Supporting information

Table S1 Fatty acid composition of DHA-PL and EPA-PL. Values are expressed as mean ± SEM (n=3).

Figure S1 Cell viability of 3T3-L1 adipocytes treated with different concentrations of DHA-PL and EPA-PL in the absence or presence of TNF-α, EX-527 or compound C. The cell viability of 3T3-L1 adipocytes treated with (A) DHA-PL and (B) EPA-PL was determined using WST-1 assay. Values are expressed as mean ± SEM (n = 4).

Figure S2 Co-treatment of compound C does not alter the activation of SIRT1 by DHA-PL or EPA-PL treatment. (A) DHA-PL and EPA-PL enhanced SIRT1 deacetylase activity in TNF-α-treated 3T3-L1 adipocytes, which was not abolished by co-treatment of compound C. (B) DHA-PL and EPA-PL increased SIRT1 protein expression in TNF-α-treated 3T3-L1 adipocytes, which was not blocked by co-treatment of compound C. Data normalization was accomplished using the endogenous reference β-actin. Values are expressed as mean ± SEM (n = 4), $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ as compared to control group; $^{*}P < 0.05$, $^{**}P < 0.01$ as compared to TNF-α group.

Figure S3 Effects of DHA-PL and EPA-PL on mRNA expression of lipolysis genes in 3T3-L1 adipocytes in the absence or presence of TNF-α. The mRNA expression of (A) C/EBP α, (B) C/EBP β, (C) PPAR γ and (D) SREBP-1c in 3T3-L1 adipocytes were measured by real-time PCR. Data normalization was accomplished using the endogenous reference β-actin. Values are expressed as mean ± SEM (n = 4), $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ as compared to control group; $^{*}P < 0.05$, $^{**}P < 0.01$ as compared to TNF-α group.

Figure S4 Glucose uptake of 3T3-L1 adipocytes treated with various concentrations of DHA-PL and EPA-PL in the absence or presence of TNF-α. The glucose uptake of 3T3-L1 adipocytes treated with (A) DHA-PL and (B) EPA-PL were evaluated by using glucose uptake cell-based assay kit following the manufacturer's instruction and normalized by the amount of protein. Values are expressed as mean ± SEM (n = 4), $^{\#}P < 0.05$, $^{\#\#}P < 0.01$ as compared to control group; $^{*}P < 0.05$, $^{**}P < 0.01$ as compared to TNF-α group.

Table S1 Fatty acid composition of DHA-PL and EPA-PL.

Fatty acid[&]	DHA-PL	EPA-PL
C14:0	3.11 ± 0.04	4.34 ± 0.01
C16:0	16.85 ± 0.43	13.78 ± 0.23
C16:1	2.67 ± 0.04	4.27 ± 0.02
C18:0	13.45 ± 0.35	11.58 ± 0.57
C18:1	7.66 ± 0.30	6.15 ± 0.31
C18:2	0.12 ± 0.01	0.45 ± 0.02
C18:3 n-3	0.24 ± 0.01	0.68 ± 0.01
C20:1	5.68 ± 0.21	7.88 ± 0.26
C20:2	0.54 ± 0.01	-
C20:3	0.11 ± 0.01	-
C20:4 (AA)	2.43 ± 0.08	4.79 ± 0.18
C20:5 (EPA)	8.88 ± 0.25	41.96 ± 0.89
C22:6 (DHA)	38.26 ± 0.65	4.12 ± 0.42
n-3 PUFAs	47.38	46.76

[&] Note: %/total fatty acids. Values are expressed as mean ± SEM (n = 3).

