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RESEARCH AREA

The aim of our group is to understand the molecular mechanisms of various human diseases, laying down the foundations for new diagnostic and therapeutic approaches. Our experiments focus on the inflammatory processes that determine the pathomechanism of a large number of major human diseases. We investigate their molecular mechanisms using transgenic animal models, healthy and patient-derived human cells and tissues, state-of-the-art molecular biology and gene editing, as well as functional and imaging methods. We have close collaboration with several prominent international research groups. Our group is supported by several programmes of excellence, including the Frontline and Topical Excellence programmes, as well as the European Union's largest international rheumatology consortium.

TECHNIQUES AVAILABLE IN THE LAB

Transgenic technologies (knockout, knock-in, gene-addition mutants) in mammals
Autoimmune and inflammatory disease models
In vitro and in vivo genetic modelling of human diseases
Molecular biology and genetics
Immune cell analysis, culture, genetic modification
Whole-genome gene editing
High-throughput confocal microscopy

SELECTED PUBLICATIONS

Szilveszter, K.P., Vikár, S., Horváth, Á.I., Helyes, Z., Sárdy, M. and **Mócsai, A.** (2022) Phospholipase Cγ2 is essential for experimental models of epidermolysis bullosa acquisita. **J Invest Dermatol 142:** 1114-1125.

Futosi, K., Kása, O., Szilveszter, K.P. and **Mócsai, A.** (2021) Neutrophil phospholipase Cγ2 drives autoantibody-induced arthritis through the generation of the inflammatory microenvironment. **Arthritis Rheumatol 73:** 1614-1625.

Orosz, A., Walzog, B. and **Mócsai, A.** (2021) In vivo functions of mouse neutrophils derived from HoxB8-transduced conditionally immortalized myeloid progenitors. **J Immunol 206:** 432–445.

Németh, T., Sperandio, M. and **Mócsai, A.** (2020) Neutrophils as emerging therapeutic targets. **Nat Rev Drug Discov 19:** 253-275.

Csete, D., Simon, E., Alatshan, A., Aradi, P., Dobó-Nagy, C., Jakus, Z., Benkő, S., Győri, D.S. and **Mócsai, A.** (2019) Hematopoietic or osteoclast-specific deletion of Syk leads to increased bone mass in experimental mice. **Front Immunol 10:** 937.