Original Article

Serum Anti-Müllerian Hormone in Polycystic Ovary Syndrome and its Relationship with Insulin Resistance, Lipid Profile and Adiponectin

Thien Jun Jun, Aniza Mohammed Jelani[1], Julia Omar[1], Rahimah Abdul Rahim[2], Najib Majdi Yaacob[3]

Department of Pathology, Sarawak General Hospital, Kuching, Sarawak, Departments of [1]Chemical Pathology and [2]Obstetric and Gynaecology, School of Medical Sciences, Universiti Sains Malaysia, [3]Unit of Biostatistic and Research Methodology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Abstract

Objectives: This study was done to estimate serum anti-Müllerian hormone (AMH) level in polycystic ovary syndrome (PCOS) patients and to correlate serum AMH level with insulin resistance, lipid profile, and adiponectin levels. **Materials and Methods:** A cross-sectional study was conducted at Hospital Universiti Sains Malaysia (Hospital USM), Health Campus, Kubang Kerian, Kelantan, Malaysia. Thirty newly diagnosed patients with PCOS attending gynecology clinic between July 2016 and April 2017 were recruited. Fasting venous blood samples were collected from the subjects. Serum AMH, insulin, adiponectin, triglycerides, high-density lipoprotein cholesterol (HDL-C), and plasma glucose levels were measured, and insulin resistance was calculated based on homeostasis model of assessment-insulin resistance (HOMA-IR). The serum AMH level was estimated, and the correlation of serum AMH level with the metabolic parameters was analyzed. **Results:** The median of serum AMH levels in women with PCOS was 6.8 ng/mL (interquartile range: 7.38 ng/mL). There was a significant negative correlation between serum AMH and HOMA-IR or triglyceride levels ($r = -0.49$, $P = 0.006$ and $r = -0.55$, $P = 0.002$, respectively). A significant positive correlation was observed between serum AMH and serum HDL-C or serum adiponectin levels ($r = 0.56$, $P = 0.001$ and $r = 0.44$, $P = 0.014$, respectively) in all study subjects. **Conclusion:** The serum AMH level is associated with HOMA-IR, triglycerides, HDL-C, and adiponectin levels, and hence it may be used as a potential cardiometabolic risk marker in women with PCOS.

Keywords: Adiponectin, Anti-Müllerian hormone, insulin resistance, lipid profile, Polycystic ovary syndrome

Introduction

Polycystic ovary syndrome (PCOS), is a disorder characterized by heterogeneous clinical presentations including hyperandrogenism, menstrual irregularity, and infertility along with metabolic disturbances manifested by hyperinsulinemia, obesity, hypertension, and dyslipidemia.[1]

Anti-Müllerian hormone (AMH), also known as Müllerian-inhibiting substance (MIS), is a dimeric glycoprotein hormone with a molecular weight of 140 kDa.[2] AMH is well-known for its role as a useful marker of ovarian reserve, as it reflects the size of the resting primordial follicle pool.[3] In PCOS, AMH is thought to be a potential surrogate marker for the diagnosis particularly in the absence of ultrasound.[4]

Recently, researchers are focusing on the relationship between AMH and metabolic factors such as homeostasis model of assessment-insulin resistance (HOMA-IR), lipid profile, and adiponectin. Knowledge on the relationship between AMH and the metabolic parameters may have significant clinical implications. The metabolic parameters represent the components of metabolic syndrome (MetS), which commonly presents in patients with PCOS.[5] IR plays an important role in PCOS pathophysiology and it leads to compensatory increase in insulin level and hyperinsulinemia. AMH level is

Access this article online

Quick Response Code:

Website: www.ijem.in

DOI: 10.4103/ijem.IJEM_305_19

Address for correspondence: Dr. Aniza Mohammed Jelani, Department of Chemical Pathology, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia.
E-mail: anizamj@usm.my

Submitted: 04-Aug-2019 **Revised:** 29-Nov-2019
Accepted: 16-Dec-2019 **Published:** 30-Apr-2020

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

How to cite this article: Jun TJ, Jelani AM, Omar J, Abdul Rahim R, Yaacob NM. Serum Anti-Müllerian hormone in polycystic ovary syndrome and its relationship with insulin resistance, lipid profile and adiponectin. Indian J Endocr Metab 2020;24:191-5.

