Evaluating the Relationship of Sulfur Mustard-Induced Cutaneous Injury and Reactive Oxygen Species

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Sulfur Mustard (SM) is an alkylating vesicant that is known to be used in chemical warfare. In recent history, SM gas was used in the Iran-Iraq war and in the Syrian conflict. It primarily affects the respiratory, ocular, and cutaneous systems. When absorbed within the skin, SM causes erythema, blistering and necrosis. While the primary mechanism of injury is not known, damage can be caused by a number of pathways including aberrant reactive oxygen species (ROS) signaling. The purpose of this review is to clarify the role of ROS in facilitating SM-induced cutaneous injury. Articles were found via an extensive literature search to compare mechanistic studies. Search terms were “Sulfur mustard”, “mustard gas” , “ROS”, “reactive oxygen species”, “oxidative stress”, “cutaneous injury”, and “skin injury”. Studies that included Nitrogen Mustard and 2-chloroethyl ethyl sulfide (CEES) were also included in evaluations because they are common analogs used to study SM injury. Information was organized and dated for a final write up. Findings indicate that SM induces oxidative stress via the upregulation of ROS and the depletion of antioxidants. This imbalance causes direct and indirect damage including DNA damage, inflammation, and eventual cell death. Results from this analysis contribute to a greater understanding of SM-induced skin injury. At present, there is no approved treatment for SM-induced skin injury. Through investigation of SM-mediated mechanisms of injury, therapeutic targets can be identified, and treatments developed to help mitigate or prevent progression of damage. Supported by ASPET SURF and NIH R25 ES020721 and AR055073.