Vitamin A Supplementation Can Yield Oxidative Damage In The Heart

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Retinoids play an important role for cardiovascular system in the development and adult life (regulating cellular events) and antioxidant properties are also reported, thus vitamin A supplementation has been sugested for treatement of diseases related with oxidative stress, as atherosclerosis. However, our group has demonstrated that retinol can generate oxidative stress in *ex vivo* and *in vivo*. Therefore, the aim of this study was to compare heart redox parameters between vitamin A-supplemented and saline-treated rats. Thirty five were used, divided into five groups, Control (saline) and four treated with different doses of Vitamin A (1000, 2500, 4500 and 9000 $IU.Kg^{-1}.day^{-1}$). Animals were treated for 28 days and thereafter were killed and its heart removed. We found increased lipoperoxidation (TBARS) levels and a decrease on Catalase activity (CAT) at 9000 $IU.Kg^{-1}.day^{-1}$ retinol-treated group. There were no observed modifications on protein damage (CARBONYL), reduced thiol (SH), non-enzimatic antioxidant activity (TRAP) and Superoxide Dismutase activity (SOD), however when we did the ratio between SOD and CAT, we observed a increase in 9000 $IU.Kg^{-1}.day^{-1}$ group. We detected a decrease in Catalase immunocontent at all doses. We concluded that the increased SOD/CAT could explain the increased lipoperoxidation, because an imbalance in these enzymes can acumulate hydrogen peroxide, that yield (in transition metals presence) hydroxil radical, which can oxidatively damages biomolecules as lipids. The decreased CAT activity was related with the decrease on its immunocontent.

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