Comparison of the effects of hydroxy-selenomethionine and selenium-enriched yeast against hydrogen peroxide-induced oxidative stress in rat intestinal epithelial cells

S. Osho, B. Higgins, A. Boyer, K. Saddoris-Clemons, B. Humphrey, M. Garcia
Phibro Animal Health Corporation, Teaneck, NJ 07666, USA

Superior bioavailability of organic selenium (Se) has been extensively studied. However, limited research has been conducted to evaluate the effect of organic forms on intestinal redox potential to support gut health and animal well-being. Two experiments (Exp) were conducted to compare the effect of either hydroxy-selenomethionine (OH-SeMet) or Se-enriched yeast (Se-yeast) on ameliorating hydrogen peroxide (H_2O_2)-induced oxidative stress in a rat intestinal epithelial cell line (IEC-6). A cell viability test was initially performed to validate the Se level (0.15, 0.3, 0.6 ug/mL) used for the reactive oxygen species (ROS) assay. In both studies, OH-SeMet or Se-yeast promoted cell viability and attenuated cell apoptosis indicating no negative impact on cell growth. However, the Se-yeast sources showed considerably greater effectiveness than OH-SeMet. To quantify redox potential, confluent cells were treated with DCFDA, an indicator of ROS production. Briefly, IEC-6 cells were seeded into 96-well plates (4 $\times 10^4$ cells per well) with 3 replicates per treatment (Trt) and incubated with a fresh solution of DCFDA. The ROS production, in response to 30µM H_2O_2, was quantified by fluorescence emission at 485 nm. Data were analyzed with GLIMMIX procedure of SAS. In both Exp, Se inclusion in all Se-Trts was 0.3 ppm. In Exp 1, Trts included: Non treated (cells only), H_2O_2, H_2O_2 + 2mM Trolox (Vit. E analog), H_2O_2 + sodium selenite, H_2O_2 + OH-SeMet, H_2O_2 + Se-yeast product A, H_2O_2 + Se-yeast product B. Cells treated with H_2O_2 had 4.6-fold increase ($P<0.05$) in ROS production relative to non-H_2O_2 treated cells. Trolox reduced ROS 7.6-fold ($P<0.05$) relative to H_2O_2 only cells, OH-SeMet and sodium selenite did not ameliorate ($P>0.35$) the negative impact of H_2O_2 on ROS production. However, cells treated with H_2O_2 + Se-yeast product A or B reduced ROS production by 15% ($P=0.07$) and 32% ($P<0.01$), respectively, compared with cells treated with H_2O_2 only. To validate the redox potential of OH-SeMet, Exp 2 included a pure form of selenomethionine. Treatments in Exp 2. were similar to Exp 1 with the inclusion of H_2O_2 + seleno-L-methionine. Selenomethionine, either as a pure source or the hydroxy analog, did not prevent ($P>0.40$) H_2O_2-induced oxidative stress. However, Exp 2 confirmed the effectiveness of Se-Yeast products on reducing ROS production. More specifically, Se-Yeast product B (52% reduction, $P=0.04$) was superior to product A (32% reduction, $P=0.17$) in comparison to cells treated with H_2O_2 only. These findings suggest that the type of organic Se source differs in their ability to regulate intestinal redox potential, with Se-yeast sources being more effective than hydroxy analogs. Selenomethionine content in Se-yeast does not appear to be the main driver in reducing ROS in intestinal cells, suggesting that livestock fed Se-yeast can support better intestinal health during oxidative stress.

Key words: Intestinal epithelial cells, oxidative stress, selenium-enriched yeast, selenomethionine