

Results from our research....

Leicester Respiratory Biomedical Research Unit

An acute worsening of lung health is not associated with the function of blood cells responsible for recognising bacterial and viral infection

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Summary (Abstract): Patients with Asthma and chronic obstructive pulmonary disease (COPD) are susceptible to acute worsening of their lung health due to infections caused by virus or bacteria. Lung defences against viral or bacterial infection utilise cells that migrate from blood to the lung to help fight infection. Previous studies in this area have shown that a certain type of immune system cell or white blood cell is impaired compared to people without respiratory disease. We have examined the frequency and function of these cells and their association with worsening lung health. We have shown that blood cells from asthma patients have a reduced response to bacterial proteins due to a reduced frequency of this cell in blood and not because they do not work properly. We also show that the function of these cells are not associated with an acute worsening of lung health.

Introduction: Patients with chronic lung disease are susceptible to acute worsening of their health (described by doctors and scientists as an 'exacerbation') due to infection caused by virus' or bacteria. Lung defences against viral or bacterial infection relies, in part, on appropriate signals to attract blood cells to the lung to help fight infection. Once they arrive they need to recognise the infection, utilising specific receptors, in order to direct appropriate killing responses by the immune system. Previous studies, conducted elsewhere, had shown that responses through these receptors were impaired in asthma (i.e. the cells were not working properly) but they had not looked at the relationship between the function of these cells and susceptibility to acute worsening of lung health. We measured the function of three of these receptors and their relationship to acute worsening of lung health.

Methods: We collected blood samples from COPD, asthma and healthy controls who visited Glenfield Hospital. In the laboratory, we exposed people's blood cells to viral and bacterial products (as a surrogate for infection) and measured their ability to produce a protein that helps fight infection. We then examined whether there was a relationship between a person's response and their susceptibility to infection. There is one white blood cell type (in particular) known to produce these responses. We measured the frequency of these cells in control and asthma blood samples utilising specific cell surface markers and cell analysing equipment. We also purified these cells and assessed their function to determine whether their function was different between health and asthma blood samples as previously described by others.

Results: We found that in patients with asthma, the blood cell response to a bacterial product was significantly reduced compared to those with COPD or controls without lung disease. This reduction, however, was not associated with susceptibility to an exacerbation. We confirmed that there is only one white blood cell type (a rare type of dendritic cell that is present in everybody's

blood) that responds to this bacterial product. We then showed that the reduced response that we saw in asthma was more likely due to a decreased number of these cells in blood rather than because these cells do not work properly.

Conclusion: We have shown that a certain type of blood cell, important for helping the fight against infection in the lung, is present at a reduced frequency in asthma blood samples but this is not associated with exacerbations. Contrary to other studies, we showed that the function of these cells from asthma and healthy people are not different.

Reference to published paper: If you would like to be sent the full paper then please get in touch with Adam Wright on aw287@le.ac.uk, or through the BRU. If you would like to receive news and events and join our mailing list please complete the online submission form on our website.



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