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RESEARCH AREA

The role of epithelial-mesenchymal (EMT) and mesenchymal-epithelial transformation (MET) during embryogenesis, tumorigenesis and inflammation is essential. In our laboratory we proved that during Freund's adjuvant-induced inflammation rat mesenteric mesothelial cells undergo epithelial-mesenchymal transition type II (EMT). During this process the mesothelial cells produce pro-inflammatory cytokines (TGF- β , GM-CSF, TNF α , IL-6, etc.) and express their receptors. After the peak of inflammation, regeneration (MET) starts, which is mediated by intense autophagy and accompanied by non-canonical signaling pathways induced by various BMP factors.

Rapid diagnosis and early treatment of peritonitis remains a challenge in emergency medicine. Both primary and secondary peritonitis have a very high mortality rate. The main regulator of these inflammatory processes is the mesothelium, which is in the focus of our investigations. Therefore, clarifying the steps and molecular regulation of inflammation and regeneration in the mesothelial cells would facilitate the diagnosis of peritonitis, the choice of therapeutic methods, and possibly enable prevention.

TECHNIQUES AVAILABLE IN THE LAB

- in vivo studies with Sprague Dawley rats
- organ and tissue preparation
- sample preparation processes used during immunohistochemical / immunocytochemical tests: fixation and embedding techniques
- preparation of semi-thin and ultra-thin sections
- immunohistochemistry / immunocytochemistry
- electron microscopic post-embedding immunocytochemistry
- light, confocal and electron microscopy
- biochemical tests: Western blot analysis, protein measurements
- densitometric analysis tests
- statistical analyses
- examination of in vitro cell and tissue cultures (surviving mesothelial cell culture)
- use of radioisotope immunoassays

SELECTED PUBLICATIONS

Zsiros, V., Katz, S., Dóczi, N., Kiss, AL. (2017) Autophagy is the key process in the re-establishment of the epitheloid phenotype during mesenchymal-epithelial transition (MET). **Exp Cell Res** **352(2)**: 382-392. .

Katz, S., **Zsiros, V.,** Dóczi, N., Kiss, AL. (2018) Inflammation-Induced Epithelial-to-Mesenchymal Transition and GM-CSF Treatment Stimulate Mesenteric Mesothelial Cells to Transdifferentiate into Macrophages. **Inflammation** **41(5)**: 1825-1834.

Zsiros, V., Katz, S., Doczi, N., Kiss, AL. (2019) Endocytosis of GM-CSF receptor β is essential for signal transduction regulating mesothelial-macrophage transition. **Biochim Biophys Acta Mol Cell Res****1866(9)**: 1450-1462.

Katz, S., **Zsiros, V.,** Kiss, AL. (2019) Under inflammatory stimuli mesenteric mesothelial cells transdifferentiate into macrophages and produce pro-inflammatory cytokine IL-6. **Inflamm Res** **68(7)**: 525-528.

Zsiros, V., Kiss, AL. (2020) Cellular and molecular events of inflammation induced transdifferentiation (EMT) and regeneration (MET) in mesenteric mesothelial cells. **Inflamm Res** **69(12)**: 1173-1179.

Zsiros, V., Dóczi, N., Petővári, G., Pop, A., Erdei, Z., Sebestyén, AL., Kiss, A. (2023) BMP-induced non-canonical signaling is upregulated during autophagy-mediated regeneration in inflamed mesothelial cells. **Sci Rep** **13(1)**: 10426.