# Article information:

Myeloid-derived MIF drives RIPK1-mediated cerebromicrovascular endothelial cell death to exacerbate ischemic brain injury | PNAS
[https://www.pnas.org/doi/10.1073/pnas.2219091120?url\_ver=Z39.88-2003=ori:rid:crossref.org=cr\_pub%20%200pubmed](https://www.pnas.org/doi/10.1073/pnas.2219091120?url_ver=Z39.88-2003&rfr_id=ori:rid:crossref.org&rfr_dat=cr_pub%20%200pubmed)

# Article summary:

1. Macrophage migration inhibitory factor (MIF) is a multifaceted protein that plays an important role in multiple inflammatory conditions.

2. This study investigates the role of MIF in promoting cerebromicrovascular endothelial cell death under inflammatory conditions.

3. The results suggest that MIF and RIPK1 can be used as potential therapeutic targets for preserving cerebromicrovascular EC survival and BBB integrity in CNS pathologies promoted by peripheral inflammation.

# 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 comprehensive overview of the role of macrophage migration inhibitory factor (MIF) in promoting receptor-interacting protein kinase 1 (RIPK1)-mediated cell death under oxygen-glucose deprivation condition, with implications for perioperative stroke (PIS). The authors provide evidence from both human and mouse studies to support their claims, which adds to the trustworthiness and reliability of the article. Furthermore, they provide detailed information on the methods used in their experiments, which allows readers to assess the validity of their findings.

However, there are some potential biases present in this article that should be noted. For example, the authors do not explore any counterarguments or alternative explanations for their findings, which could lead to a one-sided reporting of their results. Additionally, while they provide evidence from both human and mouse studies, it is unclear if these results can be generalized to other populations or contexts. Finally, there is no discussion of possible risks associated with using MIF inhibitors or RIPK1 inhibitors as therapeutic targets for PIS, which should be addressed before any clinical applications are considered.

# Topics for further research:

* Risks associated with MIF inhibitors
* Risks associated with RIPK1 inhibitors
* Alternative explanations for MIF-RIPK1 cell death
* Generalizability of MIF-RIPK1 cell death findings
* Clinical applications of MIF-RIPK1 cell death
* Perioperative stroke treatment strategies

# Report location:

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