

ANDROGEN EXCESS AND PCOS SOCIETY

Quarterly Review for Androgen Excess-PCOS Society
July 1st – September 30th, 2011

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Brief overviews of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Hirvikoski T, Lindholm T, Lajic S, Nordenström A. Gender role behavior in prenatally dexamethasone-treated children at risk for congenital adrenal hyperplasia—a pilot study. Acta Paediatr. 2011 Sep;100(9):e112-9. doi: 10.1111/j.1651-2227.2011.02260.x. Epub 2011 Apr 8. 23

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PCOS – Etiology and Animal Models

Hogg K, Wood C, McNeilly AS, Duncan WC. The in utero programming effect of increased maternal androgens and a direct fetal intervention on liver and metabolic function in adult sheep. PLoS One. 2011;6(9):e24877. Epub 2011 Sep 14. PubMed PMID: 21935484; PubMed Central PMCID: PMC3173482. 24

PCOS – Ovary

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reserve? A randomized controlled trial. Fertil Steril. 2011 Nov;96(5):1128-33. Epub 2011 Sep 13. PubMed PMID: 21917254. 24

Premature Adrenarche

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Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

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Jordan-Young RM. Hormones, context, and "brain gender": a review of evidence from congenital adrenal hyperplasia. Soc Sci Med. 2012 Jun;74(11):1738-44. Epub 2011 Sep 17

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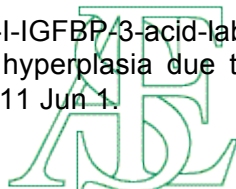
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Insulin resistance and metabolic syndrome



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Polycystic ovary syndrome (PCOS)

PCOS – Adolescence

Angioni S, Sanna S, Magnini R, Melis GB, Fulghesu AM. The quantitative insulin sensitivity check index is not able to detect early metabolic alterations in young patients with polycystic ovarian syndrome. *Gynecol Endocrinol*. 2011 Jul;27(7):468-74. Epub 2010 Jun 14. PMID: 20540677.

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PCOS – Dermatology and Body Hair Complications

Gómez Vázquez M, Navarra Amayuelas R, Lamarca M, Baquedano L, Romero Ruiz S, Vilar-Checa E, Iniesta MD. Ethinylestradiol/Chlormadinone acetate for use in dermatological disorders. *Am J Clin Dermatol*. 2011 Sep 6;12 Suppl 1:13-9. PubMed PMID: 21895045.

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PCOS – Endocrine Disrupters

None.

PCOS – Etiology and Animal Models

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PCOS – General Health Concerns

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PCOS – Genetics

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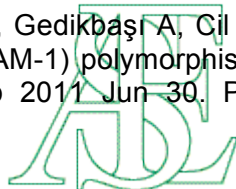
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None.

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PCOS – Phenotypic Variation

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PCOS – Psychology

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PCOS – Thyroid Complications

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PCOS – Uterus

Cloke B, Christian M. The role of androgens and the androgen receptor in cycling endometrium. Mol Cell Endocrinol. 2012 Jul 25;358(2):166-75. Epub 2011 Jul 2. PubMed PMID: 21745536.

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Makker A, Goel MM, Das V, Agarwal A. PI3K-Akt-mTOR and MAPK signaling pathways in polycystic ovarian syndrome, uterine leiomyomas and endometriosis: an update. Gynecol Endocrinol. 2012 Mar;28(3):175-81. Epub 2011 Sep 15. Review. PubMed PMID: 21916800.

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Premature Adrenarche

Blumenthal H, Leen-Feldner EW, Babson KA, Gahr JL, Trainor CD, Frala JL. Elevated social anxiety among early maturing girls. *Dev Psychol.* 2011 Jul;47(4):1133-40. PMID: 21604866

Ghizzoni L, Cappa M, Vottero A, Ubertini G, Carta D, Di Iorgi N, Gasco V, Marchesi M, Raggi V, Ibba A, Napoli F, Massimi A, Maghnie M, Loche S, Porzio O. Relationship of CYP21A2 genotype and serum 17-hydroxyprogesterone and cortisol levels in a large cohort of Italian children with premature pubarche. *Eur J Endocrinol.* 2011 Aug;165(2):307-14. Epub 2011 Jun 6. PMID: 21646284

Ibáñez L, López-Bermejo A, Díaz M, Marcos MV, de Zegher F. Early metformin therapy (age 8-12 years) in girls with precocious pubarche to reduce hirsutism, androgen excess, and oligomenorrhea in adolescence. *J Clin Endocrinol Metab.* 2011 Aug;96(8):E1262-7. Epub 2011 Jun 1. PMID: 21632811

Idkowiak J, Lavery GG, Dhir V, Barrett TG, Stewart PM, Krone N, Arlt W. Premature adrenarche: novel lessons from early onset androgen excess. *Eur J Endocrinol.* 2011 Aug;165(2):189-207. Epub 2011 May 26. Review. PMID: 21622478

Brief summaries of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Hirvikoski T, Lindholm T, Lajic S, Nordenström A. Gender role behaviour in prenatally dexamethasone-treated children at risk for congenital adrenal hyperplasia--a pilot study. *Acta Paediatr.* 2011 Sep;100(9):e112-9. doi: 10.1111/j.1651-2227.2011.02260.x. Epub 2011 Apr 8.

