**SUPPLEMENTARY INFORMATION**

# E-cadherin expression in macrophages dampens their inflammatory responsiveness *in vitro,* but does not modulate M2-regulated pathologies *in vivo*

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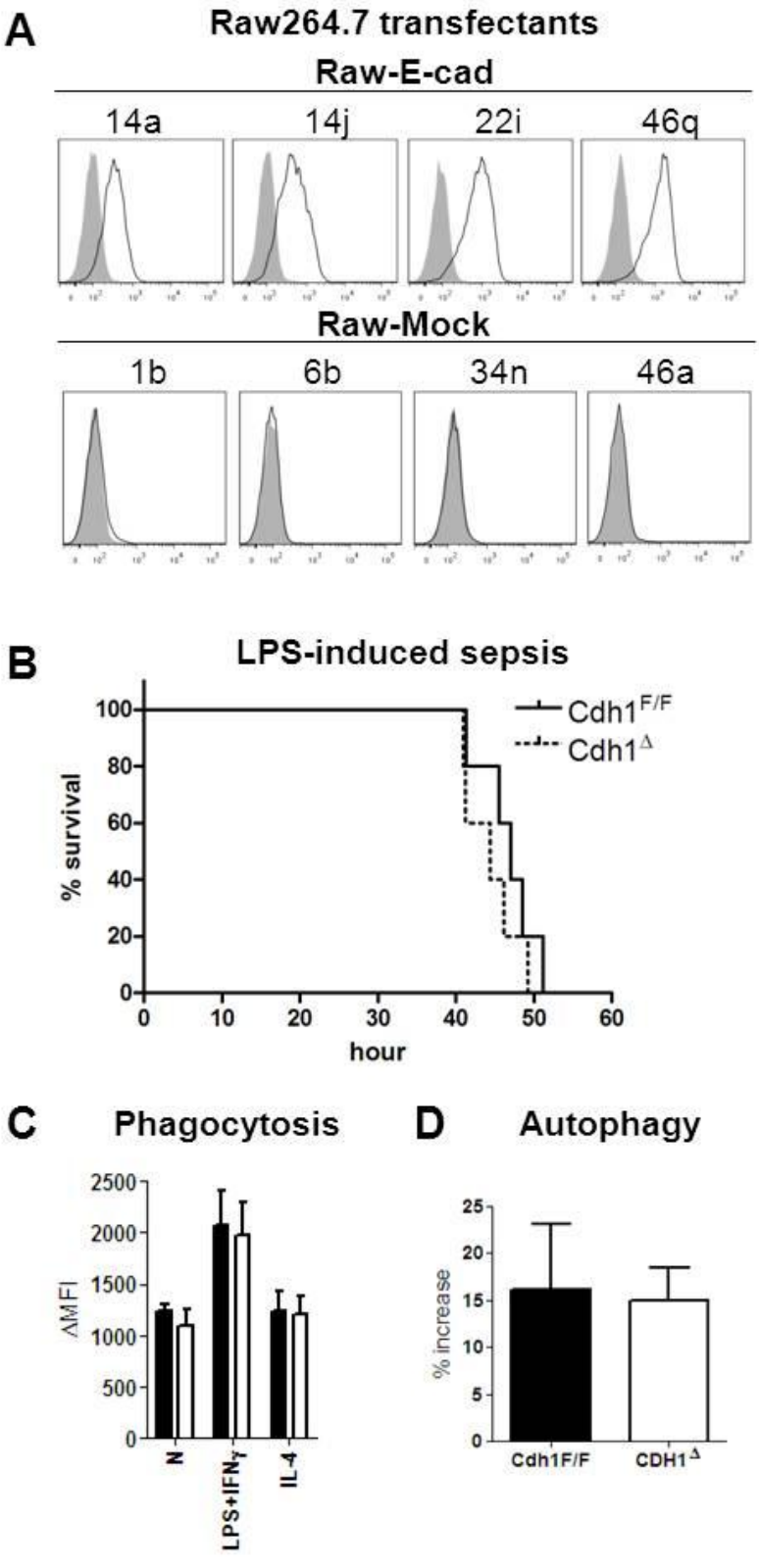
A
Raw264.7 transfectants
Raw-E-cad
14a
14j
22i
46q
Raw-Mock
1b
6b
34n
46a
B
LPS-induced sepsis
Cdh1F/F
Cdh1Δ
% survival
hour
C
Phagocytosis
ΔMFI
N
LPS+IFNγ
IL-4
D
Autophagy
% increase
Cdh1F/F
CDH1Δ

