

ANDROGEN EXCESS AND PCOS SOCIETY

Quarterly Review for Androgen Excess-PCOS Society
October 1st – December 31st, 2012

Contents

AEPCOS-related Publication

1. Roe AH, Prochaska E, Smith M, Sammel M, Dokras A. Using the Androgen Excess-PCOS Society Criteria to Diagnose Polycystic Ovary Syndrome and the Risk of Metabolic Syndrome in Adolescents. *J Pediatr.* 2012 Dec 19. PubMed PMID: 23260096.

The diagnosis of PCOS in adolescent girls is not easy and is still controversial. The issue: adolescent girls can mimic the diagnostic criteria for PCOS without maturing into a PCOS phenotype. Adolescents have naturally occurring clinical signs of high androgen levels, intermittent/absent menstrual cycles and polycystic ovaries as they transition to reproductive maturity. In this paper, Roe and colleagues use AEPCOS criteria to diagnose PCOS in 148 adolescents in the USA (mostly Caucasian) and examined the incidence of metabolic syndrome (MS) in these girls compared to 57 controls (recruited adolescents who failed diagnosis for PCOS). All girls were recruited from adolescents referred to a specialty clinic, so this referral bias likely contributes to the high incidence of PCOS. Only girls with >2 years postmenarche were included as having intermittent/absent cycles (≤ 9 per year) and polycystic ovaries were identified from at least one ovary with a volume $\geq 10 \text{ cm}^3$ and not antral follicle counts. Girls were classified with MS if they showed 3 of the following 5 modified Cook criteria: body mass index $>90^{\text{th}}$ percentile, serum triglycerides $\geq 150 \text{ mg/dL}$, serum high-density lipoprotein cholesterol (HDL-C) $\leq 40 \text{ mg/dL}$, blood pressure at $\geq 90^{\text{th}}$ percentile for age (or taking antihypertensives), and fasting plasma glucose $\geq 100 \text{ mg/dL}$. Of the 205 adolescents included in the study, $\sim 66\%$ were diagnosed with PCOS by AEPCOS criteria, with intermittent/absent cycles as the most frequent criterion. Almost all ($\sim 64\%$) adolescents identified as PCOS had at least one risk factor for MS, compared to about half that incidence in controls, and $\sim 11\%$ of girls identified as PCOS were classified as MS compared to $\sim 2\%$ of controls. These data, the authors suggest, should encourage early diagnosis of PCOS during adolescence to enable timely metabolic risk assessment and early intervention to diminish onset of cardiovascular disease.

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Brief overviews of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Chen W, Xu Z, Nishitani M, Van Ryzin C, McDonnell NB, Merke DP. Complement component 4 copy number variation and CYP21A2 genotype associations in patients with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Hum Genet. 2012 Dec;131(12):1889-94..... 25

Insulin resistance

Han MS, Jung DY, Morel C, Lakhani SA, Kim JK, Flavell RA, Davis RJ. JNK expression by macrophages promotes obesity-induced insulin resistance and inflammation. Science. 2013 Jan 11;339(6116):218-22. Epub 2012 Dec 6..... 25

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List of Publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Bachelot A, Chakhtoura Z, Plu-Bureau G, Coudert M, Coussieu C, Badachi Y, Dulon J, Charbit B, Touraine P; CAHLH study group. Influence of hormonal control on LH pulsatility and secretion in women with classical congenital adrenal hyperplasia. *Eur J Endocrinol*. 2012 Oct;167(4):499-505. PubMed PMID: 22893695.

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PCOS – Adolescence

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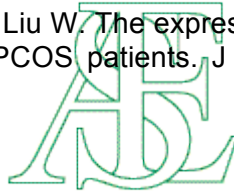
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PCOS – Endocrine Disrupters

None.

PCOS – Etiology and Animal Models

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PCOS – Pregnancy Complications

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PCOS – Protocol Reviews

None.

PCOS – Psychology

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PCOS – Thyroid Complications

Benetti-Pinto CL, Berini Piccolo VR, Garmes HM, Teatin Juliato CR. Subclinical hypothyroidism in young women with polycystic ovary syndrome: an analysis of clinical, hormonal, and metabolic parameters. *Fertil Steril*. 2012 Oct 25. PubMed PMID: 23103018.

