## 961 Recovery of Peanut Allergens From Ventilation Filters of Commercial Airliners. RT Jones BS, DF Stark MD, GL Sussman MD, JW Yunginger MD, Rochester MN, Vancouver BC, and Toronto, ONT

To help address whether peanut-sensitive travelers are exposed to peanut aeroallergens during airline flights on which peanuts are served, we obtained filter units (23" x 23" x 6") from the ventilation systems of two commercial airliners at the time of their annual replacement after approximately 5000 flight hours. The filter papers were torn into pieces, extracted 1:10 w:v in 1.0M NaCl-20mM phosphate buffer (pH 7.4) overnight at 4°C, dialyzed against 100mM NH<sub>4</sub>HCO<sub>3</sub>, and lyophilized. A new, non-exposed filter paper was extracted in the same fashion as a negative control. Non-dialyzable solids from each of the three extracts were dissolved in phosphate-buffered saline (100 mg/ml) for testing.

In an inhibition immunoassay for peanut allergens employing a crude peanut standard extract (arbitrary potency = 100,000 AU/ml), both airliner filter extracts contained measurable quantites of peanut allergens (317 AU/ml) and 611 AU/ml), but not the unexposed filter. Two-fold dilutions of the peanut standard extract and the three filter extracts were used to prepare dot immunoblots, which were probed with the same peanut IgE antibody pool. Peanut allergens were again identified in each exposed filter extract, but not in the unexposed filter extract. Control peanut dot blots probed with serum from a non-allergic person were negative, and no filter extract showed activity in two unrelated allergen blots (natural rubber latex and honey bee venom).

We conclude that peanut allergens can be eluted from ventilation system filters in commercial airliners. The most likely source of these allergens are the peanuts served during flights.

## 963 In Vivo Expression of Interleukin-12 and Interleukin-13 in Atopic Dermatitis. T. Naseer, QA Hamid, EM Minshall, YL Song, M Boguniewicz, DYM Leung. Montreal, Canada and Denver, CO, USA.

Previous studies in atopic dermatitis (AD) have shown that acute and chronic skin lesions are associated with a Th2-type profile of cytokine expression. Interleukin 12 (IL-12) and IL-13 are recently described cytokines which possess Th1 and Th2-like actions respectively. We have used the technique of in situ hybridization to examine the expression of IL-12 and IL-13 messenger RNA (mRNA) in skin biopsies from acute and chronic skin lesions and uninvolved skin from patients with AD. When compared with normal control skin, the acute and chronic skin lesions and unaffected skin from AD patients had significantly greater numbers of cells which were positive for IL-13 mRNA (p < 0.05). Acute AD skin lesions expressed a higher number of positive cells than those observed in chronic AD skin lesions (p < 0.05) or psoriasis skin lesions (p < 0.05). There was a significant increase in the numbers of IL-12 mRNA positive cells in chronic skin lesions compared to uninvolved skin from AD patients (p < 0.05). These data demonstrate that acute AD skin lesions are associated with an increased expression of IL-13 mRNA. In contrast, the relative increase in IL-12 mRNA in chronic AD skin lesions suggests a possible role for IL-12 producing cells in modulating chronic inflammation.

## Ten Minutes in a Clothes Dryer Kills All Mites in Blankets JD Miller MD, A Miller BA Ridgefield CT

Hot water washing of blankets at 130°C has been shown to kill all dust mites in the blanket, while cold water washing allows 10% of the mites to survive. Because high water temperatures may be unavailable, can be dangerous to children, and are not suitable for washing all soft items, we studied the effect of dry heat alone on mites in blankets.

Twin size Vellux<sup>tm</sup> and Snuggable<sup>tm</sup> brand blankets were inoculated with dense cultures of *D. pteronyssimus* mites spread over the center 40x40cm<sup>2</sup>. Six 5x5cm sections were cut from the blanket as pre-heat controls and again after the blanket was in a home clothes dryer at "medium" setting for 10, 20 and 30 minutes (Whirlpool model LE7680XS). A probe revealed peak temperatures of 105°C during drying. All sections were then studied with the heat escape method, and the number of surviving mites was counted.

In three experiments, there were >750 mites/10cm<sup>2</sup> in each of the sections cut before drying, but no surviving mites in sections taken after the blanket was heated for 10 minutes or more. This was also the case in a fourth experiment, where the inoculated blanket had been hung in 98% relative humidity for one hour prior to drying.

The use of dry heat in a clothes dryer for 10 minutes killed all mites in these blankets.

## 964 Dichotomy of blood and skin derived interleukin-4 producing nickel specific T-cells and restricted Vβ repertoire in allergic contact dermatitis. T Werfel, M Hentschel, A Kapp, H Renz, Hannover and Berlin, Germany

In this study we compared phenotype and cytokine pattern of nickel specific T cell clones (TCC) derived from blood and positive patch test reactions. A total of 269 TCC that reacted in a specific manner with nickel sulfate were prepared from three nonatopic patients with allergic contact dermatitis caused by nickel. All TCC expressed TCR2 ( $\alpha/\beta$ ) and 77% were CD4+ compared to 21% CD8+ TCC. The majority of nickel specific CD4+ or CD8+ TCC derived from peripheral blood displayed a type 1 cytokine pattern but surprisingly, the majority of skin-derived TCC produced IL-4 either in combination with IFN- $\gamma$  (type 0 cytokine pattern) or exclusively (type 2 pattern). Analysis of TCR-VB repertoire indicated that more than 40% of the tested TCC expressed one of the following  $V\beta$  elements: Vβ13.1/13.2, Vβ20, Vβ2, Vβ6.7 or Vβ14. Compared to unstimulated T cells from peripheral blood of the same individual, nickel-reactive TCC expressed an increased frequency of V\u03b36.7, V\u03b313, V\u03b314 and Vβ20 which suggests a selection of certain TCR Vβ elements by nickelsulfate. In contrast to the compartmentalization of IL-4 production there were no major differences in the expression of TCR-Vβ elements between blood and skin derived nickel specific TCC. These results point to a possible modulation of the cytokine production pattern of T lymphocytes after their migration from peripheral blood into the skin in allergic contact dermatitis.