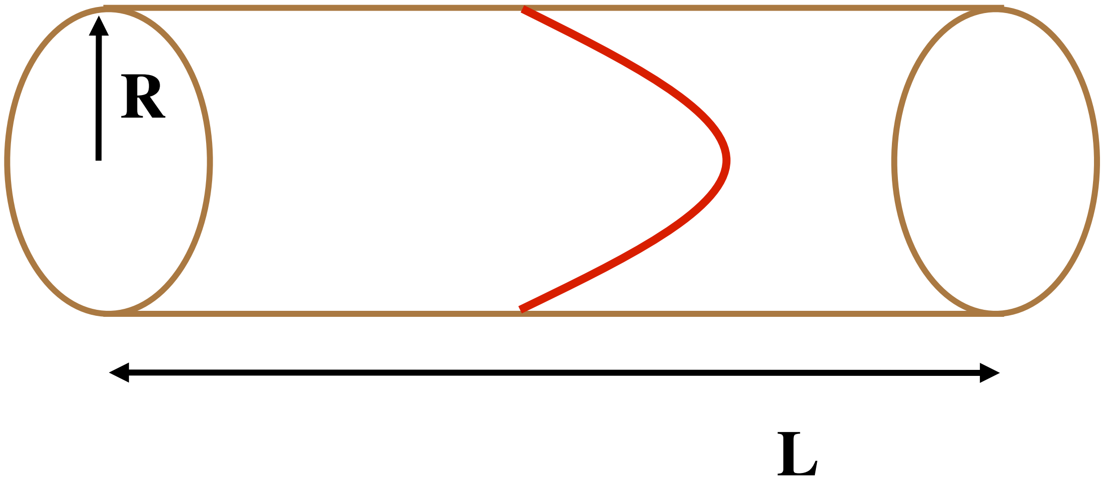
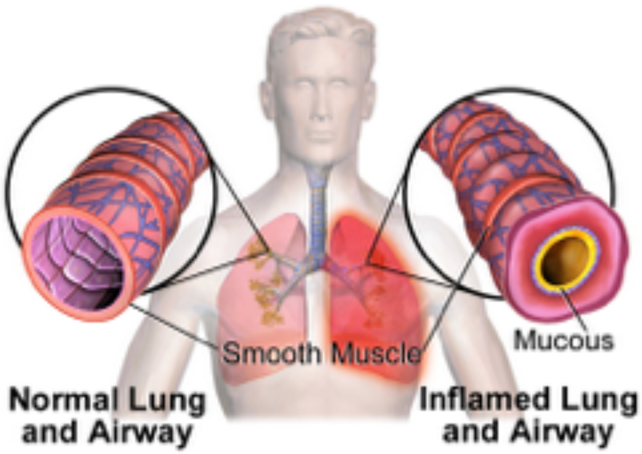
Biology 427 Biomechanics
Lecture23. Blood flow

- Some course details -- posters et al.
- Recap tube flow
- Problems of unsteadindess and pulsatile flows
- Blood flow and hematocrit variations
- Farheaus-Lindqvist effect
- Blood viscosity in the microcirculation

Circular vessel of constant radius R



$Q = \pi R^4 \Delta P / 8\mu L$ Hagen-Poiseuille law



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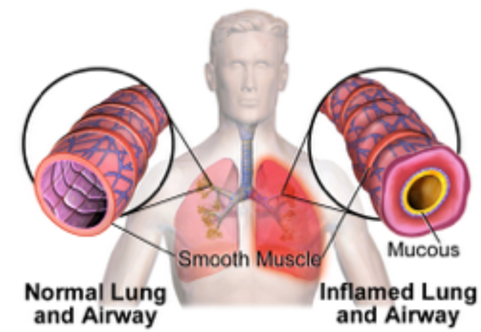
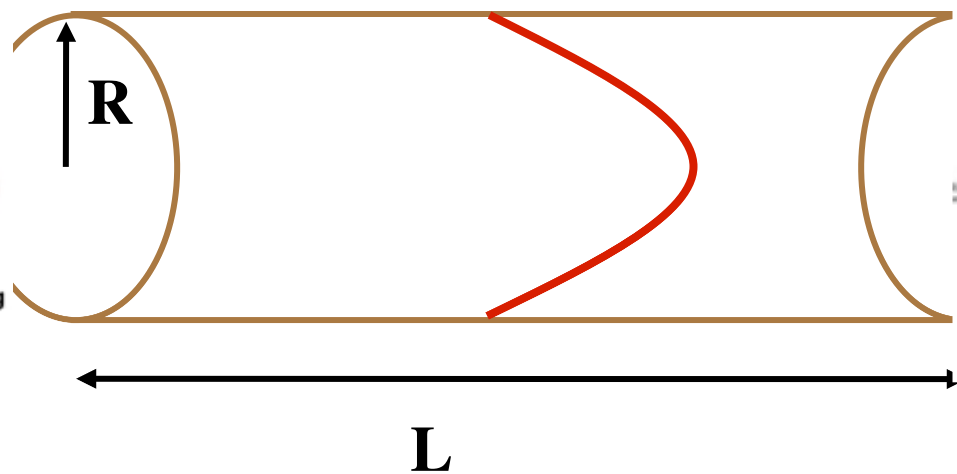
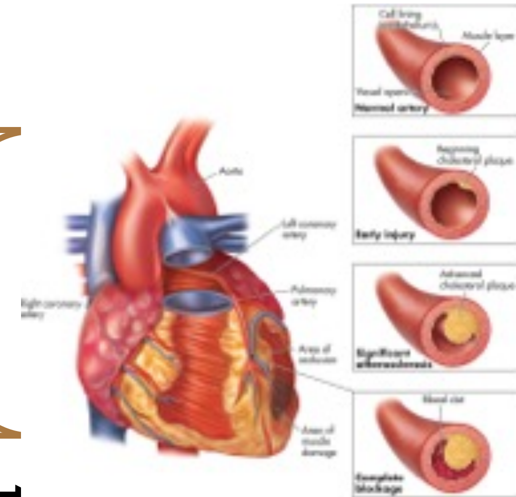
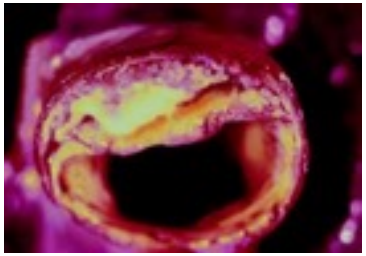


Asthma







$u(r) = \Delta P\,(R^2 - r^2)/4\,\mu\,L$

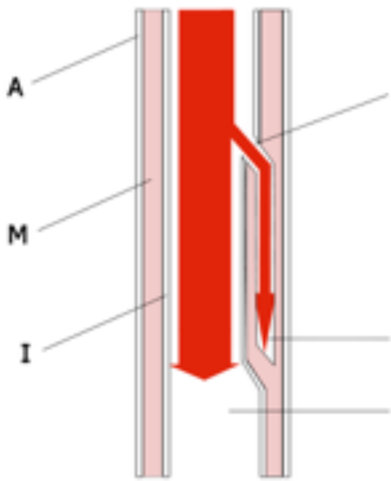
$\tau = \mu\, du/dr = -\Delta P\, r/\, 2\, L \qquad \tau = -\Delta P\, R/\, 2\, L$

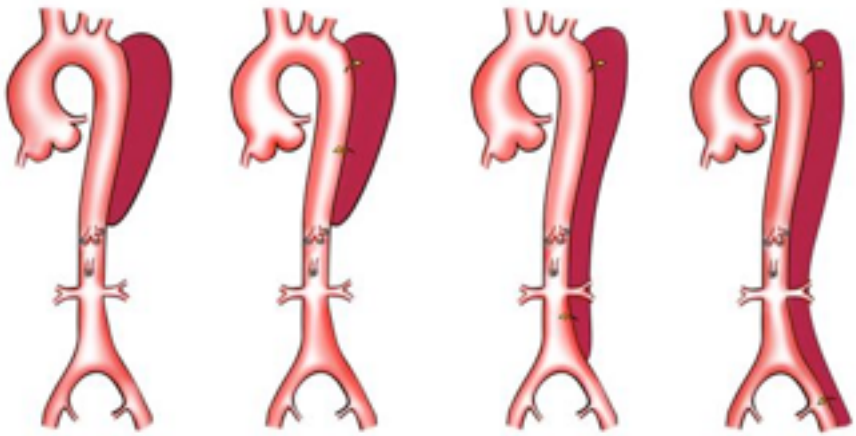
$\tau = -Q\, 2\mu/\pi R^3$

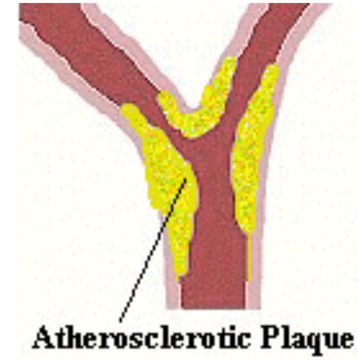
Atherosclerosis

Aortic dissection

$$\tau = \mu \frac{du}{dr}$$







Factors:
tube shape, unsteadiness, secondary flows, turbulence and unique features of blood cells

