Letter to the Editor

Susceptibility to influenza virus infection of bronchial biopsies in asthma

To the Editor:

Influenza causes significant morbidity and mortality, especially in patients with chronic lung diseases.[1] Infection results in inflammatory cell influx and leads to either resolution or increased lung immunopathology and resulting morbidity,[2] especially in patients with chronic airways diseases where viruses exacerbate inflammation and, subsequently, symptoms. Those with asthma are more susceptible to influenza and are, therefore, the most common population hospitalized, although, interestingly, they are less likely to develop severe disease or

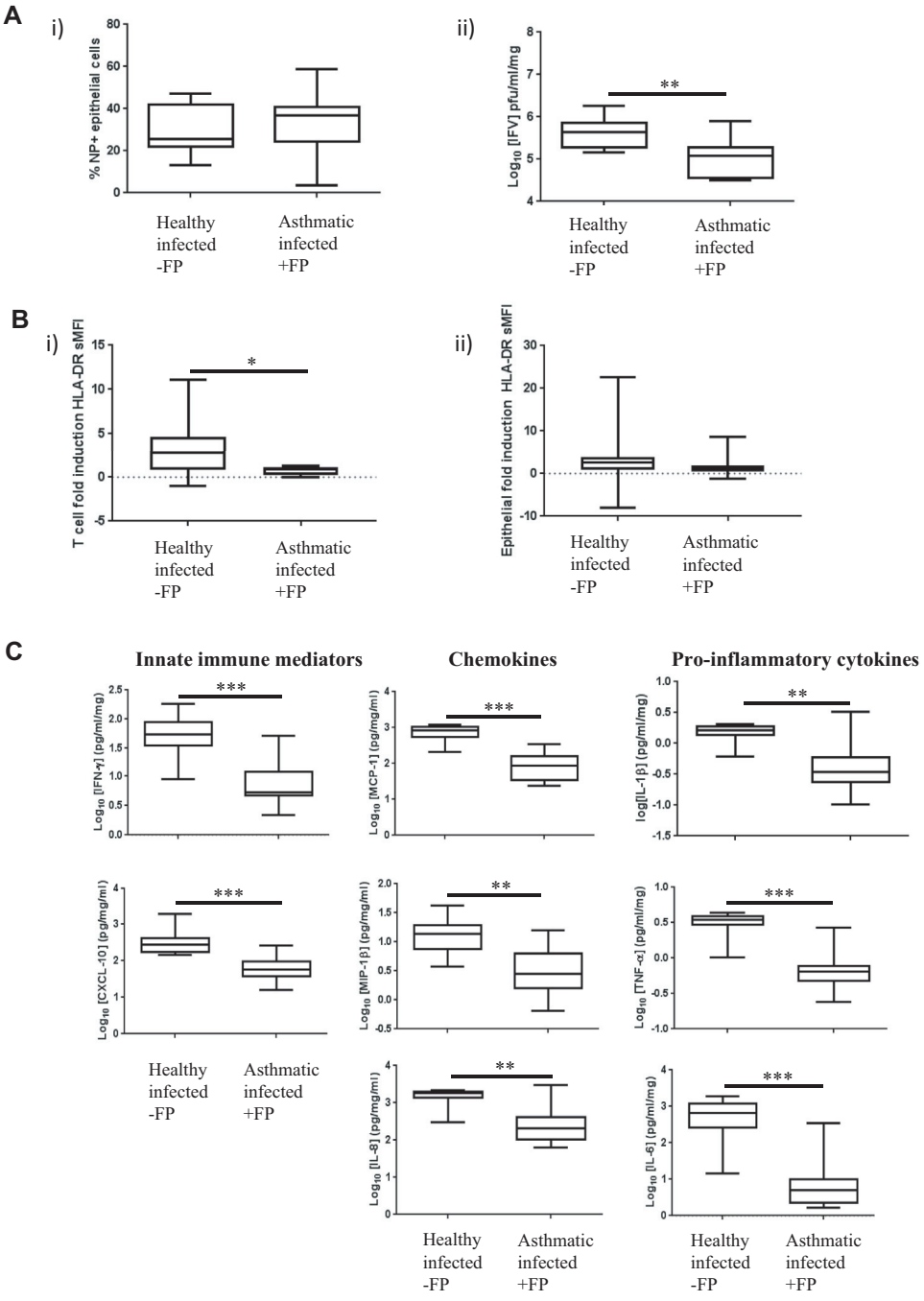

FIG 1. Comparison of bronchial biopsies from healthy subjects and subjects with asthma following influenza virus exposure. **A,** Effects on (i) epithelial cell infection and (ii) viral shedding. **B,** Fold change in MHC class II cell surface expression on (i) T lymphocytes and (ii) epithelial cells. **C,** Mediator secretion from infected biopsies. N = 10 per group compared using Mann-Whitney test. *MCP*, Monocyte chemoattractant protein; *MIP*, macrophage inflammatory protein; *NP*, Influenza A virus nucleoprotein. **P* < .05, ***P* < .005, and ****P* < .001.

