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.

638  EFFECTS OF IL-15 ADDITION ON THE SUPPRESSED INDUCTION OF CTL UPON EXPOSURE TO ASBESTOS

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Introduction Asbestos exposure can cause malignant mesothelioma and lung cancer. However, in contrast, its effect on anti-tumour immunity remains unclear. Our previous study reported that asbestos exposure suppressed the induction of CTL during mixed lymphocyte reactions (MLR), accompanied by the decrease in proliferation of CD8+ T cells. Recently, we reported that IL-2 showed a tendency to increase% granzyme B+ cells in the CFSE-positive CD8+ lymphocytes without profound changes of CD8+ T cells during mixed lymphocyte reactions (MLR). Therefore, we investigated whether IL-15 addition might improve the suppressed induction of CTL upon exposure to asbestos.

Methods For MLR, human PBMCs were cultured with irradiated allogenic PBMCs upon exposure to chrysotile B asbestos at 5 μg/ml for 7 days. After 2 days of culture, IL-15 was added at 1 ng/ml. After 7 days of MLR, PBMCs were collected and analysed for phenotypic and functional markers of CD8+ T cells with fluorescence-labelled anti-CD3, anti-CD8, anti-CD45RA, anti-CD45RO, and anti-granzyme B Abs using flow cytometry.

Result IL-15 didn’t recover the asbestos-caused decreases in% CD25+ and% CD45RO+ cells and increase% CD45RA+ cells, but recovered the decrease in cell numbers of CD3+CD8+ cells and% granzyyme B+ cells, in contrast to IL-2.

Discussion These results indicate that IL-15 is more effective on recovery from asbestos-caused suppressed induction of CTL than IL-2, although the interfered expressions of cell surface markers were not recovered even by addition of IL-15. Further study about the characteristics of CD3+CD8+ granzyyme B+ cells induced by addition of IL-15 will contribute to clarification of the mechanism of asbestos-caused suppression in CTL induction and to finding out a clue to restore it.

640  EFFECT OF LONG-TERM EXPOSURE TO ASBESTOS ON FUNCTIONAL PROPERTIES OF HUMAN CD8+ T CELL LINE

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Introduction The tumorigenicity of asbestos, which is thought to cause mesothelioma, has been clarified, whereas 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