

Metabolic activity of neutrophils is elevated in chronic obstructive pulmonary disease: Are we sure?

Sir,

Vaidyanathan and Damodar have recently reported that there is an increase in metabolic activity of neutrophils in COPD, which possibly contributes toward lung tissue damage in chronic obstructive pulmonary disease (COPD) subjects.^[1] This letter involves a critical analysis of the observations made by Vaidyanathan and Damodar.

First, the authors have claimed that there are no published studies that had assessed the metabolic activity of neutrophils in Indian COPD patients by nitro blue tetrazolium test. However, I would like to draw the attention toward the most recent published research by Shanmugam *et al.*,^[2] who happen to report similar findings as that of Vaidyanathan and Damodar.^[1]

Second, the authors have compared the tendency for activation of neutrophils on exposure to *Escherichia coli* endotoxin of COPD patients and healthy individuals.^[1] However, it was difficult to understand why the authors particularly select *E. coli* endotoxin to activate the neutrophils, rather than any other bacterial or chemical stimulation.

Third, the authors have recruited the COPD subjects according to Global initiative for Chronic Obstructive Lung Disease (GOLD) and observed that 87% of the COPD patients had a smoking history.^[1] There is no explanation regarding how the remaining 13% COPD subjects were recruited in the study. In addition, the authors have reported that the duration of smoking history ranged from 0 to 50 years,^[1] indicating the variability of COPD subjects recruitment and the introduction of sample biases.

Fourth, it can be inferred from the observations that neutrophil percentage among stimulated COPD group is higher than nonstimulated group.^[1] Furthermore, the authors have mentioned that the 33% of COPD subjects have received doxofylline concurrently.^[1] There is a lack of information whether the increase in neutrophil percentage among stimulated group is significantly higher compared to nonstimulated group. If there is an insignificant increase, then it could be possibly attributable to doxofylline. Prior animal studies have reported that doxofylline is found to inhibit neutrophil recruitment upon lipopolysaccharide stimulation.^[3]

Finally, in this study, it would have been interesting to find out, if there is a correlation of metabolic activity of neutrophils with that of lung function among COPD subjects, this would further help in understanding the COPD pathogenesis.

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Conflicts of interest

There are no conflicts of interest.

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