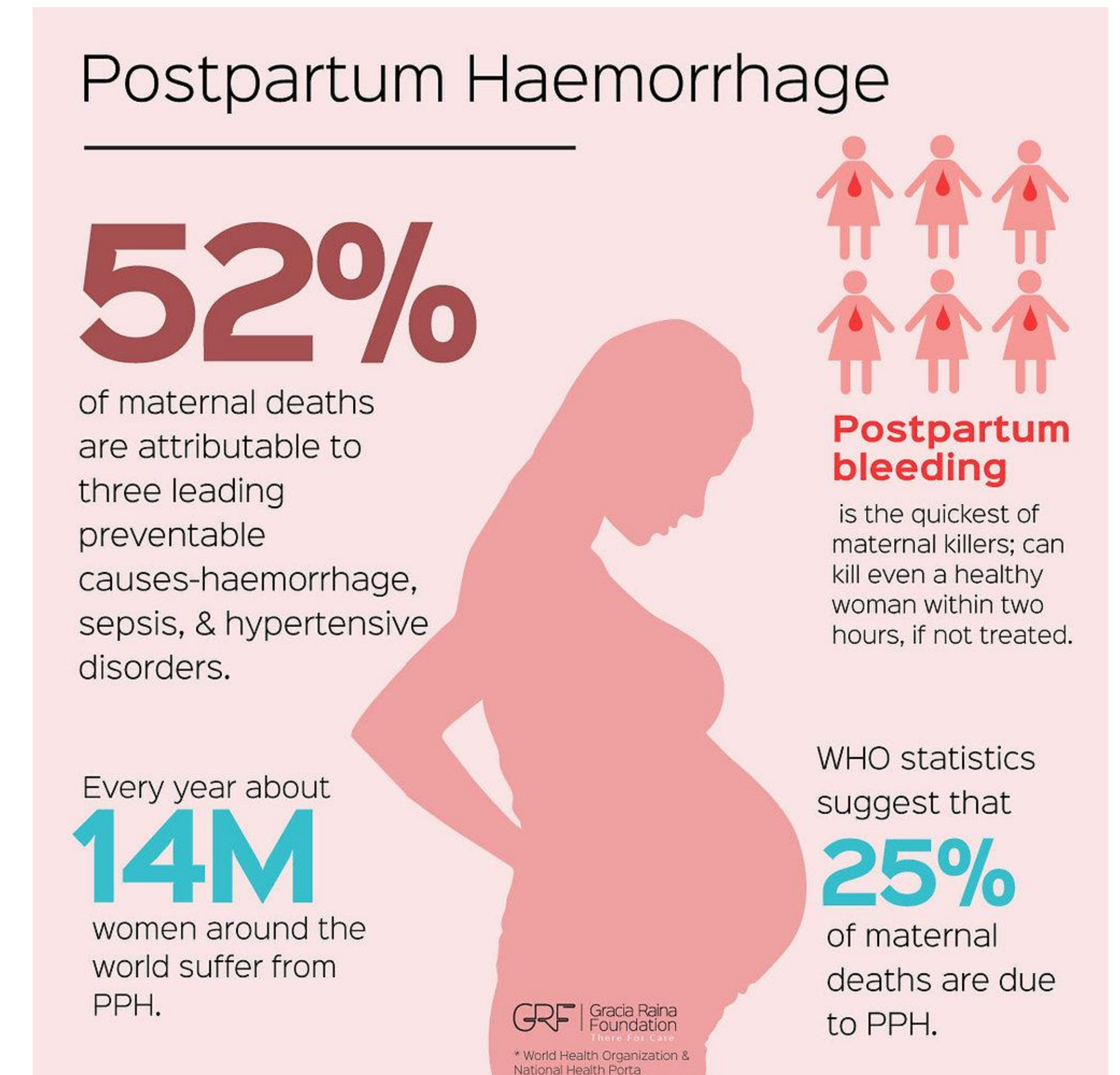


Platelet dysfunction in postpartum hemorrhage and transfusion approaches assessed in a microfluidic platform

