

Body composition of lean women with polycystic ovary syndrome

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ABSTRACT: Polycystic ovary syndrome (PCOS) is one of the most frequent endocrine and metabolic disorders in reproductive age women, and it is related to changes in body size, shape and composition. Anthropometric somatotype is a quantitative description of the individual's body shape and composition classified as endomorphy, mesomorphy or ectomorphy. Since PCOS somatotype has never previously been studied, here we evaluate body shape and composition phenomena in lean women with polycystic ovary syndrome and assess relationships with metabolic parameters. The study of 20–35 year-old women was carried out at the Department of Anatomy, Histology and Anthropology at Vilnius University. Standard anthropometric instruments and methods were used, and J. Matiegka's equations calculated skeletal mass, skin and subcutaneous adipose tissue and muscles and internal organs. In addition, Heath – Carter's somatotypes were computed, and the participants' glucose, insulin, testosterone, sex hormone-binding globulin and lipid levels were established. We analysed data from 120 women with a mean age of 27.30 ± 3.68 years. Lean women with PCOS had greater skeletal mass by 0.47 kg ($p < 0.05$, Cohen's $d = 1.14$), greater skin and subcutaneous adipose tissue mass by 2.79 kg ($p < 0.05$, Cohen's $d = 6.07$) and lower muscle mass by 1.47 kg ($p < 0.05$, Cohen's $d = 2.84$) compared to control women ($p < 0.05$). The mean PCOS somatotype ratio was 4.96–4.38–3.00 (SD 1.50–1.26–1.11). This classified women with PCOS as mesomorphic endomorphs, in contrast to healthy women who were endomorphic mesomorphs. The PCOS subjects' skin and subcutaneous adipose tissue and endomorphy/mesomorphy somatotype positively correlated with insulin levels and the HOMA-IR. It was established that lean women with polycystic ovary syndrome had a mesomorphic endomorph somatotype and higher skin and subcutaneous adipose tissue mass, but less muscle mass than healthy lean women. In addition, skin and subcutaneous adipose tissue positively correlated with insulin level and HOMA-IR in lean PCOS women.

KEY WORDS: PCOS, somatotype, adipose tissue

Introduction

Polycystic ovary syndrome (PCOS) is one of the most frequent endocrine and metabolic disorders in reproductive age women with a frequency of 4 to 17.8% in over 100 million women globally (Padmanabhan 2009; March et al. 2010). PCOS is a heterogeneous clinical syndrome characterised by the presence of hyperandrogenism and/or excess androgen in the blood, anovulation, menstrual cycle disturbance and polycystic ovary morphology. PCOS determined by complex pathogenic mechanisms is also related to bone mineral density, body size, and shape and compositional changes (Azziz et al. 2009; Allahbadia and Merchant 2011).

Current anthropometric indices of adipose tissue amount and accumulation site in PCOS women have revealed there is a greater adipose tissue mass in the internal organs, waist and arms of PCOS women than in control subjects. A tendency to android fat distribution was observed even in lean PCOS women (Gennarelli et al. 2000; Kirchengast and Huber 2001; Crosignani et al. 2003; Hashimoto et al. 2003; Snijder et al. 2004; Toscani et al. 2007; Moran and Teede 2009; March et al. 2010 and Penaforte et al. 2011). However, studies analysing skeletal, muscle and internal organ masses in PCOS women is currently lacking.

Anthropometric somatotype is a quantitative description of individual body shape and composition related to height: endomorphy, mesomorphy and ectomorphy. Endomorphy expresses relative body fat, mesomorphy captures muscularity and ectomorphy refers to the body's leanness. The commonly used differentiation is Heath-Carter's anthro-

pometric somatotype (Carter and Heath 1990). Somatotype is associated with physical fitness level, a variety of health risk factors and chronic degenerative pathologies (Williams SR et al. 2000; Saitoglu et al. 2007), and it is extremely important in public health studies because of its relationship with cardiovascular risk factors (Malina et al. 1997). PCOS Somatotype has never previously been studied.

The aim of this study is to evaluate body shape and composition and assess their relationship with metabolic parameters in lean women with polycystic ovary syndrome.

Materials and Methods

Study population

This cross-sectional study was conducted in 2007–2011 at the Department of Anatomy, Histology and Anthropology (Faculty of Medicine of Vilnius University) in cooperation with Outpatient Clinics in Vilnius. The study population was consisted of 20–35 year-old women aged 20–35 residing in Vilnius and its surrounding districts. Written informed consent was obtained from patients prior to the study which was approved by the Lithuanian Bioethics Committee.

