DIFFERENTIAL PROTEIN KINASE C AND EXTRA CELLULAR REGULATED KINASE ACTIVATION IN COMPENSATED RIGHT VENTRICULAR HYPERTROPHY SECONDARY TO EMPHYSEMA

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Based on models of previously constructed micro-patients with chronic obstructive pulmonary disease (COPD) frequently develop right ventricular hypertrophy. Right ventricular hypertrophy (RVH), resulting from COPD is an area of cardiovascular research that has gained interest in the University of Utah Health Sciences Community. Causes and implications of RVH resulting from emphysema are unknown and controversial.

A Syrian golden hamster model was used to characterize the development of RVH secondary to emphysema. Emphysema was created in 17 hamsters and 15 age matched animals were used as controls. Hamsters were evaluated after nine months to determine right ventricular contractility using echocardiograms; activation of protein kinases that have been previously identified to mediate other models in left ventricular hypertrophy, and size of cardiac myocytes. Current analysis is focused on final determination of myocyte size.

RV cardiac myocytes were isolated through a two-step process using a KDH solution and enzymatic digestion with trypsin. Using light microscopy and digital photography, pictures have been taken of individual myocytes from the right ventricle of the control and emphysema hamsters. Myocyte dimensions will be analyzed using the software Scion Image. Myocyte area and perimeter will be determined on approximately 100 cells from each hamster and means will be calculated to determine if there was an increase in myocyte size that led to right ventricular hypertrophy.