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RESEARCH AREA

Nod-like receptors are intracellular pattern recognition receptors that recognize pathogen- or danger-associated molecules and initiate cellular responses in order to protect the cell. Either directly (via inflammasome formation) or indirectly (via signal pathway), they regulate various cellular functions including pro-inflammatory cytokine secretion (like IL-1beta), cell division or cell death. NLRs function as potential therapeutic and diagnostic target since most of the already-characterized members of the family have been associated to some form of autoimmune-, autoinflammatory-, allergic- or cancer diseases. This is proven by the fact that many pharmaceutical company focuses on the molecular manipulation of NLRs by drug development. Despite of the intensive studies many questions are still open, including the cell specific function of NLRs and the molecular mechanisms that modify the functions. Our research team aims to study (1) the expression and the molecular mechanisms of the action in various macrophage subpopulations; (2) the role of skeletal muscle NLRs in the cytokine (myokine) production and insulin resistance.

TECHNIQUES AVAILABLE IN THE LAB

In vitro methods: isolation of monocytes from human blood and differentiation of macrophages subpopulations; differentiation of various mouse macrophages (eg. from bone marrow) or isolation of mouse macrophages (alveolar, peritoneal, brain); cultivation and treatment of primary cells and cell lines(human, mouse); RNA isolation, quantitative PCR; Western blot; ELISA; flow cytometry; microscopy; study of signaling pathways; staudy of metabolism; enzyme activity measurements. In vivo methods: mouse models of systemic and local inflammation; injection and isolation of murine Tibialis anterior muscle; stable and conditional knock-out mouse.

SELECTED PUBLICATIONS

Tóth, K., Lénárt, N., Berki,P., Fekete, R., Szabadits, E., Pósfai, B., Cserép, C, Alatshan, A., **Benkő, S.**, Kiss, D., Hübner, C.A., Gulyás, A., Kaila, K., Környei, Z., Dénes, Á.(2022) The NKCC1 ion transporter modulates microglial phenotype and inflammatory response to brain injury in a cell-autonomous manner. **PLoS Biol 27:** 3001526.

Kovács, E.,G., Alatshan, A., Budai, M.,M., Czimmerer, Z., Bíró, E., **Benkő, S.** (2021) Caffeine Has Different Immunomodulatory Effect on the Cytokine Expression and NLRP3 Inflammasome Function in Various Human Macrophage Subpopulations. **Nutrients 13:** 2409.

Szekanecz, Z., McInnes, I.,B., Schett, G., Szamosi, S., **Benkő, S.**, Szűcs, G. (2021) Autoinflammation and autoimmunity across rheumatic and musculoskeletal diseases. **Nat Rev Rheumatol 17:** 585-595.

Alatshan, A., Kovács, G.,E., Aladdin, A., Czimmerer, Z., Tar, K., **Benkő, S.** (2020) All-Trans Retinoic Acid Enhances both the Signaling for Priming and the Glycolysis for Activation of NLRP3 Inflammasome in Human Macrophage. **Cells 9:** 1591.

Czimmerer Z, Daniel B, Horvath A, Rückerl D, Nagy G, Kiss M, Peloquin M, Budai MM, Cuaranta-Monroy I, Simandi Z, Steiner L, Nagy B Jr, Poliska S, Banko C, Bacso Z, Schulman IG, Sauer S, Deleuze JF, Allen JE, **Benko S**, Nagy L. (2018) The Transcription Factor STAT6 Mediates Direct Repression of Inflammatory Enhancers and Limits Activation of Alternatively Polarized Macrophages. **Immunity 48:** 75-90.