# Article information:

A regulatory network controlling ovarian granulosa cell death | Cell Death Discovery
[https://www.nature.com/articles/s41420-023-01346-9?utm\_source=xmol=affiliate=meta=DDCN\_1\_GL01\_metadata](https://www.nature.com/articles/s41420-023-01346-9?utm_source=xmol&utm_medium=affiliate&utm_content=meta&utm_campaign=DDCN_1_GL01_metadata)

# Article summary:

1. Transforming growth factor-β (TGF-β) signaling plays an important role in mammalian development and homeostasis.

2. MiR-187 directly targets the 5' UTR of the sow TGFBR2 gene, maintaining its stability and increasing its levels in granulosa cells.

3. MiR-187 suppresses apoptosis in granulosa cells by activating TGF-β signaling, and lncRNA NORHA suppresses TGFBR2 expression by sponging miR-187.

# 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:

The article “A Regulatory Network Controlling Ovarian Granulosa Cell Death” provides a comprehensive overview of the role of miR-187 in regulating TGFBR2 expression and its effects on ovarian granulosa cell death. The authors provide evidence for their claims through experiments such as luciferase reporter assays, subcellular localization assays, RNA pulldown assays, mRNA stability assays, western blotting, and FACS detection of apoptosis rate. The article is well written and easy to understand, with clear explanations of the methods used and results obtained from each experiment.

The article does not appear to be biased or one-sided; it presents both sides equally by providing evidence for both miR-187's role as an activator of TGF-β signaling and lncRNA NORHA's role as a suppressor of TGFBR2 expression. Furthermore, the authors have explored counterarguments by discussing potential modifying factors that can activate or maintain levels of activated TGFBR2 protein through neddylation, ubiquitination, glycosylation modifications, and lysosomal degradation.

The article does not appear to contain any promotional content or partiality; it is focused solely on presenting scientific evidence for its claims without any bias towards any particular viewpoint or opinion. Additionally, possible risks are noted throughout the article; for example, dysregulation of TGF-β signaling can result in lethality, embryo-fetal malformations, and multiple diseases [3].

In conclusion, this article is reliable and trustworthy due to its comprehensive coverage of the topic at hand and lack of bias or promotional content.

# Topics for further research:

* Ovarian Granulosa Cell Death
* miR-187 Regulation
* TGFBR2 Expression
* Luciferase Reporter Assays
* Neddylation Modifications
* Embryo-Fetal Malformations

# Report location:

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