All women referred to the outpatient clinics for suspected PCOS because of hirsutism, menstrual cycle disturbance or infertility were offered a place in the study. Inclusion criteria for the study group were: (1) participation consent; (2) women aged 20–35; (3) diagnosis of PCOS determined according to Rotterdam criteria in: (a) clinical and/or biochemical hyperandrogenism, (b) oligoovulation or anovulation and (c) poly-

cystic ovaries defined by ultrasound, after exclusion of adrenal androgen excess, androgen secreting tumours, hyper-prolactinaemia and thyroid dysfunction (The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group 2004; Azziz et al. 2009). The control group involved healthy women who agreed to take part in the study and their inclusion criteria were: (1) participation consent; (2) women aged 20–35; (3) no evidence of clinical or biochemical androgen excess of any origin; (4) normal regular menstrual cycles, where the presence of normal ovulation was assessed by serum progesterone level on cycle days 21–25 and (5) no first-degree family history of PCOS. Women with type 2 diabetes mellitus, presence of chronic renal or hepatic diseases, taking contraceptive pills were not enrolled in the study.

In total, 235 women (135 women with PCOS and a 100 control women without hyperandrogenism or menstrual cycle disturbances) were studied. The data of 38 women were excluded from further analysis: 15 women withdrew consent and 18 women were excluded due to abnormal laboratory tests revealing different other diseases. 197 women composed the final study sample: 116 PCOS women and 81 controls.

Clinical hyperandrogenism was defined as the presence of hirsutism in nine body areas by a modified Ferriman-Gallwey score cut-off point ≥ 6 . Biochemical hyperandrogenism was defined as testosterone serum level ≥ 1.68 nmol/l, dehydroepiandrosterone sulphate (DHEAS) ≥ 10.42 μ mol/l and free androgen index (FAI) ≥ 2.94 (Zabulienė et al 2012). The menstrual cycle was considered impaired when meters; (1) it was shorter than 25 days or longer than 35 days, (2) there were fewer than 9 bleeding episodes per

year and (3) there were at least two consecutive cycles where serum progesterone level on days 21–25 was lower than 10 nmol/l (Azziz et al. 2009).

Anthropometrics

Standard anthropometric instruments from Siber Hegner and standard methods were employed (Martin and Saller 1959; Knussmann et al. 1988; Anthropometrica 2002; Tutkuvienė and Jakimaviciene 2004 and Jakimaviciene and Tutkuvienė 2004).

The following 16 anthropometric measurements were performed: body mass, height, 4 transverse skeletal measurements (widths of elbow, wrist, knee and ankle), 4 body circumferences (circumference of upper arm, forearm, wrist, thigh and calf) and 6 skinfolds (subscapular, triceps, biceps, suprailiac, thigh and calf skinfolds). Weight to the nearest 0.1 kg and height to the nearest 0.1 cm were measured in participants wearing light clothing, without shoes, after voiding and following 10 to 12 hours overnight fast. All participants were weighed on the same Gamma scales (Soehnle, Germany) and the identical spreading and sliding callipers were used for their widths. Circumferences were measured with plastic tape and skinfolds with the Holtain calliper. All measurements were performed thrice by one investigator and mean values were analysed.

Body mass index (BMI) was calculated as the ratio of weight in kilograms to height squared in meters. The estimated mass of skeleton, skin and subcutaneous adipose tissue, muscles and the internal organs and remainders were calculated according to J. Matiegka's equations, while the relative and absolute passive mass and absolute active mass were ob-

tained using J.V.G.A. Durnin et J. Womersley's equations (Matiegka 1921 cited in *Anthropometrica* 2002; Durnin et Womersley 1974 cited in *Anthropometrica* 2002).

Heath-Carter's somatotypes were computed according to the following equations (Carter JEL 2002); where skinfolds are in mm, widths in mm and height and circumferences in cm:

Endomorphy = $-0.7182 + 0.1451 (X) - 0.00068 (X^2) + 0.000014 (X^3)$, where $X = (\text{triceps skinfold} + \text{subscapular skinfold} + \text{suprailiac skinfold}) \times (170.18/\text{height})$.

Mesomorphy = $0.858 \times \text{elbows width} + 0.601 \times \text{knee width} + 0.188 \times \text{corrected upper arm circumference} + 0.161 \times \text{corrected calf circumference} - \text{height} \times 0.131 + 4.5$.

Three different equations were used to calculate ectomorphy according to the height to weight ratio (HWR), calculated by dividing height by the cubed root of the weight:

- HWR greater than or equal to 40.75 defined *ectomorphy* = $0.732 \text{ HWR} - 28.58$;
- HWR less than 40.75 but greater than 38.25 gave *ectomorphy* = $0.463 \text{ HWR} - 17.63$; HWR equal to or less than 38.25 defined *ectomorphy* = 0.1 .

