BRONCHIAL REACTIONS TO EXPOSURE TO WELDING FUMES. Contreras GR, Chan-Yeung M. UBC Department of Medicine, Vancouver, Canada.

Occupational asthma has been reported among welders exposed to stainless steel (SS) and galvanized (G) fumes but not to mild steel (MS) fumes. The mechanism of asthma induced by welding is not clear. This study examined the airway response to welding fumes in six welders with respiratory symptoms. Nonspecific airway hyperresponsiveness (NSAR) was measured using methacholine challenge test. Exposure to welding fumes that provoked their symptoms was done in a chamber. Spirometry was monitored before and at intervals during and after exposure. Exposure to MS fumes was carried out as a control test on separate day. A fall in FEV_1 >15% within the 24 hr after exposure was considered a positive test. The concentration of welding fumes during the exposure test were measured. RESULTS:

	Exposure min		Welding fumes mg/m3		FEV_1 fall %		PC_{20} mg/ml
	MS	SS	MS	SS	MS	SS	
Sub 1	60	60	3.37	7.75	23	18	<0.125
Sub 2	60	60	3.89	2.96	7	10	3.0
Sub 3	60	60	2.24	5.19	3	9	4.54
Sub 4	60	60	---	7.80	3	6	>64.0
	MS	G	MS	G	MS	G	
Sub 5	60	45	1.33	150	2	17	0.95
Sub 6	60	15	12.6	43.9	16	20	0.5

Subjects 2,3,4 did not respond to MS or MS welding exposure. Subjects 1,5,6 had a marked degree of NSAR and an immediate reaction to welding. Subject 1 had positive reaction to MS and SS welding, subject 5 to G welding and subject 6 to both MS and G welding. G welding generated very high concentration of fumes. The findings suggest that the airways response to welding in these subjects is an irritant one. Monitoring the level of exposure during challenge test is important in determining the mechanism of airway response.

ALLERGIC RHINITIS AND OCCUPATIONAL ASTHMA CAUSED BY EXPOSURE TO CHORISTONEURA FUMIFERANA (SPRUCE BUDWORM) IN AN ELECTRIC POWER PLANT. Côté J, Hébert J. Laval Hospital and CHUL, Ste-Foy, Canada.

Background: Occupational asthma can sometimes be caused by exposure to insects. We conducted a descriptive study among the 29 workers of an electric power plant who complained of ocular, nasal and respiratory symptoms when exposed to a moth named Choristoneura fumiferana (CF) during summer time. These flies were attracted in the power plant by the numerous electric lights. Because of their short life span, the flies died inside the plant; workers got exposed to the remnant bodies during 3 or 4 months each summer. Methods: A questionnaire was administered by a physician to the 29 workers; to determine the presence of symptoms of conjunctivitis, rhinitis and asthma and their relationship to CF exposure. Skin prick tests were done with 26 common airborne allergens and 2 different extracts of CF. The 29 workers were asked to fill a diary card for daily symptoms of rhinoconjunctivitis and asthma and morning and evening PEFR during a 8 week period, starting on the third week of June. A methacholine challenge test was done at the end of this period. Bronchial specific challenge test with the CF extract was offered to those workers having asthma symptoms and/or PEFR decrease when exposed to CF. Results: According to the questionnaire 14/29 workers had allergic symptoms when exposed to CF (group A), 11 had allergic rhinitis with or without conjunctivitis, 1 had isolated allergic conjunctivitis and 2 complained of both nasal and chest symptoms. Group B included 15 workers who remained free of symptoms during summer. In group A, 12 out of 14 had a positive skin test to the CF extract while in group B only 3 out of 15 had a positive skin test ($p = < 0.001$). None of the 12 office workers of this company who lived in the same area tested positive with the CF extract. Four workers had symptoms and/or PEFR decrease which suggested occupational asthma. Two of them volunteered for the specific bronchial challenge test with the CF extract. On the first day, these 2 workers were blindly challenged with placebo (saline extract) and did not have any decrease in FEV_1. On the second day, they were challenged with the CF extract and both showed an immediate asthmatic response with an FEV_1 decrease of 20 and 34%. One control subject with a similar degree of bronchial responsiveness inhaled the CF extract and no asthmatic reaction was observed. CONCLUSION: Seasonal exposure to Choristoneura fumiferana (spruce budworm) in men can cause occupational rhinoconjunctivitis and asthma.

