**MECHANISM OF CARDIOVASCULAR REMODELING IN HEART FAILURE**

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Remodeling and myocardial matrix metabolism contributes to cardiac endothelium–myocyte (perivascular fibrosis), myocyte–myocyte (interstitial fibrosis), and mitochondrion–myocyte (fusion and fission) coupling. Matrix metalloproteinases (MMPs), and tissue inhibitor of metalloproteinases (TIMPs) play differential roles in different tissues and diseases. For example, although present in the heart, MMP-3 is known as stromelysin (i.e., stromal tissue enzyme). Interestingly, TIMP-3 causes apoptosis. Exercise and nutrition are synergistic in the mitigation of diseases: exercise releases exosomes containing miRNAs. Nutrition/vitamins B6 and B12 regulate the metabolism of homocysteine (an epigenetic byproduct of DNA/RNA/protein methylation). Thus, epigenetic silencing is an important therapeutic target. The statistical analysis of cohorts may be less indicative for the treatment of a disease, particularly if the 2 twins are different in terms of responding to the medicine for the same disease, therefore, personalized medicine is the future of therapy.