Targeting AMPK and the Nrf2/HO-1 axis: a promising therapeutic strategy in acute lung injury

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Therapeutic targeting of AMPK can improve acute lung injury *via* upregulation of the cytoprotective enzyme heme oxygenase-1 https://bit.ly/3DLf1xT

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Acute lung injury (ALI) is defined as pulmonary conditions leading to severe hypoxaemia, with human acute respiratory distress syndrome (ARDS) representing its most severe manifestation [1]. The primary causes of ARDS are trauma, bacterial and viral pneumonia, sepsis and adverse drug reactions [2]. The development of effective therapeutics for ALI/ARDS remains an urgent unmet need [3]. The overall crude incidence of ALI from all sources has been estimated at 76.9 per 100 000 person-years (86.2 after age adjustment) [4]; or ∼200 000 cases per year in the USA [1]. ALI is generally associated with endothelial and epithelial cell injury, loss of alveolar–capillary membrane integrity, neutrophil influx into the lung, and release of pro-inflammatory mediators [1]. Exposure to halogen gases, such as bromine (Br_2) and chlorine (Cl_2), as well as to halogenated organic compounds, can cause severe ALI. Although many experimental animal models of ALI have been developed [5], halogen gas-induced ALI has been less widely studied. Halogen gas-induced ALI/ARDS remains a public health concern as it can arise from industrial occupational and accidental exposures. Despite individual case reports, there is a lack of more precise epidemiological data on the incidence of halogen-gas specific ALI [6].