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The Impact of Cigarette Smoking on the Therapeutic Potential of Mesenchymal Stem Cells

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Abstract

Cigarette smoking is a global health concern associated with a multitude of adverse health effects, including increased susceptibility to various diseases and reduced overall life expectancy. Recent research has uncovered a novel dimension to the detrimental consequences of smoking by examining its impact on the therapeutic potential of mesenchymal stem cells. MSCs have gained prominence in regenerative medicine due to their regenerative and immunomodulatory properties. This review explores the intricate relationship between cigarette smoking and MSCs, shedding light on how smoking may compromise the effectiveness of MSC-based therapies. We discuss the molecular mechanisms underlying this phenomenon, encompassing alterations in MSC proliferation, differentiation and immunomodulation in the presence of cigarette smoke constituents. Moreover, we delve into the potential strategies to mitigate these adverse effects, thus optimizing the therapeutic utility of MSCs in the context of smoking-related diseases. Understanding the intricate interplay between cigarette smoking and MSCs is crucial for advancing regenerative medicine and improving the clinical outcomes of patients affected by smoking-related conditions.

Keywords: Cigarette smoking • Mesenchymal stem cells • Regenerative medicine

Introduction

Mesenchymal Stem Cells (MSCs) have garnered considerable attention in regenerative medicine due to their remarkable immunoregulatory properties and their ability to differentiate into multiple cell lineages. However, various behavioral risk factors and lifestyle choices can perturb the metabolic and growth signaling pathways within MSCs, subsequently impacting their phenotype and functionality. Therefore, identifying and mitigating these factors' effects on transplanted MSCs can significantly enhance their therapeutic efficacy [1,2]. A wealth of experimental and clinical studies has underscored the detrimental impact of cigarette smoke and nicotine on MSC behavior, including proliferation, homing and chondrogenic and osteogenic differentiation. Cigarette smoke exerts inhibitory effects on the synthesis of crucial transcription factors governing cell cycle progression, metabolism, migration, chondrogenesis and osteogenesis. It also diminishes the expression of chemokine receptors and suppresses the activity of antioxidant enzymes within MSCs [3].

Description

Furthermore, cigarette smoke and nicotine have been shown to induce oxidative stress in MSCs by elevating superoxide radicals and depleting intracellular glutathione levels, resulting in adverse effects on osteogenic differentiation. Although nicotine and cotinine do not actively generate reactive oxygen species (ROS), they do hinder catalase and glutathione reductase activity, thereby contributing to ROS accumulation caused by cigarette smoke exposure. Co-incubation with N-acetylcysteine or L-ascorbate has shown promise in ameliorating impaired osteogenesis induced by cigarette smoke exposure, through activation of the nuclear factor erythroid 2-related factor 2 (Nrf2) signaling pathway and ROS scavenging, suggesting potential therapeutic avenues for supporting fracture healing in smokers [4].

Electronic cigarettes (e-cigarettes), often marketed as lower-risk alternatives to traditional cigarettes, remain an area of uncertainty with regard to the chronic inhalation of potentially toxic emissions. The consequences of prolonged exposure to e-cigarette emissions on stem cell function remain largely uncharted, raising concerns about cellular injury in the absence of effective stem cell repair mechanisms. Osteoarthritis (OA), a chronic joint ailment characterized by progressive cartilage degeneration, is associated with tobacco use as one of its environmental risk factors. However, the role of tobacco smoking in OA development remains a subject of debate, with nicotine emerging as one of the most physiologically active components in cigarette smoke. In the context of cell-based therapeutics, stem cell sources are typically screened for infectious agents and genetic disorders prior to implantation. Nonetheless, certain overlooked risk factors, such as exposure to cigarette smoke or nicotine, can compromise the regenerative potential of MSCs, affecting their proliferation, migration and differentiation abilities [5].

Conclusion

MSCs hold immense promise in the realms of regenerative medicine and tissue engineering. However, there is a need to comprehensively understand how their phenotype and differentiation capacity evolve with age. Any decline in functionality associated with aging could have profound implications for tissue quality and viability. Proteomics offers a powerful tool for deciphering the protein profiles responsible for specific cell phenotypes, shedding light on the mechanisms underlying age-related changes in musculoskeletal tissues.

Acknowledgement

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Conflict of Interest

None.

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