and ectomorphy (F = 3.71*) and non-significant for height (F = 0.76) as is evident from their F-values.

Similar trend was witnessed in the Whisker plots of lean and obese PCOS women as is portrayed in *Figures 1 and 2*. To ascertain the effect of age, one way MANOVA analysis has been carried out on the somatotype components. The results have depicted non-significant (Wilk's lambda 0.94) component dominance among lean PCOS women, while obese PCOS exhibited significant values for Wilk's lambda (0.86*) and confirming significant changes in component dominance.

The individual somatotypes of PCOS women as per their BMI categorizes were plotted on the two-dimensional somatochart (*Figures 3 and 4*). Majority of individual somatotypes of the obese PCOS women lie outside the western border of the endomorphic axis with their mean somatotype (5.7-4.7-0.4) fall in mesomorphic-endomorph sector. Most of the

TABLE 2: Mean and standard deviation of height, weight and somatotype components of obese and lean PCOS women. Level of significance p <.05*, p <.01 **, p <.001***

Age group (in years)	Categories	Height Mean ±S.D	Weight Mean ±S.D	Somatotype Mean ±S.D	SAM Mean ±S.D
18–22	Obese = 25	157.17 (±7.2)	72.14** (±13.8)	5.5-4.7-0.4 ** (0.9-1.4-0.4)	0.10 (±1.7)
	Lean = 18	158.65 (±4.8)	52.00 (±6.3)	4.0-2.2-2.6 (1.1-0.8-1.0)	0.19 (±1.6)
23–27	Obese = 50	155.99 (±5.4)	66.96** (±10.2)	5.6-4.3-0.4** (0.9-1.2-0.4)	0.05 (±1.6)
	Lean = 21	156.43 (±4.8)	50.89 (±4.3)	4.1-2.5-2.3 (0.8-0.7-0.9)	0.41 (±1.4)
28–35	Obese = 29	155.09 (± 6.2)	74.5** (±12.9)	6.0-5.4-0.2** (0.9-1.5-0.3)	0.12 (±2.0)
	Lean = 7	159.22 (±6.2)	54.51 (±4.2)	4.6-2.3-2.1 (0.7-1.2-0.8)	0.54 (±1.1)
Total	Obese = 104	156.02 (±6.1)	70.30** (±12.3)	5.7-4.7-0.4** (0.9-1.4-0.4)	0.02 (±1.8)
	Lean = 46	157.72 (±5.1)	51.87 (±5.2)	4.2-2.4-2.4 (0.9-0.8-0.9)	0.07 (±1.4)

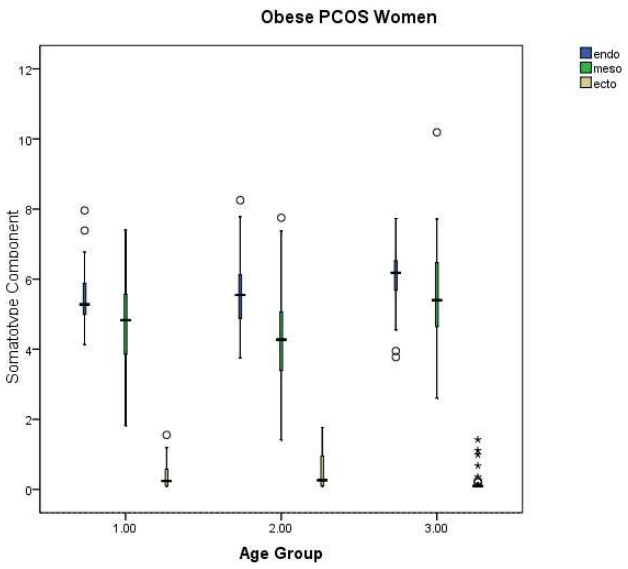

FIGURE 1: Whisker plot: Mean value and standard errors of somatotype components in obese women.

