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The Effects of Cigarette Smoking on Mesenchymal Stem Cell Therapeutic Potential

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Editorial

Mesenchymal stem cells (MSCs) have been used as new therapeutic agents in regenerative medicine due to their immunoregulatory properties and capacity for multi-lineage differentiation. Numerous behavioural risk factors and lifestyle habits may modulate metabolic and cell growth signalling pathways in MSCs, affecting their phenotype and function. As a result, identifying these factors and minimising their impact on the viability and function of transplanted MSCs may significantly contribute to their improved therapeutic efficacy.

A large number of experimental and clinical studies have shown that cigarette smoke and nicotine have negative effects on MSC proliferation, homing, chondrogenic and osteogenic differentiation. Cigarette smoke inhibits the synthesis of transcriptional factors that regulate the cell cycle, metabolism, migration, chondrogenesis, and osteogenesis, as well as the expression of chemokine receptors and the activity of anti-oxidative enzymes in MSCs.

Cigarette smoke and nicotine have been shown in numerous experimental and clinical studies to have negative effects on MSC proliferation, homing, chondrogenic and osteogenic differentiation. Cigarette smoke inhibits the synthesis of transcriptional factors that control the cell cycle, metabolism, migration, chondrogenesis, and osteogenesis, as well as the expression of chemokine receptors and anti-oxidative enzyme activity in MSCs.Cigarette smoke, on the other hand, causes oxidative stress in MSCs by increasing superoxide radicals and decreasing intracellular glutathione, negatively affecting osteogenic differentiation. Although nicotine and cotinine do not actively produce reactive oxygen species (ROS), they do inhibit catalase and glutathione reductase activity, which contributes to the accumulation of ROS caused by cigarette smoke exposure. Co-incubation with N-acetylcysteine or L-ascorbate improves impaired osteogenesis caused by cigarette smoke exposure by activating nuclear factor erythroid 2-related factor 2 (Nrf2) signalling and scavenging ROS, suggesting that these compounds could be therapeutic targets to support fracture healing in smokers.

Electronic cigarettes (e-cigarettes) are marketed as low-risk substitutes for traditional cigarettes. The effects of chronic inhalation of potential toxicants emitted by e-cigarettes, on the other hand, are largely unknown. In the absence of effective repair by stem cells, smoking-induced chronic diseases may result in cellular injury. Osteoarthritis (OA) is a chronic joint disease characterized by progressive and irreversible cartilage degeneration. Tobacco consumption is prominent among the environmental risk factors for OA, though the role of tobacco smoking in OA development is controversial. Nicotine is one of the most physiologically active molecules among the numerous chemicals found in cigarette smoke.

Prior to implantation, stem cell sources for cell-based therapeutics are frequently screened for infectious agents and genetic diseases; however, there are other risk factors that are frequently overlooked, which may result in less efficacious clinical outcomes. Exposure of mesenchymal stem cells (MSCs) to cigarette smoke or nicotine is one such risk factor. Recent research has shown that cigarette smoke or nicotine exposure reduces the regenerative potential of MSCs, specifically their proliferation, migration, and differentiation potential.

Mesenchymal stem cells (MSCs) have potential applications in regenerative medicine and tissue engineering, but it is unclear how phenotype and differentiation capacity change with age. As a result, any loss of functionality with age would have serious implications for tissue viability and tissue quality. Proteomics enables the identification of the set of proteins responsible for a specific cell phenotype, as well as insights into the mechanisms underlying age-related changes in musculoskeletal tissues [1-5].

Conflict of Interest

None.

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