also found to be increased in patients with PCOS. Insulin and AMH are believed to have influence on steroidogenesis and folliculogenesis.[6-9] Based on this, there is a possibility that AMH has a relationship with IR and other MetS components including lipid profile particularly triglycerides (TG) and HDL-C. AMH might also have a relationship with adiponectin, a novel biomarker for its relationship with IR and other MetS components.[10]

Studies regarding the correlation between AMH and IR using HOMA-IR have been done, but the findings have been mixed. A study by Park *et al*. in women without PCOS found that there was a negative correlation between AMH and HOMA-IR.[11] However, Bleil *et al*. demonstrated no association between both parameters.[12] In PCOS, inconsistent results have also been reported, and there were studies that demonstrated positive, negative, and even nil association between AMH and IR.[13-16]

Regarding correlation between AMH and lipid profile, very few studies have been reported and the findings were controversial.[16,17] Adiponectin, an important marker of MetS, was noted to have positive correlation with AMH in women without PCOS, but no such association was noted in patients with PCOS.[18]

In view of contradicting results from previous studies done in patients with PCOS, this study was conducted to show the type of relationship between AMH and these markers (HOMA-IR, TG, HDL-C, and adiponectin) in our population.

Materials and Methods

Subjects

This cross-sectional study was conducted at Hospital Universiti Sains Malaysia (Hospital USM), Kelantan, Malaysia. Newly diagnosed patients with PCOS (age 18–40 years) who attended gynecology clinic, Hospital USM, between July 2016 and April 2017 were recruited. The diagnosis of PCOS was made according to Rotterdam ESHRE-ASRM (2003) criteria. Patients with endocrinopathies such as thyroid disorder, Cushing's syndrome, and diabetes mellitus were excluded. Patients on medications such as oral contraceptives, hormonal treatment, antidiabetics, lipid-lowering agent, and weight reduction therapy were also excluded.

The study was conducted with the approval of the Human Research Ethics Committee USM (HREC), and written informed consent was obtained from all subjects.

Specimen collection

A fasting venous blood of 10 mL was collected in the morning after an overnight fast of 10–12 h irrespective of the day of menstrual cycle. The blood was dispensed into two tubes, a plain tube for AMH, insulin, high-density lipoprotein cholesterol (HDL-C), TG, and adiponectin analyses and an oxalate/fluoride tube for glucose measurement. Both tubes were centrifuged and the samples were stored at − 80°C until analysis. Serum HDL-C, serum TG, and plasma glucose level were analyzed by batch.

Laboratory measurements

AMH and insulin measurements were carried out on Cobas e 411 analyzer (Roche Diagnostics). The intra- and interassay variability at AMH concentration of 2.44 and 12.3 ng/mL was 1.2%, 3.3% and 1.1%, 3.7%, respectively. For insulin, the intra- and interassay variability at concentration of 21.9 and 74.3 μU/mL was 3.2%, 4.2% and 3.7%, 4.6%, respectively. Glucose, TG, and HDL-C were measured on an ARCHITECT C8000 chemistry analyzer (Abbott Diagnostics). The interassay variability of TG at 1.00 and 2.65 mmol/L was 0.89% and 1.54%, respectively. For HDL-C, at concentration of 0.54 and 2.04 mmol/L, the intra- and interassay variability was 1.7%, 1.0% and 1.1%, 0.5%, respectively. Adiponectin measurement was based on enzyme-linked immunosorbent assay (ELISA) method using RayBio® Human Acrp30 ELISA Kit (RayBiotech). The intra- and interassay variability was <10% and <12%, respectively. HOMA-IR index was calculated based on fasting plasma glucose concentration and serum insulin with standard formula: HOMA-IR = fasting concentration of insulin (μU/mL) × fasting concentration of glucose (mmol/L)/22.5.