Laboratory tests

Laboratory tests were performed under standard laboratory procedure in the *Medicina Practica* laboratory in Vilnius, Lithuania. Venous blood samples were taken thrice between 7 and 9 a.m from each study participant following 10 to 12 hours overnight fasting. These were, (1) during the follicular phase at days 3–6 of spontaneous or progestin-induced bleeding in anovulatory patients, (2) in the middle of the menstrual cycle and (3) on days 21–25. Participants were tested

for fasting glucose, fasting insulin, total testosterone, sex hormone-binding globulin (SHBG), dehydroepiandrosterone sulphate (DHEAS), progesterone, total cholesterol, high density lipoprotein (HDL) cholesterol, low density lipoprotein (LDL) cholesterol and triglycerides (TG).

Fasting plasma glucose was measured by the glucose oxidase technique (Roche Diagnostics GmbH, Cobas Integra 400 plus, Mannheim, Germany). Plasma total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides levels were determined by the enzymatic colorimetric method (Roche Diagnostics GmbH, Cobas Integra 400 plus, Mannheim, Germany). Insulin was measured by chemiluminiscent immunoassay kits (Abbott Laboratories, Architect 8200, Abbott Park, IL, USA). Total testosterone, SHBG, DHEAS and progesterone were measured by luminescence immunoassay (ECLIA) (Roche Diagnostics GmbH, Elecsys 2010; Mannheim, Germany). Intra-assay and inter-assay coefficients of variation for the tests were less than 5%.

Free androgen index (FAI) was defined according to A. Vermeulen et al.: $\text{FAI} = (\text{total testosterone (nmol/l)} \times 100) / \text{sex hormone-binding globulin (SHBG) (nmol/l)}$ (Vermeulen et al. 1999). The glucose tolerance test was carried out according to the methodology suggested by the World Health Organization (WHO) in 2006, with results assessed following WHO and American Diabetes Association (ADA) guidelines (World Health Organization 2006; ADA 2011; Bartoli et al. 2011). The following indices were calculated for insulin resistance evaluation: The HOMA-IR index from the homeostasis model of assessment-insulin resistance formula $\text{HOMA-IR} = (\text{insulin } (\mu\text{IU/$

ml)) \times (fasting plasma glucose (mmol/l)) / 22.5) (Matthews et al. 1985) and Quantitative Insulin Sensitivity Check Index $QUICKI = 1/(\log(\text{insulin concentration } (\mu\text{IU/ml})) + \log(\text{plasma fasting glucose (mmol/l)/0,0555}))$ (Katz et al. 2000). Insulin resistance was determined when HOMA-IR was higher than 2.5.

Statistical analysis

Statistical analysis was performed by the SPSS version 20 for Windows (SPSS Inc., Chicago, IL) and the Student's t-test for independent samples was used to compare means. Cohen's d was calculated to evaluate the effect size and Pearson's correlation coefficient (r) was calculated to establish the relationship between continuous variables. Correlation was

ranked as very weak when r was lower than 0.2, weak when r was from 0.2 to 0.39, moderate when r ranged from 0.4 to 0.69, strong when r was from 0.7 to 0.79 and very strong when r exceeded 0.8. A p value of < 0.05 was considered significant.

Somatotype plotting and analysis was performed using Heath-Carter methods with the special Somatotype – Calculation and Analysis programme (Sweat Technologies, M E R Goulding Software Development).

Results

We analysed data from 120 lean women with a mean age of 27.30 ± 3.68 years. The youngest participant was 20 and the oldest was 34. The samples consisted of

Table 1. Clinical, hormonal and biochemical data of studied women (number of patients, mean \pm SD)

Variable	PCOS n=50	Control n=70	p
Age, years	26.54 \pm 3.65	27.84 \pm 3.63	0.055
Height, cm	168.31 \pm 5.10	166.49 \pm 7.06	0.104
Weight, kg	59.75 \pm 6.13	58.41 \pm 6.51	0.258
BMI, kg/m ²	21.10 \pm 2.02	21.05 \pm 1.65	0.876
Testosterone, nmol/l	1.91 \pm 0.75	0.99 \pm 0.38	<0.0001
SHBG, nmol/l	62.34 \pm 30.17	75.84 \pm 22.15	0.009
FAI	4.07 \pm 2.85	1.42 \pm 0.71	<0.0001
Total cholesterol, mmol/l	4.78 \pm 0.89	4.70 \pm 0.73	0.591
HDL cholesterol, mmol/l	1.72 \pm 0.31	1.85 \pm 0.30	0.021
LDL cholesterol, mmol/l	2.67 \pm 0.79	2.51 \pm 0.70	0.220
TG, mmol/l	0.88 \pm 0.40	0.75 \pm 0.33	0.047
Glucose, mmol/l	4.94 \pm 0.41	4.75 \pm 0.48	0.020
Insulin, $\mu\text{U/ml}$	7.35 \pm 4.34	6.20 \pm 2.85	0.082
HOMA-IR	1.64 \pm 1.05	1.32 \pm 0.65	0.056
QUICKI	0.36 \pm 0.03	0.37 \pm 0.03	0.052

