Available online at www.sciencedirect.com
ScienceDirect
Metabolism Clinical and Experimental 56 (2007) 992 – 997
Metabolism
Clinical and Experimental
www.elsevier.com/locate/metabol

Estimation of truncal adiposity using waist circumference or the sum of trunk skinfolds: a pilot study for insulin resistance screening in hirsute patients with or without polycystic ovary syndrome

Mariana Toscani[a,b], Raphaella Migliavacca[a,b], José Augusto Sisson de Castro[a], Poli Mara Spritzer[a,b,*]

[a]*Gynecological Endocrinology Unit, Division of Endocrinology, Hospital de Clínicas de Porto Alegre, Porto Alegre, Brazil*
[b]*Department of Physiology, Universidade Federal do Rio Grande do Sul, Porto Alegre, Brazil*

Received 5 May 2006; accepted 29 March 2007

Abstract

Insulin resistance (IR) is an independent risk factor for cardiovascular disease and is a prevalent metabolic disturbance among women with polycystic ovary syndrome (PCOS). Central adiposity, a marker of IR and an accurate anthropometric method to estimate truncal adiposity, may represent a key clinical tool for IR screening in subpopulations at higher metabolic and cardiovascular risk, such as women with PCOS. The aims of the present study were (1) to investigate the influence of androgens on IR and central obesity in overweight or obese hirsute women with or without PCOS and (2) to test the reliability of the sum of trunk skinfolds (subscapular, suprailiac, and abdominal) to estimate truncal adiposity. This observational, cross-sectional study included 37 hirsute patients with body mass index of 25 kg/m^2 or greater and aged between 14 and 41 years. Twenty-four had PCOS, and 13 had ovulatory cycles, normal androgen levels, and isolated hirsutism, named idiopathic hirsutism (IH). Nutritional, anthropometric, clinical, and laboratory evaluations were performed. Body composition was assessed by measurement of waist circumference and skinfold thickness and by dual-energy x-ray absorptiometry (DXA). Both groups presented similar ages, body mass index, and hirsutism score. The PCOS group had higher androgen levels, homeostasis model assessment (HOMA) index, and fasting insulin levels. Free androgen index was positively associated with HOMA, independent of truncal adiposity ($r = 0.441$, $P = .009$). Strong correlations were also observed between truncal adiposity measured by DXA and both the sum of trunk skinfolds ($r = 0.863$, $P = .0001$) and waist circumference in hirsute patients ($r = 0.947$, $P = .0001$). In our study, IR (HOMA index $\geq$3.8) was associated with truncal obesity, with a more androgenic profile, and with an unfavorable lipid profile. In conclusion, hirsutism per se appears not to be a risk for IR and related cardiovascular disease unless there is presence of central adiposity and/or abnormal androgen profile as observed in patients with PCOS. Waist circumference and the sum of trunk skinfolds represent accurate methods to estimate truncal adiposity, but waist circumference measurement seems to be the simplest method of clinical screening for IR in hirsute women.

1. Introduction

Obesity, a major public health concern, is a chronic disorder that results from the interaction of numerous social, physiologic, metabolic, and cellular factors [1]. This multifactorial illness is a risk factor for type 2 diabetes mellitus, cardiovascular disease, osteoarthritis, several types of cancer, and certain reproductive and metabolic disorders. It is generally accepted that this risk relates more to the central distribution of fat than to the total amount of body fat [2-4]. Abdominal obesity is thought to play an important role in insulin resistance (IR) because the increased production of free fatty acids may interfere with the action of insulin [5].

There is strong epidemiologic and clinical evidence [2,6,7] that sex steroid hormones greatly influence the

Presented in part as a poster at the 88th Annual Meeting of the Endocrine Society, Boston, MA, June 2006.

* Corresponding author. Department of Physiology, Universidade Federal do Rio Grande do Sul, 90050-170 Porto Alegre RS, Brazil. Tel.: +55 51 3308 3671; fax: +55 51 3308 3656.
E-mail address: spritzer@ufrgs.br (P.M. Spritzer).