BAKER'S ASTHMA: SENSITISATION TO WHEAT FLOUR AND α-AMYLASE AND RELATION TO ALLERGEN EXPOSURE

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Since 1990 a cohort study among bakery workers is in progress. The primary aim of this project is to study the incidence of baker's asthma and to explore exposure-incidence relationships between allergen exposure and asthma. Five-hundred bakery workers of several bread-factories and small bakeries participated in a health survey (health questionnaire, spirometry, repeated peakflow measurements, skin prick tests and specific IgE), and will be followed the next three years. Personal 6-8 hour exposure levels to inhalable flour dust were monitored in all bakeries on more than 500 occasions with the Dutch PAS-6 sampling head. In all samples wheat-allergen levels were analyzed (EIA-inhibition) using human IgG_4-antibodies. The development of an immuno-assay to measure α-amylase-allergens (α-amylase is used as a dough improver) is in progress and results will be available soon. In this abstract, results of a pilot study are presented. In this pilot study special attention is given to the assessment of allergen exposure.

In one of the large bakeries (156 workers) 9% reported work-related allergic symptoms (rhinitis, chest tightness) related to wheat-flour exposure. Skin prick tests were performed with common and occupational allergens. In this bakery 45% had a positive skin prick test for one of the common allergens and 15% were positive for one or more occupational allergens. Sensitization rates to these allergens varied from 1 % for baker's yeast, 5% for wheat- and rye flour and 11% for α-amylase. Most of the positive skin prick tests were found in a group of 30 bakers who worked in the dough production section, where flour improvers are added. In this group 40% had a positive skin prick test for one of the occupational allergens. Most of the work related symptoms were found in this group. The important role of α-amylase in baker's allergy was confirmed by specific IgE-tests and skin prick tests in other bakeries.

Comparisons of exposure levels to flour dust and wheat antigens were made for all occupational titles. Considerable difference in the relation between dust- and wheat antigen exposure were found for several groups, depending on the variety of products that were handled. For some occupational titles considerable differences in antigen exposure existed, where no difference in dust exposure levels could be found, and vice versa. ANOVA revealed that between group, within group and within individual variance components differed. This suggests that exposure estimates based on flour dust levels alone will lead to serious exposure misclassification if occupational wheat allergies are studied. For α-amylase allergen exposure, even larger differences in variance components are expected, because high exposure to this enzyme is limited to a few jobs.

In conclusion, wheat flour and α-amylase are the main allergens involved in baker's asthma. To study exposure-incidence relationships in bakery workers it is necessary to measure both allergens in personal dust samples.

MACROPHAGE STIMULATION WITH FREE RADICAL RELEASE IN THE EARLY STAGE OF LUNG INJURY DUE TO ACUTE TOLUENE DIISOCYANATE EXPOSURE. *Y Meisheri, HV Dedhia, DE Banks, V Castranova, F Masri, JYC Ma, WH Pailes, H Doshi, R. Burrell, M Billie, V Vallyathan. Departments of Medicine, Chemistry, Anesthesiology, Microbiology and Pathology, West Virginia University, and Division of Respiratory Disease Studies, NIOSH, Morgantown, WV, USA.*

The role of pulmonary macrophages (AM) and free radical (FR) formation in acute toluene diisocyanate exposure (TDI) inhalation injury is unknown. The goal of our study was to characterize the inflammatory events leading to acute lung injury after a single sublethal, irritant dose of TDI. 21 rats were exposed to 1.7 to 2 ppm of TDI for 4 hours in an inhalation chamber. 10 rats served as a control on room air (Gr I). TDI exposed rats were sacrificed at 1 (Gr II, n=5), 20 (Gr III, n=5) and 48 hours (Gr IV, n=11) after exposure. Bronchoalveolar lavage (BAL) was analysed for total and differential cell count, cell viability, and lipid and protein content. AM function was assessed by chemiluminescence (CL) at rest and with zymosan stimulation (ZCL) in the absence or presence of the nitric oxide synthase inhibitor L-NAME. L-NAME dependent CL reflects nitric oxide synthase dependent activity (NOSCL). O_2 based FR were measured by electron spin resonance technique using a PBN trap. Lungs were examined microscopically for quantification of severity of injury. No statistically significant differences in total cell counts, differential counts, viability, protein and lipid measurements, or lung histology were recognized among the 4 groups. ZCL increased 113% 20 hours after TDI exposure but was not elevated 1 hour post-exposure. Likewise, NOSCL was significantly elevated by 630 % 20 hours post exposure. FR formation increased from 32±8 SEM (Gr I) to 55 ±4 ($p<0.05$) in Gr II to 70±8 SEM ($p<0.01$) in Gr III. We conclude that the early lung response to an acute irritant TDI exposure is stimulation of AM as demonstrated by increased CL, induction of NOS and FR formation. This occurs before other measurable markers of acute lung injury. The relationship between this type of TDI-induced lung injury and the initiating mechanisms of TDI sensitization and TDI-induced asthma may be related but has not been addressed.

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