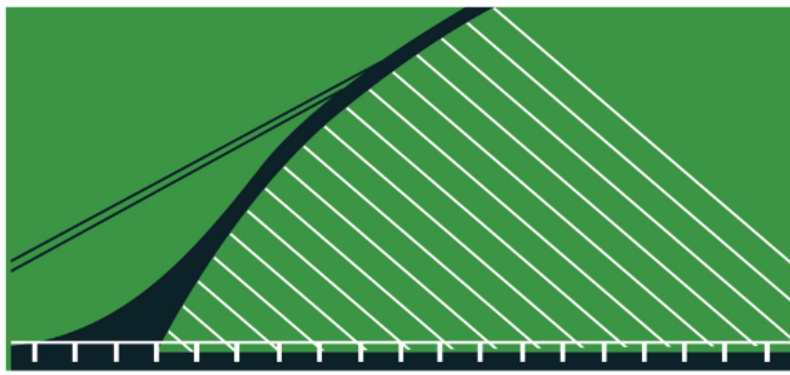


EAHAD
European Association for Haemophilia
and Allied Disorders

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19th ANNUAL CONGRESS
OF THE EUROPEAN ASSOCIATION
FOR HAEMOPHILIA AND ALLIED DISORDERS

VGA039, a protein S-targeting monoclonal antibody, restores hemostasis in von Willebrand disease patient blood in an *ex vivo* bleeding model

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Disclosure for Yumiko Sakurai

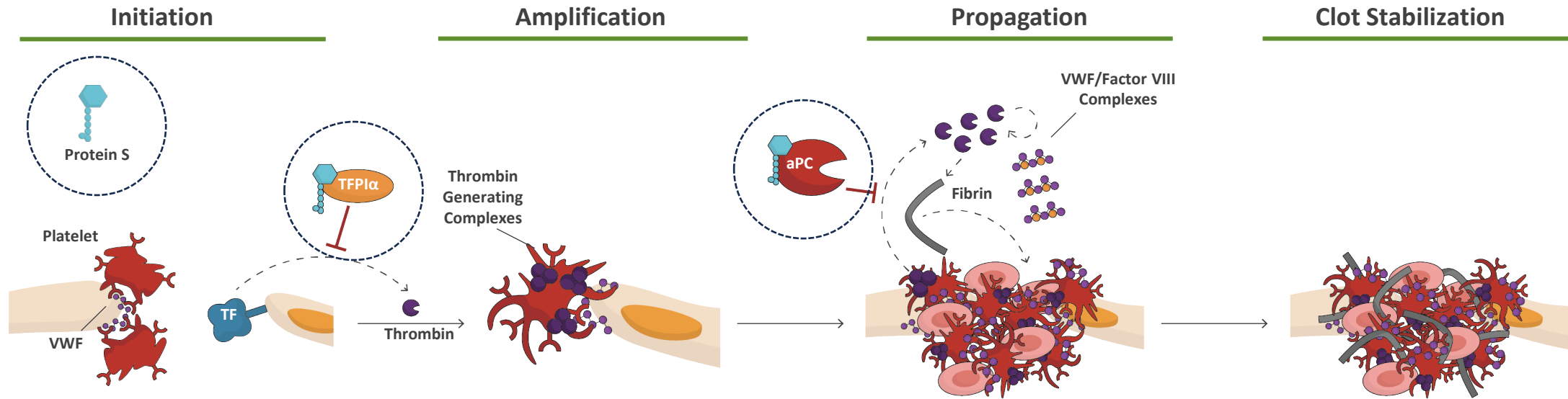
In compliance with COI policy, ESPEN requires the following disclosures to the session audience:

Shareholder	No relevant conflicts of interest to declare.
Grant / Research Support	Star Therapeutics
Consultant	No relevant conflicts of interest to declare.
Employee	No relevant conflicts of interest to declare.
Paid Instructor	No relevant conflicts of interest to declare.
Speaker Bureau	No relevant conflicts of interest to declare.
Other	No relevant conflicts of interest to declare.

