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RESEARCH AREA

Developing anti-cancer therapies requires ensuring that treatments selectively destroy cancer cells while sparing healthy ones. New therapeutic approaches exploit the phenomenon known as synthetic lethality: cells can tolerate certain defects in individual molecular pathways, but they die when two critical pathways are simultaneously impaired. One such chemotherapeutic drug is a PARP enzyme inhibitor, which induces synthetic lethality in cancer cells carrying mutations in the BRCA (Breast Cancer Susceptibility) proteins, such as in certain types of breast, ovarian, and prostate cancers. Our research group aims to identify additional targets and has demonstrated the role of the small subunits of DNA Polymerase Epsilon, POLE3 and POLE4, in regulating the sensitivity of cancer cells to PARP inhibitors. Based on these findings, the group is now working on developing a strategy to inhibit the POLE3 and POLE4 proteins in any cancer cell, potentially extending PARP inhibitor therapy to additional cancer types.

TECHNIQUES AVAILABLE IN THE LAB

- In vitro cell culture.
- High-throughput viability assays.
- Gene silencing and protein overexpression in primary and cancer cell lines.
- Monitoring protein levels and post-translational modifications using Western blotting.
- Immunofluorescent labeling with detection by confocal microscopy and flow cytometry.

SELECTED PUBLICATIONS

Mamar, H., **Fajka-Boja, R.**, Mórocz, M., Jurado, E. P., Zentout, S., Mihuţ, A., Kopasz, A. G., Mérey, M., Smith, R., Sharma, A. B., Lakin, N. D., Bowman, A. J., Haracska, L., Huet, S., & Timinszky, G. (2024). The loss of DNA polymerase epsilon accessory subunits POLE3-POLE4 leads to BRCA1-independent PARP inhibitor sensitivity. **Nucleic Acids Res 52**(12): 6994–7011.

Longarini, E. J., Dauben, H., Locatelli, C., Wondisford, A. R., Smith, R., Muench, C., Kolvenbach, A., Lynskey, M. L., Pope, A., Bonfiglio, J. J., Jurado, E. P., **Fajka-Boja, R.**, Colby, T., Schuller, M., Ahel, I., Timinszky, G., O'Sullivan, R. J., Huet, S., & Matic, I. (2023). Modular antibodies reveal DNA damage-induced mono-ADP-ribosylation as a second wave of PARP1 signaling. **Mol Cell 83**(10): 1743–1760.e11.

Fajka-Boja, R., Szebeni, G. J., Hunyadi-Gulyás, É., Puskás, L. G., & Katona, R. L. (2020). Polyploid Adipose Stem Cells Shift the Balance of IGF1/IGFBP2 to Promote the Growth of Breast Cancer. **Front Oncol 10**: 157.

Li, Y., James, S. J., Wyllie, D. H., Wynne, C., Czibula, A., Bukhari, A., Pye, K., Bte Mustafah, S. M., **Fajka-Boja, R.**, Szabo, E., Angyal, A., Hegedus, Z., Kovacs, L., Hill, A. V. S., Jefferies, C. A., Wilson, H. L., Yongliang, Z., & Kiss-Toth, E. (2019). TMEM203 is a binding partner and regulator of STING-mediated inflammatory signaling in macrophages. **Proc Nat Acad Sci U S A 116**(33): 16479–16488.

Fajka-Boja, R., Marton, A., Tóth, A., Blazsó, P., Tubak, V., Bálint, B., Nagy, I., Hegedűs, Z., Vizler, C., & Katona, R. L. (2018). Increased insulin-like growth factor 1 production by polyploid adipose stem cells promotes growth of breast cancer cells. **BMC Cancer 18**(1): 872.