EFFECT OF LYCOPENE ON RENAL DAMAGE INDUCED BY MERCURIC CHLORIDE IN RATS

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Oxidative stress has been pointed as an important molecular mechanism for kidney injury in mercury poisoning and the interaction of the metal with endogenous thiol-containing molecules such as d-aminolevulinate desidratase (d-ALA-D), seems to contribute to this process. Lycopene, a plentiful carotenoid in tomatoes, has been studied because of its large antioxidant properties. This study evaluated the ability of lycopene to prevent $HgCl_2$ toxicity, assessing parameters like d-ALA-D activity, non protein sulfhydrylic groups content, creatinine plasma levels and histopathological analyses. Rats received lycopene, by gavage (10, 25 and 50 mg/kg) six hours prior to the administration of 5 mg/kg $HgCl_2$. Twelve hours after exposure to $HgCl_2$ renal ALA-D activity was inhibited (~35%) and creatinine plasma levels were increased (~123%). Moreover, tubular necrosis was observed in animals treated with $HgCl_2$ when compared to control. Although 10 and 50 mg/kg lycopene has prevented ALA-D inhibition, it did not prevent increase in creatinine levels or tubular necrosis caused by $HgCl_2$. Neither $HgCl_2$ nor lycopene affected non protein sulfhydrylic groups content. Our results indicate that although lycopene did not prevent $HgCl_2$-induced renal failure, it could have a beneficial role against $HgCl_2$ toxicity by preventing changes in the activity of δ-aminolevulinate dehydratase.

Keywords: d-aminolevulinic acid dehydratase, creatinine, tubular necrosis, rats.

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