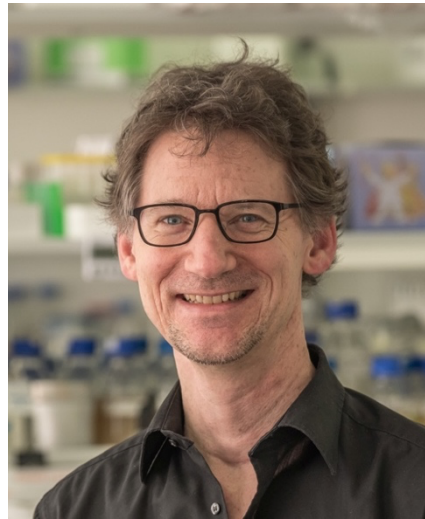


[Seminar] INF2 drives actin reorganization during plasma membrane repair

Date-Time:

Tuesday, November 17, 2026 - 10:40 to 11:20

Location: Seminar room C700, Lab 3



Description

Title: INF2 drives actin reorganization during plasma membrane repair

Speaker: Prof. Roland Wedlich-Söldner

Professor and Director,
Institute of Cell Dynamics and Imaging
University of Münster

Website: <https://www.uni-muenster.de/Cells-in-Motion/people/all/wedlich-soeldner-r.php>

Summary

Plasma membrane (PM) wounds are frequent cellular insults that trigger diverse repair responses across organisms. A common feature of these pathways is the rapid coupling of calcium influx to actin remodeling. Our previous identification of the calcium-mediated actin reset (CaAR) suggested a mechanism linking these two fundamental repair signals, but its molecular basis remained unknown. Here, we identify the formin INF2 as a key mediator of actin modulation during PM repair. Quantitative imaging reveals that injury severity determines distinct repair regimes, with modest wounds eliciting local calcium and actin responses, whereas severe wounds trigger global calcium elevation and cell-wide actin remodeling. INF2 drives both responses through its actin polymerization activity and is recruited to wound sites through a short C-terminal wound-localizing peptide (WOLOP). Structural modelling and mutational analysis indicate that WOLOP mediates interaction with Annexin-S100 complexes at damaged membranes. Loss, hyperactivation, or disease-associated INF2 variants disrupt injury responses, linking altered actin regulation to defective membrane repair. Together, our findings establish INF2 as a molecular bridge between calcium signaling and actin remodeling during membrane repair and provide a mechanistic connection between Annexin-dependent repair pathways and the actin cytoskeleton.

Key references for this seminar

Bayraktar S, Nehrig J, Menis E, Karli K, Janning A, Struk T, Halbritter J, Michgehl U, Krahn MP, Schuberth CE*, Pavenstädt H*, Wedlich-Söldner R* (2020) A deregulated stress response underlies distinct INF2 associated disease profiles. *JASN* 31, 1296-1313.

Wales P, Schuberth, CE, Aufschnaiter R, Fels J, García-Aguilar I, Janning A, Dlugos CP, Schäfer-Herte M, Klingner C, Wälte M, Kuhlmann J, Menis E, Hockaday Kang L, Maier KC, Hou W, Russo A, Higgs HN, Pavenstädt H, Vogl T, Roth J, Qualmann B, Kessels MM, Martin DE, Mulder B and Wedlich-Söldner R (2016). Calcium-mediated actin reset (CaAR) mediates acute cell adaptations. *Elife* 5, e19850.

Klingner C, Cherian AV, Fels J, Diesinger PM, Aufschnaiter R, Maghelli N, Keil T, Beck G, Toliü-Nørrelykke IM, Bathe M and Wedlich-Söldner R (2014). Isotropic acto-myosin dynamics promote organization of the apical cell cortex in epithelial cells. *J Cell Biol.* 207, 107-21.

