## Supplemental Figure

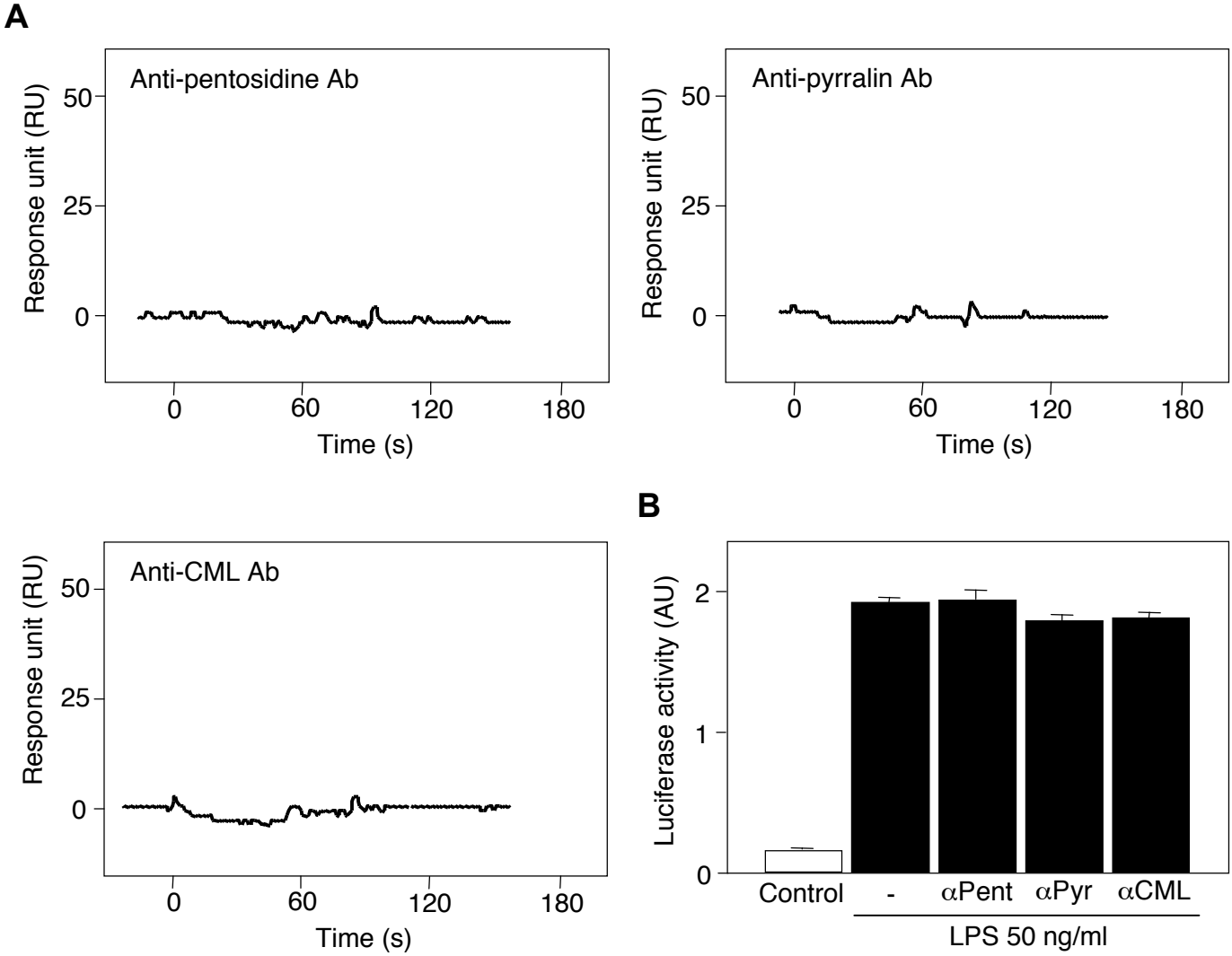


**FIGURE S1. *A*, SPR assays with anti-AGE antibodies.** After immobilization of LPS from *E. coli* 0111:B4 (Sigma L3024) on the surface of a CM5 research grade sensor chip, anti-AGE antibodies such as anti-pentosidine antibody (clone No. PEN-12, TransGenic Inc., Japan), anti-pyrraline antibody (clone No. H-12, TransGenic Inc.) or anti-$N^{\varepsilon}$-carboxymethyllysine (CML) antibody (clone No. 6D12, TransGenic Inc.) was injected at a concentration of 1.0 mg/ml. SPR assay was performed as described under Materials and Methods. ***B*, NFκB promoter assay with the RAGE expressing C6 glioma cells.** The cells were stimulated by LPS (*E. coli* 0111:B4 ) for 4 h. NFκB-luciferase assay was performed as described under Materials and Methods. Control, no stimulation; αPent, anti-pentosidine Ab (1 μg/ml) treatment; αPyr, anti-pyrralin Ab (1 μg/ml) treatment; αCML, anti-CML Ab (1 μg/ml) treatment. AU, arbitrary unit. Data are presented as mean ± SEM.

