

CCR – Lecture Series

Monday, March 2nd, 2026, 13:00 PM

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Tumor-ECM crosstalk in cancer progression

Tissue homeostasis relies on the spatial organization of distinct cell types and the surrounding extracellular matrix (ECM), where integrin-mediated adhesion and actin cytoskeleton dynamics regulate cellular identity, migration, and invasion. Our previous work highlighted that normal stromal architecture can suppress breast cancer aggression and even reprogram malignant cells towards a more benign state. Expanding this concept, we investigated mechanobiological crosstalk in two distinct malignancies. In vocal fold cancer (VFC), we identified increased ECM deposition and tissue stiffening correlating with disease progression, receptor heterogeneity, and collective migration. Physiological mechanical cues, such as stretching and vibration, attenuate nuclear β -catenin and YAP signaling, suggesting that VFC is a mechanically sensitive tumor. YAP-TEAD inhibition emerges as a potential therapeutic strategy. In mucinous colorectal carcinoma (MUC CRC), we uncover a collagen- α 2 β 1-integrin-SorLA axis regulating tumor polarity. This pathway reinforces HER2/HER3 signaling and maintains apical-in polarity, with HER2-targeting antibodies reversing polarity and impeding metastasis. Together, our findings highlight diverse, tissue-specific mechanotransduction mechanisms that reshape tumor-stroma interactions and offer novel avenues for therapeutic intervention.

Venue: Lecture Hall B2, Borschkegasse 4a

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Host: Juliane Winkler



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