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RESEARCH AREA

In our research, we are working on characterizing the immunometabolic role of poly(ADP-ribose) polymerases (PARPs), an enzyme family of great pharmacological significance, in the pathomechanism of psoriasis and eczema. The concept of immunometabolism offers a new approach to understanding the processes underlying inflammatory diseases. The direct correlation between obesity and the severity of these skin conditions suggests that systemic metabolic disorders, and consequently the reprogramming of immune cells' own metabolism, may play a significant role in the development of both psoriasis and eczema. Therefore, therapeutic strategies that can intervene in the pathological metabolic processes specific to these diseases could simultaneously reduce the inflammatory burden on the skin and the risk of disease progression, without the undesirable effects of immunosuppressive biological drugs. Our goal is to demonstrate that PARP inhibitors, currently used as anti-cancer therapies, may have a potentially new indication in the treatment of inflammatory skin diseases.

TECHNIQUES AVAILABLE IN THE LAB

We use mouse models of psoriasis and eczema for our in vivo studies. In our in vitro cell culture experiments, we routinely perform RNA and protein isolation, followed by quantitative PCR, Western blotting, and ELISA methods to examine gene and protein expression. Additionally, we use flow cytometry to identify cell death, mitochondrial functions, lipid content, and cell surface markers. For investigating cell metabolism, we utilize a Seahorse analyzer.

SELECTED PUBLICATIONS

Antal, D., Pór, Á., Kovács, I., Dull, K., Póliska, S., Ujlaki, G., Demény, M., Szöllősi, A., Kiss, B., Szegedi, A., Bai, P., **Szántó**, **M.** (2023) PARP2 promotes inflammation in psoriasis by modulating estradiol biosynthesis in keratinocytes. **J Mol Med (Berl) 101(8):** 987-999.

Antal, D., Janka, E., Szabó, J., Szabó, I., Szegedi, A., Gáspár, K., Bai, P., **Szántó, M.** (2022) Culture-based analyses of skin bacteria in lesional moist, and unaffected dry and sebaceous skin regions of hidradenitis suppurativa patients. **J Eur Acad Dermatol Venereol 36 (9):** e731-e733.

Szántó, M., Oláh, A., Szöllősi, A., Tóth, K., Páyer, E., Czakó, N., Pór, Á., Kovács, I., Zouboulis, C., Kemény, L., Bíró, T., Tóth, I. (2019) Activation of TRPV3 inhibits lipogenesis and stimulates production of inflammatory mediators in human sebocytes: a putative contributor to dry skin dermatoses. **J Invest Dermatol 139(1):** 250-253.

Bai, P., Cantó, C., Brunyánszki, A., Huber, A., **Szántó, M.**, Cen, Y., Yamamoto, H., Houten, S., Kiss, B., Oudart, H., Gergely, P. (2011) Menissier-de, M., Schreiber, V., Sauve, A., Auwerx, J.: PARP-2 regulates SIRT1 expression and whole-body energy expenditure. **Cell Metab 13 (4):** 450-460.

Kiss, B., **Szántó, M.**, Hegedűs, C., Antal, D., Szödényi, A., Márton, J., Méhes, G., Virág, L., Szegedi, A., Bai, P. (2020) Poly(ADP-ribose) polymerase-1 depletion enhances the severity of inflammation in an imiquimod-induced model of psoriasis. **Exp Dermatol 29 (1):** 79-85.