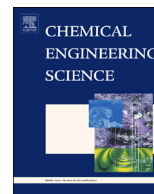




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# Unequal granular temperature model for motion of platelets to the wall and red blood cells to the center



Dimtri Gidaspow\*, Vishak Chandra

Department of Chemical and Biological Engineering, Illinois Institute of Technology, Chicago, IL 60616, USA

## HIGHLIGHTS

- A kinetic theory based analytical solution for platelet concentration was obtained.
- The model explains the motion of platelets and RBCs in the opposite direction.
- The results agree with observations.
- Shear enhanced diffusivities are related to computed granular temperatures.
- Diffusivities alone do not explain the observed migration.

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## ABSTRACT

The experimentally measured motion of platelets to the wall and red blood cells to the center is explained by unequal granular temperature kinetic theory. The migration of platelets to walls of the blood vessels in the presence of red blood cells is caused by the high granular pressure produced by the random oscillations of the red blood cells and the dissipation of platelet random energy at the walls. At the wall the shear has the highest value. This produces a high granular pressure and temperature which drive the red blood cells towards the center. An analytical solution for the platelet concentration was derived.

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## 1. Introduction

Blood is a multiphase mixture of plasma, platelets and 45% of red blood cells (Sherwood, 2004). There are 4–5 million red blood cells per micro-liter and only 0.15–0.4 million platelets per micro-liter. Hence modeling the motion of individual blood cells and platelets will produce experimentally observed diffusion coefficients, but will not explain the flow and migration in blood vessels. For example, recently Erickson (2010) used a Lattice Boltzmann-Immersed Boundary Approach to compute diffusion coefficients in a mixture of red blood cells (RBC) and platelets. However, Turitto (Goldsmith and Turitto, 1986; Turitto, 2014) believes that diffusion cannot explain the motion of red blood cells towards the center and the platelets towards the wall. In view of the large number of red blood cells and platelets, a continuum approach was used in the past to model blood flow. As already reviewed in Gidaspow and Huang (2009) paper, the early studies treated blood as a single phase fluid and used commercial

computational fluid dynamics (CFD) codes to compute blood flow. For example Gibson et al. (1993), studied the effect of vessel wall shear stress on the progression of atherosclerosis in human coronary arteries. Lyczkowski's group at the Argonne National Laboratory (Jung et al., 2006a; Jung et al., 2006b) was the first to model blood flow as a mixture of plasma and red blood cells. To obtain the motion of red blood cells and platelets, it is necessary to use the more advanced CFD codes using the unequal granular temperature kinetic theory recently reviewed in the book by Gidaspow and Jiradilok (2009). In China, Huilin's group (2003) developed such preliminary CFD codes based on the CFD code fully described in the Gidaspow and Jiradilok book (2009). At Morgantown, WV, Department of Energy Laboratory, Benyahia (2008) has extended the publicly available MFIX code (Syamlal, 1988) to unequal granular temperatures. He has reproduced the granular temperature and red blood cells concentration profiles in Gidaspow and Huang (2009) paper, but as yet was not able to obtain the profiles for the platelets computed in this manuscript.

In this study we extended the Poiseuille flow approximate model of Gidaspow and Huang (2009) to compute the concentration profiles of the red blood cells and the platelets. In agreement with the Aarts

\* Corresponding author. Tel.: +1 312 567 3045.

E-mail address: [gidaspow@iit.edu](mailto:gidaspow@iit.edu) (D. Gidaspow).

et al. (1988) experiment, we show that the platelets move towards the wall while the red blood cells move towards the center. For each phase, the driving force for migration is the granular pressure. For fully developed flow, the conventional boundary layer approximation shows that we can compute this driving force using the platelets and RBCs granular temperatures only through the use of the equations of state for the platelets and red blood cells.

## 2. Methods

The kinetic theory model of Gidaspow and Huang (2009) for fully developed flow is extended here to include platelets, whose concentration is lower than that of the red blood cells. Hence the oscillations of the particles in blood are caused by the red blood cells, whose viscosity is larger than that of the platelets. The dissipation parameter 'a' in the Gidaspow and Huang (2009) model is much larger for red blood cells than that of the more dilute platelets. This leads to the different shape of the granular temperatures for red blood cells than that for the platelets, as shown in Figure 8 in Gidaspow and Huang's paper (2009).

### 2.1. Unequal granular temperatures for RBC and platelets

The random kinetic energy balance for fully developed flow for red blood cells is as follows:

Production of oscillations due to shear + conduction

– dissipation due to inelastic collisions = 0

$$\mu_{RBC} \left( \frac{dv}{dr} \right)^2 + \frac{1}{r} \frac{d}{dr} \left( \kappa_{RBC} r \frac{d\theta}{dr} \right) - \frac{12(1-e^2)\theta_{RBC}^2 \epsilon_{RBC}^2 g_o \rho_{RBC}}{\sqrt{\pi} d_p} = 0 \quad (1)$$

where  $\kappa_{RBC} = \kappa'_{RBC} \theta^{1/2}$

$$\kappa'_{RBC} = \frac{150\sqrt{\pi} d_p \epsilon_{RBC} \rho_{RBC}}{384(1+e)g_o} \left[ 1 + \frac{6}{5}(1+e)g_o \epsilon_{RBC} \right]^2 + \frac{2\epsilon_{RBC}^2 \rho_{RBC} d_p (1+e)g_o}{\sqrt{\pi}} \quad (2)$$

