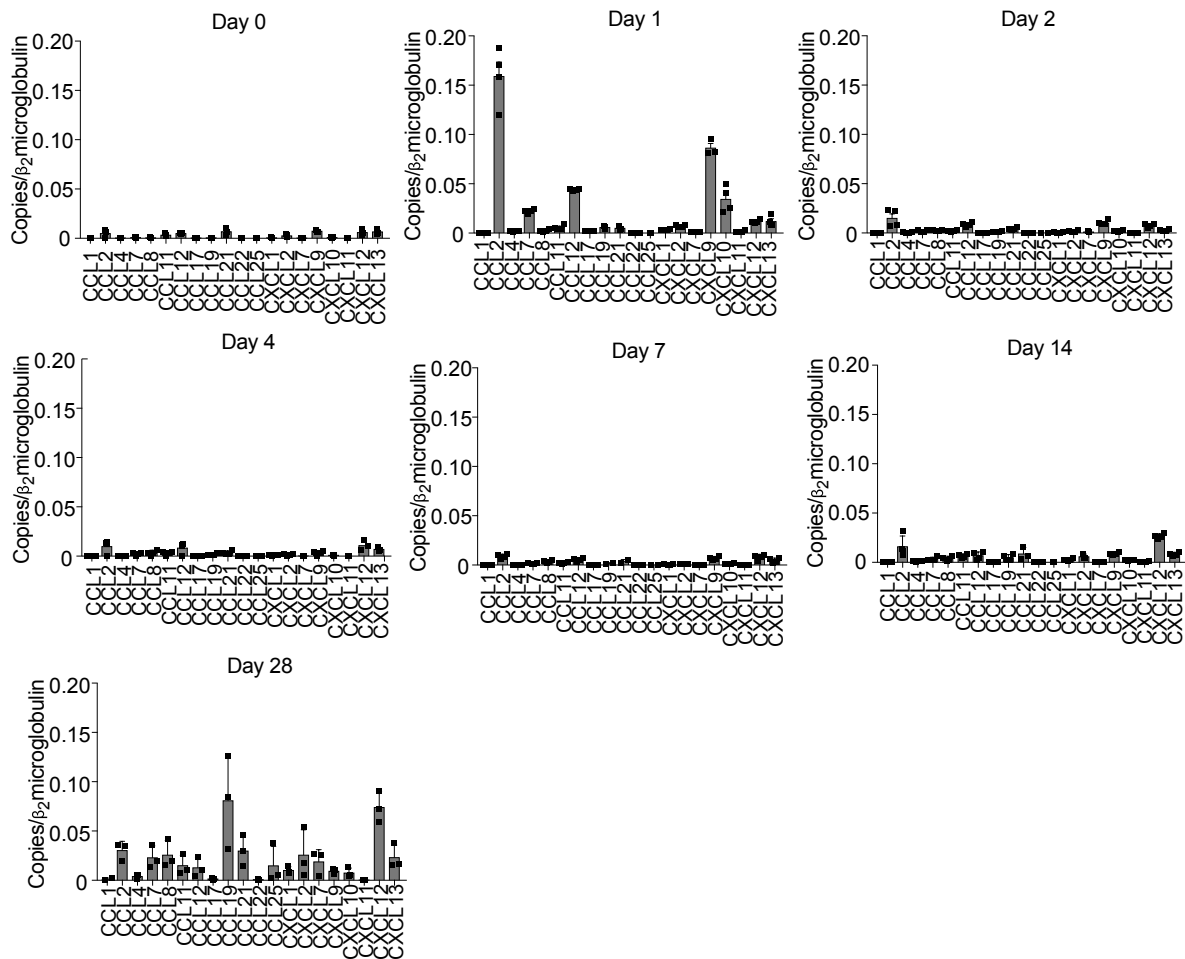
