Table Supplemental Digital Content 1: Serial echocardiographic measurements of left ventricular function at baseline and 6 h after lipopolysaccharide adminstration

	WT		TLR4^{def}	
	Baseline	**LPS-6 h**	**Baseline**	**LPS-6 h**
HR, beats/min	716±12	611±16††	684±14	683±12**
LVIDd, mm	2.8±0.1	3.1±0.08	2.8±0.1	2.8±0.1
LVIDs, mm	1.3±0.1	2.4±0.1†††	1.2±0.02	1.4±0.04***‡‡
FS, %	56±1	23±2†††	59±1	51±2***

Baseline echocardiography was measured 1 day prior to lipopolysaccharide adminstration (baseline) and again 6 h after lipopolysaccharide administration (LPS-6 h). Values are presented as mean±SE. n=5 in each group. ** P<0.01 ***P<0.001 *vs* WT LPS-6 h, ††P<0.01, †††P<0.001 *vs* WT baseline, ‡‡ P<0.01 *vs* TLR4^{def} baseline. HR, heart rate; WT, wide type; TLR4^{def}, Toll-like receptor 4 deficient; LVIDd, left ventricular internal diameters at end-diastole; LVIDs, left ventricular internal diameters at end-systole; FS, fractional shortening.

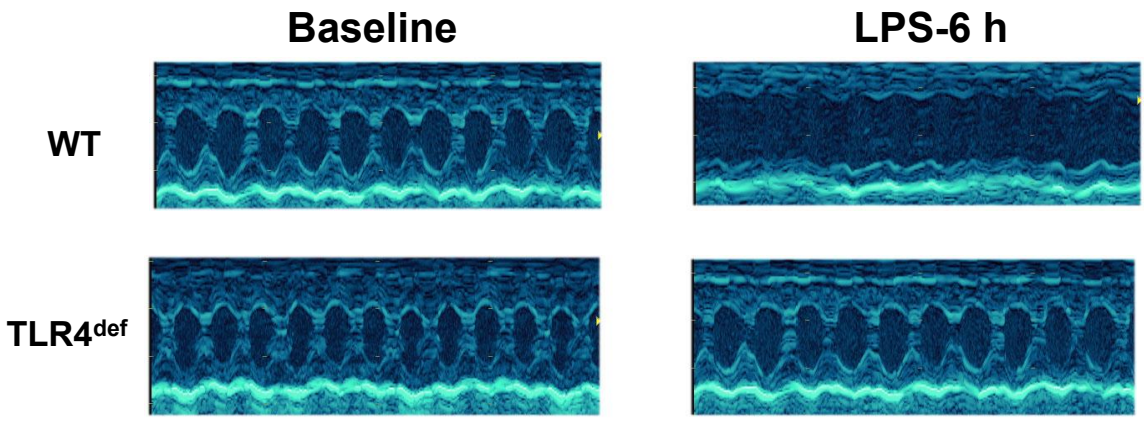

Figure Supplemental Digital Content 1. TLR4 deficiency protects the heart against endotoxin-induced dysfunction.

Six h after LPS administration, the end-systolic diameter of left ventricule in WT mice is markedly increased and the fraction shortening had dramatically decreased. TLR4-deficient mice have normal LV diameters and contractile function compared with WT mice subjected to LPS treatment. TLR, Toll-like receptor; LPS, lipopolysaccharide; WT, wide type.