

# Modifications de l'hémostase sous assistance circulatoire mécanique (ACM)

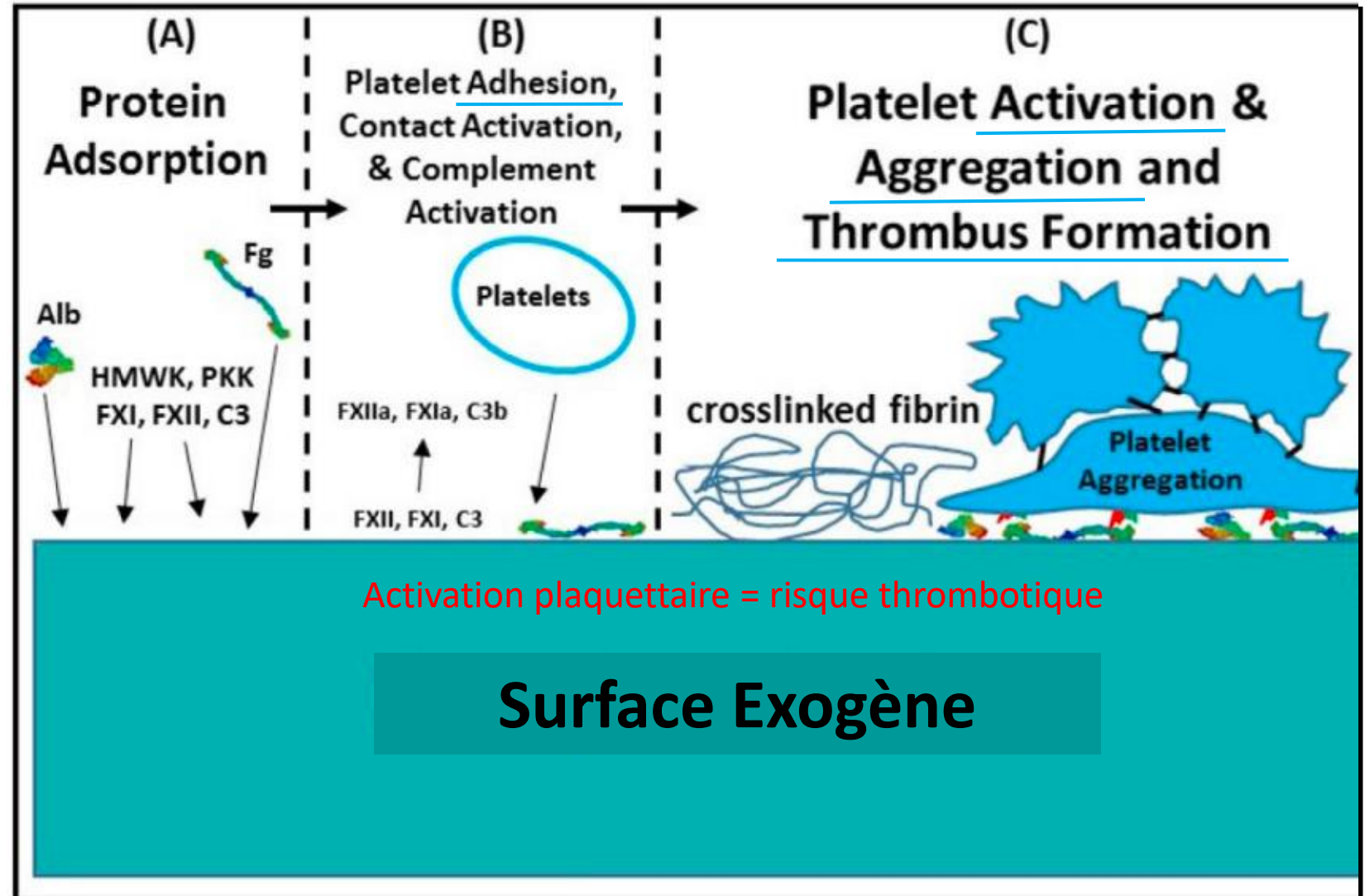
# Conflit d'intérêt

- Aucun

## Adsorption protéique

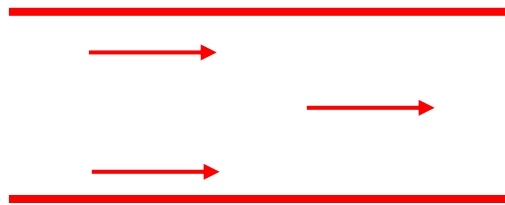
favorisée par les interactions électrostatiques et hydrophobes:

