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Respiratory Pathogens Inflammation and Virulence

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Abstract

Changes in bacterial phenotype associated with virulence were observed following exposure to e-cig vapour. In some cases the observed phenotypic changes were less than those observed with cigarette smoke-exposed bacteria. However, in general, there was little difference in the effect on exposure of bacteria to cigarette smoke or e-cig vapour, with exposure to either resulting in increased virulence and inflammatory potential of the bacterial isolates.

Keywords: Bacterial phenotype; Bacterial isolates; Asthma exacerbations; H.Influenza; E-cigar vapour; Immune evasion;

Introduction

Several studies have suggested an effect of ECVE on cultured lung cells, ranging from increased inflammation, measured by increased cytokine production, to changes in the microvasculature. Increased cytokine production and evidence of lung injury has also been observed following exposure of mice to e-cig vapour and nicotine, together with a reduced capacity to clear either bacterial or viral infection [1]. These findings suggest an inflammatory lung environment similar to that observed following cigarette smoking. Many e-cig users have previously been cigarette smokers; therefore, it is difficult to attribute any changes in lung function to e-cigs alone. However, perhaps driven by concerns over cigarette safety, many adolescents who have never smoke, are now taking up vaping, resulting in evidence of an association between e-cigarette use or exposure, and increased asthma exacerbations. There is therefore a need to understand the long-term impact of e-cigarette use and second hand ECV exposure, particularly on the lung health of vulnerable populations. Bacterial colonization and infection of the airways is a contributing factor to lung function decline across a range of chronic lung diseases and a recognized risk of tobacco smoke exposure [2]. However, the extent to which cigarette smoke, or e-cig vapour drives the establishment of bacterial colonization and aids persistence of these bacteria has not been extensively studied in all key pathogens implicated in chronic lung disease. H. influenza, S. pneumonia, P. aeruginosa and S. aureus are consistently associated with lung function decline, increased severity of disease and increased rate of exacerbation in chronic lung diseases in which smoking also plays an important role [3]. Establishment of biofilm by these pathogens is a significant virulence determinant in the pathophysiology of chronic lung disease, and is associated with establishment and persistence of infection, resistance to antibiotics and evasion of the host immune system. In this study, biofilm formation increased in all isolates in response to both cigarette smoke and e-cig vapour. Furthermore, the degree of biofilm formation observed following exposure of bacterial isolates to either cigarette smoke or e-cig vapour, was similar and suggests that bacterial exposure to either cigarette smoke or e-cig vapour may promote bacterial adhesion, biofilm formation and thus establishment of persistent infection [4]. This reflects previous studies, which demonstrated similar findings following cigarette smoke exposure of lung and oral pathogens. In all cases, genes associated with biofilm formation were found to be up regulated, and this was linked to oxidative stress resultant from cigarette smoke exposure. Changes were also observed in expression of genes encoding for bacterial cell surface structures, resulting in increased bacterial adhesion to epithelial cells. MRSA exposed to cigarette smoke had increased hydrophobicity and altered surface charge, which resulted in increased adherence to epithelial cells and decreased bacterial susceptibility to antimicrobial peptides, respectively [5]. In the case of P. gingivalis, increased expression of fimbrial proteins induced TLR2 hyposensitivity and hence altered immune responses. The effect of e-cig vapour was not investigated in these studies, and further work will be required to determine if the observed increases in biofilm following e-cig vapour exposure occur by similar mechanisms. In this study, there was limited evidence of structural change by electron microscopy, following exposure of bacteria to either cigarette smoke or e-cig vapour. Future work will therefore more fully investigate changes in bacterial transciptomes following exposure to vape or tobacco smoke. Increased biofilm formation subsequent to cigarette smoke / e-cig vapour bacterial exposure is suggestive of increased isolate virulence, and this hypothesis was further explored in the G.mellonella model [6]. Numerous studies have shown that microbial pathogenesis and bacterial virulence are comparable in humans, mice and G. mellonella, Statistically significant decreases in larvae survival, were observed for all bacteria exposed to cigarette smoke, and for all bacteria exposed to e-cig vapour, except H. influenza. Mammalian models of lung infection will be required to more fully assess changes in host pathology following infection with cigarette smoke / e-cig vapour exposed bacteria; however, our aim in this study was to assess gross changes in bacterial virulence. A particularly striking finding of this study was the change in lung inflammation observed following infection of A549 cells with bacteria exposed to either cigarette smoke or e-cig vapour. Dys-regulation of the lung inflammatory response is a hallmark of chronic lung disease, such as COPD, where it is persistent, observed long after exposure to cigarette smoke has ceased, and attributed to bacterial colonization. With the exception of S. pneumonia, IL-8 secretion from A549 cells was significantly increased in all isolates following infection with bacteria exposed to cigarette smoke and e-cig vapour, compared to infection with non- cigarette smoke / e-cig vapour exposed bacteria. Of particular note, was that there was no difference observed between levels of IL-8 produced following infection with bacteria + cigarette smoke vs. bacteria + e-cig vapour, with the exception of S.aureus [7]. In this case,