Presentation includes discussion of the following off-label use of a drug or medical device:

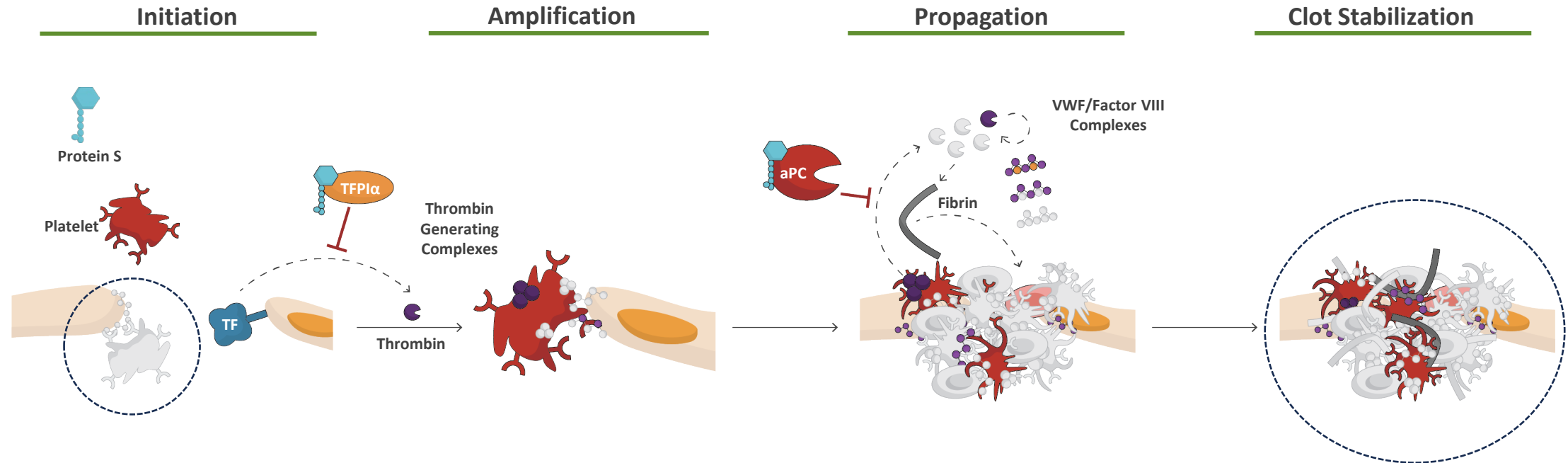
Not applicable

Protein S is a cofactor for TFPI α and activated protein C pathways



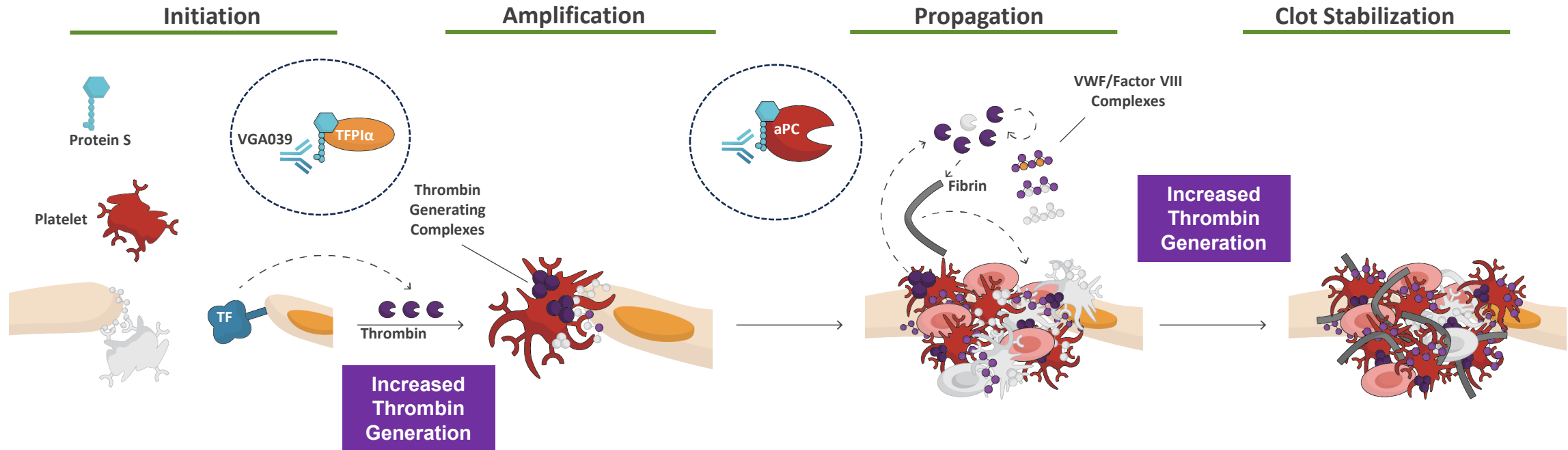
Leong et al., Blood 2022