0026-0495/$ – see front matter © 2007 Elsevier Inc. All rights reserved.
doi:10.1016/j.metabol.2007.03.006

regulation of adipose tissue distribution. Androgens may also affect central obesity in women during their reproductive years. The hyperandrogenism that is often seen in patients with polycystic ovary syndrome (PCOS) is associated with obesity of the abdominal phenotype [8-11]. Hirsutism (excessive hair growth in women in places in which terminal hair is normally not found), one of the major symptoms of PCOS, may result from an overproduction of androgens by the ovaries and/or adrenal glands or by increased sensitivity of the pilosebaceous unit to normal levels of circulating androgens [8,12,13]. Thus, overweight and obese hirsute patients with normal or increased androgen levels may serve as a reliable model to assess the relationship of androgens and IR with central adiposity.

Measuring body fat is still a challenge for researchers and clinicians. Fat depots are mainly subcutaneous and intra-abdominal; however, considerable amounts of fat can also reside among and inside muscles, particularly in the elderly [14]. Because fat is widespread and inaccessible, it is not possible to directly and accurately measure whole-body adiposity. Measurement of the waist-hip ratio (WHR) has been shown to reflect the amount of abdominal fat and is widely used to investigate the relations between abdominal fat and metabolic profile [15]. More recently, waist circumference alone has been reported to be more closely correlated with the amount of abdominal fat than with WHR in men and women [16-18]. Finally, the body mass index (BMI) is the main tool to evaluate total body adiposity. However, studies have shown that a large amount of visceral fat may represent a cardiovascular risk factor even within normal BMI values [5,18]. Actually, it seems to be better to consider the combined BMI and waist circumference for estimating cardiovascular risk [19].

Currently, the most accurate in vivo method of measuring abdominal adipose tissue is computed tomography (CT). Although CT represents a technological advance and is used as a reference standard, its application in routine clinical practice and research is limited by cost, availability of equipment, and exposure to significant amounts of ionizing radiation [20]. Other techniques that are comparable to CT in terms of accuracy include magnetic resonance imaging [16] and dual-energy x-ray absorptiometry (DXA), a simple method that exposes subjects to minimal amounts of radiation [16,20]. In addition, one of the simplest methods to evaluate total body fat is the measurement of skinfold thickness [21]. However, there is a dearth of studies evaluating the use of specific skinfolds to determine truncal obesity [22,23]. Therefore, the aims of the present study were (1) to investigate the influence of androgens on IR and central obesity in overweight or obese hirsute women with or without PCOS and (2) to test the reliability of the sum of trunk skinfolds (subscapular, suprailiac, and abdominal) to estimate truncal adiposity.

2. Patients and methods

2.1. Patients

The study population included women consulting for hirsutism at the Gynecological Endocrinology Unit at Hospital de Clínicas de Porto Alegre, Brazil. Late-onset (nonclassical) congenital adrenal hyperplasia, Cushing syndrome, and androgen-secreting tumors were excluded by appropriate tests as previously described [8,24,25]. Patients with diabetes mellitus and hyperprolactinemia (serum prolactin concentrations >20 μg/L on 2 different occasions) were also excluded.

Thirty-seven hirsute patients with BMI of 25 kg/m^2 or greater and aged between 14 and 41 years were enrolled in the study. None had received any drugs known to interfere with hormonal levels for at least 3 months before the study. Thirteen patients had regular ovulatory cycles (luteal-phase progesterone levels greater than 3.8 ng/mL) and normal androgen levels and were classified as having idiopathic hirsutism (IH). Twenty-four were oligo/amenorrheic (<9 cycles per year), had increased levels of serum testosterone and/or free androgen index, and were diagnosed as having PCOS [26]. All patients were stratified according to the presence of IR by using the homeostatic model assessment (HOMA). The HOMA value correlates well with clamp techniques and has been used to study IR in patients with PCOS of different ethnic origins [27].

