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RESEARCH AREA

My research focuses on investigating the genetic background of tumors and their mutational patterns, with the aim of understanding how these factors influence antitumor immune responses. Tumor development is driven by genetic alterations; however, it is becoming increasingly clear that not only the number of mutations, but also their nature and underlying origins jointly determine the immunological behavior of tumors. Distinct mutational processes give rise to characteristic amino acid changes, which shape the protein composition of tumors and the signals that become visible to the immune system.

The altered proteins generated by somatic mutations can give rise to so-called neopeptides, which may be recognized by the immune system through antigen presentation pathways. The efficiency of this process, however, strongly depends on the genetic background of the individual, particularly on the variability of human leukocyte antigen (HLA) molecules. A central question of my research is how tumor-specific mutational patterns interact with patient-specific genetic features, and how this interaction ultimately shapes the quality of the antitumor immune response.

The overarching goal of my research is to establish a biologically grounded framework that contributes to a more precise understanding of tumor immunological behavior and supports the development of personalized immunotherapeutic strategies. Due to its interdisciplinary nature—integrating genomics, immunology, and computational data analysis—this research area offers students valuable insight into the conceptual and methodological foundations of modern cancer research.

TECHNIQUES AVAILABLE IN THE LAB

Students will gain experience in the analysis of large-scale biological data, including the integrated processing of genomic, mutational, and gene expression datasets. The research involves the application of statistical and computational modeling approaches aimed at describing and predicting complex biological processes. The work includes programming for data analysis (R, Python) as well as data visualization techniques that support system-level interpretation of results.

SELECTED PUBLICATIONS

Juhász, S., **Papp, B. T.**, Fülöp, A. T., Farkas, Z., Kókai, D., Gyémánt, D. A., Tóth, F., Nacsa, Z., Spekhardt, D., Koncz, B., Burkovics, P., Pál, C., & Manczinger, M. (2026). Five dominant amino acid substitution signatures shape tumour immunity. **Mol Syst Biol** 10.1038/s44320-026-00193-x. Epub ahead of print.

Papp, B. T., Toplenszky, K., Ócsai, H., Csányi, I., Kemény, L., Gyulai, R., Oláh, J., & Baltas, E. (2025). Ten Years of Euromelanoma in Hungary: Nationwide Trends and Risk Factors for Skin Cancer in Central-Eastern Europe. **Cancers 17**(23): 3749.

Balogh, G. M., Koncz, B., Asztalos, L., Ari, E., Gémes, N., Szebeni, G. J., **Papp, B. T.**, Tóth, F., Papp, B., Pál, C., & Manczinger, M. (2025). C > U mutations generate immunogenic peptides in SARS-CoV-2. **Nat Commun 16**(1): 10156.

Manczinger, M., Koncz, B., Balogh, G. M., **Papp, B. T.**, Asztalos, L., Kemény, L., Papp, B., & Pál, C. (2021). Negative trade-off between neoantigen repertoire breadth and the specificity of HLA-I molecules shapes antitumor immunity. **Nat Cancer 2**(9): 950–961.

Koncz, B., Balogh, G. M., **Papp, B. T.**, Asztalos, L., Kemény, L., & Manczinger, M. (2021). Self-mediated positive selection of T cells sets an obstacle to the recognition of nonself. Proc **Natl Acad Sci U S A 118**(37): e2100542118.