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Introduction

Asbestos exposure causes a variety of tumors such as malignant mesothelioma and lung cancers [1,2], in addition to laryngeal, gastrointestinal and bladder cancers [3-5].

Issues involving asbestos usage are also varied. Although the majority of developed countries have already banned the use of asbestos, a few nations are still exporting asbestos to developing countries and people in these asbestos-importing nations encounter many chances of being exposed to asbestos. Moreover, asbestos is still present in the buildings, water-pipes and other structures of nations that have even banned the material, and many workers such as the wrecking crews of buildings are under threat of attack from asbestos exposure.

In Japan, the asbestos issue erupted in the summer of 2005 [6-8]. Residents were suddenly informed that asbestos, which was used in large amounts from the early 1950s up to the early 1990s in Japan with a maximum usage of approximately 352,000 tons in 1974, caused malignant mesothelioma (MM). Residents that lived near the asbestos handling manufacturer Kubota Corporation, in Amazasaki City, Hyogo Prefecture, developed MM. ey had never worked in the asbestos-handling manufacture industry. In addition, medical information regarding MM induced anxiety in Japanese people, since the prognosis is very poor and there is no certain way to detect the cancer in the very early stage of the disease. Furthermore, people could not remember being exposed to asbestos 30 to 40 years ago. ese matters increased the anxiety of residents. Furthermore, there is concern for people who erResidents thao

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receptor 3 (CXCR3) and interferon- were examined since cDNA array, pathway and signaling canonical analysis, as well as network analysis, indicated that these molecules were involved and altered in all sublines when compared with the original (never exposed) MT-2 line [46,47].

us, the expression of CXCR3 in MT-2 sublines and in long-term exposure to chrysotile using freshly isolated peripheral CD4+ T cells were analyzed. All of the MT-2 sublines showed reduction of CXCR3 expression in mRNA and protein (examined via Western blotting, ow cytometry, and uorescent immunohistological staining). In addition, four weeks of exposure to chrysotile caused reduction of CXCR3 expression when freshly isolated CD4+ T cells derived from six HDs were cultured with IL-2 and chrysotile. Similar to CXCR3, other 1 type molecules such as IFN- , C-X-C motif chemokine 10 (CXCL10)/IFN- -induced protein 10 (IP-10) as the ligand for CXCR3, and chemokine (C-C motif) ligand 4 (CCL4)/Macrophage in ammatory protein-1 (MIP-1)- exhibited reduced production or mRNA expression in all of the MT-2 sublines. In addition, the peripheral CD4+ T cells from patients with PP or MM exhibited increased IL-6 production compared with cells from HDs when these cells were stimulated

among these molecules is extracellular signal-regulated kinase (ERK). us, the phosphorylation status of ERK1/2 was examined using the YT-A1 subline continuously exposed to chrysotile. In spite of the enhancement of ERK1/2 phosphorylation when YT-A1 original cells were cultured with target tumor cells, the K562 subline did not show any increase of phosphorylation of ERK1/2. In addition, the reduction of phosphorylation of ERK1/2 in the YT-A1 cell line was also obtained when cells were treated with wortomannin and PP2, inhibitors of phosphoinositide 3-kinase (PI3K) and Src-family kinase, respectively, and when NK cells were cultured with target K562 cells and anti-2B4 or NKp46 antibodies were added to the culture [51].

ese results strongly suggest that NK cells exposed to asbestos reduced their cytotoxic activity via inhibition of signaling pathways involving the ERK1/2 molecule and decrease of degranulation in perforin and granzyme B as shown in gure 3. Further investigation will be conducted regarding modi cation of NK cell activity in uenced by asbestos-exposed dendritic cells or monocyte/macrophage lineage cells, since their production of cytokines may a ect K cell activity and production may be altered by asbestos exposure onto these types of cells [50-52].

Conclusion

As described above, the e ects of asbestos on T cells and NK cells reduce their activity against tumor cells. However, other immunocompetent cells such as Treg, 17, CD8+ cytotoxic T cells (CTL), and natural killer T (NKT) cells, as well as antigen presenting