The study protocol was approved by the local ethics committee (institutional review board equivalent), and written informed consent was obtained from all subjects.

2.2. Study protocol

Anthropometric measurements were determined twice by 2 different authors (M.T. and R.M.) and included body weight, height, waist circumference, WHR (waist measured at the midpoint between the lower rib margin and the iliac crest), hip circumference (widest circumference over the buttocks) [28], and BMI (current measured weight in kilograms divided by height in meters squared). Hirsutism was assigned by the original method of Ferriman and Gallwey [29]. Blood pressure was measured after a rest period of 10 minutes, with women in the supine position.

The hormonal and metabolic assessment was made between days 2 and 10 of the menstrual cycle or on any day when the patients were amenorrheic. After an overnight fast, blood samples were drawn from an antecubital vein for determination of plasma cholesterol, high-density lipoprotein cholesterol (HDL-C), and triglycerides at baseline, and glucose and insulin before and 2 hours after the ingestion of 75 g of oral glucose (oral glucose tolerance test). Impaired glucose tolerance was determined by glucose levels between 140 and 200 mg/mL, as defined by the World Health Organization [30].

Blood samples were also drawn for measurements of sex hormone–binding globulin (SHBG) and total testosterone.

All samples were obtained between 8:00 and 10:00 AM. The free androgen index (FAI) was estimated by using the following formula: T (nmol/L)/SHBG (nmol/L) × 100, where T is total testosterone. HOMA index was calculated by multiplying insulin (μIU/mL) by glucose (mmol/L) and dividing the product by 22.5.

The cutoff point to define IR was arbitrarily defined as a HOMA index of 3.8 or greater [31].

2.3. Biochemical and hormonal assays

Total cholesterol, HDL-C, triglyceride, and glucose levels were determined by colorimetric-enzymatic methods using the Bayer 1650 Advia System (Mannheim, Germany). Low-density lipoprotein cholesterol (LDL-C) was determined indirectly by using the formula LDL = total cholesterol − HDL + triglycerides/5.

Total serum testosterone levels were measured with the radioimmunoassay method (ICN, Costa Mesa, CA) with intra- and interassay coefficients of variation (CVs) of 10% and 11.6%, respectively. SHBG was measured by a chemiluminescent enzyme immunoassay (DPC, Los Angeles, CA) with sensitivity of 0.2 nmol/L and intra- and interassay CVs of 6.1% and 8.0%, respectively. Serum insulin levels were measured with an electrochemiluminescence immunoassay (Roche Diagnostics, Mannheim, Germany) with an assay sensitivity of 0.20 μIU/mL and intra- and interassay CVs of 1.8% and 2.5%, respectively.

2.4. Measurement of skinfold thicknesses

Skinfold thickness was estimated by using a caliper (Cescorf, Mitutoyo, Porto Alegre, Brazil) with a 0.1-mm scale and pressure of 10 g/mm^2. Measurements were performed by the same author (M.T.) at the triceps, subscapular, abdominal, and suprailiac regions. Values were the mean of 3 measurements for each skinfold. To estimate truncal adiposity, the sum of 3 skinfold measurements—subscapular, suprailiac, and abdominal—was considered (referred to as "sum of trunk skinfolds," expressed in millimeters).

The percentage of total body fat was calculated by the Faulkner formula [32]: percent total body fat = (triceps + subscapular + suprailiac + abdominal skinfolds × 0.153) + 5.783.

Table 1
Anthropometric and clinical characteristics of hirsute women with or without PCOS

Variable	PCOS (n = 24)	IH (n = 13)	*P*
Age (y)	23 ± 1.4	27 ± 2.5	.190
BMI (kg/m^2)	34 ± 1	30.2 ± 1	.081
Hip circumference	117 ± 15	110 ± 11	.204
WHR	0.82 ± 0.01	0.77 ± 0.01	.007
Ferriman hirsutism score	15 ± 1	15 ± 1	.719
Systolic blood pressure	127 ± 3	121 ± 4	.320
Diastolic blood pressure	83 ± 3	78 ± 4	.469

Values are expressed as mean ± SEM (Student *t* test).

