Supplemental Figure Legends

Figure S1. Suppression of ER stress-induced apoptosis by 3'-deoxyadenosine. BEAS-2B cells were exposed to TM or TG with or without 3DA and subjected to phase-contrast microscopy (A) and assessment of cell death (B).

Figure S2. Suppression of ER stress-induced apoptosis by post-treatment with 3'-deoxyadenosine. NRK-52E cells were pretreated with TM or TG for 8 h, treated with 3DA together with TG or TM and subjected to microscopic analysis (A) and assessment of cell death (B).

Figure S3. Lack of involvement of A_1/A_2 adenosine receptors in the anti-apoptotic effect of 3'-deoxyadenosine. NRK-52E cell were treated with TM (A) or TG (B) together with 3DA in the absence or presence of 1 μM DPSPX, and subjected to phase-contrast microscopy.

Figure S4. Suppression of ER stress markers by post-treatment with 3'-deoxyadenosine. NRK-52E cells were pretreated with TM or TG for 12 h, treated with 3DA for additional 10 h and subjected to Northern blot analysis.

Figure S5. Lack of suppression of *SEAP* mRNA by 3'-deoxyadenosine. SM/SV-SEAP cells were treated with 20 μg/ml 3DA for up to 48 h, and the level of

SEAP mRNA was evaluated.

Figure S6. Lack of suppression of *EF-1α* and *GAPDH* mRNAs by 3'-deoxyadenosine. Cells were stimulated for 8 h with TG in the absence or presence of 3DA, and expression of *GRP78*, *EF-1α* and *GAPDH* was evaluated by Northern blot analysis.

Figure S7. Lack of suppression of luciferase driven by viral and house-keeping gene promoters in 3'-deoxyadenosine-treated cells. NRK-52E cells were transiently transfected with pEF-1α-Luc (left) or pCMV-Luc (right), treated with or without 3DA for 12 h and subjected to chemiluminescent assay.

Figure S8. Phosphorylation of eIF2α by salubrinal. NRK-52E cells were treated with salubrinal, and phosphorylation of eIF2α was examined.

Figure S9. Establishment of NRK/PERK-DN cells. (A) NRK-52E cells were stably transfected with a dominant-negative mutant of PERK (PERK-DN), and NRK/PERK-DN cells were established. Mock-transfected NRK/Neo cells and NRK/PERK-DN cells were subjected to Northern blot analysis of *PERK*. (B) NRK/Neo cells and NRK/PERK-DN cells were treated with TM or TG and subjected to Western blot analysis of phosphorylated PERK. (C) NRK/Neo cells and NRK/PERK-DN cells were transfected with pCHOP-Luc, treated with SubAB (10 ng/ml) for 12 h and

subjected to luciferase assay.

Figure S10. Lack of attenuation of ER stress by the treatment with 3'-deoxyadenosine: assessment by ESTRAP assay. NRK-52E cell were transiently transfected with pSEAP2-Control and pGL3-Control that introduce *SEAP* and *luciferase* under the control of the SV40 promoter. The cells were pretreated with or without 3DA for 8 h, fed with fresh medium containing TG and incubated for 4 h. After the incubation, culture media and cells were subjected to SEAP assay and luciferase assay, respectively. The values of SEAP (ESTRAP activity) were normalized by the levels of luciferase (Luc).

Figure S11. Up-regulation of *MCP-1* mRNA by 3'-deoxyadenosine in differentiating preadipocytes. 3T3-L1 preadipocytes were treated with differentiation medium (10 μg/ml insulin, 0.25 μM dexamethasone and 500 μM, 3-isobutyl-1-methylxanthine; IDI) in the absence or presence of 100 μg/ml 3DA for 12 h, and expression of *MCP-1* was evaluated by Northern blot analysis. The level of 28S ribosomal RNA is shown at the bottom as a loading control.