

Glutathione depletion and in vitro lipid peroxidation in mercury or maleate induced acute renal failure.

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[Gstraunthaler G](#), [Pfaller W](#), [Kotanko P](#).

Nephrotoxic acute renal failure was experimentally induced in male rats by s.c. application of mercuric chloride and i.p. administration of maleate, respectively. Mercuric chloride and maleate are known to enhance the formation of free radicals and peroxides, which presumably overload the cell's natural elimination mechanisms for these highly reactive intermediates. In addition, a reduction in activities of superoxide dismutase, catalase and glutathione-peroxidase, enzymes responsible for the protection of cells against peroxidative action of superoxide anions and hyperperoxides was found. In both models of acute renal failure, enhanced lipid peroxidation in kidney homogenates in vitro, monitored as malondialdehyde production, was observed. Furthermore, HgCl₂ and maleate may react with free SH-groups and thus lead to a depletion of glutathione in tubular cells. Indeed, renal cortical contents of reduced and oxidized glutathione were drastically diminished. These results suggest that alterations in membrane integrity, possibly caused by peroxidative processes, can be considered the cause underlying the well-known disturbances in renal function commonly observed during the initiation phase of HgCl₂ and maleate induced acute renal failure.

