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Middle East Research Journal of Medical Sciences
ISSN: 2789-7699 (Print) & ISSN: 2958-2024 (Online)
Frequency: Bi-Monthly
DOI: https://doi.org/10.36348/merjms.2025.v05i06.003

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Aspirin and Pancreatic Ductal Adenocarcinoma: A Comprehensive Narrative Review of Chemoprevention and Clinical Outcomes

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Letter to Editor

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How to cite this paper:
Hadia Ishtiaq & Nadia Ishtiaq (2025). Aspirin and Pancreatic Ductal Adenocarcinoma: A Comprehensive Narrative Review of Chemoprevention and Clinical Outcomes. *Middle East Res J. Med. Sci*, *5*(6): 394-395.

Article History:
| Submit: 16.10.2025 |
| Accepted: 14.11.2025 |
| Published: 18.11.2025 |

To The Editor,

We read with great interest the narrative review by Azan Khan *et al*., titled "Aspirin and Pancreatic Ductal Adenocarcinoma: A Comprehensive Narrative Review of Chemoprevention and Clinical Outcomes"[1]. As final-year medical students passionate about immunology and oncology, we found the authors' discussion of the complex relationship between aspirin and PDAC to be both enlightening and clinically relevant. The sections on targeting the "undruggable" KRAS mutation and the role of diabetes are especially informative.

This review prompted us to consider this topic in relation to the modern immunology curriculum. While the authors have skillfully explained the COX-dependent and COX-independent pathways, we would like to suggest an important area for future research: the potential role of aspirin in changing the immune environment of PDAC.

The authors have correctly described the PDAC tumor microenvironment (TME) as immunosuppressive. Building on this, the well-known effect of aspirin on platelets may play a significant role in reducing metastasis, beyond its cardiovascular benefits. Platelets cover circulating tumor cells, protecting them from immune detection by Natural Killer (NK) cells[2]. By disrupting this platelet-mediated protection, aspirin may help "unmask" cancer cells, allowing the immune system to recognize and destroy them, thereby preventing metastasis.

Moreover, recent studies suggest that aspirin can help reduce inflammation by promoting pro-resolving mediators, such as resolvins, which can affect macrophage behavior within the TME[3]. This finding represents a compelling non-COX-related mechanism that supports the pathways discussed in this narrative review.

Therefore, the "precision prevention" strategy suggested by Khan *et al*., could be significantly strengthened by exploring biomarkers associated with the host immune response. For instance, are the benefits of aspirin more significant in patients with specific immune profiles, such as those with high baseline NK cell activity or distinct platelet characteristics? Understanding this could help identify the best candidates for aspirin therapy.

We thank the authors for their insightful review. It adeptly links pharmacological knowledge to complex clinical challenges. Considering aspirin's potential to affect the immune system may open new possibilities for its use, not only in prevention but also as a complementary treatment to enhance the effectiveness of new immunotherapies in PDAC.

Author Contributions: Both authors contributed equally to the conception and writing of this letter.

Conflict of Interest: There are no conflicts of interest to declare.

Funding Statement: No specific funding was received for this work.

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