**MECHANISMS OF DCM DUE TO CELL CYCLE STRESS**

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Dilated cardiomyopathy (DCM) is associated with diminished cardiac output due to defective performance of ventricular muscle. While genetic mutations in genes critical for ventricular function as well as various insults such as alcohol, infection and drugs are known to induce DCM, a significant portion of the cases of the disease remain idiopathic. The E2F/Rb pathway is a major regulator of the cardiac cell cycle and dictates growth, death and differentiation. We have utilized the repressor E2F6 to interfere with this pathway in the postnatal myocardium to impose stress on the cardiac cell cycle and interrogate impact on the heart. Transgenic mice with cardiac specific expression of E2F6 exhibit an early DCM in the absence of any hypertrophy or cell death in the postnatal myocardium. E2F6 expression impacted the cardiac cell cycle dynamics with an extended S phase. This was accompanied by major changes in the differentiation program which impacted cellular metabolism and beta-adrenergic signaling with the appearance of DCM. Thus selective pressure on the cell cycle of the postnatal myocardium can induce DCM.