

ANDROGEN EXCESS AND PCOS SOCIETY

Quarterly Review for Androgen Excess-PCOS Society
January 1st – March 31st, 2012

Contents

AEPCOS publications in January – March, 2012

1. Escobar-Morreale HF, Carmina E, Dewailly D, Gambineri A, Kelestimur F, Moghetti P, Pugeat M, Qiao J, Wijeyaratne CN, Witchel SF, Norman RJ **Epidemiology, diagnosis and management of hirsutism: a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome Society. Hum Reprod Update. 2012 Mar-Apr;18(2):146-170. 8**

Hirsutism is a common clinical feature of disorders associated with androgen excess. The prevalence is approximately 10% in most populations. For women, this increased hair growth in androgen dependent areas negatively impacts self-esteem and quality of life. Expert and experienced members of the Androgen Excess-PCOS Society performed a systematic review and critical assessment of available data related to epidemiology, pathophysiology, diagnosis, and management of hirsutism. For this comprehensive analysis, multiple databases were searched. The routine use of the nine body area modified Ferriman-Gallwey (mFG) score to quantify hirsutism was recommended. Further, the use of a cut-off value as the 95th percentile for the relevant population was recommended. In the absence of population norms, mFG scores ≥ 8 should be used for White and Black women; lower scores are appropriate for Far East and South East Asian women. The pathophysiology of hair growth and role of androgens in the hair follicle growth cycle were described. Careful scrutiny and exhaustive analysis regarding differential diagnosis, laboratory studies, and management recommendations are provided. This comprehensive document provides evidenced-based contemporary strategies to treat women with hirsutism.

2. Proceedings from the 9th Annual Meeting of the Androgen Excess & Polycystic Ovary Syndrome (AE-PCOS) Society, Orlando, FL, USA, October 13–15, 2011.

Steroids, Volume 77, Issue 4, Pages 289-354 (10 March 2012)

Advances in PCOS and Androgen Excess Disorders

Edited by Daniel Dumesic and Enrico Carmina

This special issue of Steroids presents keynote presentations from the 9th Annual Meeting of the Androgen Excess & Polycystic Ovary Syndrome (AE-PCOS) Society held in Orlando, FL, USA on October 13–15, 2011. The issue serves as an important source of information on new developments and clinical recommendations related to metabolic, reproductive, immunological and psychological issues and each publication is authored by internationally-recognized authorities in the field of PCOS and other androgen excess disorders. AEPCOS intends that the knowledge provided in this special edition of Steroids enables improved care of women affected with these common and important disorders. Individual speaker topics are listed below:

Carmina E, Dumesic D. Androgen disorders. Steroids. 2012 Mar 10;77(4):289. 27

Ehrmann DA. Metabolic dysfunction in PCOS: Relationship to obstructive sleep apnea. Steroids. 2012 Mar 10;77(4):290-294. 17

Wild RA. Dyslipidemia in PCOS. Steroids. 2012 Mar 10;77(4):295-9. 23

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González F. Inflammation in Polycystic Ovary Syndrome: underpinning of insulin resistance and ovarian dysfunction. <i>Steroids</i> . 2012 Mar 10;77(4):300-305.	18
Duleba AJ. Medical management of metabolic dysfunction in PCOS. <i>Steroids</i> . 2012 Mar 10;77(4):306-311.	17
Escobar-Morreale HF. Surgical management of metabolic dysfunction in PCOS. <i>Steroids</i> . 2012 Mar 10;77(4):312-316.	17
Jones MR, Chua AK, Mengesha EA, Taylor KD, Chen YD, Li X, Krauss RM, Rotter JI; Reproductive Medicine Network, Legro RS, Azziz R, Goodarzi MO. Metabolic and cardiovascular genes in polycystic ovary syndrome: a candidate-wide association study (CWAS). <i>Steroids</i> . 2012 Mar 10;77(4):317-322. ...	13
Jakubowicz D, Froy O, Wainstein J, Boaz M. Meal timing and composition influence ghrelin levels, appetite scores and weight loss maintenance in overweight and obese adults. <i>Steroids</i> . 2012 Mar 10;77(4):323-331. Erratum in: <i>Steroids</i> . 2012 Jul;77(8-9):887-889.	19
Burt Solorzano CM, Beller JP, Abshire MY, Collins JS, McCartney CR, Marshall JC. Neuroendocrine dysfunction in polycystic ovary syndrome. <i>Steroids</i> . 2012 Mar 10;77(4):332-337.	23
Dokras A. Mood and anxiety disorders in women with PCOS. <i>Steroids</i> . 2012 Mar 10;77(4):338-41.	30
Trapp CM, Oberfield SE. Recommendations for treatment of nonclassic congenital hyperplasia (NCCAH): an update. <i>Steroids</i> . 2012 Mar 10;77(4):342-346.	6
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Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Kamrath C, Hochberg Z, Hartmann MF, Remer T, Wudy SA. Increased activation of the alternative "backdoor" pathway in patients with 21-hydroxylase deficiency: evidence from urinary steroid hormone analysis. *J Clin Endocrinol Metab.* 2012 Mar;97(3):E367-75. Epub 2011 Dec 14 32

