Water, Aquaporins and Red Blood Cells

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Throughout the history of medicine, water was always considered to be the `elixir of life`. Major aspects of the body´s water household have been studied extensively and were as we know today only partially understood. Some of the today´s concepts on the role of water are nevertheless widely accepted. However, when listening across the borders of medicine we are hearing ´breaking news` from physicists, chemists and biologist about a hidden pool (compartment) of water in the human body which we did not know about: Protein bound water. We began asking ourselves new questions, as for example what role does this `extra water` play in our body, what role does it have in protein and enzyme function and etc.?
Being invited as speaker and medical doctor to the `Fifth Annual Conference on the Physics, Chemistry & Biology of Water 2010` at Mount Snow, Vermont, one recalls right away a frequent disease prominent in the US: Sickle Cell Anemia, a red blood cell disease. Below a certain oxygen level slightly genetically disordered hemoglobin polymerizes in the cytosol. Suddenly the colloid osmotic imbalance with the blood plasma causes a rapid cell water outflow and a water-less (?) `sickle cell` is left. However, what happens to hemoglobin bound water in this process? In another disease, in sepsis, causing a high dead toll every year in all countries, the endotoxin Lipopolysaccaride is released. Why do RBCs swell upon this event and why does the total plasma water go down? If molecular bound water was released during these events as well then how and why? How about the ´liquidity` of water in RBCs at all? RBCs have the highest cytosolic protein concentration (Hb molecule per number of water molecules) as compared to all other cells of the body. Neutron scattering data suggest about 90% liquid water and 10% bound water being present in RBCs on the one hand. On the other hand when a hemoglobin ´solution` of powdered hemoglobin was ´dissolved in water` in vitro making a physiological Hb- concentration of 33g/dL water this solution is definitely highly viscous. Why are then RBCs so flexible in the microcirculation although `water bridges` forming between hemoglobin molecules should cause high cytosolic viscosity? What role do Aquaporins (`water channels`) play in all of that? There remain even more guesses. Quite unusual experiments revealed a structural temperature transition of hemoglobin occurring at body temperature. One surprising result was that this structural transition was observed with Hb of many other species and was strongly correlated to the body temperature of those species. Why is this? Setting on at the transition temperature RBCs release cytosolic water to the blood plasma upon cytosolic hemoglobin aggregation when temperature was raised. What physiological function does this effect have? Many answers have yet to be found.