The ECM as a driver of fibroblast senescence and disrupted epithelial repair in IPF

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1. Cellular senescence can be both a friend, as well as a foe, depending on the ECM environment in development, wound repair and fibrosis. (Chapter 2)

2. Matrix proteins regulate cell-cycle arrest, as well as the release of cytokines, contributing to cellular senescence. (Chapter 2)

3. Tissue stiffness is an important driver of the activation of fibroblasts, which contributes to increased ECM deposition and cytokine secretion. (Chapter 3)

4. Tissue stiffness modulates the osteoprotegerin - receptor activator of NF-κB ligand pathway. (Chapter 3)

5. Cellular senescence and the pro-fibrotic response share common pro-inflammatory and pro-fibrotic cytokines. (Chapters 2 and 4)

6. ECM deposited by fibroblasts in the presence of disease related stimuli provokes a pro-fibrotic response in fibroblasts. (Chapter 4)

7. Senescent fibroblasts induce cell-cycle arrest and inhibit the proliferation of alveolar epithelial cells.
(Chapter 5)

8. It takes two to tango: Communication between alveolar epithelial cells and fibroblasts is necessary to induce cellular migration during alveolar epithelial wound healing. (Chapter 5)

9. There are no problems, only solutions. (John Lennon)