PCOS - Etiology and Animal Models

Amalfi S, Velez LM, Heber MF, Vighi S, Ferreira SR, Orozco AV, Pignataro O, Motta AB. Prenatal hyperandrogenization induces metabolic and endocrine alterations which depend on the levels of testosterone exposure. *PLoS One.* 2012;7(5):e37658. PubMed PMID: 22655062; PubMed Central PMCID: PMC3360026. 32

McGee WK, Bishop CV, Bahar A, Pohl CR, Chang RJ, Marshall JC, Pau FK, Stouffer RL, Cameron JL. Elevated androgens during puberty in female rhesus monkeys lead to increased neuronal drive to the reproductive axis: a possible component of polycystic ovary syndrome. *Hum Reprod.* 2012 Feb;27(2):531-40. PubMed PMID: 22114112; PubMed Central PMCID: PMC3258033. 33

Veiga-Lopez A, Ye W, Padmanabhan V. Developmental programming: prenatal testosterone excess disrupts anti-Müllerian hormone expression in preantral and antral follicles. *Fertil Steril.* 2012 Mar;97(3):748-56. PubMed PMID: 22245531; PubMed Central PMCID: PMC3292625. 33

PCOS – Ovary

Homburg R, Hendriks ML, König TE, Anderson RA, Balen AH, Brincat M, Child T, Davies M, D'Hooghe T, Martinez A, Rajkhowa M, Rueda-Saenz R, Hompes P, Lambalk CB. Clomifene citrate or low-dose FSH for the first-line treatment of infertile women with anovulation associated with polycystic ovary syndrome: a prospective randomized multinational study. *Hum Reprod.* 2012 Feb;27(2):468-73. PubMed PMID: 22128296. ... 33

PCOS – Phenotypic Variation

Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Consensus on women's health aspects of polycystic ovary syndrome (PCOS). *Hum Reprod.* 2012 Jan;27(1):14-24. PubMed PMID: 22147920.

AND

Fauser BC, Tarlatzis BC, Rebar RW, Legro RS, Balen AH, Lobo R, Carmina E, Chang J, Yildiz BO, Laven JS, Boivin J, Petraglia F, Wijeyeratne CN, Norman RJ, Dunaif A, Franks S, Wild RA, Dumesic D, Barnhart K. Consensus on women's health aspects of polycystic

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ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. *Fertil Steril*. 2012 Jan;97(1):28-38.e25. PubMed PMID: 22153789.

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Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Auchus RJ, Miller WL. Congenital adrenal hyperplasia—more dogma bites the dust. *J Clin Endocrinol Metab*. 2012 Mar;97(3):772-5.

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Berenbaum SA, Bryk KL, Beltz AM. Early androgen effects on spatial and mechanical abilities: evidence from congenital adrenal hyperplasia. *Behav Neurosci*. 2012 Feb;126(1):86-96

Cheng TQ, Speiser PW. Treatment outcomes in congenital adrenal hyperplasia. *Adv Pediatr*. 2012;59(1):269-81.

Choi JH, Jin HY, Lee BH, Ko JM, Lee JJ, Kim GH, Jung CW, Lee J, Yoo HW. Clinical phenotype and mutation spectrum of the CYP21A2 gene in patients with steroid 21-hydroxylase deficiency. *Exp Clin Endocrinol Diabetes*. 2012 Jan;120(1):23-7. Epub 2011 Oct 21.

