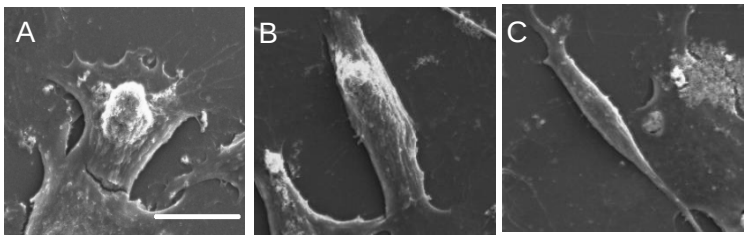
Supplementary Results:

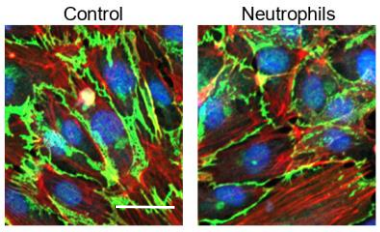
Supplementary Figure S1

ECs turned to spindle under stimulation of APL/NB4 cells. (A) ECs cultured with RPIM1640 medium. (B-C) The morphology change of ECs cultuered with NB4/APL-CM. Scale bar represents 10μm.



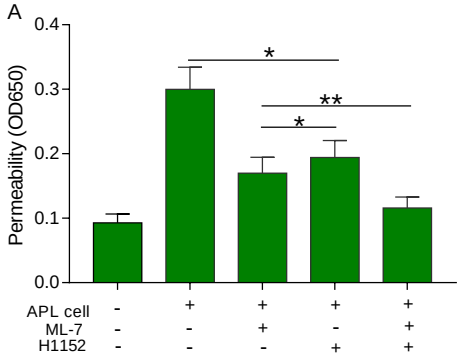
Supplementary Figure S2

We have performed experiments exploring the effect of neutrophils on endothelial cells (ECs). We found that they didn't damage ECs under normal conditions. Scale bar represents 10μm.



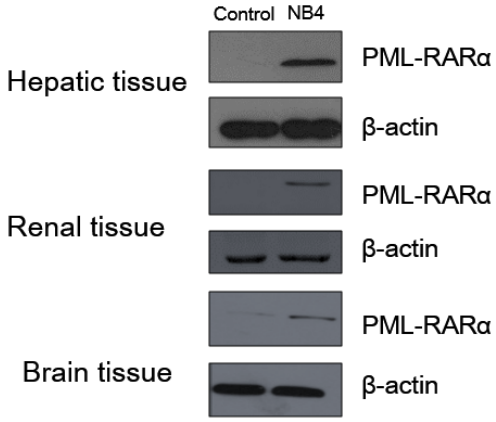
Supplementary Figure S3

MLCK and ROCK contribute to impaired barrier function. Pretreatment with MLCK inhibitor ML-7(10-4 mol/L) or/and with ROCK inhibitor H1152 (2.5μmol) for 1h, then incubated with APL cells for 16h. (A) Permeability of experimental endothelium to albumin. *p<0.05, **p<0.01. Data representive are from 6 independent experiments and represent as mean ± SD.



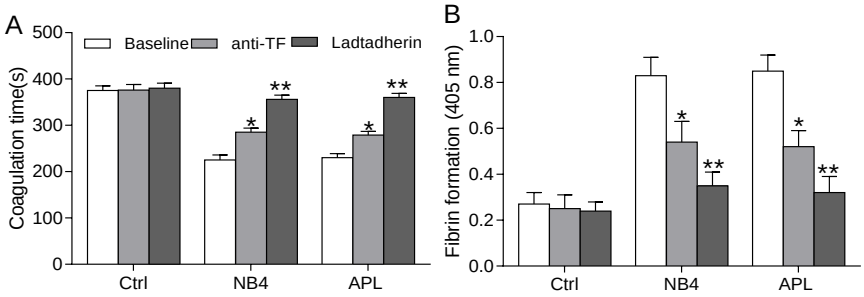
Supplementary Figure S4

Expression of PML-RARα fusion protein in mice tissue. Compared with control group, experimental group revealed expression of PML-RARα fusion protein in hepatic, renal and brain tissue.



