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Investigation of host and pathogen responses to estrogen in cystic fibrosis

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Introduction

A 'gender gap' exists in Cystic Fibrosis (CF). Females acquire earlier microbial infections; have worse lung function and poorer survival rates [1]. The sex-hormone estrogen (estradiol, E2) has recently been highlighted as a key molecule responsible for the CF gender dichotomy [2]. *Pseudomonas aeruginosa* which colonises the CF lung and dominates at end stage disease undergoes mucoid conversion in response to E2 [2,3]. The aim of this project was to study other roles of E2 in host and pathogen responses by investigating its effects on the growth rate of *Ps. aeruginosa* and the expression of catalase and superoxide dismutase (SOD) in CF bronchial epithelial cells.

Methods

Growth rate of *Ps. aeruginosa* (PA01) in the presence or absence of E2 was measured by recording optical density (OD_{600nm}) at different time points and by calculating cfu/ml. Measurements of catalase and SOD gene expression in E2-treated CFBE410- airway epithelial cells were carried out using real time qRT-PCR. Results were analysed using Graphpad PRISM 5.0.

Results

E2 had no effect on the growth of *Ps. aeruginosa* when compared to control. The expression of catalase mRNA in CFBE410- cells in response to E2 was not altered however, there was two-fold increase in SOD gene expression in response to 10 nM E2, 24hr (p= 0.0057).

Conclusion

Estradiol has no effect on the growth of *Ps. aeruginosa in vitro*. In CF bronchial epithelial cells although catalase gene expression remains unchanged, E2 increases SOD expression, potentially increasing hydrogen peroxide levels and contributing to *Ps. aeruginosa* mucoid conversion.

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