# Article information:

NUF2 overexpression contributes to epithelial ovarian cancer progression via ERBB3-mediated PI3K-AKT and MAPK signaling axes - PMC
<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9811817/>

# Article summary:

1. NUF2 is upregulated in epithelial ovarian cancer (EOC) and is associated with poor prognosis.

2. NUF2 downregulation decreases cell proliferation, migration, invasion and tumor growth in EOC.

3. NUF2 promotes EOC progression through ERBB3-induced activation of the PI3K-AKT and MAPK signaling axes.

# 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 “NUF2 overexpression contributes to epithelial ovarian cancer progression via ERBB3-mediated PI3K-AKT and MAPK signaling axes” is a well-written and comprehensive review of the role of NUF2 in EOC progression. The authors provide evidence from both clinical studies and laboratory experiments to support their claims that NUF2 is upregulated in EOC tissues and cell lines, and that its downregulation can inhibit cell proliferation, migration, invasion, and tumor growth in vivo. Furthermore, they demonstrate that NUF2 promotes EOC progression through ERBB3-induced activation of the PI3K-AKT and MAPK signaling pathways.

The article does not appear to have any major biases or unsupported claims. All of the evidence presented is supported by data from clinical studies or laboratory experiments, which makes it reliable and trustworthy. The authors also provide a thorough discussion of their findings, exploring potential mechanisms for how NUF2 may be involved in EOC progression as well as possible therapeutic implications for targeting this protein in future treatments for ovarian cancer patients.

The only potential issue with the article is that it does not explore any counterarguments or alternative explanations for the observed effects of NUF2 on EOC progression. While this does not necessarily detract from the overall quality of the article, it would have been beneficial to include some discussion of other possible explanations for these effects or potential limitations to their findings.

# Topics for further research:

* NUF2 ovarian cancer therapeutic implications
* ERBB3-mediated PI3K-AKT signaling pathways
* NUF2 expression regulation mechanisms
* NUF2 role in EOC metastasis
* NUF2-induced cell migration pathways
* NUF2-targeted drug development strategies

# Report location:

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