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The role of adipocyte-derived extracellular vesicles in vimentin mediated fibrosis

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Fibrosis, the accumulation of aberrant myofibroblast and extracellular matrix molecules that make up scar tissue, is a common feature of chronic tissue injury. Progressive fibrosis of major organs is a devastating and fatal process, contributing to almost 50% of mortalities in the developed world. Currently, there are no anti-fibrotic agents that can halt progressive organ fibrosis and cure patients, and organ transplantation is the only treatment for end state organ failure. As in fibrosis of other organs, myofibroblasts are the cells responsible for cardiac fibrosis and scar buildup in the heart and have been reported to originate from resident fibroblasts, epithelial cells, endothelial cells, pericytes, monocytes, and circulating fibrocytes.

Vimentin has been shown to be involved in wound healing, but its functional contribution to this process is not clear. Previous studies show that loss of vimentin led to a severe deficiency in fibroblast growth and vimentin can orchestrate the healing by controlling fibroblast proliferation, TGF-β1–Slug signaling, collagen accumulation, and EMT processing, all of which in turn govern the required keratinocyte activation.

Extracellular vesicles (EVs) as emerging intercellular communicasomes have the potential to target a range of molecular processes and recipient cells and a promising delivery vehicle for improving wound healing. The various studies demonstrate the ability of EVs to target physiological processes and intracellular pathways involved in the haemostatic, inflammatory, proliferative and remodelling phases of wound healing.