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A modular joint-on-chip approach to study cellular cross-communication in a simulated osteoarthritic micro-environment

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INTRODUCTION: Cartilage degeneration and synovitis are key hallmarks of joint degenerative diseases, such as osteoarthritis (OA). The communication between these two tissues is fundamental to maintain both homeostasis and disease onset. However, studying this communication has remained challenging. Herein, we propose a novel modular precision microfluidic platform, that combines a synovial membrane-on-chip and a cartilage-on-chip platform. This seamless strategy enables the facile and in depth study of cross-communication between these two joint components, specifically via inflammatory mediators, aiming to replicate the onset of OA.

METHODS: Two types of microfluidic PDMS-based chips were designed with actuation chambers to emulate the mechanical forces of the cartilage and synovial membrane. For the cartilage chip, human healthy and OA affected chondrocytes were seeded in an ECM-like hydrogel. The synovial membrane chip was composed of a hydrogel re-enforced by an elastic membrane, which was seeded with synovium fibroblasts. Both chips were connected by a common channel, where synovium mimicking medium (culture medium supplemented with HA) was flowing (60 µl/hr). After both tissues achieved phenotypical maturation, human macrophages were added to the system. The behavior of the immune cells were a key read-out, focusing on their mobility, cytokine and proteinase release profile (ELISA) and polarization ratio between M1 and M2 (qPCR). On-line and end-point analysis were conducted after 1, 3 and 7 days.

RESULTS & DISCUSSION: The unique chip actuator designs allowed for physiologically relevant stimulation of the cartilage and synovial membrane, by cyclic compression and stretching, respectively. The effect of the mechanical load was determined on the release of inflammatory mediators. The proteinases and inflammatory cytokine profiles are the basis of the fingerprint of the influence of the variables: static vs. mechanically stimulated, healthy vs. OA-affected chondrocytes, and, presence vs. absence of macrophages.

CONCLUSIONS: This modular joint-on-chip platform has the potential to provide unprecedented insights in the effects of inflammation of a single joint tissue, the performance of the various joint tissues, and the joint function itself.

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