CHRD 2021: Abstract & Poster Submission Form

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Research Category:

- ◉ Basic Science
- ○ Clinical
- ○ Community Health / Policy

What was your role in the project?

- ☑ Design
- ☑ Perform Experiments
- ☑ Analyze Data
- ☑ Write Abstract
- ☐

Presenter Status:

- ◉ Undergraduate Students
- ○ Masters Student
- ○ PhD Student
- ○ Post-Doctoral Fellows
- ○ Residents
- ○ Non-Trainee

Title

A 3D Bioprinted Model of Airway Smooth Muscle to Replicate Airway Stiffening in Asthma

Background

Asthma is the leading cause of hospitalization in Canadian children. It is characterized by excessive contraction of airway smooth muscle (ASM) and stiffening of the airway walls. Conventional experimental models ineffectively replicate airway stiffening, making it difficult to understand asthma pathogenesis.

Objective

We used novel 3D bioprinting technology to develop an experimental model of ASM to simulate the effect of stiffened airway walls.

Methods

ASM cells were resuspended at 2.5×10^7 cells/mL in a bioink comprised of RGD-coupled-alginate (0.375% w/v), fibrinogen (5 mg/mL) and collagen-I (1 mg/mL). Cells were printed as a ring constrained within a “spiderweb” acellular frame (0.75-1.25% w/v alginate) and secured in a specially designed tissue holder. Luminal area was used to assess muscle shortening. Contractile function was assessed using potassium chloride and cytochalasin-D, while the depolymerization of the acellular structure with citrate/EDTA was used to assess the relative strength of the structure versus the muscle.

Results

ASM rings exhibited excellent physical stability in the tissue holder during culture. ASM minimally constricted during serum withdrawal, while KCl caused a predictable, but modest contraction (<5% lumen area reduction). Cytochalasin-D was capable of reversing the KCl contraction and allowed the tissue to expand to a higher luminal area than pre-KCl levels, indicating the structure was highly elastic. The effect of acellular stiffness on contraction and relaxation was not significant ($p=0.7883$). Depolymerization resulted in a rapid (<1 minute) and large reduction in luminal area (>50%), indicating the acellular structure was providing a very strong load opposing contraction. Reductions in the lumen area were significantly higher in the 0.75% acellular tissues ($p<0.05$).

Conclusion

Our bioprinted model of ASM is capable of replicating very stiff airway walls. Future work will characterize how ASM responds to varying stiffnesses, providing potential insight into the role of airway stiffening in asthma pathogenesis.

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