**Abstract for NIEHS T32 – Samuel Arriola**

Chloropicrin (CP) is a toxic industrial chemical used as a soil fumigant and has been identified as a high priority chemical threat agent due to its potential for accidental or intentional release. While CP is known to cause acute lung injury, the dose-dependent cellular targets and mechanisms underlying injury and repair are not well defined. This project will investigate the pulmonary effects of CP using a mouse model to define dose-response relationships, characterize acute injury patterns, and examine temporal dynamics of lung repair. Specific Aim 1 will establish the primary cellular targets of injury across a range of CP doses using histopathology, oxidative stress markers, and inflammatory profiling. Aim 2 will assess how CP-induced injury evolves or resolves over time, focusing on epithelial regeneration, fibrosis, and immune cell dynamics. Aim 3 will test whether local estrogen production by macrophages regulates lung repair, using a macrophage specific CYP19A1 knockout model. Together these studies will identify mechanisms of CP-induced lung damage and recovery, providing insight into potential therapeutic targets and supporting the development of medical countermeasures to mitigate the health effects of chemical exposures.