Coulm B, Coste J, Tardy V, Ecosse E, Roussey M, Morel Y, Carel JC; DHCSF Study Group. Efficiency of neonatal screening for congenital adrenal hyperplasia due to 21-hydroxylase deficiency in children born in mainland France between 1996 and 2003. *Arch Pediatr Adolesc Med*. 2012 Feb;166(2):113-20

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Idkowiak J, Randell T, Dhir V, Patel P, Shackleton CH, Taylor NF, Krone N, Arlt W. A missense mutation in the human cytochrome b5 gene causes 46,XY disorder of sex development due to true isolated 17,20 lyase deficiency. *J Clin Endocrinol Metab*. 2012 Mar;97(3):E465-75. Epub 2011 Dec 14.

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Jeandron DD, Sahakitrungruang T. A novel homozygous Q334X mutation in the HSD3B2 gene causing classic 3 β -hydroxysteroid dehydrogenase deficiency: an unexpected diagnosis after a positive newborn screen for 21-hydroxylase deficiency. *Horm Res Paediatr*. 2012;77(5):334-8. doi: 10.1159/000336004. Epub 2012 Feb 9

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Khalid JM, Oerton JM, Dezateux C, Hindmarsh PC, Kelnar CJ, Knowles RL. Incidence and clinical features of congenital adrenal hyperplasia in Great Britain. *Arch Dis Child.* 2012 Feb;97(2):101-6.

Krone N, Reisch N, Idkowiak J, Dhir V, Ivison HE, Hughes BA, et al. Genotype-phenotype analysis in congenital adrenal hyperplasia due to P450 oxidoreductase deficiency. *J Clin Endocrinol Metab.* 2012 Feb;97(2):E257-67. Epub 2011 Dec 7

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Vigliani MB, Buster JE. Nonclassic 21-hydroxylase deficiency presenting as endometrial hyperplasia with uterine bleeding in a 67-year-old woman. *Fertil Steril*. 2012 Apr;97(4):950-2. Epub 2012 Jan 23

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Polycystic ovary syndrome (PCOS)

PCOS – Adolescence

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PCOS – Dermatology and Body Hair Complications

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PCOS – Endocrine Disrupters

None.

PCOS – Etiology and Animal Models

Abbott AD, Colman RJ, Tiefenthaler R, Dumesic DA, Abbott DH. Early-to-Mid Gestation Fetal Testosterone Increases Right Hand 2D:4D Finger Length Ratio in Polycystic Ovary Syndrome-Like Monkeys. *PLoS One*. 2012;7(8):e42372. PubMed PMID: 22927929; PubMed Central PMCID: PMC3425513.

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PCOS – Psychology

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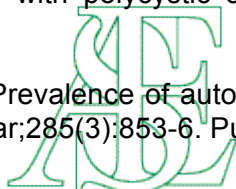
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PCOS – Thyroid Complications

Celik C, Abali R, Tasdemir N, Guzel S, Yuksel A, Aksu E, Yilmaz M. Is subclinical hypothyroidism contributing dyslipidemia and insulin resistance in women with polycystic ovary syndrome? *Gynecol Endocrinol.* 2012 Aug;28(8):615-8. PubMed PMID: 22329744.

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PCOS – Uterus

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Premature Adrenarche

Oron T, Lebenthal Y, de Vries L, Yackobovitch-Gavan M, Phillip M, Lazar L. Interrelationship of extent of precocious adrenarche in appropriate for gestational age girls with clinical outcome. *J Pediatr.* 2012 Feb;160(2):308-13. doi: 10.1016/j.jpeds.2011.08.009.

Sontag-Padilla LM, Dorn LD, Tissot A, Susman EJ, Beers SR, Rose SR. Executive functioning, cortisol reactivity, and symptoms of psychopathology in girls with premature adrenarche. *Dev Psychopathol.* 2012 Feb;24(1):211-23. doi: 10.1017/S0954579411000782

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Williams RM, Ward CE, Hughes IA. Premature adrenarche. Arch Dis Child. 2012 Mar;97(3):250-4. PubMed PMID: 21835833.

