Angiogenesis is the formation of new blood vessels from pre-existing blood vessels. In clinical applications, angiogenesis is an excellent therapeutic target to treat different forms of cardiovascular disease, such as wounds resulting from ischemia. Angiogenesis is regulated by a variety of proteins. One of these proteins, Angiopoietin-1 (ANGPT1) contributes to vessel maturation and stability. ANGPT1 is thought to be constitutively expressed by pericytes on the endothelial cell layer of blood vessels. Our previous experimental results show that in addition to pericytes, fibro/adipogenic progenitors (FAPs) contribute to angiogenesis and express ANGPT1. FAPs are tissue-resident mesenchymal stromal cells (MSCs) that proliferate in response to muscle damage. The proliferation of FAPs are thought to help repair damaged muscle, but in pathological conditions, FAPs differentiate into fibroblasts and adipocytes. This specific study aims to show that adipogenesis in cardiac muscle is a result of the depletion of the ANGPT1 gene in cardiac FAPs following cardiac muscle damage. Greater understanding of the roles of pericytes and FAPs behind angiogenesis may allow for new ways to advance therapeutic solutions to cardiac ischemia.

Themes:

Check (highlight) the most applicable theme according to the abstract.

| Innovation and Technology | Health and Wellness | Culture and Society | Sustainability and Conservation |

Comments: Very well written with sufficient background to promote understanding to those outside of biomedical research. Consider providing detail on experimental methods if this is a study that will be conducted prior to MURC. All the best!