behaviour. This was related to the basic psychological needs, mainly due to a higher of the need for autonomy ($p < 0.01$). **Conclusion** Need for autonomy important in the motivation to reduce sitting. Presenting different to reduce sitting seems to autonomy. Future studies are warranted to evaluate if more emphasis on the need relatedness by organising e.g. group discussions during the interventions, results in a larger reduction sitting behaviour.

Allergy and Immunotoxicology

1053

AUGMENTED PROLIFERATION OF MESOTHELIAL CELLS CAUSED BY SECRETORY FACTORS DERIVED FROM IMMUNE CELLS UPON EXPOSURE TO ASBESTOS

¹Y Nishimura*, ²Y Maki, ¹N Kumagai-Takei, ¹S Lee, ¹H Matsuzaki, ¹K Yoshitome, ¹T Otsuki. ¹Department of Hygiene, Kawasaki Medical School, Kurashiki, Japan; ²Department of General Thoracic Surgery, Okayama University Graduate School of Medicine, Dentistry and Pharmaceutical Sciences, Okayama, Japan

10.1136/oemed-2018-ICOHabstracts.135

Introduction Our studies have demonstrated suppressive effects of asbestos exposure on anti-tumour immunity related with malignant mesothelioma. On the other hand, whether there is a potential interaction between immune and mesothelial cells remains to be clear. The present study examined the effect of secretory factors produced by human peripheral blood mononuclear cells (PBMC) upon exposure to chrysotile A (CA) or crocidolite (CR) asbestos on human mesothelial cell line of MeT-5A.

Methods PBMC were cultured with antibodies to CD3 and CD28 upon exposure to CA or CR at 5 or 20 µg/ml. After 2 or 7 days, culture supernatants were harvested and stored. MeT-5A cells were cultured with 8-fold diluted culture supernatants for 48 hours. Cell proliferation was assayed by WST-1. Cytokines in the culture supernatants were assayed by luminex. G-CSF, GM-CSF, IL-1α, IL-1β, IL-3, IL-5, IL-13 and IL-17A were added into the parts of MeT-5A cell cultures.

Results In contrast to 2 days, the supernatants of 7 days PBMC cultures with CA or CR at 20 µg/ml significantly increased MeT-5A cell proliferation. The productions of IL-1α, IL-1β, IL-3, IL-5, IL-13 and IL-17A in the culture of PBMC were high upon CA or CR exposure. The supplementation with G-CSF and GM-CSF into the culture did not increase proliferation of MeT-5A, whereas IL-1α, IL-3, IL-5, IL-13 and IL-17A augmented it. In contrast, the combined addition of these cytokines did not change MeT-5A cell proliferation.

Conclusion These results indicate that there is an interaction between immune and mesothelial cells, in which secretory factors derived from immune cells exposed to asbestos augmented mesothelial cell proliferation. Actually, asbestos-exposed immune cells showed increased production of cytokines, some of which individually augmented MeT-5A cell proliferation. Those findings suggest that asbestos-exposed immune cells might let mesothelial cells proliferate in an uncontrolled manner, leading to generation of transformed cells.

1097

IMPORTANCE OF SKIN EXPOSURE IN A SUB-CHRONIC MOUSE MODEL OF CHEMICAL-INDUCED ASTHMA

¹Jeroen AJ Vanoirbeek, ¹Lore Pollaris, ²Tatjana Deceasterker, ¹Sofie Van Den Broucke, ¹Tsui Hung-chang, ³Sven Seys, ¹Peter HM Hoet, ¹Benoit Nemery. ¹Centre for Environment and Health, Department of Public Health and Primary Care; ²Pneumology, Department of Clinical and Experimental Medicine; ³Laboratory of Clinical Immunology, Department of Microbiology and Immunology, University of Leuven, Leuven, Belgium

10.1136/oemed-2018-ICOHabstracts.136

Introduction Toluene 2,4-toluene diisocyanate (TDI) is well-known chemical sensitizer and occupational asthmagogen. In a chronic mouse model, with dermal sensitisation and five weeks of intranasal challenges with TDI we were able to induce several key hallmarks of occupational asthma, including airway hyperreactivity (AHR), and a predominant Th2 immune responses. Yet, no features of airway inflammation, nor airway remodelling were present. Therefore, we altered the TDI mouse model and introduced endotracheal challenge to investigate lung inflammation and remodelling.

Methods On days 1 and 8, BALB/c mice were dermally treated (20 µl/ear) with 0.5% TDI or the vehicle AOO (3:2). From day 15, the mice received under light isoflurane anaesthesia, a total of five oropharyngeal challenges with 20 µl of the chemical or AOO (1:4) (days 15, 17, 19, 23 and 24). Two days after the last challenge, airway hyperreactivity (AHR) to methacholine was assessed, followed by an evaluation of pulmonary inflammation in bronchoalveolar lavage (BAL). As immunological parameters, lung dendritic cells, lymphocyte subpopulations (T- and B-cells) and the cytokine production profile (Th1 vs Th2) in auricular lymph nodes were measured. Blood was sampled to determine total serum IgE, IgG1 and IgG2a.

Results Mice dermally sensitised and challenged with TDI showed significant increased proliferation of the auricular lymph nodes, characterised by Th-, Tc- and B-cells. The cytokine production profile of the auricular lymph nodes showed increased levels of IL4, IL13, IL10 and IFNλ. Furthermore, mice sensitised and challenged with TDI showed significantly increased serum IgE and IgG1 levels. These TDI-sensitised and challenged mice showed pronounced airway hyperreactivity, along with a mixed eosinophilic and neutrophilic inflammation and recruitment of several dendritic cell subpopulations. Mice that were not dermally sensitised, but only received the TDI challenges did not show airway hyperreactivity, but had a neutrophilic lung inflammation, compared to the complete control mice. These mice also did not show any signs of immune sensitisation, yet dendritic cell recruitment to the lungs was identical to the TDI-sensitised and TDI-challenged mice.

Conclusion Endo-tracheal instillation with TDI leads to lung inflammation, without AHR, probably due to the irritant properties of TDI. Yet, mice dermally sensitised with TDI, followed by TDI challenges showed a predominant Th2 response, with AHR and eosinophilic inflammation. These data confirm the important role of dermal sensitisation in the development of chemical-induced asthma.