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Body: The inflammatory phenotype of acute asthma exacerbations has been reported to be characterized by a heterogeneous infiltrate with eosinophils and large numbers of neutrophils in sputum as well as broncho-alveolar lavage (BAL). Epidemiological surveys suggested viral infection of the respiratory tract as the main trigger. Double stranded RNA is produced as an intermediate during replication of respiratory viruses and can be sensed by toll-like receptor 3 (TLR3). TLR3 could therefore play a key role in the pathogenesis of acute asthma exacerbation. Our study aimed at i) establishing a mouse model of TLR3 trigged asthma exacerbation and ii) clarifying the underlying mechanisms. Intra-nasal (i.n.) application of the TLR3 ligand poly (I:C) induced an exacerbation of experimental allergic asthma in mice characterized by enhanced release of pro-inflammatory cytokines, mucus production, and pronounced airway hyperresponsiveness (AHR). Exacerbation included marked infiltration of neutrophils and T helper 17 (TH17) cells. . None of these characteristics could be observed in mice lacking IL-17A, indicating an essential role of this cytokine in mediating TLR3 triggered asthma exacerbation. Furthermore, in IL-23p19 mice, which lack IL-17 producing γδ T cells and TH17 cells, local application of poly (I:C) again provoked asthma exacerbation. Therefore, we suggest IL-17 producing NKT cells as main cell type involved in TLR3 triggered exacerbation of allergic asthma in mice.