Statistical analysis

The sample size was calculated using G*power software version 3.1.9.2 (test family: exact; statistical test: correlation, bivariate normal model). For a correlation coefficient of 0.5 under alternative hypothesis (ρHa = 0.5) to be statistically significant with two tail type I error of 0.05, type II error of 80%, and correlation coefficient under null hypothesis of 0 (ρ H0 = 0), the required sample size was 29. Anticipating 10% dropout due to preanalytical error, the adjusted number of study participants required in this study was 33 patients with PCOS. It was expected that the number of patients with PCOS receiving treatment in Hospital USM throughout the study period is equal to the number of patients required based on sample size calculation. Therefore, no sampling method was applied in this study and all eligible patients with PCOS who fulfilled the eligibility criteria were included.

Data analysis was performed using SPSS Statistical Package version 22. The distribution of numerical variables was examined using test of normality (Shapiro–Wilk test) and histogram with overlap normal curve. Normally distributed numerical variables were described as mean and standard deviation (SD), whereas non-normally distributed numerical variables were described as median and interquartile range (IQR). Serum AMH level was estimated using descriptive statistics function in SPSS to obtain the measure of central tendency and dispersion. The estimated level was reported as median (IQR) together with range (minimum and maximum values). To determine linear relationship between AMH and other metabolic parameters (insulin, TG, HDL-C, HOMA-IR), assumption for Pearson's correlation was assessed during data analysis. Assessment of normal distribution of the variables was examined by histogram and scatterplot. This assumption was violated because

AMH, HOMA-IR, TG, and adiponectin were non-normally distributed. Bivariate correlation analyses were therefore conducted using Spearman's correlation. *P* value < 0.05 was used as statistically significant.

Results

Thirty women who met the criteria for PCOS were included in the study. The mean age was 27.8 ± 4.08 years. The range of serum AMH level measured was between 2.4 and 17.8 ng/mL with a median of 6.8 ng/mL and IQR of 7.38 ng/mL. The general characteristics of the study subjects with PCOS are given in Table 1.

Correlation analyses were performed between serum AMH and metabolic parameters in women with PCOS as shown in Table 2. There was a negative moderate correlation between serum AMH and HOMA-IR, which was statistically significant ($r = -0.49$, $P = 0.006$). There was also a significant inverse moderate correlation between serum AMH and serum TG ($r = -0.55$, $P = 0.002$). A significant positive moderate correlation was observed between serum AMH and serum HDL-C in all study subjects ($r = 0.56$, $P = 0.001$). There was a positive fair correlation found between serum AMH and serum adiponectin in women with PCOS, which was statistically significant ($r = 0.44$, $P = 0.014$).

Table 1: General characteristics of study subjects with PCOS (*n*=30)

Characteristic	Mean±SD/ median (IQR)	Range of values
Age (years)	27.8±4.08	21-36
BMI (kg/m^2)	31.2±6.22	24.3-44.1
AMH (ng/mL)	6.8 (7.38)	2.5-17.9
HOMA-IR	5.0 (5.52)	1.1-19.5
Triglycerides (mmol/L)	1.4 (1.22)	0.6-5.1
HDL-C (mmol/L)	1.2±0.21	0.8-1.7
Adiponectin (µg/mL)	2.5 (2.01)	0.9-6.8
Fasting blood glucose (mmol/L)	4.9 (0.90)	3.7-6.6
Fasting insulin (µU/mL)	20.7 (17.72)	4.7-48.4

PCOS, polycystic ovary syndrome; SD, standard deviation; IQR, interquartile range; BMI, body mass index; AMH, anti-Müllerian hormone; HOMA-IR, homeostasis model assessment for insulin resistance; HDL-C, high-density lipoprotein cholesterol

Table 2: Correlations between serum AMH and HOMA-IR, triglycerides, HDL-C, and adiponectin in women with polycystic ovary syndrome

Variable	*R*	*P*
HOMA-IR	-0.49	0.006
Triglycerides (mmol/L)	-0.55	0.002
HDL-C (mmol/L)	0.56	0.001
Adiponectin (µg/mL)	0.44	0.014

AMH, anti-Müllerian hormone; HOMA-IR, homeostasis model assessment for insulin resistance; HDL-C, high-density lipoprotein cholesterol. *P*<0.05

Discussion

PCOS is a complex reproductive and hormonal disorder associated with derangement in metabolic parameters which indicates a risk for long-term clinical complications.

