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Flash Talk - Also Poster # 14

IN VITRO MODELS OF HUMAN PATHOLOGICAL CARDIAC TISSUE VIA BIOARTIFICIAL SCAFFOLDS

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Background Myocardial infarction causes the loss of billions of cardiomyocytes and the remodelling of local extracellular matrix (ECM), leading to the progressive formation of a stiff fibrotic tissue mainly populated by cardiac fibroblasts. *In vitro* models of human pathological cardiac tissue able to closely reproduce post-infarct microenvironment could greatly improve preclinical experimentation on human heart, providing predictive tools to study new therapies. In this work, the structure and composition of bi- and three-dimensional scaffolds were tailored to obtain models of human pathological cardiac tissue with different thicknesses and severity degrees.

Methods electrospinning (Linari Engineering) and fused-deposition modelling (Rokit Invivo) to obtain 2D and 3D scaffolds, respectively processed Polycaprolactone (PCL). Gelatin (G) was grafted on scaffold surface through a mussel-inspired approach based on two steps: (i) 3, 4-Dihydroxy-D, L-phenylalanine (DOPA) polymerisation on PCL surface; (ii) incubation in G solution. After each functionalization step, physicochemical, morphological and mechanical characterizations were performed. Cardiac fibroblasts isolated from human ventricle (HCFs, PromoCell) were cultured on the scaffolds at a density of 7×10^4 cells/cm², and their adhesion, proliferation and protein expression were analysed.

Results SEM analysis showed that 2D electrospun membranes consisted of a nanofiber network free of defects, while a reproducible interconnected porous structure was obtained for 3D scaffolds. QCM-D, ATR-FTIR and XPS analyses confirmed successful surface modification after each step, while the amount of grafted G was quantified by a colorimetric assay. Mechanical and thermal properties of scaffolds did not vary after functionalization. HCFs cultured on G grafted scaffolds showed better attachment and proliferation compared to non-functionalized scaffolds. The expression of fibroblast markers (α -SMA, DDR2) and secretion of typical cardiac ECM proteins (Fibronectin, Laminin, Tenascin and Collagen IV) were confirmed by immunofluorescence and western blot analysis.

Conclusions 2D and 3D bioartificial scaffolds supported long-term HCF culture, and their composition and structure affected HCF markers and protein deposition. In the future, new therapies will be tested *in vitro* using such constructs. This project has received funding from the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation programme grant agreement No 772168.