Self margination in sickle cell anemia blood flow

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The origin of sickle cell disease (SCD) lies in a recessive point mutation in the gene that encodes for the hemoglobin molecule of its carrier called HbS. Although HbS affinity for oxygen is not much different from its healthy homologe HbA, when it releases its oxygen, the HbS polymerizes into long rope like fibers that give to the cells it chacarteristic sickled shape. This shape was believed in the sixties to be responsible of the characteristic vaso-occlusion phenomena known for sickle cell disease since the more rigid cells will have difficulty to pass in the microcirculation like normal red blood cells do.

However, in the last three decades, SCD pathobiology has been proven to be more complex to explain general vaso-occlusion than the logical and recently, simulations are shown that the rigid, crescent-shaped red blood cells that are the hallmark of sickle cell disease don't cause the red cell blockages on their own [1].

Then, SCD is still a perplexing disease and almost no consequent cellular scale approaches of the study of capillary obstruction dynamics have been proposed in microflow, although the problem of obstruction is in essence a circulatory one.

Knowing that stiffer cells like white blood cells [2] or plaquelets migrate toward the vessel walls in blood flow through a process called margination, that depends mainly on local hematocrit, flow rate, red blood cell aggregation, deformability of different cell components [3], in this research we investigate experimentally the collective behavior of oxygenated arteriol-like sickle red blood cells and their margination process on flow through cylindrical channels with inner diameters comparable in size to a human arteriol. The cells are labelling accordingly to their density, that is associated to their rigidity and flash under different flow conditions on pressure and solutions, including solutions inducing and non-inducing red blood cell aggregation.

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