

# Seminar series TRR 305 – Striking a moving target: From mechanisms of metastatic organ colonisation to novel systemic therapies



**Wednesday, 30 November 2022**

**15.00 h**

**hybrid (on site in Erlangen)**

TRC Auditorium Translational Research Center, Raum 0.010  
Schwabachanlage 12, 91054 Erlangen

**Prof. Dr. med. Dr. rer. nat.  
Alpaslan Tasdogan**

Institute for Tumor Metabolism, Department of Dermatology,  
University Hospital Essen & German  
Cancer Consortium, Partner Site, Essen



## Targeting Metabolic Liabilities in Cancer Metastasis

Distant metastasis is responsible for over 90% of deaths in patients with cancer. Around 30% of all diagnosed melanoma patients will develop metastases in different visceral organs, and eventually approximately 80% of those patients will die from metastatic disease. So far, there is no cure for metastatic cancer and no pharmacological therapy is available. Therefore, it is critical that we better understand how cancer cells metastasize and discover novel treatment strategies. My lab's focus is to better understand how metastatic melanoma cells adapt their metabolism at different stages of metastasis and to characterize the metabolic plasticity in metastatic melanoma tumors to dissect stage-specific metabolic adaptations for novel therapeutic strategies. For that reason, we developed novel strategies to identify metabolic vulnerabilities in cancer cells during metastasis. Applying these approaches, we uncovered previously not reported metabolic dependencies in metastasizing melanoma cells. We are currently testing these dependencies by both pharmacological and genetic inhibition to identify metabolic inhibitors that block metastasis. In the next steps we will test our new findings in melanoma patients as a novel metabolic target for cancer therapy.

Tasdogan, A., Kumar, S., Allies, G., Bausinger, J., Beckel, F., Hofemeister, H., Mulaw, M., Madan, V., Scharffetter-Kochanek, K., Feuring-Buske, M., Doehner, K., Speit, G., Stewart, A. F., & Fehling, H. J. (2016). DNA Damage-Induced HSPC Malfunction Depends on ROS Accumulation Downstream of IFN-1 Signaling and Bid Mobilization. *Cell stem cell*, 19(6), 752–767.

<https://doi.org/10.1016/j.stem.2016.08.007> Tasdogan, A., Faubert, B., Ramesh, V., Ubellacker, J. M., Shen, B., Solmonson, A., Murphy, M. M., Gu, Z., Gu, W., Martin, M., Kasitinon, S. Y., Vandergriff, T., Mathews, T. P., Zhao, Z., Schadendorf, D., DeBerardinis, R. J., & Morrison, S. J. (2020). Metabolic heterogeneity confers differences in melanoma metastatic potential. *Nature*, 577(7788), 115–120. <https://doi.org/10.1038/s41586-019-1847-2>

## Zoom-Meeting-Link

<https://fau.zoom.us/j/66415728567?pwd=WEtqOTg0a1VIN08rSFYxeGdqWmkwQT09>

Meeting-ID: 664 1572 8567

Kenncode: 207 552

