



MEETING ABSTRACT

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# B cell Semaphorin 4c expression mitigates the airway hyperresponsiveness and acute inflammation which characterize allergic airway disease

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## Background

Semaphorin signaling proteins, initially examined in the context of neuronal axon development, have recently been implicated as regulators of immune cell migration. Our laboratory has determined that expression of Semaphorin 4C (Sema4C) is strongly induced on B cells exposed to Th2 stimulation, and we seek to elucidate its mechanism of controlling allergic airway disease.

## Methods

Wild-type and Sema4C<sup>-/-</sup> mice were sensitized intraperitoneally using 100 µL OVA (0.5 mg/mL ovalbumin and 4 mg/mL aluminum hydroxide in PBS) on days 0 and 14, and were challenged intranasally using 20 µL OVA (10 mg/mL ovalbumin in PBS) from days 28 to 30. Sacrifice and analysis of Airway Hyperresponsiveness via flexiVent was performed on day 31. Serum IgE and IL-10 expression levels were measured by ELISA. B cells were phenotyped by fluorescence-activated cell sorting (FACS). B cell motility was measured by migration assays.

## Results

Please see figure 1.

## Conclusions

Semaphorin 4C regulates the allergic airway disease through immune synapse-governed cytoskeletal rearrangements in B cells, and minimizes the inflammatory cellular lung infiltration that contributes to airway hyperresponsiveness.

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