**Figure S1**

**Figure S1. (A) Raw264.7 transfectants.** Four independent E-cadherin over-expressing Raw264.7 transfectants and four E-cadherin-negative mock transfectants were generated as described earlier [6] and the E-cadherin surface expression of these clones was assessed by flow cytometry. **(B) LPS-induced sepsis.** An LPS-induced sepsis experiment was performed as typical M1 response *in vivo*. Hereto, 5 $Cdh1^{\Delta}$ and 5 $Cdh1^{F/F}$ mice (n=5) were injected with 12 mg/kg LPS (from *Escherichia coli* 0111:B4, Sigma) after which their survival was recorded. No differences were observed between the $Cdh1^{\Delta}$ mice and their WT ($Cdh1^{F/F}$) counterparts.**(C) Phagocytosis.** BMDM from naive $Cdh1^{\Delta}$ and $Cdh1^{F/F}$ mice (n=3) were treated with 10 ng/ml LPS + 10 U/ml IFNγ or with IL-4, or remained untreated for 24 h. Next, macrophages were cultured for 1 h at 4°C (control) or 37°C in the presence of green fluorescent latex beads (Polyscience, 1/5000 dilution). Latex bead uptake was assessed by flow cytometry and is shown as ΔMFI = [median fluorescence intensity] $_{37°C}$ - [median fluorescence intensity] $_{4°C}$. **(D) Autophagy.** $Cdh1^{\Delta}$ and $Cdh1^{F/F}$ BMDM were treated for 24 h with 500 nM rapamycin to induce autophagy or were left untreated. Next, a Cyto-ID Autophagy Detection Kit (Enzo Life Sciences) was used according to the manufacturer's protocol, followed by flow cytometry. Data show the rapamycin-induced increase in autophagy, compared to untreated macrophages.

