N-Acetyl-L-cysteine enhances the effect of selenium nanoparticles on cancer cytotoxicity by increasing the production of selenium-induced reactive oxygen species

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Figure S1. Body weight and cancer cell number in peritoneal cavity of mice. Experiment. To inspect the development of body weight and cancer cells in the mice peritoneal cavity post the inoculation, healthy mice were i.p. injected with saline as a control, or two millions of viable H22 cells as a moderate tumor model. (A) Body weight. (B) Viable cells. * P < 0.05, *** P < 0.001, compared to control.



Figure S2. Time effect of SeNP-triggered apoptosis of H22 cells in peritoneal cavity of mice. Experiment. Highly malignant H22 model mice (n = 4/group) were i.p. injected with saline as control or SeNPs (4 mg Se/kg); mice were sacrificed at the indicated time post-injection, and cells were collected. Graph showing viable cells. Data are presented as the mean \pm SEM. * P < 0.05, ** P < 0.01, compared to control.



Figure S3. Comparison between GSH and Cys in driving SeNPs to produce ROS at a molar ratio of 20 and 80 (GSH/Se or Cys/Se). (A) ROS production. (B) ROS formation kinetics. Experiments were carried out in 50 mM PBS (1 mM EDTANa2, pH 7.5) at 37°C in the presence or absence of 50 μ M DCFH-DA. Data are presented as the mean of two replicates; the error bar represents the range. In most data points, the range was smaller than the symbol. The vehicle control has been subtracted from the treatments. * P < 0.05, compared to GSH/SeNPs group.