**Supplementary information**

***IL-1β derived Th17 immune responses are a critical factor for neutrophilic-eosinophilic airway inflammation on psychological stress-induced immune tolerance breakdown in mice***

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**Supplementary Material and Methods**

*Mice and Protocols for Tolerization, Sensitization, Antigen Challenge, and Stress Exposure*

We used female BALB/c IL-17A knockout (KO) mice (generated by Yoichiro Iwakura) [30] for the asthma model as described in the Material and Methods section of the manuscript. Mice were housed under a 12-h light/dark cycle at a constant temperature of 23±1℃. Sterilized food and water were provided ad libitum.

**Supplementary Figure legends**

Fig. S1. IL-17A KO mice (the BALB/c background) showed decreased neutrophilic inflammation but not eosinophilic inflammation. BAL was collected on day 5 after OVA inhalation from the mice in the four groups. The cells in BAL fluid were stained with a Diff-Quick solution, and cell composition was determined via light microscopy. (A) Schematic representation of the protocols used for tolerization, stress exposure, sensitization, and antigen challenge. (B) Cell composition in BAL fluid of mice.

**Reference**

30 Nakae S, Komiyama Y, Nambu A, Sudo K, Iwase M, Homma I, et al. Antigen-specific T cell sensitization is impaired in IL-17-deficient mice, causing suppression of allergic cellular and humoral responses. Immunity. 2002 Sep;17(3):375–87.

