Introduction

Asthma is a complex pulmonary disease affecting around 300 million individuals globally. It is characterized by chronic inflammation and hyper-responsiveness to various stimuli leading to airway remodeling and increased smooth muscle mass. The primary cells responsible for this process are the fibroblasts, which can remodel and contract collagen, allowing for the structural changes observed in asthma.

Hypothesis

Non asthmatic airway fibroblasts significantly contract collagen I gels more than fibroblasts from asthmatic airways. Both cell types show similar rates of contraction.

Methods

Both parenchymal and airway fibroblasts used in this study were obtained from donor lungs deemed not suitable for transplant. Both cell types were obtained in three passages and expanded to higher densities. Collagen gels were prepared in serum free DMEM at pH 7.4. Both cell types were seeded onto collagen I gels and allowed to settle for 20 minutes. Both cell types were then analyzed for protein levels using western blot analysis.

Summary

Non asthmatic airway fibroblasts significantly remodel collagen fibers into more dense collagen fibers as compared to asthmatic derived fibroblasts.

Future Directions

In order to determine why asthmatic airway fibroblasts could not contract collagen I gels as efficiently as their non asthmatic counterparts, western blot analysis for the proteins involved in collagen remodeling and contraction will be conducted.

A difference in these protein levels may help to explain why asthmatic airway fibroblasts do not remodel the collagen fibers as densely and then do not contract the gels as much as non asthmatic airway fibroblasts.

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