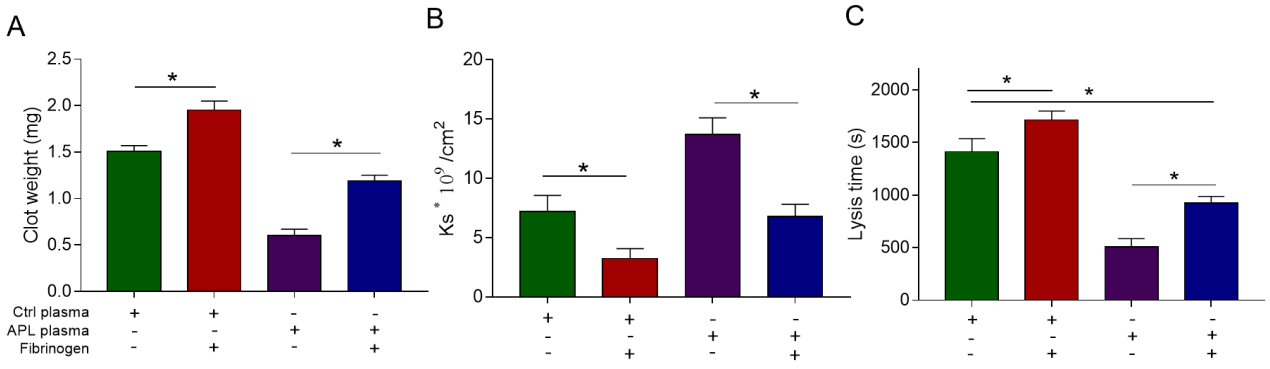
Supplementary Figure S5

Inhibition assay of procoagulant activity of ECs treated by APL/NB4 cell medium. (A-B) Inhibition assay using ECs treated by RPIM1640 medium as control. Stimulated ECs were incubated with anti-TF antibody and ladtadherin. Every data represents 3 independent experiments and showed as mean ± SD. *p<0.05 vs Baseline group; **p<0.01 vs anti-TF group.



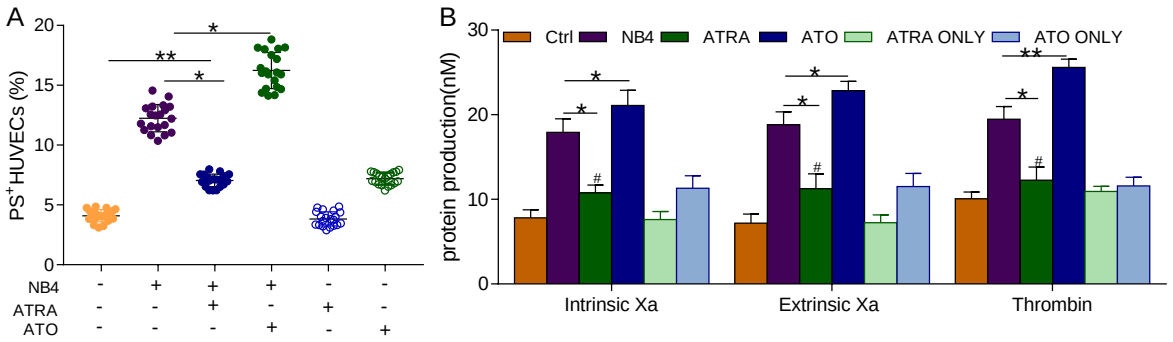
Supplementary Figure S6

Additional Fbg elevates the character of plasma clot. (A) The thrombus weight of the blood samples from healthy volunteers and APL patients with or without additional fibrinogen. (B) The average fibrin clot pore size (Ks) of the samples from (A). (C) Clot lysis assays were performed using 1 nM t-PA, and the lysis time was recorded for the four groups. *p<0.05. Data are from 6 independent experiments and represent as mean ± SD.



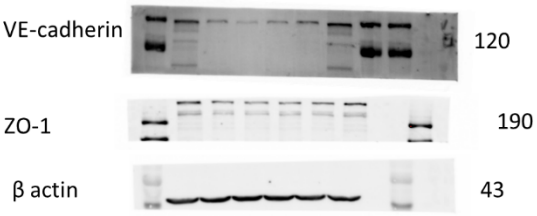
Supplementary Figure S7

ATRA decreases the procoagulant activity of ECs via inhibiting PS exposure. (A) PS exposure on HUVECs over the course of treatment with NB4 only or NB4 with ATRA or ATO was measured using lactadherin labeling and flow cytometry. (B) The production of coagulation proteins for HUVECs was measured from different experimental groups. *p<0.05, **p<0.01; #p<0.05 vs the data in the control group.

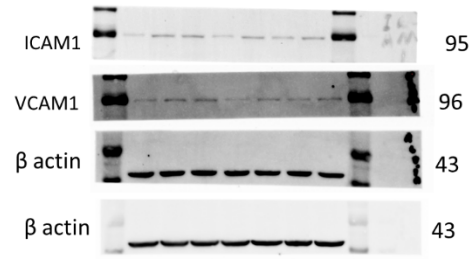


Supplementary Figure S8

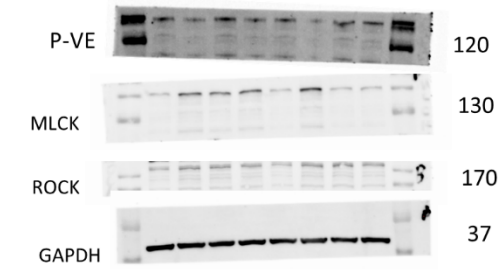
F1b



F3a+F4e



F3g and F3j



F5f