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Anisodamine Inhibits Bleomycin-induced Pulmonary Fibrosis through Inhibition against Endoplasmic Reticulum Stress

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SUMMARY. **The objective of this study is to investigate the inhibition effect of anisodamine on idiopathic pulmonary fibrosis (IPF) and endoplasmic reticulum (ER) stress. Fifty healthy male SD rats were randomly divided into 5 groups: the pulmonary fibrosis model group induced by bleomycin (BLM), low concentration of anisodamine group (5 mg/kg), middle concentration of anisodamine group (10 mg/kg), high concentration of anisodamine group (20 mg/kg), and the control group. Histological study was conducted to evaluate the effect of anisodamine on pulmonary fibrosis. Both RT-PCR and Western blot were used to determine the expression of fibrosis related factors α-SMA, collagen I and ER stress markers CHOP and GRP78. Histological study showed that anisodamine could significantly inhibit the fibrosis of lung tissues induced by BLM. When treated with BLM, expression of fibrosis related factors α-SMA, collagen I and ER stress markers CHOP and GRP78 significantly increased compared with the control. However, anisodamine could inhibit expression of all α-SMA, collagen I, CHOP and GRP78 at both mRNA and protein levels under all concentrations, and the effect was in a dose-dependent manner. Anisodamine can inhibit bleomycin-induced pulmonary fibrosis and the effect may be through the inhibition of endoplasmic reticulum stress.**

RESUMEN. El objetivo de este estudio es investigar el efecto inhibidor de la anisodamina sobre la fibrosis pulmonar idiopática (FPI) y el estrés del retículo endoplásmico (ER). Cincuenta ratas SD macho sanas se dividieron aleatoriamente en 5 grupos: el grupo modelo de fibrosis pulmonar inducido por bleomicina (BLM), el grupo de baja concentración de anisodamina (5 mg/kg), el grupo de concentración media de anisodamina (10 mg/kg), el grupo de alta concentración de anisodamina (20 mg/kg) y el grupo control. Se realizó un estudio histológico para evaluar el efecto de anisodamina sobre la fibrosis pulmonar. Tanto RT-PCR como Western blot se usaron para determinar la expresión de factores relacionados con la fibrosis α-SMA, colágeno I y marcadores de estrés CHOP y GRP78. El estudio histológico mostró que la anisodamina podría inhibir significativamente la fibrosis de los tejidos pulmonares inducida por BLM. Cuando se trató con BLM, la expresión de los factores relacionados con la fibrosis α-SMA, los marcadores de estrés con colágeno I y CHOP y GRP78 aumentaron significativamente en comparación con el control. Sin embargo, la anisodamina podría inhibir la expresión de todos los α-SMA, colágeno I, CHOP y GRP78 tanto a nivel de ARNm como de proteína en todas las concentraciones, y el efecto fue dependiente de la dosis. La anisodamina puede inhibir la fibrosis pulmonar inducida por bleomicina y el efecto puede ocurrir por la inhibición del estrés del retículo endoplásmico.

KEY WORDS: anisodamine, endoplasmic reticulum stress, pulmonary fibrosis.

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