



Polycystic Ovarian Syndrome in Taiwanese Women

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Polycystic ovary syndrome (PCOS) is an extremely common disorder in women of reproductive age. Diagnosis of PCOS is principally based on clinical and physical findings. Diagnostic criteria and PCOS definitions used by clinicians and researchers are almost as heterogeneous as the syndrome. Of those diagnosed with PCOS using the 2003 Rotterdam criteria, 61% fulfilled the 1990 National Institutes of Health criteria for unexplained hyperandrogenic chronic anovulation. Patient populations with the new phenotypes have less severe ovulatory dysfunction and less androgen excess than patients diagnosed using the 1990 National Institutes of Health criteria. These findings might be common across all female populations with PCOS, in Asian or Western countries. Data for clinical hyperandrogenism indicates that the prevalence of hirsutism in Taiwanese PCOS women is lower than that for Caucasian/Western women. The extent of metabolic abnormalities in women with PCOS may vary with phenotype, age and ethnicity. Obesity represents a major risk factor for metabolic syndrome and insulin resistance. Approximately 40–50% of all women with PCOS are overweight or obese. Obese subjects with PCOS have a higher risk of developing oligomenorrhea, amenorrhea and biochemical hyperandrogenemia than non-obese women with PCOS. Moreover, obese women with PCOS have significantly more severe insulin resistance, lower serum luteinizing hormone levels, and lower luteinizing hormone to follicle-stimulating hormone ratios than those of non-obese women with PCOS. PCOS women in Taiwan have a higher luteinizing hormone to follicle-stimulating hormone ratio and lower insulin resistance than those in PCOS women in Western countries. However, the average body mass index is significantly lower in Taiwanese PCOS women than in Western women, which might partially explain the difference between these two populations in terms of clinical and biochemical presentations. To further document the ethnic variation between women with PCOS in Taiwan and Western countries, future studies should pay attention to the effect of obesity on the diagnosis and clinical presentations of PCOS-related syndromes.

1. Introduction

Polycystic ovary syndrome (PCOS), which is an extremely common disorder in women of reproductive age, is a heterogeneous collection of signs and symptoms that,

gathered together, form a spectrum of a disorder. Investigating PCOS is crucial as it is now considered to increase metabolic and cardiovascular risk, thereby impacting the health of women to a much greater extent than previously thought. Diagnosis of PCOS is principally

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based on clinical and physical findings. The first comprehensive definition arose from a conference sponsored by the National Institutes of Health–National Institute for Child and Human Development (NIH-NICHD) in April 1990.¹ The recommended NIH diagnostic criteria are as follows: clinical or biochemical evidence of hyperandrogenism, chronic anovulation and exclusion of other known disorders. However, most gynecologists are more likely to use morphological analysis of ovaries by ultrasonography to facilitate the diagnosis in clinical practice. Therefore, redefining PCOS to incorporate an appropriate definition of a polycystic ovary is required.² In 2003, new guidelines for diagnosing PCOS were suggested at a joint meeting of the European Society for Human Reproduction and the American Society of Reproductive Medicine. A diagnosis of PCOS can be reached when at least two of three criteria are met: hyperandrogenism and/or hyperandrogenemia (HA); oligomenorrhea or amenorrhea, or anovulation (oligo-an); polycystic ovary morphology (PCOM); and the exclusion of any androgen excess disorders.³ The Rotterdam 2003 criteria have expanded rather than replaced the 1990 NIH criteria. These new diagnostic criteria have added the following two PCOM phenotypes:¹ (1) PCOM with HA and without oligo-an;² and (2) PCOM with oligo-an and without HA.⁴ However, The Androgen Excess and PCOS Society recently suggested that PCOS should be defined by the presence of hyperandrogenism (clinical and/or biochemical), ovarian dysfunction (oligo-an and/or polycystic ovaries), and the exclusion of related disorders.⁵ The prevalence of PCOM in women of reproductive age has been reported to be 22–33%.^{2,6} PCOS diagnosed using the 1990 NIH criteria occurs in roughly 6–8% of women of reproductive age.⁷ The prevalence and clinical manifestations of PCOS should be reevaluated after applying these new diagnostic criteria. Moreover, ethnic differences in clinical presentations of PCOS have been demonstrated in several studies.^{8–11}

