Immunome Res 2018, Volume 14
DOI: 10.4172/1745-7580-C1-015

9th Molecular Immunology & Immunogenetics Congress

March 08-09, 2018 | London, UK

Th1 chemokine secretion by vanadium pentoxide in primary dermal fibroblasts

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Statement of the Problem: Vanadium is a silvery-grey and soft metal with different oxidation states (-1, 0, +2, +3, +4 and +5). The most common commercial form is vanadium pentoxide (V_2O_5). All vanadium compounds are considered toxic. The Occupational Safety and Health Administration have set an exposure limit for the workplace (considering about a 40 h work week), of 0.05 mg/m3 for V_2O_5 dust and 0.1 mg/m3 for V_2O_5 fumes. The exposure dose of vanadium that is considered life-threatening is 35 mg/m3 (as shown by The National Institute for Occupational Safety and Health (NIOSH)), that can cause serious and perpetuating health issues, and death. The most vulnerable to vanadium toxicity is the respiratory system, while the effect on the gastrointestinal system is minimal owing to the low gut absorption rate. An increase in skin rashes has been observed in certain vanadium workers, including the development of atopic dermatitis. However, to the best of our knowledge, no prior *in vivo* or *in vitro* studies have evaluated the effect of vanadium exposure in dermal fibroblasts.

Methodology & Theoretical Orientation: Here, we evaluate the effect of V_2O_5 on proliferation and chemokine secretion in dermal fibroblasts.

Findings: The results showed that V_2O_5 has no effect on fibroblasts viability or proliferation, but it induces the secretion of T-helper (Th)1 chemokines from dermal fibroblasts, synergistically increasing the effect of important Th1 cytokines, as interferon-gamma and tumor necrosis factor-alpha. In this way, V_2O_5 can lead to the induction and perpetuation of an inflammatory reaction in dermal tissue.

Conclusion & Significance: Additional studies are required to evaluate dermal integrity and manifestations in subjects who are occupationally exposed or living in polluted areas.

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