# Article information:

DsbA-L interacts with VDAC1 in mitochondrion-mediated tubular cell apoptosis and contributes to the progression of acute kidney disease - ScienceDirect
<https://www.sciencedirect.com/science/article/pii/S2352396422000433>

# Article summary:

1. DsbA-L is involved in the progression of acute kidney injury (AKI).

2. DsbA-L interacts with VDAC1 to induce renal cell apoptosis and mitochondrial injury.

3. The PT-DsbA-L-KO mice showed amelioration of AKI progression via the downregulation of VDAC1.

# 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:

This article provides a detailed overview of the role of DsbA-L in acute kidney injury (AKI). The authors provide evidence that DsbA-L interacts with VDAC1 to induce renal cell apoptosis and mitochondrial injury, and that this interaction can be used as a potential therapeutic target to attenuate the pathological effects caused by AKI. The authors also present data from experiments conducted on PT-DsbA-L and VDAC1 KO mice, which demonstrate that these proteins are involved in the progression of AKI.

The article is generally reliable and trustworthy, as it provides evidence for its claims through experiments conducted on both mouse models and human biopsies taken from patients with AKI. Furthermore, the authors provide a comprehensive list of funding sources for their research, which adds to its credibility. However, there are some points that could be improved upon in order to make the article more reliable and trustworthy. For example, while the authors discuss potential therapeutic targets for AKI based on their findings, they do not explore any possible risks associated with such treatments or interventions. Additionally, while they discuss potential counterarguments to their findings, they do not provide any evidence or data to support these counterarguments. Finally, while they provide a comprehensive list of funding sources for their research, they do not mention any potential conflicts of interest that may have influenced their results or conclusions.

# Topics for further research:

* Acute kidney injury therapeutic targets
* Mitochondrial injury in AKI
* VDAC1 knockout mice
* Potential risks of AKI treatments
* Evidence for counterarguments to AKI research
* Conflicts of interest in AKI research

# Report location:

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