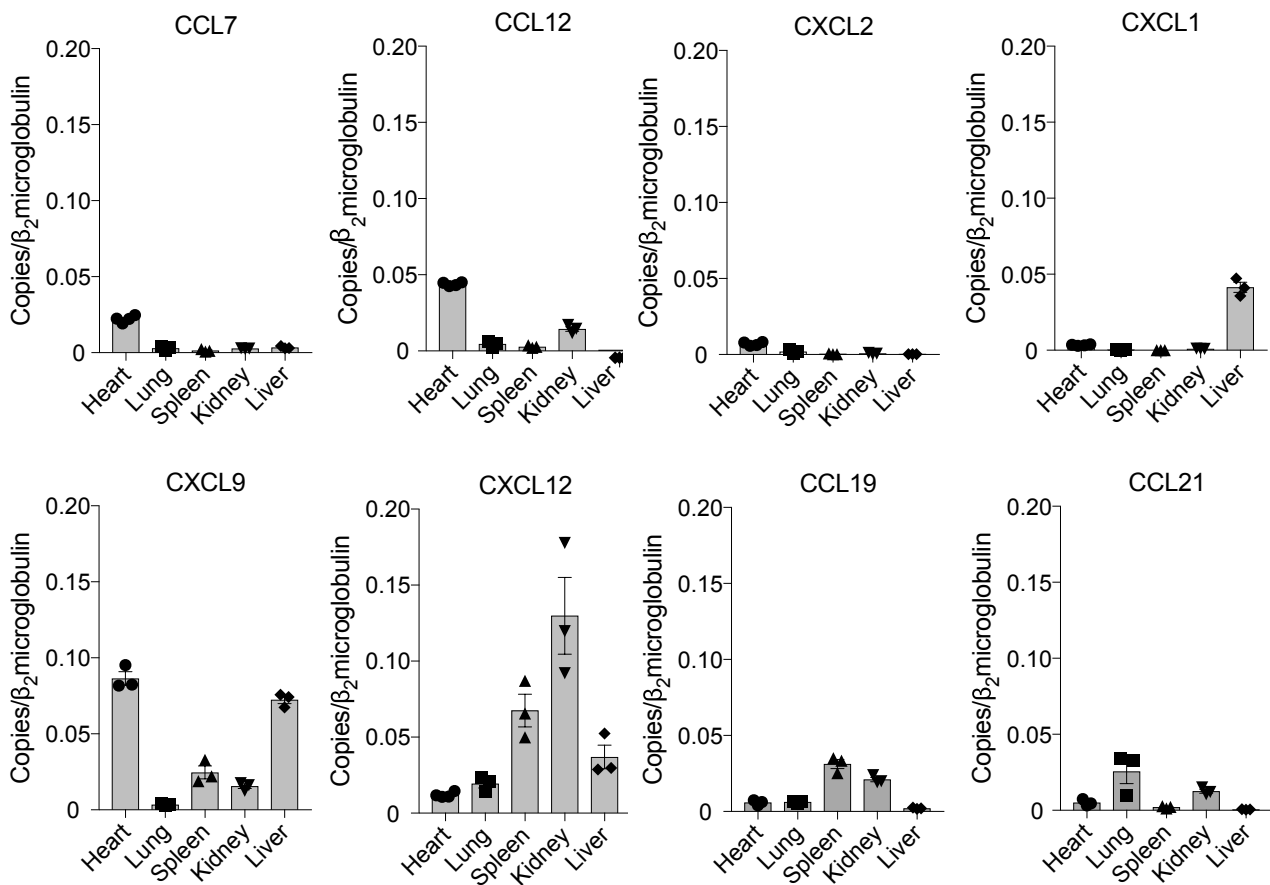


