TLR4/MyD88/NF-κB signaling in the rostral ventrolateral medulla is involved in the depressor effect of candesartan in stress-induced hypertensive rats

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Supporting Information

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Figure S1. Chronic intracerebroventricular infusion of Toll-like receptor (TLR) 4 inhibitor VIPER attenuated the increases on phosphorylated nuclear factor κB subunit P65 (P65) expression in the rostral ventrolateral medulla (RVLM) of stress induced hypertension (SIH) rats. Values are presented as mean ± SE, n = 6. **P < 0.01 vs. control + CP; #P < 0.05 vs. SIH + CP (One-way ANOVA with turkey’s post hoc test). CP: control peptide; VIPER: TLR4 inhibitor; p-p65: phosphorylated P65.
Figure S2. Chronic intracerebroventricular infusion of AT$_1$R antagonist candesartan (CAN) attenuated the increases on phosphorylated nuclear factor κB subunit P65 (P65) expression in the rostral ventrolateral medulla (RVLM) of stress induced hypertension (SIH) rats. Values are presented as mean ± SE, n = 6. **$P < 0.01$ vs. control + aCSF; $^\#P < 0.05$ vs. SIH + aCSF (One-way ANOVA with turkey’s post hoc test). aCSF: artificial cerebrospinal fluid; CAN: candesartan, an AT$_1$R antagonist; p-p65: phosphorylated P65.
**Figure S3.** The TLR4 binds to MyD88 directly in the rostral ventrolateral medulla (RVLM) of control and stress induced hypertension (SIH) rats by using the co-immunoprecipitation method. The binding degree of TLR4 and MyD88 increased in the RVLM in SIH rat compared to in the control rat. TLR4: Toll-like receptor-4; MyD88: myeloid differentiation factor 88.