SUPPLEMENTARY MATERIAL

Supplementary Table 1 Characteristics of the subjects included in this study

	Primary APS		Controls	
N	40		47	
Female	24	(60%)	30	(64%)
Age (y)*	44 ± 5	(21-77)	42 ± 4	(22-67)
Disease duration (y)*	9 ± 3	(1-28)		
White/Caucasian	38	(95%)	42	(89%)
IgG anti-β_2GPI	20/40	(50%)		
IgM anti-β_2GPI	7/40	(18%)		
IgA anti-β_2GPI	14/40	(35%)		
IgG anticardiolipin	30/40	(75%)		
IgM anticardiolipin	13/40	(33%)		
Lupus anticoagulant	29/36	(81%)		
"Triple-positive"	19/36	(53%)		
Venous thrombosis	18/40	(45%)		
Arterial thrombosis	16/40	(40%)		
Small vessel	4/40	(10%)		
Pregnancy morbidity	7/24	(29%)		
Thrombocytopenia	9/40	(23%)		
Livedo	11/40	(28%)		
Warfarin	19/40	(48%)		
Aspirin	18/40	(45%)		
LMWH	2/40	(5%)		
Factor Xa inhibitor	4/40	(10%)		
Hydroxychloroquine	14/40	(35%)		
Immunosuppressant	1/40	(3%)		

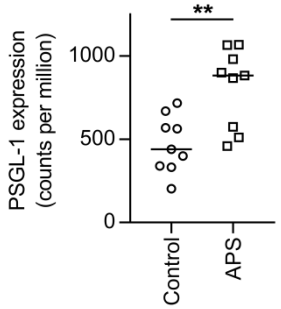
***** mean ± 95% confidence interval (range)

Supplementary Table 2 Differentially expressed genes in neutrophils from patients with primary APS as compared with healthy matched controls

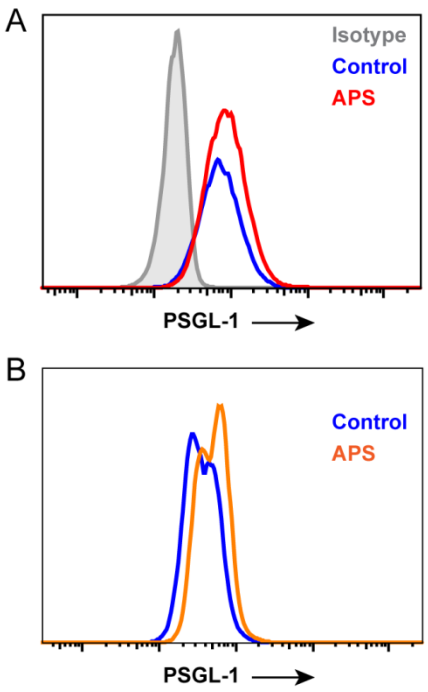
(See separate attachment)

Supplementary Table 3 Gene ontology analysis of genes upregulated and downregulated in neutrophils from patients with primary APS as compared with healthy matched controls

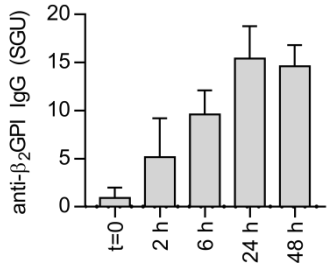
(See separate attachment)



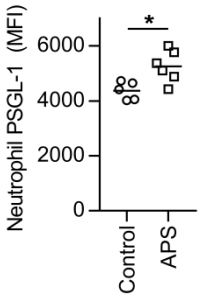
Supplementary Figure 1 Details of PSGL-1 gene expression from the RNA sequencing data set, presented as counts per million. The mean for each group is denoted by a solid horizontal line, while each data point represents a unique control/patient; ***P*<0.01 by t test.



Supplementary Figure 2 Conditioning control neutrophils with APS patient serum induces surface expression of PSGL-1. Control neutrophils were conditioned with either control or APS serum, as described in Methods. Neutrophils were then collected, and surface expression of PSGL-1 was quantified by flow cytometry. **A** and **B** demonstrate results for two different APS serum samples (red and orange, respectively). These data are representative of three independent experiments, all with similar results.



Supplementary Figure 3 Human anti-β_2GPI is detectable in the blood of mice following APS IgG injection. 2 mg of APS IgG was administered by intraperitoneal injection, and blood was collected for serum preparation at various time points after injection. SGU=standard IgG units, calculated according to manufacturer's instructions (Inova Diagnostics). Mean ± standard deviation are presented for n=3 mice per group.



Supplementary Figure 4 Increased surface expression of PSGL-1 *in vivo*, following administration of APS IgG. 2 mg of either control IgG or APS IgG was administered by intraperitoneal injection, and blood was collected 24 hours later for flow cytometry. MFI=mean fluorescence intensity. The mean for each group is denoted by a horizontal line, while each data point represents a unique mouse; *P<0.05 by t test.

Supplementary Video 1 Leukocyte adhesion and rolling in WT mice treated with APS IgG. The five-second video contains 150 frames. Leukocytes are stained red as described in Figure 5. Rolling leukocytes move along the vessel-wall surface at a rate that is slower than the background flow of blood.

(See separate attachment)

Supplementary Video 2 Attenuated leukocyte adhesion and rolling in PSGL-$1^{-/-}$ mice treated with APS IgG. The five-second video contains 150 frames. Leukocytes are stained red as described in Figure 5. Rolling leukocytes move along the vessel-wall surface at a rate that is slower than the background flow of blood.

(See attachment)