SD – standard deviation, PCOS – women with polycystic ovary syndrome BMI – body mass index, SHBG – sex hormone – binding globulin, FAI – free androgen index, HDL – high density lipoprotein, LDL – low density lipoprotein, TG- triglycerides, HOMA-IR – homeostasis model of assessment-insulin resistance, QUICKI – Quantitative Insulin Sensitivity Check Index.

50 lean women with PCOS (according to Rotterdam criteria) and 70 healthy lean control subjects. No significant lean differences existed between the PCOS and control women's average age, height, weight and BMI (Table 1).

The results determined that androgen levels differed significantly between PCOS women and controls: SHBG were lower in women with PCOS, while testosterone and FAI were greater in PCOS women than controls ($p < 0.001$) (Table 1). There was no difference in total cholesterol and LDL cholesterol levels between these groups ($p > 0.05$). The HDL cholesterol in PCOS women was statistically significantly lower by 0.13 mmol/l and TG higher by 0.13 mmol/l than controls ($p < 0.05$). Fasting glucose was statistically significantly higher in PCOS women than controls ($p < 0.05$), and finally no differences were determined in insulin, HOMA-IR and QUICKI indices between the PCOS and control women ($p > 0.05$).

Body composition parameters, comprising estimated skeletal mass, skin and subcutaneous adipose tissue mass, muscle mass and relative and absolute

passive mass in PCOS women, differed to control values ($p < 0.05$) (Table 2). Lean PCOS women had slightly greater mean skeletal mass by 0.47 kg ($p < 0.05$, Cohen's $d = 1.14$), greater mean skin and subcutaneous adipose tissue mass by 2.79 kg ($p < 0.05$, Cohen's $d = 6.07$) and lower mean muscle mass by 1.47 kg ($p < 0.05$, Cohen's $d = 2.84$) compared to controls ($p < 0.05$). Although the relative and absolute passive mass derived from Durnin and Womersley's equations was higher in PCOS women than in controls ($p < 0.05$, Cohen's $d = 4.68$ and 4.08 accordingly), estimated viscera and remainder mass and the absolute active mass estimated in this manner did not differ in PCOS and control women.

While the mean PCOS somatotype was 4.96-4.38-3.00 (SD 1.50-1.26-1.11), thus characterizing women with PCOS as mesomorphic endomorphs, the mean control women's somatotype was 4.17-4.59-2.89 (SD 1.17-1.18-0.95). Healthy women presented as endomorphic mesomorphs. The mean somatotypes for each group were compared and found to be significantly different ($p = 0.031$) due to endomorphic difference ($p = 0.002$). The

Table 2. Body composition of lean PCOS and control women

Variable	PCOS n=50	Control n=70	p
Mass estimated by J. Matiegka's equations			
Skeletal mass	9.92 ± 1.07	9.45 ± 1.21	0.028
Skin and subcutaneous adipose tissue	22.12 ± 6.40	19.33 ± 5.7	0.014
Muscles mass	22.30 ± 2.89	23.77 ± 2.80	0.006
Viscera and remainders	12.31 ± 1.26	12.03 ± 1.34	0.258
Mass estimated by Durnin and Womersley's equations			
Relative passive mass	31.10 ± 4.95	27.42 ± 4.39	<0.0001
Absolute passive mass	18.74 ± 4.35	16.15 ± 3.79	0.001
Absolute active mass	41.01 ± 3.57	42.26 ± 4.11	0.087

SD – standard deviation, PCOS – women with polycystic ovary syndrome.

