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# STEM CELL & CELL-FREE APPROACHES IN AIRWAY EPITHELIAL REPAIR

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## SUMMARY

The development in stem cell technology provides a promising alternative treatment strategy that gives new hope to patients. The ability of endogenous airway repair reduces with age, therefore treatment with external source of cells will be beneficial in the context of organ repair especially the lungs. Various preclinical and clinical evident have shown that treatment with stem cells are not only involved in tissue regeneration and repair by direct differentiating into specific cells and tissues, but they are also involved in recruiting and promoting homing process of the endogenous cells to the site of injured tissues and organs. This phenomenon has raised a question whether do the stem cells are the only reasons for tissue to be repaired or whether the repair process is mainly contributed by paracrine effects that are released by the stem cells in which their immunomodulatory properties promotes endothelial and epithelial homing process during tissue repair. Our preclinical studies of chronic and acute lung injury models have proven that cell therapy either using progenitor or stem cells promote airway repair, reduce inflammatory cell responses and regulate the expression of certain inflammatory markers. However, there is still a lack of fundamental mechanism on how the stem cell-secreted compounds especially mesenchymal stem cells (MSCs) play a role in airway regeneration and repair. Recently, MSC derived extracellular vesicles (EVs) have gathered worldwide interest as a new cell-free based therapy as the EVs do not contain nucleus and do not proliferate, hence avoiding the potential risk of tumour formation. In a rat model of smoke-induced chronic obstructive pulmonary disease (COPD) revealed that the treatments with umbilical cord-mesenchymal stem cells (uc-MSCs), uc-MSCs-extracellular vesicles (uc-MSC-EVs), uc-MSC-conditioned media (uc-MSC-CM) were significantly reduced the effects of inflammation on peribronchiol and perivascular of the lung, reduced the alveolar septal thickening with mononuclear inflammation as well as reduced the number of goblet cells as compared to injury and self-healing groups. The uc-MSCs, uc-MSC-EVs, and uc-MSC-CM treatment groups were also shown to alleviate the loss of alveolar septa in emphysematous lung of COPD rats. These histological results suggest that MSC-EV are as effective as MSC in ameliorating the inflammation and thus provide a new opportunity in utilizing the MSC-EVs in the treatment of COPD. Despites huge evident to support the use of stem cells as a source of cell for therapy, a major challenge remains to which extent the stem cell is safe in clinical setting. Therefore, the use of EVs could be an alternative approach for future cell-free therapy especially for lung diseases.