Table 2
Hormonal and metabolic data of hirsute women with or without PCOS

Variable	PCOS (n = 24)	HI (n = 13)	*P*
Testosterone (ng/mL)	0.9 ± 0.1	0.6 ± 0.1	.004
SHBG (nmol/L[a])	17 (13-57)	30 (21-40)	.24
Free androgen index	19.6 ± 3.5	9.7 ± 2.8	.034
Fasting glucose (mg/dL)	88 ± 2	83 ± 3	.258
2-h glucose (mg/dL)	122 ± 6	121 ± 8	.888
Fasting insulin (μUI/mL)[a]	28 (15-50)	13 (8-41)	.019
HOMA[a]	6 (3-10)	3 (2-7)	.008
Total cholesterol (mg/dL)	195 ± 10	185 ± 9	.501
HDL-C (mg/dL)	50 ± 2	50 ± 3	.966
LDL-C (mg/dL)	171 ± 11	156 ± 9	.372
Triglycerides (mg/dL)[a]	114 (67-161)	88 (73-140)	.276

Values are expressed as mean ± SEM (Student *t* test) or median and interquartile range.

[a] Median and interquartile range (25%-75%) (Mann-Whitney *U* test).

2.5. Body composition by DXA

Subjects were scanned in whole-body mode with a DXA device (HOLOGIC QDR 4500A, Waltham, MA) at the Division of Radiology at Hospital de Clínicas de Porto Alegre. Precision errors were estimated by repeated measurements with complete repositioning for the following regions: subtotal body mass density (head excluded) CV = 1.06%; subtotal body fat = 1.03%; and subtotal body lean mass (bone excluded) = 0.92%. Trunk fat mass was equal to the subtotal body fat mass minus the fat mass values for the arms and legs. Patients laid supine on the DXA table with arms adequately separated from the trunk and were instructed to remain still throughout the scanning procedure. All DXA procedures were performed by the same technician and interpreted by the same author (J.A.S.C.).

2.6. Statistical analysis

Results are presented as mean ± SEM. Nonparametric data are presented as median and interquartile range. Two-tailed Student *t* tests were used to compare the differences between means of parametric continuous variables and the Mann-Whitney *U* test was used for comparisons of nonparametric data. Statistical significance for categorical variables was calculated by Pearson χ^2 test. Spearman rank correlation coefficient was calculated between variables by using a 2-tailed significance test for variables with non-Gaussian distribution. Log_{10} transformation was used to normalize the distribution of non-Gaussian variables related to HOMA and FAI, and Pearson correlation test was performed. Mean values were back-transformed for presentation.

All analyses were performed using the Statistical Package for the Social Sciences (SPSS, Chicago, IL). Data were considered significant at $P < .05$.

3. Results

The clinical and anthropometric characteristics of hirsute patients with PCOS or IH are summarized in Table 1. The 2

Table 3
Comparison of skinfold thickness and DXA in hirsute patients with or without PCOS

Variable	PCOS (n = 24)	IH (n = 13)	*P*
Skinfold thickness total body fat (%)	34 ± 1	29 ± 2	.050
Sum of trunk skinfolds (mm)	144 ± 7	119 ± 9	.034
DXA trunk fat (kg)	17 ± 1	14 ± 1	.038
Total DXA (kg)	36 ± 2	30 ± 3	.150
DXA total fat (%)	41 ± 1	40 ± 1	.510
Waist circumference (cm)	96 ± 2.5	86 ± 2.9	.016

Values are expressed as mean ± SEM (Student *t* test).

groups were similar in terms of age, severity of hirsutism, and blood pressure, but WHR was higher in the group with PCOS. Patients with IR constituted 67% of the PCOS group and 30% of the group with IH. Ten patients had impaired glucose tolerance (7 in the group with PCOS and 3 in the group with IH).