Brief summaries of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Kamrath C, Hochberg Z, Hartmann MF, Remer T, Wudy SA. Increased activation of the alternative "backdoor" pathway in patients with 21-hydroxylase deficiency: evidence from urinary steroid hormone analysis. J Clin Endocrinol Metab. 2012 Mar;97(3):E367-75. Epub 2011 Dec 14

In this study, Kamrath and colleagues collected urine samples from 142 untreated individuals with 21-hydroxylase deficiency. The subjects varied in age from one day to 25 years. Urine samples were also obtained from 138 control subjects. The urine samples were analyzed by gas chromatography-mass spectrometry to compare metabolites reflecting Δ^5 , Δ^4 , and backdoor pathway for metabolism of steroid hormones. These investigators found that the untreated 21-OHD subjects had increased urinary ratios of 5 α -pregnane-3 α ,17 α -diol-20-one (pdiol) reflecting the backdoor pathway compared to the Δ^4 and Δ^5 pathway metabolites. Thus, the backdoor pathway of steroid hormone metabolism is active postnatally with maximum activity during early infancy.

PCOS - Etiology and Animal Models

Amalfi S, Velez LM, Heber MF, Vighi S, Ferreira SR, Orozco AV, Pignataro O, Motta AB. Prenatal hyperandrogenization induces metabolic and endocrine alterations which depend on the levels of testosterone exposure. PLoS One. 2012;7(5):e37658. PubMed PMID: 22655062; PubMed Central PMCID: PMC3360026.

In this study, authors aimed to see the impact of prenatal testosterone levels on metabolic and endocrine alterations during adult life. They injected pregnant Sprague Dawley rats prenatally with either 2 or 5 mg free testosterone (groups T2 and T5, respectively) from day 16 to day 19 day of gestation. Female offspring from T2 and T5 displayed different phenotypes of PCOS during adult life. Offspring from T2 showed hyperandrogenism, ovarian cysts and ovulatory cycles, whereas those from T5 displayed hyperandrogenism, ovarian cysts and anovulatory cycles. Both these groups showed increased circulating glucose levels after an intraperitoneal glucose tolerance test which was higher in T5 rats and directly correlated with body weight at prepubertal age. A decrease in body weight at prepubertal age in both T2 and T5 rats, however, was compensated during adult life. At the molecular level, the higher dose of prenatal testosterone (T5) enhanced the expression of both the protein that regulates cholesterol availability (the steroidogenic acute regulatory protein (StAR)) and the protein expression of the transcriptional factor: peroxisome proliferator-activated receptor gamma (PPAR gamma). Prenatal hyperandrogenization induced an anti-oxidant response that prevented a possible pro-oxidant status and also the higher dose of prenatal testosterone (T5) induced a pro-inflammatory state in ovarian tissue mediated by increased levels of prostaglandin E (PG) and the protein expression of cyclooxygenase 2 (COX2, the limiting enzyme in PG synthesis). The authors concluded that prenatal injection of testosterone in female rats modulates the uterine environment and that, in turn, results in endocrine and metabolic abnormalities that resemble PCOS phenotypes during adult life.

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McGee WK, Bishop CV, Bahar A, Pohl CR, Chang RJ, Marshall JC, Pau FK, Stouffer RL, Cameron JL. Elevated androgens during puberty in female rhesus monkeys lead to increased neuronal drive to the reproductive axis: a possible component of polycystic ovary syndrome. Hum Reprod. 2012 Feb;27(2):531-40. PubMed PMID: 22114112; PubMed Central PMCID: PMC3258033.

These investigators assessed whether androgen exposure in the childhood and adolescent period could lead to pubertal alterations in LH secretory patterns in female rhesus monkeys. Subcutaneous testosterone implants were placed beginning at 1 year of age to maintain elevated circulating testosterone concentrations during the post-pubertal period. Subsequently, pulsatile secretion of LH was measured over 12 h during the early follicular phase of a menstrual cycle. In addition, GnRH-stimulated gonadotropin responses were determined. Although the timing of menarche was comparable between the treated and control monkeys, the testosterone-treated animals had a significantly greater LH pulse frequency during the early follicular phase compared with controls ($P = 0.039$) when measured at 5 years of age. The GnRH-stimulated LH response was greater in the testosterone-treated animals at 4 years of age ($P = 0.042$), but not when the animals were 5 years old ($P = 0.57$). Both groups showed similar rates of ovulation in early adulthood. One limitation of this study is the small number of animals. Nevertheless, these data suggest that increased androgen exposure during puberty may alter the HPG axis to resemble the HPG axis features previously noted in girls with hyperandrogenism and obesity.

