# BAW 2026

## Multiscale Biology and Disease Pathways

### Insights into cells, systems and therapeutics

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**Jointly Organized by**

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Dr. **Nilabja Sikdar** (born 16 September 1974) is a molecular geneticist whose research focuses on the **genomics and genetic basis of cancer in the Indian population**. He completed his **Ph.D. in Human Genetics at the Indian Statistical Institute, Kolkata (1999–2005)**. Following his doctoral training, he pursued **postdoctoral research at the National Human Genome Research Institute (USA) from 2005 to 2010 and at the National Cancer Institute (USA) from 2010 to 2013**, where he worked on genomic mechanisms related to DNA repair, chromosomal rearrangements, and cancer biology. After returning to India, he served as a **Ramalingaswami Re-entry Fellow and Ramanujan Fellowship awardee (equivalent to Scientist-D) under the Department of Biotechnology, Government of India, from August 2013 to December 2022**. Since **June 2023**, he has been working as a **Scientist-G at the Estuarine and Coastal Studies Foundation**. Dr. Sikdar has led major genomics projects including a **comprehensive genetic characterization of pancreatic cancer in the Indian population** and studies on **somatic driver mutations in gall bladder cancer**, supported by national funding agencies. His research output includes **over 36 peer-reviewed research articles and 20 book chapters and reviews**, with **more than 1,400 citations and an h-index of 21**. He has also supervised several Ph.D. and postgraduate students and mentored numerous research trainees. Dr. Sikdar serves as an **Associate Section Editor for *Frontiers in Oncology* (Gastrointestinal Cancers section)** and is a **registered editorial board member of the *World Journal of Gastroenterology***. His contributions have been recognized through several national and international honors, including the **FARE (Fellows Award for Research Excellence) from the U.S. National Institutes of Health**, the **Keystone Symposia Scholarship**, and prestigious **Ramalingaswami and Ramanujan Re-entry Fellowships from the Government of India**.

# Multi-Omics Integration and Machine Learning Reveal Prognostic Methylation Biomarkers in Pancreatic Cancers

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Background and Objective: Pancreatic cancer (PanCa) continues to exhibit poor survival rates, underscoring the urgent need for novel prognostic markers and therapeutic targets. Here, we applied integrative computational approaches combining machine learning with multi-omics data to identify methylation-regulated differentially expressed genes (MeDEGs) that may hold prognostic and therapeutic relevance in PanCa.

Methods: Multi-omics datasets were obtained from TCGA-PAAD, GEO, and ICGC (RNA-Seq and 450K methylation arrays) and were analyzed by comparing pancreatic tumors and adjacent normal tissues. Differential expression and methylation detection analyses resulted in a final set of MeDEGs, which were further inspected through correlation filtering. Full PCA/K-means clustering was performed on MeDEGs and the risk evaluation model was developed using Adaptive LASSO Cox regression. Predictions were validated through Random Forest and kNN classifiers with assessments through AUC-ROC, Precision, Recall, F1-score and survival analyses in independent cohorts.

Results: A study resulted in through identification of 27 differentially expressed genes (MeDEGs) associated with altered methylation, in which three major pathways include lipid metabolism (MOGAT2) and immune modulation (CD68, MIA), and cancer signaling (MOGAT2). The classification model using Random Forest trained on the methylated data presented strong predictive performance (AUC-ROC = 0.924, Precision = 0.769, Recall = 0.748, F1 = 0.95, variance = 0.1091), and demonstrated stability and generalizability. A prognostic risk score (PRS) model based on Adaptive LASSO regression analysis identified five highly significant MeDEGs, an optimal $\lambda = 0.7588$, and and AUC of 0.66 for survival discrimination. Next, whole genome bisulfite sequencing (WGBS) validated results by confirming hypomethylation status of CD68 and MIA, and site-specific methylation in MOGAT2 gene associated with liver metastases. The original findings were further validated in independent GEO datasets: MOGAT2 ($p = 0.002$) and CD68 ($p = 0.001$) were significantly associated among the Western cohorts. Finally, molecular docking analyses predicted therapeutic interactions with the MeDEGs to epigallocatechin-gallate, dorzagliatin, and to 5-FU in that order.

Conclusions: Our study presents a computational framework for identifying methylation-driven biomarkers in pancreatic cancer. While the findings are promising, they remain preliminary and hypothesis-generating. Future experimental validation is essential to establish clinical utility.