ABSTRACT SUBMISSION FORM

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SEX + GENDER

Exploring the role of sex and gender on health research

CHRD
CHILD HEALTH RESEARCH DAYS

CHRD 2020: Abstract Submission Form

Submitter Name
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Title
COPD and Diabetes: The impact of hyperglycemia on fibroblast phenotype

Background
Chronic obstructive pulmonary disease (COPD) is a chronic obstructive pulmonary disease with an increasing mortality in Canada. COPD has many co-morbidities which increase disease mortality. An underappreciated comorbidity in COPD is diabetes, which increases both hospitalizations and mortality. In the lung, fibroblasts inflammation and senescence may be important for disease progression and exacerbations.

Objective
We hypothesized that hyperglycemia causes cellular senescence and inflammation in COPD fibroblasts, when compared to non-COPD.

Methods
Human airway fibroblasts, obtained from COPD and non-COPD patient lungs were cultured in low glucose (LG) (5.5 mM) and high glucose (HG) (25mM) conditions. Fibroblasts from three females and one male patient were used. Hoechst dye was used to visualize count cells for proliferation over 72 hours. qPCR was used to measure the relative abundance of p21, collagen (COL1A1), and interleukin-8 (IL-8) with or without transforming growth factor stimulation (2.5ng/mL) (TGF-β) for 24 hours. RNA abundance was normalized to three housekeeping genes. Data presented as mean ± SEM, significance defined as $p<0.05$.

Results
Exposure to HG conditions did not alter proliferation in non-COPD cells, but may slow proliferation in COPD cells (n=1).The addition of TGF-β to cells caused a significant increase in p21 expression in both LG (1.7±0.17 fold, n=2) and HG exposures (1.5±0.19 fold, n=2). HG media, regardless of disease status, increased IL-8 mRNA abundance (2.5±0.71 fold, p=0.06, n=4). COPD cells displayed a significant reduction in TGF-β induced COL1A1 production. Furthermore, HG suppressed TGF-β induced COL1A1 expression in non-COPD cells by 4-fold (9.6± 0 to 2.1±0.02 fold, n=1-2) but only by 1.4-fold in COPD cells

(3.4±0.01 to 2.5±0.1 fold, n=2).

Conclusion
This data suggests HG promotes inflammation and impairs the normal wound-healing response in fibroblasts. Further measurements of senescence need to be evaluated, and the mechanism relating HG exposure and TGF-β insensitivity needs to be explored.

Theme:
Basic Science

Do you have a table/figure to upload?
No

Are you willing to participate in Goodbear's Den?
Yes

Presenter Status:
Undergraduate Students

What was your role in the project?
Perform Experiments

Authors

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