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PCOS – Uterus

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Leonhardt H, Gull B, Kishimoto K, Kataoka M, Nilsson L, Janson PO, Stener-Victorin E, Hellström M. Uterine morphology and peristalsis in women with polycystic ovary syndrome. *Acta Radiol.* 2012 Dec 1;53(10):1195-201. PubMed PMID: 23081959.

Ormazabal P, Romero C, Gabler F, Quest AF, Vega M. Decreased Phosphorylation of Y14Caveolin-1 in Endometrial Tissue of Polycystic Ovary Syndrome Patients may be Related with an Insulin Resistant State in this Tissue. *Horm Metab Res.* 2012 Dec 7. PubMed PMID: 23225242.

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Woodard TL, Awonuga AO, Puscheck E. Malignant transformation of endometrioma in a woman with a history of ovulation induction and in vitro fertilization. *Case Report Med.* 2012;2012:497362. PubMed PMID: 23304157.

Premature Adrenarche

Kılıç A, Durmuş MS, Ünüvar E, Yıldız I, Aydın BK, Uçar A, Bundak R, Baş F, Darendeliler F, Oğuz F, Sidal M, Yekeler E. Clinical and laboratory characteristics of children referred for early puberty: preponderance in 7-8 years of age. *J Clin Res Pediatr Endocrinol.* 2012 Dec;4(4):208-12

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Brief summaries of selected publications

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Chen W, Xu Z, Nishitani M, Van Ryzin C, McDonnell NB, Merke DP. Complement component 4 copy number variation and CYP21A2 genotype associations in patients with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Hum Genet. 2012 Dec;131(12):1889-94

The gene encoding 21-hydroxylase (CYP21A2) is located in a complex genetic locus on chromosome 6 at the central region of the human major histocompatibility complex. There is a highly homologous pseudogene in close physical proximity and genes that encode complement 4 (C4A and C4B). Two other genes, RP1 and TNXB, are in tandem with CYP21 and C4 gene from the telomeric to centromeric ends constituting a genetic module termed RCCX (R P-C 4-C YP21-TN X). The RCCX modules are characterized by modular duplication or deletion events in which each duplicated or deleted module usually covers a CYP21A1P-TNXA-RP2-C4 unit. The most common haplotype is a bimodular RCCX format. The RCCX modules can be considered as copy number variants. These investigators found that the most common CYP21A2 mutation associated with the nonclassic form of CAH, V281L, was associated with high C4 copy number. A large CYP21A2 deletion leads to a complete loss of function mutation and the classic salt-losing form of CAH. The large deletion was associated with low C4 copy number. Monomodular RCCX with a short C4 gene is a risk factor for autoimmune disease and was found to be less frequent in patients with CAH compared to population estimates. The investigators concluded that CAH patients have increased C4 CNV, with mutation-specific associations that may be protective for autoimmune disease. Thus, CNV involving C4 may influence propensity to develop autoimmune disorders.

Insulin resistance

Han MS, Jung DY, Morel C, Lakhani SA, Kim JK, Flavell RA, Davis RJ. JNK expression by macrophages promotes obesity-induced insulin resistance and inflammation. Science. 2013 Jan 11;339(6116):218-22. doi: 10.1126/science.1227568. Epub 2012 Dec 6.

The cJun NH(2)-terminal kinase (JNK) signaling pathway contributes to inflammation and plays a key role in the metabolic response to obesity, including insulin resistance. This pathway participates in a cell signaling cascade that senses metabolic stress leading to activation of obesity-induced inflammatory responses. Hirosumi and colleagues (Nature 2002;420:333) generated JNK1 and JNK2 knock-out mice and reported that JNK2 knockout mice were protected from weight gain, whereas no metabolic effect was noted for the JNK1 knockout mice. In this report, Han and colleagues generated mice with selective JNK1 and JNK2 deficiencies in macrophages. They found that feeding a high-fat diet to control and JNK-deficient mice caused similar obesity. Only mice with JNK-deficient macrophages, however, remained insulin-sensitive. Despite the improved insulin sensitivity, other aspects of metabolic dysfunction such as circulating fatty acid concentrations were unaffected. The protection of mice with macrophage-specific JNK deficiency against insulin resistance was associated with reduced tissue infiltration by macrophages. It remains to be determined if myeloid JNK signaling contributes to insulin resistance and inflammatory responses to a similar extent in other obese animals, e.g. humans.