VWD pathophysiology is associated with insufficient platelet adhesion and unstable clot formation



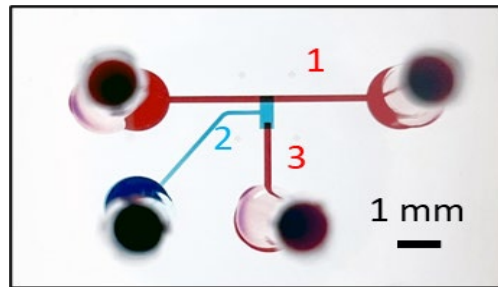
Leong et al., Blood 2022

VGA039 is a fully human monoclonal antibody targeting protein S with dual action on the TFPI α and activated protein C (aPC) pathways

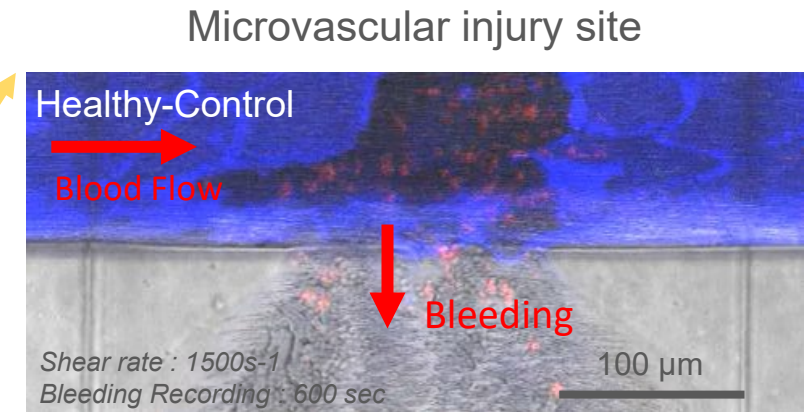
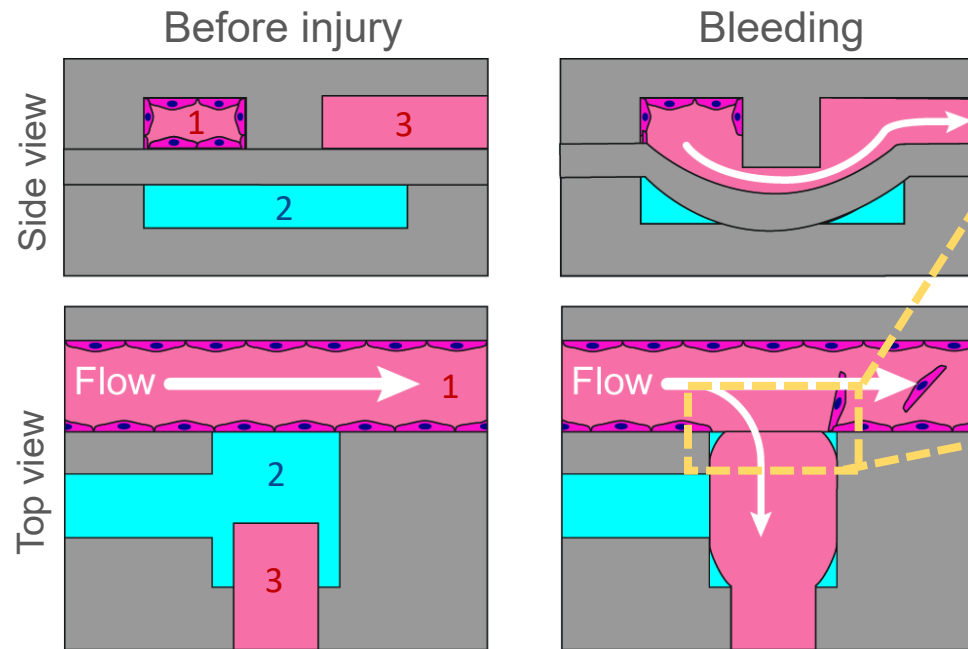


