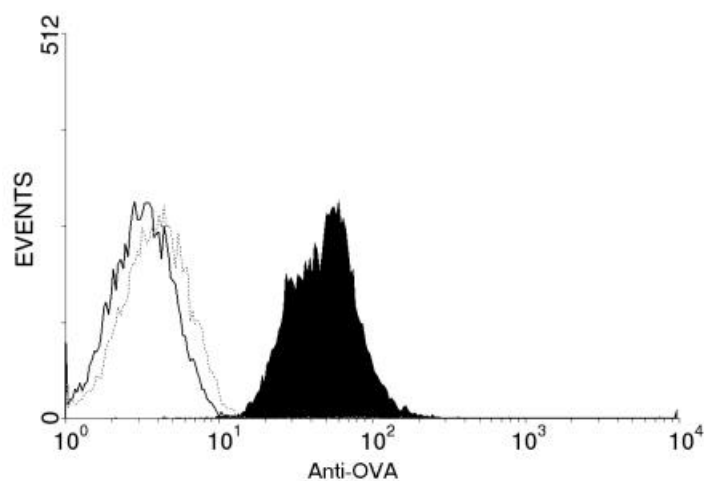
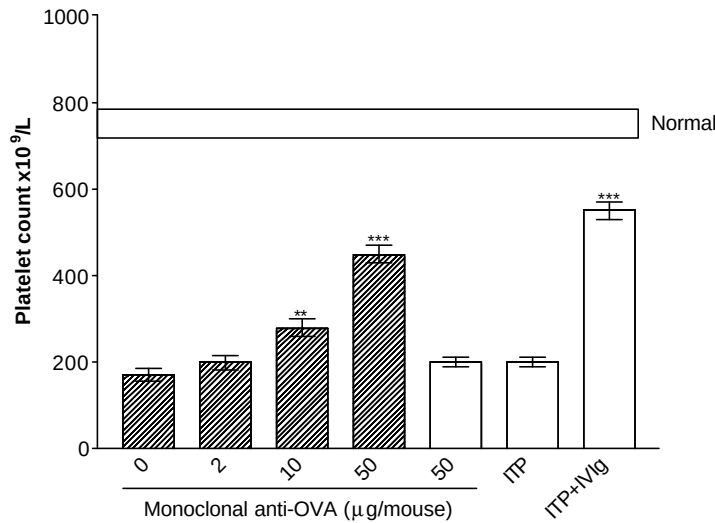


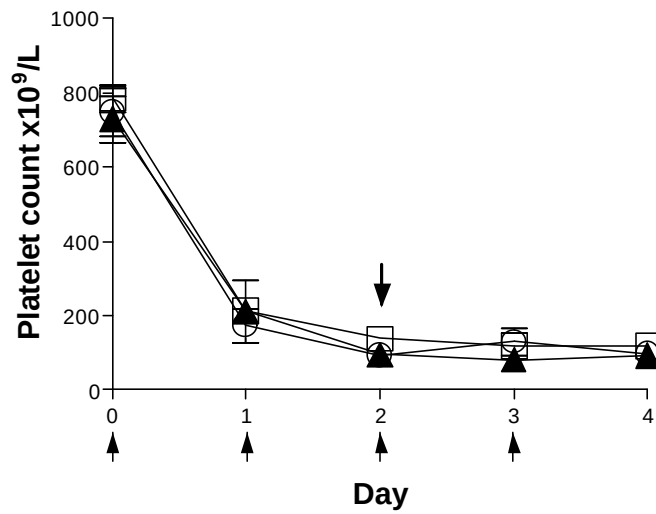
Supplementary Figures**Supplementary Figure 1. OVA is detected on the surface of RBC after coupling with EDAC.**

OVA-RBC prepared with EDAC were stained with anti-OVA (solid histogram), or control IgG (solid line), followed by the appropriate FITC conjugated secondary antibody (dashed line, secondary antibody only). RBC were then analyzed for fluorescence by flow cytometry. The x axis shows relative fluorescence intensity; y -axis represents cell number. Histogram is representative of 3 experiments.