## Supplemental Figure

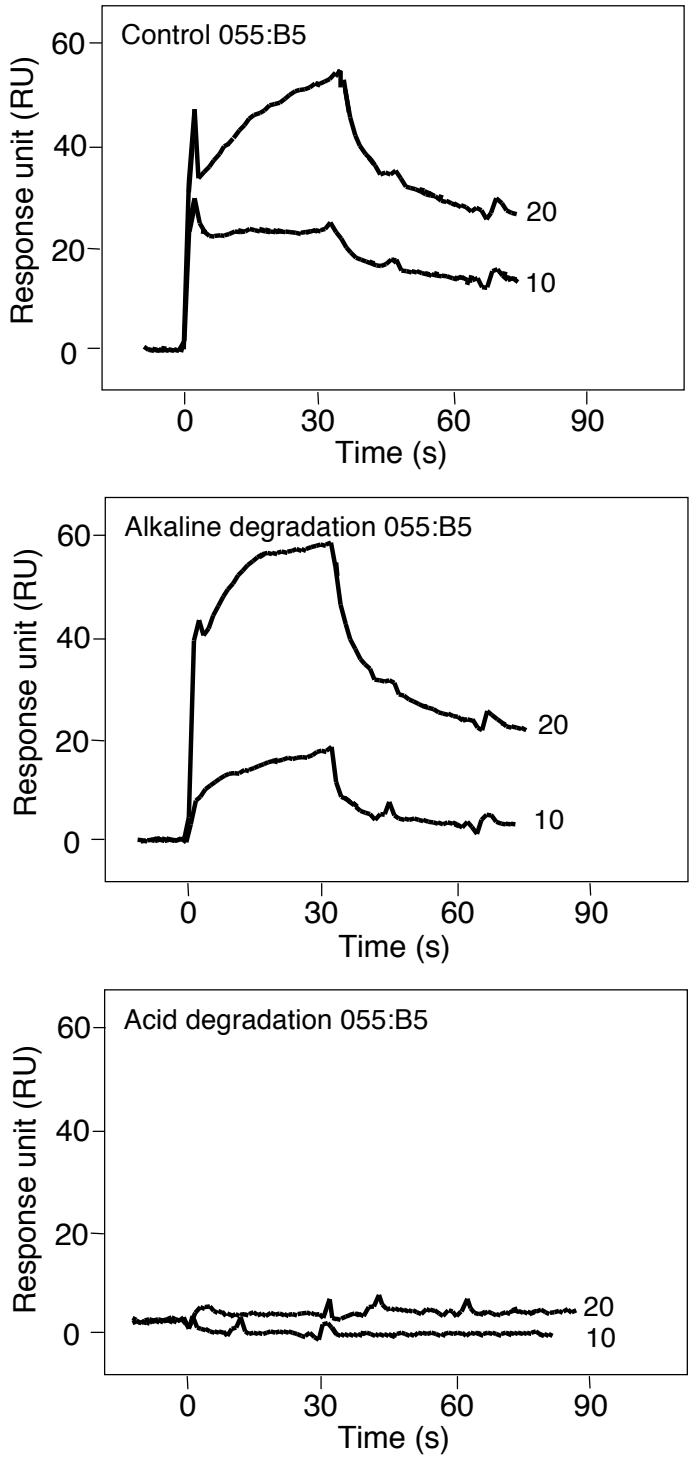


**FIGURE S2. SPR assays with degradated LPS.** After immobilization of purified esRAGE on the surface of a CM5 research grade sensor chip, non-degradated (Control), alkaline degradated (Alkaline degradation) or acid degradated (Acid degradation) LPS (E. coli 055:B5) was injected at a concentration of 10 or 20 ng/ml. The alkaline degradation of LPS was performed as previously described (33). Briefly, LPS (1.5 mg) was incubated in 0.03 N NaOH at 30°C for 20 min. After incubation, the reaction was stopped and neutralized by adding an equivalent amount of acetic acid solution. The acid degradation of LPS was carried out according to the previous experiment (34). Briefly, LPS (1.5 mg) was hydrolyzed with 0.1 M sodium acetate buffer (pH 4.4) at 100°C for 2 h. After incubation, the LPS solution was neutralized. Control LPS was incubated in 0.1 M sodium acetate buffer (pH 7.0) at 30°C for 20 min.