Leong et al., Blood 2022

Hemostasis-on-a-chip



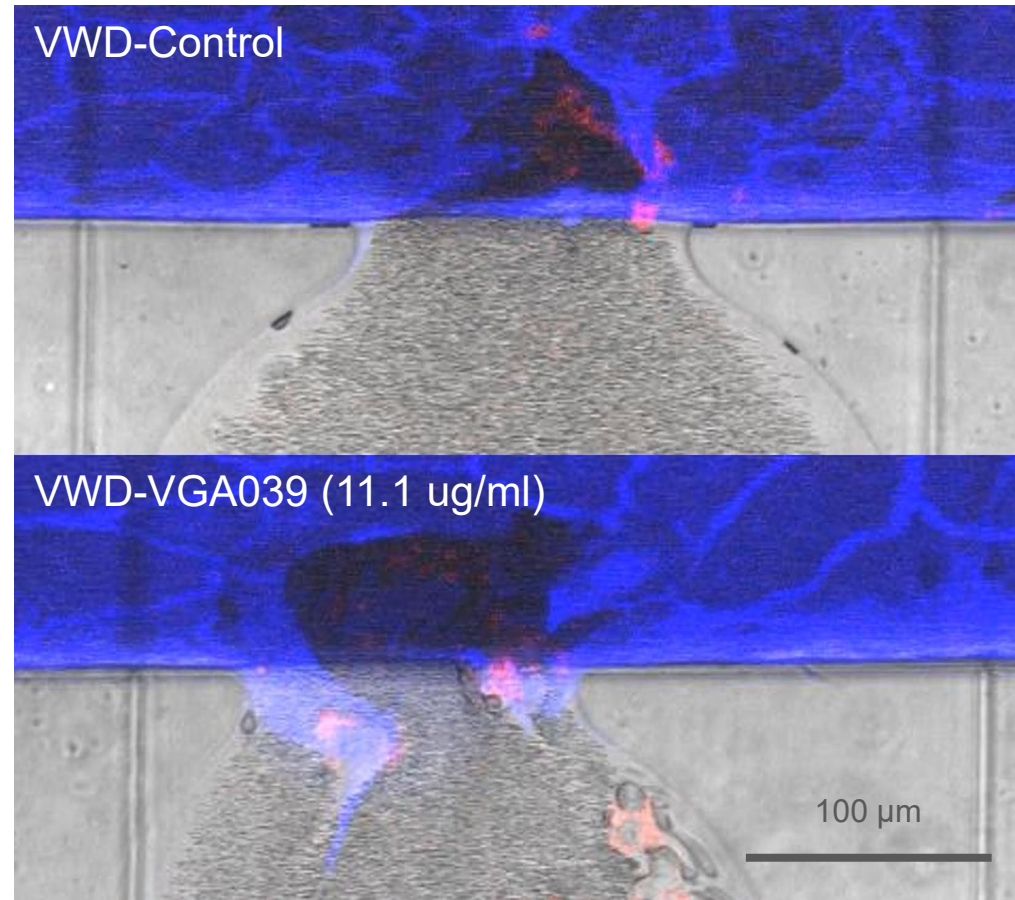
1. Endothelialized microvascular channel
2. Valve channel under flexible membrane
3. Bleeding outlet channel



