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## Metformin: Friend or foe of the polycystic ovary?

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Polycystic ovary syndrome (PCOS) is one of the most prevalent endocrine disorders in women. It is diagnosed by hyperandrogenemia, menstrual disturbances i.e., oligo- or anovulation, and/or polycystic ovaries on ultrasound (Rotterdam criteria). It is associated with insulin resistance, hyperinsulinemia and obesity. In the USA, the medical cost of its diagnosis and treatment during 2005 was around $4 billion, with a similar prevalence in the UK. With increasing rates of obesity, the number of women diagnosed with PCOS is likely to rise. The defining abnormality and the morphological feature of the polycystic ovary is the increase in the number of follicles compared with normal ovaries. The follicles in these ovaries over-produce androgens, leading to hyperandrogenemia. The high insulin levels present in a substantial number of women with insulin-resistant PCOS further exacerbate the over-production of androgens. Metformin, an insulin sensitizer, is widely prescribed in PCOS is primarily aimed at improving insulin sensitivity. It is well established however, that metformin can decrease hyperandrogenemia, improve menstrual cycles and ovulation rates without major changes of systemic insulin sensitivity. Could metformin act directly on the ovary? This talk will outline numerous lines of evidence from in vitro studies on human ovarian cells, showing that metformin directly alters ovarian steroidogenesis- in terms of androgen and oestrogen output. It can also alter insulin signaling in these cells affecting glucose uptake and hence potentially follicle growth. We will discuss whether this can be translated to an evidence-based approach to metformin's use in treating women with PCOS.

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