Table 2 shows hormonal and metabolic data of hirsute patients. As expected, the PCOS group presented higher androgen levels. HOMA and insulin levels were also higher in the PCOS than in the IH group. In addition, HOMA index was positively correlated with total testosterone concentrations ($r = 0.408$, $P = .010$), as well as with triglycerides ($r = 0.328$, $P = .041$), total cholesterol ($r = 0.417$, $P = .008$), and LDL-C ($r = 0.473$; $P = .002$). On the other hand, a negative correlation was observed between HOMA and SHBG levels ($r = -0.401$, $P = .012$).

Data concerning body fat composition are presented in Table 3. Patients with PCOS had significantly higher values of sum of trunk skinfolds, DXA trunk fat, and waist circumference measurements than those with IH. Positive correlations were found between HOMA and truncal adiposity as determined by the sum of trunk skinfolds (millimeters) ($r = 0.466$, $P = .003$), DXA trunk fat (kilograms) ($r = 0.412$, $P = .01$), and waist circumference (centimeters) ($r = 0.334$, $P = .038$).

Waist circumference presented significant correlations with both skinfold thickness ($r = 0.870$, $P \leq .001$) and DXA measurements ($r = 0.818$, $P \leq .001$). In addition, the sum of skinfold thickness was positively correlated with DXA trunk fat measurements ($r = 0.863$, $P = .001$). Waist circumference also presented the same magnitude of positive and significant correlations with DXA trunk fat measurements ($r = 0.947$, $P = .0001$).

HOMA was found to be significantly associated with waist ($r = 0.441$, $P = .007$) and with FAI ($r = 0.430$, $P = .010$) (Fig. 1) and this association remained significant even after adjustment for waist circumference ($r = 0.440$, $P = .009$).

4. Discussion

In the present study, hirsute patients with PCOS had a higher percentage of truncal adiposity, greater testosterone levels, and free androgen index than women of similar age and degree of hirsutism without PCOS. Moreover, a positive correlation was found between HOMA index and androgens, independent of central adiposity, indicating that hyperandrogenism in PCOS may be additive to elevated waist circumference in increasing risk of IR and seems to be an independent predictor of IR. These findings are also

