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CLINICAL ARTICLE

Resting metabolic rate and exercise capacity in women with polycystic ovary syndrome

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Abstract

Objective: To assess the resting metabolic rate (RMR) and exercise capacity (or maximal oxygen consumption [VO_2 max]) of women with polycystic ovary syndrome (PCOS) and central adiposity. *Method:* In a cross-sectional study, RMR was measured by indirect calorimetry and VO_2 max by the Astrand test for 31 women with PCOS and 29 controls matched for age and body mass index, but with a different body fat distribution. Differences between the means were analyzed. *Results:* There were no significant differences in RMR or VO_2 max values between the 2 groups. *Conclusion:* Central adiposity was not predictive of an altered RMR or of decreased exercise capacity in women with PCOS.

1. Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrinopathies, with a prevalence ranging from 5% to 10%. It causes anovulation and infertility in women of reproductive age [1] and many other health risks are associated with it. Women with PCOS are at increased risk for features of metabolic syndrome such as obesity, insulin resistance, and hyperlipidemia, and for developing type 2 diabetes and hypertension, with a subsequent risk of cardiovascular disease [2]. Approximately 50% of women with PCOS are overweight or obese, with body fat mostly centrally located [2]. Although lifestyle modifications, in the form of exercise and proper nutrition, decrease the risk of developing the nonreproductive conditions just mentioned [3], dietary restrictions are generally difficult to maintain in the long-term. Very low calorie diets have been shown to alter the body's metabolism adversely by decreasing the resting metabolic rate (RMR), and it has been theorized that the decrease in RMR is a consequence of the decrease in lean mass associated with diet-induced weight loss [4].

The aim of this study was to compare the RMR and exercise capacity (or maximum oxygen consumption [VO_2 max]) of women with PCOS and central adiposity—an indicator of cardiovascular morbidity—with those of women

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without PCOS matched for age and body mass index (BMI), but with different patterns of body fat distribution.

2. Methods

2.1. Study participants

In this prospective study carried out in 2007, 31 consecutive women with PCOS were matched for age and BMI with 29 women who did not have the condition. The diagnosis of PCOS was defined according to the consensus criteria reached in May 2003 in Rotterdam, the Netherlands, among representatives of the European Society of Human Reproduction and the American Society of Reproductive Medicine [5]. The criteria are the following: clinically evident or biochemical hyperandrogenism; oligomenorrhea; polycystic ovaries as defined by ultrasonography; and exclusion of other etiologies such as congenital adrenal hyperplasia, androgen-secreting tumors, or Cushing syndrome. The control group consisted of 29 women presenting for common gynecological complaints such as vaginal discharge or pruritus. The controls had regular menstrual cycles and no clinical or biochemical evidence of hyperandrogenism.

2.2. Measurements taken and tests performed

The women were weighed in their underwear to the nearest 0.1 kg and their height was recorded to the nearest 0.5 cm. Body mass index was calculated as weight in kilograms divided by height in meters squared. Using a metal measuring tape 1 cm wide, waist circumference was measured as the minimum value between the iliac crest and the lateral costal margin and hip circumference as the maximum value over the buttocks. The waist to hip ratio was then calculated. None of the participants were on any special diet.

Table 1 Demographic, hormonal, and physical characteristics of the study population[a]

Characteristic	PCOS group ($n=31$)	Control group ($n=29$)	P value
Age, y	25.9 ± 5.3	27.1 ± 4.8	NS
Gravidity	2 (0–4)	2 (0–4)	NS
Parity	2 (0–4)	2 (0–4)	NS
BMI	26.97 ± 5.12	26.03 ± 5.66	NS
FSH, mIU/mL	5.24 ± 1.89	7.69 ± 2.98	0.03
LH, mIU/mL	7.01 ± 3.88	6.23 ± 5.21	NS
LH/FSH	1.34 ± 0.61	0.81 ± 0.65	0.001
Estradiol, pg/mL	56.76 ± 20.44	47.65 ± 22.56	NS
Free testosterone, pg/mL	3.01 ± 0.95	0.96 ± 0.71	0.001
DHEA-S, μg/dL	227.56 ± 76.45	215.51 ± 81.67	NS
Fasting glucose, mg/dL	95.64 ± 11.03	92.49 ± 10.66	NS
Fasting insulin, mIU/mL	16.13 ± 9.86	7.25 ± 3.01	0.03
Fasting glucose to fasting insulin ratio	6.01 ± 3.72	13.56 ± 6.13	0.001
Waist to hip ratio	0.81 ± 0.07	0.78 ± 0.08	0.04

Abbreviations: BMI, body mass index, calculated as weight in kilograms divided by height in meters squared; DHEA-S, dehydroepiandrosterone sulfate; FSH, follicle stimulating hormone; LH, luteinizing hormone; NS, not significant; PCOS, polycystic ovary syndrome.
[a] Values are given as mean ± SD or as median (range) unless otherwise indicated.

