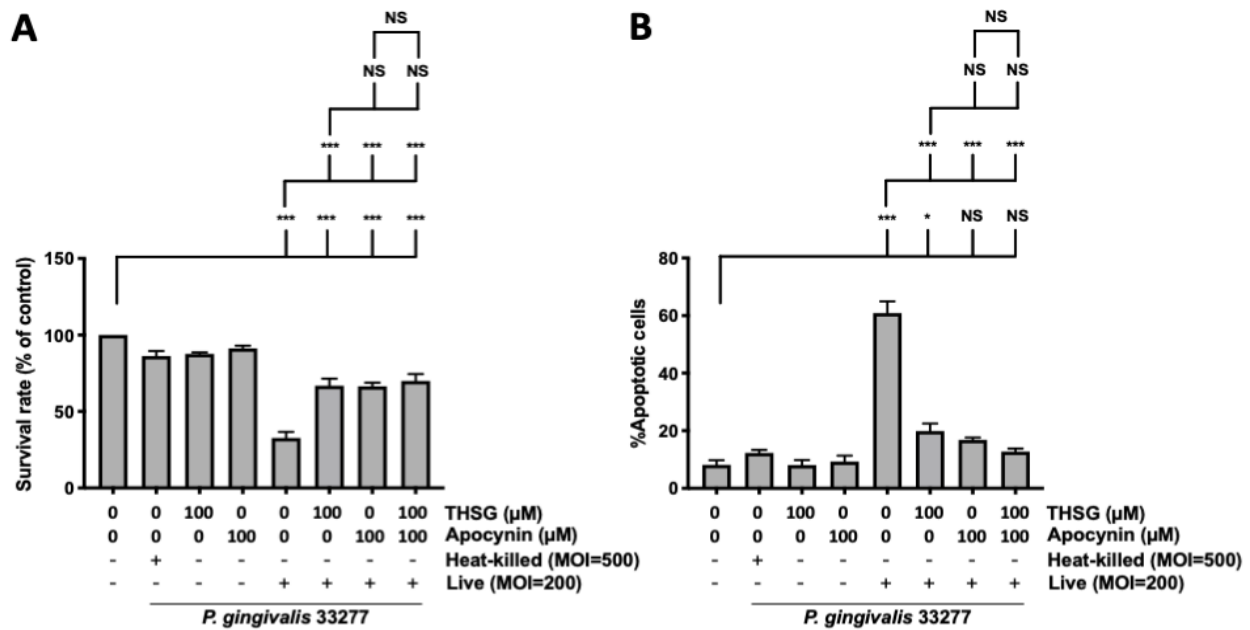


**Supplementary Figure S1.** Apocynin optimum dosage determination in brain endothelial cells. bEnd.3 cells were cultured with apocynin at 0, 50, 100, 200, and 300  $\mu\text{M}$  before inoculating with *P. gingivalis* (heat-killed MOI 500 or live MOI 200). **(A)** The survival rate at 24 h post-infection was determined by MTT assay. **(B)** DCFH-DA (50  $\mu\text{M}$ ) was added to the cells before the infection to examine the intracellular production of ROS. The fluorescence intensity of DCF was calculated using a flow cytometer and presented as a gated histogram in the percentage of the P2 area. Data in the bar graph are shown as mean values  $\pm$  SEM ( $n = 4$ ). Significant difference of the control and apocynin 0  $\mu\text{M}$  group are presented as \*,  $p < 0.05$ ; \*\*,  $p < 0.01$ ; \*\*\*, and  $p < 0.001$ . NS: not significant.



**Supplementary Figure S2.** Effect of THSG and apocynin co-treatment on *P. gingivalis*-stimulated cell death in brain endothelial cells. bEnd.3 cells were treated for 2 h with 100 μM THSG, 100 μM apocynin, or the combination of THSG and apocynin before being infected with heat-killed (MOI 500) or live (MOI 200) *P. gingivalis* for another 90 minutes. **(A)** Twenty-four hours after the infection, the survival rate was quantified by MTT assay and presented as a percentage of the control. **(B)** Annexin V FITC/PI was used to stain the cells to determine the percentage of apoptotic cells. Cells were analyzed using a flow cytometer. Data in bar graphs are represented as means ± SEM ( $n = 4$ ). Significant difference of the control, infection with THSG 0 μM, infection with THSG 100 μM, and infection with apocynin 100 μM group are expressed as \*,  $p < 0.05$ ; \*\*,  $p < 0.01$ ; \*\*\*,  $p < 0.001$ . NS: not significant .