Figure S1. rHMGB1 recovers MHC-I expression. Expression of MHC-I in (A) HPV.PSV-infected HaCaT and (B) CaSki cells added with rHMGB1 via flow cytometry. *P<0.05. rHMGB, recombinant high mobility group box; MHC-I, major histocompatibility complex class I; HPV.PSV, human papillomavirus 16 pseudovirus; FSC-H, forward scatter height; MFI, mean fluorescence intensity.

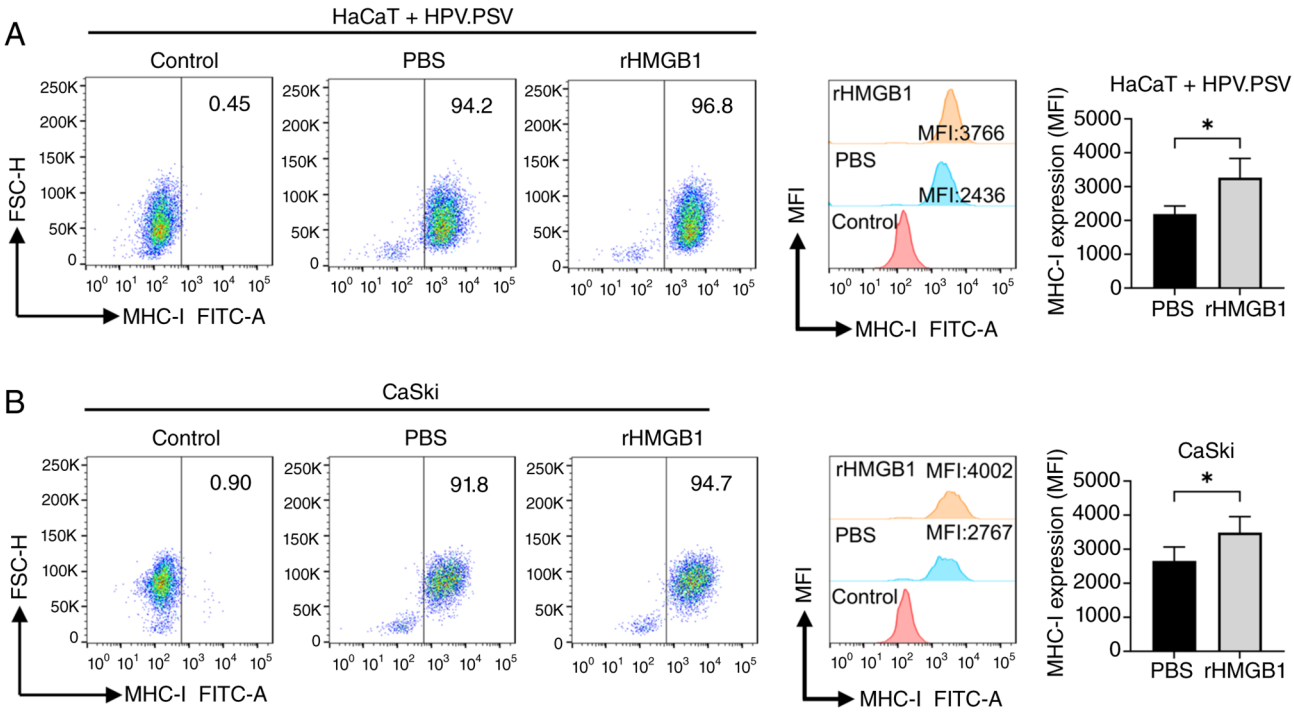

Figure S2. Hyperthermia does not influence tapasin expression. Western blot to detect the expression of tapasin in HPV.PSV-infected (A) HaCaT and (B) CaSki cells. Con, cells were not treated by hyperthermia. GAPDH was used as loading control. Tapasin, TAP (transporter associated with antigen processing) binding protein; HPV.PSV, HPV 16 pseudovirus; ns, not significant; Con, control.