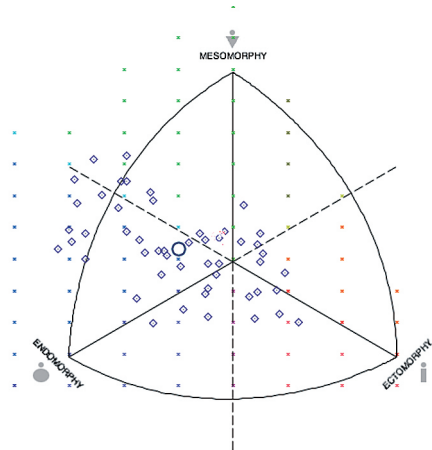


Fig. 1. Somatotype of lean PCOS subjects
O – mean somatotype: 4.96–4.38–3.00

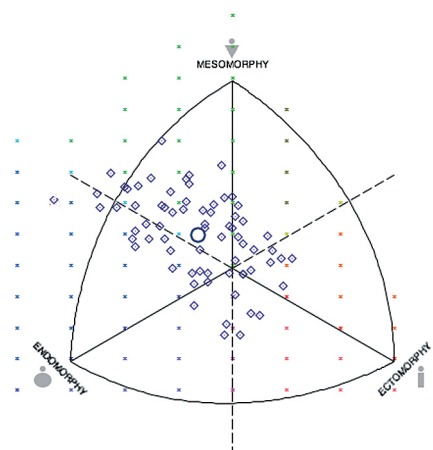


Fig. 2. Somatotype of healthy women
O – mean somatotype: 4.17–4.59–2.89

individual PCOS and controls somatotypes are presented graphically in Figure 1 and 2 somatoplots.

Distribution of PCOS and healthy women’s somatotypes are presented in Table 3. Prevailing PCOS somatotypes were

mesomorphic endomorph (31%), mesomorph-endomorph (17%) and central (13%), while the dominant proportions of the control group were classified as endomorphic-mesomorph (29%) and balanced mesomorph and central (14% both).

Table 3. Distribution of PCOS and healthy women’s somatotypes

Somatotype categories	PCOS n=50 n (%)	Control n=70 n (%)	p
Endomorph-ectomorph	0 (0)	3 (4%)	0.138
Ectomorphic endomorph	2 (4%)	0	0.092
Balanced endomorph	3 (6%)	3 (4%)	0.671
Mesomorphic endomorph	15 (31%)	8 (11%)	0.011
Mesomorph-endomorph	8 (17%)	8 (11%)	0.468
Endomorphic mesomorph	4 (8%)	20 (29%)	0.006
Balanced mesomorph	2 (4%)	10 (14%)	0.064
Ectomorphic mesomorph	0	0	
Mesomorph-ectomorph	2 (4%)	3 (4%)	0.939
Mesomorphic ectomorph	1 (2%)	2 (3%)	0.767
Balanced ectomorph	5 (10%)	3 (4%)	0.216
Endomorphic ectomorph	0	0	
Central	6 (13%)	10 (14%)	0.717

PCOS – women with polycystic ovary syndrome.

Table 4. Correlations between body composition, glucose and insulin resistance parameters

Variable	Glucose	Insulin	HOMA-IR	QUICKI
PCOS women n=50				
Mass estimated by J. Matiegka's equations				
Skeletal mass	0.14	0.21	0.22	-0.14
Skin and subcutaneous adipose tissue	0.16	0.52**	0.51**	-0.49**
Muscles mass	0.20	0.23	0.23	-0.23
Viscera and remainders	0.25	0.48**	0.49**	-0.43**
Mass estimated by Durnin and Womersley's equations				
Relative passive mass	0.15	0.43**	0.44**	-0.42**
Absolute passive mass	0.23	0.53**	0.53**	-0.49**
Absolute active mass	0.16	0.18	0.19	-0.14
Somatotype components				
Endomorphy	0.13	0.45**	0.45**	-0.45**
Mesomorphy	0.29*	0.41**	0.41**	-0.42**
Ectomorphy	-0.32*	-0.47**	-0.48**	0.44**
Controls n=70				
Mass estimated by J. Matiegka's equations				
Skeletal mass	0.03	0.11	0.11	-0.10
Skin and subcutaneous adipose tissue	0.04	0.21	0.21	-0.22
Muscles mass	-0.00	0.05	0.05	-0.03
Viscera and remainders	0.06	0.10	0.10	-0.11
Mass estimated by Durnin and Womersley's equations				
Relative passive mass	-0.06	0.17	0.15	-0.17
Absolute passive mass	-0.01	0.15	0.15	-0.16
Absolute active mass	0.11	0.01	0.03	-0.02
Somatotype components				
Endomorphy	-0.08	0.15	0.12	-0.15
Mesomorphy	0.04	0.07	0.08	-0.07
Ectomorphy	-0.02	0.03	0.02	-0.03

PCOS – women with polycystic ovary syndrome, HOMA-IR – homeostasis model of assessment-insulin resistance, QUICKI – Quantitative Insulin Sensitivity Check Index; * $p < 0.01$; ** $p < 0.001$.

The PCOS body composition parameters, comprising skin and subcutaneous adipose tissue, viscera and reminders and passive mass and also the endomorphic/mesomorphic somatotypes, positively correlated with insulin and HOMA-IR (Table

4). Although, ectomorphy was negatively correlated with glucose, insulin and HOMA-IR, in complete contrast, no control group correlations were established for body composition parameters and glucose and insulin resistance parameters.