In our study, the median HOMA-IR was 5.0 (5.52) which suggests the presence of IR. This is in line with the findings from previous studies regarding IR in PCOS.[19,20] The association between AMH levels and HOMA-IR in patients with PCOS was inconsistent based on previous studies.[13-16] The contributing factors to the mixed results in previous studies were due to the possibility of differences in ethnicity, sample size, and anthropometry of the study subjects. However, the actual mechanism of action of insulin on AMH production is still not clear. In our study, a significant negative moderate correlation between serum AMH and HOMA-IR ($r = -0.49$, $P = 0.006$) was found. This observation is similar to studies done by Chen *et al*.[14] and Feldman *et al*.[21] The negative correlation between AMH and HOMA-IR could be due to the effect of oxidative stress in IR on ovarian granulosa cell function. AMH is produced by the granulosa cells of ovaries and IR has been associated with oxidative stress.[22,23] The role of oxidative damage to granulosa cells has been hypothesized by Park *et al*. and De Bruin *et al*.[11,24] Therefore, the same hypothesis could be applied in this study indicating that oxidative stress in patients with IR may induce injury to granulosa cells and subsequently decrease AMH production. However, this hypothesis needs further evaluations.

Hypertriglyceridemia and low HDL-C are common metabolic abnormalities found in women with PCOS.[7] A study by Aye *et al*. suggested that the presence of hypertriglyceridemia could contribute to atherothrombosis via platelet hyperactivation in PCOS.[25] The high prevalence of low HDL-C in women with PCOS also contributes to the risk of cardiovascular disease.[26] In our study, there was a significant negative moderate correlation between serum AMH and serum TG ($r = -0.55$, $P = 0.002$). This finding is contrary to the results published by Cui *et al*. and Feldman *et al*. which stated no significant correlation was found in women with PCOS.[16,21] There was a significant positive moderate correlation observed between serum AMH levels and serum HDL-C levels in this study ($r = 0.56$, $P = 0.001$). This result is consistent with a study done by Feldman *et al*.[21] However, Cui *et al*. and Skalba *et al*. showed negative and no correlation, respectively, which are contradictory to our finding.[16,17] The correlation between AMH and lipid profile in our study might suggest the potential utility of AMH as a biomarker for cardiovascular risk assessment. Further evaluation is needed to confirm these findings.

The median level of adiponectin in our study was 2.5 µg/mL, which was lower than normal population. This is consistent with a number of studies demonstrating lower adiponectin levels in PCOS.[27,28] The hormone possesses insulin sensitizing and anti-atherosclerotic properties which may reduce the development of metabolic disorders such as type 2

diabetes mellitus, obesity, and cardiovascular diseases.[28] The association between serum adiponectin and MetS is also noted in patients with PCOS based on study done by Ko *et al*.[29] There was a significant positive fair correlation between serum AMH and serum adiponectin levels in this study ($r = 0.44$, $P = 0.014$). Woo *et al.* reported no association between serum AMH and adiponectin in patients with PCOS; however, positive correlation was described in patients without PCOS.[18] The findings in our study could be explained by the relationship between adiponectin and IR which affect AMH production. Adiponectin was found negatively correlated with IR (HOMA-IR) in overweight subjects and in patients with PCOS.[30-32] IR is closely related to PCOS pathophysiology and it may lead to reduced AMH production as mentioned earlier. Therefore, due to the impact of adiponectin on improving insulin sensitivity, there is possibility that adiponectin will be able to increase AMH production.

A number of studies have evaluated the level of AMH in patients with PCOS. The value of AMH is known to be higher in patients with PCOS than without PCOS.[33] There are evidences that serum AMH level varied across different ethnicities.[13,16] There could be both genetic and environmental factors that affect the serum AMH levels in different ethnic groups.[34,35] A recent study on serum AMH levels across different ethnic group by Bhide *et al.* reported that the levels of serum AMH are higher in South Asian Women than in white European and Afro-Caribbean women and described the influence of smoking status on AMH level.[34] In this study, the median level of AMH represents all patients with PCOS attending the gynecological clinic in Hospital USM. Further studies with larger sample size including recruitment from other hospitals in Kelantan State are needed for better estimation of AMH in women with PCOS in the population.