## Supplemental Figure

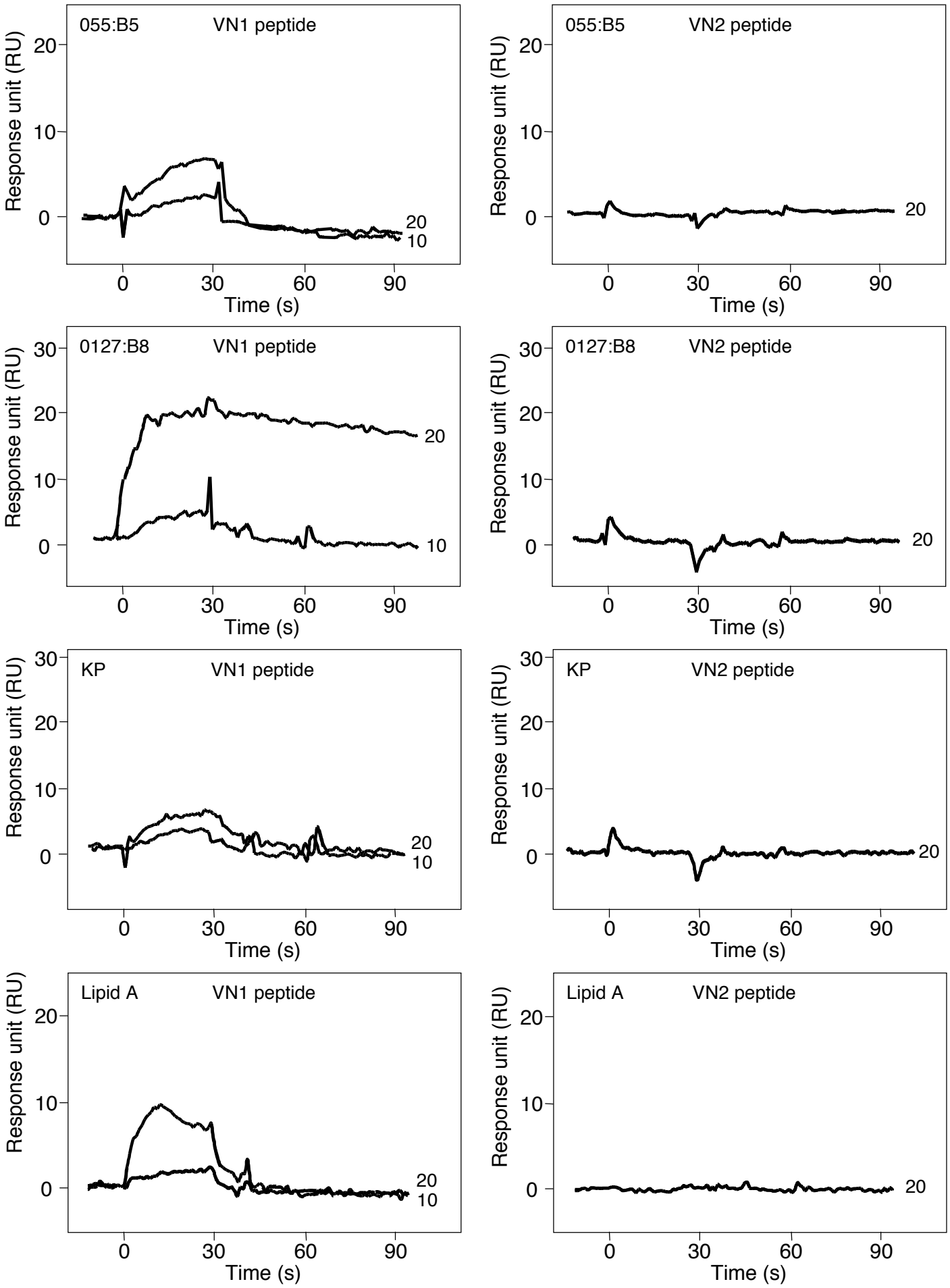


**FIGURE S3. SPR assays using synthetic peptides.** Synthetic peptides of human RAGE protein were employed to identify potential LPS-binding sites of RAGE. After immobilization of VN1 peptide (KGAPKKPPQRLEWKLN) or VN2 peptide (WKLNTGRTEAWKVLSPQG) on the surface of a CM5 research grade sensor chip, LPS from *E. coli* 055:B5 (055:B5), *E. coli* 0127:B8 (127:B8), *Klebsiella pneumonia* (KP), or 3-deoxy-D-manno-octulosonic acid $(KDO)_2$-lipid A (Lipid A) was injected at a concentration of 10 or 20 ng/ml.

## Supplemental Figure

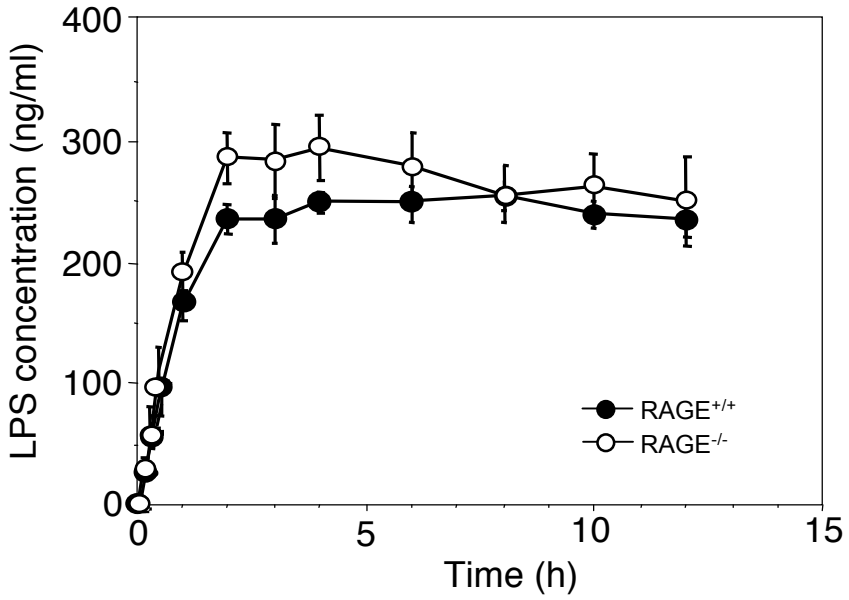


**FIGURE S4. Plasma concentrations of LPS.** FITC-labeled LPS (50 mg/kg) were i.p. injected. Samples were taken at the indicated time points after the injection. Data are presented as mean±SEM.