Emily P. Mihalko, PhD, Riya Sharma, Morgan Mollen, Hyagriv N. Simhan, MD MS,
Susan M. Shea, PhD, Matthew D. Neal, MD FACS, Grace Lim, MD MSc MBA

Background & Hypothesis

- Postpartum hemorrhage (PPH) affects **14 million women** annually and accounts for **27.1%** of maternal deaths
- Non-targeted, non-specific, and largely unoptimized therapies
- Acute obstetric coagulopathy can exacerbate bleeding and is associated with high rates of maternal morbidity and fetal death
- Primary hemostatic mechanisms have not been characterized
- Antifibrinolytics less effective in anemic states highlights the limitation of fibrin-centric models and underscores the importance of platelet-dependent mechanisms



We hypothesize that platelet dysfunction may exacerbate PPH and contribute to the failure of current treatment strategies to effectively control bleeding.

[1] Say et al., *Lancet Global Health*, 2014.

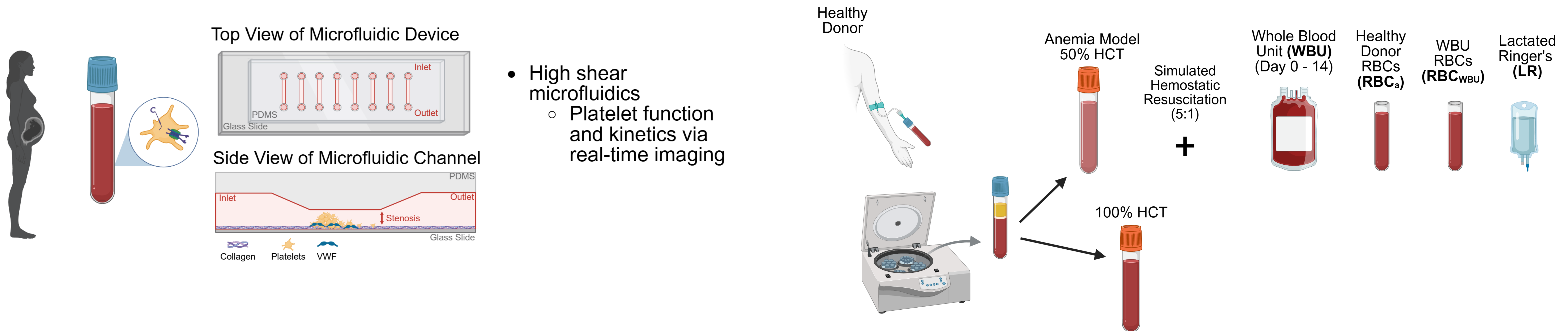
[2] Chiara M Corbetta-Rastelli et al., *Obstet Gynecol*, 2023.

[3] de Lloyd et al., *J Thromb Haemost.*, 2023.