- Fibrinogen
- Fibronectin
- von Willebrand factor
- FXI-FXII
- Kininogen
- Prekallikrein

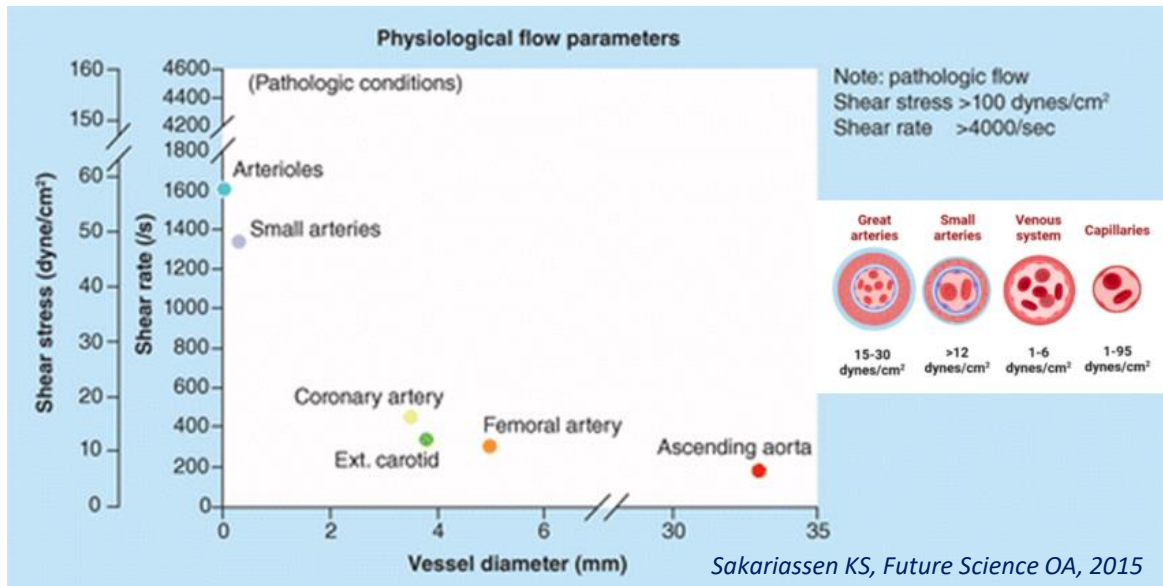
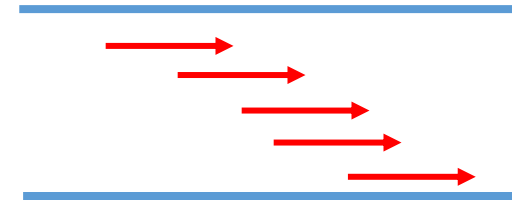


**Contrainte de cisaillement (shear stress):** forces frictionnelles exercées par le flux sanguin contre la paroi vasculaire, déterminé par le **débit sanguin**, la **viscosité du sang** et le **diamètre du vaisseau**

