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Red blood cell aggregation – why blood is thicker than water

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Interactions between biological particles, including red blood cells (RBC), white blood cells, platelets and endothelial cells lining blood vessels, are governed by a complex interplay of various specific and non-specific forces. Numerous studies have detailed several "lock-key" mechanisms (e.g., antigen-antibody) leading to cell-cell attraction and adhesion, and specific molecules and binding sites have been identified. Conversely, the non-specific interactions of macromolecules with various cell types have received less attention. However, it has recently been shown that interactions between red blood cells are influenced by the presence of non-adsorbing, non-bridging macromolecules in the suspending phase. For these polymers, their near-surface concentration is less than in the bulk phase, and hence depletion of macromolecules near the RBC surface and the resulting attractive force appears to be the most likely mechanism for RBC aggregation. RBC are the most numerous cells in blood, have an important biological function (i.e., transport of oxygen), and enhanced attractive interaction between RBC increases low shear blood viscosity and adversely affects blood flow in small vessels. Depletion-generated attractive forces can also have a marked impact on the adhesion of red blood cells to endothelial cells, thereby presenting an alternative mechanism by which plasma proteins could regulate cell-cell interactions. Understanding the in vivo physiological implications of these findings should aid in developing new approaches and therapy for clinical conditions associated with enhanced cell-cell adhesion (e.g., myocardial infarction, nephrotic syndrome, sickle cell disease).

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