**Supplemental Figure 1. Temporal and spacial distribution of inflammatory cells in the heart.** Hearts were isolated from naive or CAWS-injected WT mice (on day 1, 7 or 28) and stained with anti-Ly6G/Ly6C or F4/80 for IHC. The number of Ly6G/Ly6C-positive cells (**A**) or F4/80-positive cells (**B**) was counted in high-power fields (600  $\mu$ m x 600  $\mu$ m) in myocardium area or aortic root area (mean  $\pm$  SEM, n= 3 mice per group, cell number of 2 fields are counted in each area of the mice, \*, p< 0.001 versus naive in each area). Scale bars in the low-power field, 1 mm; Scale bars in the high-power field, 100  $\mu$ m. All p values were calculated using unpaired two-tailed Student's t test.

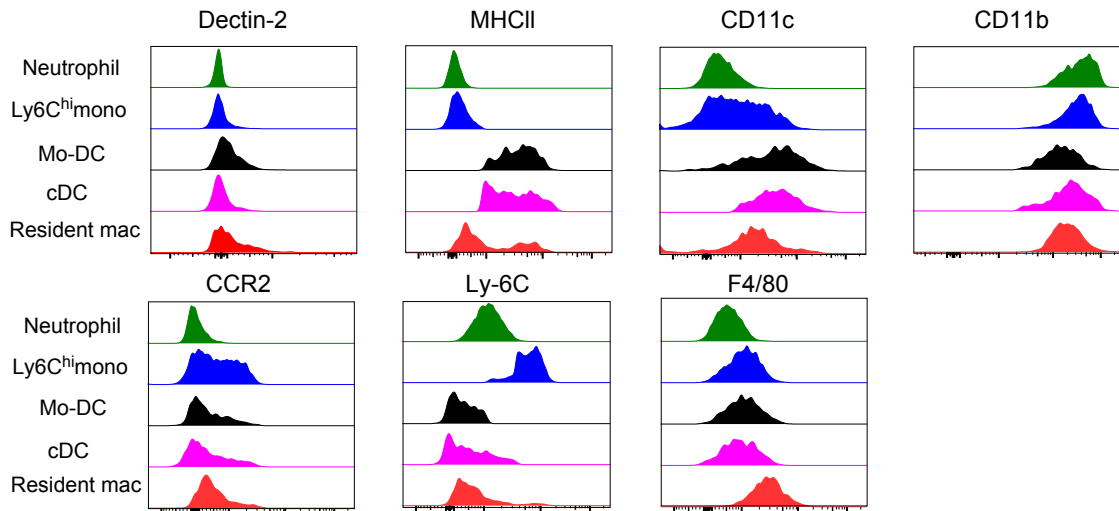


**Supplemental Figure 2. Chemokines levels in the heart over the course of CAWS-induced vasculitis.** Real-time qPCR analysis for chemokine levels on RNA isolated from heart tissue on day 1, 2, 4, 7, 14, 21 and 28 after CAWS injection (mean ± SEM, n = 3-4 per group).



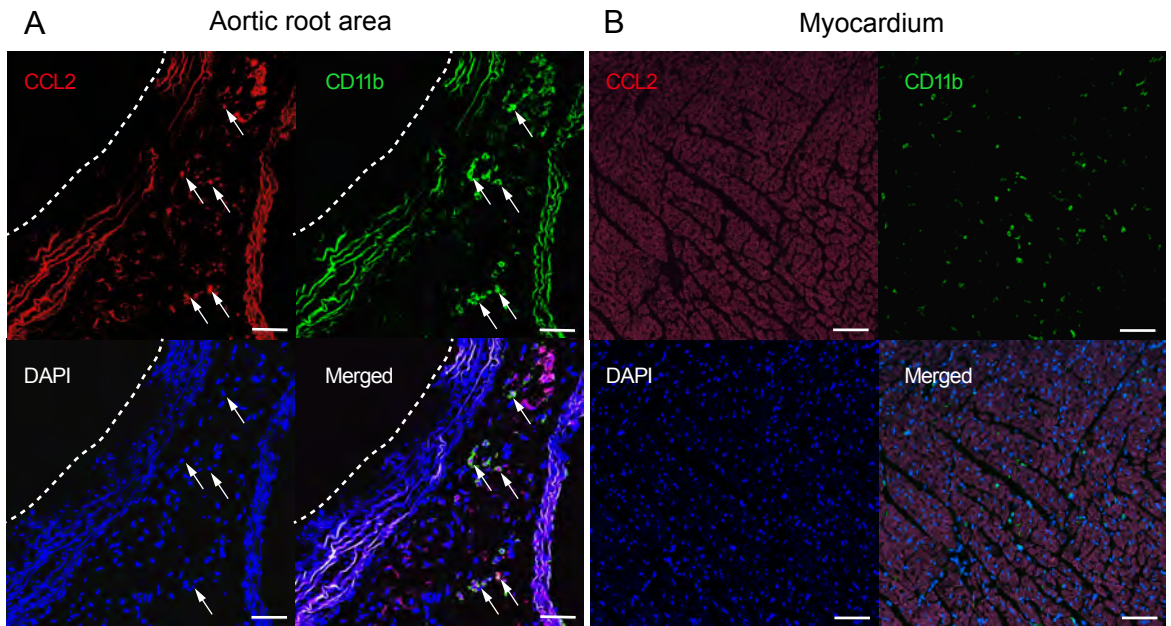
**Supplemental Figure 3. Chemokine levels in various organs day 1 after CAWS**

