Supporting Information

Olm et al. 10.1073/pnas.0902204106

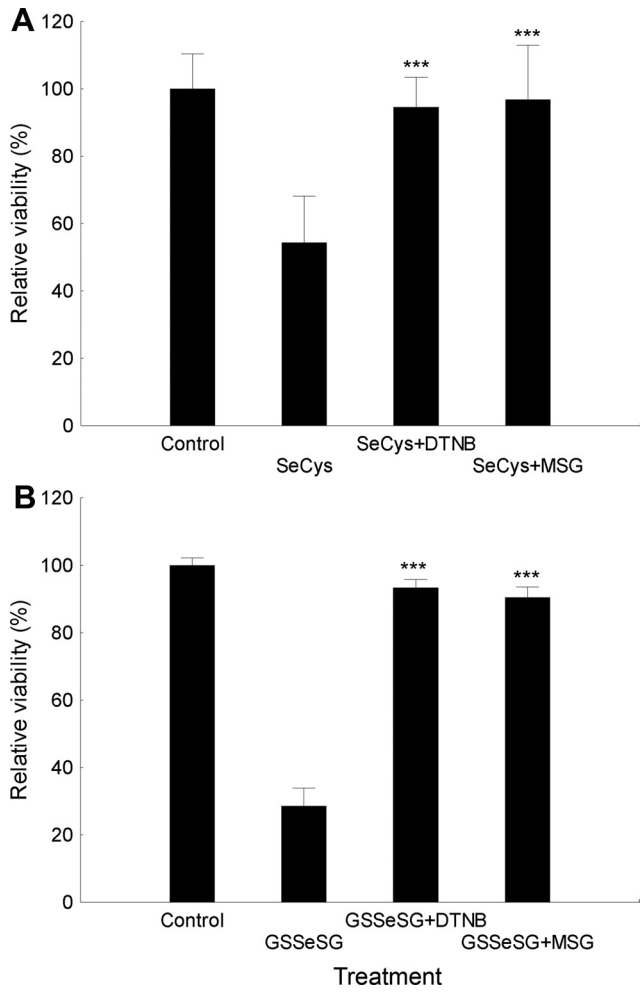

Fig. S1. Cytotoxicity of redox-active selenocompounds is inhibited by DTNB and MSG. (*A*) H157 cells treated with seleno-L-cystine (10 μM) and in combination with DTNB (500 μM) or MSG (60 μM). (*B*) H157 cells treated with GSSeSG (3 μM) and in combination with DTNB (500 μM) or MSG (60 μM). ***$P < .001$ in relation to treatment with selenocompounds.

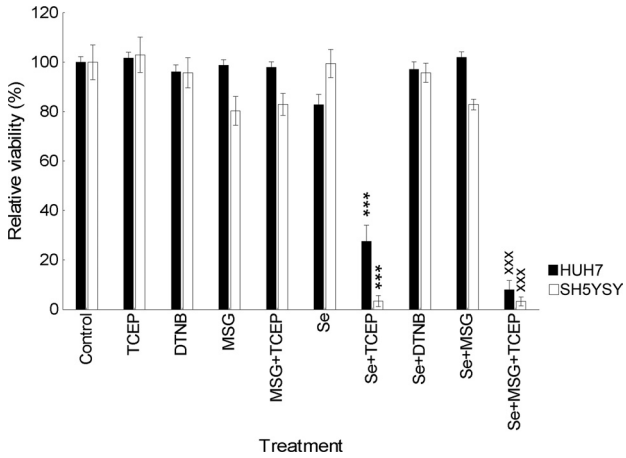

Fig. S2. Hepatoma and neuroblastoma cells display similar patterns in response to selenite compared with lung cancer cells when extracellular redox state or x_c^- ystine antiporter activity is modulated. Viability was measured by XTT in HUH7 hepatoma or SH5YSH neuroblastoma cells treated with 5 μM selenite for 20 h. TCEP (75 μM) was used as an extracellular reductant, and DTNB (500 μM) was used as an extracellular thiol scavenger. MSG (60 mM) was used as an inhibitor of the x_c^- cystine/glutamate antiporter. TCEP (75 μM) with MSG was used as an extracellular thiol impact control. Error bars display $\pm$ 0.95 confidence intervals. ***(^{xxx})$P < .001$, *in relation to selenite, ^{x}in relation to selenite + MSG.

Table S1. Selenite sensitivity and selenium uptake by human lung cancer cell

Cell line	Type	IC-50 selenite, μM	Selenium uptake, ng/mg total protein
H157	Non–small cell lung carcinoma	4	280 $\pm$ 6
U2020	Small cell lung carcinoma	6	60 $\pm$ 9
H611	Non–small cell lung carcinoma	22	NA ($<$10)