Short Bio

Roland Wedlich-Söldner studied Biology in Munich and graduated with Regine Kahmann and Gero Steinberg on microtubule-dependent transport in the plant pathogenic fungus *Ustilago maydis*. During his postdoc with Rong Li at Harvard Medical school he studied the mechanisms of spontaneous cell polarization. He then headed a research group on “Cellular Dynamics and Cell patterning” at the Max Planck Institute of Biochemistry before joining the University of Münster in 2014 as full professor and director of the Institute of Cell Dynamics and Imaging. His group uses high resolution live cell microscopy to identify fundamental concepts of cellular organization, with a focus on membrane organization and cytoskeletal dynamics.

Key publications, * co-corresponding

Haase D, Rasch C, Keller U, Tsytsyura Y, Glyvuk N, Elting A, Wittmar J, Janning A, Kahms M, Wedlich N, Schuberth C, Heuer A, Klingauf J, Wedlich-Soldner R. (2023) Tetraspanner-based nanodomains modulate BAR domain-induced membrane curvature. *EMBO Rep*, e57232.

Bayraktar S, Nehrig J, Menis E, Karli K, Janning A, Struk T, Halbritter J, Michgehl U, Krahn MP, Schuberth CE*, Pavenstädt H*, Wedlich-Söldner R* (2020) A deregulated stress response underlies distinct INF2 associated disease profiles. *JASN* 31, 1296-1313.

Busto JV, Elting A, Haase D, Spira F, Kuhlman J, Schäfer-Herte M and Wedlich-Soldner R (2018) Lateral plasma membrane compartmentalization links protein function and turnover. *EMBO J* 37, pii: e99473.

Wales P, Schuberth, CE, Aufschnaiter R, Fels J, García-Aguilar I, Janning A, Dlugos CP, Schäfer-Herte M, Klingner C, Wälte M, Kuhlmann J, Menis E, Hockaday Kang L, Maier KC, Hou W, Russo A, Higgs HN, Pavenstädt H, Vogl T, Roth J, Qualmann B, Kessels MM, Martin DE, Mulder B and Wedlich-Söldner R (2016). Calcium-mediated actin reset (CaAR) mediates acute cell adaptations. *Elife* 5, e19850.

Klingner C, Cherian AV, Fels J, Diesinger PM, Aufschnaiter R, Maghelli N, Keil T, Beck G, Toliü-Nørrelykke IM, Bathe M and Wedlich-Soldner R (2014). Isotropic acto-myosin dynamics promote organization of the apical cell cortex in epithelial cells. *J Cell Biol.* 207, 107-21.

Freisinger T, Klünder B, Johnson J, Müller N, Pichler G, Beck G, Costanzo M, Boone C, Cerione RA, Frey E and Wedlich-Soldner R (2013). Establishment of a robust single axis of cell polarity by coupling multiple positive feedback loops. *Nat Commun* 4, 1807.

Spira F, Mueller NS, Beck G, von Olshausen P, Beig J, Wedlich-Soldner R (2012). Patchwork organization of the yeast plasma membrane into numerous coexisting domains. *Nat Cell Biol* 14, 640-8.

Domínguez-Escobar J, Chastanet A, Crevenna AH, Fromion V, Wedlich-Soldner R* and Carballido-López R* (2011). Processive Movement of MreB-Associated Cell Wall Biosynthetic Complexes in Bacteria. *Science* 333, 225-8. * shared corresp. authors.

Riedl J, Crevenna AH, Kessenbrock K, Yu JH, Neukirchen D, Bista M, Bradke F, Jenne D, Holak TA, Werb Z, Sixt M and Wedlich-Soldner R (2008). Lifeact - a versatile marker for the visualization of F-actin. *Nature Methods* 5, 605-7.

Short abstract

Plasma membrane wounds trigger rapid calcium influx and actin remodeling, but how these signals are coupled has remained unclear. We identify the formin INF2 as a key mediator of this process. Quantitative imaging shows that wound severity determines local or cell-wide repair responses. INF2 drives both through its actin-polymerization activity and is recruited to damaged membranes by a short C-terminal wound-localizing peptide that interacts with Annexin-S100 complexes. These findings link calcium-dependent membrane repair to actin regulation and reveal how INF2 dysfunction may impair repair.