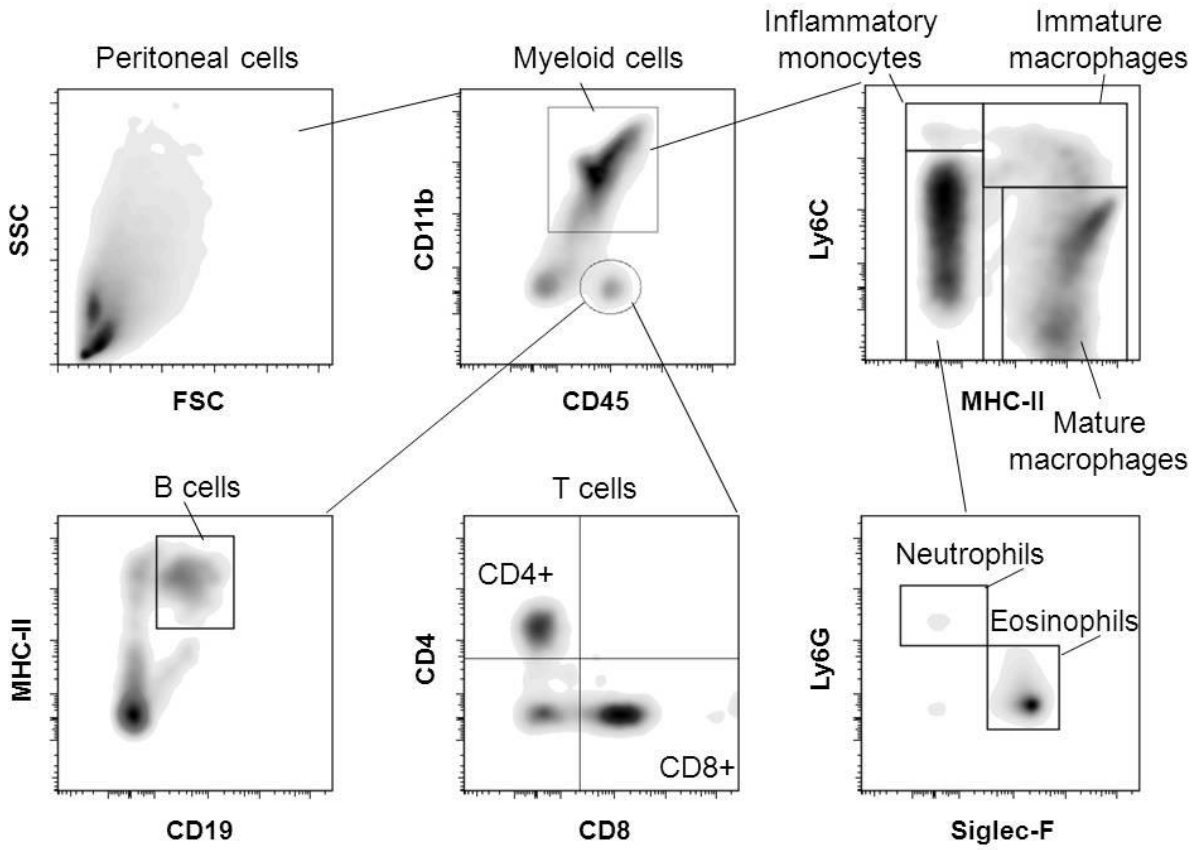


**Figure S2. FACS gating strategy on freshly isolated peritoneal cells from *Taenia crassiceps* infected Cdh1$^{F/F}$ mice.** Myeloid cells and other leukocytes were first separated based on CD11b/CD45 expression. Within the CD11b$^{-}$/CD45$^{+}$ leukocyte gate, B cells were gated as MHC II$^{high}$/CD19$^{+}$ and T cells were separated based on their differential CD4 and CD8 expression. Within the CD11b$^{+}$ myeloid cell gate Ly6C/MHC II staining discriminates between Ly6C$^{high/}$MHC II$^{-}$ inflammatory monocytes, Ly6C$^{high/}$MHC II$^{+}$ immature macrophages and Ly6C$^{low\ to\ int/}$MHC II$^{high}$ mature macrophages. Within the Ly6C$^{lo\ to\ int}$/MHC II$^{-}$ gate, neutrophils were selected as Ly6G$^{+}$ and eosinophils were gated as Siglec-F$^{+}$.

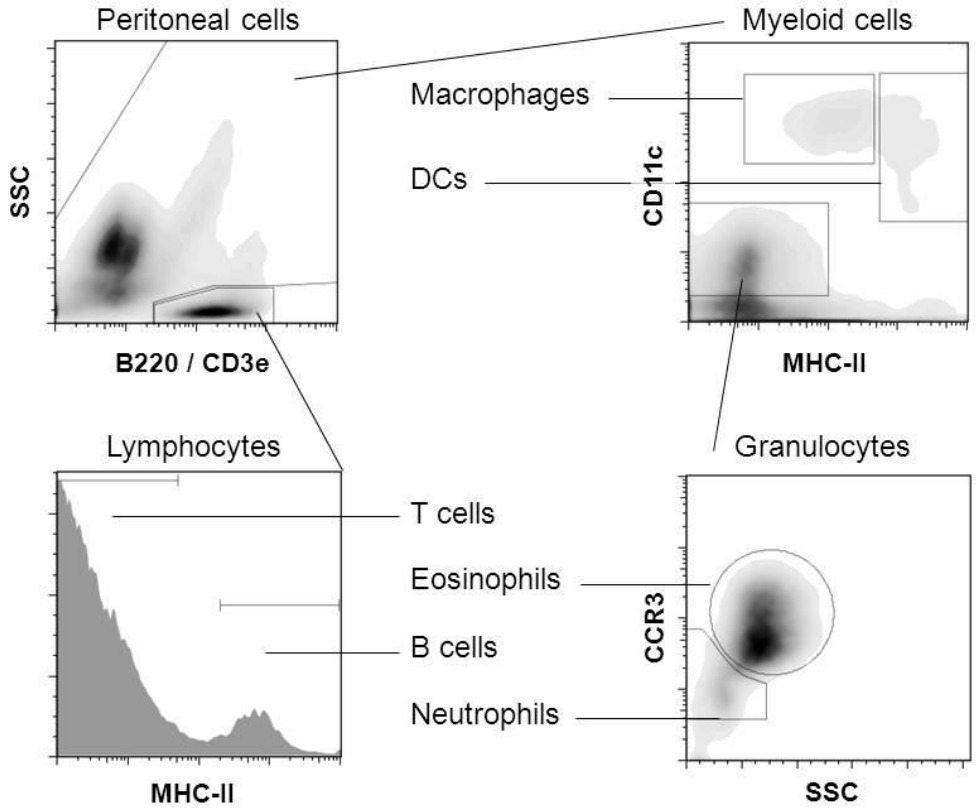


**Figure S3. FACS gating strategy on cells of the bronchoalveolar lavage (BAL) of Cdh1$^{F/F}$ mice (4x OVA).** First, lymphocytes were gated based on their B220/CD3 expression and low side scatter and further selected as B or T cells according to their high of low MHC II expression, respectively. Within the myeloid cell gate, macrophages were selected as CD11c$^{hi}$/MHC II$^{int}$ and DCs as CD11c$^{high}$/MHC II$^{high}$. Within the CD11c$^{-}$/MHC II$^{-}$ gate, eosinophils were gated as CCR3$^{hi}$ and the remaining SSC$^{int}$ population was marked as neutrophils.