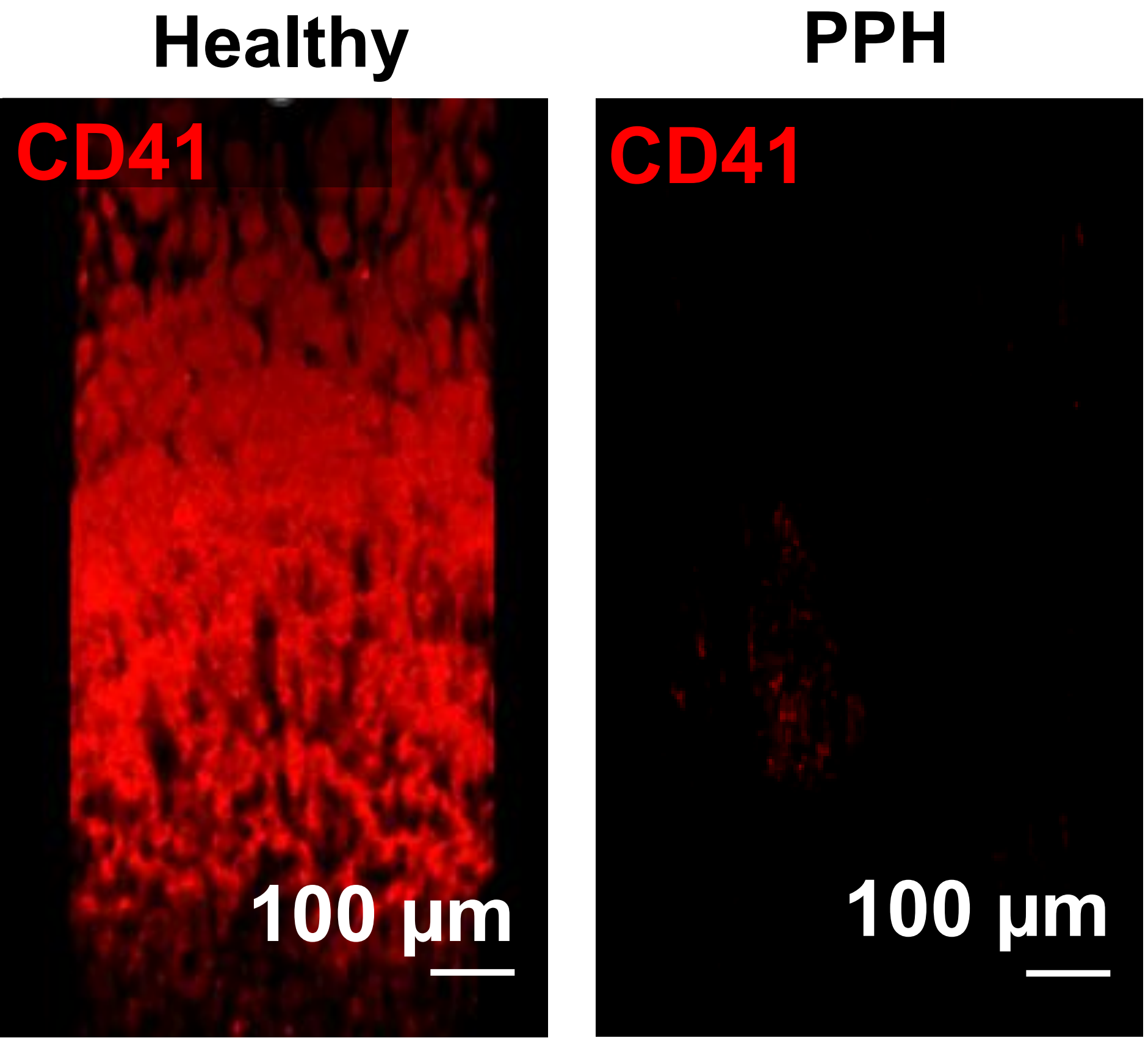
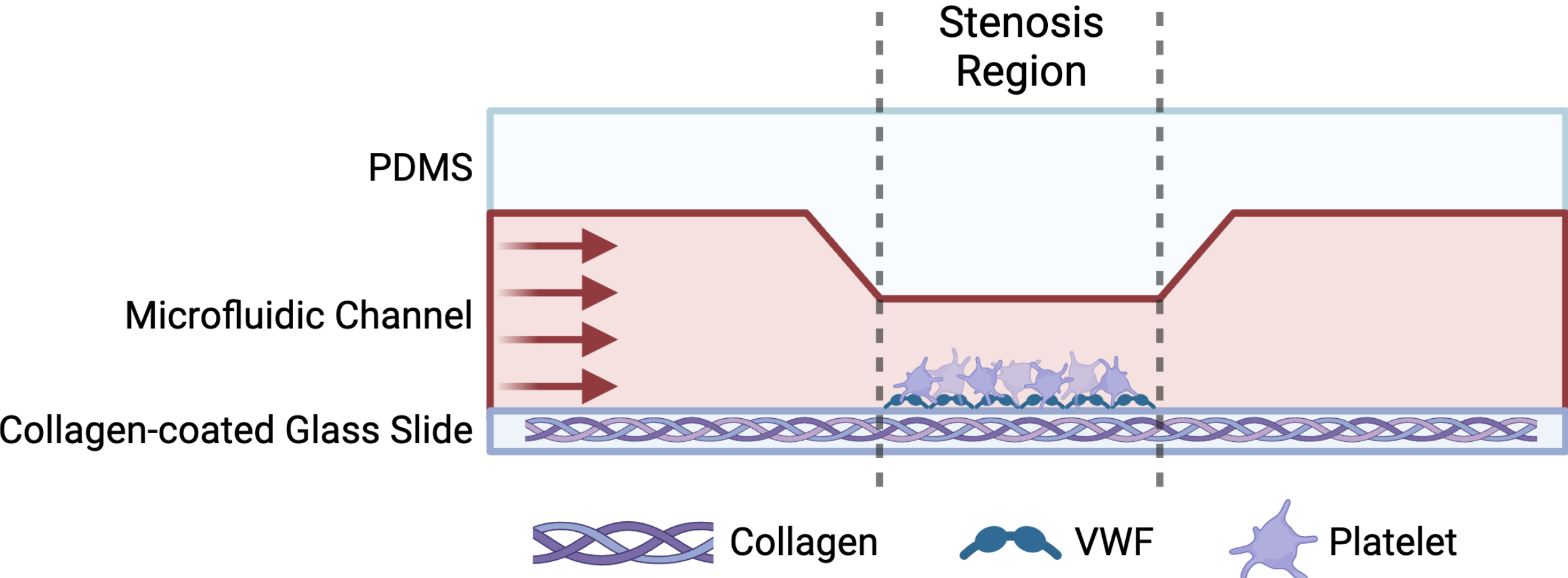
[4] Collins et al., *Blood Adv*, 2025.