**injection.** Wild type mice were injected with CAWS and 1 day later, heart, lung, spleen, kidney and liver were harvested and assessed for chemokine RNA levels by qPCR (mean  $\pm$  SEM, n = 3-4).



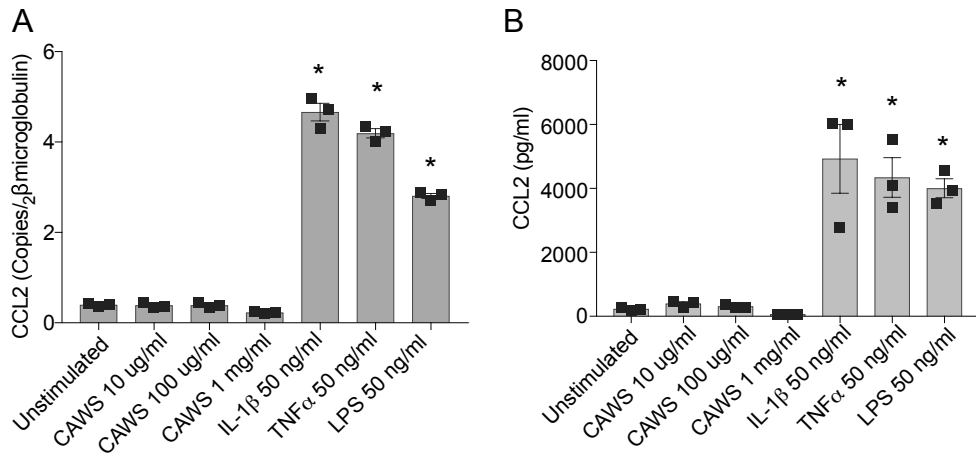
**Supplemental Figure 4. Phenotype of myeloid subsets recovered from WT hearts day 1 after CAWS injection.** Representative histograms of cardiac cell population labeled with antibodies to Dectin-2, MHC-II, CD11c, CD11b, CCR2, Ly6C and F4/80. The data is one representative of 3 independent experiments.



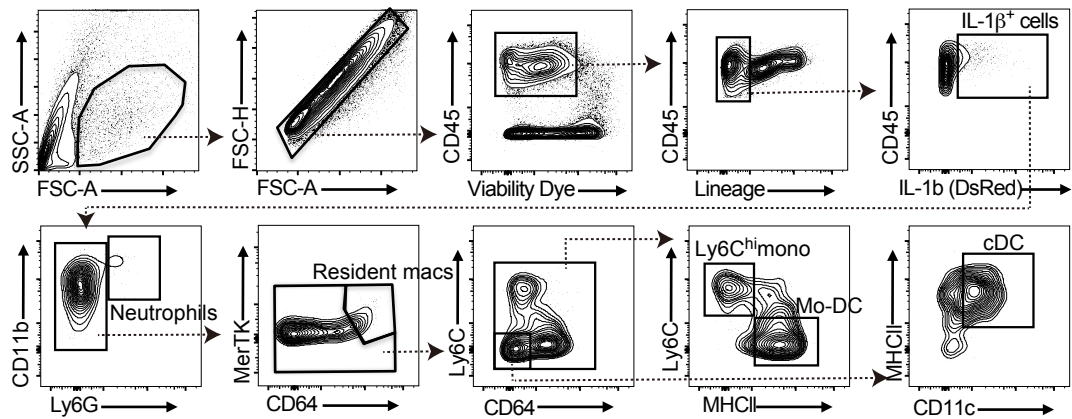


**Supplemental Figure 5. CCL2 is preferentially produced by CD11b+ cells in the aortic root compared to the myocardium following i.p. CAWS injection.**