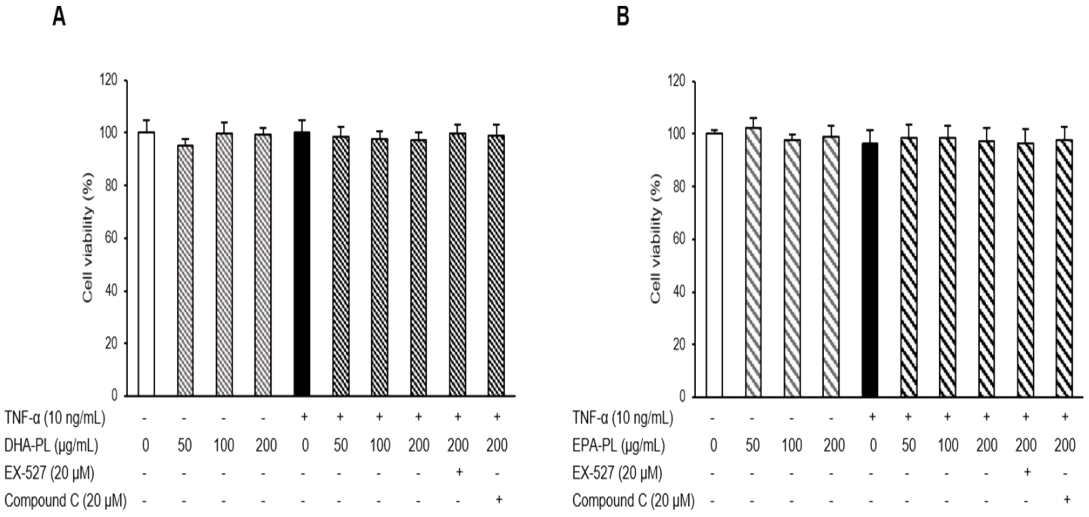

Figure S1 Cell viability of 3T3-L1 adipocytes treated with different concentrations of DHA-PL and EPA-PL in the absence or presence of TNF-*α*, EX-527 or compound C.

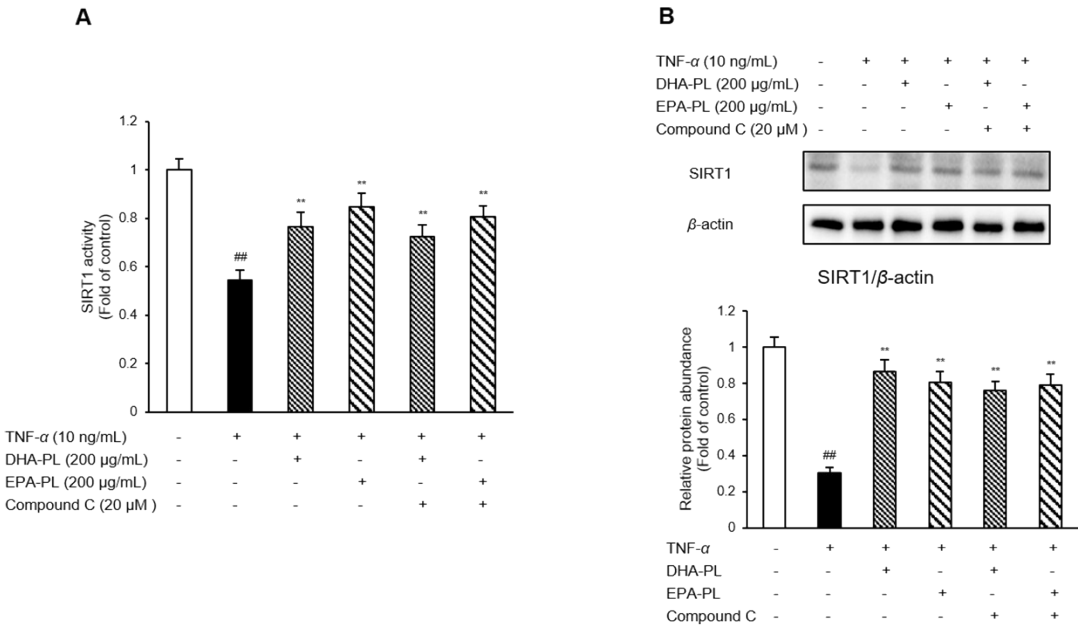

Figure S2 Co-treatment of compound C does not alter the activation of SIRT1 by DHA-PL or EPA-PL treatment.

