AI/ML AND VIRTUAL HUMAN PLATFORMS FOR THREAT AGENT HAZARD ASSESSMENT AND MEDICAL COUNTERMEASURE DISCOVERY AND DRUG DEVELOPMENT

Regulation Of Actin Depolymerization Resulting From Exposure To Choking Agent

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Agents of lung injury result in Acute Respiratory Distress Syndrome (ARDS) through proinflammatory signaling leading to actin depolymerization. In order to determine if chemical warfare choking agents act in a similar way, we developed a mouse model of lung injury using CEES, a sulfur mustard analog. We found that the toxin affects the alveolar proinflammatory signaling pathway that deteriorates the air-liquid barrier formed by the alveolar epithelium and the adjoining endothelium. We found two key points along the pathway leading to actin-dependent barrier loss that are involved in the response to exposure of CEES. We are now focused on regulating these steps to prevent severe lung injury.

This work was supported by HDTRA1-18-1-0049.