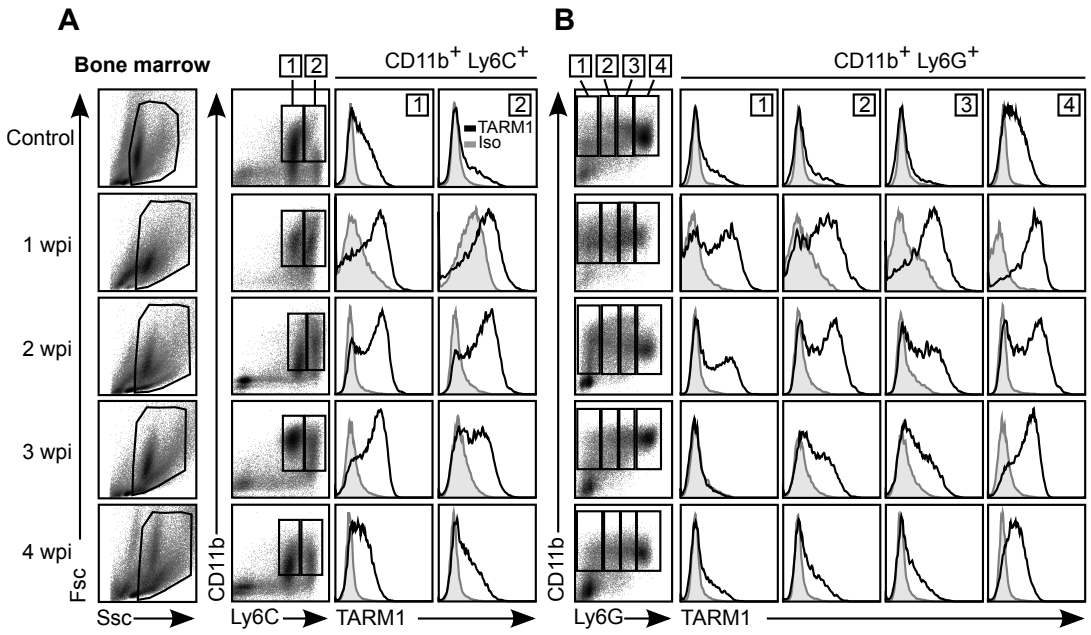
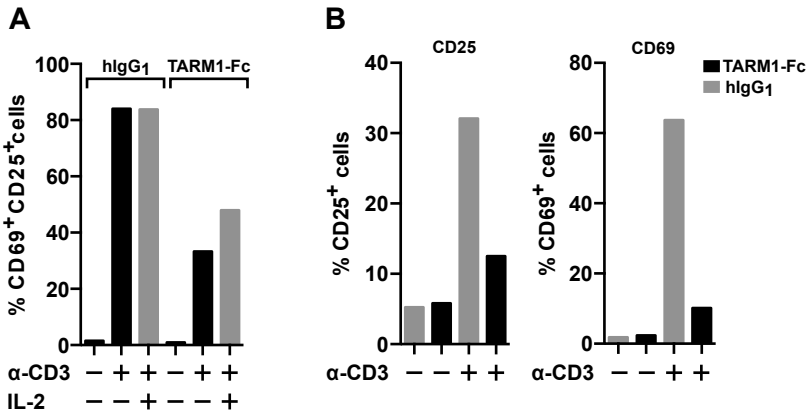
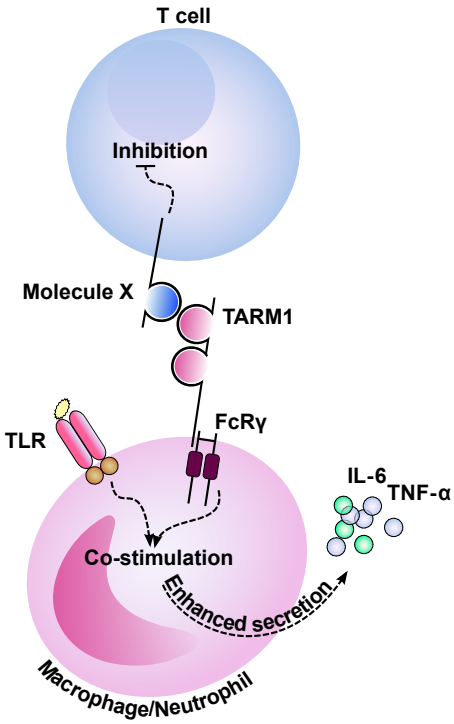


Suppl. Figure 1. Systemic infection with S. Typhimurium causes upregulation of TARM1 expression on CD11b $^{+}$Ly6G^{+} and CD11b^{+}Ly6C^{+} bone marrow neutrophils. C57BL/6 mice (n=3) were injected i.v. with either PBS or *aroA* attenuated S. Typhimurium strain SL3261 (1 x 10^{6} CFU) and sacrificed at the indicated time points. TARM1 expression was determined by flow cytometry. Representative plots from one of three independent experiments are shown. CD11b^{+} cells were gated according to thier expression of Ly6C (gates 1 and 2) (A) and Ly6G (gates 1-4) (B). Histogram overlays show TARM1 expression in corresponding Ly6C and Ly6G gates.



Suppl. Figure 2. TARM1-Fc inhibits human $CD4^+$ T cell activation *in vitro*. T cells were isolated immunomagnetically by positive selection (Miltenyi) from peripheral blood of healthy donors (n=2) and activated with plate-bound anti-CD3 (1.2 µg/ml, clone OKT3) in the presence of plate-bound TARM1-Fc (10 µg/ml) or human IgG_1 ($hIgG_1$) (10 µg/ml). Recombinant human IL-2 (50 U/ml) was added to some wells. T cells were cultured for 3 days and the expression of activation markers CD25 and CD69 was analyzed by flow cytometry. (A) donor 1, (B) donor 2.



Suppl. Figure 3. Model for TARM1-mediated bi-directional signaling in the immune system. TARM1 receptor signaling synergizes with specific inflammatory stimuli, e.g. TLR agonists, such as Pam3CK4 (TLR1/2), Poly I:C (TLR3), LPS (TLR4) or Imiquimod (TLR7), to enhance the secretion of pro-inflammatory cytokines, such as TNF-α and IL-6, by TARM1-expressing cells. Conversely, engagement of the TARM1 ectodomain with an as yet unidentified ligand expressed by T cells results in the inhibition of T cell activation and proliferation.