Table 5. Correlations between body composition and lipids metabolism parameters

Variable	Total cholesterol	HDL cholesterol	LDL cholesterol	TG
PCOS women n=50				
Mass estimated by J. Matiegka's equations				
Skeletal mass	0.03	-0.11	0.03	0.60
Skin and subcutaneous adipose tissue	0.06	-0.15	0.06	0.31*
Muscles mass	-0.11	-0.12	-0.10	0.02
Viscera and remainders	-0.04	-0.20	-0.02	0.18
Mass estimated by Durnin and Womersley's equations				
Relative passive mass	0.19	-0.11	0.18	0.36**
Absolute passive mass	0.10	-0.18	0.10	0.33*
Absolute active mass	-0.18	-0.13	-0.17	-0.10
Somatotype components				
Endomorphy	0.18	-0.11	0.17	0.38**
Mesomorphy	0.10	-0.19	0.15	0.19
Ectomorphy	-0.07	0.24	-0.11	-0.28
Controls n=70				
Mass estimated by J. Matiegka's equations				
Skeletal mass	0.15	-0.16	0.24*	0.05
Skin and subcutaneous adipose tissue	0.29*	-0.14	0.32**	0.19
Muscles mass	0.03	-0.10	0.10	-0.10
Viscera and remainders	0.21	-0.15	0.27*	0.09
Mass estimated by Durnin and Womersley's equations				
Relative passive mass	0.24*	-0.15	0.28*	0.18
Absolute passive mass	0.28*	-0.18	0.33**	0.16
Absolute active mass	0.08	-0.08	0.12	-0.01
Somatotype components				
Endomorphy	0.24*	-0.15	0.28*	0.18
Mesomorphy	0.26*	0.26	0.23	0.11
Ectomorphy	-0.28*	-0.07	0.21	-0.24*

PCOS – women with polycystic ovary syndrome, HDL – high density lipoprotein cholesterol, LDL – low density lipoprotein cholesterol, TG – triglycerides; * $p < 0.01$; ** $p < 0.001$.

PCOS skin and subcutaneous adipose tissue, passive mass and endomorphic body composition parameters positively correlated with triglycerides (Table 5).

The following weak correlations were determined in the control group: (1) between subcutaneous adipose tissue, passive mass, endomorphy and mesomorphy and the total cholesterol level and (2) for skeletal mass, subcutaneous ad-

ipose tissue, viscera and reminders and the passive mass endomorphy and LDL cholesterol level.

Discussion

In recent years, attention has centred on determining body composition in PCOS women with different body mass index, quantity and quality of bone mass and

muscles and adipose tissue (Zborowski et al., 2000; Kirchengast et Huber 2001; Faulds 2003; Puder et al. 2005; To and Wong 2005; Toscani et al. 2007; Barber et al. 2008; Cosar et al. 2008; Moran and Teede 2009; Kassanos et al 2010; March et al. 2010; Manneras-Holm et al. 2011; Villa et al. 2011 and Barber and Franks 2013). Our study presents analytic results of investigations into body composition and somatotype in lean PCOS women.

PCOS women had similar bone mineral density (BMD) levels to the control group (To and Wong 2005; Zborowski et al. 2000). Biochemical hyperandrogenism and elevated circulating insulin levels, directly through stimulation of osteoblastic activity, or indirectly via its effect on PCOS associated sex hormone-binding globulin or insulin-like growth factor binding proteins, have a positive effect on BMD alleviating negative impacts associated with anovulation (Zborowski et al. 2000). Our study showed that lean women with PCOS had greater mean skeletal mass than control women. Peripheral quantitative computerised tomography established significantly higher distal-tibial cortical density in lean PCOS than that in lean control women, although trabecular bone density did not differ (Kassanos et al 2010). Good et al. (1999) reported higher BMD in the left and right arms and left ribs of a lean PCOS group compared to control values. These authors suggested that regional differences in PCOS bone mass, and particularly the significant upper skeletal BMD increase, indicates lean mass accretion in the trunk and upper extremities (Good et al. 1999).

Dual energy X-ray absorptiometry results for total body fat in lean PCOS women and healthy controls were dis-

cordant. Although an Austrian study reported that lean women with PCOS had significantly higher total fat mass than controls matched for age, weight and BMI (21.2 kg vs. 14.8 kg, $p=0.002$) (Kirchengast and Huber 2001), Puder et al. (2005) determined that total fat mass did not differ between PCOS subjects and controls (25.6 kg vs. 25.2 kg, $p=0.9$). While Carmina et al. examined total, trunk and central abdominal fat quantity by total-body dual energy X-ray absorptiometry in 40 lean PCOS women and weight-matched controls (BMI=22.4 kg/m²) and agreed that there was no difference in total fat (Carmina et al. 2007), our study revealed that lean PCOS women had higher skin and subcutaneous adipose tissue and greater relative and absolute passive masses than our lean healthy controls.

