Supplementary Materials: Docosahexaenoic acid enhances Oxaliplatin-induced autophagic cell death via the ER stress/SESN2 pathway in colorectal cancer

Soyeon Jeong, Dae Yeong Kim, Sang Hee Kang, Hye Kyeong Yun, Jung Lim Kim, Bo Ram Kim, Seong Hye Park, Yoo Jin Na, Min Jee Jo, Yoon A Jeong, Bu Gyeom Kim, Dae-Hee Lee and Sang Cheul Oh

Figure S1. Other ω3-PUFAs or ω6-PUFAs have no effect with Oxaliplatin. (A-C) Cell viability was measured by WST-1 after treatment with 10 µM Oxaliplatin and 10 µM ω6-PUFAs arachidonic acid (A) or ω3-PUFAs alpha-linolenic acid (B) and eicosapentaenoic acid (C).
Figure S2. DHA enhances Oxaliplatin-induced autophagy. (A) HCT116 cells were treated with Oxaliplatin and DHA treatment and p62 expression was observed. (B) Formations of GFP-LC3 puncta following Oxaliplatin and DHA treatment were analyzed via confocal microscopy (Scale Bar, 10 µm). (C) HCT116 cells were exposed to Oxaliplatin and DHA with or without CQ or rapamycin for 24, and 48 h. The autophagic cells were analyzed by flow cytometry and quantified. (D) The protein levels of p62 and LC3 were evaluated by western blotting in DLD-1 (upper) and SW620 (lower) cells.
Figure S3. Combinatorial treatment with DHA and Oxaliplatin reduces viability and results in SESN2-mediated autophagy in PDC cells. (A-B) PDC cells were exposed to Oxaliplatin and DHA, and analyzed via WST-1 assay (A), and qRT-PCR (B). ***, p < 0.001.
The densitometry readings/intensity ration of all western blot bands. Unfortunately, we do not have a whole blot showing all molecular weight markers because we cut a single membrane to identify expression of several molecules. So we only have a fragmented band film with a molecular weight.