**THE CANCER ASSOCIATED FIBROBLAST AND THE HEART FAILURE ASSOCIATED FIBROBLAST- SIMILAR PHENOTYPE, SIMILAR PATHWAYS LEADING TO POTENTIALLY SIMILAR TREATMENT**

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While morbidity, mortality, and health care costs associated with heart failure (HF) are increasing, advancements in early diagnosis and treatment strategies have not been forthcoming. Firstly, there is a need to differentiate HF phenotypes into different disease processes, not dissimilar to what is done in cancers. Secondly, move beyond conventional thought regarding biological pathways that regulate myocardial growth and function. Thirdly, harness the insight gained from advances in biological pathways affected by chemotherapeutics that may hold relevance to HF. HF myocardial fibroblasts express transcriptional and protein markers similar to those observed in a process of mesenchymal-epithelial transformation described in cancer. These cancer associated fibroblasts (CAFs) have been shown to contribute to cancer growth and alterations in normal tissue structure/function through the release of growth factors, signaling molecules, and proteases. This laboratory and others have identified that HF associated fibroblasts (HFAFs) express a similar profile of growth factors and signaling molecules in patient and animal models. The aims: (1) Identify the phenotype classifications of clinical HF and relate these to abnormalities in fibroblast growth/function, (2) present new findings on common signaling pathways and altered expression profiles between CAFs and HFAFs, putting forward the postulate that these cell types are the same, (3) examine new translational studies of localized approaches to target HFAF and how chemotherapeutics hold a place in the treatment of HF. The imminent conclusion is that new findings in cancer research can be translated to target the transdifferentiated fibroblast in HF as a form of interstitial cancer.