# Article information:

Peptide analogues PKHB1 and 4N1K induce cell death through CD47‐independent mechanisms - Leclair - 2020 - Cancer Science - Wiley Online Library
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# Article summary:

1. Uscanga-Palomeque et al reported that PKHB1, a peptide analog of 4N1K with increased stability in serum, directly induced T-leukemic cell death by engaging the CD47 receptor.

2. It is well documented that CD47 ligation with antibodies can invoke cell death in the absence of effector cells.

3. PKHB1 and 4N1K induce CD47-independent cell death in Jurkat and MOLT4 cells, as evidenced by decreased size and increased granularity of treated cells, as well as significant annexin V binding.

# Article rating:

Appears moderately imbalanced: The article provides some useful information, but is missing several important points or pieces of evidence that would be required to present the discussed topics in a balanced and reliable way. You are encouraged to seek a more balanced perspective on the presented issues by exploring the provided research topics and looking at different information sources.

# Article analysis:

The article provides an interesting insight into the potential of peptide analogues PKHB1 and 4N1K to induce cell death through CD47-independent mechanisms. The authors provide evidence for their claims through experiments conducted on WT and CD47−/− Jurkat and MOLT4 cell lines, showing that both peptides induced significant cell death in a CD47-independent manner. However, there are some points of consideration that should be taken into account when assessing the trustworthiness and reliability of this article.

First, the authors do not provide any evidence for their claim that “CD47 is commonly upregulated in tumor cells” or any other sources to back up this statement. This could lead to readers making assumptions about the prevalence of CD47 upregulation without any supporting evidence. Additionally, while the authors do mention previous studies conducted on 4N1K which showed its propensity to bind nonspecifically to proteins in vitro as well as to the plasma membrane, they do not explore any counterarguments or alternative explanations for these findings which could weaken their argument for PKHB1 having similar effects.

Furthermore, while the authors do mention possible risks associated with using these peptides (such as nonspecific antibody binding), they do not provide any further information on how these risks can be mitigated or avoided when using them in clinical settings. Additionally, it is unclear whether all possible side effects have been explored or if there are any long-term implications associated with using these peptides which could potentially affect patient safety if used clinically.

In conclusion, while this article provides an interesting insight into the potential use of peptide analogues PKHB1 and 4N1K to induce cell death through CD47-independent mechanisms, there are some points of consideration which should be taken into account when assessing its trustworthiness and reliability such as lack of evidence for certain claims made by the authors and lack of exploration into counterarguments or alternative explanations for findings presented in this article.

# Topics for further research:

* CD47 upregulation in tumor cells
* Nonspecific antibody binding risks
* Long-term implications of peptide analogues
* Mitigation of peptide analogues risks
* Alternative explanations for 4N1K findings
* Clinical applications of peptide analogues

# Report location:

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