Abstract:
Indium is an important metal used in many modern-day technologies such as flat panel displays, solar panels and other microelectronic devices. Over the last decade, the industrial use of indium has grown appreciably due to increased demands for consumer and commercial electronics. The increased industrial use of indium has also lead to a greater number of potential occupational exposures to indium. Case reports have emerged describing a progressive and often irreversible lung disease in a small number of workers primarily employed in indium production and reclamation facilities. OSHA has not yet established an occupational exposure limit (OEL) for indium. Both NIOSH and ACGIH have each recommended an OEL for indium but these recommendations were made more than 20 years ago. The purpose of this evaluation was to comprehensively review the current toxicological information pertaining to indium to derive an updated OEL for indium and indium compounds. Based on the current data, three critical health endpoints were identified: alveolar proteinosis (animal and human), bronchiolo-alveolar hyperplasia (animal), and lung tumors (animal). The available data suggest that cellular infiltration such as accumulation of alveolar macrophages, chronic inflammation, and remodeling (e.g., proteinosis, fibrosis, hyperplasia) are precursor events to neoplasia. For this analysis, two different approaches were used to derive an OEL. Depending on the dose-response curve for each end-point, a LOAEL approach (alveolar proteinosis) or bench mark dose approach (hyperplasia, lung tumors) was used to derive a point-of-departure (POD) from chronic inhalation studies conducted in rats exposed to indium tin oxide. Each POD was evaluated based on a weight-of-evidence consideration of toxicokinetics, toxicodynamics, and other uncertainties. Overall our analysis supports the use of animal data for the derivation of an OEL for respirable indium of 0.03 μg/m³.