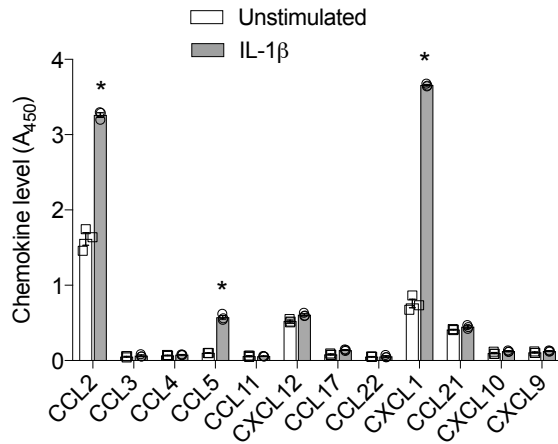
(A) Aortic root area or (B) myocardium isolated from *Ccl2-RFP<sup>fl/fl</sup>* reporter mice on day 1 after CAWS injection was stained for CD11b (green) and analyzed by confocal microscopy. Arrows indicate co-localization (yellow) of CD11b (green) with CCL2 (red). Scale bars= 50  $\mu$ m.



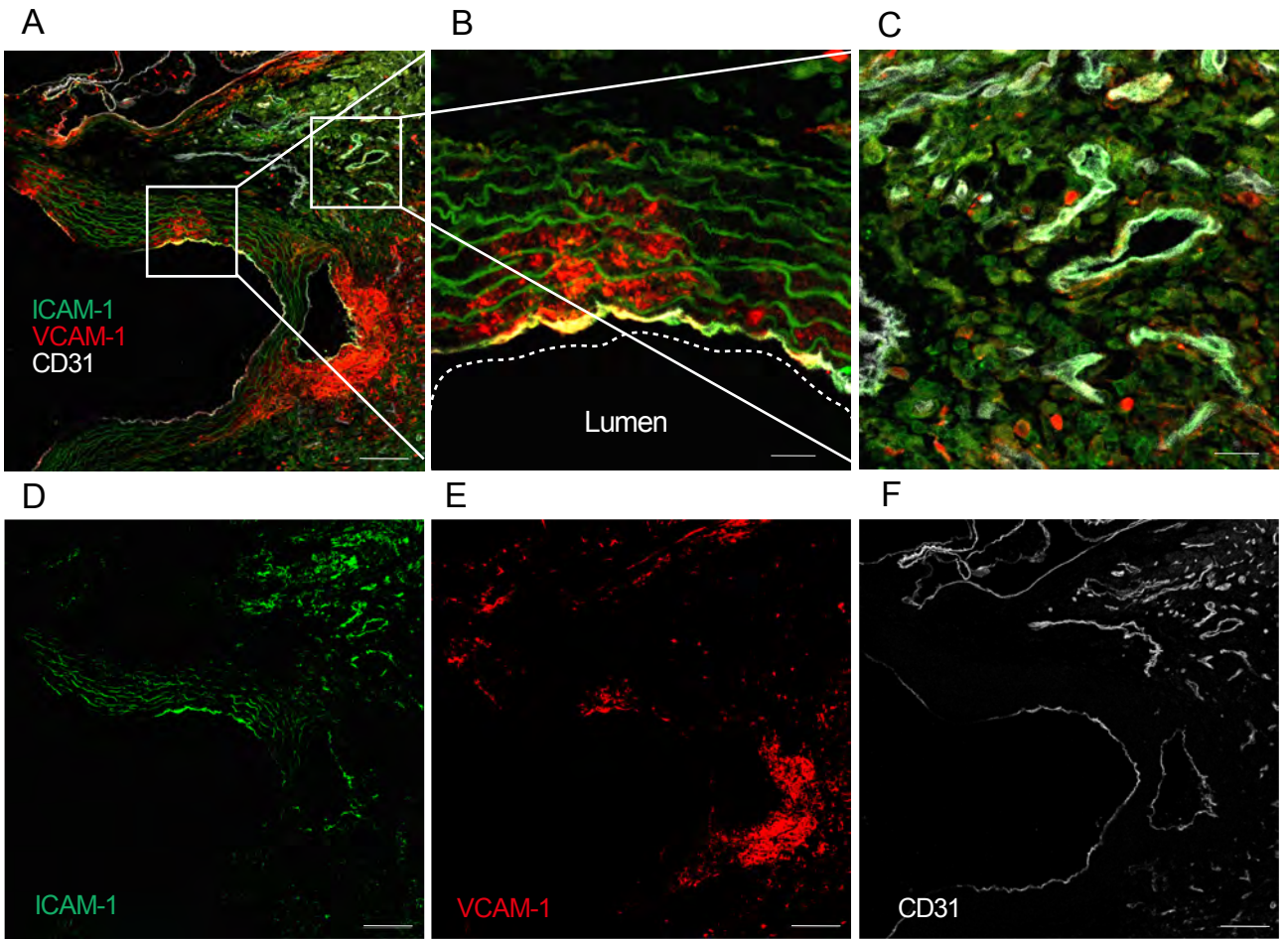
**Supplemental Figure 6. Cardiac fibroblasts did not produce CCL2 in response to CAWS stimulation.** Primary mouse cardiac fibroblasts were stimulated with IL-1 $\beta$ , TNF $\alpha$ , LPS, or CAWS for 18 hours. **(A)** CCL2 RNA levels were assessed by qPCR and **(B)** protein release by ELISA (mean  $\pm$  SEM, n=3, \*, p< 0.0001 versus Unstimulated). All p values were calculated using unpaired two-tailed Student's t test.