Methods

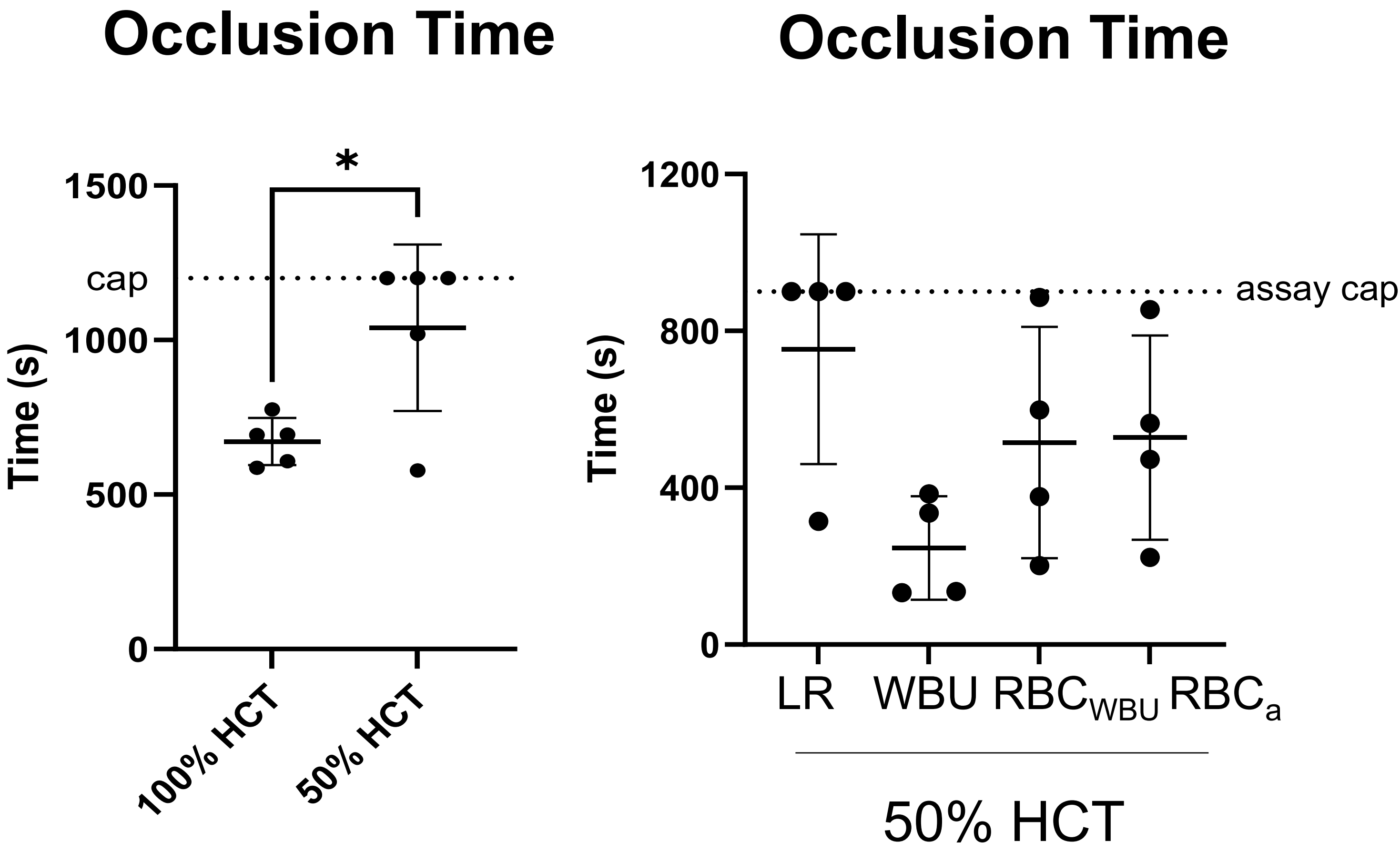
- PPH patient citrated whole blood sample capture prior to hemostatic resuscitation
 - Blood loss $>1000\text{mL}$ or blood loss accompanied by hemodynamic instability
- Microfluidic assay for physiologic high shear platelet function testing
- Anemia model and hemostatic resuscitation simulation in microfluidic device