According to the three diagnostic criteria for PCOS, all women can be classified into three subgroups: (1) healthy women who have a normal ovary appearance, do not have oligomenorrhea or amenorrhea, and have no clinical or biochemical presentations of androgen excess; (2) PCOS risk factor-positive (PCOS-rf) women who have only one of three PCOS components: (i) PCOM only, (ii) oligo-an only, and (iii) HA only; (3) PCOS women who present with at least two of the following criteria: (i) PCOM, (ii) oligo-an, or (iii) HA (Figure 1).³

Both healthy control and PCOS-rf groups belong to the group of non-PCOS subjects. Our previous study showed that disturbances caused by several clinical presentations in PCOS-rf women were less severe than in women with PCOS but were still significantly different from those in healthy control women.¹²

Using the Rotterdam 2003 criteria, four PCOS phenotypes can be classified as NIH subgroups and non-NIH subgroups as follows.

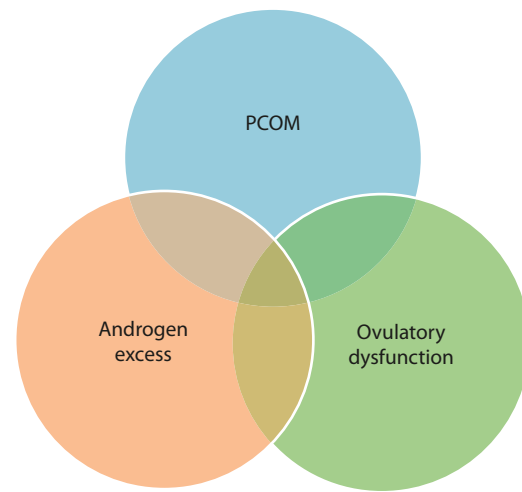


Figure 1 Diagnostic criteria and phenotypes of polycystic ovary syndrome.

A. NIH subgroup

- (HA + oligo-an type): hyperandrogenism and/or hyperandrogenemia and oligomenorrhea/amenorrhea without polycystic ovary morphology; this phenotype is PCOS without PCOM.
- (HA + oligo-an + PCOM type): hyperandrogenism and/or hyperandrogenemia and oligomenorrhea/amenorrhea with polycystic ovary morphology; this phenotype is PCOS with all three diagnostic components.

B. Non-NIH subgroup

- (HA + PCOM type): hyperandrogenism and/or hyperandrogenemia and polycystic ovary morphology without oligomenorrhea/amenorrhea; this phenotype is PCOS without oligo-an.
- (Oligo-an + PCOM type): oligomenorrhea/amenorrhea and polycystic ovary morphology without hyperandrogenism and/or hyperandrogenemia; this phenotype is PCOS without HA.

Compared with the NIH 1990 criteria, polycystic ovary morphology is a new diagnostic criterion. In a study of Taiwanese women using the 2003 Rotterdam criteria, our results showed that of those women diagnosed with PCOS, 61% fulfilled the 1990 NIH criteria for unexplained hyperandrogenic chronic anovulation.⁸ The percentages of patients with the two newly extended phenotypes were as follows: 18% had PCOS without HA and 21% had PCOS without oligo-an.⁸ However, the patient populations with the new phenotypes had less severe ovulatory dysfunction and less androgen excess than patients diagnosed using the 1990 NIH criteria. The prevalence of PCOM has been suggested to be higher than 20% in both Asian and Western women.^{13,14} PCOM is the most common PCOS component in patients; 91% of patients diagnosed with the Rotterdam criteria and 85% diagnosed with the NIH criteria have PCOM.

