Supplemental Material for

NANO-LIQUID CHROMATOGRAPHY-ORBITRAP MS-BASED QUANTITATIVE PROTEOMICS REVEALS DIFFERENCES BETWEEN THE MECHANISMS OF ACTION OF CARNOSIC ACID AND CARNOSOL IN COLON CANCER CELLS

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Supplemental Material, Table S6. Phosphorylated peptides detected in HT-29 cells after the treatment with different concentrations of CA and CS at different times.





Figure S1. Chemical structures of CA (A) and CS (B).



Figure S2. Scatter plot of mRNA versus protein expression ratios (log2) of HT-29 cells exposed to GI50 concentration of CA for 24 h.



Figure S3. Top ten molecular and cellular functions significantly over-represented in protein datasets obtained at 24 h with different concentrations of CA (**A**, GI50; **B**, TGI; **C**, LC50) or CS (**D**, GI50; **E**, TGI; **F**, LC50). Fisher's exact test was used to calculate a p-value determining the probability that the association between the genes in the dataset and the canonical pathway is explained by chance.



Figure S4. Protein levels of HSPA5 after treatment of HT-29 cells with different concentrations of CA (GI50, TGI, LC50) or control (0.2% DMSO) for 24 h. (**A**) Blots showing HSPA5 and β -actin levels from forty micrograms of protein loaded per lane. (**B**) Expression ratio of HSPA5 normalized to β -actin from three independent experiments. Bars represent mean \pm SEM (* indicates significant differences between the treated and control samples as determined by t-test, p-value < 0.05).



Figure S5. mRNA levels of PSMC1 normalized to GAPDH in HT-29 cells exposed to TGI concentration of CS or control (0.2% DMSO) for 2, 6, and 24 hours. Bars represent mean \pm SEM of three independent experiments (* indicates significant differences between the treated and control samples as determined by t-test, p-value < 0.05).