# Article information:

The Hyaluronidase, TMEM2, Promotes ER Homeostasis and Longevity Independent of the UPRER - ScienceDirect  
<https://www.sciencedirect.com/science/article/pii/S0092867419311687>

# Article summary:

1. A CRISPR-based knockout (KO) screen was used to identify genes important for cells to survive ER-based protein misfolding stress, which identified the cell-surface hyaluronidase (HAase), Transmembrane Protein 2 (TMEM2), as a potent modulator of ER stress resistance.

2. TMEM2 alters intracellular ER stress resistance through changes in ECM metabolism and promotes ER stress resistance independent of UPRER through MAPK signaling.

3. Ectopic expression of human TMEM2 promotes lifespan and immunity in C. elegans independent of canonical UPRER activation but dependent on the ERK ortholog mpk-1 and the p38 ortholog pmk-1.

# 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 is generally reliable and trustworthy, as it provides evidence from multiple sources such as Cell Metabolism, Volume 30, Issue 6, 3 December 2019, Cells Volume 179, Issue 6, 27 November 2019, and Highlight sections that support its claims. The article also provides detailed information on the methods used to conduct the research such as CRISPR-Cas9 screening in human fibroblasts and ectopic expression of human TMEM2 in C. elegans. Furthermore, it presents both sides of the argument by discussing how TMEM2 alters intracellular ER stress resistance through changes in ECM metabolism while also promoting ER stress resistance independent of UPRER through MAPK signaling.

However, there are some potential biases present in the article that should be noted. For example, there is a lack of discussion about possible risks associated with using CRISPR-Cas9 technology or ectopic expression of human TMEM2 in C. elegans that could potentially lead to unintended consequences or side effects that were not discussed in the article. Additionally, there is a lack of exploration into counterarguments or alternative explanations for the findings presented in the article which could provide further insight into its claims and conclusions. Finally, there is a lack of evidence provided for some of the claims made throughout the article which could weaken its overall reliability and trustworthiness if not addressed properly.

# Topics for further research:

* Risks associated with CRISPR-Cas9 technology
* Side effects of ectopic expression of human TMEM2
* Counterarguments to TMEM2 altering intracellular ER stress resistance
* Alternative explanations for TMEM2 promoting ER stress resistance
* Evidence for TMEM2 altering ECM metabolism
* Evidence for TMEM2 promoting MAPK signaling

# Report location:

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