Conclusion

In summary, our study demonstrates that serum AMH has association with HOMA-IR, TG, HDL, and adiponectin levels. Thus, it may be used as a potential cardiometabolic risk marker in patients with PCOS.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Balen AH. Polycystic ovary syndrome (PCOS). The Obstet Gynaecol 2017;19:119-29.
2. Rzeszowska M, Leszcz A, Putowski L, Hałabiś M, Tkaczuk-Włach J, Kotarski J, *et al*. Anti-Müllerian hormone: Structure, properties and appliance. Ginekologia Polska 2016;87:669-74.
3. Weenen C, Laven JS, Von Bergh AR, Cranfield M, Groome NP, Visser JA, *et al*. Anti-Müllerian hormone expression pattern in the human ovary: Potential implications for initial and cyclic follicle recruitment. Mol Hum Reprod 2004;10:77-83.
4. Conway G, Dewailly D, Diamanti-Kandrakis E, Escobar-Morreale HF, Frank S, Gambineri A, *et al*. The polycystic ovary syndrome: A position statement from the European society of endocrinology. Eur J Endocrinol 2014;171:1-29.
5. Ranasinha S, Joham A, Norman R, Shaw J, Zoungas S, Boyle J, *et al*. The association between polycystic ovary syndrome (PCOS) and metabolic syndrome: A statistical modelling approach. Clin Endocrinol 2015;83:879-87.
6. Grynnerup AG, Lindhard A, Sørensen S. The role of anti-Müllerian hormone in female fertility and infertility–An overview. Acta Obstetricia Gynecologica Scand 2012;91:1252-60.
7. Durlinger AL, Gruijters MJ, Kramer P, Karels B, Kumar TR, Matzuk MM, *et al*. Anti-Mullerian hormone attenuates the effects of FSH on follicle development in the mouse ovary. Endocrinology 2001;142:4891-9.
8. Khan KA, Stas S, Kurukulasuriya LR. Polycystic ovarian syndrome. J Cardiometab Syndr 2006;1:125-32.
9. Diamanti-Kandarakis E. Polycystic ovarian syndrome: Pathophysiology, molecular aspects and clinical implications. Expert Rev Mol Med 2008;10:e3.
10. Kadowaki T, Yamauchi T, Kubota N, Hara K, Ueki K, Tobe K. Adiponectin and adiponectin receptors in insulin resistance, diabetes, and the metabolic syndrome. J Clin Invest 2006;116:1784.
11. Park HT, Cho GJ, Ahn KH, Shin JH, Kim YT, Hur JY, *et al*. Association of insulin resistance with anti-mullerian hormone levels in women without polycystic ovary syndrome (PCOS). Clin Endocrinol 2010;72:26-31.
12. Bleil ME, Gregorich SE, McConnell D, Rosen MP, Cedars MI. Does accelerated reproductive aging underlie pre-menopausal risk for cardiovascular disease? Menopause 2013;20:1139.
13. La Marca A, Orvieto R, Giulini S, Jasonni VM, Volpe A, De Leo V. Müllerian-inhibiting substance in women with polycystic ovary syndrome: Relationship with hormonal and metabolic characteristics. Fertil Steril 2004;82:970-2.
14. 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.
15. Bayrak A, Terbell H, Urwitz-Lane R, Mor E, Stanczyk FZ, Paulson RJ. Acute effects of metformin therapy include improvement of insulin resistance and ovarian morphology. Fertil Steril 2007;87:870-5.
16. Cui Y, Shi Y, Cui L, Han T, Gao X, Chen ZJ. Age-specific serum antimüllerian hormone levels in women with and without polycystic ovary syndrome. Fertil Steril 2014;102:230-6.e2.
17. Skałba P, Cygal A, Madej P, Dąbkowska-Huć A, Sikora J, Martirosian G, *et al*. Is the plasma anti-müllerian hormone (AMH) level associated with body weight and metabolic, and hormonal disturbances in women with and without polycystic ovary syndrome? Eur J Obstet Gynecol Reprod Biol 2011;158:254-9.
18. Woo HY, Kim KH, Rhee EJ, Park H, Lee MK. Differences of the association of anti-müllerian hormone with clinical or biochemical characteristics between women with and without polycystic ovary syndrome. Endocr J 2012;59:781-90.
19. Kumar A, Barki S, Raghav V, Chaturvedi A, Kumar KH. Correlation of vitamin D with metabolic parameters in polycystic ovarian syndrome. J Family Med Prim Care 2017;6:115.
20. Song DK, Hong YS, Sung YA, Lee H. Insulin resistance according to β-cell function in women with polycystic ovary syndrome and normal glucose tolerance. PLoS One 2017;12:e0178120.
21. Feldman RA, O'Neill K, Butts SF, Dokras A. Antimüllerian hormone

