Supplemental Material

Table S1. Primer sequences.

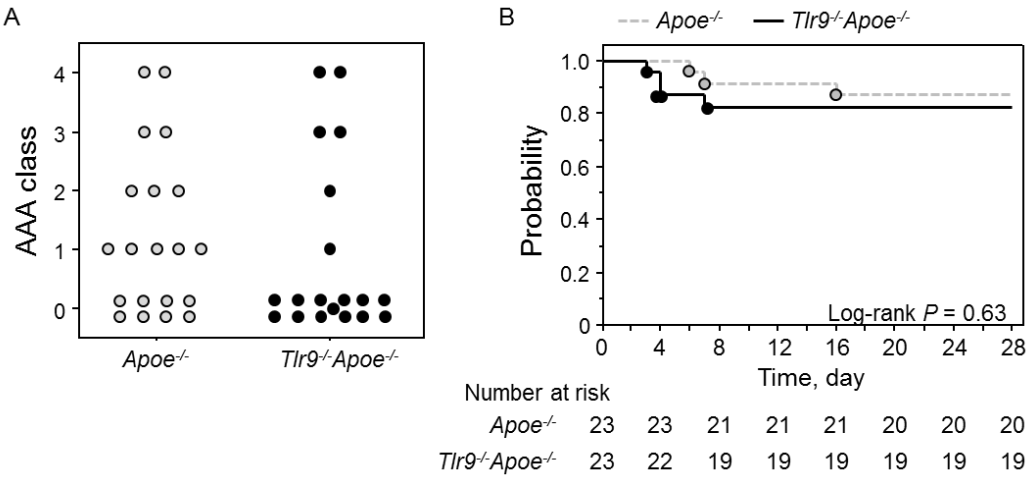
	Forward	Reverse
F4/80	5’- TGCATCTAGCAATGGACAGC -3’	5’- GCCTTCTGGATCCATTTGAA -3’
ICAM-1	5’- TTCACACTGAATGCCAGCTC -3’	5’- GTCTGCTGAGACCCCTCTTG -3’
MCP-1	5'-CCACTCACCTGCTGCTACTCAT-3’	5'-TGGTGATCCTCTTGTAGCTCTCC-3’
TLR9	5'-ATGGACGGGAACTGCTACTACA-3’	5'-GACCTTGGAACCAGGAAGAGTT-3’
TNF-α	5'-ACCCTCACACTCAGATCATCTTC-3’	5'-TGGTGGTTTGCTACGACGT-3’
VCAM-1	5'-GCCCATCCTCTGTGACTCAT-3’	5'-AGGCCACAGGTATTTTGTCG-3’
β-actin	5'-CCTGAGCGCAAGTACTCTGTGT-3’	5'-GCTGATCCACATCTGCTGGAA-3’

Table S2. Effect of genetic deletion of TLR9 on metabolic parameters in vehicle infused mice.

	Apoe$^{-/-}$	*Tlr9*$^{-/-}$*Apoe*$^{-/-}$	*P*-value
Body weight, g	31.7±0.8	31.8±1.1	0.72
Heart rate, bpm	750±5	725±12	0.05
Systolic blood pressure, mmHg	100.6±2.6	98.0±2.2	0.56
Diastolic blood pressure, mmHg	64.7±1.9	58.6±2.3	0.05
Total choletesterol, mg/dl	1034.9±80.4	1193.1±135.4	0.29
Triglyceride, mg/dl	140.9±10.9	150.0±23.1	0.71
HDL cholesterol, mg/dl	20.6±1.5	20.1±2.4	0.86

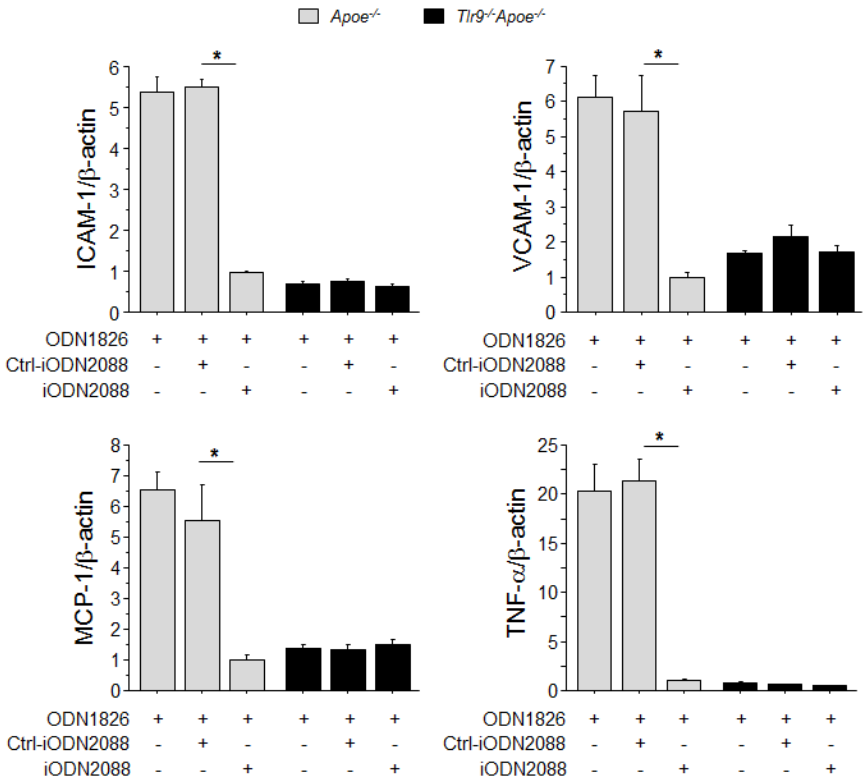
All values are mean ± SEM. HDL; high-density lipoprotein

Figure S1. Effect of genetic deletion of TLR9 on abdominal aortic aneurysm formation and survival curve.