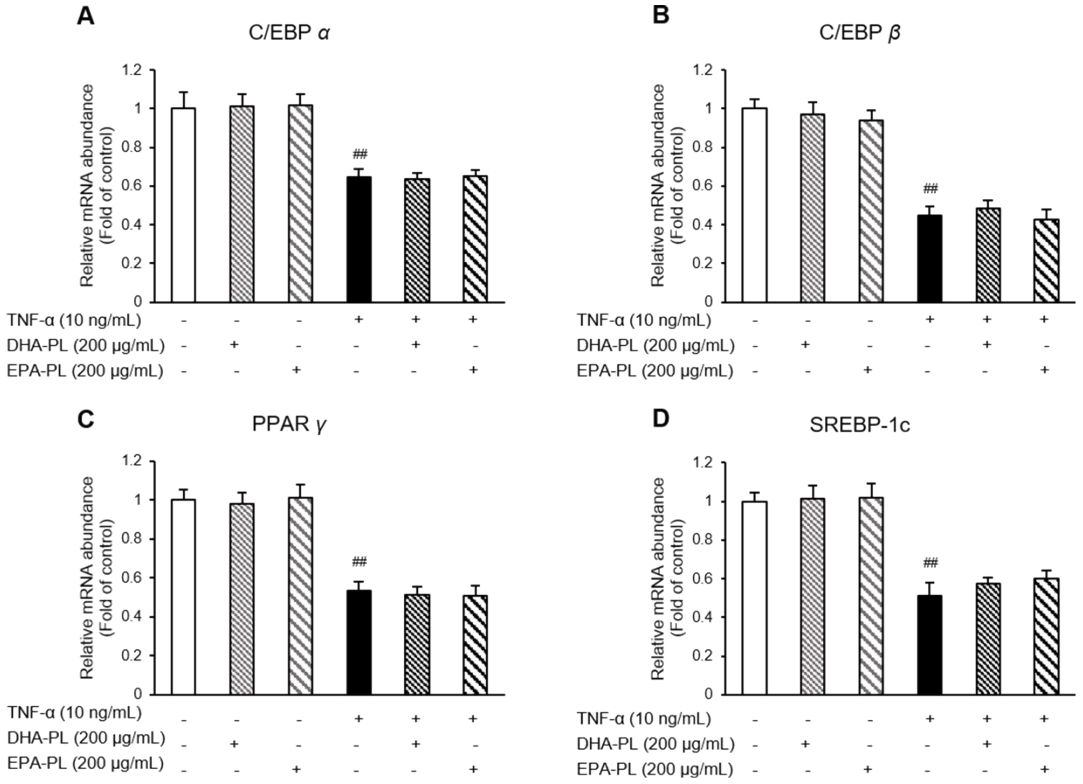

Figure S3 Effects of DHA-PL and EPA-PL on mRNA expression of lipolysis genes in 3T3-L1 adipocytes in the absence or presence of TNF-α.

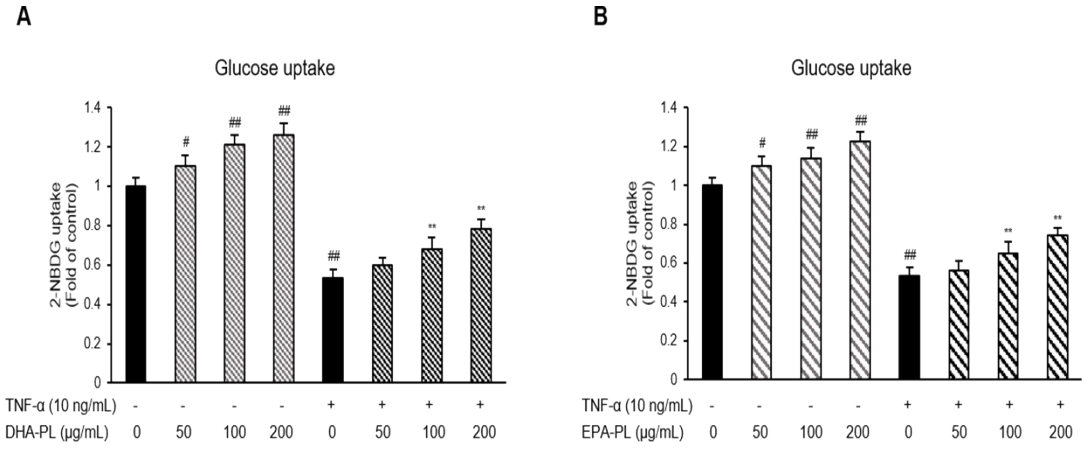

Figure S4 Glucose uptake of 3T3-L1 adipocytes treated with various concentrations of DHA-PL and EPA-PL in the absence or presence of TNF-*α*.