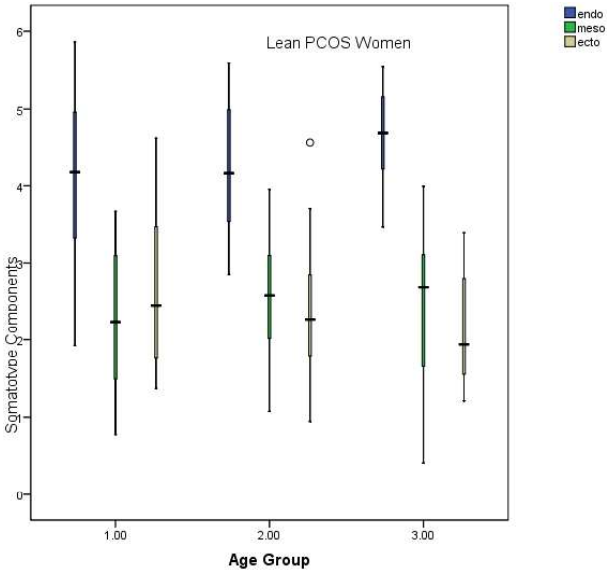

FIGURE 2: Whisker plot: Mean value and standard errors of somatotype components in lean women.

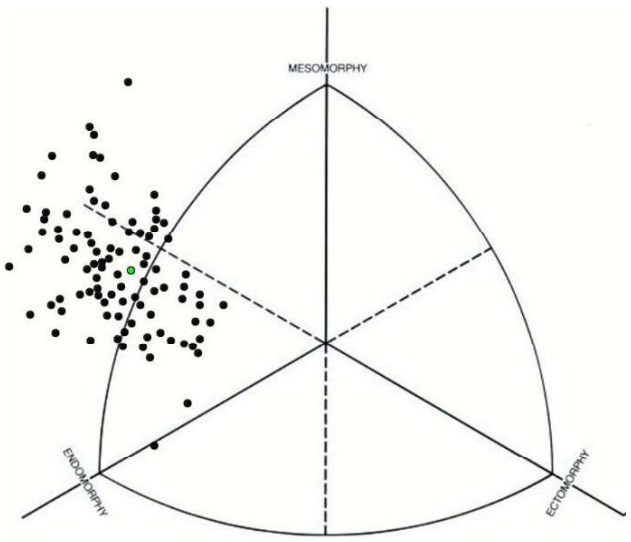

FIGURE 3: Somatotype distribution of individual and mean somatotype of obese PCOS women.

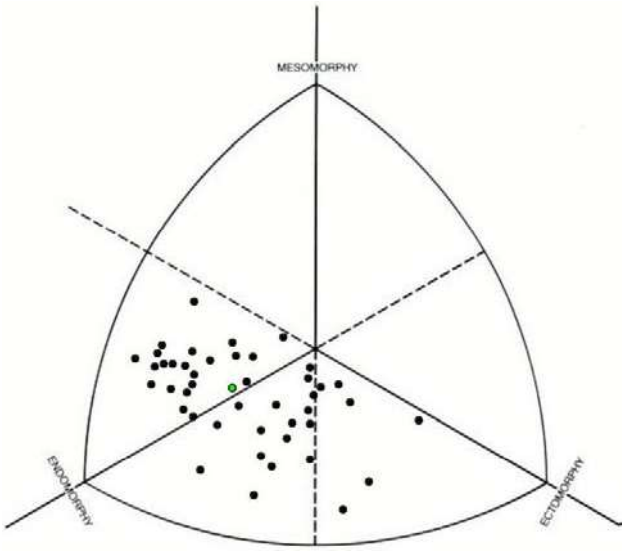

FIGURE 4: Somatotype distribution of individual and mean somatotype of lean PCOS women.

individual somatotypes of the lean PCOS women were scattered in the south west region of the endomorphy with their mean somatotype (4.2-2.4-2.4) fall in balanced endomorph sectors of somatochart.

Table 3 represents the distribution of individual somatotypes of obese and lean PCOS women in different categories of somatochart. Out of the total obese PCOS patients 63.46% were in mesomorphic-endomorph, 20.19% in mesomorph-endomorph and 14.42% in endomorphic-mesomorph sectors, while lean PCOS participants presented a considerable proportion of subjects (39.13%) in mesomorphic-

TABLE 3: Distribution of obese and lean PCOS women somatotypes in different categories of somatochart.

Categories	Obese PCOS N (%)	Lean PCOS N (%)
Endomorph-ectomorph	0	5 (10.86%)
Ectomorphic-endomorph	0	7 (15.22%)
Balanced endomorph	1(0.96%)	3 (6.52%)
Mesomorphic-endomorph	66 (63.46%)	18 (39.13)
Mesomorph-endomorph	21 (20.19%)	3 (6.52%)
Endomorphic-mesomorph	15 (14.42%)	1 (2.17%)
Mesomorph-ectomorph	1 (0.96%)	2 (4.34%)
Endomorphic-ectomorph	0	5 (10.86%)
Central	0	2 (4.34%)

endomorph category and 15.22% in ectomorphic-endomorph and low frequency of participants were noted in other categories i.e mesomorph-ectomorph (4.34%),balanced endomorph (6.52%), endomorphic-ectomorph (10.86%) and central (4.34%).

