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Protective Effects of Polypodiside as a Nrf2 Activator on Beas-2B Cells Against the Injury Induced by LPS

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SUMMARY. Acute lung injury (ALI) followed by acute respiratory distress syndrome (ARDS) are the lethal lung diseases. It was found activating Nrf2 could attenuate inflammation, oxidative stress and apoptosis in ALI/ARDS. To discover novel therapeutic for ALI/ARDS, polypodiside was explored using Beas-2B cells injured by LPS. The results showed polypodiside improved the viability of Beas-2B cells treated with LPS and suppressed oxidative stress via reducing ROS and MDA, and elevating SOD, CAT and GPx. Meanwhile, the inflammation resulting from LPS in Beas-2B cells was ameliorated by polypodiside through decreasing the pro-inflammatory cytokines including TNF-α IL-1β and IL-6. It was also found NF-ϰB was inactivated by polypodiside. In addition, polypodiside down-regulated caspase-3 and Bax and up-regulated Bcl-2 to inhibit apoptosis of Beas-2B cells induced by LPS. Then the activation of Nrf2 in Beas-2B cells was associated with the protective effects of polypodiside.

RESUMEN. La lesión pulmonar aguda (ALI) seguida del síndrome de dificultad respiratoria aguda (SDRA) son enfermedades pulmonares letales. Se descubrió que la activación de Nrf2 podría atenuar la inflamación, el estrés oxidativo y la apoptosis en ALI/ARDS. Para descubrir una nueva terapia para ALI/ARDS, se exploró el polipodísido utilizando células Beas-2B lesionadas por LPS. Los resultados mostraron que el polipodisido mejoró la viabilidad de las células Beas-2B tratadas con LPS y suprimió el estrés oxidativo mediante la reducción de ROS y MDA, y el aumento de SOD, CAT y GPx. Mientras tanto, la inflamación resultante del LPS en las células Beas-2B mejoró con el polipodísido al disminuir las citocinas proinflamatorias, incluidas TNF-α, IL-1β e IL-6. También se descubrió que el polipodísido inactivaba el NF-ϰB. Además, el polipodisido disminuyó la caspasa-3 y Bax y aumentó la Bcl-2 para inhibir la apoptosis de las células Beas-2B inducida por LPS. Luego, la activación de Nrf2 en las células Beas-2B se asoció con los efectos protectores del polipodísido.

KEY WORDS: apoptosis, Beas-2B cells, inflammation, Nrf2, oxidative stress, polypodiside*:*

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