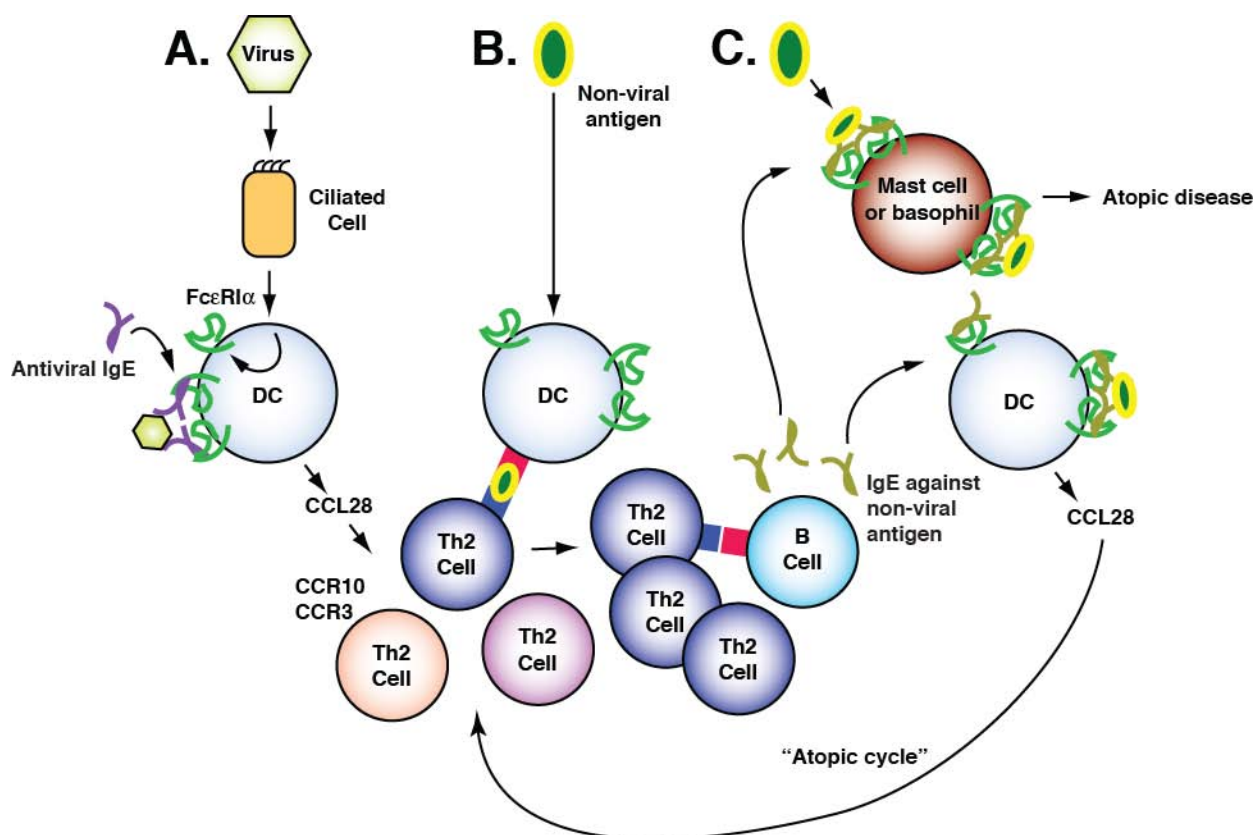


**Online supplemental information****Development of atopy: severe paramyxoviral infection is sufficient to induce atopic disease against non-viral antigens in a mouse model**

Dorothy S. Cheung, MD, Sarah J. Ehlenbach, BS, Tom Kitchens, BS, Desire A. Riley, BS, and Mitchell H. Grayson, MD

**eFigure 1. Proposed model of development of atopy.**

**eFigure 1. Proposed model of development of atopy.** (A) Viral respiratory infection drives expression of FcεRI on lung cDC, and IgE against virus leads to cross-linking of FcεRI on the cDC. This leads to release of chemoattractant (CCL28) for the recruitment of Th2 cells in an antigen non-specific fashion. (B) Exposure to a non-viral antigen could lead to expansion of Th2 cells, which would instruct B cells to make non-viral antigen specific IgE. (C) Subsequent exposure to the antigen would lead to clinical relevant atopic disease. If IgE against the non-viral antigen is cross-linked on the cDC in this second exposure, then further antigen non-specific recruitment of Th2 cells could occur, leading to an “atopic cycle”. DC refers to conventional dendritic cells.



eFigure 1.