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Title: Oxidative stress and lung permeability during chlorine-induced acute lung injury in mice

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Body: Clinical diagnosis and prognosis of respiratory injury after accidental inhalation of chlorine is complicated by unknown exposure levels of the individuals, delayed symptoms and a wide range in pathological out-come. Here we used a murine model for chlorine-induced lung injury to evaluate if indicators of oxidative stress and lung permeability can be used as biomarkers of acute lung injury. Mice was exposed to 200 ppm Cl2 during 15 min by nose-only inhalation. Bronchoalveolar lavage fluid (BALF) and serum was sampled from 2 h up to 14 days post exposure and analysed with ELISA regarding oxidation of phospholipids (8-isoprostane PGF2α), albumin, surfactant protein D (SP-D) and Clara cell specific protein 16 (CC16). The dose-response of the markers was also determined in mice exposed to 25, 50, 100 and 200 ppm Cl2 and their specificity was evaluated in another murine model for chemical-induced lung injury (mustard gas analogue). Exposure to Cl2 caused a dose-dependent increase of 8-isoprostane PGF2α in BALF and the highest concentration of Cl2 significantly increased ($p<0.001$ at 2 h) the levels of 8-isoprostane PGF2α up to 12 h after exposure. In contrast levels of SP-D were unaffected by Cl2 exposure in BALF but were significantly decreased in serum up to 72 h after exposure. A significant increase of albumin in BALF and CC16 in the circulation indicates alterations in lung permeability. Inhalation of the mustard gas analogue caused changes in lung permeability but did not increase phosopholipid oxidation. We conclude that 8-isoprostan PGF2α represents a promising local biomarker for chlorine-induced lung injury that potentially could be used as a diagnostic tool.