Table 2 Comparison of cardiovascular fitness and RMR values between study and control groups[a]

Variable	PCOS group ($n=31$)	Control group ($n=29$)	P value
VO_2 max, mL kg^{-1} min^{-1}	29.15 ± 7.02	27.33 ± 9.41	0.397
RMR, kcal/d	1166.87 ± 370.98	1045.52 ± 295.86	0.168

Abbreviations: PCOS, polycystic ovary syndrome; RMR, resting metabolic rate; VO_2 max, maximum volume of oxygen consumed, expressed in milliliters per kilogram of body weight per minute.
[a] Values are given as mean ± SD unless otherwise indicated.

The RMR was measured using an indirect calorimeter (Quark b^2; Cosmed, Rome, Italy) with a computerized metabolic card, which analyzed oxygen consumption and carbon dioxide production. The participants were instructed not to exercise, eat, or smoke, respectively, in the 24 h, 12 h, and 2 h preceding the test, which was performed between 8:30 AM and 10:30 AM in a silent, dark, and temperate room after the participants had rested for 15 min. Lying in a supine position and wearing a facemask, the participants did not move their limbs during the 15 to 20 min necessary to perform the test.

The maximum volume of oxygen consumed to produce energy over 1 min (VO_2 max), expressed in milliliters per kilogram of body weight, was determined from heart rate and workload using the Astrand test. The test was monitored by the same person and performed in the same order and conditions for all participants on a computerized cycle ergometer (Monark 839; Monark Exercise AB, Valberg, Sweden). The participants had been instructed not to eat in the 2 h preceding the test and were wearing convenient clothes. Their heart rate was measured using a chest belt telemetry system (Polar model; Monark Exercise AB). After performing the submaximal exercise test for 6 min, the participants were asked to reach their steady-state heart rate.

Serum levels of follicle stimulating hormone, luteinizing hormone, 17-β-estradiol, 17-OH-progesterone, basal prolactin, total and free testerone, dehydroepiandrosterone sulfate, androstenodione, total cholestrol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, fasting glucose, and insulin were measured in all participants during the early follicular phase (from the second to the fifth day of the menstrual cycle). The hormonal tests were by chemiluminescent enzyme immunoassay using the AxSYM System (Abbott Diagnostics, Abbott Park, IL, USA). The mean interassay and intraassay coefficients of variation were 6% and 6.7%. The serum concentration of dehydroepiandrosterone sulfate, 17-OH-progesterone, free testosterone, free thyroxine, and thyroid stimulating hormone were measured according to standard radioimmunassays. Immune-reactive insulin levels were determined using a microparticle enzymatic immunoassay. Levels of total cholesterol, high-density lipoprotein cholesterol, and triglycerides were determined by standard methods. Insulin sensitivity was estimated by calculating the fasting glucose to fasting insulin ratio.

The research ethics board of the Medical School of Afyon Kocatepe University reviewed and approved the study. All

participants signed an informed consent form after the aim of the study had been explained.

2.3. Statistical analysis

Statistical analyzes were performed with SPSS, version 13.0 (SPSS, Chicago, IL, USA). The Shapiro–Wilk statistic was calculated to test for normal distribution. Differences between means were analyzed by the *t* test or the Mann–Whitney *U* test depending on data distribution. The results are given as mean ± SD, and $P<0.05$ was accepted as statistically significant.

3. Results

Demographic characteristics, physical measurements, and mean serum biochemical and hormonal levels are given in Table 1. There were no significant differences in age ($P>0.05$) and BMI ($P>0.05$) between the groups. Free testosterone levels were significantly higher in the PCOS than in the control group ($P=0.001$). Compared with the control group, the fasting insulin level was significantly higher and the fasting glucose to fasting insulin ratio was significantly lower in the PCOS group ($P=0.03$ and $P=0.001$, respectively). The waist to hip ratio was significantly higher in the PCOS group ($P=0.04$). There were no significant differences in sex hormone-binding globulin levels ($P>0.05$) or in lipid profile between the 2 groups ($P>0.05$). Thyroid stimulating hormone levels were within normal ranges in both groups.

No significant difference in RMR was observed between the 2 groups ($P=0.168$) and the value of VO_2 max was not associated with PCOS ($P=0.397$) and, therefore, with central adiposity (Table 2).

4. Discussion

The results of this study provide no evidence of an altered RMR or exercise capacity in women with PCOS and excessive central body fat.

