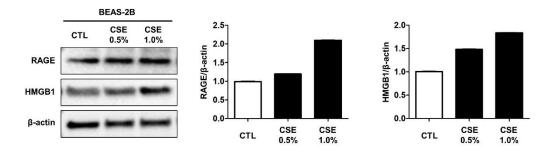
## **Data Supplement**

## Inhibition of RAGE attenuates cigarette smoke-induced lung epithelial cell damage via RAGE-mediated Nrf2/DAMP signaling

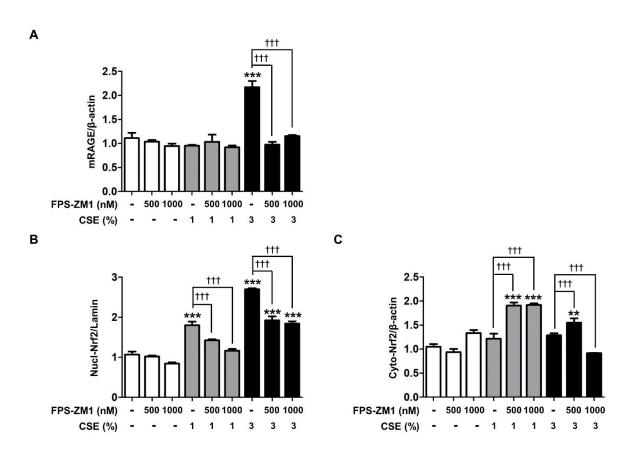
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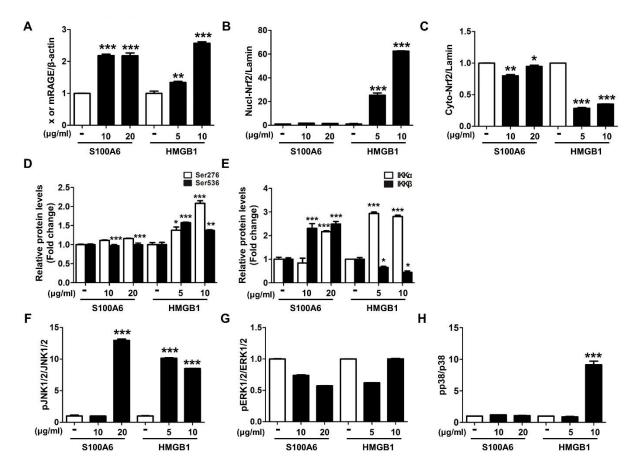
Supplementary files



Supplementary Figure 1. The RAGE and HMGB1 were determined by western blotting in 0.5% and 1.0% CSE-exposed BEAS-2B for 24h. Equal protein loading amounts were proved by  $\beta$ -actin. The corresponding density ratio was determined using Image J. CTL, no treatment.



**Supplementary Figure 2.** A549 cells were treated with CSE in presence of FPS-ZM1 for 8h. The Lamin A/C and β-actin were used for loading control of the nuclear protein and cytoplasmic protein, respectively. Values are presented as the mean  $\pm$ SE of three independent experiments performed in triplicate. CTL, no treatment; n.s, not significant. \*\*\*p<0.001 vs. CTL; †††p<0.001 vs. 1% CSE treatment; ‡‡‡p<0.001 vs. 3% CSE treatment. CTL: control.



**Supplementary Figure 3.** A549 cells were treated with S100A6 (10 and 20μg/ml) and HMGB1 (5 and 10 μg/ml) for protein expression of DAMP signaling. The Lamin A/C and  $\beta$ -actin were used for loading control of the nuclear protein and cytoplasmic protein, respectively. Values are presented as the mean ±SE of three independent experiments performed in triplicate. CTL, no treatment; n.s, not significant. \*p<0.05 and \*\*\*p<0.001 vs. no treatment