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The contribution of fibronectin to cell-induced tissue morphogenesis

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When engineering fibrous tissues, attaining an appropriate distribution of cells and matrix to ascertain *in vivo* functionality and durability remains a challenge. Since increased cell density towards tissue surfaces was recently correlated with the distribution of fibronectin in 3D microtissues, we now asked how fibronectin impacts cell-induced tissue morphogenesis and heterogeneity.

To address this question, microtissues were engineered in a high-throughput model system containing rectangular arrays of 12 posts. Posts constrained fibroblast-populated collagen gels that self-assembled into trampoline-shaped tissues with dimensions of ~750x750x40µm. Fibronectin contribution was assessed using fibronectin-knockout mouse embryonic fibroblasts (MEFs-Fn-/-) and floxed equivalents (MEFs-Fnf/f), in fibronectin-depleted growth medium with/without exogenously added plasma or cellular fibronectin.

In the absence of fibronectin, MEF-Fn-/- were homogenously distributed throughout the collagen matrix. Contrary, in the presence of fibronectin, both cell types produced shell-like tissues with a cell-free collagen core and cell/fibronectin-rich surfaces. By incorporating a stretch-sensitive FRET-sensor, (plasma) fibronectin-stretch was observed to increase from the tissue center towards surfaces. We therefore suggest cells are attracted by fibronectinin a stretch-magnitude dependent manner. In addition, excessive assembly of (plasma) fibronectin by MEF-Fn-/- was observed at the tissue surface, speculated to originate from the absence of (cellular) fibronectin, which was indeed abolished after addition of cellular fibronectin.

Results suggest that tissue morphogenesis/heterogeneity is governed by fibronectin, providing stretch-dependent migration cues while cellular fibronectin provides biological cuesreducing excessive ECM assembly leading to tissue homeostasis. Implications can be found in accounting for the role of fibronectin in tissue engineering that may explain developing tissue heterogeneity.

Biography

Foolen Jasper has completed his PhD from the department of biomedical engineering at Eindhoven University of Technology, followed by a postdoctoral study of which part was conducted in the lab of Prof. Chris Chen at the University of Pennsylvania. He is now a Post-doctoral fellow in the lab of Viola Vogel at ETH Zurich and was recently awardeda Marie Curie fellowship.

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