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Title: LSC 2013 abstract - The role of mast cells, IL-13 and TRP channels in a mouse model of chemical-induced airway hyperresponsiveness

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Body: Occupational asthma is the most common work-related lung disease in industrialized countries. The mechanisms of occupational asthma caused by chemicals are still not completely understood. Therefore, we used a mouse model of chemical-induced asthma to examine the role of the neurogenic system as well as the role of IL-13 and mast cells by using different knock-out mice. On days 1 and 8, wild type C57Bl/6 mice, IL-13, Transient Receptor Potential (TRP)V1, TRPA1 and mast cell deficient mice were dermally sensitized with 1% toluene-2.4-diisocyanate (TDI) or vehicle (acetone/olive oil) on both ears. On day 15, the mice received a single intranasal challenge with 0.1% TDI or vehicle. In a second experiment TDI or vehicle sensitized wild type C57Bl/6 mice received an intraperitoneal injection of the NK1R antagonist RP67580 (1µg/µl) prior to the challenge. Airway reactivity to methacholine, lung inflammation, lymphocyte subpopulations in the draining auricular lymph nodes and total serum IgE were assessed 24h after the challenge. IL-13, TRPV1, TRPA1 and mast cell deficient mice showed a significant lower airway hyperreactivity compared to wild type mice, 24h after TDI challenge, without any sign of lung inflammation. Treatment with the NK1R antagonist also resulted in a significant decrease in airway hyperreactivity. In the auricular lymph nodes T-helper cells, T-cytotoxic cells and B-cells were significantly lower in mast cell deficient and IL-13 deficient mice, compared to wild type mice. These results indicate the importance of IL-13, TRPA1 and TRPV1 channels and mast cells in the development of immune-mediated bronchial hyperreactivity.