For clinical presentations, excess androgen has a principal role in diagnostic criteria. It has been demonstrated that PCOS without excess androgen comprises one of the least severe phenotypes.^{5,8} Chae et al's study of Korean women found that PCOS without HA is a common phenotype in Korea and these women are less likely to have metabolic dysfunction, insulin resistance and elevated blood pressure.⁹ Our study and previous reports support the proposal that excess androgen is the condition directly responsible for the signs and symptoms that are recognized as PCOS.^{5,8,9} Therefore, we can summarize the severity in clinical and biochemical syndromes of PCOS-related disturbances in women using various inclusion criteria as shown in Figure 2.

2. Clinical Presentation of Hyperandrogenism in Taiwanese Women With PCOS

The most apparent signs of androgen excess are external manifestations, including oily skin, acne, hirsutism, android obesity, and androgenic alopecia. Hirsutism is not only a function of circulating androgen levels, it can also be determined by genetic factors. Only 28–35% of Asian women with PCOS have hirsutism.^{8,15} DeUgarte et al applied the 1990 NIH diagnostic criteria to investigate 271 PCOS patients with average modified Ferriman-Gallwey (mF-G) scores of 8.0 and determined that 77%

had hirsutism.¹⁶ We utilized the same criteria (NIH diagnostic criteria) and a similar scoring system to evaluate 95 Taiwanese women, and found that 35% had hirsutism and average mF-G scores of 4.4. Although 53% of PCOS patients under the Rotterdam 2003 criteria have excess clinical androgen, the incidence of hirsutism (28%) was less than that for acne (35%) in our study of Taiwanese women.⁸

To study and document the clinical presentation of hyperandrogenism in Taiwanese women, we recently reported a study of 627 women in the outpatient clinic of Wan-Fang Hospital.¹⁷ Of the 627 women studied, 55% (346/627) had PCOS, 33% (210/627) had hyperandrogenemia, 38% (240/627) had acne, 19% (121/627) had hirsutism, and 34% (213/627) were obese. Our survey showed that women suffering from obesity had a higher incidence of PCOS (64% vs. 51%) and hyperandrogenemia (51% vs. 25%), a lower incidence of acne (31% vs. 42%), and a higher serum total testosterone than non-obese subjects. Even though obese women had significantly higher serum total testosterone levels, they still had a lower incidence of acne than non-obese women, and this was found uniformly in PCOS, non-PCOS, and hyperandrogenemic and non-hyperandrogenemic subjects.¹⁷

The effect of hyperandrogenemia on cutaneous manifestations, e.g., acne, might be alleviated by obesity. This phenomenon could be explained by aromatase activity in adipose tissue. The increase in adipose tissue



Figure 2 Diagram of the severity in clinical and biochemical syndromes of polycystic ovary syndrome-related disturbance in women using various inclusion criteria.

is associated with an increase in the enzyme aromatase.¹⁸ Aromatase converts testosterone to estradiol, and is localized to sebaceous glands and to both outer as well as inner root sheath cells of anagen terminal hair follicles. Aromatase may play a “detoxifying” role by removing excess androgens.¹⁹ Estrogens may act by several mechanisms that negatively influence sebaceous gland growth or lipid production.²⁰ Therefore, obese women might present with fewer clinical manifestations of hyperandrogenism than non-obese women.

3. Inappropriate Gonadotropin Secretion in Taiwanese Women With PCOS

Inappropriate gonadotropin secretion, especially increased mean luteinizing hormone (LH) levels, is a very common finding in women with PCOS.²¹ Although LH levels and the LH to follicle-stimulating hormone (FSH) ratio are not required for diagnosis in Rotterdam criteria,¹ they are still major criteria in the diagnosis of PCOS in Japanese women. Disturbance of the hypothalamic-pituitary-ovarian axis is still thought to have a key etiologic role in PCOS patients.²²

