Group	Interleukin- 6	TNF-a	PAI-1
WT + SAL	1.3 ± 0.6	1.2 ± 0.4	0.6±0.1
WT + LPS	8.5 ± 1.3*	1.0 ± 0.2	0.8±0.2
iNOS KO + SAL	2.4 ± 1.2	2.4 ± 0.6	0.9±0.3
iNOS KO + LPS	11.6 ± 2.7 †	1.7 ± 0.4	0.8±0.2

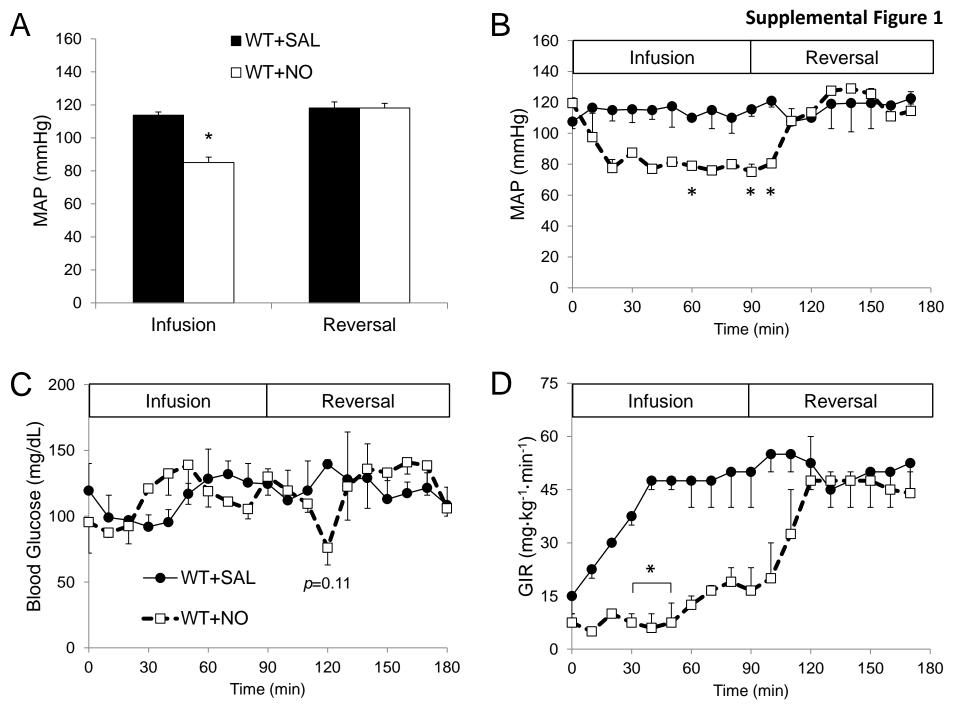
Table S1. Gene expression of pro-inflammatory markers in white adipose tissue of wild type and iNOS knockout mice treated with LPS and after the hyperinsulinemic-euglycemic clamp. * p<0.05 vs WT+SAL; † p<0.05 vs KO+SAL

SUPPLEMENTAL FIGURE LEGENDS

Supplement 1. Mean arterial blood pressure (MAP in mmHg, Panel A), glucose infusion rate (GIR in mg·kg⁻¹·min⁻¹, Panel B), and blood glucose (in mg/dL, Panel C) for saline-treated (SAL) or sodium nitroprusside (NO)-treated wild-type (WT; iNOS^{+/+}) mice during experimental induction (infusion) and restoration of hypotension in an extended (180 min) hyperinsulinemic-euglycemic clamp (Group 1A). Mice were treated with IV saline or NO infusion 90 min prior to clamp onset (t=0 min). Infusion was stopped at t=90 min (Reversal). MAP (Panel A) was summarized as mean during infusion period (t=0-60 min) and reversal period (t=90-180). Data are expressed as mean \pm SEM (n=2). *p \leq 0.05 vs. WT+SAL compared by t-test; NS = not significant (p \geq 0.05).

Supplement 2. Continuous mean arterial blood pressure (MAP) monitoring during infusion of sodium nitroprusside (NO)-infused iNOS^{+/+} mice (Group 1B).

Supplement 3. Effects of LPS on skeletal muscle insulin signaling in wild-type (WT; INOS^{+/+}) and iNOS knock-out (iNOS^{-/-}) mice after a 2 h hyperinsulinemic-euglycemic clamp (Group 2A). β -tubulin was used as a loading control. Western blotting was performed for gastrocnemius and vastus lateralis extract for total and tyrosine phosphorylation of IRS1 and Insulin receptor β (IR β).



Supplemental Figure 2