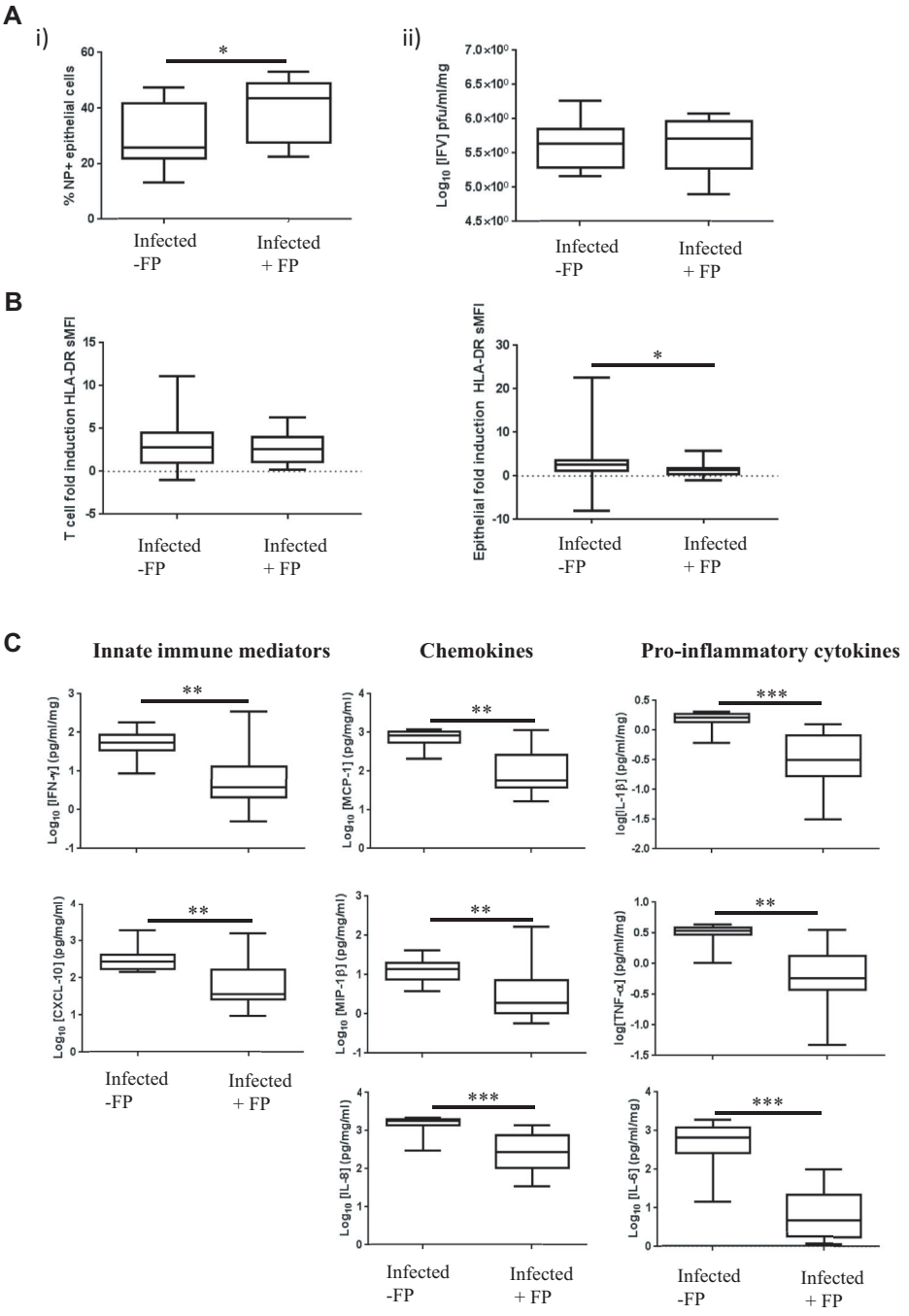

FIG 2. A, Effect of addition of exogenous corticosteroid to bronchial explants from healthy subjects on (i) epithelial cell infection and (ii) viral shedding. **B,** Fold change in MHC class II cell surface expression following infection on the cell surface of (i) T lymphocytes and (ii) epithelial cells. **C,** Mediator secretion from infected biopsies. N = 10 per group. Data were compared using Wilcoxon matched pairs signed rank test. *MCP*, Monocyte chemoattractant protein; *MIP*, macrophage inflammatory protein; *NP*, Influenza A virus nucleoprotein. **P* < .05, ***P* < .005, and ****P* < .001.

die than those without asthma.[3] The mechanisms underlying the increased susceptibility to viral infections in those with asthma are poorly understood, but it has been suggested that the skewing toward T_H2 immunity results in deficient T_H1 antiviral immunity.[4] Understanding of antiviral immunity in asthma is also complicated by the immunosuppressive effects of inhaled corticosteroids (ICSs) or oral corticosteroids, standard treatments in asthma.[5] The effectiveness of ICSs during exacerbations is unclear because doubling their dose at the time of upper respiratory tract infection fails to prevent asthma exacerbations.[6] Corticosteroids may protect against severe outcomes in those with asthma with influenza infection, whereas systemic corticosteroids in individuals without asthma cause delayed viral clearance.[7]