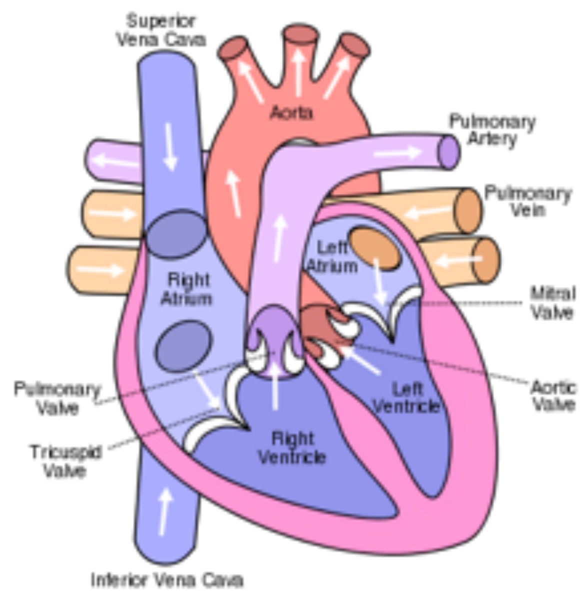
Hagen-Poiseuille law $Q = \pi R^4 \Delta P/(8\mu L)$

$\Delta P = Q\, 8\mu L/(\pi R^4)$

Shear stress $\tau = -Q\, 2\mu/(\pi R^3)$

Equivalent lengths

1.0	straight tube
1.4	45 deg bend
1.4	T junction
2.5	90 deg. bend



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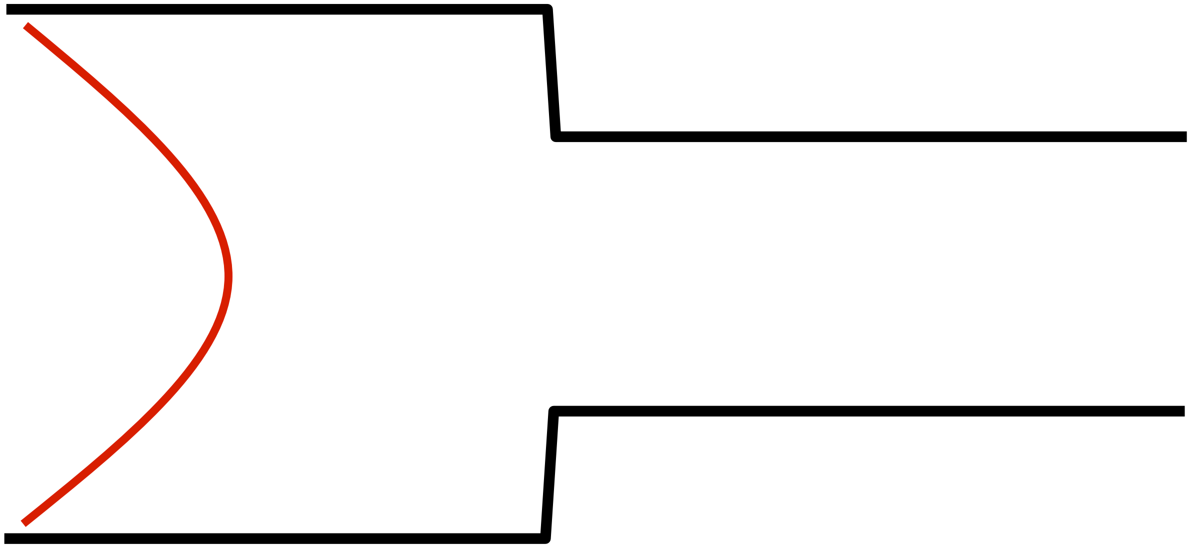
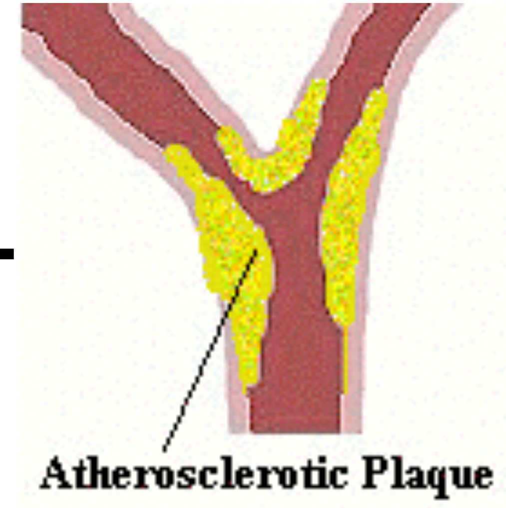
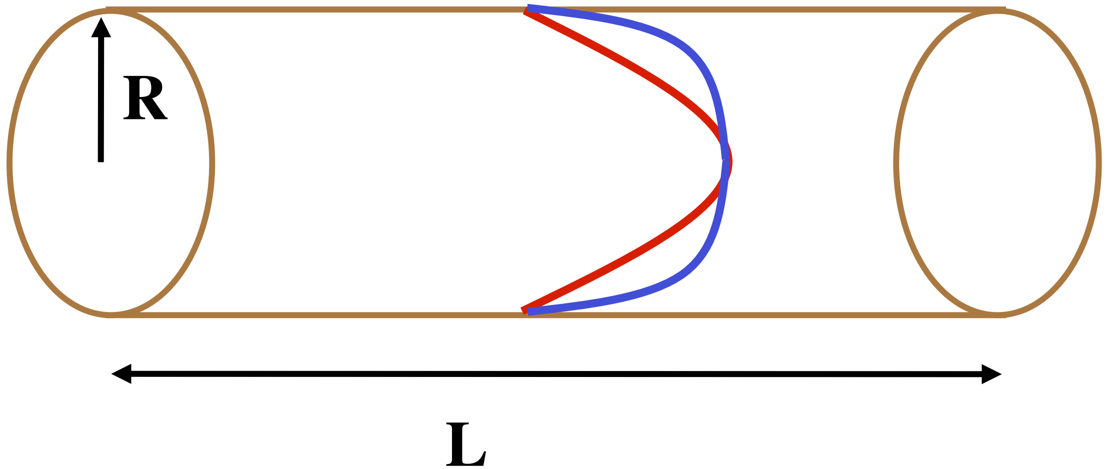




$$\tau = \mu \frac{du}{dr}$$

What does the velocity gradient look like as blood enters the constriction? Where is shear stress greatest?

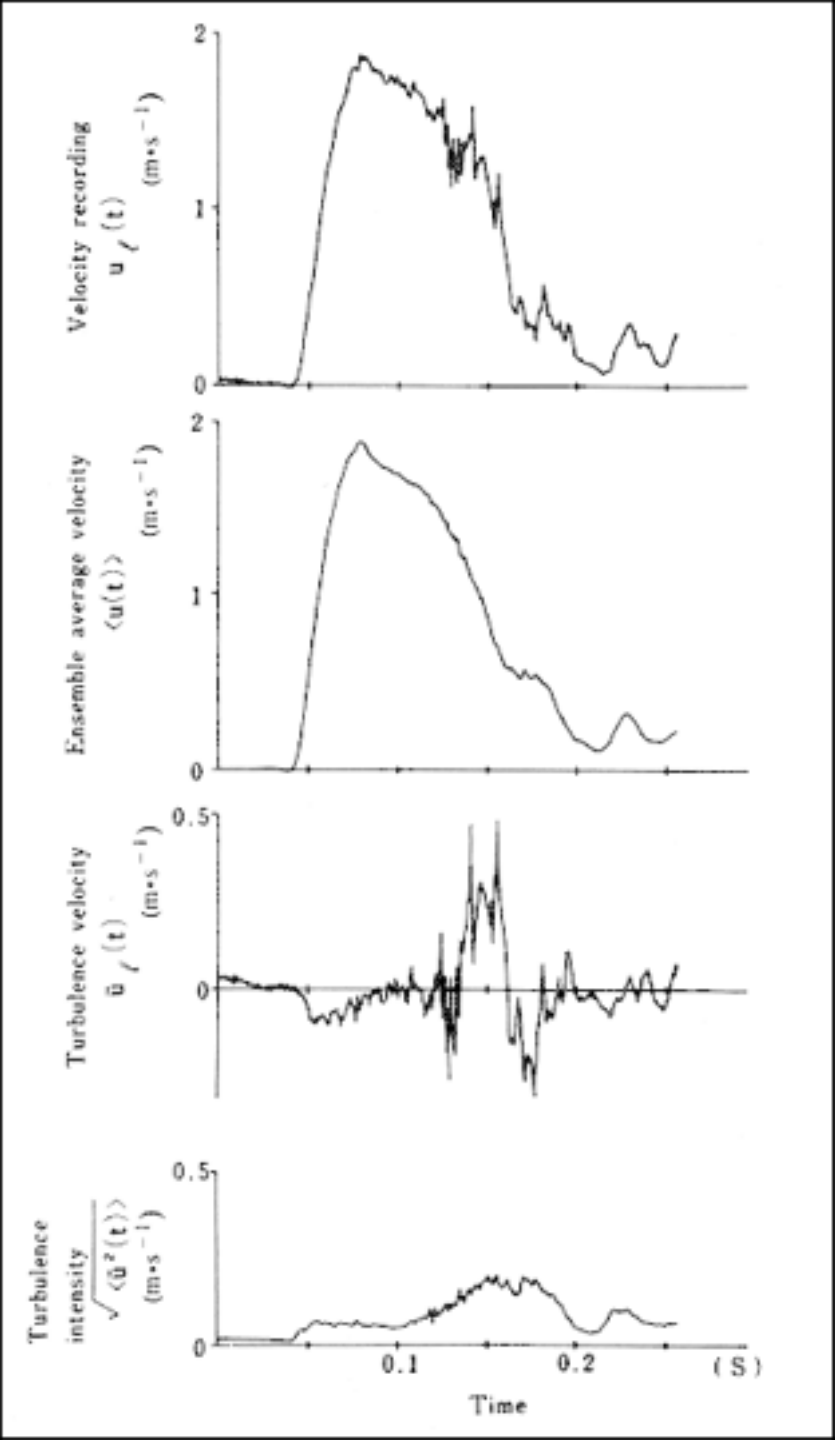
Factors:
tube shape, unsteadiness, secondary flows, turbulence and unique features of blood cells