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exposure to e-cig vapour resulted in increased IL- 8 levels compared to cigarette smoke. Levels of TNF-α were similarly increased following e-cig vapour exposure with H.influenza, S. pneumonia and S.aureus. These data indicate that bacteria exposed to cigarette smoke promote a greater inflammatory response in A549 cells than in non-exposed bacteria, but that this is closely matched and in some cases exceeded by the level of inflammation observed following exposure to e-cig vapour. Altered immune responses, which promote bacterial persistence, have previously been observed with S. pneumonia, following airway cell-cigarette smoke exposure and with cigarette smoke - exposed MRSA. MRSA exposure to e-cig vapour has also been described as altering immune-modulatory cytokines in the airways of mice. Our findings expand on this work to show that exposure of other key respiratory pathogens to both cigarette smoke and, in particular, e-cig vapour has the potential to modulate host response to infection and we speculate that this could contribute to the increased inflammation and bacterial persistence characteristic of smoking-related chronic lung disease. The epithelial cell-line A549 were considered to be suitable for this study since the epithelium is the major source of lung immune-modulatory factors and is hence critical in the modulation of inflammatory diseases such as COPD and bronchiectasis. Furthermore, they are well characterized and standardized, allowing for rigorous comparison of bacterial infections. Future studies will more fully analyse the host response to cigarette smoke / e-cig vapour exposed bacteria in a range of primary cell cultures, but this is outside the scope of the present study. Addition of a range of immune pathway inhibitors suggested that the cell-signalling pathway utilized in response to infection is dependent on the bacterial species involved [8]. Furthermore, the results did not indicate that increased cytokine production in response to bacterial exposure to e-cig vapour was occurring via an alternative cell-signalling pathway, compared to bacterial infection alone or cigarette smoke-exposed bacteria. Moreover, bacterial cigarette smoke / e-cig vapour exposure enhanced the immune-modulatory effect observed. Increased activation of both NFκB and MAPK signalling pathways have been implicated in the pathogenesis of COPD and asthma, with NFκB up regulation further associated with steroid insensitivity, but the potential contribution of bacterial infection to this pathway is still poorly understood. Our findings clearly indicate that these pathways may be further up-regulated by exposure of key lung pathogens to cigarette smoke or e-cig vapour. The bacterial lung community is complex and increased airway inflammation subsequent to bacterial exposure to cigarette smoke / e-cig vapour is likely to be mediated via a range of signalling pathways. Understanding each of these, and their respective contribution to inflammation in vivo may provide insight into potential therapies to reduce the effects of persistent bacterial-induced inflammation. A recurring theme of this study is the similarity observed in the effect of exposure to cigarette smoke compared to e-cig vapour on bacterial phenotype and virulence. Cigarette smoke was generated in accordance with previously published and accepted protocols: however, this is a potential limitation of this study [9]. In order to ensure comparability, cigarette smoke and e-cig vapour were prepared using a similar method. This may not represent a true reflection of differences between smoking and vaping, e.g. it fails to take account of the differences in puffing topography between conventional and electronic cigarettes, and between individuals. E-cigarette users take larger and longer puffs, compared to conventional cigarette users, which may increase nicotine delivery. Our model may therefore underestimate the exposure of respiratory pathogens to e-cig vapour. Our current protocol is also based on a one-off exposure to cigarette smoke / e-cig vapour, and used a brand of e-cigarettes with no added flavour: however, flavourings and e-cigarettes additives have been associated with changes in the bronchial epithelia and impairment in respiratory innate immunity. Further studies are therefore required to investigate the effect of both common e-cigarette flavourings and long-term exposure of bacteria to cigarette smoke / e-cig vapour. Furthermore, only reference isolates were used in this study and further work investigating a wider range of clinical isolates is required [10]. Exposure of respiratory pathogens to e-cigarette vapour induced changes in phenotype and virulence, which may increase bacterial persistence and inflammatory potential. These changes were similar, and in some cases exceeded, those observed following bacterial exposure to cigarette smoke and suggest that there is little difference between the effect of cigarette smoke and e-cig vapour.

Conclusion

There is therefore an urgent need for further robust clinical studies investigating and clarifying the long-term effect of e-cigarette use on both airway cells and respiratory pathogens to enable a better informed judgment to be made regarding their safety.

Acknowledgement

None

Conflict of Interest

None

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