# Article information:

Inhibition of guanosine monophosphate synthetase (GMPS) blocks glutamine metabolism and prostate cancer growth - Wang - 2021 - The Journal of Pathology - Wiley Online Library
<https://onlinelibrary.wiley.com/doi/10.1002/path.5665>

# Article summary:

1. Glutamine is an important nutrient for cancer cells, and its availability is regulated by the glutamine transporter ASCT2.

2. Glutamine can be used for the TCA cycle or to synthesize purine nucleotides through the de novo biosynthetic pathway.

3. Guanosine monophosphate synthetase (GMPS) is one of three glutamine amidotransferases involved in de novo purine biosynthesis and has been shown to be overexpressed in metastatic human melanoma cells, suggesting it may be a potential therapeutic target in prostate cancer.

# 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 “Inhibition of guanosine monophosphate synthetase (GMPS) blocks glutamine metabolism and prostate cancer growth” by Wang et al., published in The Journal of Pathology in 2021, provides an overview of the role of GMPS in prostate cancer growth and suggests that it may be a potential therapeutic target. The authors provide evidence from clinical samples, cell lines, bioinformatics analysis, statistical analysis, immunohistochemistry, lentiviral shRNA expression, western blotting, cell viability assays, cell growth assays (IncuCyte), cell counting, BrdU incorporation assays, apoptosis assays, immunofluorescence staining of cells, cell metabolism assays, in vitro l-[15N-(amide)]glutamine and l-[U-13C5]glutamine tracing and metabolite extraction as well as targeted metabolomics using LC–MS to support their claims.

The article appears to be reliable overall; however there are some points that could have been addressed more thoroughly. For example, the authors do not discuss any potential risks associated with targeting GMPS or any possible side effects that could arise from such treatments. Additionally, while they provide evidence from multiple sources to support their claims about GMPS expression being increased in prostate cancer compared to normal tissue samples as well as correlating with higher Gleason scores and metastatic castration-resistant prostate cancer cell cycle genes CDK1 CDC20 and UBE2C; they do not explore any counterarguments or present both sides equally which could have strengthened their argument further.

In conclusion this article appears to be reliable overall but could have been improved by exploring counterarguments more thoroughly and discussing potential risks associated with targeting GMPS therapeutically

# Topics for further research:

* Potential risks of targeting GMPS therapeutically
* Counterarguments to GMPS expression in prostate cancer
* Side effects of targeting GMPS
* Clinical implications of targeting GMPS
* Metabolomics analysis of GMPS expression
* Glutamine metabolism and prostate cancer growth

# Report location:

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