## Supplemental Figure

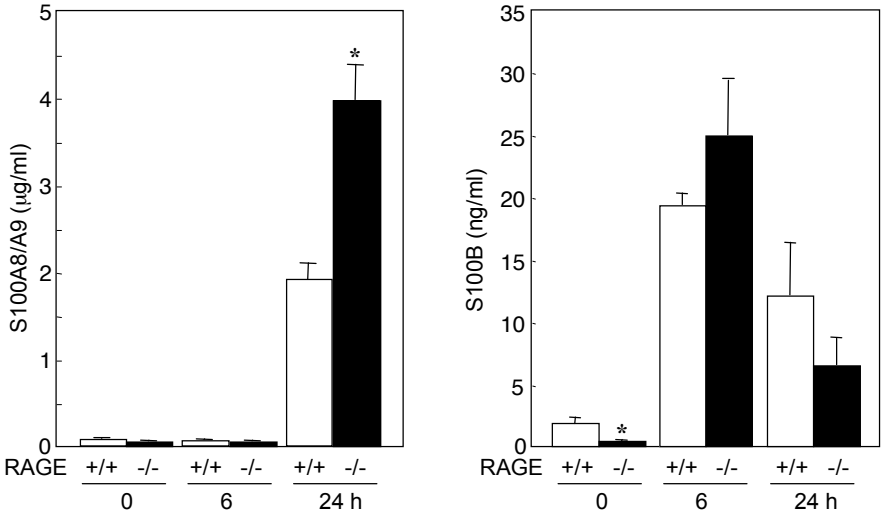


**FIGURE S5. Serum levels of S100A8/A9 and S100B.** Serum concentration of S100A8/A9 and S100B were measured with Immundiagnostik AG (Germany) and Uscn Life Science Inc. (China) ELISA systems, respectively.

## Supplemental Figure

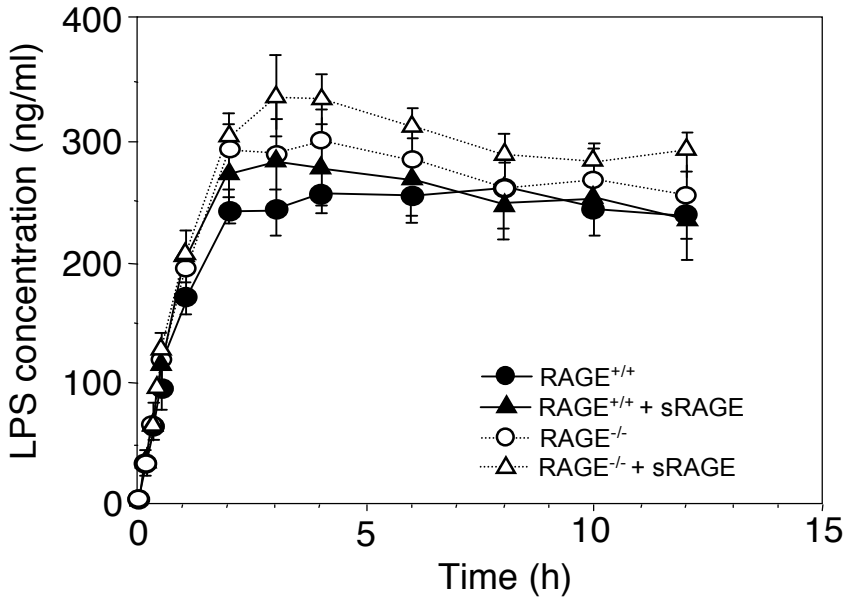


**FIGURE S6. Plasma concentrations of LPS.** FITC-labeled LPS (50 mg/kg) were i.p. injected. Samples were taken at the indicated time points after the injection. Data are presented as mean±SEM.