- Blue: Endothelial Cells
- Red: Platelet
- Yellow: Platelet/fibrin colocalization
- Green: Fibrin

We assessed the effect of VGA039 in whole blood from patients with VWD

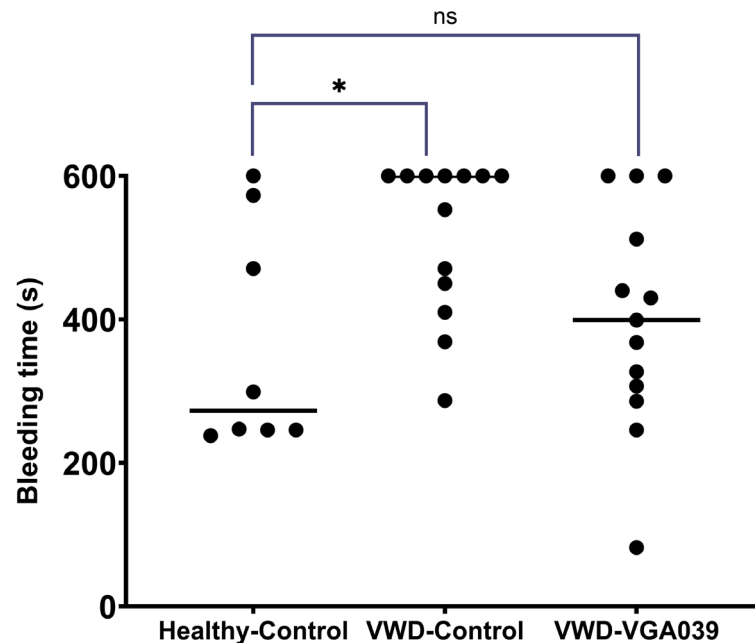
VGA039 improves *ex vivo* bleeding time and accelerated platelet deposition and fibrin accumulation in blood samples from VWD



Blue: Endothelial Cells
Red: Platelet
Yellow: Platelet/fibrin colocalization
Green: Fibrin

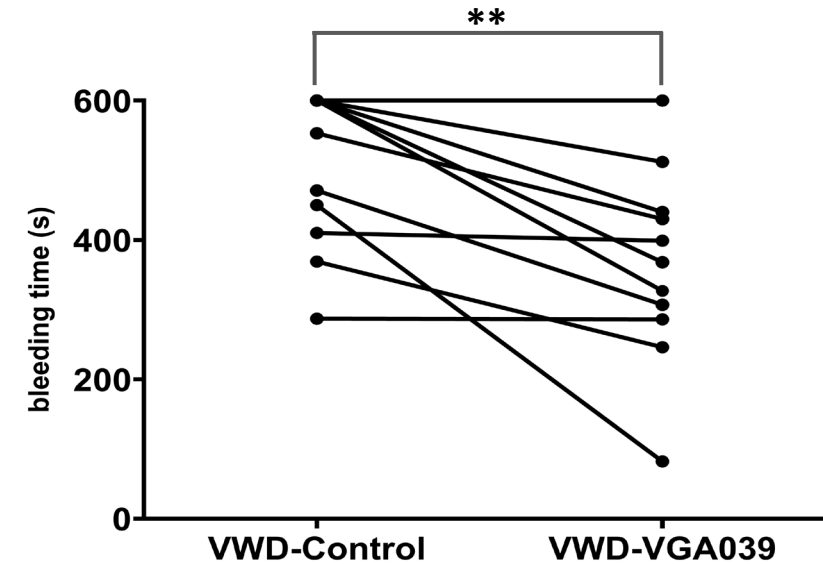
Median bleeding time of VWD blood treated with VGA039 demonstrates the ability of VGA039 to restore hemostasis

