

Vicenin-1 Ameliorated House Dust Mite-Induced Allergic Response in Neonatal Mice Via Regulation of the TGF- β /Collagen-1 Pathway

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SUMMARY. Asthma is a chronic disorder that often develops early in life when the respiratory and immune systems are still developing. Several glycosides have been demonstrated to have therapeutic effects on airway hyperresponsiveness (AHR). The aim was to investigate the potential of vicenin-1 in an experimental model of asthma in house dust mite (HDM)-exposed neonatal mice. Allergen challenge was performed in neonatal Balb/c mice via exposure to 10 μ g HDM extract (thrice/week, three weeks) followed by 15 μ g from 3-12 weeks. They were treated with vicenin-1 (5, 10, and 20 mg/kg) for 28 days. Chronic exposure to HDM caused induction of AHR reflected by elevated levels of BALF inflammatory cell count, total protein, albumin, HDM-specific IgE, LDH, and ALP in serum. However, vicenin-1 (10 and 20 mg/kg) treatment ameliorated these HDM-induced elevated levels of markers. Vicenin-1 inhibited elevated oxidative stress (SOD, GSH, MDA, and NO levels) and down-regulated pro-inflammatory cytokines (TNF- α , IL-1 β , and IL-6) mRNA expressions in the lung. HDM-induced up-regulated mRNA expressions of collagen-1 and TGF- β in the lung were also attenuated by vicenin-1. HDM-induced histopathological aberrations in lung tissue were reduced after vicenin-1 treatment. The findings of the present investigation suggested that vicenin-1 exerts its antiasthmatic potential against HDM-induced AHR in neonatal mice via amelioration of elevated levels of HDM-specific IgE, pro-inflammatory cytokines (TNF- α , and ILs), oxidative stress (SOD, GSH, MDA, and NO), fibrotic markers (collagen-1 and TGF- β).

RESUMEN. El asma es un trastorno crónico que a menudo se desarrolla temprano en la vida cuando los sistemas respiratorio e inmunológico aún se están desarrollando. Se ha demostrado que varios glucósidos tienen efectos terapéuticos sobre la hiperreactividad de las vías respiratorias (AHR). El objetivo era investigar el potencial de vicenina-1 en un modelo experimental de asma en ratones recién nacidos expuestos a ácaros del polvo doméstico (HDM). La provocación con alérgenos se realizó en ratones Balb/c recién nacidos a través de la exposición a 10 μ g de extracto de HDM (tres veces por semana, tres semanas) seguido de 15 μ g de 3 a 12 semanas. Fueron tratados con vicenina-1 (5, 10 y 20 mg/kg) durante 28 días. La exposición crónica a HDM causó la inducción de AHR reflejada por niveles elevados de recuento de células inflamatorias BALF, proteína total, albúmina, IgE específica de HDM, LDH y ALP en suero. Sin embargo, el tratamiento con vicenina-1 (10 y 20 mg/kg) mejoró estos niveles elevados de marcadores inducidos por HDM. Vicenin-1 inhibió el estrés oxidativo elevado (niveles de SOD, GSH, MDA y NO) y citocinas proinflamatorias reguladas a la baja (TNF- α , IL-1 β e IL-6) expresiones de ARNm en el pulmón. Vicenina-1 también atenuó las expresiones de ARNm reguladas al alza inducidas por HDM de colágeno-1 y TGF- β en el pulmón. Las aberraciones histopatológicas inducidas por HDM en el tejido pulmonar se redujeron después del tratamiento con vicenina-1. Los hallazgos de la presente investigación sugirieron que la vicenina-1 ejerce su potencial antiastmático contra la AHR inducida por HDM en ratones recién nacidos a través de la mejora de los niveles elevados de IgE específica de HDM, citocinas proinflamatorias (TNF- α e IL), estrés oxidativo (SOD, GSH, MDA y NO), marcadores fibróticos (colágeno-1 y TGF- β).

KEY WORDS: airway hyperresponsiveness, asthma, collagen-1, neonatal, TGF- β , vicenin-1

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