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Effect of metformin on insulin resistance (IR), insulin-like growth factor-1 (IGF-1) and insulin-like growth factor -binding protein- 1 (IGFBP-1) in obese females having acanthosis nigricans with or without polycystic ovarian syndrome

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Insulin resistance (IR) and associated hyperinsulinemia (HI) are the main pathogenic factors of acanthosis nigricans (AN) associated with obesity and or polycystic ovarian syndrome (PCOS). Metformin, as an insulin sensitizing drug, may offer a selective therapeutic approach for AN. We have evaluated the correlations of AN severity grade to body mass index (BMI), waist-to-hip ratio (WHR), IR, serum levels of insulin-like growth factor-1 (IGF-1) and IGF-1 binding protein (IGFBP-1) in obese females having AN with or without PCOS compared to obese females without AN and determine the efficacy of metformin in the treatment of AN. We concluded that PCOS is more prevalent among obese females with AN. So, obesity together with AN could be early predictors of PCOS in such patients. Also metformin therapy not only clinically improved AN lesions but also restores normal levels of insulin and decreases the pool of free-bioactive IGF-1 by increasing the levels of circulating IGFBP-1in AN patients with and without PCOS. Therefore, hyperinsulinemic women with AN+PCOS may benefit from a trial of metformin for improvement of skin lesions, signs and symptoms of PCOS and underlying HI.

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Expression of the RNA binding protein SAM 68 in granulosa cells after insulin and leptin induction in PCO women

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Introduction: Sam68 is a RNA-binding protein expressed in granulosa cells which may be recruited to insulin and leptin receptors. This study aimed to study the participation of Sam68 in granulosa cells of normal and PCO women in response to leptin and insulin in vitro.

Material & Methods: Granulosa cells obtained during follicle puncture from normal and PCO patients who underwent IVF cycles in the clinic IVI Sevilla are recruited and sent to Virgen Macarena University Hospital to be studied by immune precipitation and immune-blot of the phosphorylated proteins. Inmuno blot and qPCR were used to measure the expression level of Sam68 after insulin/leptin induction.

Results: Sam 68 is tyrosine phosphorylated by insulin or leptin stimulation in granulosa cells. Sam68 is recruited to the signaling complexes and reducing its RNA binding capacity. Besides, we found that both insulin and leptin amplify the expression of Sam68 in granulosa cells. Finally, full expression of Sam68 is needed for the activation of PI3K and MAPK signaling pathways by insulin or leptin in granulosa cells.

Conclusions: Leptin and insulin receptor signaling recruits Sam68. Both hormones induce its expression and Sam68 is required for the whole activation of the insulin and leptin receptor signal pathway in granulosa cells. Our data suggest that Sam 68 may be a new key element in the ovarian insulin resistance and the impaired fertility found in PCO women.

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