

**AE & PCOS
QUARTERLY PUBLICATION LIST
JULY- SEPT 2015**

Highlighted articles

Kauffman AS, Thackray VG, Ryan GE, Tolson KP, Glidewell-Kenney CA, Semaan SJ, Poling MC, Iwata N, Breen KM, Duleba AJ, Stener-Victorin E, Shimasaki S, Webster NJ, Mellon PL. A Novel Letrozole Model Recapitulates Both the Reproductive and Metabolic Phenotypes of Polycystic Ovary Syndrome in Female Mice. Biol Reprod. 2015 Sep;93(3):69.

Rodent models of PCOS commonly do not fully recapitulate the complexity of the human disorder. Recently, a PCOS rat model using letrozole (LET), a nonsteroidal aromatase inhibitor, was shown to exhibit multiple PCOS phenotypes, including metabolic features absent in other rodent models. Given the advantages of using genetic and transgenic mouse models, this study explored whether LET could produce a similar PCOS phenotype in mice. Pubertal female C57BL/6N mice were treated for 5 wk with LET (at about 2 mg/kg/day), which resulted in increased serum testosterone and normal diestrus levels of estradiol, similar to the hyperandrogenemia and follicular phase estrogen levels of PCOS women. As in PCOS, ovaries from LET mice were larger, polycystic, and lacked corpora lutea versus controls. Most LET females were acyclic, and all were infertile. LET females displayed high serum levels of LH and higher LHb mRNA in the pituitary. In contrast, serum FSH and FSHb were reduced in LET females, demonstrating differential effects on gonadotropins, as in PCOS. Within the ovary, LET females had higher CYP17, CYP19, and FSH receptor mRNA expression. In the hypothalamus, LET females had higher kisspeptin receptor mRNA expression, but lower progesterone receptor mRNA levels. LET females also gained more weight than controls, had increased abdominal adiposity and adipocyte size, elevated adipose inflammatory mRNA levels, and impaired glucose tolerance, mirroring the metabolic phenotype in PCOS women. This is the first report of a LET paradigm in mice that recapitulates both reproductive and metabolic PCOS phenotypes. One issue with such LET models, however, is the absence of obvious aromatase deficiency in PCOS women. For example, highly successful first line treatments for ovulation induction in PCOS now increasingly use LET to induce endogenous gonadotropin stimulation of ovarian follicle growth commonly resulting in pre-ovulatory estradiol stimulation of an LH surge and ovulation enabling subsequent pregnancy. Further, elevated endometrial aromatase and over-exposure to estradiol are linked to endometrial hyperplasia in PCOS. So, while LET animal models are useful for understanding reproductive endocrine and metabolic complications of estrogen deficient conditions, their relevance to PCOS is unclear.(DA)

Legro RS, Dodson WC, Kris-Etherton PM, Kunselman AR, Stetter CM, Williams NI, Gnatuk CL, Estes SJ, Fleming J, Allison KC, Sarwer DB, Coutifaris C, Dokras A. Randomized Controlled Trial of Preconception Interventions in Infertile Women With Polycystic Ovary Syndrome. J Clin Endocrinol Metab. 2015Nov;100(11):4048-58. doi: 10.1210/jc.2015-2778. Epub 2015 Sep 24. PubMed PMID:26401593.

There is a general consensus that weight loss in PCOS improves ovulation, pregnancy rate and live birth, but the ability to show this in randomized clinical trials has been difficult. Most trials have been significantly underpowered for live birth rates. In this large RCT, Dr. Legro and colleagues compared an intensive weight loss regimen with lifestyle modification combined with medications for weight loss if indicated to a short course of oral contraceptives alone or in combination with the weight loss regimen for the impact on ovulation and pregnancy rates in overweight or obese women with PCOS. They randomized 159 women and achieved a little more than 6% weight reduction in the combined group and the weight loss only group. Cumulative ovulation rates were superior after weight loss: OCP, 46%; Lifestyle, 60%; and Combined, 67% (P < .05). Live birth rates trended toward higher rates

in the weight loss groups compared to the OCP, but there was no difference between the combined and weight loss only group: OCP, 12%; Lifestyle, 26%; and Combined, 24% ($P = .13$). While the study had to be terminated early by the DSMB because of the similarity in live birth rates in the two groups which achieved weight loss, as a value of information analysis supported that additional study would not lead to the ability to detect the difference the authors previously hypothesized, the authors noted “the study can be viewed as an important and innovative investigation that follows a group of infertile women to delivery and provides a model for future studies on this critical life transition. This publication supports the prior evidence from non-randomized trials that weight loss prior to conception improves the ovulatory rate and does not hinder the ability to achieve a pregnancy. In fact there was significant metabolic benefit seen in the weight loss groups and a detriment in the group assigned only to OCP, further supporting recommendations for lifestyle modification even if using OCP for cycle control in overweight or obese women with PCOS. Additional study is needed to see if pre-pregnancy weight reduction can be maintained during pregnancy and improve pregnancy complications that are associated with pregnancy in PCOS women. (KH)

