

ANDROGEN EXCESS AND PCOS SOCIETY

Quarterly Review for Androgen Excess-PCOS Society
July 1st – September 30th, 2012

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Brief overviews of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Meyer-Bahlburg HF, Dolezal C, Haggerty R, Silverman M, New MI. Cognitive outcome of offspring from dexamethasone-treated pregnancies at risk for congenital adrenal hyperplasia due to 21-hydroxylase deficiency. *Eur J Endocrinol.* 2012 Jul;167(1):103-10..... 24

Insulin resistance

Guo X, Cui J, Jones MR, Haritunians T, Xiang AH, Chen YD, Taylor KD, Buchanan TA, Davis RC, Hsueh WA, Raffel LJ, Rotter JI, Goodarzi MO. Insulin clearance: confirmation as a highly heritable trait, and genome-wide linkage analysis. *Diabetologia.* 2012 Aug;55(8):2183-92..... 24

PCOS – Adolescence

Trottier A, Battista MC, Geller DH, Moreau B, Carpentier AC, Simoneau-Roy J, Baillargeon JP. Adipose tissue insulin resistance in peripubertal girls with first-degree family history of polycystic ovary syndrome. *Fertil Steril.* 2012 Dec;98(6):1627-34. PubMed PMID: 22985947..... 24

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PCOS – Ovary

Catteau-Jonard S, Bancquart J, Poncelet E, Lefebvre-Maunoury C, Robin G, Dewailly D. Polycystic ovaries at ultrasound: normal variant or silent polycystic ovary syndrome? *Ultrasound Obstet Gynecol.* 2012 Aug;40(2):223-9. PubMed PMID: 22648908..... 25

PCOS and 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..... 25

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Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

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Matsubara Y, Ono M, Miyai K, Takizawa F, Takasawa K, Onishi T, Kashimada K, Mizutani S. Longitudinal analysis of growth and body composition of Japanese 21-OHD patients in childhood. *Endocr J.* 2013;60(2):149-54. Epub 2012 Sep 28.

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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

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PCOS – Etiology and Animal Models

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PCOS – General Health Concerns

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Bonny AE, Appelbaum H, Connor EL, Cromer B, DiVasta A, Gomez-Lobo V, Harel Z, Huppert J, Sucato G; NASPAG Research Committee. Clinical variability in approaches to polycystic ovary syndrome. *J Pediatr Adolesc Gynecol.* 2012 Aug;25(4):259-61. PubMed PMID: 22840936.

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PCOS – Genetics

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PCOS – Immunological Considerations

None.

PCOS – After the Menopause

None.



PCOS – Metabolic Dysfunction/Cardiovascular Disease/Inflammation

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PCOS – Neuroendocrine Dysfunction

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PCOS – Ovary

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PCOS – Pregnancy Complications

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None.

PCOS – Psychology

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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.

PCOS – Uterus

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Premature Adrenarche

Bird IM. In the zone: understanding zona reticularis function and its transformation by adrenarche. *J Endocrinol*. 2012 Aug;214(2):109-11.

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Brief summaries of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Meyer-Bahlburg HF, Dolezal C, Haggerty R, Silverman M, New MI. Cognitive outcome of offspring from dexamethasone-treated pregnancies at risk for congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Eur J Endocrinol. 2012 Jul;167(1):103-10

This manuscript reports the observational follow-up on children who received prenatal dexamethasone treatment because of their high risk to inherit CAH (sibs known to have congenital adrenal due to 21-hydroxylase deficiency). Their first study included 140 children aged 5-12 years with 67 DEX-exposed (long-term: eight CAH girls) and 73 unexposed (with 15 CAH girls). Their second study included 20 participants aged 11-24 years with seven DEX-exposed (long-term: one CAH woman) and 13 unexposed (with four CAH women). Although the vast majority of group comparisons were not significant, long-term DEX exposure was associated with slower mental processing than in controls on several neuropsychological variables. However, partial correlations of DEX exposure duration with cognitive outcome did not corroborate this association. These investigators concluded that their findings regarding cognitive function in CAH girls with long-term DEX exposure raised concerns about potentially adverse cognitive outcome following prenatal DEX. Nevertheless, they felt that replications in larger samples are required.

This manuscript investigates the outcome of prenatal dexamethasone treatment for pregnancies at risk for congenital adrenal hyperplasia. Accumulating data obtained from both animal and human investigations indicate the potential for deleterious consequences following prenatal DEX exposure. Based on the 2010 Endocrine Society Guidelines statement regarding prenatal DEX therapy, this use of this treatment should be re-evaluated (Speiser et al, Journal of Clinical Endocrinology & Metabolism 2010;95:4133-60).

Insulin resistance

Guo X, Cui J, Jones MR, Haritunians T, Xiang AH, Chen YD, Taylor KD, Buchanan TA, Davis RC, Hsueh WA, Raffel LJ, Rotter JI, Goodarzi MO. Insulin clearance: confirmation as a highly heritable trait, and genome-wide linkage analysis. Diabetologia. 2012 Aug;55(8):2183-92.