To evaluate the predictive value of PCOS diagnostic criteria, we performed a receiver operating characteristic (ROC) curve analysis and showed that the area under the ROC curve for the LH to FSH ratio is similar to the areas under the ROC curve for total testosterone and for average ovarian volume.¹² Because total testosterone serum levels and average ovarian volume are both used as inclusion criteria for PCOS diagnosis, the LH to FSH ratio might be a useful marker of PCOS in Taiwanese women.¹² We further evaluated the role of the LH to FSH ratio in patients with oligomenorrhea or anovulation by using ROC analysis. For the diagnosis of oligomenorrhea or anovulation, the LH to FSH ratio appears to have better prediction power than total testosterone and average ovarian volume.¹²

To set the threshold of the LH to FSH ratio for identifying women with PCOS, poor sensitivity is the most critical component in diagnosis.²³ Our results showed a sensitivity of 13% and specificity of 96% at an LH-FSH ratio of >3 , and 35% and 90%, respectively, at a ratio of >2 .¹² The decision thresholds of the LH to FSH ratio in PCOS diagnosis shows that an LH to FSH ratio of >1 results in the best combination of sensitivity (69%) and specificity (80%).¹² Therefore, we propose that the threshold LH to FSH ratio could be set as >1 for the best combination of sensitivity and specificity for the diagnosis of both PCOS and oligomenorrhea or amenorrhea.

4. PCOM in East Asian Women With PCOS

Classically, PCOM has been described as enlarged ovaries containing numerous peripheral antral follicles with

increased ovarian volume. The ultrasound appearance of PCOM has been recently codified as ≥ 12 follicles in either ovary measuring 2–9 mm in diameter, and/or increased ovarian volume of >7 mL.²⁴ However, Chen et al studied 432 Chinese women with PCOS using NIH criteria and compared them with 153 age-matched women as controls, and found that the thresholds for PCOS were 6.4 cm³ for mean ovarian volume and 10 for mean follicle number.²⁵ Both mean ovarian volume and mean follicle number of Asians might be different compared with Western PCOS women.²⁵ Women with PCOM only have a significantly lower ovarian volume, serum LH, testosterone and dehydroepiandrosterone sulfate levels but higher serum sex hormone-binding globulin levels compared with PCOS women; PCOM only represents a milder end of the PCOS spectrum.²⁶ Chen et al's study in Taiwanese women reported that the number of antral follicles and ovarian volume showed a significant correlation with anti-müllerian hormone, total testosterone and the free androgen index; and anti-müllerian hormone, body mass index (BMI) and total testosterone were independently related to the number of antral follicles.²⁷

5. Insulin Resistance and Glucose Intolerance in Taiwanese Women With PCOS

Shi et al reported that 1040 Chinese women with PCOS had an impaired glucose tolerance (IGT) prevalence of 17.2% and a type 2 diabetes prevalence of 16.6%.²⁸ Chen et al evaluated 102 PCOS patients and reported that the prevalence of IGT was 20.5% and that of non-insulin-dependent diabetes mellitus was 1.9%.²⁹ Lee et al reported that in Korean women with PCOS, the prevalence of IGT was 17.0% and the prevalence of type 2 diabetes was 1.0%.³⁰ Wei et al reported 356 Taiwanese with PCOS and 974 control infertility patients and found that the prevalence of IGT was 7.6% and diabetes mellitus was 3.1% in patients with PCOS compared with 2.9% and 0.2% in the control group, respectively.³¹ Legro et al studied 254 women with PCOS and found that the IGT prevalence was 31.1% and the type 2 diabetes prevalence was 7.5%.³² Chinese PCOS women have a lower prevalence of IGT than American PCOS women.^{28,29,31,32} Cheung et al reported that the prevalence of metabolic syndrome in Hong Kong Chinese women with PCOS increased from 16.7% at under 30 years of age to 53.3% at over 40 years.³³ Women with PCOS have a fivefold increase in the risk of developing metabolic syndrome compared with women without PCOS, even after controlling for age and BMI, suggesting that PCOS alone is an independent risk factor for metabolic syndrome.³³ Chen et al reported that the free androgen index, total testosterone and sex hormone-binding globulin levels are significantly correlated with

both systolic blood pressure and diastolic blood pressure in Taiwanese women with PCOS.³⁴

