

04.12.

18:00

FESTSAAL



POPPELSDORFER
SCHLOSSGESPRÄCHE

ALL STUDENTS, COLLEAGUES AND
INTERESTED MEMBERS OF THE PUBLIC ARE
WELCOME TO ATTEND

PROF. DR. MARCUS CONRAD

FERROPTOSIS AS A ROOT CAUSE OF NEURODEGENERATIVE DISEASES

POPPELSDORFER SCHLOSSGESPRÄCHE
WITH THE SUPPORT OF GESELLSCHAFT FÜR
BIOCHEMIE UND MOLEKULARBIOLOGIE GBM.
IN COOPERATION WITH THE BONNER FORUM
FÜR BIOMEDIZIN.



FOOD AND DRINKS
PROVIDED



PANEL DISCUSSION



FESTSAAL IN THE MAIN
BUILDING OF THE
UNIVERSITY - ACCESS
VIA THE ROUNDABOUT
FROM THE INNER
COURTYARD OR
COURTYARD GARDEN



PROF. DR. MARCUS CONRAD



Background

Ferroptosis is a novel form of regulated necrotic cell death marked by iron-dependent lipid peroxidation and metabolic constraints. Our contribution to this intriguing and emerging field began with the early discovery that the genetic knockout of glutathione peroxidase 4 (GPX4) causes a caspase-independent, non-apoptotic form of cell death, which could be prevented by the lipophilic antioxidant vitamin E both in cells and in mice to some extent (Seiler et al., Cell Metab 2008). Meanwhile, it is known that GPX4 is at the heart of ferroptosis due to its unique activity to prevent detrimental lipid peroxidation. Systems that sustain full functionality of GPX4 can be regarded as important nodes to control ferroptosis. Cellular processes, such as iron handling, polyunsaturated fatty acid metabolism, endoplasmic reticulum stress, glutaminolysis and the mevalonate pathway, have been recently recognized to impinge on this form of cell death as well.

Genetic approaches

We are using state-of-the-art genetic technologies including genome-wide CRISPR/Cas9 screening to unravel novel ferroptosis players. Gain- and loss-of function approaches performed both in vitro and in vivo aim to investigate and validate the importance and relevance of these ferroptosis players in physiology and disease development. Moreover, their potential as putative future stratification markers in various disease contexts is also a focus in our laboratory.

Small molecule discovery

We are constantly applying phenotypic and target-based screening efforts to identify and develop novel cell death modulators for both inhibiting and inducing ferroptosis in the context of (neuro)degenerative diseases and cancer, respectively.

Publications

Nakamura, Toshitaka, and Marcus Conrad. "Exploiting ferroptosis vulnerabilities in cancer." *Nature cell biology* vol. 26,9 (2024): 1407-1419. doi:10.1038/s41556-024-01425-8

Ito, Junya et al. "PRDX6 dictates ferroptosis sensitivity by directing cellular selenium utilization." *Molecular cell* vol. 84,23 (2024): 4629-4644.e9. doi:10.1016/j.molcel.2024.10.028

Zheng, Jiashuo, and Marcus Conrad. "Iron raider: Tumors in the bone marrow." *Cell* vol. 188,22 (2025): 6107-6108. doi:10.1016/j.cell.2025.09.028

Wu K, Vaughan AJ, Bossowski JP, Hao Y, Ziogou A, Kim SM, Kim TH, Nakamura MN, Pillai R, Mancini M, Rajalingam S, Han M, Nakamura T, Wang L, Chung S, Simeone D, Shackelford D, Kang YP, Conrad M, Papagiannakopoulos T (2025) Targeting FSP1 triggers ferroptosis in lung cancer. *Nature*. 2025 Nov 5. doi: 10.1038/s41586-025-09710-8.

Palma M, Chaufan M, Breuer CB, Müller S, Sabatier M, Fraser CS, Szylo KJ, Yavari M, Carmona A, Kaur M, Melo LMN, Cansiz F, Monge-Lorenzo J, Flores M, Mishima E, Nakamura T, Proneth B, Labrado M, Liang Y, Cayting N, Zheng L, Cañeque T, Colombeau L, Wahida A, Friedmann Angeli JP, Tasdogan A, Hui S, Rodriguez R, Conrad M, Reticcker-Flynn NE, Ubellacker JM (2025) Lymph node environment drives FSP1 targetability in metastasizing melanoma. *Nature*. 2025 Nov 5. doi: 10.1038/s41586-025-09709-1.