The Influence of Non-Steroidal Anti-Inflammatory Drugs and Paracetamol on the Expression of the Lung Coronavirus (COVID-19) Attachment Receptor and Enzyme Genes in Mice.

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Abstract

Introduction: There are several reports that favor the use of paracetamol over non-steroidal anti-inflammatory drugs in the management of respiratory viral infections, including COVID-19.

Aims: This study aimed to find out the effect of the most commonly used analgesics paracetamol, ibuprofen, and diclofenac on the mRNA expression of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) entry associated genes and arachidonic acid metabolizing genes in the lungs of mice.

Methods: Twenty eight Balb/c mice were divided into 4 groups and were treated separately with the vehicle, paracetamol, ibuprofen, and diclofenac in clinically equivalent doses for 14 days. Then, the expression of SARS-cov2 entry genes ace2, tmpress, and cathepsin genes, in addition to the arachidonic acid metabolizing genes cyp450, cox, and lox were analyzed using real-time PCR assay.

Results: It was found that paracetamol significantly downregulated ($P < 0.05$) the expression of tmpress and cathepsin genes by 8.5 and 5.6 folds, respectively, while ibuprofen and diclofenac significantly upregulated ($P < 0.05$) the expression of the ace2 gene by more than 2.5 folds. In addition, all tested drugs downregulated ($P < 0.05$) cox2 gene expression, and paracetamol reduced the mRNA levels of cyp4a12 and 2j5 genes by 3 and 4.2 folds, respectively. Lastly, diclofenac induced the expression of the drug metabolizing gene cyp2c29 by 4.8 folds. Conclusion: It can be concluded that the most commonly used analgesics significantly affected the mRNA expression of SARS-cov2 entry associated genes and arachidonic acid metabolizing genes in the lungs mice. Further clinical studies are needed to confirm findings of this study.

Keywords: Analgesics, arachidonic acid, gene expression, lung, SARS-cov2.