6. Obesity and PCOS in Taiwanese Women

Obesity is a prominent feature of PCOS, occurring in 40–50% of those patients.^{8,35} Obesity appears to exert an additive, synergistic impact on the manifestations of PCOS, independently and negatively affecting insulin sensitivity, risk of diabetes and cardiovascular profile.^{36,37} The prevalence of obesity in PCOS patients is greater than that of the general female population. Conversely, the prevalence of PCOS is greater in overweight and obese women than in lean women.³⁸ Obesity worsens both biochemical hyperandrogenemia and chronic anovulation, which are the two most important diagnostic criteria of PCOS.³⁸

In a study of women with PCOS, BMI had a significantly positive correlation with serum total testosterone and a negative correlation with serum LH.³⁵ This study also found that obese subjects with PCOS had a higher risk of developing oligomenorrhea [odds ratio (OR), 2.2; 95% confidence interval (CI), 1.3–3.7] and biochemical hyperandrogenemia (OR, 2.6; 95% CI, 1.6–4.2) than non-obese women with PCOS.³⁵ Moreover, obese women with PCOS have significantly higher serum total testosterone levels, and more prolonged menstrual intervals than non-obese women with PCOS. Notably, obese women with PCOS present with less acne than non-obese subjects (OR, 0.5; 95% CI, 0.3–0.9).³⁸ We recently studied 273 women in four phenotypes of PCOS and found that the insulin sensitivity index and the percentage of insulin resistance were not significantly different among discrete phenotypes of PCOS groups and normal controls. However, obesity (OR, 14.0; 95% CI, 7.5–26.5) results in a higher risk for developing insulin resistance than hyperandrogenemia (OR, 2.1; 95% CI, 1.3–3.6), oligomenorrhea (OR, 1.8; 95% CI, 1.0–3.3), and PCOM (OR, 1.4; 95% CI, 0.8–2.7).³⁹ Compared with hyperandrogenemia, chronic anovulation, and PCOM, obesity is the most important risk factor in determining insulin resistance in women with PCOS. However, the average BMI is significantly lower in Taiwanese PCOS women than in Western women, which might partially explain the difference in clinical and biochemical presentations in women between East Asia and Western countries.^{8,17,39,40}

7. Summary

The following points are summarized from this review. (1) PCOM is the most frequently used criterion in PCOS diagnosis. PCOS without androgen excess may be a mild phenotype of PCOS. (2) East Asian women with PCOS have a smaller mean ovarian volume and fewer mean

follicle numbers than Western women. (3) Hirsutism has a lower prevalence in Taiwanese women with PCOS than in Western women. (4) The LH to FSH ratio is a valuable diagnostic tool for evaluating Taiwanese women with PCOS, especially in the diagnosis of oligomenorrhea or anovulation. (5) Obese subjects with PCOS have a higher risk of developing oligomenorrhea, amenorrhea, biochemical HA, insulin resistance, and lower LH to FSH ratios than non-obese women with PCOS. (6) Taiwanese women with PCOS have a lower BMI and lower prevalence of IGT than Western women with PCOS.