$Q = \pi R^4 \Delta P / 8\mu L$ Hagen-Poiseuille law

Valid until the flow is turbulent! $Re = UD/\nu$ ($Re > \sim 2000$)

Vessel	(cm / sec.)	Reynolds Number
Aorta	48	3,400
Artery	45	500
Arteriole	5	0.7
Capillary	0.1	0.002



Turbulence and unsteadiness

Womersely Number

$$Wo = \frac{\omega R^2}{\nu} = \frac{2\pi f R^2 \rho}{\mu}$$

when large, acceleration effects are important

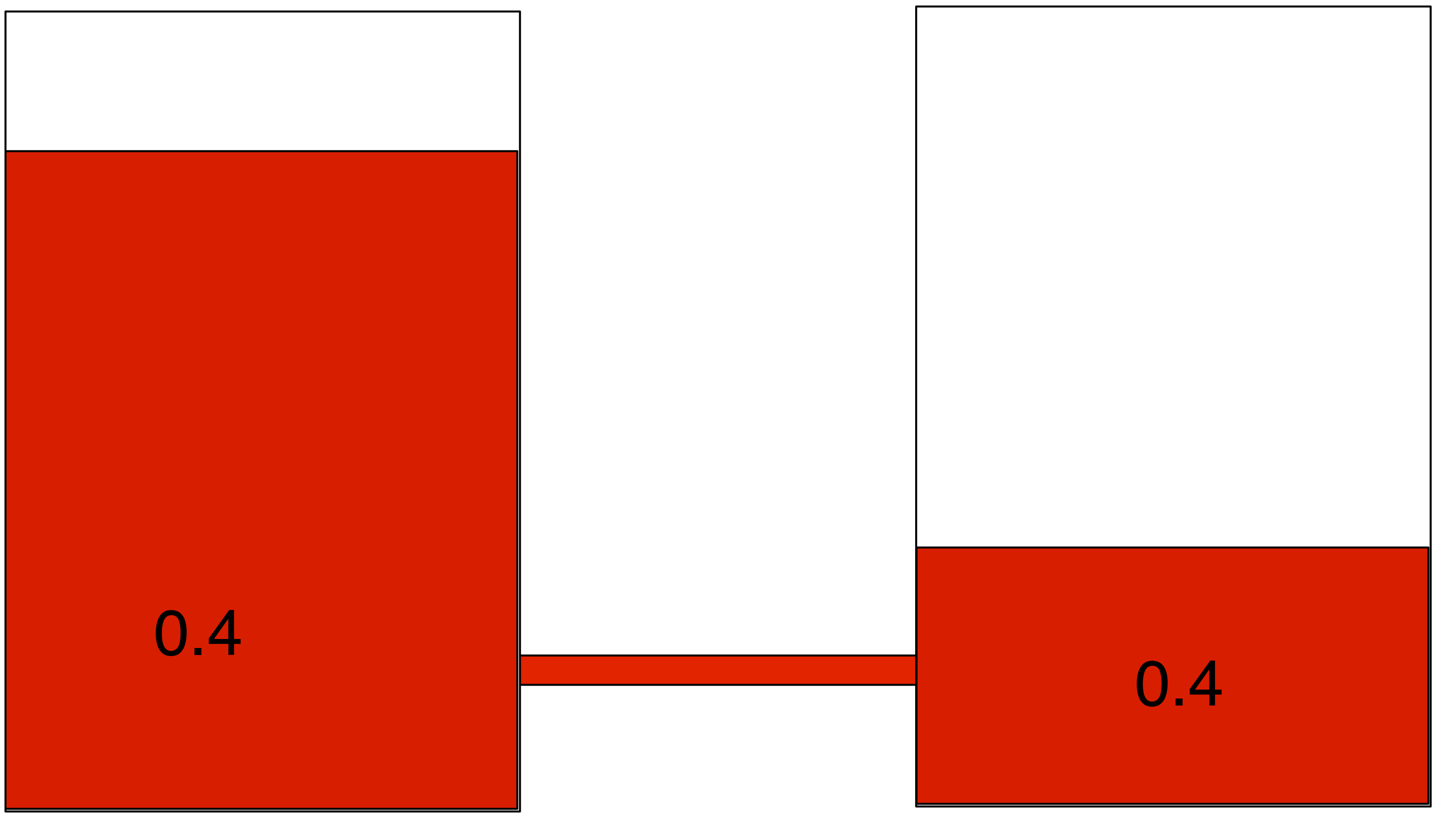
- Ascending Aorta -- 13.2
- Descending Aorta -- 11.5
- Abdominal Aorta -- 8
- Femoral Artery -- 3.5
- Carotid Artery -- 4.4
- Arterioles --.04
- Capillaries -- 0.005
- Venules -- 0.035
- Inferior Vena Cava -- 8.8
- Main Pulmonary Artery -- 15

Poiseuille flow in a small tube: s the hematocrit in the tube

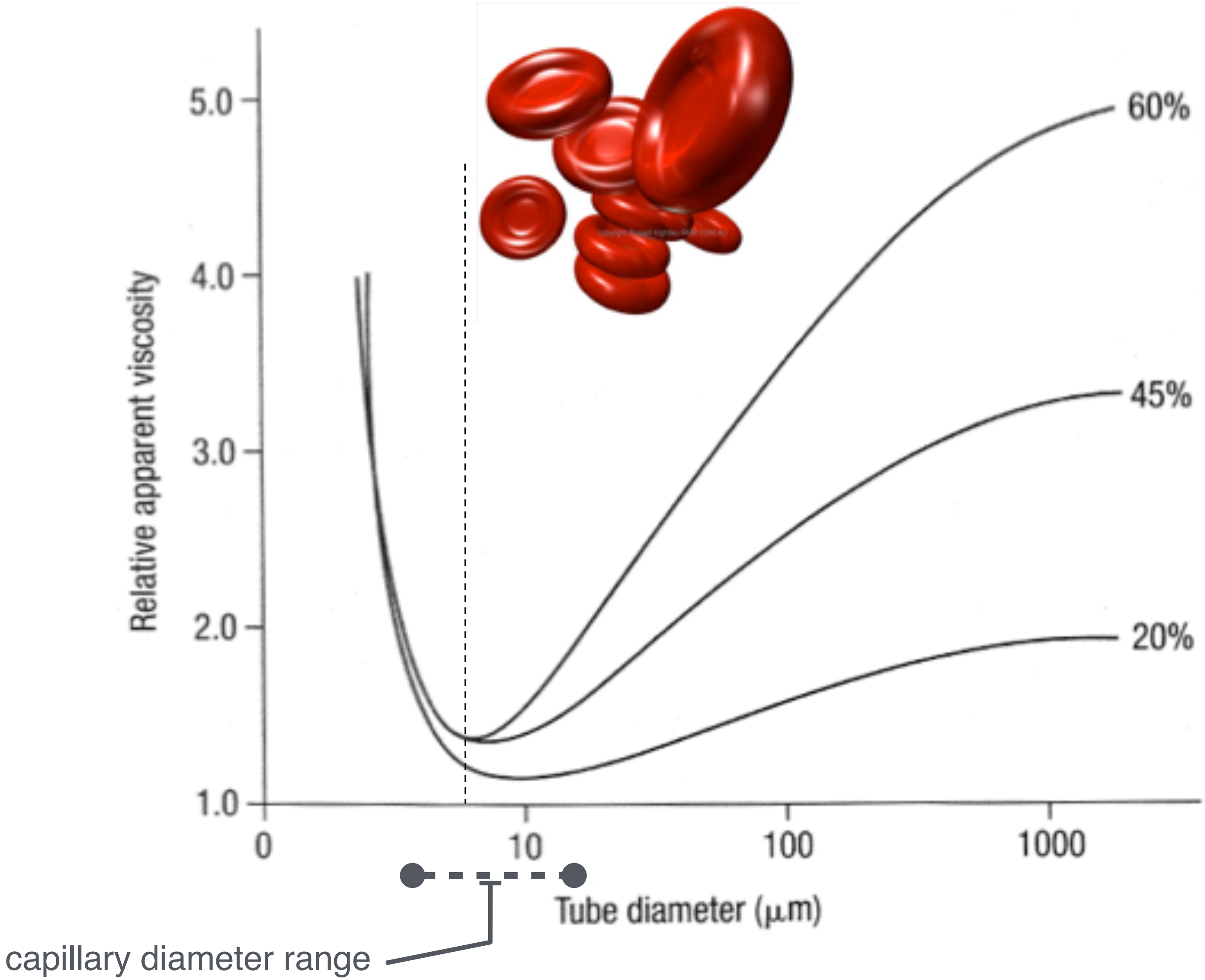
a. 0.4

b. less than 0.4

c. greater than 0.4   why?