Ex-vivo Bleeding Time in Healthy, VWD controls and VWD-VGA039 treated samples



(*: $p=0.02$, Mann-Whitney U test, ns: not significant)

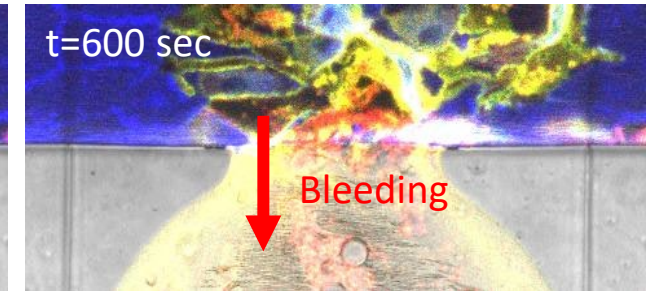
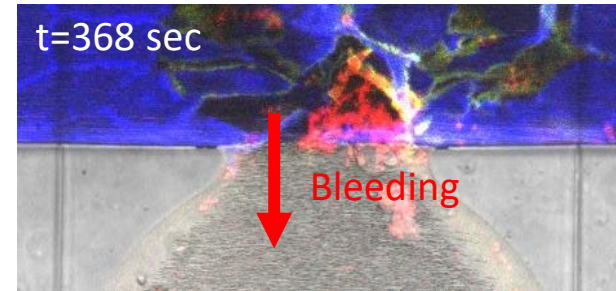
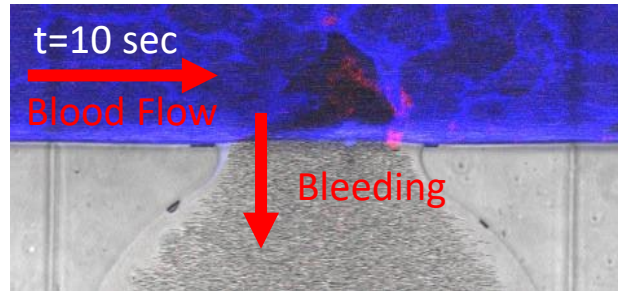
Change in *Ex-Vivo* Bleeding Time in VWD control and VWD-VGA039 treated samples



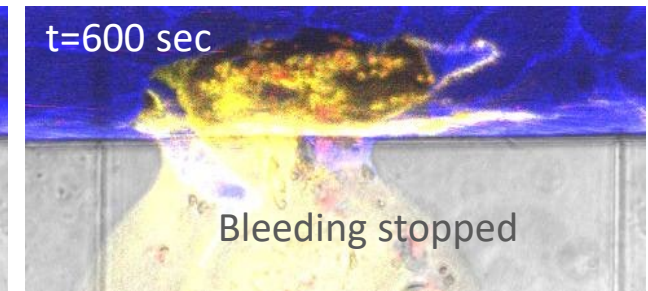
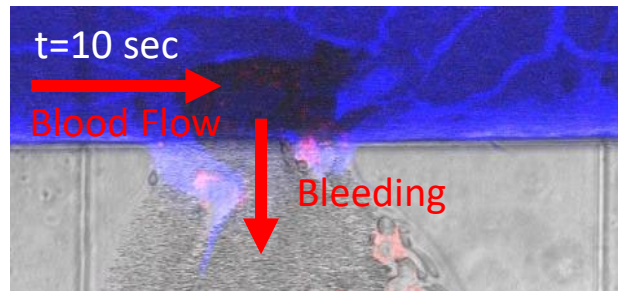
(**): $p=0.002$, Wilcoxon test)

VGA039 accelerate platelet deposition and fibrin accumulation in blood samples from VWD

