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Insights on Targeted Therapeutic Approaches for Cardiovascular Disease Using Nanocarriers

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Description

Ischemic heart disease kills more people worldwide than all cancers combined. Myocardial infarction (MI) is the leading cause of death in both the elderly and the nonelderly (65-year-old) population. Atherosclerotic plaque buildup in the coronary arteries, which supply oxygenated blood to the myocardium, is caused by lifestyle and genetic factors. These plaques can become unstable, resulting in the formation of a sudden thrombus that blocks blood flow and deprives the myocardium of oxygen and respiratory substrates, resulting in cardiomyocyte (CM) necrosis and apoptosis. Up to a billion CMs are lost in a typical adult human MI. Adult mammals have unusually low rates of CM proliferation, which results in poor regeneration after injury because parenchymal cells cannot be replaced in sufficient quantity. Most acute MIs are not fatal right away, but CMs are replaced by collagenous scar tissue, which preserves the structural integrity of the heart but does not contribute to contractile function. Patients develop heart failure as a result of this cardiac remodeling [1-3]. The current gold standard therapy for MI is to restore blood flow as quickly as possible using percutaneous coronary intervention (PCI) or thrombolytic drugs, followed by supportive treatments such as lipid-lowering agents, anti-hypertensives and anti-coagulants to reduce stress on the heart and the risk of future events. A model treatment could focus on protecting CMs after reperfusion and modulating long-term inflammation to reduce scar formation, promote angiogenesis and improve regeneration. There are, however, no clinically available treatments that can directly stimulate cardiac regeneration.

Many steps in the complex MI pathogenesis, which includes cell apoptosis, inflammation and remodelling, could potentially be exploited for therapeutic benefit. Agents that activate the Akt pro-survival pathway, such as insulin-like growth factor 1 (IGF-1), may reduce CM apoptosis. Pro-angiogenic factors such as vascular endothelial growth factor (VEGF), interleukin-8 (IL-8) and basic fibroblast growth factor (FGF-) can promote blood vessel formation, stabilization and maturation, which can improve tissue perfusion. TGF-1, MCP-1, TNF- and G-CSF are chemotactic molecules that promote host monocyte recruitment and differentiation into macrophages, thereby influencing local inflammation.

Tissue remodelling can be manipulated by varying the activity of matrix metalloprotease (MMP) and tissue inhibitors of metalloproteinases (TIMP) in the vicinity of the infarct. Anti-inflammatories, antioxidants and immunomodulatory agents have also been investigated as potential treatments for damage caused by hypoxia and reperfusion. There is also growing interest in the use of RNAs, which play an important role in normal heart physiology and are involved in angiogenesis, apoptosis and fibrosis following a myocardial infarction. Some RNAs may help resolve the inflammatory process, promoting cardiac repair. These potential treatment modalities, however, are hampered by poor delivery to the infarct area and difficulties in achieving temporospatial control [4,5].

There are numerous causes of poor myocardial delivery. To begin with, the heart is not easily accessible for drug administration. Myocardial uptake after intravenous administration is extremely low, as will be demonstrated later in this review. The most direct route is intramyocardial injection, but it requires invasive procedures and carries higher risks due to the unstable nature of the ventricle wall after MI. Even so, therapeutics are rapidly lost into circulation, aided by rapid heart contractions. This route is not suitable for a course of treatment that requires more than one injection because multiple intramyocardial injections are impractical. Some therapeutics can be delivered via catheter into the coronary artery, which supplies the myocardium. However, peptides like VEGF and IGF-1 degrade quickly in the bloodstream or after direct injection into tissues. Regeneration and remodelling take months and acute inflammation is both harmful and necessary for long-term wound healing. As a result, a single delivery of an isolated growth factor, cytokine, microRNA, or small molecules is unlikely to make a significant difference in long-term outcomes.

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Conflict of Interest

Authors declare no conflict of interest.

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