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Autores	Jackeline A. Mendes, Matheus C. Ribeiro, Gustavo J. M. V. Reis Filho, Thalita Rocha, Marcelo N. Muscará, Soraia K. P. Costa, Heloisa H. A. Ferreira
Autor (es) USF	Thalita Rocha
Autores Internacionais	
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Resumo	Studies suggest that hydrogen sulfide (H2S) plays a relevant and beneficial role in the pathophysiology of pulmonary allergic diseases, such as asthma. These diseases may be triggered by changes in airway epithelium caused by repeated exposure to environmental allergens. This study aimed to investigate whether H2S protects against bronchial epithelium apoptosis in allergic inflammation in mice. The effects of H2S on the production of Th2 cytokines and on the infiltration of pulmonary inflammatory cells were also studied. Female BALB/c mice previously sensitized with ovalbumin (OVA) were treated with H2S donor (sodium hydrosulfide [NaHS]) 30 min prior to OVA challenge. After euthanasia (48 h post challenge), the right lung was homogenized to study apoptosis protein expression and to analyze cytokine levels in lung tissue. The left lobe was fixed in formalin for morphological analysis of lung tissue and verification of apoptosis in situ by the TUNEL assay. Histological results showed that NaHS reduced the airway inflammatory infiltrate and prevented an increase in the IL-4, IL-5 and IL-25 levels caused by OVA challenge. Activation of caspase 3 and FasL in response to the allergen was also fully prevented by NaHS treatment. TUNEL staining showed that the challenge from OVA significantly increased the rate of apoptosis in the bronchiolar epithelium, and that this incremental apoptosis was abolished by NaHS treatment. In conclusion, our results showed that H2S donor has a protective effect against airway epithelium damage caused by an allergic reaction, and represents a potential agent in treating allergic lung disorders, such as asthma.
Fomento	