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| **The effect of extracellular matrix produced from epithelial cells as a model of idiopathic pulmonary fibrosis** |
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| **Introduction/Aim:** Idiopathic pulmonary fibrosis (IPF) is non-resolving fibrosis caused by repetitive injury and subsequent accumulation of extracellular matrix (ECM). IPF accounts for the development of 4.4% to 48% of lung cancer in patients, and one-third of lung cancer patients have pulmonary fibrosis. Developing lung cancer in IPF patients undergone a lung transplant is 20 times more than the general population. The relentless presence of TGF-β1 is considered the main trigger, and evidence suggests that during the inflammation, AEC2 cells are the major TFG-ß1 producers in IPF patients. Airway epithelial cells (AECs) may potentially exert a more direct influence on these physiological processes through the alteration of ECM composition, which is not elucidated. Therefore, adopting an approach to enlighten the function of epithelial-derived ECM as an IPF model on lung cancer cell behaviour may propose a therapeutic approach for lung tumours with IPF.  **Methods:** In order to establish the epithelial-derived ECM as an IPF model *in vitro*, we used BEAS2B and A549 as human alveolar and bronchial epithelial cell lines, respectively. The cells were cultured at the desired density, and after 24h, they were stimulated with TGF-β1 for 72h to induce the proliferation and ECM deposition; furthermore, decellularization process was carried out to leave the intact ECM behind. The three types of lung cancer cells (NCI-H358, NCI-H1573, and NCI-H520) were utilized for culturing on top of the epithelial-derived ECM. The ECM ELISA, proliferation assay, and attachment assay were carried out to determine the effect of epithelial-derived ECM on lung cancer cell behaviour.  **Results:** The ECM ELISA results illustrated that fibronectin and Col IV were increased in the ECM derived from BEAS2B and A549. On the other hand, the proliferation assay showed that the growth of NCI-H358 cultured on the BEAS2B-derived ECM decreased in 48 and 72h after culture. Also, the proliferation of the cultivated NCI-H358 (48h) and NCI-H520 (48, 72h) declined on the A549-derived ECM. The attachment assay results were consistent with the proliferation assay.  **Conclusion:** The crucial role of ECM in both the IPF, and lung cancer development has been investigated intensively. However, the effect of ECM on lung cancer cells' behaviour has remained ambiguous. In this regard, we will investigate the effect of this ECM on invasiveness and related cellular signaling.    **Grant Support:** None. |