# Article information:

Intrauterine hyperglycemia impairs endometrial receptivity via up-regulating SGK1 in diabetes - PubMed
<https://pubmed.ncbi.nlm.nih.gov/35287185/>

# Article summary:

1. Diabetes can adversely affect reproductive function, and SGK1 is up-regulated in multiple tissues of diabetic patients.

2. This study established a streptozotocin-induced diabetic mouse model to observe the effects of diabetes on endometrial SGK1 expression and endometrial receptivity.

3. Hyperglycemia was found to lead to diminished embryo implantation and dysregulated SGK1, LIF and MUC1, with hyperglycemia-activated SMAD2/3 being responsible for the enhancement of SGK1.

# Article rating:

Appears well balanced: The article presents the information in a reliable and balanced way, without biases and prejudices. The claims made in the article are well supported and, where applicable, all sides of the argument are given opportunity to present their point of view. The article appears trustworthy and reliable.

# Article analysis:

This article provides a comprehensive overview of the effects of diabetes on endometrial receptivity via up-regulating SGK1 in diabetes. The authors have conducted extensive research into the topic, including establishing a streptozotocin-induced diabetic mouse model and injecting the uterine lumen of normal mice with high-glucose solution as well as culturing endometrial cells in high-glucose medium to mimic intrauterine hyperglycemia. The results from these experiments provide compelling evidence that hyperglycemia could lead to diminished embryo implantation and dysregulated SGK1, LIF and MUC1. Additionally, through over-expression of SGK1 in vivo and in vitro, it was found that enhanced SGK1 also decreased LIF expression, increased MUC1 expression, and attenuated embryo implantation rate. Furthermore, it was identified that hyperglycemia-activated SMAD2/3 might be responsible for the enhancement of SGK1.

The article is generally reliable and trustworthy due to its comprehensive research methods used to investigate the effects of diabetes on endometrial receptivity via up-regulating SGK1 in diabetes. The authors have provided detailed explanations for their findings as well as potential sources for any biases or unsupported claims made throughout the article. Furthermore, all possible risks are noted throughout the article which adds to its trustworthiness and reliability. There does not appear to be any one-sided reporting or missing points of consideration within this article either which further adds to its trustworthiness and reliability.

# Topics for further research:

* Endometrial receptivity and diabetes
* Hyperglycemia and embryo implantation
* SMAD2/3 and SGK1
* Streptozotocin-induced diabetic mouse model
* Intrauterine hyperglycemia and LIF expression
* MUC1 expression and diabetes

# Report location:

<https://www.fullpicture.app/item/276ad9818f9863e833f5e745537d9fc9>