Insulin resistance shows an inverse correlation with insulin clearance which is considered to be an adaptive response to maintain euglycemia (Haffner et al. Eur J Clin Invest 1992;22:147-53). These authors had previously reported a high heritability of insulin clearance in a Hispanic cohort. In this manuscript, they validated their prior finding by performing hyperinsulinemic-euglycemic clamps in 513 participants from 140 Hispanic families. The most significant linkage peaks for steady state plasma insulin concentrations were observed at chromosome 15 and chromosome 20. Single nucleotide polymorphisms at these loci carry several promising candidate genes. Genes mapped to chromosome 15 include MEGF11, CYP1A2, CSK, LMAN1L, CHRN4, and ADAMTSL7. The locus on chromosome 20 included portions of the PLCG1 and ZHX3 genes. Understanding the mechanism(s) responsible for insulin clearance may benefit elucidation of the molecular etiologies of insulin resistance in PCOS.

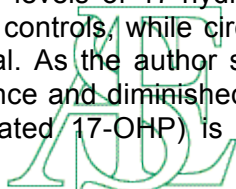
PCOS – Adolescence

Trottier A, Battista MC, Geller DH, Moreau B, Carpentier AC, Simoneau-Roy J, Baillargeon JP. Adipose tissue insulin resistance in peripubertal girls with first-degree family history of polycystic ovary syndrome. Fertil Steril. 2012 Dec;98(6):1627-34. PubMed PMID: 22985947

This study adds to increasing reports from these and other investigators on insulin-related defects in pre- and peri-pubertal girls who are either daughters or sisters of women with PCOS. This is a small, cross-sectional

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study with controls obtained from girls in general pediatric clinics with stable conditions, such as well-controlled hypothyroidism. The girls with close PCOS relatives were insulin resistant compared to controls and showed diminished insulin-mediated suppression of circulating levels of non-esterified fatty acids. The degree of insulin resistance correlated positively with circulating levels of 17-hydroxyprogesterone (17-OHP). PCOS relatives also had greater BMI and 17-OHP levels than controls, while circulating levels of androgens such as DHEA, androstenedione and testosterone were normal. As the author suggest, an increased genetic risk of PCOS may manifest before puberty as insulin resistance and diminished adipocyte response to insulin. Whether the accompanying latent hyperandrogenism (elevated 17-OHP) is a consequence or a cause of early onset metabolic defects requires elucidation.



PCOS - Ovary

Catteau-Jonard S, Bancquart J, Poncelet E, Lefebvre-Maunoury C, Robin G, Dewailly D. Polycystic ovaries at ultrasound: normal variant or silent polycystic ovary syndrome? Ultrasound Obstet Gynecol. 2012 Aug;40(2):223-9. PubMed PMID: 22648908.

The applicability of ovarian ultrasound criteria in the diagnosis PCOS, and its role in the long-term sequelae of the syndrome continue to stimulate controversy. Wrapped in this controversy is a large group of women who on ultrasound who exhibit a polycystic appearance to their ovaries (PCO), but do not satisfy any PCOS diagnosis based on NIH, Rotterdam or AEPCOS criteria. Despite multiple publications in the literature, there is no clear evidence for endocrine abnormalities in this group. The group from Lille, France, which has contributed greatly to our understanding of ovarian ultrasound and PCOS over many years, now presents interesting data concerning women with at least polycystic ovary. After strictly characterizing this subset of women, they report significantly higher levels of AMH in these women versus controls, and that they are intermediate between controls and those with PCOS. FSH levels were also reduced in these women, similar to those with PCOS. Inhibin levels were not studied, which would have been of interest regarding the FSH finding. The occurrence of ovulatory dysfunction, such as luteal deficiency, was not studied and would be another worthy area for study, particularly for infertile women in this group. The finding of increased AMH levels in these women is of clinical interest as it could have a negative impact on reproductive outcomes as well as a potential risk for ovarian hyperstimulation syndrome. PCO on ultrasound in otherwise normal phenotypes could therefore represent an intermediate condition, or “milder” PCOS phenotype as proposed by the authors. “Trust not too much to appearances” - [Virgil](#).

PCOS and Thyroid complications

Celik C, Abali R, Tasdemir N, Guzel S, Yuksel A, Aksu E, Yılmaz 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.

In a small, but interesting study, Celik and colleagues investigated lipid parameters, insulin resistance, and glucose tolerance in subclinical hypothyroidism (SCH) women with and without polycystic ovary syndrome (PCOS). The topic is debatable as previous data have been controversial. They studied 20 patients with PCOS and SCH (Group I) and 39 patients with PCOS and normal thyroid function (Group II) and 53 healthy women with normal thyroid function (Group III). The results indicated significantly higher triglyceride levels (143.26 ± 99.86 mg/dL) in group I compared to both group II (88.56 ± 37.56 mg/dL) and group III (83.71 ± 31.94 mg/dL). All other lipid components including total cholesterol, HDL-cholesterol, LDL-cholesterol was comparable in the groups. HOMA-IR was significantly different in groups I (2.92 ± 2.34), II (1.95 ± 1.52) and III (1.60 ± 0.86). Similarly, serum insulin levels were significantly different ($P = 0.027$) among the groups I

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($12.45 \pm 8.62 \mu\text{U/mL}$), II ($8.60 \pm 5.35 \mu\text{U/mL}$) and III ($7.04 \pm 3.55 \mu\text{U/mL}$). The authors concluded that women with PCOS and subclinical hypothyroidism should be evaluated for dyslipidemia and insulin resistance. This finding was a little unexpected as earlier studies on a larger cohort with multiple insulin indices (Ganie et al Fertil Steril. 2011;95:2039-43) showed no significant differences related to thyroid function. A prospective, well designed, randomized study with levothyroxine replacement and a placebo-controlled arm is needed to answer this question more definitively.

