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.

**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 in nickel. Their findings were compared with a control group of 37 male office workers (mean age=46.7±10.6), employed in nickel. The tumorigenicity of asbestos, which is thought to cause mesothelioma and lung cancer. However, in contrast, its effect on anti-tumour immunity remains unclear. In ICOH Congress 2015, we have reported the enhanced decrease in% perforin+ cells of stimulated CD8+ cells of the patients with malignant