Vascular system



ACM

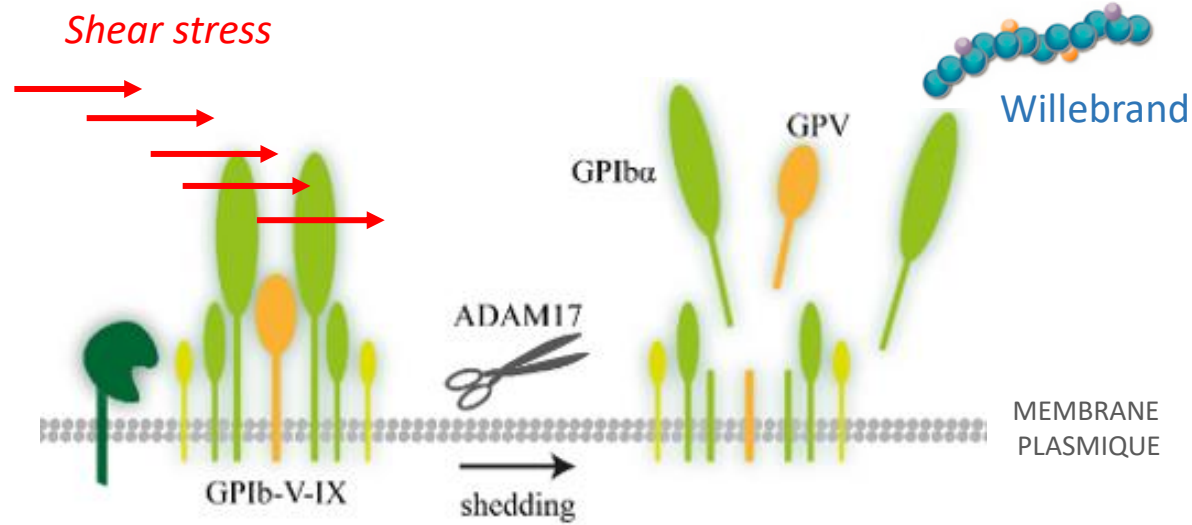


**Table 1. Typical range of wall shear rates and stresses in blood vessels**

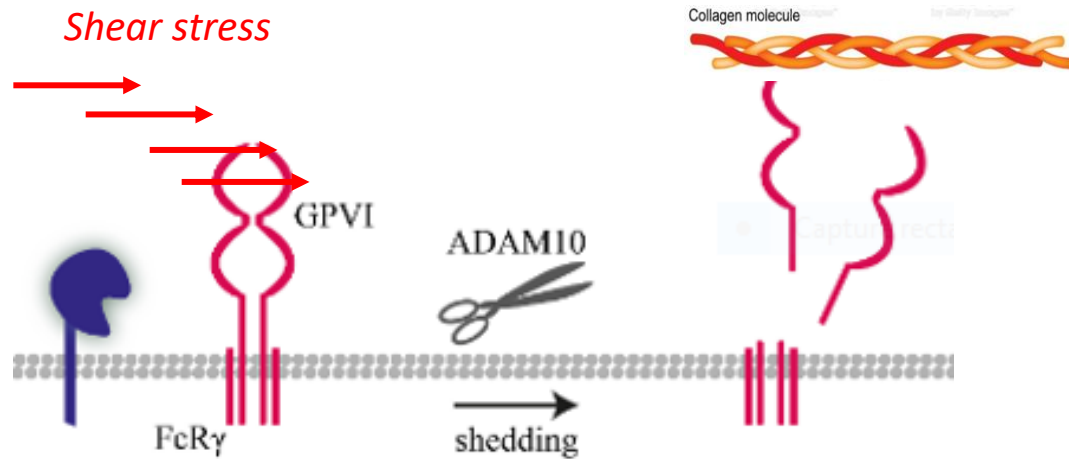
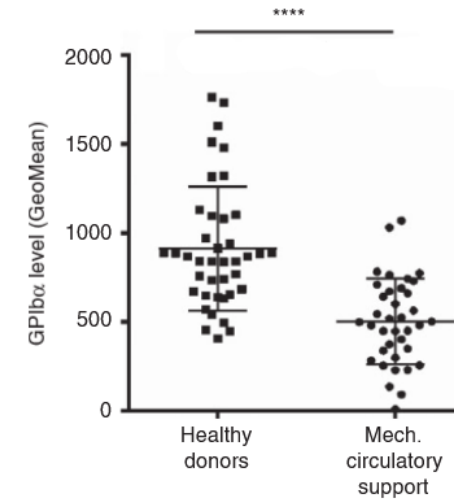
Blood vessel	Shear rate (/s)	Shear stress (Pa)
Large arteries*	300-800	1.4-3.6
Arterioles*	450-1600	2-7.2
Veins*	15-200	0.07-0.9
Stenotic vessels*	800-10 000	3.6-45
Axial flow LVAD†	—	600
Centrifugal flow LVAD†	—	150-230

