The immunological effects of asbestos exposure on various lymphocytes such as the regulatory T cell (Treg), responder CD4+ T helper cell (Tresp), CD8+ cytotoxic T lymphocytes (CTL) and natural killer (NK) cells were investigated. Results show that asbestos exposure impairs anti-tumour immunity through enhancement of regulatory T cell function and volume, reduction of CXCR3 chemokine receptor in responder CD4+ T helper cells, and impairment of the killing activities of CD8+ cytotoxic T lymphocytes (CTL) and NK cells. These findings were used to explore biological markers associated with asbestos exposure and asbestos-induced cancers, and suggested the usefulness of serum/plasma IL-10 and TGF-β, surface CXCR3 expression in Tresp, the secreting potential of IFN-γ in Tresp, intracellular perforin level in CTL, and surface expression NKp46 in NK cells. Although other unexplored cytokines in serum/plasma and molecules in these immunological cells, including Th17, should be investigated by experimental procedures in addition to a comprehensive analysis of screening methods, biomarkers based on immunological alterations may be helpful in clinical situations to screen the high-risk population exposed to asbestos and susceptible to asbestos-related cancers such as mesothelioma.

**Abstracts**

**CONTACT DERMATITIS AMONG WORKERS OCCUPATIONALLY EXPOSED TO FERRONICKEL ALLOYS**


**Introduction**

Many studies have shown that nickel and its alloys can be potential irritants and sensitizers among workers engaged in ferronickel alloy production, and provoke occupational contact dermatitis. 

**Objective**

To assess the prevalence of contact dermatitis focusing on allergic contact dermatitis in workers exposed to nickel while producing ferronickel alloys.

**Methods**

A cross-sectional study included 103 male workers (mean age=49.1±10.1) employed as ferronickel smelters (duration of exposure 18.2±11.9) with direct contact to nickel. Their findings were compared with a control group of 37 male workers (mean age=46.7±10.6), employed in the same facility, without direct nickel contact, matched for age, smoking habits and socioeconomic status. Evaluation of examined subjects included Nordic Questionnaire on Occupational Skin Diseases (NOSQ-2002/long), physical examination of the skin changes on hands, wrists and forearms, and patch test with NiSO4 (5%).

**Results and discussion**

Skin rash during six months was registered in 21 (20.4%) exposed worker, and in 4 (10.8%) controls. The prevalence of skin changes, chronic rhinitis, conjunctivitis, and asthma was higher in exposed workers, but without statistical significance. Hand skin fluorescence due to non-occupational substances were present in 10 (9.8%) of exposed workers, and among 2 (5.5%) of controls. There was no significant difference concerning urticaria between two groups, and non-occupational nickel sensitisation (metal buttons, jewellery, etc.). Positive patch test by 5% NiSO4 was registered in 20 (19.5%) exposed workers and in 2 (5.4%) controls (p<0.05). Significant difference was found between the two groups concerning improvement of skin lesions after temporary elimination of workplace exposure. Positive elimination test was registered among 5 (4.9%) exposed workers with hand contact dermatitis.

**Conclusion**

Our data confirmed that workplace nickel exposure can cause occupational allergic contact dermatitis among workers producing ferronickel alloys, and determined the need of preventive activities in order to decrease the pathogenic dermal effect of nickel.