Inhibition of Ascites Tumor Growth in Mice by Sodium Orthovanadate Mediated by Oxidative Stress

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Several studies report that vanadate possesses antineoplasic effects and it was also showed that vanadate derivatives lead to oxidative stress in animals. The aim of this work was to evaluate the sodium orthovanadate (SO) antitumor activity *in vivo* and its potential relationship with oxidative stress. Antitumor activity of SO was investigated using the Ehrlich Ascites Carcinoma (EAC) inoculated intraperitoneally in isogenic Balb/c male mice (20.83±0.98 g b.w., n=6) after treated during 9 days with SO (37.5 mg/kg body weight per day, treated group=TG). Negative Control (NC), received only DMSO 10% (50µL) daily. 10^{th} day animals were sacrificed and the ascitic fluid was collected and oxidative stress antitumoral index were evaluated. Results were expressed by means and standard deviation and they were analyzed using one-way ANOVA and Tukey-Kramer test. SO treatment caused decreased tumor (4.0±1.0 mL), packed cell volume (1.7±0.4 mL) and the viable cell count (79±5 x 10^6 cell/mL) when compared to NC (8.7±2.4 mL; 3.0±0.5 mL; 93±15 x 10^6 cell/mL, respectively) demonstrating a SO antitumor potential. The catalase (TG=18.71±0,65; NC=6.0±0.35 nmol/mg protein), glutathione-S-transferase (TG=3.48±0.05; NC=2.48±0.22 µmol/mg protein) and superoxide dismutase (TG=1.20±0.13; NC=0.97±0.05 USOD/mg protein) activities increased with SO treatment. Also lipid peroxidation (TG=1.40±0.17; NC=0.94±0.12 nmol/mg protein) increased. SO treatment did not cause significant difference at the GSH content (TG=7.06±0.36; NC=6.50±0.68 nmol/mg prot). The results suggest that SO exhibited antitumor activity and could be associated to oxidative stress in the tumor.

Keywords: *sodium orthovanadate, antitumoral effect, oxidative stress*