Veiga-Lopez A, Ye W, Padmanabhan V. Developmental programming: prenatal testosterone excess disrupts anti-Müllerian hormone expression in preantral and antral follicles. Fertil Steril. 2012 Mar;97(3):748-56. PubMed PMID: 22245531; PubMed Central PMCID: PMC3292625.

This study investigated the ovarian impact of fetal testosterone exposure on the expression of selected ovarian regulators implicated in follicular recruitment and persistence using a sheep model of PCOS. Immunohistochemical determination of expression of anti-Müllerian hormone (AMH), kit ligand, and growth differentiation factor 9 (GDF9) in fetal, prepubertal, and adult ovarian tissues showed that, in adult ovaries, fetal testosterone exposure reduced AMH protein expression in granulosa cells of preantral follicles, while increasing its expression in antral follicles, compared to age-matched ewes. These differences were not evident in prepubertal animals. Ovarian protein expression of GDF9 and kit ligand were not altered. Fetal testosterone exposure may thus contribute to over-expression of ovarian AMH in a sheep model for PCOS, analogous to ovarian findings in women with the syndrome. Fetal testosterone exposure may induce abnormal folliculogenesis in the sheep PCOS model (increased antral follicle persistence and anovulation), at least in part, through changes in AMH regulation.

PCOS – Ovary

Homburg R, Hendriks ML, König TE, Anderson RA, Balen AH, Brincat M, Child T, Davies M, D'Hooghe T, Martinez A, Rajkhowa M, Rueda-Saenz R, Hompes P, Lambalk CB. Clomifene citrate or low-dose FSH for the first-line treatment of infertile women with anovulation associated with polycystic ovary syndrome: a prospective randomized multinational study. Hum Reprod. 2012 Feb;27(2):468-73. PubMed PMID: 22128296.

Clomiphene induction of ovulation has been the primary option for induction of ovulation for infertile women with PCOS for decades. Modest pregnancy rates, significant side effects and adverse effects on the endometrium and cervical mucus by clomiphene have led to several peer-reviewed reports evaluating low dose gonadotropins as an alternative. This RCT, using a low-dose approach with recombinant FSH as a first-

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line treatment, yielded excellent results. Cumulative pregnancy rates were significantly higher and time to pregnancy faster. OHSS was avoided and a very low multiple birth rate was noted. The same monitoring protocols were used for both treatment arms and the monitoring costs were not different. Gonadotropin medication costs continue to be an impediment to this approach in many countries. For example the heterogeneity of infertility treatment coverage in the U.S. (if infertility treatment is covered by a health plan at all) would restrict gonadotropins for a significant amount of patients. The advantages of a faster time to conception, higher overall pregnancy rate and subsequently greater patient satisfaction (with low OHSS and multiple gestations), however, may offset any cost disadvantage for partially covered or self-paying patients. It would also be of interest to evaluate this approach in obese PCOS patients (who are the more common in the U.S.) as the treatment response and overall doses of medications required may prove to be different than in this study, where the BMI was essentially normal.

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Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Consensus on women's health aspects of polycystic ovary syndrome (PCOS). Hum Reprod. 2012 Jan;27(1):14-24. PubMed PMID: 22147920.

AND

Fauser BC, Tarlatzis BC, Rebar RW, Legro RS, Balen AH, Lobo R, Carmina E, Chang J, Yildiz BO, Laven JS, Boivin J, Petraglia F, Wijeyeratne CN, Norman RJ, Dunaif A, Franks S, Wild RA, Dumesic D, Barnhart K. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertil Steril. 2012 Jan;97(1):28-38.e25. PubMed PMID: 22153789.

These are two identical publications that represent considerations and conclusions arising from the recent consensus update conference on the diagnostic criteria required for PCOS, as well as the deliberations that went into inclusion of traits in the diagnosis. Perhaps more importantly, these two papers include consideration of relevant, but non-diagnostic, traits relevant to PCOS.