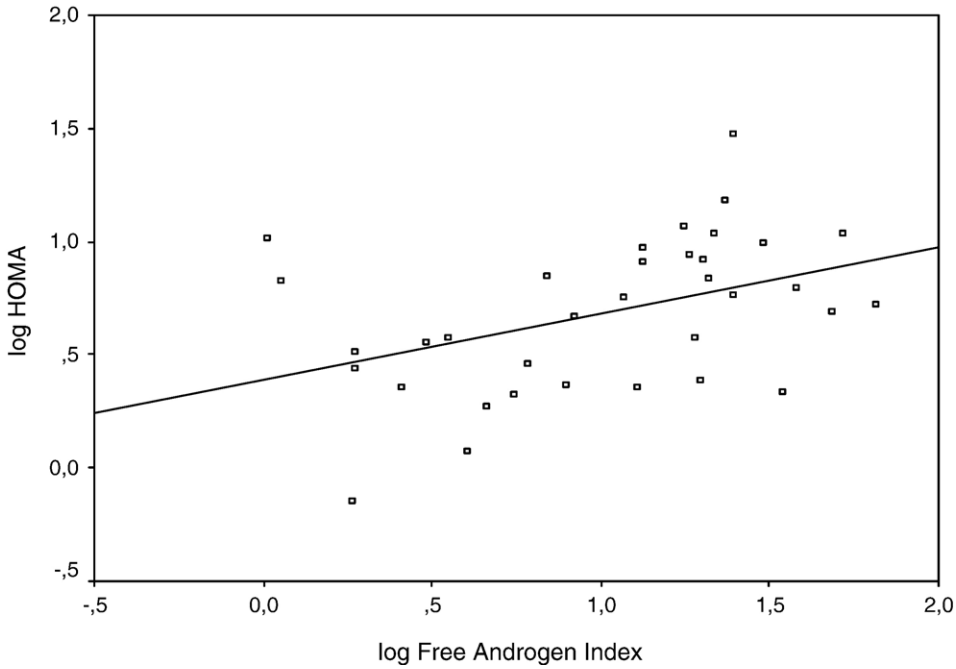

Fig. 1. Correlation between HOMA and FAI in hirsute patients (Pearson correlation test). $r = 0.440$, $P = .009$, adjusted for waist circumference.

consistent with our previous observations on the association of IR and androgenicity in pre-, peri-, and postmenopausal women with no evidence of clinical disease [7,33,34].

Waist circumference, the sum of trunk skinfolds, and DXA trunk fat were higher in our patients with PCOS. Previous studies have shown that a greater accumulation of abdominal adipose tissue is associated with glucose intolerance and hyperinsulinemia resulting from IR [18]. The pattern of fat deposition in obese subjects might be influenced by the relative amounts of androgenic and estrogenic sex hormones. Women with greater WHR seem to have higher total androgen levels [6,35]. Associations between androgens and abdominal fat or hyperinsulinemia in postmenopausal women have been previously described [33,34,36]. Obesity, particularly that of abdominal distribution, can directly affect the severity of hyperandrogenism in women with PCOS by reducing SHBG levels and increasing free androgen levels [11,37]. Moreover, hyperandrogenism by itself can favor IR and the simultaneous development of abdominal obesity in patients with PCOS [9,10].

Dual-energy x-ray absorptiometry is a well-accepted method for evaluating body composition, presenting high precision and simplicity. Whereas CT can well identify both subcutaneous and visceral adipose compartments, DXA may be used to measure total abdominal adiposity and to validate other methods of measuring body fat [5]. Evidence from different studies shows significant correlations between skinfold thickness and DXA in several groups of subjects [38-40]. Warner et al [41] have shown an excellent correlation of abdominal and thigh skinfold thickness with DXA in female athletes. A similar correlation between body fat and DXA has been reported in adolescent girls with type 1 diabetes mellitus [42]. We observed a strong correlation between DXA trunk fat measurements and both waist circumference and sum of specific trunk skinfolds.

From a clinical point of view, estimating regional fat distribution is easy and cheap, and thus, anthropometric measurements may be preferred over imaging methods. In this sense, the proposed sum of trunk skinfolds as an index of truncal adiposity could be an advantageous option. However, it is important to stress that the validity and reliability of skinfold measures are directly related to the individual ability of the appraiser, type of caliper, and individual characteristics (race, presence of obesity, age) [43]. It is also well known that the possibility for interobserver differences increases with the number of measurements. In turn, waist circumference is a simple and single measurement that showed a strong correlation with DXA trunk fat measurement when applied to our hirsute patients. Moreover, although skinfolds directly measure only subcutaneous tissue, waist circumference reflects both subcutaneous and deeper tissue masses.

In conclusion, the present results indicate that (1) hirsutism per se appears not to be a risk for IR and related cardiovascular disease unless there is presence of central adiposity and/or abnormal androgen profile; (2) waist circumference and the sum of trunk skinfold measurements represent accurate methods for estimating truncal adiposity, but waist circumference measurement seems to be the simplest method of clinical screening for IR in subpopulations at higher metabolic and cardiovascular risk, such as women with PCOS.

Acknowledgment

This study was supported by grants from Conselho Nacional de Desenvolvimento Científico e Tecnológico and PRONEX 26/98 (Programa de Apoio aos Núcleos de Excelência em Pesquisa).