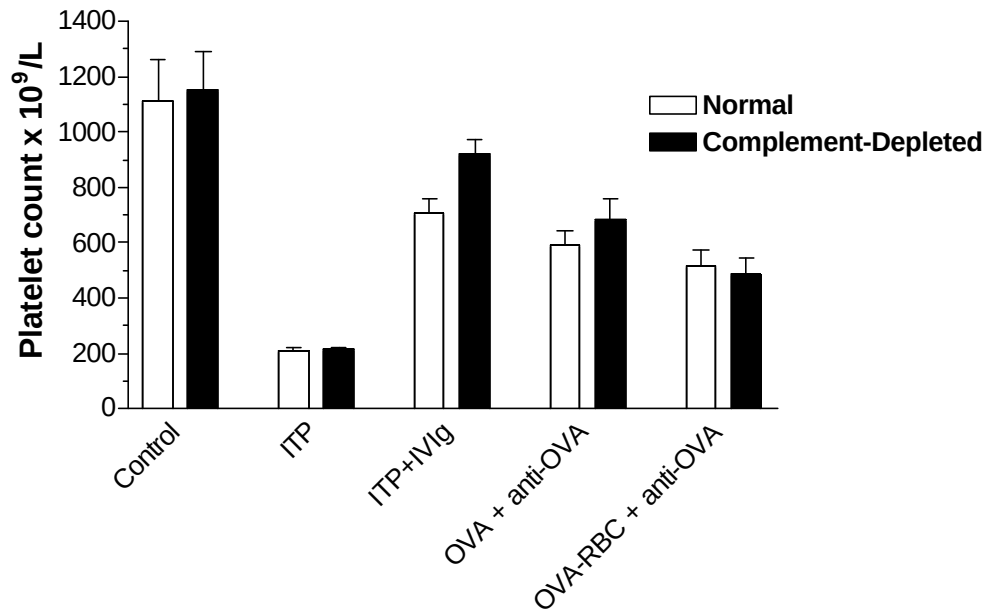
Supplementary Figure 2. Monoclonal anti-OVA reactive with soluble OVA ameliorates immune thrombocytopenia.

CD1 mice were pretreated by intravenous injection of 1 mg OVA (Hatched Bars) that had been pre-incubated with the indicated dose of monoclonal anti-OVA (IgG1, clone OVA-14, Sigma), followed 24 hours later by injection of 2 µg anti-platelet antibody. The x-axis denotes the treatment; y-axis denotes platelet counts taken 24 hours later by injection of 2 µg anti-platelet antibody. Normal: unmanipulated mice; ITP: mice injected with anti-platelet antibody only; ITP+IVIg: mice pretreated with 50 mg IVIg followed by antiplatelet antibody; n=9 mice for each group from 3 independent experiments. *** $P < 0.001$, ** $P < 0.01$ vs. ITP mice. Data are presented as mean $\pm$ SEM.



Supplementary Figure 3. Antibodies to albumin and transferrin require the expression of FcγRIIB to ameliorate immune thrombocytopenia.

FcγRIIB^{-/-} mice were injected with 2 μg anti-platelet antibody on days 0 through 3 denoted by the arrow (↑). On day 2 (↓) mice were injected intraperitoneally with 50 mg IVIg (□), or intravenously with 1 mg anti-albumin antibody (▲), or 1 mg anti-transferrin antibody (○). Mice were bled daily for platelet counting; n=3 mice for each group. Data are presented as mean ± SEM (see Figure 6B for complementary experiment in normal mice).



Supplementary Figure 4. Antibody reactive with soluble OVA or OVA-RBC ameliorates immune thrombocytopenia independent of complement activity.

CD1 mice were injected intraperitoneally with 5U of cobra venom factor (CVF, Cedarlane Laboratories Ltd) followed by a second injection of 5U CVF after 4 hours to deplete complement, or were left untreated. CVF successfully depleted complement to below detectable levels as assessed in a haemolytic assay (data not shown). After 24 hours, mice were treated with the preparations indicated on the x axis. The induction of thrombocytopenia and counting of platelets were as described in Supplementary Figure 2. Control: mice receiving no therapeutic treatment; ITP: mice treated with 2 μ g anti-platelet antibody only; ITP+IVIg: mice treated intraperitoneally with 50 mg IVIg followed by anti-platelet antibody; OVA + anti-OVA: mice treated intravenously with OVA + anti-OVA; OVA-RBC + anti-OVA: mice treated intravenously with 10^8 OVA-RBC + anti-OVA; n=5 mice for each group from 3 independent experiments. There is no statistically significant difference ($P>0.05$) between normal and complement depleted group. Data are presented as mean $\pm$ SEM.