

Exploring Diabetic Cardiomyopathy  
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Integrins have been found to be important mechanoreceptors between the myocardium and the extracellular matrix (ECM), serving an essential role for normal cardiovascular function in the adult heart. Integrin signaling triggers the activation of downstream molecules such as focal adhesion kinase (FAK) and paxillin. Altered integrin localization has been linked to the altered expression of extracellular matrix components as a result of cardiac hypertrophy, cardiomyopathy, and myocardial infarction.

Hyperglycemia has been associated to many myocardial diabetic complications. The present study investigates the effects of high and low glucose, with and without insulin, on cellular adhesions and signaling to the ECM through changes in FAK, paxillin, and  $\beta_1$ D integrin. Because heart muscle is not a continuous cell line, skeletal muscle from mouse soleus (Sol 8) cells was used for this *in vitro* study and treatments were assessed using densitometry analysis of western blots, immunocytochemistry, and an MTT assay was used to determine cell viability.