

Polycystic Ovarian Syndrome Conference

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Adipose tissue dysfunction in PCOS

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Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders and affects $\geq 7-9\%$ of reproductive-aged women. 60-70% of PCOS patients demonstrate insulin resistance (IR) above and beyond that predicted by body mass, race or age, resulting in compensatory hyperinsulinemia and an increased risk for Type-2 diabetes (T2DM) and cardiovascular disease. The underlying cellular mechanisms leading to IR in PCOS remain unclear. Subcutaneous adipose tissue (SAT) molecularly functions including the size of adipocytes, the stimulation of glucose transport, GLUT4 production, lipolysis, adipogenesis and insulin resistance related gene expression, adipokines secretion, adipogenesis, microRNAs profile appear to be defective in this disorder. However, no defects in insulin signaling have been found; including insulin signaling related genes expression, insulin binding, insulin receptor expression and the IRS-1/PI3K/AKT pathway. In genetic study, certain polymorphisms and SNPs have been found to be directly and indirectly associated with PCOS. Understanding the unique mechanisms of adipose tissue dysfunction in PCOS patients may point to potential new therapeutic avenues for this very common disorder.

Biography

Yen Hao Chen has obtained his PhD degree at the University of California, Riverside in 2006. He has joined Dr. Ricardo Azziz's lab in California and studied Polycystic Ovary Syndrome (PCOS) in 2008. He got his full-time tenure-track Faculty Position as an Assistant Professor on November 1, 2012 in Georgia Regents University. His research focuses on studying insulin resistance in adipose (fat) tissue from PCOS patients. He has discovered different microRNAs expression profile in adipose tissue from PCOS. Among these microRNAs, he found a specific small RNA called miR-93 which is over-expressed and may cause insulin resistance in fat tissue.

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