The RMR is the amount of energy expended while at rest in a temperate environment in the postabsorptive state (meaning that the digestive system is inactive, which requires about 12 h of fasting in humans). The release of energy in this state is only sufficient for the functioning of the vital organs, such as the heart, lungs, brain, and the rest of the nervous system, and the maintaining of the other parts of the body. The RMR decreases with age and the loss of lean body mass can increase with cardiovascular exercise and an increased muscle mass. An illness, the intake of food and beverages, environmental temperature, and stress levels can affect the overall energy expenditure. An accurate RMR measurement requires the person to be awake but at complete rest, with the sympathetic nervous system unstimulated. Resting metabolic rates also differ among populations. In a comparison between young Gambian and Swiss men, basal metabolism adjusted for body composition was found to be lower among the Gambians [6]. Environmental factors such as climate, diet, and physical activity do not influence RMR, however, and RMR is not likely to contribute to differences in obesity prevalence between countries [7].

Segal et al. [8] investigated whether the high frequency of obesity in women with PCOS was related to a lower energy expenditure, RMR, and/or thermic response to a standard meal in these women. Their results support the hypothesis that thermogenesis is blunted in obese individuals, but provide no evidence of altered RMR or postprandial thermogenesis in women with PCOS compared with women without the condition but with a similar degree of obesity [8]. Faloia et al. [9] analyzed the possible causes and effects of obesity, such as resting energy expenditure, postprandial thermogenesis, and insulin resistance in women with PCOS. Their results confirm previous reports on hyperinsulinemia and insulin resistance in these women. Furthermore, in that study, reduced postprandial thermogenesis was statistically related to reduced insulin sensitivity in the women with PCOS. The authors concluded that the reduced postprandial thermogenesis in women with PCOS might predispose them to weight gain [9].

Although PCOS is described as a cause of infertility [1], women with PCOS and controls had the same parity in our study, perhaps because of the small number of participants, all of whom were overweight but none of whom were obese (all had a BMI <30). All of the women with PCOS had adopted lifestyle changes to improve fertility or lessen menstrual cycle irregularities at some time in their lives, but they had not been following any diet or exercise regimen in the previous 6 months.

Exercise induces positive changes in body composition, and an increased fitness level may prove beneficial for women with PCOS, as endurance exercise training and increase in relative and absolute VO_2 max values have been shown to improve insulin action in obese individuals [10].

In conclusion, although the narrow range for age and BMI is a limitation to the study, the RMR was the same for women with PCOS and controls with a similar BMI. In this cross-sectional analysis, compared with other patterns of body fat distribution, the central adiposity characteristic of women with PCOS was not predictive of a lower RMR or of a lower VO_2 max value. Motivating women with PCOS to adopt positive lifestyle modifications for the long-term would be beneficial to them; however, further studies are needed to evaluate RMR and VO_2 max in women with PCOS of different BMI ranges and age groups.

References

[1] Archer JS, Chang RJ. Hirsutism and acne in polycystic ovary syndrome. Best Pract Res Clin Obstet Gynaecol 2004;18:737–54.
[2] Norman RJ, Dewailly D, Legro RS, Hickey TE. Polycystic ovary syndrome. Lancet 2007;25:685–97.
[3] Diabetes Prevention Program Research Group. The diabetes prevention program: description of lifestyle intervention. Diabetes Care 2002;25:2165–71.
[4] Ross R, Janssen I, Tremblay A. Obesity reduction through lifestyle modification. Can J Appl Physiol 2000;25:1–18.
[5] The Rotterdam ESHRE/ASRM-sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome (PCOS). Hum Reprod 2004;19:41–7.
[6] Minghelli G, Schutz Y, Charbonnier A, Whitehead R, Jequier E. Twenty four hour energy expenditure and basal metabolic rate measured in a whole-body indirect calorimeter in Gambian men. Am J Clin Nutr 1990;51:563–70.
[7] Luke A, Rotimi CN, Adeyemo AA, Arvizu RAD, Prewitt TE, Kayser LM. Comparability of resting energy expenditure in Nigerians and US blacks. Obes Res 2000;8:351–9.

[8] Seagl KR, Dunaif A. Resting metabolic rate and postprandial thermogenesis in polycystic ovarian syndrome. Int J Obes 1990;14:559–67.
[9] Faloia E, Canibus P, Gatti C, Frezza F, Santangelo M, Garrapa GG, et al. Body composition, fat distribution and metabolic characteristics in lean and obese women with polycystic ovary syndrome. J Endocrinol Invest 2004;27:424–9.
[10] Després JP, Lamarche B. Physical activity and the metabolic complications of obesity. In: Bouchard, C, editor. Physical activity and obesity. USA: Human Kinetics; 2000. p. 331–54. Champaign, Illinois.