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Myocardial hypertrophy, usually caused by a combination of increased load and reduced function, plays a critical role in the development of heart failure, arrhythmias and sudden cardiac death. Despite the importance of this disease, little is known about the molecular signals that trigger cardiac hypertrophy. Myocyte stretch (swelling), a pathological condition observed in many types of heart disease, plays an important role in the development of cardiac hypertrophy. However, the molecular mechanisms that allow cardiac hypertrophy in response to mechanical stress remain unknown. In this proposal, we will address the hypothesis that mechanical stress of the myocyte activates a small GTP binding protein, RhoA, to stimulate the phosphorylation and activation of the focal adhesion kinase (FAK), a protein tyrosine kinase that plays a central role in the activation of many signal transduction pathways. We further hypothesize that RhoA plays a central role in the generation of the increase in cardiomyocyte protein synthesis, the molecular determinant of cardiac hypertrophy. Our recent studies show that activation of RhoA is required for cardiomyocyte hypertrophy in response to various stresses, such as volume overload and mechanical stretch. Moreover, we have identified a small GTPase inhibitor, Rhosin, that inhibits the activation of RhoA and the induction of cardiomyocyte hypertrophy in response to myocyte stretching. We also have shown that the activation of RhoA and the increase in cardiomyocyte protein synthesis are preceded by a rapid activation of FAK. Furthermore, we have identified one molecular intermediate, phosphatidylinositol 3-kinase (PI3K), that is important in the activation of FAK by mechanical stress. Using hypertrophying models in vivo and in vitro, we have shown that FAK, PI3K and MAP kinase are essential for the induction of cardiomyocyte hypertrophy by mechanical stress. Since RhoA is a molecular effector of PI3K, we will

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