X100  
X20-30

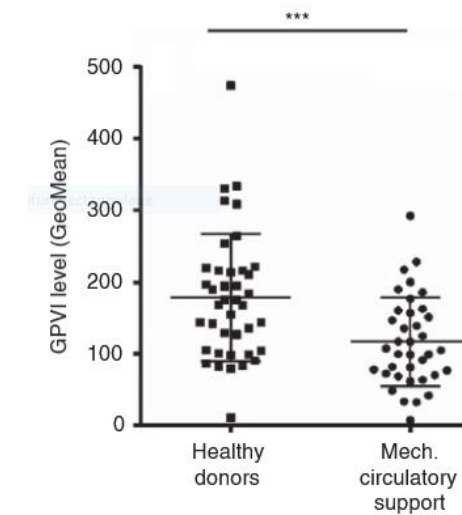
# TAUX DE CISAILLEMENT ÉLEVÉS ET PERTE DES RÉCEPTEURS PLAQUETTAIRES



Récepteur  
Willebrand

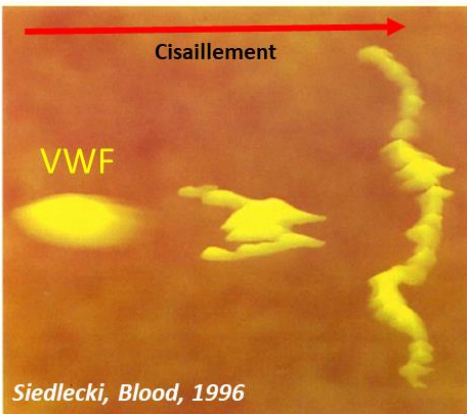


Récepteur  
collagène



# HÉMOSTASE, FACTEUR WILLEBRAND ET CONTRAINTES DE CISAILLEMENT (SHEAR STRESS)

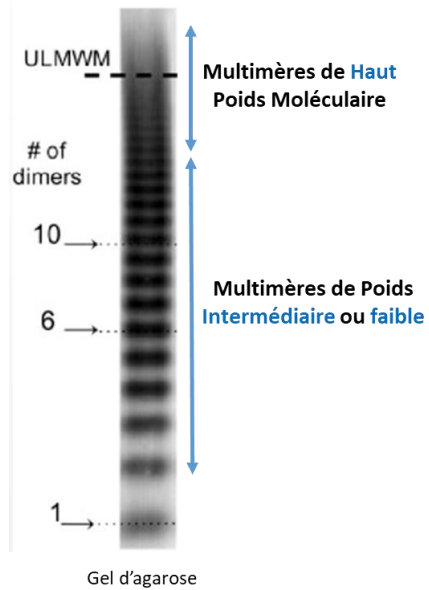
No Shear → Shear Applied by AFM Probe → 35 Dyn/cm<sup>2</sup> Applied by Rotating Disk



Globular vWF      Short Extended Chain      Extended Chain

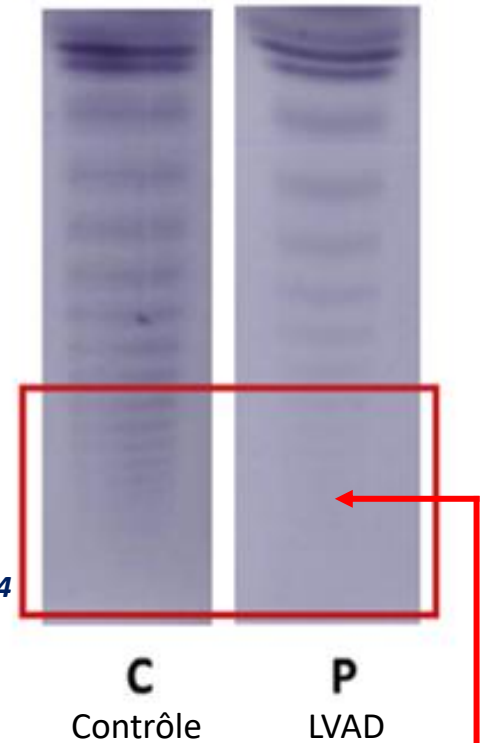
Shear-Dependent Changes in the Three-Dimensional Structure of Human von Willebrand Factor