For constant  $\kappa'_{RBC}$ , Eq. (1) takes the form

$$\frac{\mu_{RBC}}{\kappa_{RBC}} \left( \frac{dv}{dr} \right)^2 + \frac{1}{2\theta} \left( \frac{d\theta}{dr} \right)^2 + \frac{d^2\theta}{dr^2} - \frac{12(1-e^2)\theta_{RBC}^2 \epsilon_{RBC}^2 g_o \rho_{RBC}}{\sqrt{\pi} d_p \kappa'_{RBC}} = 0 \quad (3)$$

In dimensionless form the red blood cell granular temperature equation becomes as follows:

$$\frac{d^2\theta'_{RBC}}{dx^2} + \frac{1}{2\theta'_{RBC}} \left( \frac{d\theta'_{RBC}}{dx} \right)^2 + \frac{1}{r} \frac{d\theta_{RBC}}{dx} - a\theta'_{RBC} = -x^2 \quad (4)$$

where

$$a = \frac{12(1-e^2)\epsilon_{RBC}^2 g_o R^2 \rho_{RBC}}{\sqrt{\pi} d_p \kappa'_{RBC}}, \quad \theta'_{RBC} = \frac{\theta_{RBC}}{4(\mu_{RBC}/\kappa_{RBC})v_{RBC,max}^2}, \quad x = \frac{r}{R} \quad (5)$$

The parameter 'a' measures the dissipation due to inelastic collision with the restitution coefficient, e. It includes the effect of rotations of the red blood cells. The RBC pressure,  $P_{RBC}$  and the shear viscosity,  $\mu_{RBC}$  are expressed as a function of granular temperature based on the kinetic theory model (Gidaspow and Huilin, 1998; Jackson, 2000).

$$P_{RBC} = \epsilon_{RBC} \rho_{RBC} \theta_{RBC} + 2\rho_{RBC}(1+e)\epsilon_{RBC}^2 g_o \theta_{RBC} \quad (6)$$

$$\mu_{RBC} = (4/5)\epsilon_{RBC}^2 \rho_{RBC} d_p g_o (1+e)(\theta/\pi)^{1/2} + \frac{10\rho_{RBC} d_p \epsilon_{RBC} \sqrt{\pi} \theta}{96(1+e)g_o} \times [1 + (4/5)g_o \epsilon_{RBC}(1+e)]^2 \quad (7)$$

where

$$g_o = \left[ 1 - \left( \frac{\epsilon_{RBC}}{\epsilon_{RBC,max}} \right)^{1/3} \right]^{-1} \quad (8)$$

For platelets the dissipation parameter is almost zero compared with that of the red blood cells, since  $\epsilon_{platelets} \ll \epsilon_{RBC}$ .

Therefore its granular temperature equation is the same as that for the red blood cells, but with no inelastic dissipations as shown below:

$$\mu_{RBC} \left( \frac{dv}{dr} \right)^2 + \frac{1}{r} \frac{d}{dr} \left( r \kappa_{RBC} \frac{d\theta_{platelets}}{dr} \right) = 0 \quad (9)$$

Then in dimensionless form it becomes as follows:

$$\frac{d^2\theta'_{platelets}}{dx^2} + \left[ \frac{1}{2} \frac{1}{\theta'_{RBC}} \frac{d\theta'_{RBC}}{dx} + \frac{1}{x} \right] \frac{d\theta'_{platelets}}{dx} = -x^2 \quad (10)$$

### 2.2. Boundary conditions

In place of using the Johnson and Jackson (1987) boundary conditions used by Gidaspow and Huang (2009), there is more flexibility by prescribing wall shear. The shear wall balance can be obtained by equating the production of oscillations at the wall to the wall dissipation. This balance given by Gidaspow (1994) is as follows:

$$(1-e)\theta_{RBC} = \frac{1}{15} \left( \frac{dv}{dr} \right)^2 d_p^2 \quad (11)$$

It shows that we can prescribe the wall red blood cells granular temperature. Hence we give  $\theta'_{RBC}$  some values at  $r=1$ . For example, for  $\theta'_{RBC,wall}=1$ ,  $e=1.0-10^{-5}$ . The wall collisions are nearly elastic. For the platelets we use the Johnson–Jackson's boundary condition (Johnson and Jackson, 1987).

$$\theta'_{platelets} = -C \left. \frac{d\theta}{dr} \right|_{r=1} \quad (12)$$

where C is the dimensionless parameter.

### 2.3. Red blood cells and platelets concentration

The RBC and platelets concentrations can be obtained from their equations of state with the known inlet concentrations. For the RBC the equation of state is given by Eq. (6). For the more dilute platelets the second term involving the square of the platelets concentration is negligible. Hence the ideal particle equation of state like the ideal gas law for molecules is applicable, as shown below:

$$P_{platelets} = \epsilon_{platelets} e_{platelets} \theta_{platelets} \quad (13)$$

In terms of the number of particles per unit volume, n and the mass, m, this equation becomes

$$P_{platelets} = nm\theta_{platelets} \quad (14)$$

With the inlet platelet concentration,  $n_{inlet}$ :

$$P_{platelets} = n_{inlet} m \theta_{platelets} \quad (15)$$

Within the accuracy of the boundary layer approximation, there is no radial pressure distribution. Therefore Eqs. (13) and (14) give the simple equation:

$$\frac{n}{n_{inlet}} = \frac{\theta_{inlet}}{\theta_{platelets}} \quad (16)$$

For constant particle viscosity and conductivity:

$$\theta_{inlet} = \frac{1}{4} \frac{\mu_s v_{max}^2}{\kappa_s} \quad (17)$$

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