References

[1] National Institutes of Health, National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation and treatment of overweight and obesity in adults. The evidence report. Bethesda: NIH, NHLBI; 1998.
[2] Bjorntorp P. Body fat distribution, insulin resistance, and metabolic diseases. Nutrition 1997;13:795-803.
[3] World Health Organization. Obesity: preventing and managing the global epidemic. Report of WHO consultation. Geneva: WHO; 1998.
[4] Després JP, Lemieux I, Prud'homme D. Treatment of obesity: need to focus on high risk abdominally obese patients. Clin Rev 2001;3:716-20.
[5] Bosello O, Zamboni M. Visceral obesity and metabolic syndrome. Obes Rev 2000;1:47-56.
[6] Mayes JS, Watson GH. Direct effects of sex steroid hormones on adipose tissues and obesity. Obes Rev 2004;5:197-216.
[7] Donato GB, Fuchs SC, Oppermann K, Bastos C, Spritzer PM. Menopausal status is associated with central adiposity measured at different cut-offs of waist circumference and waist-to-hip ratio. Menopause 2006;13:280-5.
[8] Spritzer PM, Poy M, Wiltgen D, Mylius LS, Capp E. Leptin concentrations in hirsute women with polycystic ovary syndrome or idiopathic hirsutism: influence on LH and relationship with hormonal, metabolic, and anthropometric measurements. Hum Reprod 2001;16:1340-6.
[9] Ehrmann DA. Polycystic ovary syndrome. N Engl J Med 2005;352: 223-36.
[10] Ek I, Arner P, Ryden M, Holm C, et al. A unique defect in the regulation of visceral fat cell lipolysis in the polycystic ovary syndrome as an early link to insulin resistance. Diabetes 2002;51:484-92.
[11] Puder JJ, Varga S, Kraenzlin M, De Geyter C, Keller U, Muller B. Central fat excess in polycystic ovary syndrome: relation to low-grade inflammation and insulin resistance. J Clin Endocrinol Metab 2005;90:6014-21.
[12] Rosenfield RL. Hirsutism. N Engl J Med 2005;353:2578-88.
[13] Oliveira IO, Lhullier C, Brum IS, Spritzer PM. Gene expression of type 2 17beta-hydroxysteriod dehydrogenase in scalp hairs of hirsute women. Steroids 2003;68:641-9.
[14] Rice CL, Cunningham DA, Paterson DH, Lefcoe MS. Arm and leg composition determined by computed tomography in young and elderly men. Clin Physiol 1989;9:207-20.
[15] Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. Arch Intern Med 2002;162:2074-9.
[16] Kamel EG, McNeill G, Van Wijk MCW. Usefulness of anthropometry and DXA in predicting intra-abdominal fat in obese men and women. Obes Res 2000;8:36-42.
[17] Dobbelsteyn CJ, Joffres MR, MacLean DR, Flowerdew G. A comparative of waist circumference, waist-to-hip ratio and body

