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Protective role of intracellular glutathione against nitric oxide-induced necrosis in rat gastric mucosal cells.

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BACKGROUND: Nitric oxide synthase activity is increased in the stomach in association with *Helicobacter pylori* infection and portal hypertension, but the mechanism by which nitric oxide contributes to mucosal damage remains unclear. **AIM:** To examine whether nitric oxide injures gastric mucosal cells and whether cellular glutathione affects nitric oxide-induced cytotoxicity. **METHODS:** A confluent monolayer of RGM-1 gastric mucosal cells was exposed to nitric oxide donors (NOC5 or NOC12). Cell viability was determined by trypan blue dye exclusion, lactate dehydrogenase release and supravital staining with Hoechst 33342 and propidium iodide. The kinetics of the reduced/oxidized forms of glutathione were also measured, as well as the effect of glutathione-depletion or glutathione-precursor treatment on nitric oxide-induced cytotoxicity. **RESULTS:** Excess exogenous nitric oxide produced by NOC5 or NOC12 induced necrosis in RGM-1 cells in a time- and concentration-dependent manner. The level of reduced glutathione drastically decreased prior to the loss of cell viability and remained low, but oxidized glutathione was not affected. Glutathione depletion increased necrosis of both NOCs in an NOC-concentration-related fashion, while pre-treatment with gamma-glutamylcysteine ethyl ester reduced their necrotic susceptibility. **CONCLUSION:** Exogenous nitric oxide induced necrosis in gastric mucosal cells, and intracellular reduced glutathione protects gastric mucosal cells from damage by nitric oxide.

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