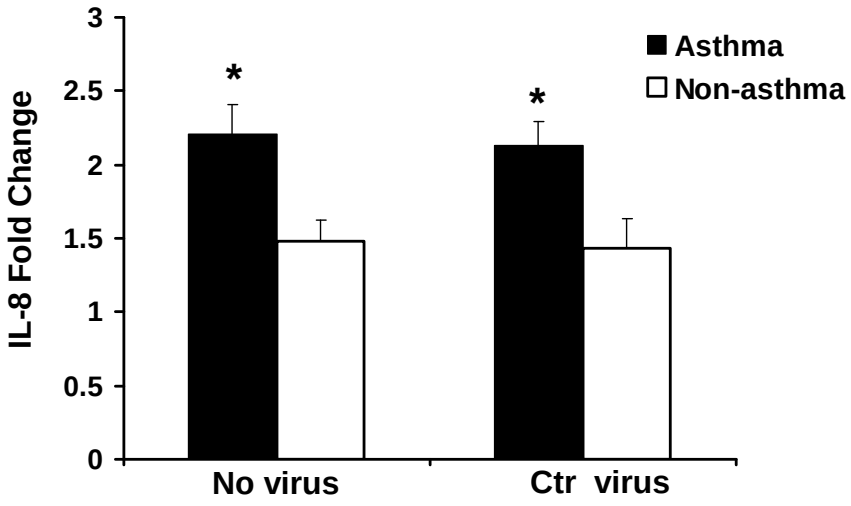
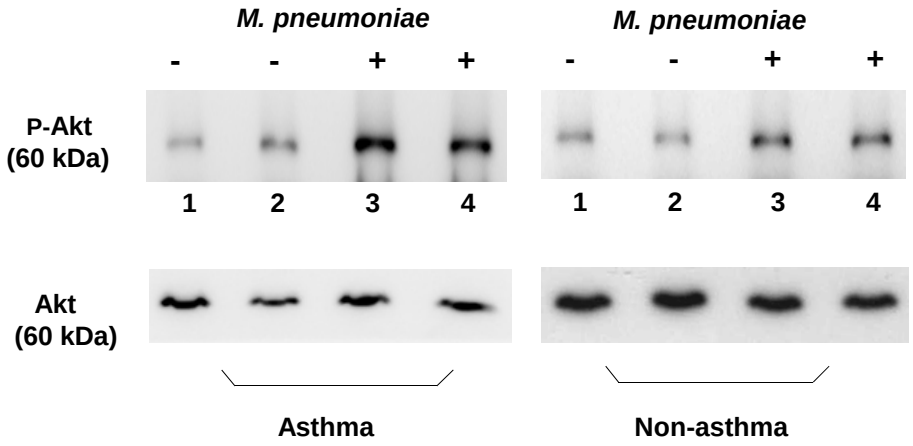
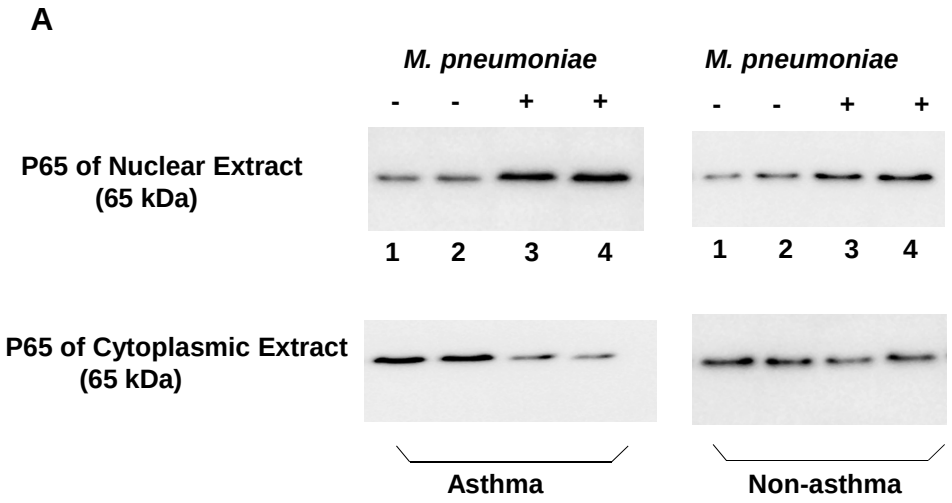
Supplemental Figure 1:

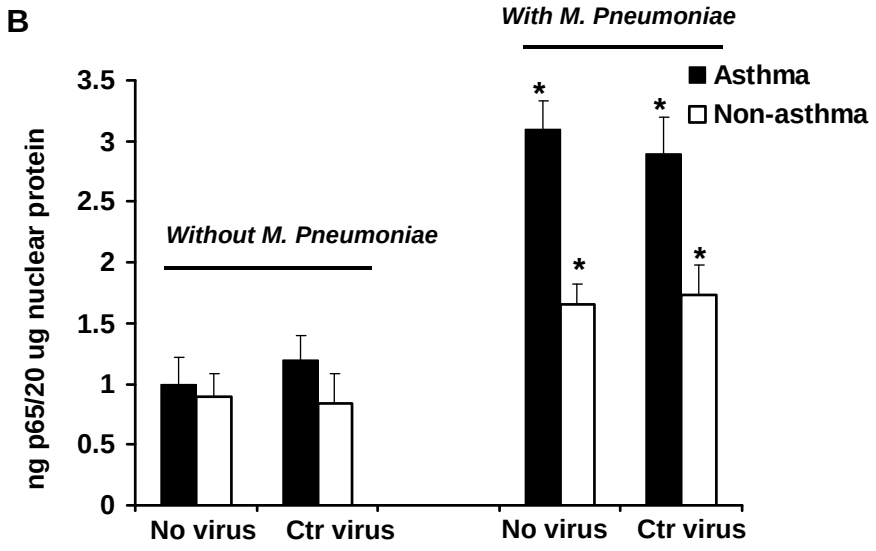


Supplemental Figure 2:

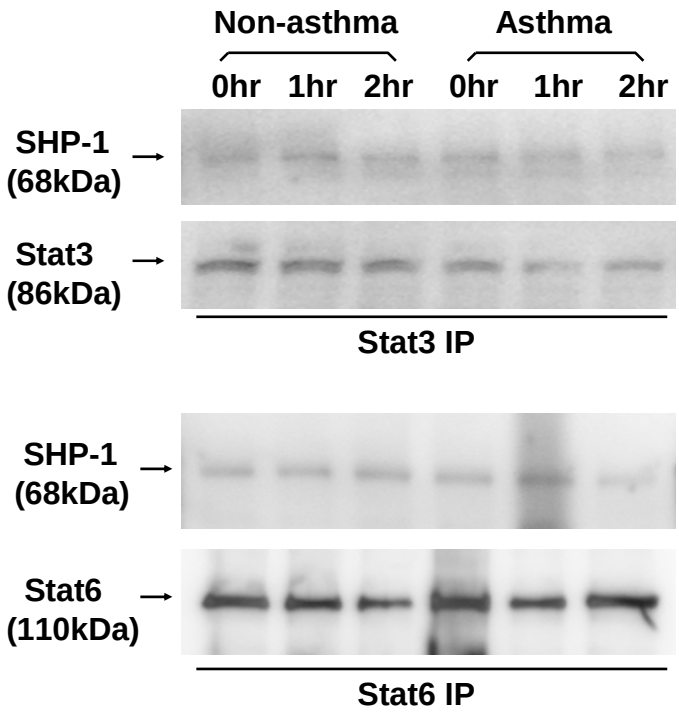


Supplemental Figure 3:





Supplemental Figure 4:



Supplemental Figure Legends

Figure S1. *M. pneumoniae*-induced IL-8 production in non-asthmatic and asthmatic airway epithelial cells with or without transduction of control adeno-associated virus. To test whether rAAV alone caused non-specific effects on IL-8 levels in epithelial cells, apical supernatant collected 48 hr after *M. pneumoniae* infection from cells with or without control rAAV treatment was used to perform Il-8 Elisa. "No virus": airway epithelial cells without control rAAV transduction were treated with *M. pneumoniae* for 48 hours; "Ctr virus": control rAAV transduced airway epithelial cells were treated with *M. pneumoniae* for 48 hours. * $p<0.01$ compared with non-asthmatic cells. Data representing the fold change of IL-8 from cells without mycoplasma infection are presented as means ± SEM (n=10 for non-asthma; n= 12 for asthma). rAAV alone did not cause significant effect on IL-8 levels in both non-asthmatic and asthmatic airway epithelial cells.

Figure S2: *M. pneumoniae*-induced Akt phosphorylation in total cell lysates of non-asthmatic and asthmatic cells with or without control adeno-associated virus transduction. Immunoblot analysis of phosphorylated Akt (P-Akt) levels in airway epithelial cells after 2 hr treatment with *M. pneumoniae*. Lane 1, 3: airway epithelial cells without control rAAV transduction; Lane 2, 4: airway epithelial cells transduced with control rAAV. rAAV alone did not cause non-specific effects on the levels of Akt phosphorylation in cells with or without *M. pneumoniae* infection.

Figure S3: *M. pneumoniae*-induced NF-κB activation in non-asthmatic and asthmatic airway epithelial cells with or without control adeno-associated virus transduction. (A) Immunoblot of cytoplasmic and nuclear protein lysates against p65 in non-asthmatic and asthmatic airway epithelial cells without mycoplasma infection or challenged with mycoplasma for 2 hr. Lane 1, 3: airway epithelial cells without control rAAV infection; Lane 2, 4: airway epithelial cells infected with control rAAV. (B) NF-κB activation was quantified in non-asthmatic and asthmatic airway epithelial cells without mycoplasma infection or 2 hours after mycoplasma infection. The NF-κB DNA binding activity is reported as ng of bound p65 protein per 20 μg of nuclear extracts. "No virus": airway epithelial cells without control rAAV transduction; "Ctr virus": control rAAV transduced airway epithelial cells. * $p<0.01$ compared with cells without mycoplasma infection in the same group. Data are presented as means ± SEM values (n=10 for non-asthma; n= 12 for asthma). rAAV alone did not cause non-specific effects on the activity of NF-κB.

Figure S4: *Mycoplasma pneumoniae* infection did not induce dynamic association of Stat3/SHP-1 and Stat6/SHP-1 in airway epithelial cells from non-asthmatic and asthmatic subjects. Following a time-course treatment employing *M. pneumoniae* infection, epithelial cells lysates (500 μg) were immunoprecipitated with 2μg of rabbit anti-human Stat3 or Stat6 antibody (Cell Signaling Technology, Danvers, MA). The immunoprecipitates were analyzed by SDS-PAGE and immunoblotting using mouse anti-human SHP-1 antibody (Santa Cruz Biotechnology, Santa Cruz, CA) to determine the total amount of SHP-1 co-immunoprecipitated with Stat3 or Stat6 in the samples. The

PVDF membrane was then stripped and re-probed with mouse anti-human Stat3 or Stat6 antibody to determine the amounts of Stat3 or Stat6 in the immunoprecipitates and quantified by densitometry. Shown are representative data from five asthmatic and five non-asthmatic subjects. There was no significant difference of the amounts of SHP-1 associated with Stat3 or Stat6 between non-asthmatic and asthmatic airway epithelial cells, before and after the treatment with mycoplasma.