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References

1. Zawadzki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: toward a rational approach. In: Dunaif A, Givens JR, Haseline F, eds. *Polycystic Ovary Syndrome*. Boston: Blackwell Scientific, 1992:377–84.
2. Balen A, Michelmore K. What is polycystic ovary syndrome? Are national views important? *Hum Reprod* 2002;17:2219–27.
3. The Rotterdam ESHRE/ASRM-sponsored PCOS workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril* 2004;81:19–25.
4. Azziz R. Controversy in clinical endocrinology: diagnosis of polycystic ovarian syndrome: the Rotterdam criteria are premature. *J Clin Endocrinol Metab* 2006;91:781–5.
5. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, Janssen OE, et al. Task Force on the Phenotype of the Polycystic Ovary Syndrome of The Androgen Excess and PCOS Society. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. *Fertil Steril* 2009;91:456–88.
6. Clayton RN, Ogden V, Hodgkinson J. How common are polycystic ovaries in normal women and what is their significance for the fertility of the population? *Clin Endocrinol (Oxf)* 1992;37:127–34.
7. Azziz R, Woods KS, Reyna R, Key TJ, Knochenhauer ES, Yildiz BO. The prevalence and features of the polycystic ovary syndrome in an unselected population. *J Clin Endocrinol Metab* 2004;89:2745–9.
8. Hsu MI, Liou TH, Chou SY, Chang CY, Hsu CS. Diagnostic criteria for polycystic ovary syndrome in Taiwanese Chinese women: comparison between Rotterdam 2003 and NIH 1990. *Fertil Steril* 2007;88:727–9.
9. Chae SJ, Kim JJ, Choi YM, Hwang KR, Jee BC, Ku SY, Suh CS, et al. Clinical and biochemical characteristics of polycystic ovary syndrome in Korean women. *Hum Reprod* 2008;23:1924–31.
10. Carmina E, Koyama T, Chang L, Stanczyk FZ, Lobo RA. Does ethnicity influence the prevalence of adrenal hyperandrogenism and insulin resistance in polycystic ovary syndrome? *Am J Obstet Gynecol* 1992;167:1807–12.
11. Ewing JA, Rouse BA. Hirsutism, race and testosterone levels: comparison of East Asians and Euroamericans. *Hum Biol* 1978;50:209–15.

