CHRD 2021: Abstract & Poster Submission Form

Submitter Name

Mitchell
First

Wilson
Last

Email

wilsonm1@myumanitoba.ca

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

Nicotine Promotes Airway Fibroblast Senescence

Background

Asthma and chronic obstructive pulmonary disease (COPD) are both associated with environmental risk factors. Exposure to maternal diabetes and/or cigarette smoke increases risk for childhood asthma and may play a role in COPD development. Premature cell aging (senescence) is important in the pathophysiology of lung disease. However, it is unclear if exposure to nicotine or hyperglycemia, particularly in utero, can promote premature cellular senescence in lung fibroblasts, a key cell in lung disease.

Objective

Exposure to hyperglycemia and/or nicotine accelerates the transition to senescence in human lung fibroblasts.

Methods

Human lung fibroblast cells (HLF) were cultured in high glucose (HG) media with or without nicotine. Cells serially grown and passaged in standard media acted as a senescence control. Cells were maintained in conditioned media for four passages (P1 to P4). Changes in proliferation were assessed using population growth curves. Relative abundance of interleukin 8 (IL8) and senescence markers P21 and Lamin-B1 were measured using qPCR. Data presented as relative abundance, n=1.

Results

Exposure of HLF to nicotine or HG+nicotine increased doubling time at every passage. By P4, doubling times were 3.48 days for HG+N, 2.8 days for LG+N, 1.97 days for HG, and 1.95 days for senescent control. In the senescent control, P21 was not elevated until P4 where it increased by 2.0-fold. Comparatively, P21 was elevated at P1 by 1.5-fold and 1.9-fold in nicotine and HG+nicotine treated cells, respectively. Furthermore, HG+nicotine decreased Lamin-B1 abundance at P1 (1.5-fold). IL-8 was only changed in the senescent control.

Conclusion

Co-exposure to hyperglycemia and nicotine increased doubling times of HLF and modified markers of senescence. These changes suggest in utero exposure to nicotine promotes senescence, which may be linked with disease pathogenesis. Further research to understand this interaction will require increased sample size and diversification of senescence markers.

Authors

- For each author, please click "**[+] Add Item**" and provide the author's information

Name	Email	Role	Profession
Mitchell Wilson	wilsonm1@myumanitoba.ca	Presenting Author	
Shana Kahnamoui Zadeh	skahnamouizadeh@chrim.ca	Co Author	
Kaylene Normand	normandk@myumanitoba.ca	Co Author	
Christopher D. Pascoe	cpascoe@chrim.ca	Co Author	Assistant Professor