Effect of orally administered reduced- and oxidized-glutathione against acetaminophen-induced liver injury in rats.

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To investigate the effects of orally administered reduced-glutathione (GSH) and oxidized-glutathione (GSSG) on liver GSH repletion and liver injury, acetaminophen (AAP) highly loaded rats were used. Orally administered GSH, dependent on dosage, indicated a protective effect against AAP-induced hepatotoxicity. This effect was associated with a recovery of liver GSH levels. Orally administered GSSG also indicated liver GSH recovery and hepatotoxicity inhibition to the same extent as orally administered GSH. Because intraperitoneally administered GSH did not indicate liver GSH recovery, the replenishment of GSH levels after orally administered GSH is thought to be produced through the degradation of GSH into its constituent amino acids in the intestine and their re-synthesis in the liver. On the other hand, orally administered GSH indicated a lower liver GSH recovery than orally administered cysteine prodrugs did, although the hepatotoxicity inhibitory degree was similar. Thus orally administered GSH may have another hepatoprotective system besides the resynthesis of its constituent amino acids. The current study establishes that orally administered glutathione, both GSH and GSSG, is a useful tool to recover liver GSH levels and to prevent liver injury.

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