Studies examining fat distribution in lean PCOS women gave contradictory results. Topographical studies revealed that the adipose tissue mass in internal organs, waist and arms was greater in PCOS women than in controls. Herein, over two thirds of total adipose tissue accumulated in the upper torso in PCOS women with different weights, and up to 70% PCOS women exhibited male fat distribution patterns (Gennarelli et al. 2000; Kirchengast and Huber 2001; Crosignani et al. 2003; Hashimoto et al. 2003; Snijder et al. 2004; Kirchengast 2005; Li and Lin 2005; Barber et al. 2006; Carmina et al. 2007; Toscani et al. 2007; Moran and Teede 2009; March et al. 2010 and Penaforte et al. 2011).

The following contrasting results were also published; (1) Kirchengast and Hubert reported that half their lean PCOS patients had android fat distribution and all lean controls were gynoid (Kirchengast 2001), (2) Carmina et al. (2007) re-

corded that young normal weight PCOS females with BMI=22.4 kg/m² had significantly higher central abdominal fat (451 g vs. 344 g, $p<0.01$) and higher trunk fat compared to their total fat percentage (36.6% vs. 32.8%, $p<0.01$), (3) Yildirim et al. (2003) demonstrated that the mean pre-peritoneal and visceral fat layer determined by ultrasound in non-obese PCOS patients with BMI<25 kg/m² was significantly greater than in controls, (4) Puder et al. (2005) accorded that the trunk to extremity fat ratio was higher in PCOS women (BMI=26.3±5.7 kg/m²) compared to controls (BMI=25.5±4.8 kg/m²) (1.06 vs. 0.79, $p=0.007$), (5) Yucel et al. (2005) showed that fat mass in the trunk and arms and the ratio of trunk fat mass to leg fat mass was significantly higher in non-obese patients with PCOS ($p<0.05$): (6) A Swedish study reported that lean PCOS women with mean BMI 23±1.5 kg/m² had significantly higher trunk to peripheral fat ratio (Svendsen et al. 2008), (7) Good et al. (1999) found no statistically significant differences in body fat distribution in lean PCOS women and controls; although the former tended to have lower mean body fat percentages, and (8) most recently, Aydin et al. (2013) documented no differences in fat distribution in lean PCOS women and healthy controls.

In addition to distribution sites, the quality of adipose tissue also differs in healthy and PCOS women. Faulds et al. (2003) showed that subcutaneous fat cells' lipolytic response to catecholamines decreased in young lean PCOS women with 24.8±4.8 kg/m² mean BMI. This increased their abdominal fat cell volume by approximately 25%. Decreased lipolytic activity and increased fat-cell lipid content is known to promote subsequent obesity in PCOS women,

and Manneras-Holm et al. (2001) contend that lean PCOS women have larger adipocytes, a lower serum adiponectin level, lower adipose tissue lipoprotein lipase activity and increased waist-to-hip ratio. These authors reported no further differences in anthropometric variables, abdominal adipose tissue volume or its distribution, and they concluded that adipocyte size, circulating adiponectin and waist circumference were the most important variables associated with insulin sensitivity in PCOS women. They further suggested that these factors are more important than biochemical hyperandrogenism in PCOS development and maintenance of insulin resistance.

Studies of lean body mass in PCOS women were also fraught with different results: (1) Carmina et al. (2009) demonstrated that muscle mass increased in PCOS women compared to weight-matched controls (mean BMI of 27.6±5.8 kg/m²). The lean mass expressed as total lean mass divided by height was significantly higher in PCOS women with PCOS than in controls at 275 vs. 256 total g/height cm, $p<0.01$. (2) Kirschengast and Hubert (2001) reported that lean PCOS women had significantly lower total and upper lean body mass than lean healthy controls at 35.6 kg vs. 38.7 kg, $p=0.04$ and 20.8 kg vs. 18.7 kg respectively, $p=0.03$, (3) while, our study showed significantly higher muscle mass in lean controls than in lean PCOS women, (4) (Dantas et al. 2013) reported that skeletal muscle plays a pivotal role in the peripheral glucose uptake and (5) Schooling et al. (2011) concurred that low muscle mass, especially from adolescence, can increase the risk of diabetes; (6) McGarry (2002) added that triglycerides in the form of ectopic fat mass accumulate in skeletal

