THE ROLE OF OXIDATIVE STRESS IN APOPTOSIS AND CELL PROLIFERATION OF HUMAN BRONCHIAL EPITHELIAL CELLS

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Oxidative stress is an important pathophysiological factor in chronic respiratory diseases. Our study aimed at elucidating through which pathway oxidative stress–mediated apoptosis occurs at the gene expression level under oxidative stress in the human bronchial epithelial cell line BEAS-2B. Suitable doses and time period were detected by exposing BEAS-2B cells to hydrogen peroxide (H_2O_2) at different doses and time periods, and the oxidative-damaged cell culture model was designed. The treatment and control groups were compared in terms of gene expression levels determined by Quantitative Real Time Polymerase Chain Reaction. The oxidative-damaged cell model was confirmed by the spectrophotometric measurement of malondialdehyde and catalase activity ($p < 0.05$). Caspase-3, caspase-9, bax, and bak gene expression levels increased significantly in the treatment group compared to the control group ($p < 0.05$). There were not any significant differences between the groups in terms of caspase-8, Bcl-2, and bik ($p > 0.05$). p53 and p21 gene expression levels were found to be significantly higher in the treatment groups ($p < 0.05$). H_2O_2-induced oxidative stress, induced apoptosis through the intrinsic pathway at gene expression level in the bronchial epithelial BEAS-2B cells was observed.

Key words: *Apoptosis, BEAS-2B, Cell proliferation, Oxidative stress, Reactive oxygen species.*

РОЛЬ ОКСИДАТИВНОГО СТРЕСУ В АПОПТОЗІ І ПРОЛІФЕРАЦІЇ КЛІТИН БРОНХІАЛЬНОГО ЕПІТЕЛІЮ ЛЮДИНИ

Оксидативний стрес є важливим патофізіологічним фактором при хронічних респіраторних захворюваннях. Мета нашого дослідження полягала у вивченні шляхів виникнення апоптозу, спричиненого оксидативним стресом, на рівні експресії генів під впливом оксидативного стресу у лінії клітин бронхіального епітелію людини BEAS-2B. Відповідні дози та часові проміжки виявляли шляхом дії різних доз перекису водню (H_2O_2) на клітини BEAS-2B з різними часовими проміжками та створення моделі культури клітин, вражених оксидативним стресом. Експериментальну та контрольну групи порівнювали щодо рівнів експресії генів, яку визначали за допомогою кількісної ПЛР у реальному часі. Модель культури клітин, вражених оксидативним стресом, було підтверджено за допомогою спектрофотометричного вимірювання активності малондиальдегіду і каталази ($p < 0,05$). Рівні експресії генів каспаза-3, каспаза-9, bax і bak суттєво підвищились в експериментальній групі порівняно з контрольною ($p < 0,05$). Не було зафіксовано значних відмінностей між групами щодо каспази-8, Bcl-2 і bik ($p > 0,05$). Рівні експресії генів p53 і p21 виявились значно вищими в експериментальних групах ($p < 0,05$). У клітинах бронхіального епітелію BEAS-2B спостерігався оксидативний стрес, викликаний H_2O_2, що призвів до апоптозу внутрішнім шляхом на рівні експресії генів.

Ключові слова: апоптоз, BEAS-2B, проліферація клітин, оксидативний стрес, активні форми кисню.

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