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ORAL PRESENTATON

Does the NLRP3 inflammasome drive inflammation in PCOS? Insights from human adipose tissue

Salih Atalah Alenezi
University of Nottingham, United Kingdom

Background: Polycystic ovary syndrome (PCOS) is a complex endocrine-metabolic condition affecting reproductive and metabolic health in women of reproductive age. Chronic low-grade inflammation is a key feature of PCOS, but its molecular drivers remain poorly understood. Among candidate mechanisms, the NLRP3 inflammasome, a central regulator of innate immune-mediated inflammation, has emerged as a potential contributor to PCOS pathology.

What was the key research question?

Does the NLRP3 inflammasome play a role in the pathogenesis of PCOS?

This study aimed to investigate whether the expression of NLRP3 and its associated inflammasome components is altered in the adipose tissue of women with PCOS, providing molecular insight into its potential involvement in disease mechanisms.

What was known before?

The NLRP3 inflammasome is activated in metabolic disorders such as obesity and type 2 diabetes, both of which are common in PCOS. It governs the maturation of IL-1β and IL-18, pro-inflammatory cytokines implicated in insulin resistance and systemic inflammation. While increased inflammasome activity has been reported in blood samples from PCOS patients, its status in adipose tissue, a metabolically active and immunologically relevant organ, has not been clearly defined.

What was done in this study?

In a case-control study, subcutaneous and visceral adipose tissue (SAT and VAT) biopsies were collected from PCOS patients and BMI-matched healthy controls. RT-qPCR was used to measure the mRNA expression of key inflammasome genes: **NLRP3, IL-1B, CASP1, and PYCARD**.

What were the main findings and role of chance?

No significant differences in gene expression of NLRP3 or its related components were observed between PCOS and control groups (all $p>0.05$). These findings suggest that NLRP3 is not transcriptionally upregulated in the adipose tissue of women with PCOS. However, the study's conclusions are tempered by potential limitations including sample size, adipose tissue variability, and the known disconnect between mRNA expression and protein function in inflammasome biology.

What is the interpretation and next step?

Although no transcriptional activation of the NLRP3 inflammasome was observed, this does not exclude a role for NLRP3 in PCOS pathogenesis. Functional validation using protein-level assays (e.g., Western blotting) and inflammasome activation studies are essential to determine whether post-transcriptional or post-translational regulation may still drive inflammatory signalling in PCOS.

Conclusion: This study provides preliminary evidence that NLRP3 gene expression is not elevated in adipose tissue from PCOS women. Nonetheless, the broader question of its role in PCOS remains open and warrants further investigation through **multi-level molecular and functional studies** to clarify its contribution and therapeutic potential.

Biography

Salih is a Clinical Lab Specialist at the Ministry of Health in the Kingdom of Saudi Arabia and a clinical PhD researcher at the University of Nottingham, UK. His doctoral research investigates the role of the NLRP3 inflammasome in polycystic ovary syndrome (PCOS) and its impact on female infertility and in vitro fertilization (IVF) outcomes. With a strong foundation in clinical laboratory science and a growing focus on reproductive immunology, Salih bridges the gap between diagnostics and translational research. His work aims to advance understanding of inflammation-driven reproductive disorders and support the development of innovative, evidence-based interventions in fertility treatment. Passionate about improving patient outcomes, Salih is committed to driving progress in reproductive medicine through scientific discovery, clinical application, and interdisciplinary collaboration. His dual expertise positions him at the forefront of efforts to enhance women's reproductive health, IVF success, and overall quality of life through precision medicine and targeted therapeutic strategies.

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