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# SODIUM THIOSULPHATE SHOWS PROMISING ANTI-INFLAMMATORY ROLE AGAINST DOXORUBICIN-INDUCED RENAL INJURY DEPENDING ON TLR4 PATHWAY INHIBITION

**Abstract** Doxorubic in nephrotoxicity is always a major cause of death for cancer patients. objectives: our study aimed at proving the potential curative mechanisms of sodium thiosulphate, on experimentally-induced nephrotoxicity in rats by doxorubicin explaining the mechanisms of the serious inflammation pathway TLR4/MAPK P38/NF-κB/TNF-α. Methods: nephrotoxicity was induced by parenteral administration of doxorubicin (5.2mg/kg/ weekly for 4 weeks). And the treatment depends on giving sodium thiosulphate (400 mg/kg, p.o.) One hour before doxorubicin injection for4 weeks. Doxorubicin injection caused severerenal dysfunction evident from a significant increase in the kidney biomarkers; urea,creatinine, KIM.1, and serum cystatin C,together with decreasing serum albumin and total protein.Besides,increased MDA and MPOassociated with a significant decrease inGSH, Nrf-2,SOD and catalase activities, heightened inflammatory markers TLR4, MAPK P38,NF-κB,IL-1β, and TNF- alpha also, induced apoptotic markers expression in renal tissues of doxorubicin group.However, treatment with sodium thiosulphate normalized oxidative markers, inflammatory markers,MDA,MPO, GSH, SOD, Nrf-2and catalase. Also, prevented apoptotic changesthroughsuppressing BAX and increasing Bcl-2. Conclusion: Our study provides a promising protective use of sodium thiosulphate against doxorubicin nephrotoxicity.

**Keywords:** Doxorubicin, Nephrotoxicity, oxidative stress, Inflammation, Apoptosis, TLR4