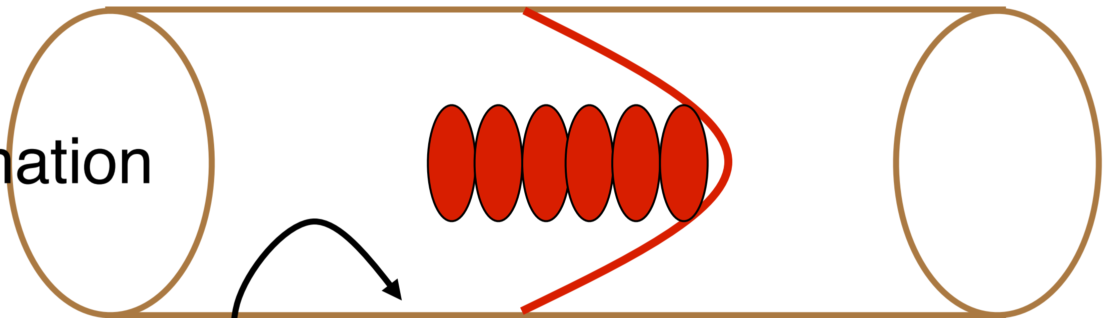


The Fahreaus-Lindqvist effect

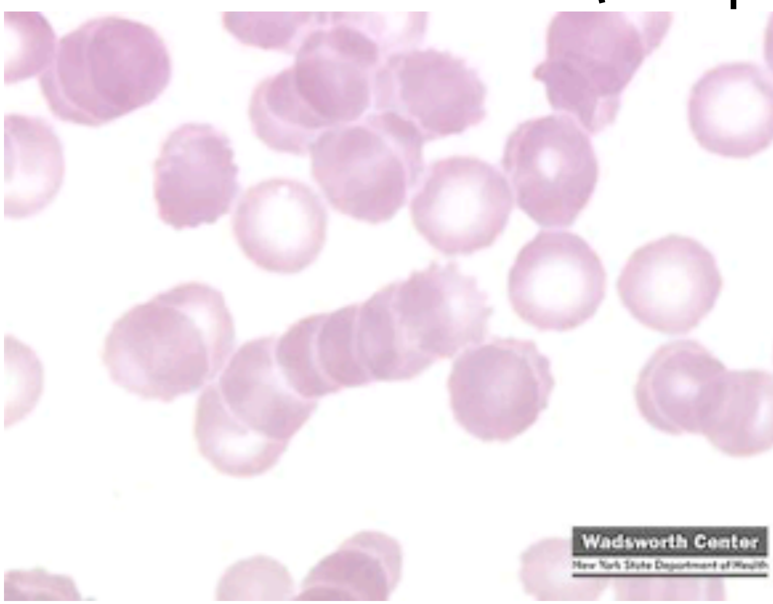


cell migration towards core
(Segré-Silberberg effect -- shear induced lift?)

