# Article information:

Hydrogen Sulfide Modulates Endothelial–Mesenchymal Transition in Heart Failure | Circulation Research
[https://www.ahajournals.org/doi/full/10.1161/CIRCRESAHA.122.321326?rfr\_dat=cr\_pub++0pubmed=Z39.88-2003=ori%3Arid%3Acrossref.org](https://www.ahajournals.org/doi/full/10.1161/CIRCRESAHA.122.321326?rfr_dat=cr_pub++0pubmed&url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org)

# Article summary:

1. Hydrogen sulfide (H2S) is an endogenous physiological signaling molecule that has protective effects in the cardiovascular system.

2. Endothelial-specific CSE knockout results in increased expression of key regulatory genes in the Endothelial-Mesenchymal transition pathway, leading to elevated cardiac interstitial and perivascular fibrosis in a transverse aortic constriction (TAC)-induced heart failure model.

3. Modulation of endothelium-derived H2S production may represent a novel therapeutic strategy for the treatment of heart failure with reduced ejection fraction and, more broadly, cardiovascular diseases involving endothelial-mesenchymal transition.

# 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 “Hydrogen Sulfide Modulates Endothelial–Mesenchymal Transition in Heart Failure | Circulation Research” is generally reliable and trustworthy as it provides evidence from multiple studies to support its claims. The article cites previous research to back up its assertions about the role of hydrogen sulfide (H2S) as an endogenous physiological signaling molecule with protective effects in the cardiovascular system, as well as its potential role in modulating endothelial–mesenchymal transition (EndoMT). Furthermore, the article provides evidence from mouse models demonstrating that endothelial cell (EC)-specific CSE knockout results in increased expression of key regulatory genes in the EndoMT pathway, leading to elevated cardiac interstitial and perivascular fibrosis in a transverse aortic constriction (TAC)-induced heart failure model. Additionally, it suggests that modulation of endothelium-derived H2S production may represent a novel therapeutic strategy for treating heart failure with reduced ejection fraction and other cardiovascular diseases involving EndoMT.

The article does not appear to have any major biases or one-sided reporting; however, there are some minor issues worth noting. For example, while the article does provide evidence from multiple studies to support its claims, it does not explore any counterarguments or alternative explanations for its findings. Additionally, while it does mention potential risks associated with modulating H2S production, it does not provide any detailed information about these risks or how they can be mitigated. Finally, while the article does present both sides of the argument equally, it could benefit from providing more detail about each side's position on this issue.

In conclusion, overall this article is reliable and trustworthy due to its use of evidence from multiple studies to support its claims; however, there are some minor issues worth noting such as lack of exploration into counterarguments or alternative explanations for its findings and lack of detail regarding potential risks associated with modulating H2S production.

# Topics for further research:

* Endothelial–Mesenchymal Transition
* Cardiovascular Diseases Involving EndoMT
* CSE Knockout
* Transverse Aortic Constriction
* Therapeutic Strategies for Heart Failure
* Mitigating Risks of Modulating H2S Production

# Report location:

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