Since the 1980's, prenatal dexamethasone treatment has been considered for women at risk for a child with classical congenital adrenal hyperplasia (CAH). This treatment, however, is really directed at only 1 out of 8 possible outcomes: a female fetus affected with CAH. Data, such as presented in this article, are accumulating indicating that prenatal dexamethasone treatment may have significant deleterious consequences. In this pilot study, these authors provide data indicating that among boys exposed to dexamethasone in utero, their gender role behavior may be altered. This report cautions regarding prenatal dexamethasone exposure. Studies of larger cohorts are necessary to draw more decisive conclusions about the safety of prenatal dexamethasone treatment. Until then, parents must be thoroughly informed that this is an experimental therapy with potential deleterious consequences, many uncertainties, and benefits that accrue only to affected female fetuses.

Nordenström A. Adult women with 21-hydroxylase deficient congenital adrenal hyperplasia, surgical and psychological aspects. *Curr Opin Pediatr.* 2011 Aug;23(4):436-42.

This is an excellent and comprehensive review that provides current updates on multiple aspects of management for women with CAH. These aspects include the outcome of feminizing surgery, infertility, quality of life, and psychosexual aspects in women with CAH. Multi-disciplinary transitional care and pregnancy are also discussed.

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PCOS – Etiology and Animal Models

Hogg K, Wood C, McNeilly AS, Duncan WC. The in utero programming effect of increased maternal androgens and a direct fetal intervention on liver and metabolic function in adult sheep. PLoS One. 2011;6(9):e24877. Epub 2011 Sep 14. PubMed PMID: 21935484; PubMed Central PMCID: PMC3173482.

Fetal testosterone (T) excess, experimentally delivered via the dam, induces a variety of PCOS-like traits in exposed female mammals from mice to monkeys. In this sheep study by Hogg and colleagues, the authors add a refinement and experimentally deliver T directly to the fetal female. Such an approach should avoid the maternal insulin resistance and transient gestational hyperglycemia induced by using the dam as the delivery vehicle for excess T. Direct fetal T delivery requires delaying treatment until at least mid-gestation when the fetus is sufficiently developed to accommodate a depot intramuscular injection. Direct fetal T delivery, in contrast to maternal T delivery, does not induce fatty liver or a whole series of gene changes involved in hepatic glucose and lipid metabolism in the exposed female when adult. The controls involved in the direct fetal approach, however, show increased fatty liver compared to maternal delivery controls, implicating the fetal approach per se in some of the metabolic findings, possibly mediated through stress or inflammatory responses. Gene expression changes related to development of fatty liver are found in fetal T delivery, and are distinct from those of maternal T delivery. Interestingly, fetal T delivery increases hepatic expression of estrogen receptor alpha, whereas maternal T delivery increases hepatic expression of androgen receptor. Fetal T exposure may thus cause some, but not all, programming of adult metabolic traits. The results of direct fetal T delivery also implicate metabolic, stress or inflammatory factors in contributing to metabolic phenotypes generated by fetal T exposure models, whether or not they deliver T directly to the fetus or via the dam.

PCOS – Ovary

Palomba S, Falbo A, Di Cello A, Cappiello F, Tolino A, Zullo F. Does metformin affect the ovarian response to gonadotropins for in vitro fertilization treatment in patients with polycystic ovary syndrome and reduced ovarian reserve? A randomized controlled trial. Fertil Steril. 2011 Nov;96(5):1128-33. Epub 2011 Sep 13. PubMed PMID: 21917254.

The use of metformin for women with PCOS undergoing IVF treatment is controversial. Proponents point to recent literature that supports its use in reducing the risk of ovarian hyperstimulation syndrome (OHSS). Earlier studies reported improved reproductive outcomes. This interesting study by Palomba et al explores a theoretical concern that metformin, in its reduction of OHSS risk for hyper-responders, could indeed worsen the outcomes for poor responders to ovarian stimulation. PCOS patients with anovulatory infertility, and predicted to have poor response due to advanced reproductive age or elevated basal FSH, were randomized to metformin or placebo. Interestingly obese patients were excluded, perhaps with the understanding of recent evidence that non-obese women may have better IVF outcomes with metformin treatment than obese women. A GnRH flare protocol using only recombinant FSH was used for stimulation. Patients in the metformin arm were found to have twice the percentage of cancelled cycles, all due to poor response. Serious reductions in peak estradiol, mature follicles and oocyte numbers were seen in this group. Significantly higher gonadotropin dosing was required as well. The adverse effects were great enough to require termination of the study. Metformin has been proposed to reduce intra-ovarian androgen production, which would be beneficial in younger women requiring stimulation. Could metformin reduce those androgens, waning in the older PCOS patient, that are necessary for early folliculogenesis? Given the frequent practice of prescribing metformin to all PCOS patients during IVF cycles, confirmation of this effect with further study would be welcome.

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Premature Adrenarche

Idkowiak J, Lavery GG, Dhir V, Barrett TG, Stewart PM, Krone N, Arlt W. Premature adrenarche: novel lessons from early onset androgen excess. Eur J Endocrinol. 2011 Aug;165(2):189-207. Epub 2011 May 26. Review. PMID: 21622478

This publication provides a thorough and comprehensive approach to the differential diagnosis and management of children with premature pubic hair development. The figures are excellent.