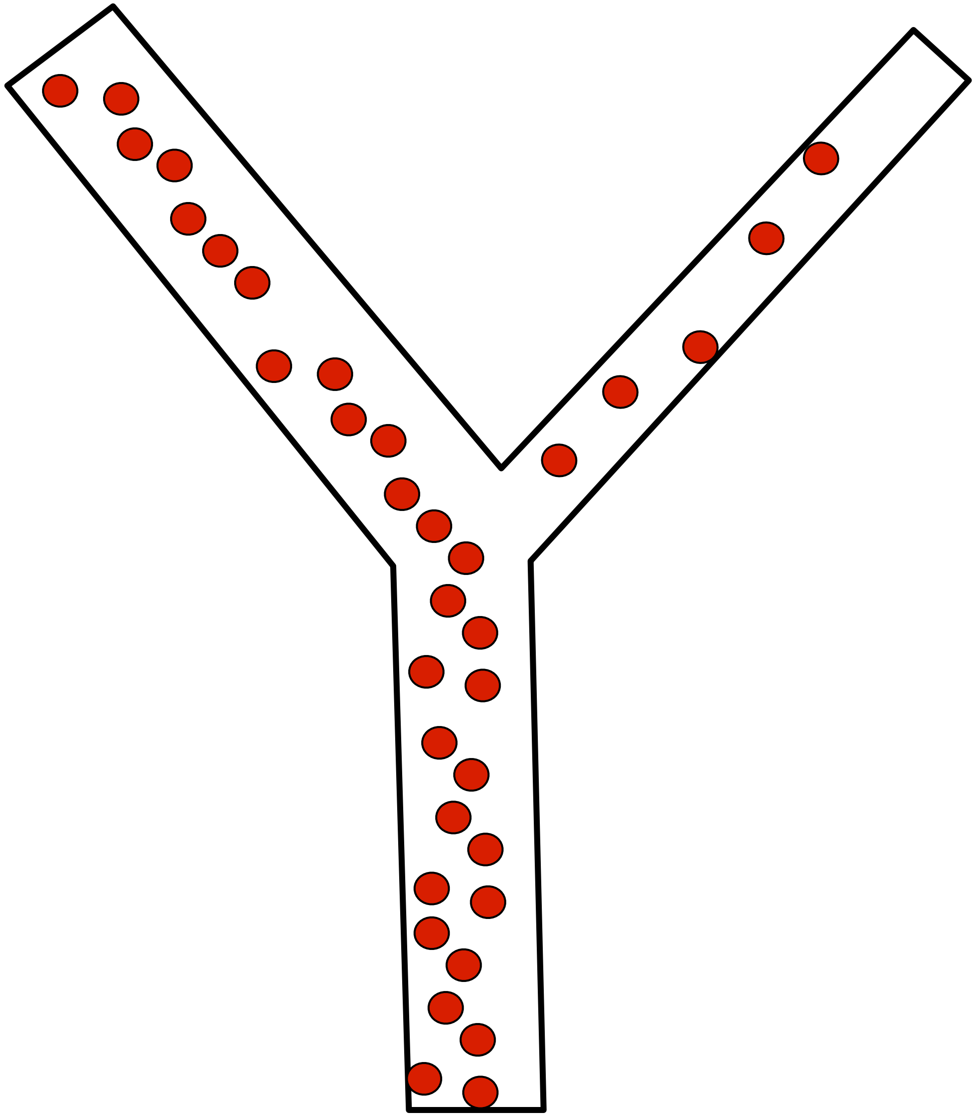
rouleaux formation



slower flow and fewer cells per volume of fluid



Entrance effects/plasma skimming



In this branched tube, is the hematocrit

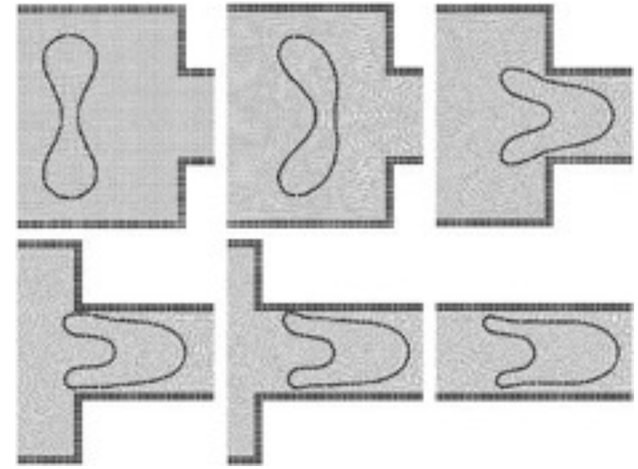
a. 0.4

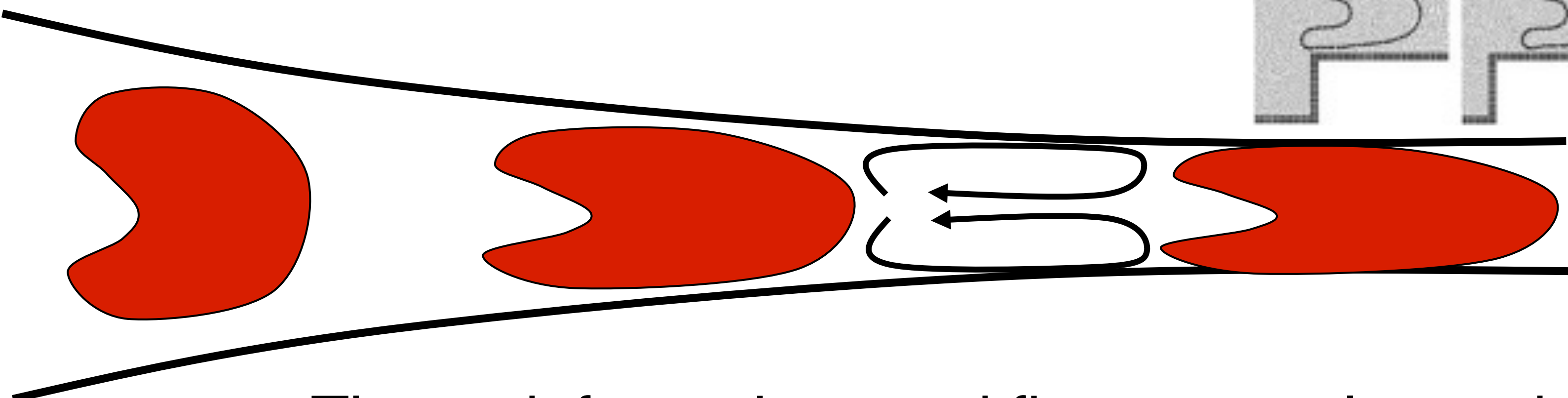
b. greater than 0.4

c. less than 0.4

“cell free” layer near perimeter leads to greater plasma/volume (fewer rbc/vol) in asymmetric branching vessels.

Cell deformations and secondary flows underlie significant increases in apparent viscosity



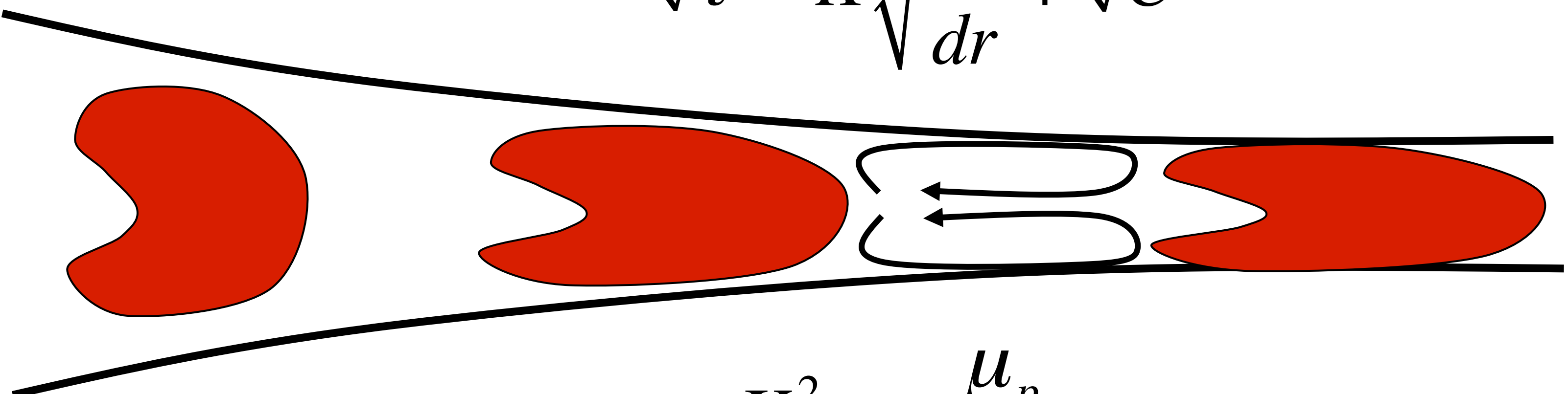


These deformations and flows may also underlie augmented flux of nutrients and gases!

Hosseini SM, Feng JJ. A particle-based model for the transport of erythrocytes in capillaries. Chemical Engineering Science 2009; 64:4488-97.

Blood viscosity varies with the shear stress

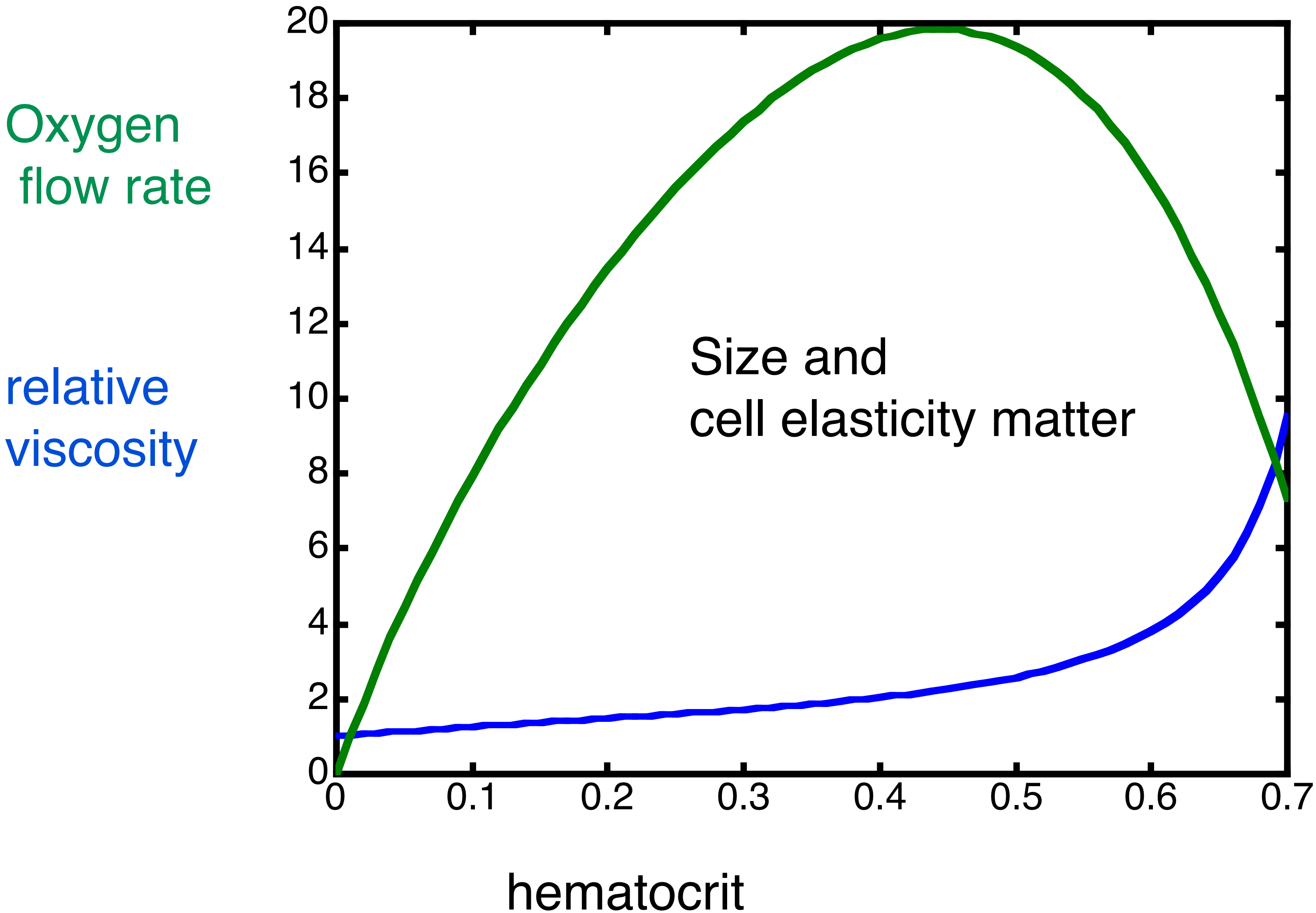
$$\sqrt{\tau} = K\sqrt{\frac{du}{dr}} + \sqrt{C}$$