DISCUSSION

PCOS is believed to be the result of endocrine as well as metabolic disorder that may leads to diverse health issues. There is a meager information in the literature about the physique of PCOS women. In the present cross-sectional study prevalence of obese PCOS was 69.33%, which was substantially higher than their lean PCOS (30.66%) counterparts. Similarly, a report of Carmina *et al.* (2006) also identified obesity as a classic characteristics of polycystic ovary syndrome with approximately 70% of PCOS women showing higher body mass index (>25kg/m^2). The prevalence of obesity among PCOS women was noted to be 74% in Birmingham females (Yildiz *et al.* 2008). A study of Mahdi *et al.* (2018) also noticed that about 40.7% and 24.7% PCOS women were over-weight and obese respectively.Most of the earlier studies (Carmina *et al.* 2007, Doh *et al.* 2016, Huber-Buchholz *et al.* 1999, Majumdar, Singh 2009) carried out on the clinical and metabolic profiles of obese and lean PCOS women illustrated that the effect of obesity and upper body fat localization may contribute to ovarian dysfunction. A study performed by Kirchengast, Huber (2001) revealed that greater amount of body fat and lesser lean mass in lean PCOS in contrast to controls and have intermediate or android fat distribution. They also assumed that obesity further worsened the hyperandrogenic and metabolic conditions and ovulation performance.

Our study results presented that lean PCOS women were taller (157.72 cm vs 156.02 cm, $p > 0.05$) and significantly lighter (51.87 kg vs 70.30 kg, $p < 0.01$) than their obese PCOS counterparts. The obese PCOS women were more endomorphic and mesomorphic, but less ectomorphic than their lean PCOS counterparts at all age groups and t-values demonstrated statistically significant ($p<0.01$) differences for all the three components of somatotypes. The mean somatotype of obese PCOS women was 5.7-4.7-0.4 (SD 0.9-1.4-0.4), whereas for lean PCOS mean somatotype was 4.2-2.4-2.4 (SD 0.9-0.8-0.9) and the women in both the categories classified as mesomorphic-endomorph and balanced endomorph respectively. The only previous study performed on lean PCOS women by Zabuliene *et al.* (2013) also showed that mean PCOS somatotype was 4.96-4.38-3.00 (SD 1.50-1.26-1.11) and was recorded in mesomorphic endomorphs category. Majority of obese (63.49%) and lean PCOS women (39.13%) fall in the mesomorphic endomorph sector, which is consistent with the study of Zabuliene *et al.* (2013) exhibiting lean PCOS in the same category.

Our study showed convergence with the findings of Zabuliene *et al.* (2013) that endomorphic component was dominant in PCOS women irrespective of their BMI category. The endomorphic component increases significantly ($p < 0.01$) in both obese as well as lean PCOS women with advancing age. According to the Kalichman *et al.* (2006) endomorphy component in females was consistently increased up to 6th decades and then decreased with age and largest differences of all somatotype appeared between 18–30 and 31–40 years.