mass index as indicators of cardiovascular risk. Int J Obes Relat Metab Disord 2001;25:652-61.
[18] Pouliot MC, Despres JP, Lemieux S, et al. Waist circumference and abdominal sagittal diameter: best simple anthropometric indices of abdominal visceral tissue accumulation and related cardiovascular risk in men and women. Am J Cardiol 1994;74:460-8.
[19] Ham TS, van Leer EM, Seidell JC, Lean MEJ. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. BMJ 1995;311:1401-5.
[20] Glickman SG, Marn CS, Supiano MA, Dengel DR. Validity and reliability of dual-energy X-ray absorptiometry for the assessment of abdominal adiposity. J Appl Physiol 2004;97:509-14.
[21] Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among U.S. adults: the National Health and Nutrition Examination Surveys, 1960 to 1991. J Am Med Assoc 1994;272:205-11.
[22] Velde SJ, Twisk JWR, van Mechelen W, Kemper HCG. Birth weight, adult body composition, and subcutaneous fat distribution. Obes Res 2003;11:202-8.
[23] Marcus MA, Murphy L, Pi-Sunyer FX, Albu JB. Insulin sensitivity and serum triglyceride level in obese white and black women: relationship to visceral and truncal subcutaneous fat. Metabolism 1999;48:194-9.
[24] Spritzer PM, Billaud L, Thalabard JC, et al. Cytoproterone acetate versus hydrocortisone treatment in late-onset adrenal hyperplasia. J Clin Endocrinol Metab 1990;70:642-6.
[25] Azziz R, Dewailly D, Owerbach D. Nonclassic adrenal hyperplasia: current concepts. J Clin Endocrinol Metab 1994;78:810-5.
[26] Comim FV, Spritzer PM. Increased growth hormone response to clonidine in nonobese normoinsulinemic patients with polycystic ovary syndrome. Fertil Steril 2004;81:108-13.
[27] Mather KJ, Kwan F, Corenblum B. Hyperinsulinemia in polycystic ovary syndrome correlates with increased cardiovascular risk independent of obesity. Fertil Steril 2000;73:150-6.
[28] World Health Organization. Physical status: the use and interpretation of anthropometry. Report of a WHO expert committee. World Health Organ Tech Rep Ser 1995;854:1-452.
[29] Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 1961;21:1140-8.
[30] World Heath Organization. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: report of a WHO consultation: diagnosis and classification of diabetes mellitus. Geneva: WHO; 1999.
[31] Ascaso JF, Romero P, Real JT, Priego A, Valdecabres C, Carmena R. Insulin resistance quantification by fasting insulin plasma values and HOMA index in a non-diabetic population. Med Clin (Barc) 2001;117:530-3.
[32] Faulkner JA. Physiology of swimming and diving. In: Falls H, editor. Exercise physiology. Baltimore: Academic Press; 1968. p. 415-45.
[33] Maturana MA, Spritzer PM. Association between hyperinsulinemia and endogenous androgen levels in peri- and postmenopausal women. Metabolism 2002;51:238-43.
[34] Oppermann K, Kohek MB, Fuchs SC, Spritzer PM. Association between hyperinsulinemia, endogenous androgens, and endometrial thickness in pre- and perimenopausal women: a population-based study. Gynecol Endocrinol 2004;18:269.
[35] Gambineri A, Pelusi C, Vicennati V, Pagotto U, Pasquali R. Obesity and the polycystic ovary syndrome. Int J Obes Relat Metab Disord 2002;26:883-96.
[36] Toth MJ, Tchernof A, Sites CK, Poehlman ET. Effect of menopausal status on body composition and abdominal fat distribution. Int J Obes Relat Metab Disord 2000;24:226-31.
[37] Legro RS, Kunselman AR, Dodson WC, Dunaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. J Clin Endocrinol Metab 1999;84:165-9.
[38] Woodrow G, Oldroyd B, Smith MA, Turney JH. Measurement of body composition in chronic renal failure: comparison of skinfold anthropometry and bioelectrical impedance with dual energy X-ray absorptiometry. Eur J Clin Nutr 1996;50:295-301.
[39] Stall SH, Ginsberg NS, De Vita MV, et al. Comparison of five body-composition methods in peritoneal dialysis patients. Am J Clin Nutr 1996;64:125-30.
[40] Evans EM, Saunders MJ, Spano MA, Arngrimsson SA, Lewis RD, Cureton KJ. Body-composition changes with diet and exercise in obese women: a comparison of estimates from clinical methods and a 4-component model. Am J Clin Nutr 1999;70:5-12.
[41] Warner ER, Fornetti WC, Jallo JJ, Pivarnik JM. A skinfold model to predict fat-free mass in female athletes. J Athl Train 2004;39:259-62.
[42] Ingberg CM, Sarnblad S, Palmer M, Schvarcz E, Berne C, Aman J. Body composition in adolescent girls with type 1 diabetes. Diabet Med 2003;20:1005-11.
[43] Lohman TG, Pollock ML, Slaughter MH, Brandon LJ, Broileau RA. Methodological factors and the prediction of body fat in female athletes. Med Sci Sports Exerc 1984;16:92-6.