KATALIN JUDIT BUDAY

Semmelweis University
Faculty of Medicine
Department of Physiology

Address: Tűzoltó u. 37-47., H-1094 Budapest, Hungary

RESEARCH AREA

The understanding of cellular stress responses and metabolic processes has become increasingly important in cancer therapy, as these factors fundamentally influence tumor survival and therapeutic outcomes. My research focuses on the molecular mechanisms of redox regulation, cellular metabolism, and stress responses in cancer cells. Particular emphasis is placed on redox-active proteins, including members of the glutathione peroxidase family, and their roles in regulating endoplasmic reticulum stress, lipid homeostasis, and inflammatory signaling pathways. Stress processes associated with dysregulated lipid metabolism, including lipotoxicity and ferroptosis-related cell death mechanisms, significantly affect the metabolic state of tumor cells and their response to cellular stress.

Part of our research is conducted in melanoma cell models, where tumor metabolism, redox balance, and non-apoptotic cell death mechanisms are studied in relation to response to immunotherapy and the development of therapeutic resistance. The aim is to identify cellular regulatory mechanisms that may reveal novel therapeutically targetable processes. To support this, functional genetic approaches, including CRISPR-based screening strategies, are applied for the identification of potential therapeutic targets.

TECHNIQUES AVAILABLE IN THE LAB

CRISPR/Cas9-based gene knockout and functional genetic approaches, establishment and optimization of pooled CRISPR screening systems, amplification of gRNA libraries and lentiviral packaging, maintenance and genetic modification of cell lines, and generation of stable knockout cell lines. Application of in vitro and in vivo tumor models, implementation of cellular stress and metabolic assays, and investigation of lipotoxicity- and ferroptosis-related cell death models. Flow cytometry measurements and data analysis, tumor dissociation and preparation of single-cell suspensions, use of fluorescent reporter systems, and application of microscopy techniques. RNA isolation, gene expression analysis using real-time PCR, protein-level analyses by Western blot, as well as basic immunohistochemical and immunofluorescent sample processing.

SELECTED PUBLICATIONS

Buday, K., & Conrad, M. (2020). Emerging roles for non-selenium containing ER-resident glutathione peroxidases in cell signaling and disease. **Biol Chem 402(3):** 271–287.

Doll, S., Freitas, F. P., Shah, R., Aldrovandi, M., da Silva, M. C., Ingold, I., Goya Grocin, A., Xavier da Silva, T. N., Panzilius, E., Scheel, C. H., Mourão, A., **Buday, K.**, Sato, M., Wanninger, J., Vignane, T., Mohana, V., Rehberg, M., Flatley, A., Schepers, A., Kurz, A., … Conrad, M. (2019). FSP1 is a glutathione-independent ferroptosis suppressor. **Nature 575(7784):** 693–698.

Rodriguez Camargo, D. C., Garg, D., **Buday, K.**, Franko, A., Rodriguez Camargo, A., Schmidt, F., Cox, S. J., Suladze, S., Haslbeck, M., Mideksa, Y. G., Gemmecker, G., Aichler, M., Mettenleiter, G., Schulz, M., Walch, A. K., Hrabě de Angelis, M., Feige, M. J., Sierra, C. A., Conrad, M., Tripsianes, K., … Reif, B. (2018). hIAPP forms toxic oligomers in plasma. **Chem Commun (Camb) 54(43):** 5426–5429.

Ingold, I., Berndt, C., Schmitt, S., Doll, S., Poschmann, G., **Buday, K.**, Roveri, A., Peng, X., Porto Freitas, F., Seibt, T., Mehr, L., Aichler, M., Walch, A., Lamp, D., Jastroch, M., Miyamoto, S., Wurst, W., Ursini, F., Arnér, E. S. J., Fradejas-Villar, N., … Conrad, M. (2018). Selenium Utilization by GPX4 Is Required to Prevent Hydroperoxide-Induced Ferroptosis. **Cell 172(3):** 409–422.e21.

Rodriguez Camargo, D. C., Tripsianes, K., **Buday, K.**, Franko, A., Göbl, C., Hartlmüller, C., Sarkar, R., Aichler, M., Mettenleiter, G., Schulz, M., Böddrich, A., Erck, C., Martens, H., Walch, A. K., Madl, T., Wanker, E. E., Conrad, M., de Angelis, M. H., & Reif, B. (2017). The redox environment triggers conformational changes and aggregation of hIAPP in Type II Diabetes. **Sci Rep 7:** 44041.