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Eosinophils are effector cells that have an important role in the pathogenesis of allergic disease. Defective removal of these cells likely leads to chronic inflammatory diseases such as asthma. Thus, there is great interest in understanding the mechanisms responsible for the elimination of eosinophils from inflammatory sites. Previous studies have demonstrated a role for certain mediators and molecular pathways responsible for the survival and death of leukocytes at sites of inflammation. Reactive oxygen species have been described as proinflammatory mediators but their role in the resolution phase of inflammation is poorly understood. The aim of this study was to investigate the effect of reactive oxygen species in the resolution of allergic inflammatory responses. An eosinophilic cell line (Eol-1) was treated with hydrogen peroxide and apoptosis was measured. Allergic inflammation was induced in ovalbumin sensitized and challenged mouse models and reactive oxygen species were administered at the peak of inflammatory cell infiltrate. Inflammatory cell numbers, cytokine and chemokine levels, mucus production, inflammatory cell apoptosis and peribronchiolar matrix deposition was quantified in the lungs. Resistance and elastance were measured at baseline and after aerosolized methacholine. Hydrogen peroxide accelerates resolution of airway inflammation by induction of caspase-dependent apoptosis of eosinophils and decrease remodeling, mucus deposition, inflammatory cytokine production and airway hyperreactivity. Moreover, the inhibition of reactive oxygen species production by apocynin or in $gp91^{phox-/-}$ mice prolonged the inflammatory response. Hydrogen peroxide induces Eol-1 apoptosis *in vitro* and enhances the resolution of inflammation and improves lung function *in vivo* by inducing caspase-dependent apoptosis of eosinophils.