VWD-Control



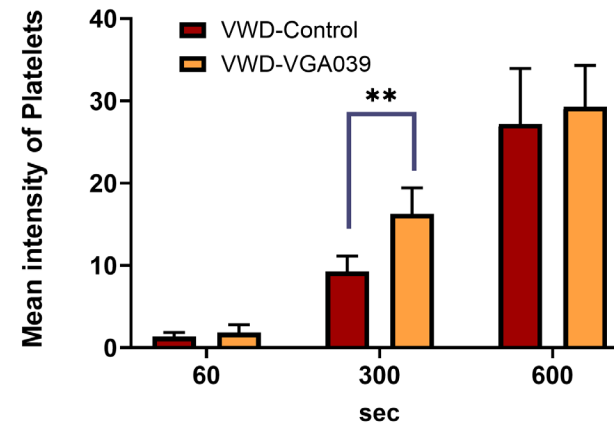
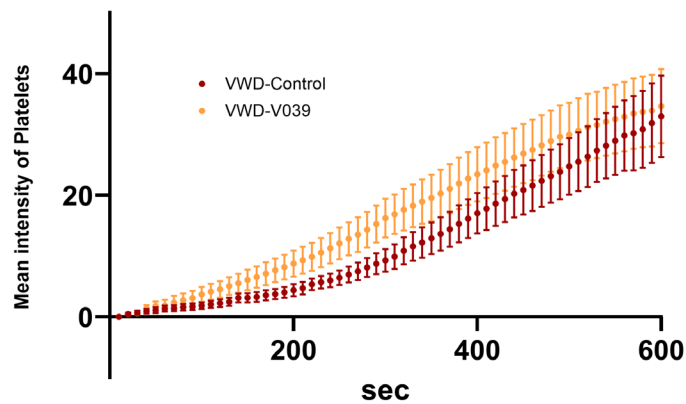
VWD-VGA039



Blue: Endothelial Cells
Red: Platelet
Yellow: Platelet/fibrin colocalization
Green: Fibrin

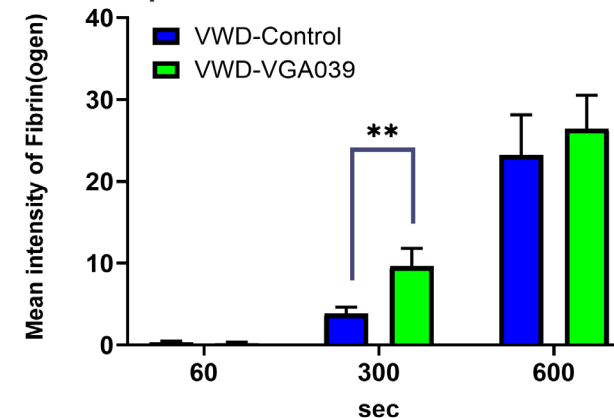
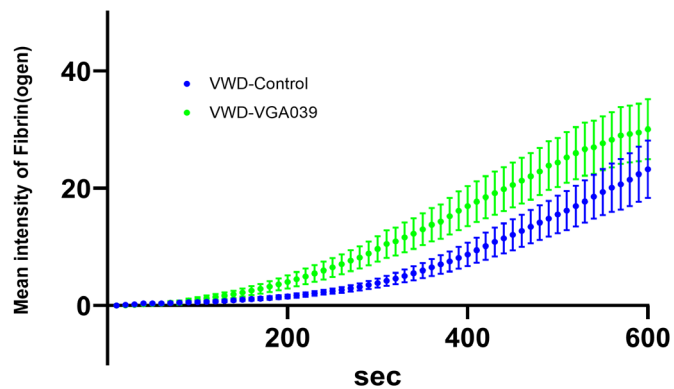
VGA039 significantly accelerates platelet deposition and fibrin accumulation in VWD patient blood samples

Mean intensity of platelet deposition



(**): p=0.004, Student t-test

Mean intensity of fibrin deposition



(**): p=0.008, Student t-test

Conclusions

- VGA039 significantly shortens *ex vivo* bleeding times in VWD blood samples in our hemostasis-on-a-chip model
- Reductions in *ex vivo* bleeding times were associated with accelerated rates of platelet deposition and fibrin accumulation
- We conclude that the dual action of VGA039 on TFPI α and activated protein C can restore hemostasis in blood from patients with bleeding disorders such as VWD