

reproductive complications such as recurrent abortions, gestational diabetes, intrauterine growth restriction, pregnancy induced hypertension that give rise to underweight newborns and condition metabolic diseases to adult life and increased risk of cancer, especially breast and endometrial cancer. Insulin resistance and hyperandrogenism are the most important etiopathogenic factors in PCOS. On the other hand, subjects exposed to an adverse microenvironment in the intrauterine stage develop compensating responses to survive, a process called fetal programming. Prenatal exposure to androgens and/or insulin resistance may act as fetal programming factors and cause restriction of intrauterine growth, obesity and insulin resistance in offspring. Newborn may have an increased risk of metabolic syndrome, increased incidence of hypertensive, type 2 diabetes, heart disease and cerebrovascular disease. Prevention of these complications will be achieved if women with Polycystic Ovary Syndrome are treated appropriately throughout their lives, but especially before and during their pregnancy. Only in this way can the risk of them be reduced, representing a better quality and greater life expectancy.

## Reproductive Endocrinology HYPERANDROGENIC DISORDERS THROUGHOUT THE LIFESPAN AND INTO THE NEXT GENERATION

### *Mother's PCOS Diagnosis Is Not Associated With Behavioral Symptoms in Offspring at 4-5 years*

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**Introduction:** Variation in prenatal sex steroid concentrations has been linked to child behavioral problems, with higher maternal total and free testosterone associated with child internalizing and externalizing symptoms. Polycystic Ovary Syndrome (PCOS) is a hyperandrogenic disorder that results in elevated testosterone levels during pregnancy among affected women. Population-based analyses suggest a higher risk of depression and anxiety diagnosis in children of women with PCOS. Animal models of prenatal hyperandrogenism further support an association with increased anxiety in offspring. Within the context of a multi-center U.S. pregnancy cohort, we examined early childhood behavioral and social responsiveness in children born to mothers with and without PCOS.

**Methods:** Pregnant women were recruited in their first trimester for The Infant Development and Environment Study (TIDES). PCOS status was determined by maternal reports of PCOS diagnosis or history of hirsutism/oligomenorrhea. Women who reported neither a history of PCOS or

symptoms of hirsutism or oligomenorrhea served as a comparison group. When participating children were age 4, mothers completed the Behavioral Assessment System for Children, 2<sup>nd</sup> Edition (BASC-2), a measure of child behavior problems, and the Social Responsiveness Scale, 2<sup>nd</sup> Edition (SRS-2), a measure of social impairment consistent with autistic traits. We fit linear regression models considering three outcomes: (1) BASC-2 externalizing composite score (e.g., hyperactivity, aggression); (2) BASC internalizing composite score (e.g., anxiety, depression, somatization); and (3) SRS-2 total score, adjusting for covariates, maternal age, child age, race, study center, income, education, alcohol and tobacco use, child sex, maternal depression. For all outcomes, higher scores indicate more problematic behaviors.

**Results:** A total of 360 mother/child pairs completed the 4-year assessment and were included in this analysis. This included 81 mothers with PCOS (37 male, 44 female) and 279 comparison mothers (132 male, 147 female). Mean maternal age at delivery was 30.7 years ( $\pm 4.7$ ) for PCOS cases and 31.9 years ( $\pm 5.4$ ) for comparison mothers. Interaction terms indicated no effect modification by child sex. In analyses combining both sexes, maternal PCOS was not associated with externalizing behaviors ( $\beta=1.81$ ; 95% CI: -2.37, 6.0;  $p=0.40$ ), internalizing behaviors ( $\beta=2.20$ ; 95% CI: -2.14, 6.53;  $p=0.32$ ), or social impairment ( $\beta=-0.34$ ; 95% CI: -3.34, 2.65;  $p=0.82$ ).

**Conclusions:** In this prospective evaluation, we observed no association between maternal PCOS and neurobehavioral symptoms in children at age 4. Given that prior literature using population databases suggested increased behavioral symptoms in school-aged and older children of PCOS mothers, these symptoms may become more apparent with development and continued assessment is warranted.

## Reproductive Endocrinology HYPERANDROGENIC DISORDERS THROUGHOUT THE LIFESPAN AND INTO THE NEXT GENERATION

### *MRNA Expression of Adipocytokines and Glucose Transporter Type 4 (GLUT4) in Adipose Tissue in Women With and Without Polycystic Ovary Syndrome*

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**Background:** The main feature of polycystic ovary syndrome (PCOS) is hyperandrogenism, adipocytes hypertrophy and chronic low-grade inflammation. It is known that PCOS is closely linked to functional dysfunction in adipose tissue, which in turn are connected with metabolic disturbances such as insulin resistance. However, there is no complete characterization of the adipose tissue in women with PCOS. **Aim:** To compare the expression of adipocytokines between women with and without PCOS. **Materials and Methods:** The total number of twenty two participants were enrolled into the study. Study group included ten women with PCOS diagnosed according to the Rotterdam criteria, the control group- twelve women