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Acidic Mammalian Chitinase (AMCase) Prevents Chitin-induced Eosinophil Recruitment to the Lung

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Chitin stabilizes the exoskeleton of arthropods and crustaceans and is a component of fungal cell walls. Mammals cannot synthesize chitin, however they express chitinases and chitinase-like proteins. Most of these proteins have lost their enzymatic activity against chitin and their functions are largely unknown, however some have been implicated in eosinophil chemoattraction and tissue remodeling. We identified two chitinase-like proteins, Ym2 and AMCase in the mouse lung as being expressed in a Stat6-dependent manner after infection with the helminth parasite *Nippostrongylus brasiliensis*. Transgenic mice overexpressing Ym2 and AMCase in the lung did not show any signs of increased inflammation or tissue damage in the lung, either spontaneously or following infection with *N. brasiliensis*. Furthermore, administration of antibodies specific for Ym2 and AMCase did not prevent lung inflammation in a murine asthma model or in the *N. brasiliensis* model. However, eosinophil recruitment to the lung induced by intranasal chitin administration was significantly reduced in AMCase transgenic mice or mice treated with AMCase-coated chitin beads, in contrast to Ym2 transgenic mice or Ym2-coated chitin beads. Therefore, AMCase functions as a negative regulator of chitin induced lung eosinophilia.