

METABOLIC TRIGGERS OF A SILENT KILLER: RETHINKING PANCREATIC CANCER RISK THROUGH THE LENS OF DIABETES AND OBESITY

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Abstract

Pancreatic cancer remains one of the deadliest malignancies globally, with late diagnosis and aggressive progression contributing to its poor prognosis. Recent studies continue to strengthen the evidence that metabolic disorders, specifically diabetes and obesity, significantly influence both the risk and outcomes of pancreatic cancer.

INTRODUCTION

A recurring theme across multiple investigations is the association between new-onset diabetes and an increased risk of pancreatic cancer. Li et al. (2025) highlights the importance of recognizing new-onset diabetes as a potential early marker for pancreatic malignancy, suggesting that diabetes diagnosis could serve as an opportunity for early cancer detection. Similarly, an Australian cohort study focusing on women with new-onset diabetes developed predictive

models to identify those at higher risk of pancreatic cancer, emphasizing the need for vigilant monitoring in this subgroup.

Type 2 diabetes, a chronic metabolic condition, was shown by the University of Manchester's large-scale population study (2025) to nearly double the risk of pancreatic cancer, especially in women. This aligns with other epidemiological data suggesting that hyperinsulinemia, insulin resistance, and chronic

inflammation, common in diabetes, may create a pro-carcinogenic environment in the pancreas. Verywell Health (2025) further supports this biological plausibility, detailing how elevated blood glucose and insulin can promote tumorigenesis.

Obesity, often intertwined with diabetes, also emerges as a critical factor influencing pancreatic cancer risk. DeWitt et al. (2024) demonstrated that obesity affects not only cancer risk but also the anatomical distribution of pancreatic adenocarcinoma, which may have implications for diagnosis and treatment strategies. The review by MDPI (2024) delves deeper into the mechanistic pathways, showing how adipose tissue dysfunction, chronic systemic inflammation, and altered secretion of adipokines contribute to carcinogenesis in pancreatic tissue.

Interestingly, metabolically healthy obesity, a phenotype without typical metabolic dysfunctions, was explored by Ahmed et al. (2024), revealing that even in the absence of metabolic derangements, obesity alone may increase pancreatic cancer risk. This challenges the notion that only metabolically unhealthy obesity is relevant in cancer risk assessment. The interplay between metabolic interventions and cancer risk is also a key focus. Patel et al. (2024) performed a meta-analysis showing that bariatric surgery, which improves metabolic profiles and reduces obesity, may lower the incidence of pancreatic cancer, suggesting therapeutic avenues beyond glucose control.

Zhou et al. (2024) provided a global perspective, analysing the burden of pancreatic cancer attributable to metabolic risk factors. Their comprehensive assessment confirmed that the rising prevalence of diabetes and obesity significantly contributes to the global pancreatic cancer burden, underscoring the urgency of addressing metabolic health in cancer prevention strategies.

CONCLUSION

Diabetes and obesity are not merely comorbid conditions but active contributors to pancreatic carcinogenesis. They reinforce the critical need for integrating metabolic monitoring into pancreatic cancer screening and developing tailored prevention and management strategies targeting metabolic health.

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