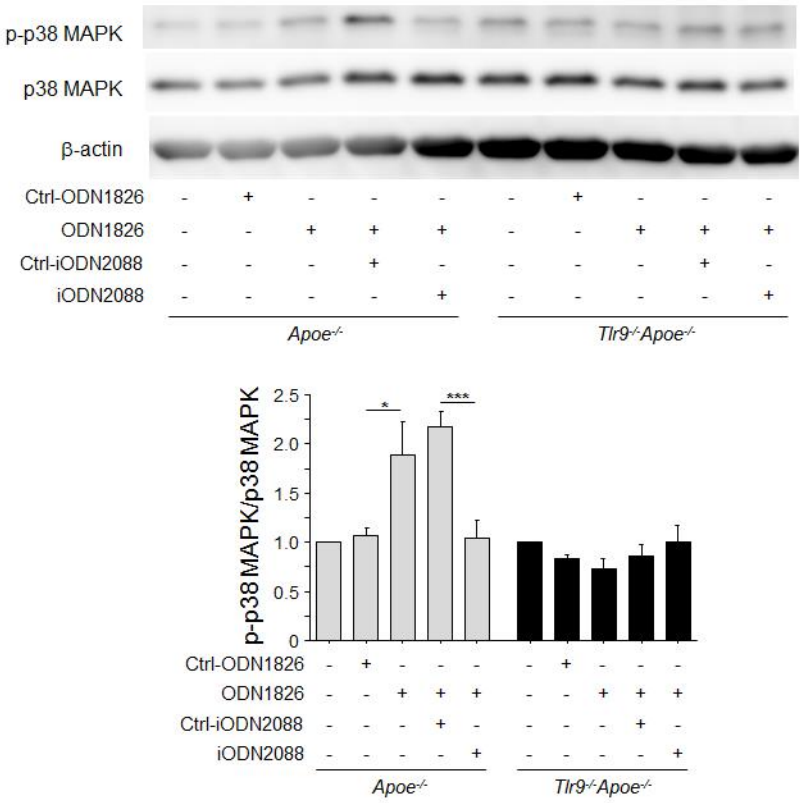
Genetic deletion of TLR9 did not affect the severity of abdominal aortic aneurysm (**A**) and survival curve **(B**) between *Apoe*$^{-/-}$ mice and *Tlr9*$^{-/-}$*Apoe*$^{-/-}$ mice. The severity of abdominal aortic aneurysm and survival curve between the two strains were analyzed by Mann-Whitney U test and Kaplan-Meier method with log-rank test, respectively.

Figure S2. iODN2088, a specific antagonist to TLR9, inhibits TLR9 activation.



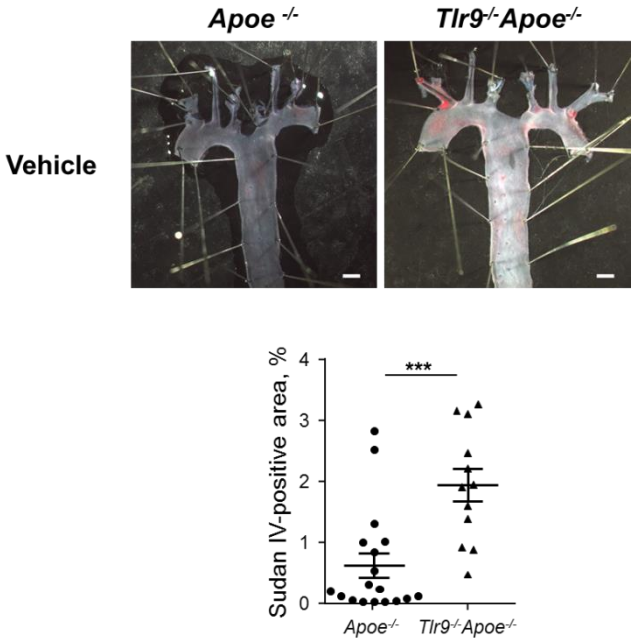
qPCR analysis demonstrated that iODN2088, an inhibitory oligonucleotide for TLR9, blocked inflammatory molecule expression induced by ODN1826. *$P < 0.05$. All comparisons between iODN2088-treated group and Ctrl-iODN-treated group were performed with Mann-Whitney U test. All values are mean ± SEM.

Figure S3. TLR9 ligation promotes p38 MAPK activation in *Apoe*$^{-/-}$ macrophages.



Western blotting analysis showed that ODN1826 increased the phosphorylation of p38 MAPK in *Apoe*$^{-/-}$ macrophages, which was blocked in the presence of iODN2088. Both ODN1826 and iODN2088 did not have any effect in *Tlr9*$^{-/-}$*Apoe*$^{-/-}$ mice. **P*<0.05, and ****P*<0.001. Comparison between ODN1826-treated group and Ctrl-ODN1826-treated group and comparison between iODN2088-treated group and Ctrl-iODN-treated group were performed with Mann-Whitney U test. All values are mean ± SEM.

Figure S4. *Tlr9*$^{-/-}$*Apoe*$^{-/-}$ mice had larger atherosclerotic lesions compared with *Apoe*$^{-/-}$ mice under vehicle infusion condition.



Genetic deletion of TLR9 promoted atherosclerotic lesion development in vehicle-infused *Apoe*$^{-/-}$ mice (n = 12-18). Scale bar; 1 mm. ***P<0.001. Comparison between *Tlr9*$^{-/-}$*Apoe*$^{-/-}$ mice and *Apoe*$^{-/-}$ mice was performed with Mann-Whitney U test. All values are mean ± SEM.