# Article information:

Journal of Cellular Physiology | Cell Biology Journal | Wiley Online Library
<https://onlinelibrary.wiley.com/doi/10.1002/jcp.31073>

# Article summary:

1. Programmed cell death (PCD) has been a hot topic in research, with various mechanisms such as necroptosis, pyroptosis, ferroptosis, and cuproptosis being discovered.

2. Necroptosis, a form of inflammatory PCD mediated by mixed lineage kinase domain-like protein (MLKL), has gained increasing attention due to its role in disease progression and development.

3. Bacterial infections can induce necroptosis, serving as both a defense mechanism against infection and a promoter of bacterial escape and inflammation. The involvement and role of necroptosis in apical periodontitis (AP) is still not fully understood.

# Article rating:

Appears strongly imbalanced: The article is written in a biased or one-sided way, and the information it provides is not trustworthy enough to be considered a reliable source. You should consult other sources to find reliable information on the presented issues.

# Article analysis:

The article titled "Journal of Cellular Physiology | Cell Biology Journal | Wiley Online Library" provides an overview of the latest research on necroptosis, a form of programmed cell death. While the article covers various aspects of necroptosis and its role in diseases, there are several areas where critical analysis is warranted.

Firstly, the article lacks proper citations for the claims made. It mentions that necroptosis has been found to play a key role in disease progression and development but does not provide specific studies or evidence to support this statement. Without proper references, it is difficult to evaluate the validity of these claims.

Additionally, the article seems to have a biased focus on bacterial pathogens and their induction and regulation of necroptosis. While this may be an important aspect to consider, it fails to explore other potential causes or triggers of necroptosis, such as viral infections or genetic factors. This one-sided reporting limits the comprehensiveness of the article and may lead to an incomplete understanding of necroptosis.

Furthermore, the article repeats certain points multiple times without providing new information or expanding on them. For example, it repeatedly discusses how bacterial pathogens induce and regulate necroptosis without delving into specific mechanisms or providing supporting evidence. This repetition adds unnecessary length to the article without adding substantial value.

Moreover, there is a lack of discussion on potential risks or drawbacks associated with targeting necroptosis as a therapeutic strategy. While the article briefly mentions potential therapeutic strategies for necroptosis in apical periodontitis (AP), it does not address any potential side effects or limitations of these approaches. A more balanced analysis would consider both the benefits and risks associated with targeting necroptosis for treatment.

Overall, while the article provides some insights into current research on necroptosis and its involvement in AP, it falls short in terms of providing comprehensive evidence-based analysis. The lack of proper citations, one-sided reporting, repetition of information, and failure to address potential risks are notable limitations. A more thorough and balanced examination of the topic would enhance the credibility and usefulness of the article.

# Topics for further research:

* Mechanisms of necroptosis in viral infections
* Genetic factors influencing necroptosis
* Alternative triggers of necroptosis beyond bacterial pathogens
* Studies on the role of necroptosis in disease progression and development
* Potential side effects of targeting necroptosis as a therapeutic strategy
* Limitations of current therapeutic approaches for necroptosis in apical periodontitis

# Report location:

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