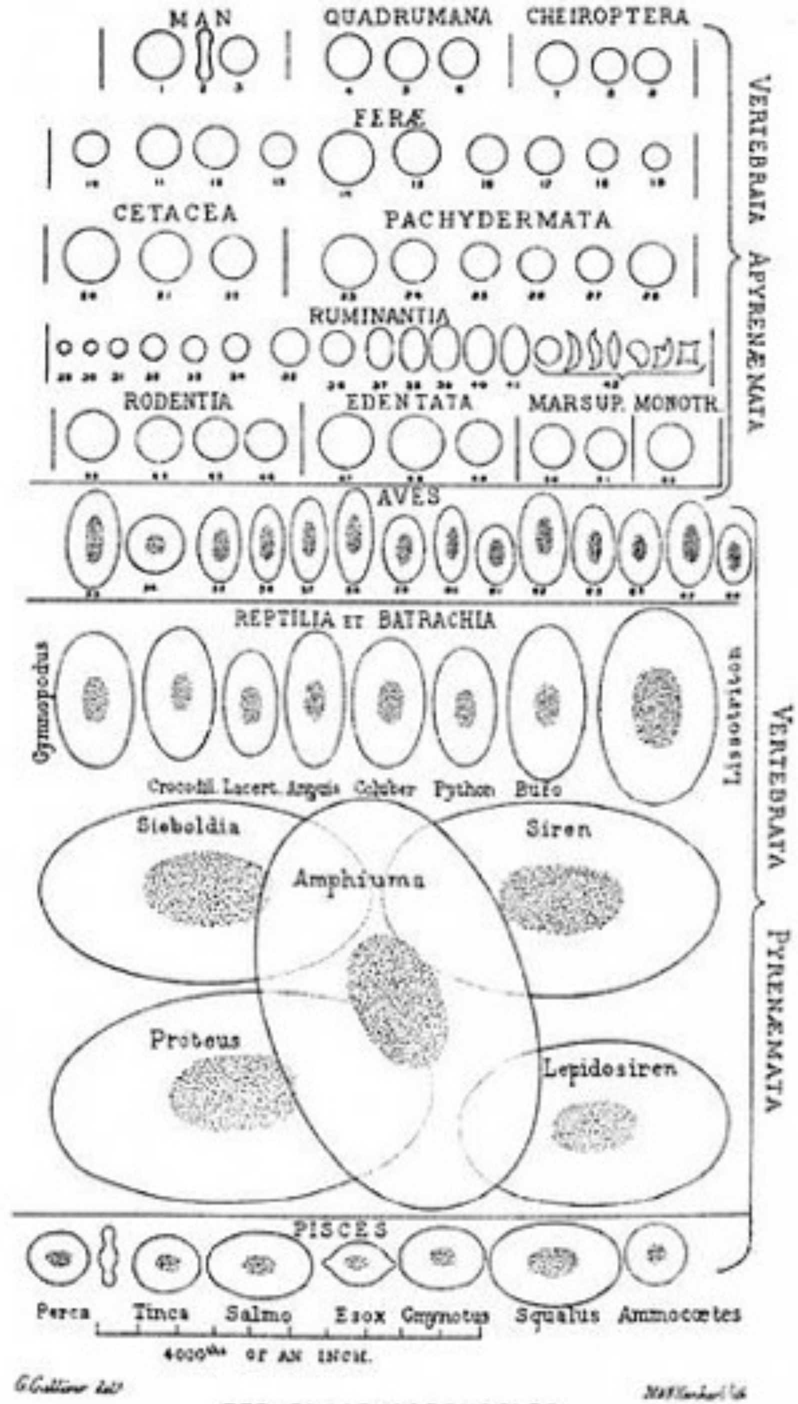
$$K^2 = \frac{\mu_p}{1 - \alpha\phi}$$

$$\alpha = 0.07\exp\{2.49\phi + 1107\exp(-1.65\phi)/T_K\}$$

Oxygen flow rate ~ $\mathbf{Q}\,\phi = \pi\,\phi\, r^4\, \Delta P/(8\,\mu L)$



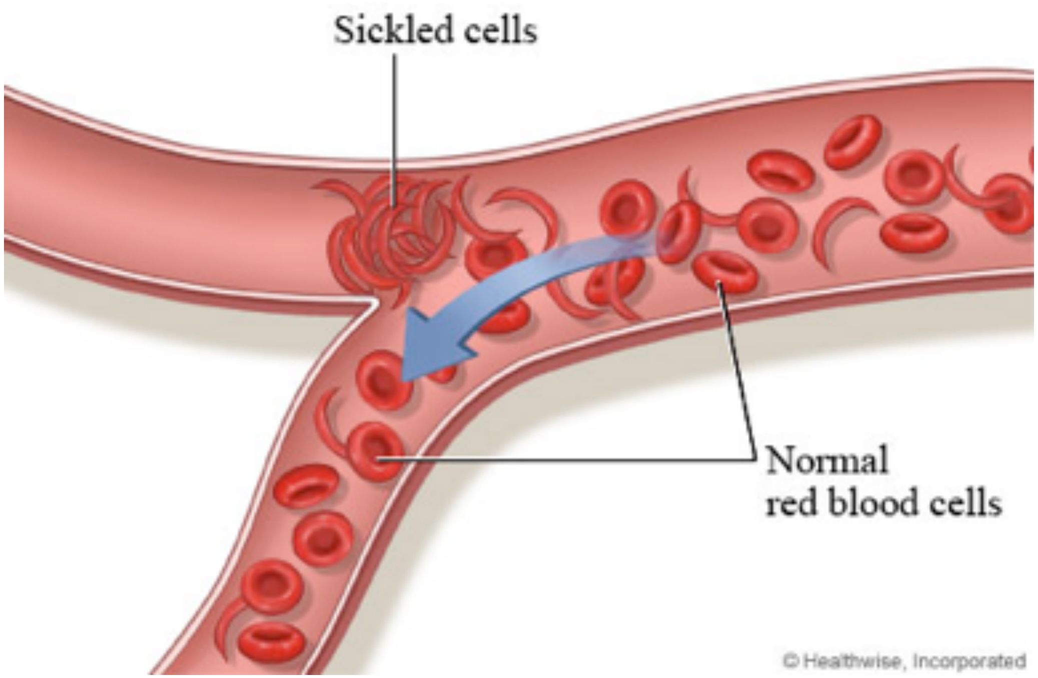
Gulliver, G. (1875). "On the size and shape of red corpuscles of the blood of vertebrates, with drawings of them to a uniform scale, and extended and revised tables of measurements". *Proceedings of the Zoological Society of London* **1875**: 474–495.