Shim U, Kim HN, Lee H, Oh JY, Sung YA, Kim HL. Pathway Analysis Based on a Genome-Wide Association Study of Polycystic Ovary Syndrome. [PLoS One](https://doi.org/10.1371/journal.pone.0136609). 2015 Aug 26;10(8):e0136609. doi: 10.1371/journal.pone.0136609. eCollection 2015. PMID: 26308735. The genetic component of polycystic ovary syndrome (PCOS) is evident, but studies aiming to identify associated genes have shown controversial results. The authors used a dataset obtained through a previous genome-wide association study (GWAS), to elucidate the biological pathways that contribute to PCOS susceptibility and the associated genes. The GWAS data included 636,797 autosomal single nucleotide polymorphisms (SNPs) from 1,221 Korean women (432 PCOS patients and 789 controls). A meta-analysis gene-set enrichment of variant associations (MAGENTA) identified biological pathways or gene sets associated with PCOS. MAGENTA implements gene-set enrichment analysis (GSEA) associated with GWAS data. Top-ranking pathways or gene sets associated with PCOS were identified, and significant genes within the pathways were analyzed. GWAS dataset identified significant pathways related to oocyte meiosis and the regulation of insulin secretion by acetylcholine (ACh) and free fatty acids (FFAs). The significant genes involved in the pathway of oocyte meiosis were *SMC3* (structural maintenance of chromosome 3), *CCNE2* (cyclin E2), *PPP2R5D* (protein phosphatase 2, regulatory subunit B, delta), *INS* (insulin), *PPP2R5C* (protein phosphatase 2, regulatory subunit B, gamma), *PLCZ1* (phospholipase C, zeta 1), *PPP2R5A* (protein phosphatase 2, regulatory subunit B, alpha), *PPP1CB* (protein phosphatase 1, catalytic subunit, beta isozyme) and *SPDYA* (speedy/RINGO cell cycle regulator family member A). In addition, *INS*, *STXBP1* (syntaxin binding protein 1), *PLCB3* (phospholipase C, beta 3), *GNAQ* (guanine nucleotide binding protein, q polypeptide) and *PLCB2* (phospholipase C, beta 2) were identified in the pathways related to the regulation of insulin secretion by ACh and the regulation of insulin secretion by FFAs. They identified pathways and candidate genes involved in PCOS. These findings may provide new insight for understanding the mechanisms underlying the development of PCOS. However, there are some limitations of this study: 1) the number of women with PCOS included in the GWAS dataset is small; 2) most of the genes are partially unknown, and their biological function still needs to be established; 3) the study is confined to one ethnic group and different phenotypes of PCOS are seen in women with different ethnic backgrounds, so the results may not be generalizable to other ethnic groups. (CM)

List of Publications

Publications were searched in PubMed with primary search criteria congenital adrenal hyperplasia, premature adrenarche or PCOS with secondary subcategory, inclusive of the quarter dates. Every attempt was made to include all papers in English in these categories but may not be an exhaustive list. If a related paper was published in this quarter and not listed below, please notify the Publications Committee so that it may be include in an upcoming Quarterly Review.

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Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Alqahtani MA, Shati AA, Zou M, Alsuheel AM, Alhayani AA, Al-Qahtani SM, Gilban HM, Meyer BF, Shi Y. A Novel Mutation in the CYP11B1 Gene Causes Steroid 11β-Hydroxylase Deficient Congenital Adrenal Hyperplasia with Reversible Cardiomyopathy. *Int J Endocrinol.* 2015;2015:595164.

Ambroziak U, Kępczyńska-Nyk A, Kuryłowicz A, Małunowicz EM, Wójcicka A, Miśkiewicz P, Macech M. The diagnosis of nonclassic congenital adrenal hyperplasia due to 21-hydroxylase deficiency, based on serum basal or post-ACTH stimulation 17-hydroxyprogesterone, can lead to false-positive diagnosis. *Clin Endocrinol (Oxf).* 2015 Aug 31. doi: 10.1111/cen.12935. [Epub ahead of print]

Bachelot A, Golmard JL, Dulon J, Dahmoune N, Leban M, Bouvattier C, Cabrol S, Leger J, Polak M, Touraine P. Determining clinical and biological indicators for health outcomes in adult patients with childhood onset of congenital adrenal hyperplasia. *Eur J Endocrinol.* 2015 Aug;173(2):175-84.

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Premature Adrenarche

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PCOS - Adolescence

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PCOS Uterus/Endometrium

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