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RESEARCH AREA

Inflammatory bowel disease (IBD) is a group of disorders (Crohn's disease and ulcerative colitis) that cause chronic inflammation in the gastrointestinal tract. In many cases IBD can be characterised already onset at a young age, and the number of affected patients has risen sharply. Therefore, there is a pressing need to understand the pathologies of IBD and create effective treatments. New experimental data have claimed that both (macro)autophagy and ferroptosis are highly involved in IBD. While in any disturbance in autophagy might cause IBD, ferroptosis participates in the development of IBD assuming that although the two processes help each other, they have opposite effects on IBD. However, the proper dynamical characteristic of autophagy and ferroptosis regulated IBD remains largely unknown. We suppose that finding a proper balance between autophagy and ferroptosis is a promising strategy for treating IBD.

In our lab both theoretical and molecular biological techniques are used to answer our scientific questions. This project is based on our previous models of autophagy induction and our recently adjusted human cell line (Caco-2) to investigate the important elements and feedback loops of ferroptosis and autophagy upon IBD.

Our goals are the following:

-To build a multicellular (tri-culture) model that we can use to mimic IBD by combining intestinal epithelial cells (Caco-2) with mucus-producing cells (HT29-MTX)and immune cells (such as THP-1 macrophages).

- To investigate the molecular mechanism of the crosstalk between autophagy and ferroptosis by using various inducers or silencers the key components of the control network.

- To test various natural compound (such as resveratrol, sulforaphane) which can reduce the negative effects of IBD through the regulation of autophagy and/or ferroptosis and suggesting novel therapeutic treatments.

TECHNIQUES AVAILABLE IN THE LAB

We use classical molecular biological techniques, such as RNA-, DNA isolation, Western blot, ELISA, qPCR, microscopic techniques.

SELECTED PUBLICATIONS

Holczer, M., Márton, M., **Stiller, I.**, Lizák, B., Bánhegyi, G., & Kapuy, O. (2025). Fine-Tuning of the Endoplasmic Reticulum Stress Response Mechanism Plays a Key Role in Cellular Survival-A Mathematical Study. **Int J Mol Sci 26(22)**: 10961.

Ahmed, S., Kovács, D., Kovács, M., Kosztelnik, M., Hotzi, B., Sigmond, T., Saskői, É., Vincze, V. V., Erdélyi, V., Deák, V., **Stiller, I.**, Vellai, T., & Barna, J. (2026). Heat shock factor-1 alleviates ER-stress in Caenorhabditis elegans. **Sci Rep 16(1)**: 9928.

Balogh, T., Lőrincz, T., **Stiller, I.**, Mandl, J., Bánhegyi, G., & Szarka, A. (2016). The Level of ALR is Regulated by the Quantity of Mitochondrial DNA. **Pathol Oncol Res 22(2)**: 431–437.

Stiller, I., Lizák, B., & Bánhegyi, G. (2014). Physiological functions of presenilins; beyond γ-secretase. **Curr Pharm Biotechnol 15(11)**: 1019–1025.

Margittai, É., Löw, P., **Stiller, I.**, Greco, A., Garcia-Manteiga, J. M., Pengo, N., Benedetti, A., Sitia, R., & Bánhegyi, G. (2012). Production of H_2O_2 in the endoplasmic reticulum promotes in vivo disulfide bond formation. **Antioxid Redox Signal 16(10)**: 1088–1099.