muscles from insulin resistance. This increases overall weight and adds to the insulin resistance found in 30–50% of normal body weight PCOS women. (7) our study established that insulin and HOMA-IR were higher in PCOS women than in controls; but not significantly so. Lean PCOS women showed lower QUICKI than lean controls, but again without significant difference (0.36 vs. 0.32, $p=0.052$). We also found a moderate positive correlation in PCOS women with skin and subcutaneous adipose tissue mass, for relative and absolute passive mass with insulin and HOMA-IR (with insulin $r=0.52$, $p<0.0001$, with HOMA-IR $r=0.51$, $p<0.0001$) and a negative correlation with QUICKI ($r=-0.49$, $p<0.0001$); (8) meanwhile, Carmina and colleagues (2007) detected strong correlations between central abdominal fat and insulin and also QUICKI in PCOS women with normal body weight (with insulin $r=0.69$, $p<0.01$; QUICKI: $r=-0.66$, $p<0.01$) and (9) Aydin and colleagues (2013) showed that HOMA-IR was positively correlated with total fat percentage, fat mass and trunk fat mass and percentage in lean PCOS women.

Our study results determined that PCOS sufferers had lower HDL cholesterol and higher TG compared to controls. We detected weak positive correlations for TG with skin, subcutaneous adipose tissue mass and relative and absolute passive mass. However, while Yildirim et al. (2003) found no correlation between subcutaneous fat density and metabolic variables in non-obese PCOS women, they related serum TG level to visceral fat and pre-peritoneal fat thickness (Yildirim et al 2003).

Human body size, structure, proportions and composition change significantly during our life span. Heath-Cart-

er's (1990) somatotyping classifies population in terms of fatness (endomorphism), muscularity (mesomorphism) and linearity (ectomorphism). Kalichman L (2006) reported the following age related somatotype changes prevalent in women; (1) mesomorphism continues to increase until the 5th decade, (2) ectomorphism tends to decrease until the 5th decade, (3) endomorphism increases until the 6th decade and decreases thereafter (4) mean endomorphism values decrease during the 7th and 8th decades, (5) 18–30 years old women with mean BMI of 23.2 kg/m² had an endomorphism-mesomorphism-ectomorphism somatotype ratio of 3.78–4.59–2.15 (Kalichman L 2006), and, (6) Sterkowicz-Przybycien and Almansba (2011) established that healthy Polish women had a balanced endomorph ratio at 4.22–2.99–3.08.

Few studies have related somatotype and physical phenomena in patients with different diseases. While type 2 diabetes mellitus sufferers were generally classified as overweight and obese with central body fat distribution pattern, diabetic women with a mean somatotype ratio of 8.6–6.4–0.2 had significantly higher endomorphic values than controls ($p<0.05$) (Buffa et al. 2007a). In contrast, Bulgarian diabetic females were predominantly mesomorphic, and Baltadjiev AG (2012) considers that this somatotype group possesses advantages in diabetic risk and prognosis (Baltadjiev AG 2012). Women Alzheimer patients with mean somatotype ratio of 7.0–5.3–0.7 were less mesomorphic and more ectomorphic than the controls who registered 7.7–6.3–0.4. These differences were significant with mesomorphism $p=0.000$ and ectomorphism $p=0.012$ (Buffa et al. 2007).

Our study identified a PCOS somatotype ratio of 4.96–4.38–3.00 compared

to 4.17–4.59–2.89 in controls, and our somatotype means were significantly different at $p=0.031$ due to endomorphic difference. Here, almost one third of PCOS women were mesomorphic endomorphs while 30% of healthy controls were endomorphic mesomorphs.

Koleva and colleagues (2002) showed that mesomorphic endomorphs most frequently suffered from digestive system diseases (40.6%, $p<0.05$), neuroses (30.1%, $p<0.05$), and lumbosacral radiculitis (15.4%), while those with the highest endomorphy and mesomorphy and the lowest ectomorphy frequently experienced arterial hypertension and liver disease. These authors concluded that dominant mesomorphy and marked endomorphy increase the risk of certain diseases, and they therefore stress the extreme necessity for body weight control.

Conclusion

Lean women with polycystic ovary syndrome had a mesomorphic endomorph=somatotype and higher skin and subcutaneous adipose tissue mass, but less muscle mass than lean healthy women. Here, skin and subcutaneous adipose tissue positively correlated with insulin and HOMA-IR in our lean PCOS women.

Author contribution

LZ data collection, analysis and article writing; JU data collection, analysis and article writing, JT main scientific supervisor of the study and reviewer of the article.

Conflict of interest

The authors declare there is no conflict of interests

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