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Atopic dermatitis-associated protein interaction network lead to new insights in chronic sulfur mustard skin lesion mechanisms

Expert Rev. Proteomics 10(5), 449–460 (2013)

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Chronic sulfur mustard skin lesions (CSMSLs) are the most common complications of sulfur mustard exposure; however, its mechanism is not completely understood.According to clinical signs, there are similarities between CSMSL and atopic dermatitis (AD). In this study, proteomic results of AD were reviewed and the AD-associated protein–protein interaction network (PIN) was analyzed. According to centrality measurements, 16 proteins were designated as pivotal elements in AD mechanisms. Interestingly, most of these proteins had been reported in some sulfur mustard-related studies in late and acute phases separately. Based on the gene enrichment analysis, aging, cell response to stress, cancer, Toll- and NOD-like receptor and apoptosis signaling pathways have the greatest impact on the disease. By the analysis of directed protein interaction networks, it is concluded that TNF, IL-6, AKT1, NOS3 and CDKN1A are the most important proteins. It is possible that these proteins play role in the shared complications of AD and CSMSL including xerosis and itching.

Keywords: acute sulfur mustard skin lesion • atopic dermatitis • chronic sulfur mustard skin lesion • protein interaction network • proteomics

Sulfur mustard & acute & chronic complaints

Sulfur mustard (SM), an alkylating agent developed in the early 19th century, has been used as a chemical agent in several wars. SM is less toxic than other chemical agents, but is used more extensively because of its low cost, ease of production, ability to disable troops in the battlefield and long-term complaints. A low dose of SM usually does not result in acute symptoms, but a high dose causes severe and acute symptoms. Some reports indicate that cumulative low doses can also lead to serious side effects [1].

SM is absorbed by inhalation, through the skin, mucous membranes or eyes and through the digestive tract following consumption of contaminated food. Exposure of the eyes to high-dose SM causes conjunctivitis, eyelid rash and sometimes temporary blindness [2]. Acute pulmonary obstruction occurs after inhalation of SM. The eyes are the most sensitive parts of the body to SM, but pulmonary obstruction is the primary cause of mortality.

The lipophilic property of the skin makes it a suitable transmission system for SM as a lipophilic agent. Following skin contact with liquid SM, 80% of SM evaporates, 2.4% remains on the skin and 17.6% is absorbed systemically [3]. The result of long-term contact is marked systemic toxicity. Acute SM skin lesions (ASMSL) seen in Iranian troops exposed to SM included erythematosus, pigmentary exfoliation, superficial vesicles, bullous, necrotic bullous, deep necrosis, allergic contact dermatitis and toxic reactions [4]. Depending upon the intensity of the SM, first, second or third degree burns occur. Where there is 50% skin contact, mortality is likely.

Blisters gradually develop on the skin after SM exposure. After 6–24 h, itching and severe sensitivity of the skin develops [5,6]. Itching and erythema appear after 2–8 h of contact

 10.1586/14789450.2013.841548 © 2013 Informa UK Ltd ISSN 1478-9450

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