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RESEARCH AREA

Skeletal muscle is a highly dynamic tissue that can undergo successful regeneration upon injury, and change in size in response to exercise, aging or due to diseases (e.g. cancer cachexia, immobilization, or denervation). The muscle stem cells, satellite cells are stimulated by local damage to proliferate extensively and form myoblasts that will subsequently migrate, differentiate and fuse to form muscle fibers. Our research aims are to study (i) the signaling pathways and mechanisms in myoblast migration, differentiation, and fusion (ii) the role of exosomes in cell migration, (iii) the biology of satellite cells. Moreover, we investigate the molecular mechanisms regulating skeletal muscle mass, and we aimed to find new nanotechnological approaches for the local treatment of muscle atrophy. Skeletal muscle has an important role in whole-body metabolism, it accounts for 40% of adult human body weight, and about 90% of insulin-stimulated glucose uptake occurs in skeletal muscle. The vesicular transport of GLUT4 glucose transporters is impaired in cases of insulin resistance and type-2 diabetes mellitus leading to decreased glucose uptake of skeletal muscle and increased blood glucose level. Our further aim is to study this mechanism and to find new signaling pathways regulating glucose uptake of skeletal muscle. Our work is mainly basic research and we have strong scientific collaborations with clinicians.

TECHNIQUES AVAILABLE IN THE LAB

Mammalian tissue culture techniques, in vivo animal models, primary cell isolation, single myofiber and satellite cell isolation, immunocytochemistry, immunohistochemistry, fluorescent microscopy techniques, image analysis, analysis of cell migration, flow cytometry, cell cycle analysis, cell proliferation assays, spectrophotometry (measurement of enzyme activities, metabolites), PCR, co-immunoprecipitation, GTP-ase activity assays, Western blotting, glucose tolerance test, insulin tolerance test.

SELECTED PUBLICATIONS

Szabo, K., Varga, D., Vegh, AG., Liu, N., Xiao, X., Xu, L., Dux, L., Erdelyi, M., Rovo, L., **Keller-Pinter, A.** (2022) Syndecan-4 affects myogenesis via Rac1-mediated actin remodeling and exhibits copy-number amplification and increased expression in human rhabdomyosarcoma tumors. **Cell Mol Life Sci 79:** 122.

Becsky, D., Szabo, K., Gyulai-Nagy, S., Gajdos, T., Bartos, Z., Balind, A., Dux, L., Horvath, P., Erdelyi, M., Homolya, L., **Keller-Pinter, A.** (2020) Syndecan-4 Modulates Cell Polarity and Migration by Influencing Centrosome Positioning and Intracellular Calcium Distribution. **Front Cell Dev Biol 15:** 575227.

Becsky, D., Gyulai-Nagy, S., Balind, A., Horvath, P., Dux, L., **Keller-Pinter, A**. (2020) Myoblast Migration and Directional Persistence Affected by Syndecan-4-Mediated Tiam-1 Expression and Distribution. **Int J Mol Sci 21:** 823.

Keller-Pinter, A., Szabo, K., Kocsis, T., Deak, F., Ocsovszki, I., Zvara, A., Puskas, L., Szilak, L., Dux, L. (2018) Syndecan-4 influences mammalian myoblast proliferation by modulating myostatin signalling and G1/S transition. **FEBS Lett 592:** 3139-3151.

Kocsis, T., Trencsenyi, G., Szabo, K., Baán, J.A., Müller, G., Mendler, L., Garai, I., Reinauer, H., Deak, F., Dux, L., **Keller-Pintér, A.** (2016) Myostatin propeptide mutation of the hypermuscular Compact mice decreases the formation of myostatin and improves insulin sensitivity. **Am J Physiol Endocrinol Metab 312:** E150-E160.