The Distinctive Effects of Acute and Chronic Psychological Stress on Airway Inflammation in a Murine Model of Allergic Asthma

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Background: Psychological stress has long been recognized to be associated with asthma symptoms. There appear to be individual differences in the susceptibility to even the same kind of stress, and furthermore, stress responses are different between the types of the stress, acute and chronic, even in the same person. However, the mechanisms linking stress to asthma are not well defined. Psychological stress upregulates the expression of endogenous opioids. The opioids stimulate the hypothalamus-pituitary-adrenal axis and sympathetic and adrenomedullary system, through the activation of μ-opioid receptor (MOR) to release stress hormones, such as cortisol and catecholamines, respectively. These hormones can modulate immune responses via the induction of Th1 immunity.

Methods: Female BALB/c and C57BL/6, wild and MOR-deficient, mice sensitized with ovalbumin (OVA) were exposed to OVA with or without either acute or chronic restraint stress. Airway inflammation was evaluated by the measurement of the number of inflammatory cells and cytokine contents in bronchoalveolar lavage fluids.

Results: In BALB/c mice, but not in C57BL/6 mice, the number of total cells, eosinophils and lymphocytes in the acute stress group were significantly decreased compared with those in the non-acute stress group. In contrast, chronic stress significantly increased the cell numbers and the contents of IL-4 and IL-5 in both mouse strains. Furthermore, these exacerbations were abolished in MOR-deficient mice.

Conclusions: These results suggest that acute stress modifies the allergic airway responses distinctively depending on the genetic background, and MOR is involved in the chronic psychological stress-induced exacerbation of allergic airway inflammation.

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