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Immunostimulation and Cancer Prevention

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Several studies have established the excessive cancer risk for workers exposed to several widespread workplace chemicals. The present study was designed to examine the immune alteration associated with exposure to toxic chemicals and possible counteracting of chemical toxicity using MGN-3. MGN-3 is a new biological response modifier (BRM) that consists of arabinoxylan compound, which is a polysaccharide that contains hemicellulose-･extract of rice bran, modified by enzymes from shiitake mushrooms.

Eleven individuals who had been exposed to chemicals in the workplace participated in the study. The participants demonstrated immune dysfunction as indicated by: low levels of natural killer (NK) cell activity (10.2±4.2 LUs), lymphocyte blastogenic responses to T-cell mitogens (PHA, 39060±12517 cpm and CONA, 36224±11922 cpm) and B-cell mitogen (PWM, 16550±6330 cpm), compared to control responses. Subjects received MGN-3 at a dose of 15 mg/kg/d for four months. Treatment with MGN-3 increased NK cell activity 4 and 7 fold at two and four months respectively, while T and B-cell functions were 130-150% higher than base line values.

NK cell immunomodulatory function by MGN-3 was also examined in ninety (90) patients with different types of malignancies: prostate(22), breast(20), multiple myeloma(16), ovary(5), and other organs(27). Patients received and completed conventional therapy such as surgery, chemotherapy, radiation or hormonal therapy prior to participation in the study. NK cell activity was serially monitored in a monthly basis using K562, a human erythroleukemic cell target. Patients received MGN-3 at dose 45 mg/kg/d for 2-5 years. In clinical MGN-3 trials, 95.5% of patients (86/90) demonstrated an augmentation of NK activity (2-10 fold increase of baseline) at 1-2 weeks post treatment. The activity was maintained at a high level for up to 5 years. The effect of stress during human NK cell immunomodulation by MGN-3 is worth investigation, since stress is known to be a strong immune depressant. During the

course of treatment with MGN-3, 11.6% of the responders (10/86) suffered from different kinds of stressors, such as: sleep deprivation, bereavement following the loss of a spouse or close relative, work overload or financial problems. These patients evidence 40-75% decrease in their NK activity as compared to the previous level. After elimination of some stressors, the NK activity increased back to the former value.

We conclude that 1) MGN-3 is a new BRM that does offer a new immunotherapeutic approach to cancer by increasing host anti-tumor responses through an augmentation of the NK cell activity. 2) Suppression in NK activity by chemical exposure can be fully restored with MGN-3. 3) Our preliminary results suggest that stress cause immune suppression during the course of MGN-3 treatment. Monitoring NK activity on a monthly basis will be able to detect this suppression which requires correction, otherwise it will have definite future clinical ramifications.