Results



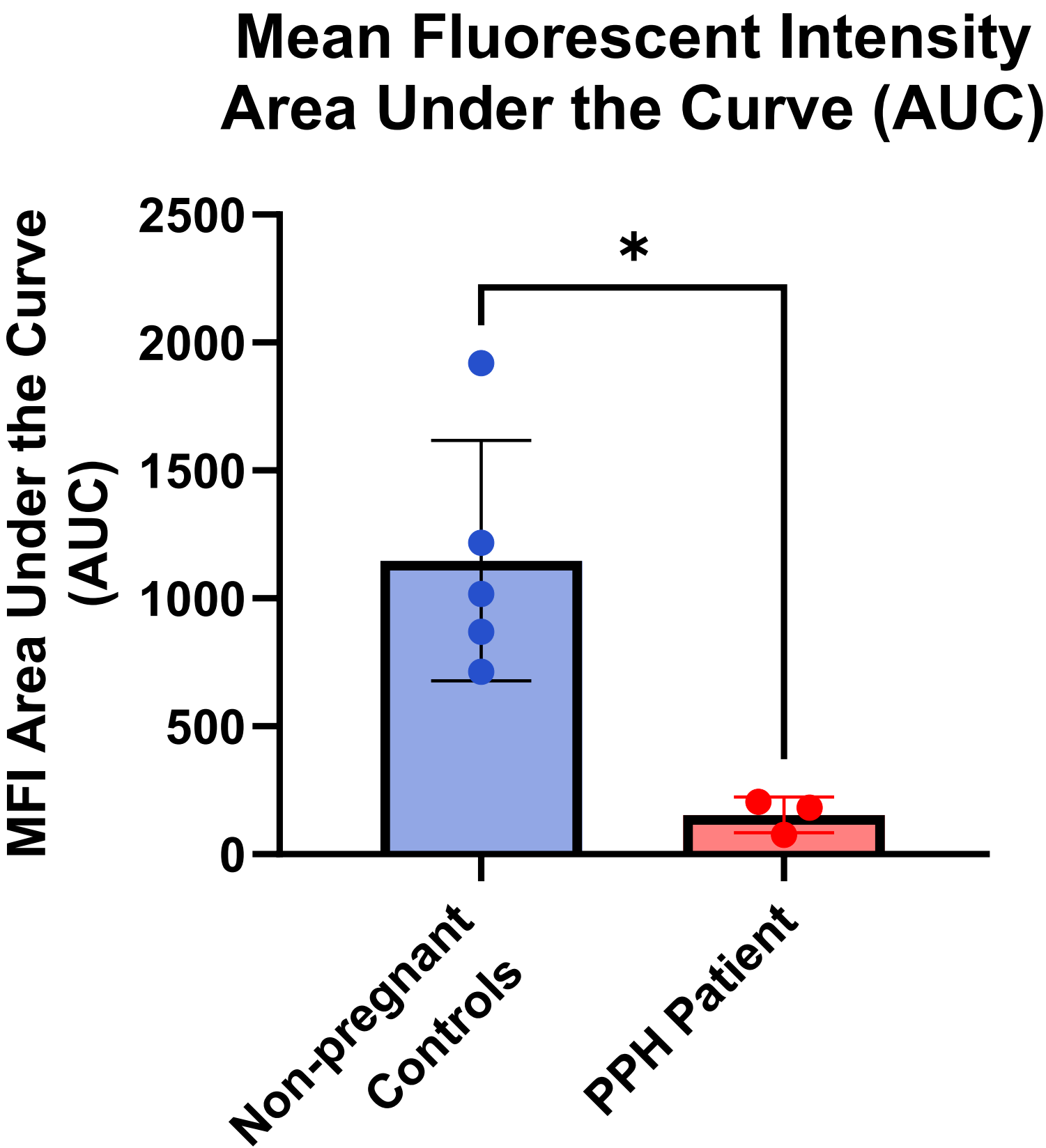
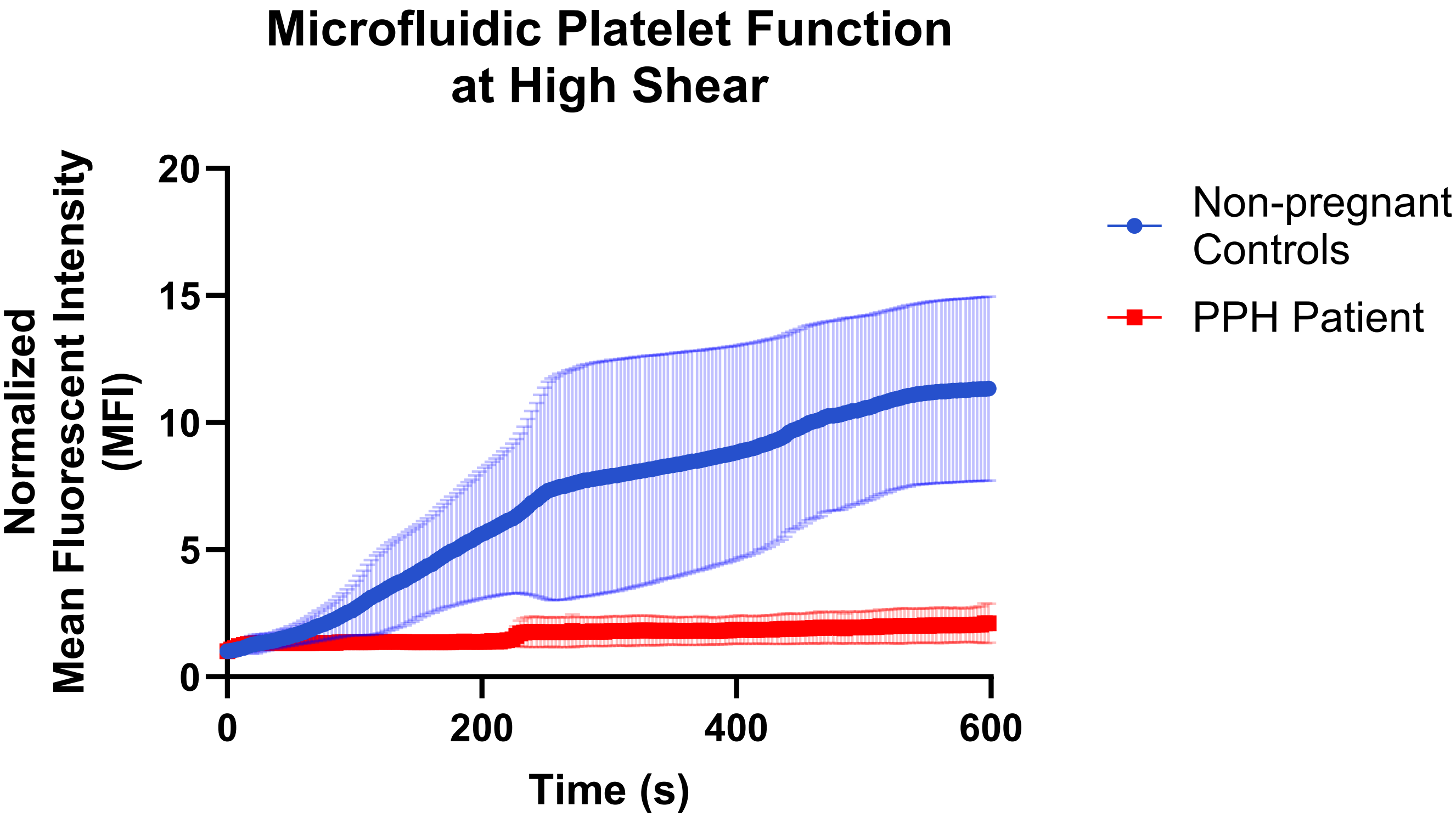
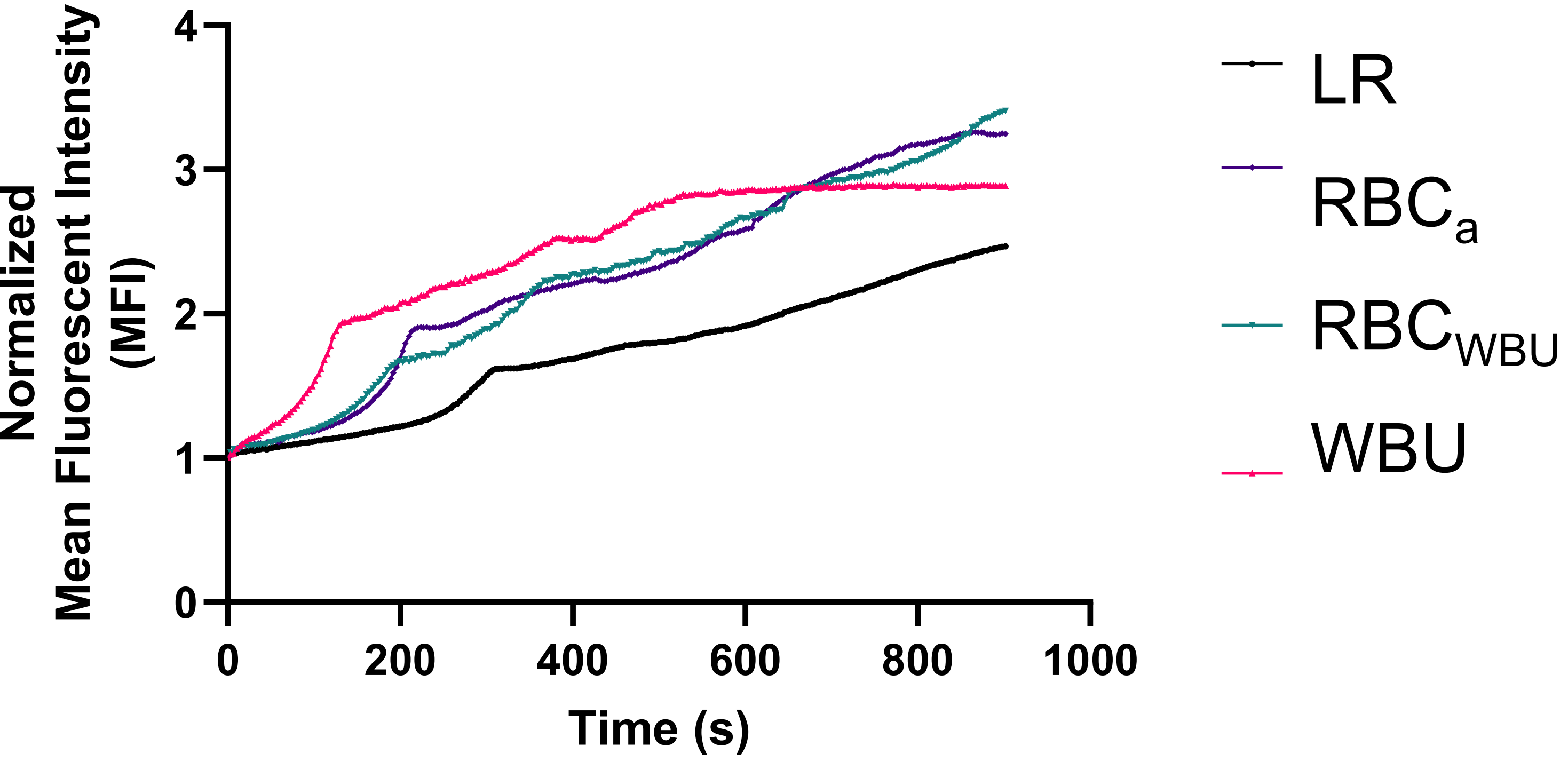
	100% HCT	50% HCT
Hemoglobin (g/dL)	11.3 $\pm$ 0.7	5.9 $\pm$ 1.2
Platelet Count ($\times 10^3$ cells/ μ L)	209 $\pm$ 33	180 $\pm$ 34
TEG 6s CK-R (min)	7.6 $\pm$ 1.9	8.7 $\pm$ 2.2
TEG 6s CK-K (min)	1.6 $\pm$ 0.4	1.2 $\pm$ 0.1
TEG 6s CK-Angle (degrees)	69.6 $\pm$ 4.6	73.9 $\pm$ 0.8
TEG 6s CK-MA (mm)	59.5 $\pm$ 3.2	64.3 $\pm$ 1.9