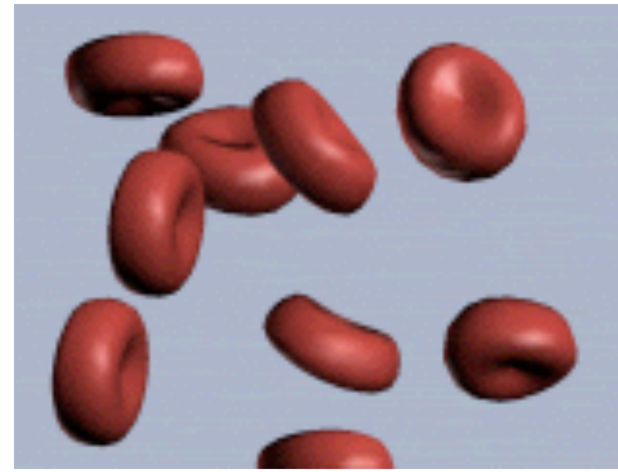
RED BLOOD-CORPUSCLES

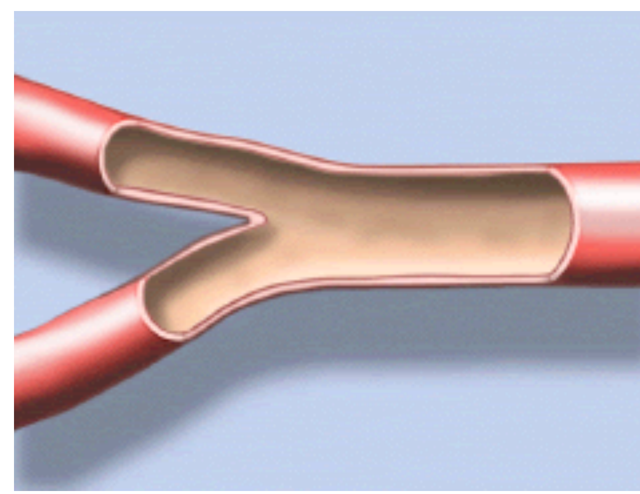
72,000 individuals

Point mutation in the hemoglobin β gene



Malaria modulates the modulus

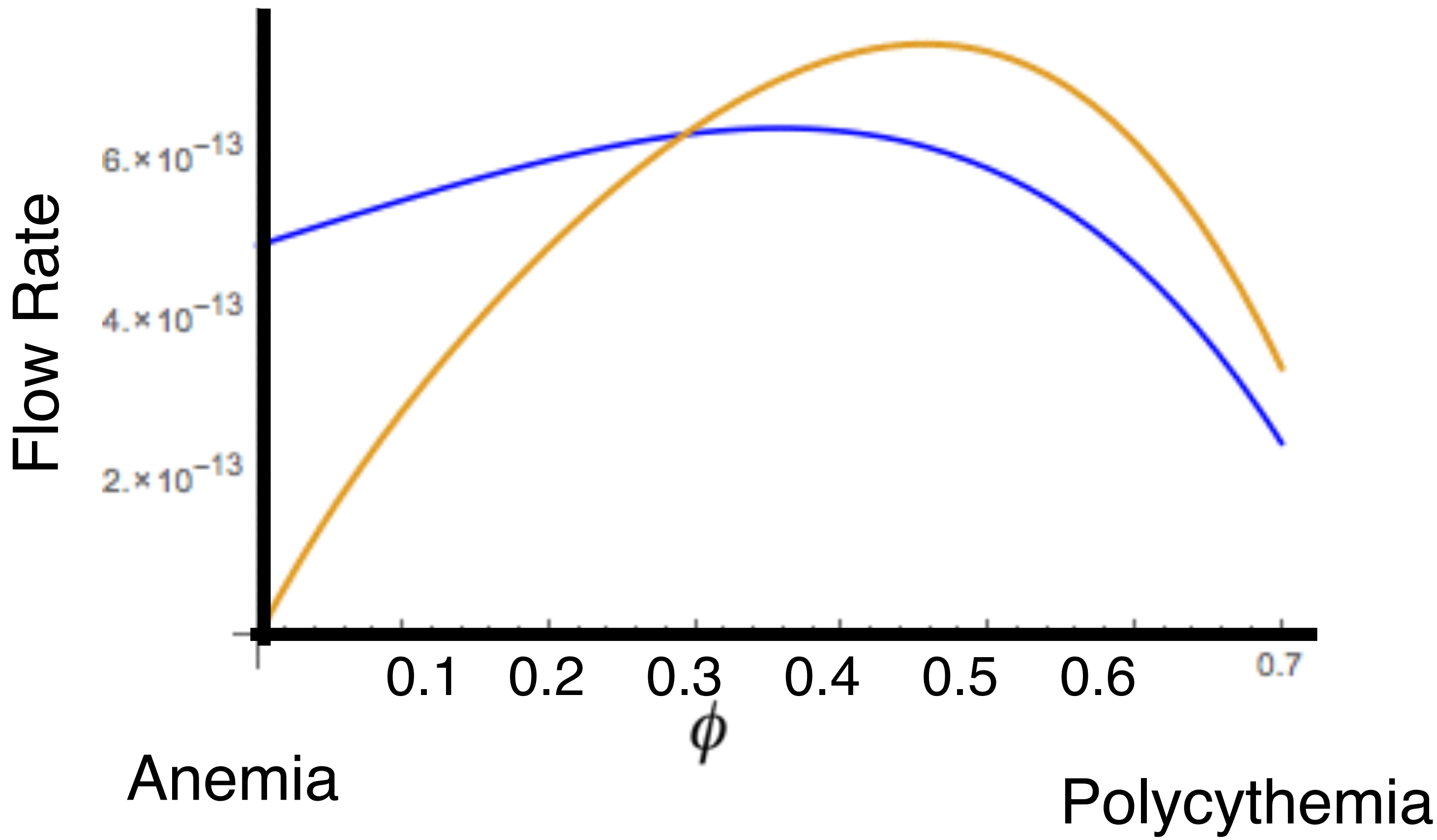




Anopheles

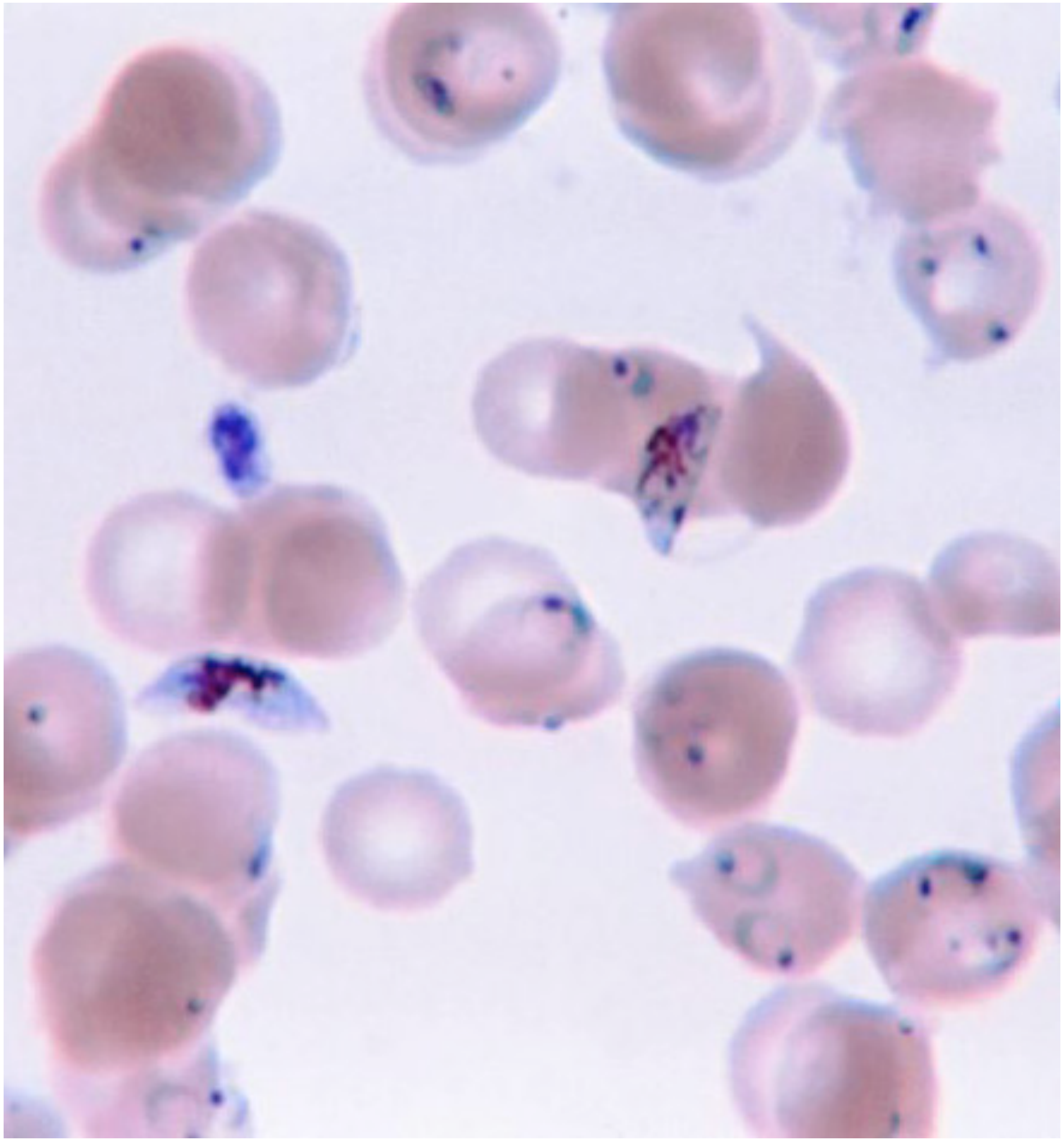

visualphotos.com

Blood viscosity depends non-linearly on red blood cell concentration (ϕ hematocrit) and Temperature