12. Hsu MI, Liou TH, Liang SJ, Su HW, Wu CH, Hsu CS. Inappropriate gonadotropin secretion in polycystic ovary syndrome. *Fertil Steril* 2009;91:1168–74.
13. Hart R, Hickey M, Franks S. Definitions, prevalence and symptoms of polycystic ovaries and polycystic ovary syndrome. *Best Pract Res Clin Obstet Gynaecol* 2004;18:671–83.
14. Chan CC, Ng EH, Tang OS, Lee CP, Ho PC. The prevalence of polycystic ovaries in Chinese women with a history of gestational diabetes mellitus. *Gynecol Endocrinol* 2006;22:516–20.
15. Lam PM, Ma RC, Cheung LP, Chow CC, Chan JC, Haines CJ. Polycystic ovarian syndrome in Hong Kong Chinese women: patient characteristics and diagnostic criteria. *Hong Kong Med J* 2005;11:336–41.
16. DeUgarte CM, Bartolucci AA, Azziz R. Prevalence of insulin resistance in the polycystic ovary syndrome using the homeostasis model assessment. *Fertil Steril* 2005;83:1454–60.
17. Yang JH, Weng SL, Lee CY, Chou SY, Hsu CS, Hsu MI. A comparative study of cutaneous manifestations of hyperandrogenism in obese and non-obese Taiwanese women. *Arch Gynecol Obstet* 2010 Apr 30. [Epub ahead of print]
18. Cohen PG. Aromatase, adiposity, aging and disease. The hypogonadal-metabolic-atherogenic-disease and aging connection. *Med Hypotheses* 2001;56:702–8.
19. Chen W, Thiboutot D, Zouboulis CC. Cutaneous androgen metabolism: basic research and clinical perspectives. *J Invest Dermatol* 2002;119:992–1007.
20. Thiboutot D. Acne: hormonal concepts and therapy. *Clin Dermatol* 2004;22:419–28.
21. Taylor AE. Gonadotropin dysfunction in women with polycystic ovary syndrome. *Fertil Steril* 2006;86(Suppl 1):S12.
22. Taylor AE, McCourt B, Martin KA, Anderson EJ, Adams JM, Schoenfeld D. Determinants of abnormal gonadotropin secretion in clinically defined women with polycystic ovary syndrome. *J Clin Endocrinol Metab* 1997;82:2248–56.
23. Cho LW, Jayagopal V, Kilpatrick ES, Holding S, Atkin SL. The LH/FSH ratio has little use in diagnosing polycystic ovarian syndrome. *Ann Clin Biochem* 2006;43:217–9.
24. Jonard S, Robert Y, Dewailly D. Revisiting the ovarian volume as a diagnostic criterion for polycystic ovaries. *Hum Reprod* 2005;20:2893–8.
25. Chen Y, Li L, Chen X, Zhang Q, Wang W, Li Y, Yang D. Ovarian volume and follicle number in the diagnosis of polycystic ovary syndrome in Chinese women. *Ultrasound Obstet Gynecol* 2008;32:700–3.
26. Ng EH, Chan CC, Ho PC. Are there differences in ultrasound parameters between Chinese women with polycystic ovaries only and with polycystic ovary syndrome? *Eur J Obstet Gynecol Reprod Biol* 2006;125:92–8.
27. Chen MJ, Yang WS, Chen CL, Wu MY, Yang YS, Ho HN. The relationship between anti-Müllerian hormone, androgen and insulin resistance on the number of antral follicles in women with polycystic ovary syndrome. *Hum Reprod* 2008;23:952–7.
28. Shi Y, Guo M, Yan J, Sun W, Zhang X, Geng L, Xu L, et al. Analysis of clinical characteristics in large-scale Chinese women with polycystic ovary syndrome. *Neuro Endocrinol Lett* 2007;28:807–10.
29. Chen X, Yang D, Li L, Feng S, Wang L. Abnormal glucose tolerance in Chinese women with polycystic ovary syndrome. *Hum Reprod* 2006;21:2027–32.
30. Lee H, Oh JY, Sung YA, Chung H, Cho WY. The prevalence and risk factors for glucose intolerance in young Korean women with polycystic ovary syndrome. *Endocrine* 2009;36:326–32.
31. Wei HJ, Young R, Kuo IL, Liaw CM, Chiang HS, Yeh CY. Prevalence of insulin resistance and determination of risk factors for glucose intolerance in polycystic ovary syndrome: a cross-sectional study of Chinese infertility patients. *Fertil Steril* 2009;91:1864–8.
32. Legro RS, Kusanman 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.
33. Cheung LP, Ma RC, Lam PM, Lok IH, Haines CJ, So WY, Tong PC, et al. Cardiovascular risks and metabolic syndrome in Hong Kong Chinese women with polycystic ovary syndrome. *Hum Reprod* 2008;23:1431–8.
34. Chen MJ, Yang WS, Yang JH, Chen CL, Ho HN, Yang YS. Relationship between androgen levels and blood pressure in young women with polycystic ovary syndrome. *Hypertension* 2007;49:1442–7.
35. Gambineri A, Pelusi C, Vicennati V, Pagotto U, Pasquali R. Obesity and the polycystic ovary syndrome. *Int J Obes* 2002;26:883–96.
36. Dunaif A, Segal KR, Futterweit W, Dobrjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. *Diabetes* 1989;38:1165–74.
37. Hoeger K. Obesity and weight loss in polycystic ovary syndrome. *Obstet Gynecol Clin North Am* 2001;28:85–97.
38. Martínez-Bermejo E, Luque-Ramírez M, Escobar-Morreale HF. Obesity and the polycystic ovary syndrome. *Minerva Endocrinol* 2007;32:129–40.
39. Su HW, Chen CM, Chou SY, Liang SJ, Hsu CS, Hsu MI. Polycystic ovary syndrome or hyperprolactinaemia: a study of mild hyperprolactinaemia. *Gynecol Endocrinol* 2010 May 26. [Epub ahead of print]
40. Liou TH, Yang JH, Hsieh CH, Lee CY, Hsu CS, Hsu MI. Clinical and biochemical presentations of polycystic ovary syndrome among obese and nonobese women. *Fertil Steril* 2009;92:1960–5.