Conformation native  
Non fonctionnelle



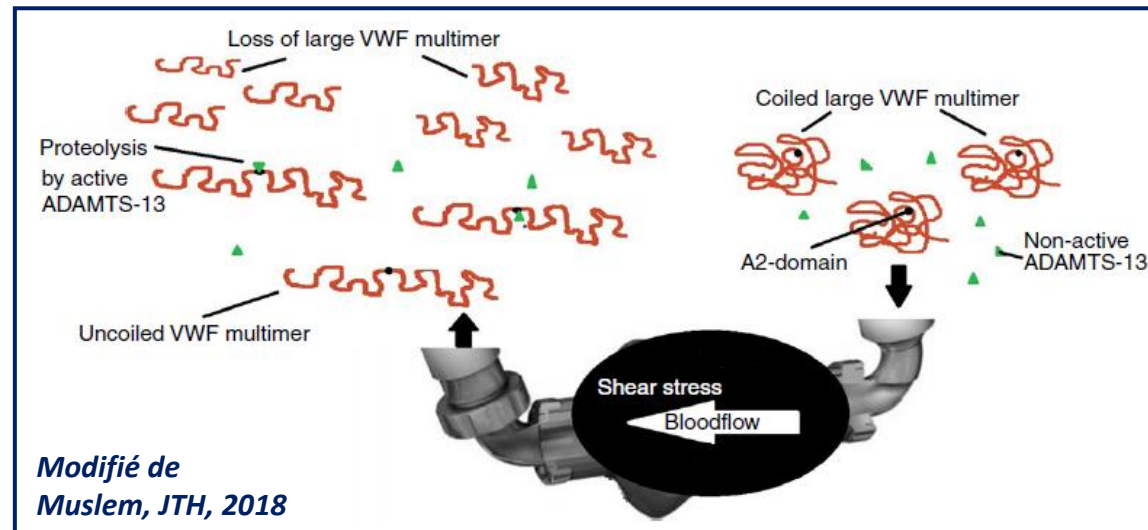
ACM

Fiore, JTCS, 2014



Perte des multimères de haut poids moléculaires et de fct du VWF

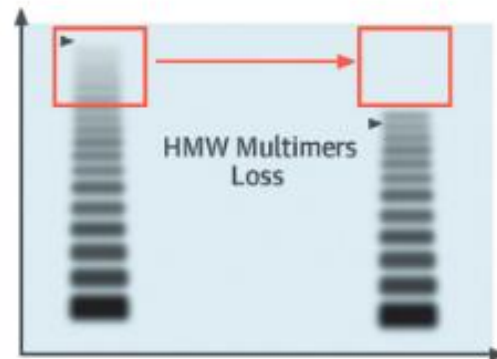
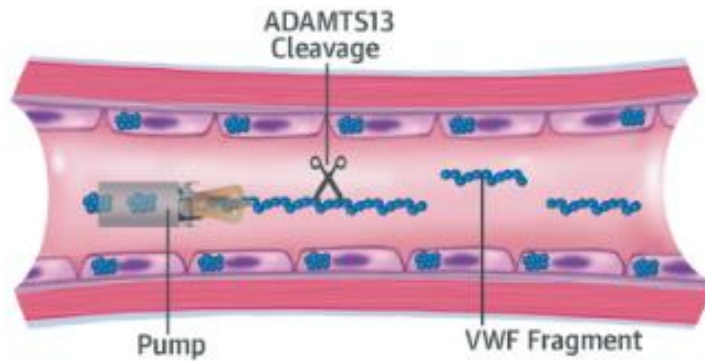
=  
**Maladie de Willebrand Acquis**  
=  
**Risque hémorragique**



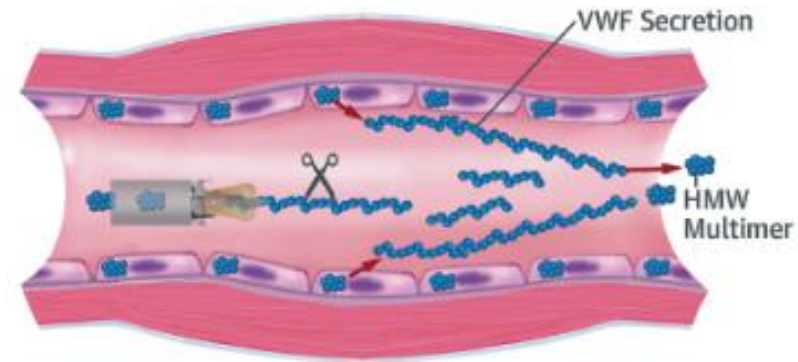


# PULSATILITÉ ET WILLEBRAND ACQUIS

## Low Pulsatility



## Normal Pulsatility







# MODIFICATIONS DE L'HÉMOSTASE SOUS ACM

