

CHRD 2024: Abstract Submission Form

Presenter Name

John Paul Aguilar

Presenter Status

Masters Student

Role in the project

Design
Perform Experiments
Analyze Data
Write Abstract

Research Category

Basic Science

Title

Amniotic Fluid as Mediator of the Maternal Environment: The AMME Study

Background

Fetal breathing movements are part of normal lung development and permit the exchange of lung fluid with amniotic fluid (AF). Changes in AF composition, including cytokines, could therefore alter lung development and health. AF may bridge the maternal external environment and the developing lungs. Exposure to cigarette smoke (CS) in-utero increases risk for chronic lung diseases like asthma. It is unknown if environmental (including CS) exposures changes AF cytokine profiles.

Objective

The aim of the study is to characterize the cytokine profile of human AF in smokers and non-smokers and determine if airway epithelial cells respond to these changes.

Methods

Matched AF, maternal (MB), cord blood (CB), and placentae (PL) were collected from patients undergoing a term caesarean delivery. Cotinine was measured using an enzyme-linked immunosorbent assay. Cytokine/chemokine profiles were measured using a 96-plex assay and profiles compared using partial least squares-discriminant analysis and t-tests. Epithelial cells were exposed to AF, and trans-epithelial electrical resistance (TEER) was measured. Data is presented as mean±SD.

Results

Cotinine was detected in 29% AF samples at levels 1.38-fold higher than in maternal blood ($p < 0.05$). AF cytokine profiles are distinct from MB, CB, and PL, characterized by higher abundances of IL-6 (63-, 293-fold), IL-1RA (29-, 104-) and MCP-1 (3-, 3-fold, relative to CB and MB respectively). Cotinine-positive AF had higher levels of IL-6 and CXCL9 ($p < 0.0001$, $p < 0.005$ respectively). Cytokines like RANTES and sCD40L are negatively correlated with epithelial barrier function.

Conclusion

AF has a distinct cytokine/chemokine profile from MB, CB, and PL. CS exposure prenatally alters the cytokine and chemokine profile of AF, increase in pro-inflammatory IL-6, and bioaccumulation of cotinine. This may provide a novel insight into the role AF plays in mediating the external and fetal environments and in understanding its role in the developmental origins of chronic lung disease.

Do you have a table/figure to upload?

No

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