

# Researchers find out why polycystic ovary syndrome and diabetes are linked

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Nearly 50 percent of women with polycystic ovary syndrome (PCOS) develop pre-diabetes or type 2 diabetes before the age of 40, but the reasons for the correlation was unclear. In a new study in the *American Journal of Physiology–Endocrinology and Metabolism*, researchers report that inflammation is the cause for the increased diabetes risk in women with PCOS. Understanding how PCOS leads to diabetes will help identify ways to intervene.

Eating causes a sharp rise in glucose level in the blood, which the body absorbs with insulin. Insulin is produced by  $\beta$ -cells in the pancreas and released in two phases: just after a meal is consumed when insulin stored in the cells is released and later after the meal when newly synthesized insulin is released. Reduced ability to secrete insulin leads to a high [blood glucose level](#).

In patients with PCOS, the blood-glucose increase after eating stimulates immune cells to activate [inflammatory responses](#), which does not happen in lean women without PCOS. The [inflammation](#) process impedes the action of insulin, leading to insulin resistance and the development of type 2 diabetes, but some studies have suggested that inflammation can also disrupt the secretion of insulin. Studies have also reported that patients with PCOS have poorly functioning  $\beta$ -cells. The research team sought to determine if inflammation was the cause of the  $\beta$ -cell malfunction.

The researchers measured glucose-stimulated release of insulin and inflammatory proteins in women who had PCOS but normal blood glucose level. According to the researchers, obese women with PCOS had greater first and second phase  $\beta$ -cell impairment compared with women without PCOS. Furthermore, lean and obese patients with PCOS had greater activation of inflammatory responses compared with lean women without PCOS. Most importantly, the researchers wrote,  $\beta$ -cell function became more impaired as the

inflammation pathway became more active. "Our findings highlight the need for further investigation to determine the mechanism by which inflammation interacts with the pancreatic  $\beta$ -cells to increase [diabetes risk](#) in PCOS," they wrote.

The article "Pancreatic  $\beta$ -cell dysfunction in [polycystic ovary syndrome](#): role of hyperglycemia-induced nuclear factor- $\kappa$ B activation and systemic inflammation" is published in the *American Journal of Physiology–Endocrinology and Metabolism*. It is highlighted as one of this month's "best of the best" as part of the American Physiological Society's APSselect program.

**More information:** "Pancreatic  $\beta$ -cell dysfunction in polycystic ovary syndrome: role of hyperglycemia-induced nuclear factor- $\kappa$ B activation and systemic inflammation." *Am J Physiol Endocrinol Metab* 308: E770 –E777, 2015. DOI: [10.1152/ajpendo.00510.2014](https://doi.org/10.1152/ajpendo.00510.2014)

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