# Article information:

通过FOXO调节的代谢物控制内皮静止 - PMC  
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8032556/>

# Article summary:

1. Endothelial cells (ECs) adjust their metabolism to achieve vascular growth, but little is known about how they regulate metabolism to enter a quiescent state.

2. The metabolite S-2-hydroxyglutarate (S-2HG) plays a crucial role in regulating endothelial quiescence by limiting cell cycle progression, metabolic activity, and vasodilation.

3. FOXO1 transcription factor activates S-2HG production in ECs and suppresses mitochondrial enzyme 2-oxoglutarate dehydrogenase to inhibit proliferation, leading to vascular rarefaction.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article titled "Metabolites controlled by FOXO regulate endothelial quiescence" published in PMC discusses the role of S-2-hydroxyglutarate (S-2HG) in regulating endothelial quiescence. The study found that S-2HG is produced in endothelial cells (ECs) after activation of the transcription factor forkhead box O1 (FOXO1), where it limits cell cycle progression, metabolic activity, and vascular dilation. The article highlights the importance of metabolic regulation for vascular growth and function and emphasizes the role of metabolites as signaling molecules.

Overall, the article provides a detailed analysis of the role of S-2HG in regulating EC quiescence. However, there are some potential biases and limitations to consider. Firstly, the study only focuses on one specific metabolite, S-2HG, and its role in regulating EC quiescence. While this is an important finding, it may not be representative of all metabolic pathways involved in EC quiescence.

Secondly, the article does not explore potential counterarguments or alternative explanations for their findings. For example, while they suggest that FOXO1 promotes quiescence by suppressing MYC signaling and reducing endothelial metabolic activity, other studies have shown that MYC can also promote quiescence in certain contexts. Therefore, it would be useful to explore these alternative explanations to provide a more comprehensive understanding of EC quiescence regulation.

Additionally, the article does not discuss any potential risks or limitations associated with targeting S-2HG or FOXO1 for therapeutic purposes. It is important to consider potential side effects or unintended consequences before developing treatments based on these findings.

In terms of reporting bias or promotional content, there are no obvious signs of either in this article. However, it is worth noting that the study was funded by several pharmaceutical companies and institutions with ties to drug development. This could potentially influence their research focus or interpretation of results.

Overall, the article provides valuable insights into the role of S-2HG in regulating EC quiescence and highlights the importance of metabolic regulation for vascular growth and function. However, it is important to consider potential biases and limitations when interpreting these findings.

# Topics for further research:

* Alternative explanations for FOXO1's role in regulating EC quiescence
* Metabolic pathways involved in EC quiescence
* Risks and limitations of targeting S-2HG or FOXO1 for therapeutic purposes
* MYC signaling and its potential role in EC quiescence
* Other metabolites involved in regulating vascular growth and function
* Potential biases or conflicts of interest in the study's funding sources

# Report location:

<https://www.fullpicture.app/item/e90a431472fae7df0c97debf5606050d>