levels and cardiometabolic risk in young women with polycystic ovary syndrome. Fertil Steril 2017;107:276-81.
22. Park K, Gross M, Lee DH, Holvoet P, Himes JH, Shikany JM, *et al*. Oxidative stress and insulin resistance. Diabetes Care 2009;32:1302-7.
23. Paneni F, Costantino S, Cosentino F. Role of oxidative stress in endothelial insulin resistance. World J Diabetes 2015;6:326.
24. De Bruin J, Dorland M, Spek E, Posthuma G, Van Haaften M, Looman C, *et al*. Age-related changes in the ultrastructure of the resting follicle pool in human ovaries. Biol Reprod 2004;70:419-24.
25. Aye MM, Kilpatrick ES, Aburima A, Wraith KS, Magwenzi S, Spurgeon B, *et al*. Acute hypertriglyceridemia induces platelet hyperactivity that is not attenuated by insulin in polycystic ovary syndrome. J Am Heart Assoc 2014;3:e000706.
26. Wild RA, Carmina E, Diamanti-Kandarakis E, Dokras A, Escobar-Morreale HF, Futterweit W, *et al*. Assessment of cardiovascular risk and prevention of cardiovascular disease in women with the polycystic ovary syndrome: A consensus statement by the Androgen excess and polycystic ovary syndrome (AE-PCOS) society. J Clin Endocrinol 2010;95:2038-49.
27. Mirza SS, Shafique K, Shaikh AR, Khan NA, Qureshi MA. Association between circulating adiponectin levels and polycystic ovarian syndrome. J Ovarian Res 2014;7:18.
28. Aroda V, Ciaraldi TP, Chang SA, Dahan MH, Chang RJ, Henry RR. Circulating and cellular adiponectin in polycystic ovary syndrome: Relationship to glucose tolerance and insulin action. Fertil Steril 2008;89:1200-8.
29. Ko JK, Li HW, Lam KS, Tam S, Lee VC, Yeung TW, *et al*. Serum adiponectin is independently associated with the metabolic syndrome in Hong Kong, Chinese women with polycystic ovary syndrome. Gynecol Endocrinol 2016;32:390-4.
30. Altinova AE, Toruner F, Bukan N, Yasar DG, Akturk M, Cakir N, *et al*. Decreased plasma adiponectin is associated with insulin resistance and HDL cholesterol in overweight subjects. Endocr J 2007;54:221-6.
31. Sepilian V, Nagamani M. Adiponectin levels in women with polycystic ovary syndrome and severe insulin resistance. J Soc Gynecol Investig 2005;12:129-34.
32. Yilmaz M, Bukan N, Demırcı H, Öztürk Ç, Kan E, Ayvaz G, *et al*. Serum resistin and adiponectin levels in women with polycystic ovary syndrome. Gynecol Endocrinol 2009;25:246-52.
33. Sahmay S, Guralp O, Senturk LM, Imamoglu M, Kucuk M, Irez T. Serum anti-müllerian hormone concentrations in reproductive age women with and without polycystic ovary syndrome: The influence of body mass index. Reprod Med Biol 2011;10:113-20.
34. Bhide P, Gudi A, Shah A, Homburg R. Serum anti-mullerian hormone levels across different ethnic groups: A cross-sectional study. BJOG 2015;122:1625-9.
35. Schuh-Huerta SM, Johnson NA, Rosen MP, Sternfeld B, Cedars MI, Reijo Pera RA. Genetic variants and environmental factors associated with hormonal markers of ovarian reserve in Caucasian and African American women. Hum Reprod 2011;27:594-608.