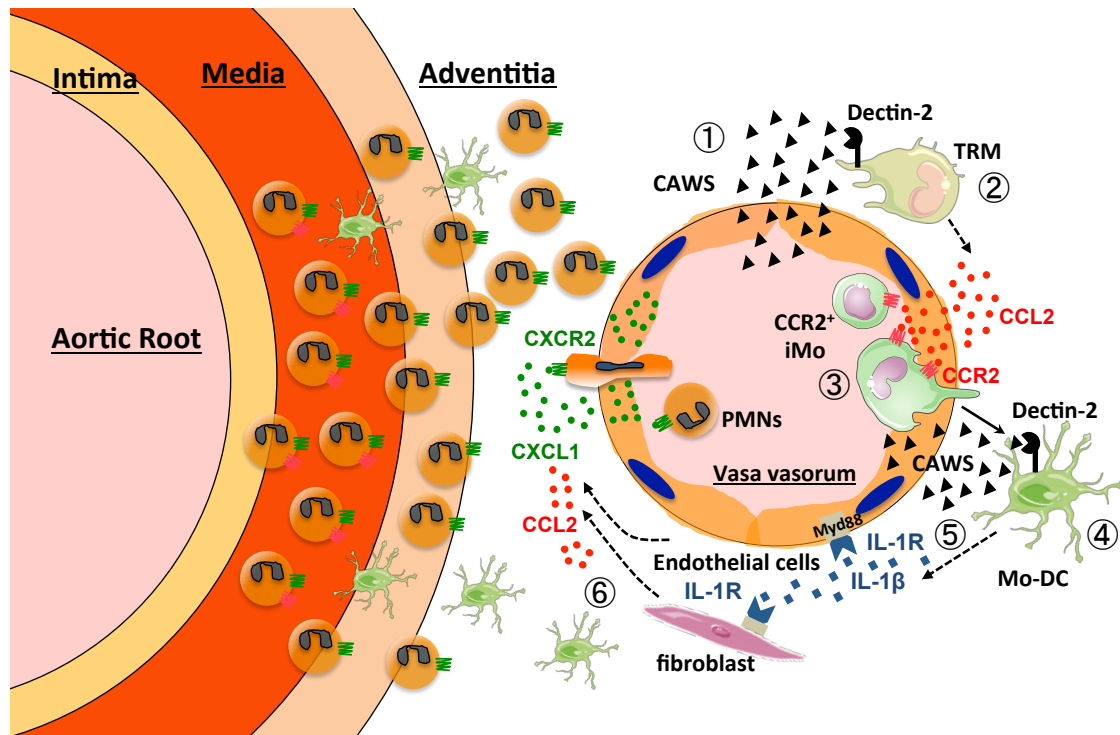
**Supplemental Figure 7. Flow cytometric gating strategy for single cell suspensions isolated from the hearts of *pIL1-DsRed* transgenic mice.** Representative contour plots of DsRed<sup>+</sup> IL-1β-expressing cells derived from WT or *pIL1-DsRed* transgenic hearts 7 days after CAWS injection. One representative of 3 independent experiments.



