Tumor-derived extracellular vesicles disrupt the blood-brain barrier endothelium following high-frequency irreversible electroporation

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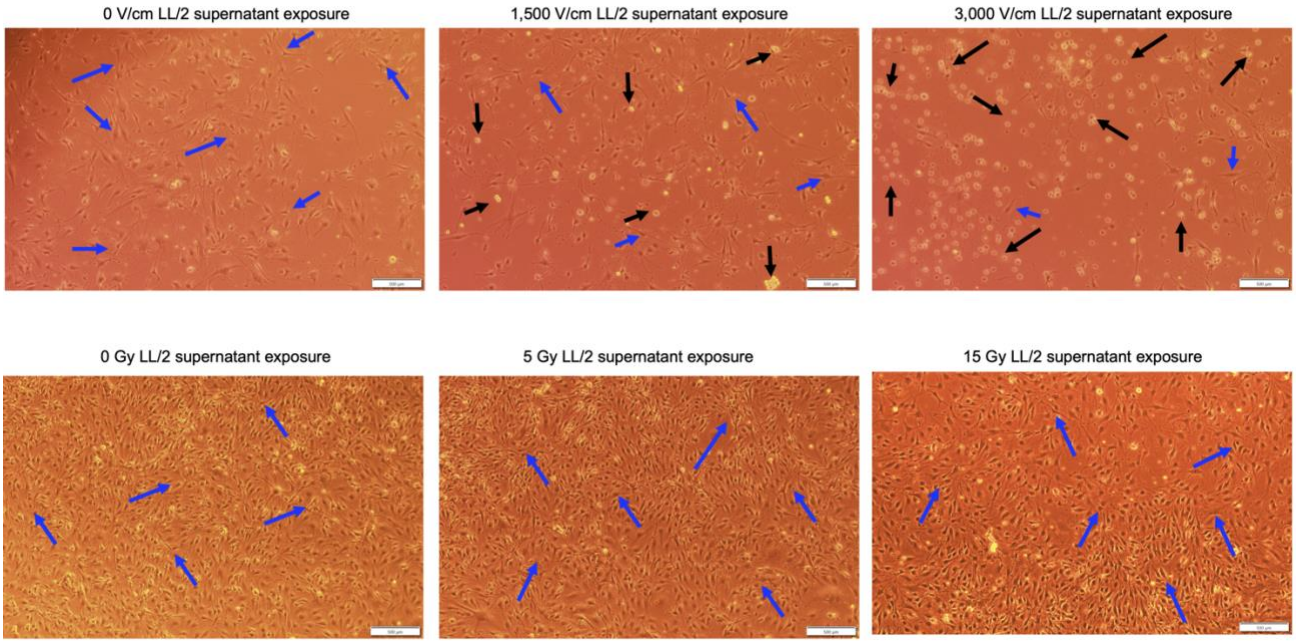
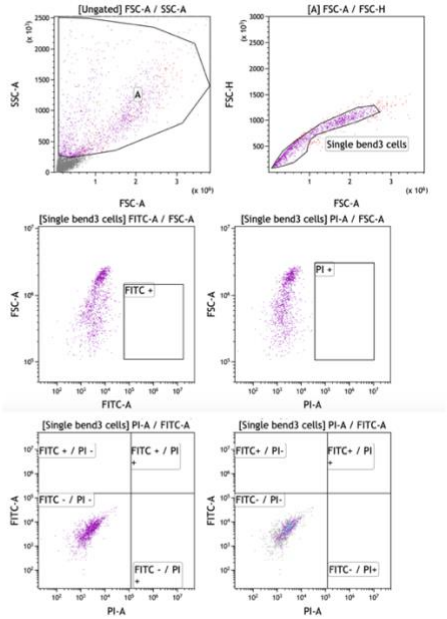


Supplemental Figure 1. Representative images of disruption of cerebral endothelial cell morphology and monolayer integrity induced by supernatants of H-FIRE- and RT-treated LL/2 Lewis lung carcinoma cells. LL/2 Lewis lung carcinoma cells were treated with H-FIRE doses of 0, 1,500, and 3,000 V/cm and RT doses of 0, 5, and 15 Gy. Immediately following treatment, supernatants were collected and cerebral endothelial cell monolayers (bEnd.3) were exposed to post-H-FIRE or post-RT tumor cell supernatants for 30 minutes. The top row depicts representative images of bEnd.3 monolayers following 30 minute exposure with supernatants of H-FIRE-treated LL/2 Lewis lung carcinoma cells (10X magnification). The bottom row depicts representative images of bEnd.3 monolayers following 30 minute exposure with supernatants of RT-treated LL/2 Lewis lung carcinoma cells (10X magnification). Blue arrows identify representative morphology of adhered bEnd.3 cells, while black arrows identify cells that have lost adherence post-supernatant exposure.



Supplemental Figure 2. Flow cytometry gating strategy for Annexin V/PI assay of bEnd.3 cells exposed to supernatants of 0 V/cm or 3,000 V/cm H-FIRE-treated F98 or LL/2 cells.