Endpoint Image
50% HCT + WBU



Platelet Deposition
CD41_{Recipient}



Conclusions

- PPH may be exacerbated by an acute platelet dysfunction that is yet to be fully investigated, potentially involving a pathology of platelet adhesion mechanisms.
- In an anemia model, microfluidic assays indicate better hemostatic resuscitation with intact whole blood compared to isolated RBC groups and LR, potentially due to RBC dysfunction during isolation or dilution of mechanistically critical hemostatic factors at high shear.
- Upcoming studies will continue to study acute platelet dysfunction in PPH and will further investigate platelet receptors and plasma mediators, including von Willebrand Factor activity.

Acknowledgements

Trauma and Transfusion
Medicine Research Lab
(TTMRL)

Faculty

Matthew D. Neal, MD FACS

Susan M. Shea, PhD

Lab Members past and present

Pearlson Prashanth, PhD

Irona Khandaker, MD PhD

Riya Sharma

Lillian Scarlett

Keller Ulrich

Pierce Shipp

Morgan Mollen

Jack Killinger

Garrett Campbell

Katelin Rahn

Kaelyn McClain

Ronit Kar

Aarushi Bajpai

Amudan Srinivasan, MD

Elizabeth Andraska, MD

Nijmeh Alsaadi, MD

Reem Younes, MD

Abiha Abdullah, MD

Rida Zakar, MD

Laith Al Khraisat, MD

Kelly Williamson, PhD

Scott Donahue

Alyssa Falcione

Lauren Jackson

Robert Voinchet

Rassam Rassam

Devin Dishong

Refael Munitz

Nick Rocchio-Giordano

Jason Brandon

Juliana Briggs

Tvisha Subhasish

Victor Young

Skye Clayton

Lisa Vavro

Casey Hicks

Javonn Musgrove

Kestrel Merritt

Tirzah Griffin

Collaborators

Dr. Grace Lim

Dr. Ashley Brown | Brown Lab

Dr. Kim Thomas | Thomas Lab

Dr. Anirban Sen Gupta | Sen Gupta Lab

Dr. Dan Shiwarski | STEL

Dr. David Ku | Ku Lab

Dr. David Vorp

Dr. Phil Spinella

Dr. Hyagriv Simhan

Dr. Shahid Nimjee

Dr. Beverley Hunt

Dr. Homa Ahmadzia

TTMRL 



Thank you!

Emily Mihalko
epm35@pitt.edu

UPMC
LIFE CHANGING MEDICINE



University of
Pittsburgh

Trauma and Transfusion
Medicine Research Center