**Supplemental Figure 8. Cardiac fibroblasts secrete chemokine proteins following stimulation with IL-1b.** Mouse cardiac fibroblasts were stimulated with IL-1b (20 ng/ml) for 18 hours and chemokine protein levels in the culture supernatant were measured by ELISA (mean $\pm$  SEM, n = 3, \*, p < 0.0001 versus Unstimulated). All p values were calculated using unpaired two-tailed Student's t test.



**Supplemental Figure 9. ICAM-1 and VCAM-1 expression in the aortic root area of the heart on day 28 after CAWS injection.** (A-F) Sections of heart tissue were stained with ICAM-1, VCAM-1 and CD31. **A**, ICAM-1; **B**, VCAM-1; **C**, CD31; **D**, Merged image of A-C; **E**, luminal endothelium; **F**, adventitial area. Green autofluorescent elastic fibers in the medial wall. Scale bars= 100 μm.



**Supplemental Figure 10. CAWS induces Dectin-2-dependent CCL2 production in tissue resident macrophages in the aortic root and coronary arteries initiating arteritis.** ① Deposition of CAWS in the adventitia of the aortic root on day 1; ② CAWS activates Dectin-2 on tissue resident macrophages (TRM) to release CCL2; ③ CCL2 induces the recruitment of CCR2<sup>+</sup> inflammatory monocytes (iMo) into the adventitia; ④ iMo differentiate into monocyte derived-dendritic cells (Mo-DC) in the adventitia; ⑤ CAWS activates Dectin-2 on Mo-DC to release IL-1 $\beta$ ; ⑥ IL-1 $\beta$  activates endothelial cells and fibroblasts in the adventitia to release CXCL1 and CCL2, which recruit neutrophils (PMN) and iMo, respectively, into the adventitia driving and amplifying vascular inflammation.

**Supplemental Table 1.**

<b>ANTIBODIES</b>	<b>SOURCE</b>	<b>Clone</b>
CD11b-BV510	BioLegend	M1/70
CD11c-BV605	BioLegend	N418
CD31-Pacific Blue	BioLegend	390
CD45-FITC	BioLegend	30-F11
CD45-BV711	BioLegend	30-F11
CD45.1-FITC	BioLegend	A20
CD45.2-APC	BioLegend	104
I-A/I-E-PerCP/Cy5.5	BioLegend	M5/114.15.2
CD62P-PE	BioLegend	RMP-1
CD64-PE/Cy7	BioLegend	X54-5/7.1
CD90.2-APC	BioLegend	Thy1.2
CD106-PE	BioLegend	429
CD106-AF488	BioLegend	429
CD172a-APC	BioLegend	P84
F4/80-BV650	BioLegend	BM8
Ly6C-BV421	BioLegend	HK1.4
Ly6G-BV786	BioLegend	1A8
XCR1-BV421	BioLegend	ZET
MERTK-FITC	BioLegend	2B10C42
CD11b-Biotin	BioLegend	M1/70
CD11c-Biotin	BioLegend	N418
CD19-BUV395	BD Biosciences	1D3
Thy1.1-BUV395	BD Biosciences	53-2.1
NK1.1-BUV395	BD Biosciences	PK136
CD54-PE	BD Biosciences	3E2
CD62E-PE	BD Biosciences	10E9.6
Ly6G-PE	BD Biosciences	1A8
Streptavidin-FITC	BD Biosciences	N/A
CCR2-AF700	R&D Systems	475301
CCR2-PE	R&D Systems	475301
MERTK-APC	R&D Systems	108928
Dectin-2-APC	R&D Systems	N/A
polyclonal Goat IgG	R&D Systems	N/A
CD24-PE	eBioscience	30-F1
gp38-PE/Cy7	eBioscience	8.1.1
Purified F4/80	Cell Signaling Technology	N/A
Purified Ly-6G/Ly-6C	Novus Biologicals	RB6-8C5
Purified Dectin-2	Bio-Rad	D2.11E4
Anti-rat IgG-AF488	Invitrogen	N/A

