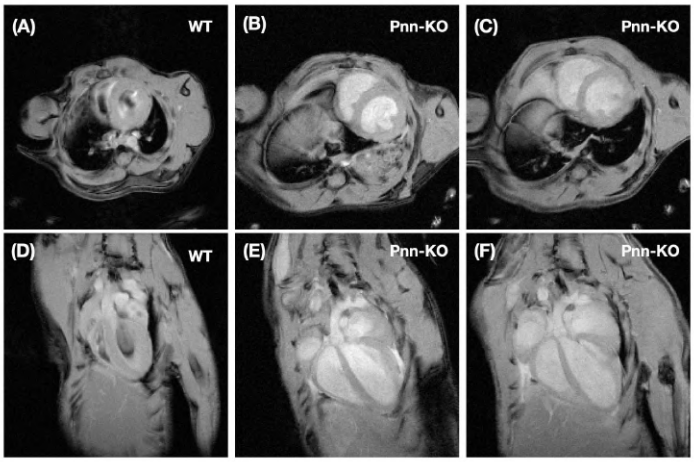
Architecture and Signaling Regulation in Cardiac Stress Response and Cardiomyopathy

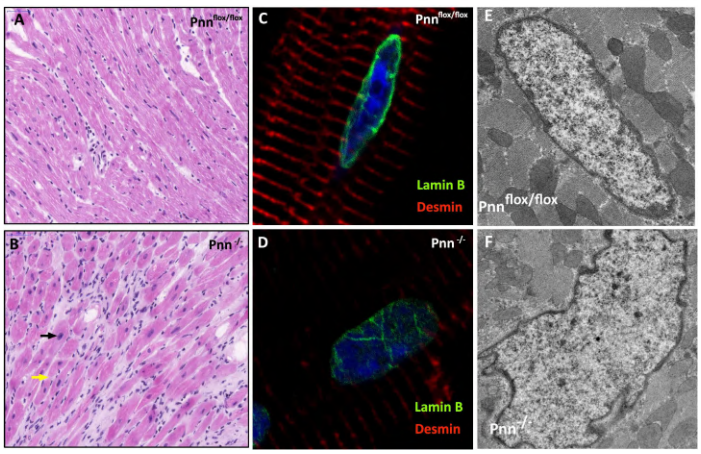
主持人：呂史提 (Steve Leu)

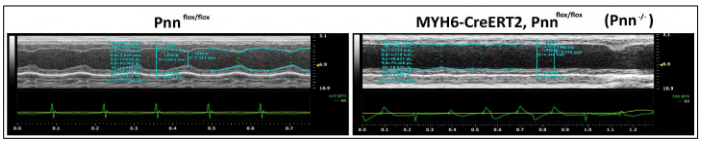
研究助理：邱妤均、陳俐璇

The PI participates in the cardiovascular translational research to investigate the myocardial stress responses, including intercellular communication and intracellular signaling, in cardiac cells post cardiac stresses caused by ischemia, pressure-overload, genetic defects, nutrition insults, or developmental programming. To determine the relationship between stress response and prognosis of heart diseases, the PI applies cardiovascular disease animal models with examination on molecular, cellular, histopathological, and functional levels. Animal models with cardiovascular diseases and gene manipulation are utilized to reveal the underlying mechanism of architecture and signaling regulation in myocardium.

Cardiomyocyte-specific Pnn deficiency results in arrhythmogenic dilated cardiomyopathy (ADCM) in mice



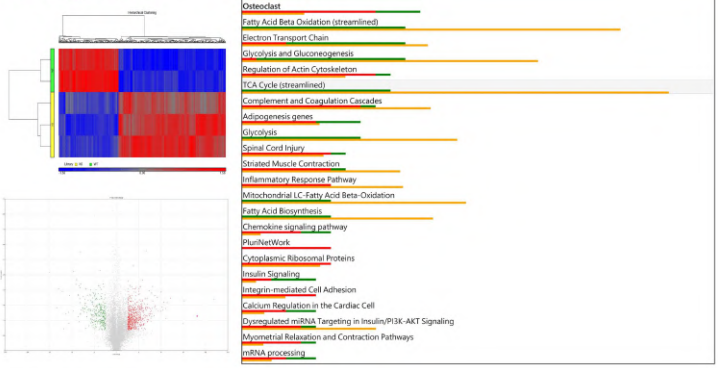




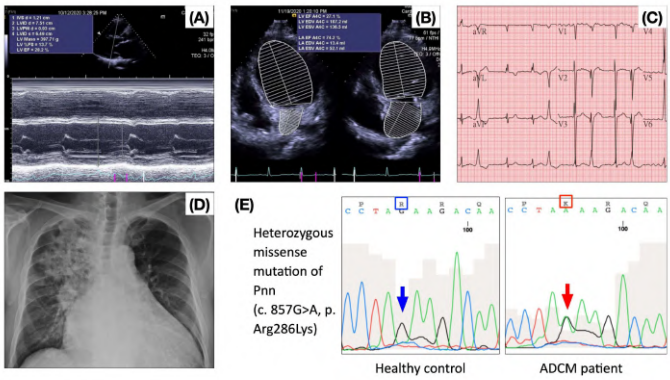
3 Pnn KO mice : 2 Flox mice

Up-regulated: 659

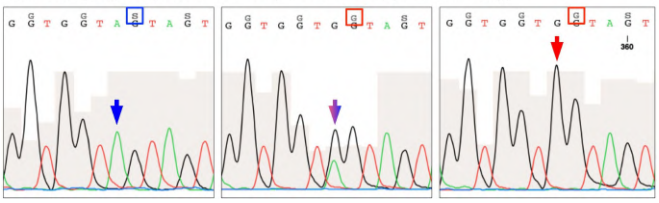
Down-regulated: 333

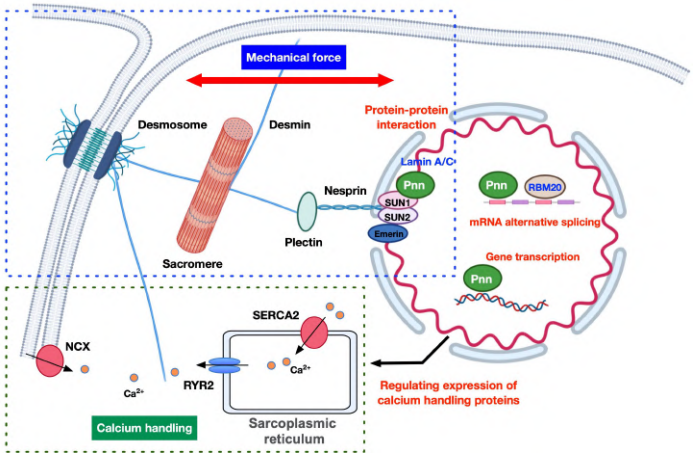


Characterization of pathogenic Pnn mutants in patients with arrhythmogenic dilated cardiomyopathy



Genetic polymorphism of Pnn (c. 2011A>G, p. Ser671Gly)





Genetic defects in Pnn lead to ADCM in human through affecting the response of cardiomyocytes against mechanical force as well as regulating the expression and mRNA alternative splicing of genes involved in intracellular calcium handling