Figure S1. Establishment of radio- and chemoresistant breast cancer cell lines. (A) MCF7-WT and MCF7-IRR cells were exposed to IR treatment, and cell sensitivity to IR was evaluated using MTT assay. (B) T47D-WT and T47D-IRR cells were exposed to IR treatment, and cell sensitivity to IR was evaluated using MTT assay. (C) MCF7-WT and MCF7-DR cells were treated with doxorubicin for 48 h, and cell sensitivity to doxorubicin was evaluated using MTT assay. (D) T47D-WT and T47D-DR cells were treated with doxorubicin for 48 h, and cell sensitivity to doxorubicin was evaluated using MTT assay. (D) T47D-WT and T47D-DR cells were treated with doxorubicin for 48 h, and cell sensitivity to doxorubicin was evaluated using MTT assay. Data represent three independent experiments. WT, wild-type; IRR, ionizing radiation-resistant; IR, ionizing radiation; DR, doxorubicin-resistant.



Figure S2. NHEJ efficiency of WT, radio- and chemoresistant breast cancer cell lines. (A-D) Quantification of GFP events generated by NHEJ in (A) MCF7-WT and MCF7-IRR, (B) T47D-WT and T47D-IRR, (C) MCF7-WT and MCF7-DR, and (D) T47D-WT and T47D-DR cells. The GFP events in the resistant groups were normalized to those in the respective WT cells. \*\*\*P<0.001 vs. WT. WT, wild-type; GFP, green fluorescent protein; IRR, ionizing radiation-resistant; DR, doxorubicin-resistant; NHEJ, non-homologous end joining.



Figure S3. MV increases the rate of apoptosis induced by IR/doxorubicin in breast cancer cells. Apoptosis was evaluated using a caspase-3/7 activity assay. (A and C) MV-Edm induced caspase-3/7 activity in (A) MCF7-IRR and (C) T47D-IRR cells exposed to IR. Cells were infected with MV-Edm at a MOI of 0, 0.1 or 0.5, followed by IR treatment. Caspase-3/7 activity was normalized to that of cells treated with 0 Gy for each virus dose group. (B and D) MV-Edm induced caspase-3/7 activity in (B) MCF7-DR and (D) T47D-DR cells exposed to doxorubicin. Cells were infected with MV-Edm at a MOI of 0, 0.1 or 0.5, followed by IR treatment. Caspase-3/7 activity in (B) MCF7-DR and (D) T47D-DR cells exposed to doxorubicin. Cells were infected with MV-Edm at a MOI of 0, 0.1 or 0.5, followed by doxorubicin treatment. Caspase-3/7 activity was normalized to that of cells treated with 0  $\mu$ M doxorubicin (DMSO) for each virus dose group. \*\*P<0.01. Data are presented as the mean ± SEM from three independent experiments. MV, measles virus; IR, ionizing radiation; MOI, multiplicity of infection; DR, doxorubicin-resistant; IRR, ionizing radiation-resistant; Edm, Edmonston-B.