**Supplemental Table 2.**

<b>Probe</b>	<b>Forward</b>	<b>Reverse</b>
<i>Ccl1</i>	AAG ATG GGC TCC TCC TGT CC	TTG AGG CGC AGC TTT CTC TAC
<i>Ccl2</i>	TTA AAA ACC TGG ATC GGA ACC AA	GCA TTA GCT TCA GAT TTA CGG G
<i>Ccl4</i>	TCT TGC TCG TGG CTG CCT	GGG AGG GTC AGA GCC CA
<i>Ccl7</i>	GCT GCT TTC AGC ATC CAA GTG	CCA GGG ACA CCG ACT ACTG
<i>Ccl8</i>	CGC AGT GCT TCT TTG CCT G	TCT GGC CCA GTC AGC TTC TC
<i>Ccl11</i>	TCC ACA GCG CTT CTA TTC CTG	GGA GCC TGG GTG AGC CA
<i>Ccl12</i>	GCT GGA CCA GAT GCG GTG	CCG GAC GTG AAT CTT CTG CT
<i>Ccl17</i>	CAG GGA TGC CAT CGT GTT TC	CAC CAA TCT GAT GGC CTT CTT
<i>Ccl19</i>	ATG CGG AAG ACT GCT GCC	CGG AAG GCT TTC ACG ATG TT
<i>Ccl21</i>	TCC CGG CAA TCC TGT TCT T	CCT TCC TCA GGG TTT GCA CA
<i>Ccl22</i>	TAC ATC CGT CAC CCT CTG CC	CGG TTA TCA AAA CAA CGC CAG
<i>Ccl25</i>	GCC TGG TTG CCT GTT TTG TT	CAG CAG TCT TCA AAG GCA CCT
<i>Cxcl1</i>	CTG GGA TTC ACC TCA AGA ACA TC	CAG GGT CAA GGC AAG CCT C
<i>Cxcl2</i>	CCA ACC ACC AGG CTA CAG G	GCG TCA CAC TCA AGC TCT G
<i>Cxcl5</i>	TGC GTT GTG TTT GCT TAA CCG	AGC TAT GAC TTC CAC CGT AGG
<i>Cxcl7</i>	CTC AGA CCT ACA TCG TCC TGC	GTG GCT ATC ACT TCC ACA TCA G
<i>Cxcl9</i>	AAT GCA CGA TGC TCC TGC A	AGG TCT TTG AGG GAT TTG TAG TGG
<i>Cxcl10</i>	GCC GTC ATT TTC TGC CTC A	CGT CCT TGC GAG AGG GAT C
<i>Cxcl11</i>	AAT TTA CCC GAG TAA CGG CTG	ATT ATG AGG CGA GCT TGC TTG
<i>Cxcl12</i>	AAA CCA GTC AGC CTG AGC TAC C	GGC TCT GGC GAT GTG GC
<i>Cxcl13</i>	CTC TCC AGG CCA CGG TAT TCT	CCG ACA ACA GTT GAA ATC ACT CC
<i>Ccr1</i>	ACC TTC GGC AGC TGT TTC A	TCC ACA GAG AGG AAG GGC AG
<i>Ccr2</i>	TTA AAA ACC TGG ATC GGA ACC AA	GCA TTA GCT TCA GAT TTA CGG G
<i>Cxcr2</i>	ATG CCC TCT ATT CTG CCA GAT	GTG CTC CGG TTG TAT AAG ATG AC
<i>Il1b</i>	ACC TGT CCT GTG TAA TGA AAG ACG	TGG GTA TTG CTT GGG ATC CA