Immune Activation and Autoantibodies in Humans with Long-Term Inhalation Exposure to Formaldehyde

Jack D. Thrasher, Ph.D., Alan Broughton, M.D., Ph.D., Roberta Madison, .PhD.

Published in: Archives of Environmental Health, Vol. 45, pp. 217-223, 1990

ABSTRACT: four groups of patients with long-term inhalation exposure to formaldehyde (HCHO) were compared with controls who had short-term periodic exposure to HCHO. The following were determined for all groups: total white cell, lymphocyte, and T cell counts; T-helper/T-suppressor ratios; total Ta1+, IL2+, and B cell counts; antibodies to formaldehyde-human serum albumin conjugate and autoantibodies. When compared to the controls, the patients had significantly higher antibody titers to HCHO-HSA. In addition, significant increases in Ta1+, Il2+, and B cells and autoantibodies were observed. Immune activation, autoantibodies, and HCHO-HSA antibodies are associated with long-term formaldehyde inhalation.

Introduction:

INHALATION EXPOSURE to formaldehyde (HCHO) is associated with symptoms of irritation to mucous membranes,[1,2] chronic health problems (e.g. asthma,[2] nasopharyngeal cancer,[3)] and multiple subjective health complaints[4,5]. Recent observations have shown that both humoral- and cell-mediated Immunologic mechanisms occur in humans with long-term HCHO exposure. Antibodies of all isotypes to HCHO conjugated human serum albumin (HCHO-HSA) are demonstrable in HCHO anaphylaxis,[6] hemodialysis patients,[7] mobile home residents,[4] persons with occupational exposures,[5,8] office workers,[9] and in person in other environments.[4] In addition, changes in cell-mediated immunity include increases in eosinophils, basophils, and T-suppressor cells following acute exposure of patients with HCHO asthma.[10] Moreover, individuals with multiple subjective health complaints associated with long-term HCHO inhalation have evidence of immune activation and the presence of autoantibodies.[4,5]

The patients in our study had symptoms and complaints related to several organs, as described previously,[4,5,9] which were similar to symptoms of workers with multiple chemical sensitivity,[11] cacosmia,[12] and other chemical exposures.[13-15] We report on the differences in humoral and cell-mediated immunity in humans with long-term inhalation exposure to HCHO vs. asymptomatic students (controls) who experienced short-term, periodic exposure to the chemical.

In conclusion, measurements of changes in WBCs, T cells, and H/S ratios in individuals with apparent chemical sensitivities appear to be inadequate immune parameters to examine. If one assumes that these individuals respond immunogically to environmental chemicals, investigations into autoimmunity and immune activation and perturbations in the interleukins, leukotreines, prostaglandins, and other immunologic mediators appear to be fruitful areas for further research.[29-32] Thus, it appears that HCHO sensitivity is a real phenomenon and requires further research.[4,27-32]

If you wish a copy of this paper contact Dr. Thrasher at toxicologist1@msn.com