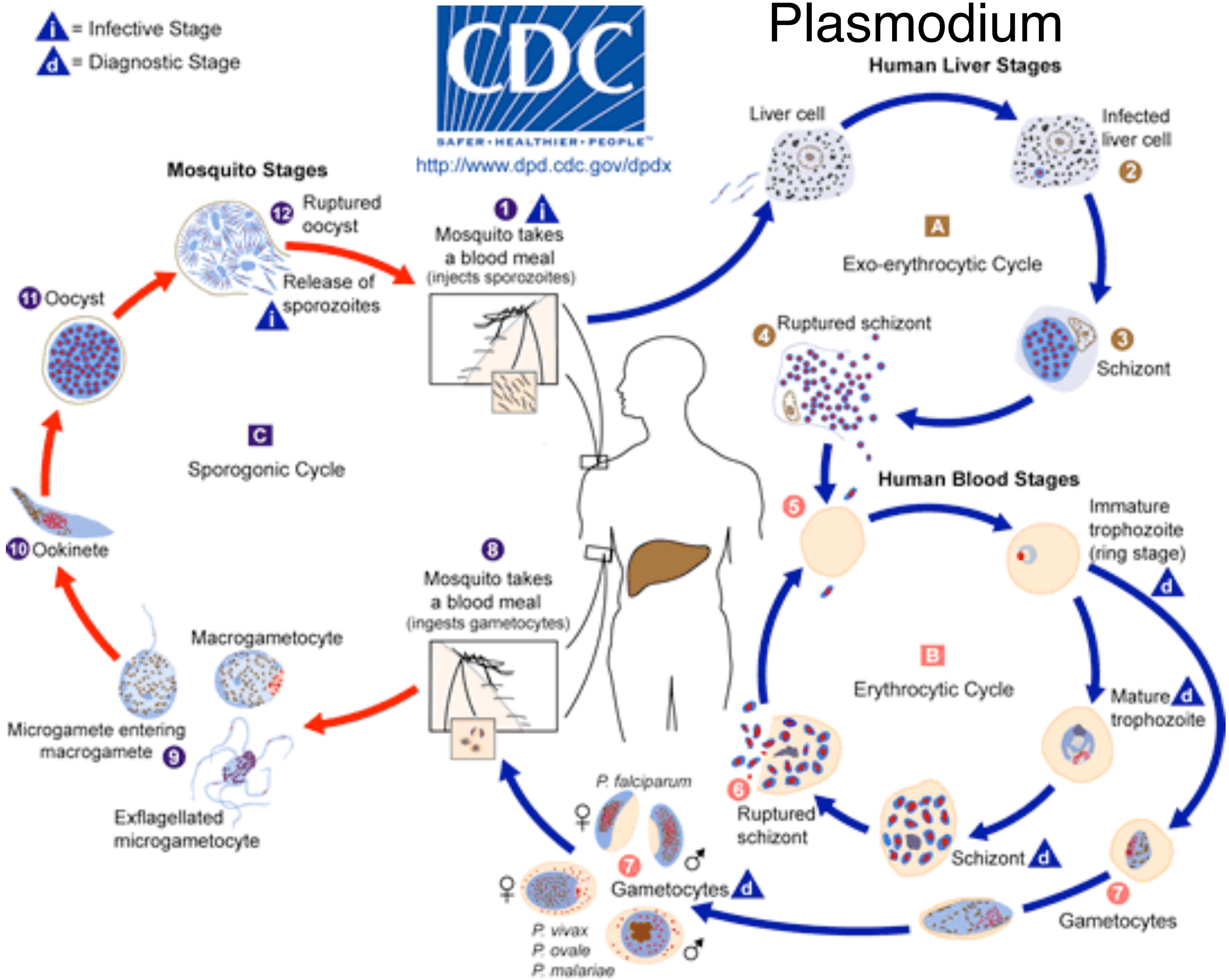


*Mathematica file is free to anyone who wants it (posted on the class website)

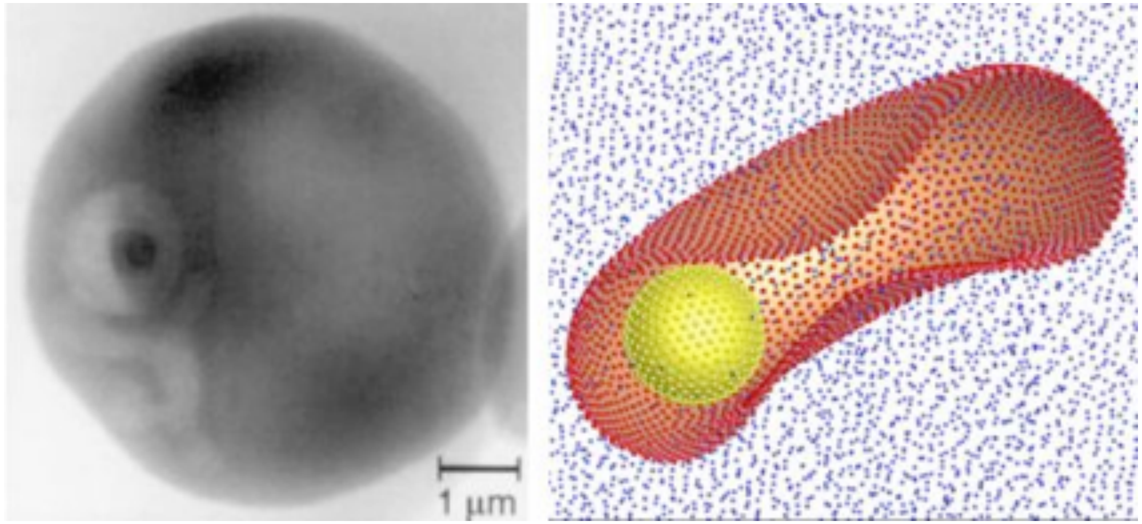
Gametocytes



Plasmodium

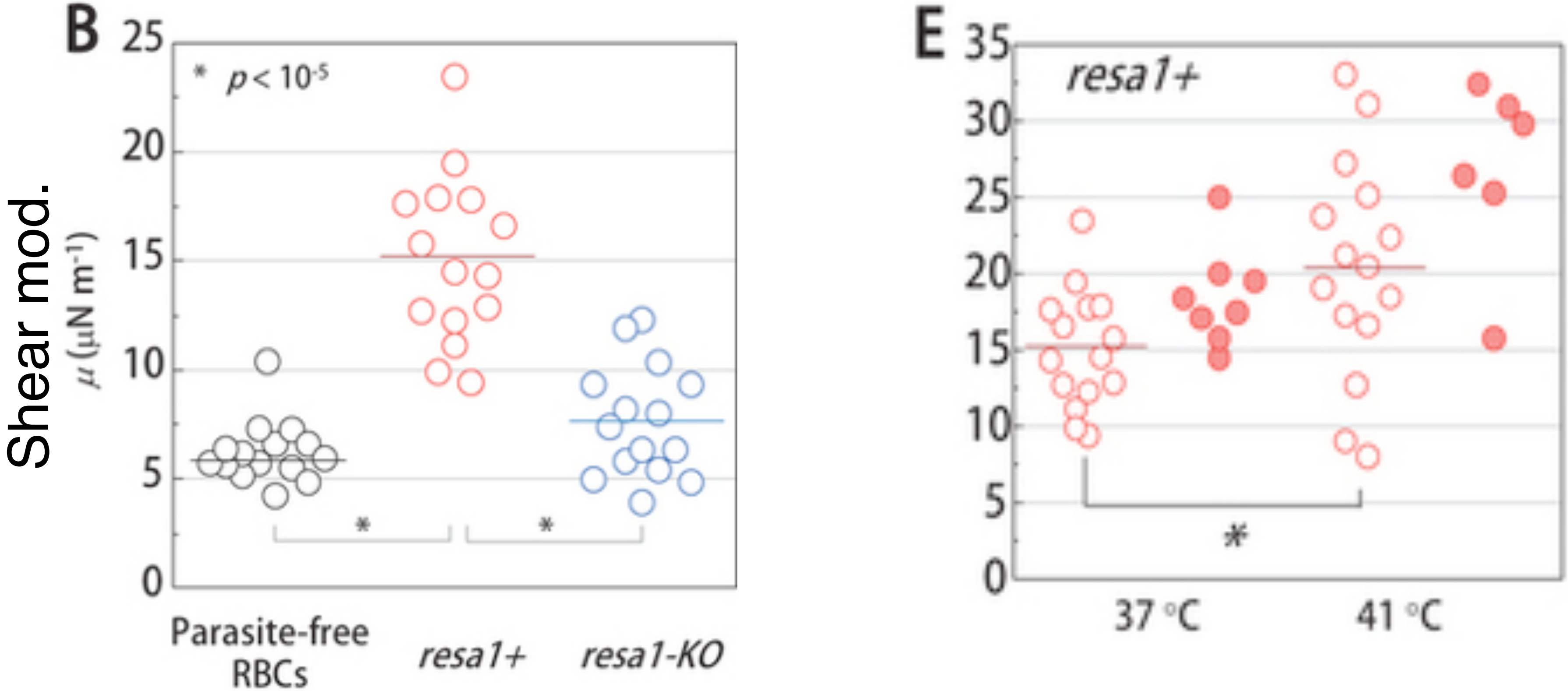


= Infective Stage
= Diagnostic Stage
http://www.dpd.cdc.gov/dpdx
Human Liver Stages
Liver cell
Infected liver cell
A
Exo-erythrocytic Cycle
Schizont
Ruptured schizont
Mosquito Stages
Ruptured oocyst
Release of sporozoites
Oocyst
Mosquito takes a blood meal (injects sporozoites)
C
Sporogonic Cycle
Ookinete
Human Blood Stages
Immature trophozoite (ring stage)
B
Erythrocytic Cycle
Mature trophozoite
Schizont
Ruptured schizont
Gametocytes
Mosquito takes a blood meal (ingests gametocytes)
Macrogametocyte
Microgamete entering macrogamete
Exflagellated microgametocyte
P. falciparum
Gametocytes
P. vivax
P. ovale
P. malariae



The pathogenicity of *Plasmodium falciparum* (*Pf*) malaria results from the stiffening of red blood cells (RBCs) and its ability to adhere to endothelial cells (cytoadherence). The dynamics of Pf-parasitized RBCs is studied by three-dimensional mesoscopic simulations of flow in cylindrical capillaries in order to predict the flow resistance enhancement at different parasitemia levels.

D. A. Fedosov, B. Caswell, S. Suresh, and G. E. Karniadakis, "Quantifying the biophysical characteristics of Plasmodium-falciparum-parasitized red blood cells in microcirculation", *Proceedings of the National Academy of Sciences USA*,, 2010.



Pf155/RESA protein influences the dynamic microcirculatory behavior of ring-stage *Plasmodium falciparum* infected red blood cells

Monica Diez-Silva, YongKeun Park, Sha Huang, Hansen Bow, Odile Mercereau-Puijalon, Guillaume Deplaine, Catherine Lavazec, Sylvie Perrot, Serge Bonnefoy, Michael S. Feld, Jongyoon Han, Ming Dao & Subra Suresh

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