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RESEARCH AREA

A common side effect of anti-tumor therapies is the development of tumor cells that are resistant to the treatment. Therefore, it is essential to develop combined therapies that target multiple cellular processes simultaneously with different compounds, as well as designing effective testing systems to screen for these. Our research group aims to identify metabolic and signaling pathways whose manipulation inhibits the progression of the Drosophila melanogaster carcinoma model we use. Previous research, including our own, has shown that this genetically induced tumor type – similar to mammalian tumors – is highly dependent on sugar and lipid metabolism, as well as the activation of various growth and inflammation signaling pathways. In addition to genetically manipulating these processes, our goal is to test a number of known and new anti-cancer agents, as well as their combinations, on this invertebrate tumor model. The advantage of our model is that it offers a faster, more efficient, and ethically more approvable alternative to vertebrate systems for the discovery and preliminary testing of new, potential anti-tumor strategies.

TECHNIQUES AVAILABLE IN THE LAB

Methods of molecular cell biology:

- Fluorescence microscopy: Examination of the subcellular localization of proteins using fluorescent reporters (e.g., GFP) and fluorescent immunolabeling.
- Testing the effects of small molecule compounds and genetic treatments in vivo on Drosophila tumors.
- Analyzing the activity of signal transduction processes by Western blotting.
- Determination of tissue lipid composition by lipidomics.

Genetic methods:

- Tumor induction in transgenic Drosophila
- Epistasis analysis through the simultaneous manipulation of parallel cellular processesSELECTED

SELECTED PUBLICATIONS

Hegedűs, K., **Takáts, S.**, Boda, A., Jipa, A., Nagy, P., Varga, K., Kovács, A. L., & Juhász, G. (2016). The Ccz1-Mon1-Rab7 module and Rab5 control distinct steps of autophagy. **Mol Biol Cell 27(20):** 3132–3142.

Khezri, R., Holland, P., Schoborg, T. A., Abramovich, I., **Takáts, S.**, Dillard, C., Jain, A., O'Farrell, F., Schultz, S. W., Hagopian, W. M., Quintana, E. M., Ng, R., Katheder, N. S., Rahman, M. M., Teles Reis, J. G., Brech, A., Jasper, H., Rusan, N. M., Jahren, A. H., Gottlieb, E., … Rusten, T. E. (2021). Host autophagy mediates organ wasting and nutrient mobilization for tumor growth. **EMBO J 40(18):** e107336.

Szenci, G., Glatz, G., **Takáts, S.**, & Juhász, G. (2024). The Ykt6-Snap29-Syx13 SNARE complex promotes crinophagy via secretory granule fusion with Lamp1 carrier vesicles. **Sci Rep 14(1):** 3200.

Takáts, S., Glatz, G., Szenci, G., Boda, A., Horváth, G. V., Hegedűs, K., Kovács, A. L., & Juhász, G. (2018). Non-canonical role of the SNARE protein Ykt6 in autophagosome-lysosome fusion. **PLoS Genet 14(4):** e1007359.