Background: Red blood cell (RBC) aggregation has been a major scientific interest of Oguz Baskurt[1]. Unanswered questions for him were: Is RBC aggregation “good” or “bad” for tissue perfusion and should enhanced RBC aggregation in inflammation be corrected therapeutically?

Methods: We analyzed the impact of RBC aggregation on the perfusion of an artificial microvascular network (AMVN) at driving pressures ranging from 2.5 to 60 cmH2O. RBCs were suspended in either 46.5 g/L dextran 40 (non-aggregating) or 35 g/L dextran 70 (aggregating) with a hematocrit of 30%. Both dextran solutions had the same viscosity (2.1 ± 0.2 mPa·s).

Results: Aggregation was observed in 5 µm “capillaries” of AMVNs for RBCs suspended in dextran 70 at driving pressures up to 40 cmH2O. AMVN perfusion rates were similar for aggregating and non-aggregation RBC suspensions. The “capillary” hematocrit was higher for dextran 70 than dextran 40 suspensions (i.e., the Fahraeus effect was reduced by RBC aggregation).

Conclusions: RBC aggregation did not affect the rate of perfusion of an AMVN. A higher hematocrit in “capillaries” of the AMVN indicates a better oxygen transport capacity in the presence of RBC aggregation. This suggests a beneficial effect of enhanced RBC aggregation in disease, which should not be corrected therapeutically.