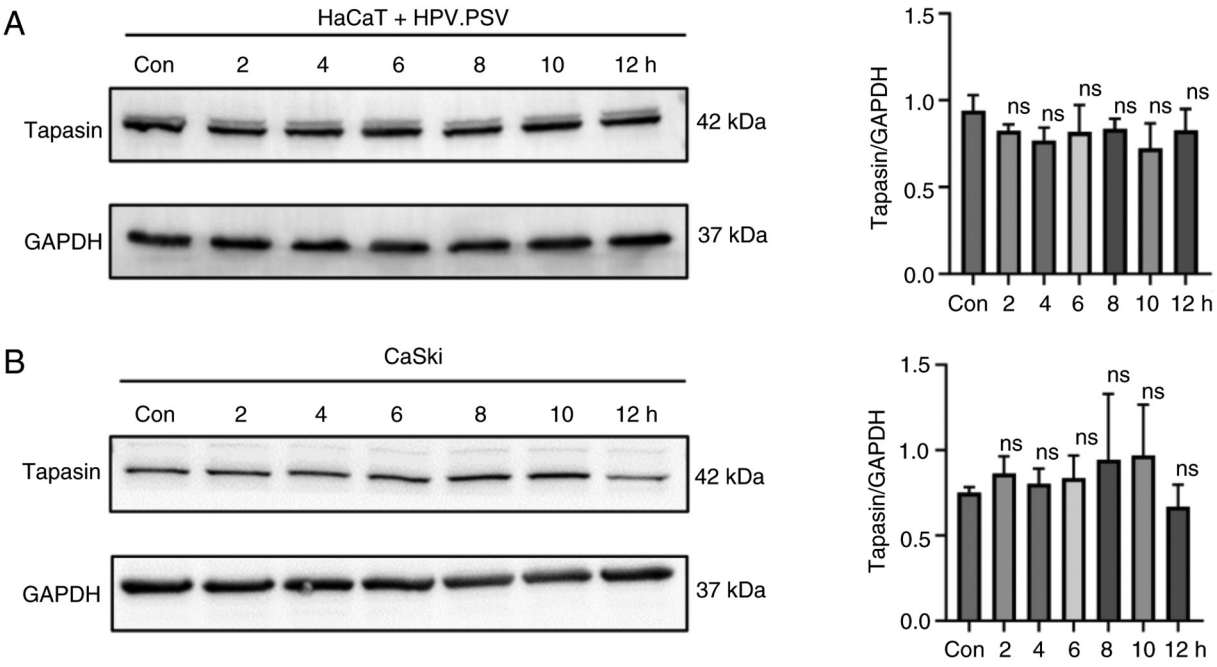

Figure S3. JNK pathway inhibitors and activators regulate affect the JNK signaling pathway. Western blotting was performed to detect the effect of different concentrations of inhibitor SP600125 on the JNK pathway in (A) HaCaT and (B) CaSki cells. Western blotting was performed to detect the effect of activator anisomycin on the JNK pathway in (C) HaCaT and (D) CaSki cells. *P<0.05, **P<0.01, ***P<0.001, ****P<0.0001 vs. 0. p-, phosphorylated; ns, not significant.

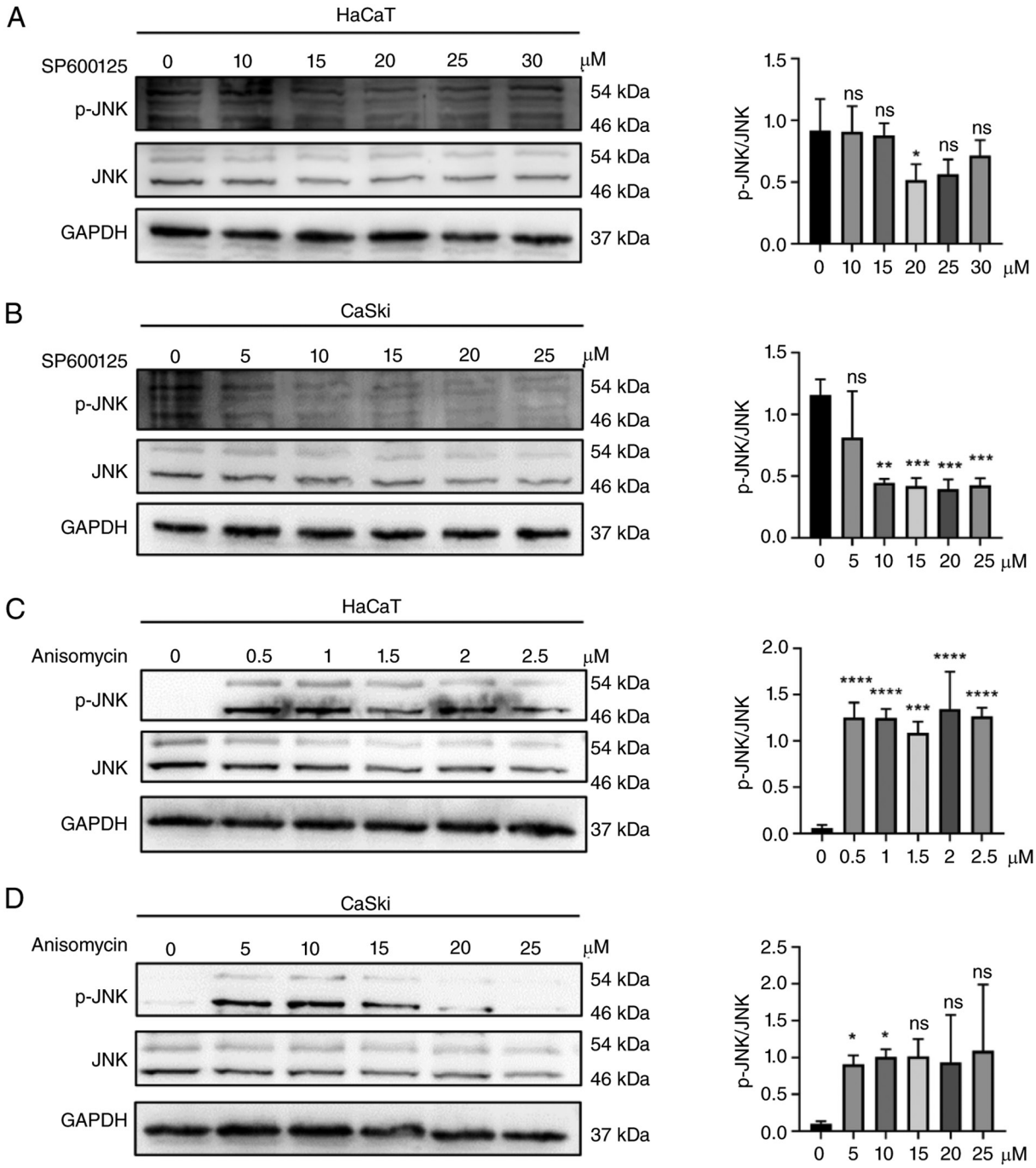

Figure S4. Hyperthermia does not influence CRM1 expression. Western blotting was performed to detect the expression of CRM1 in HPV.PSV-infected (A) HaCaT and (B) CaSki cells following hyperthermia. β-tubulin was used as loading control. CRM1, chromosome region maintenance 1; HPV.PSV, HPV 16 pseudovirus; ns, not significant; Con, control.