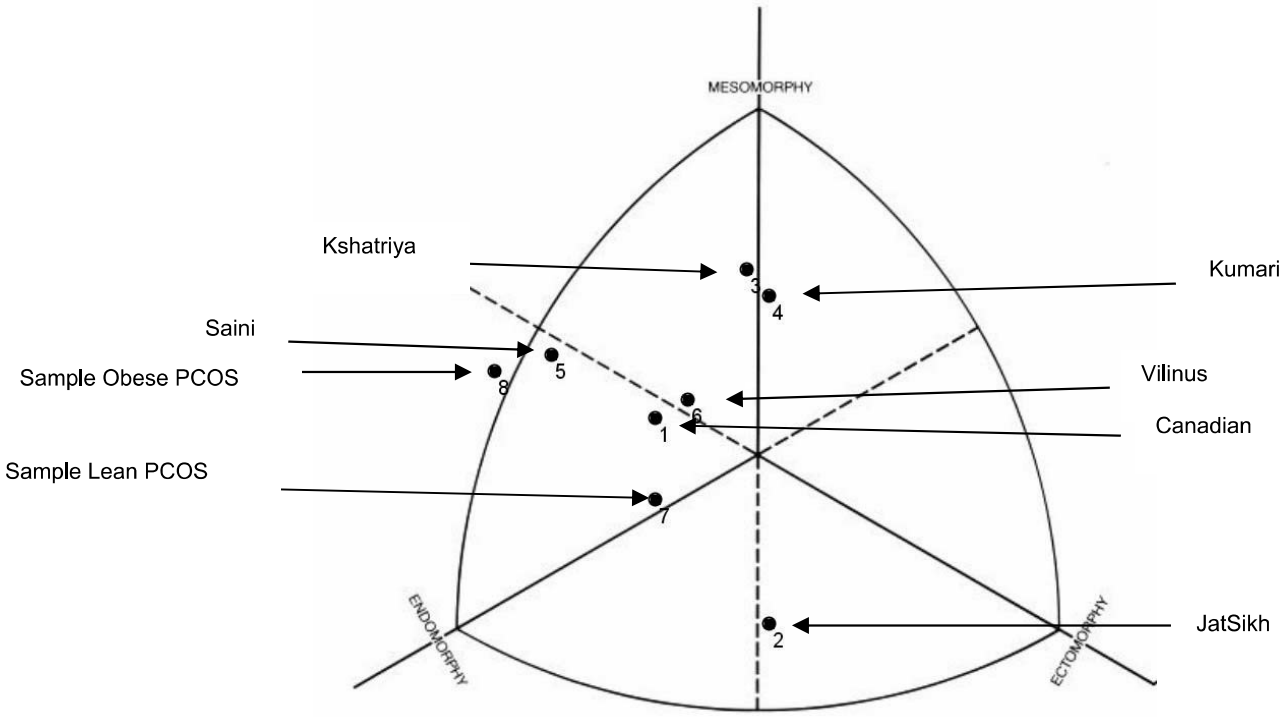

FIGURE 5: Comparison of mean somatotype of obese and lean PCOS women with preexisting data. 1, Canadian; 2, Jat Sikh; 3, Kshatriya; 4, Kumari; 5, Saini; 6, Vilinus; 7, Sample Lean PCOS women; 8, Sample Obese PCOS women.

The mean somatotypes of obese and lean PCOS women of present study have been compared with pre-existing data on the somatotypes of healthy and normal sample of Canadian females (5.5-4.6-1.8; Bailey *et al.*1982), Jat Sikh females (4.1-2.3-3.6; Sidhu *et al.* 1982), Kshatriya females (2.5-4.7-2.1) and Kumari females (2.3-4.7-2.9; Chandel, Malik 2012), Saini females (5.7-5.7-1.8; Kaur, Malik 2016) and Vilinus females (4.2-4.6-2.9; Zabuliene *et al.* 2013). The comparative account summarized that the somatotype of present obese sample was classified as mesomorphic endomorph, while Jat Sikh females also lie in mesomorphic endomorph sector, but the obese PCOS women of present study fall in the outer boundary of western border of endomorphic axis of the somatochart. Somatotypes of Vilinus females fall in mesomorph endomorph, while Kshatriya females and Kumari females were found in balanced mesomorph and Saini females lie in endomorph mesomorph sectors of somatochart.

It is evident from the comparison that the obese PCOS women exhibited maximum development of endomorphic component and least ectomorphic component than all the pre-existing samples. Even the lean PCOS women demonstrated higher endomorphy than Jat Sikh females, Kshatriya females, Kumari females and comparable to Vilinus females. This may be attributed to tendency of PCOS patients towards more fat deposition. Finding of Crosignani (2003) also supported this trend and indicated that endomorphy signifies the fatness and increased obesity is one of the most reliable predictors of PCOS women and it also escalates severity of manifestation.

CONCLUSION

It is evident from the above discussion that somatotype profile of obese (5.7-4.7-0.4) and lean (4.2-2.4-2.4) PCOS women was grouped in mesomorphic-endomorph sector and balanced endomorph sector respectively, thus indicating a dominance of endomorphic component and least ectomorphic component irrespective of their body mass index category. Hence, the findings of present study highlighted somatotypes as an efficient marker of body morphology of PCOS women.

PCOS is one of the emerging lifestyle diseases throughout the world with multiple health implications

particularly targeting women in their reproductive age. Present study will be helpful in filling the gap in the understanding of this escalating health problem affecting reproductive, metabolic and psychological characteristics, since the literature in this domain is still scanty. Hence, it is pertinent to generate awareness among females to bring behavioural management / lifestyle modification as well as dietary changes to tackle this silent but escalating health concern.

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