Role of calcium in the rheological abnormalities induced by repeated deoxygenation of sickle cells

University of Birmingham - The role of blood rheology in sickle cell disease

Description: -
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Frontiers

An outline consistent with current knowledge suggests that the swelling tendency resulting from the declining activity of the sodium pump is opposed by net KCl and fluid losses resulting from periodic Gardos channel activation during capillary passages, elicited by brief surges in cell calcium via stretch-activated PIEZO1channels. This narrative outlines a hypothesis for the evolution and mechanism of the extended longevity of human RBCs consistent with current knowledge.

Scientific & Academic Publishing: The article detailed information

The movement from curve A to C , illustrates that, the behavior of the cell represented by curve B is less abnormal than that represented by curve C. In general, the study of the viscoelastic property of sickle RBC is used to ascertain the relative contributions of RBC membrane and the internal HbS solution to viscous and elastic components of total deformability.

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Several genetic and cellular modulators of blood rheology in SCD are discussed, as well as unresolved questions and perspectives. When the 59Fe activity ratios between top and bottom cell-density layers were monitored for up to 200 days following tracer injections, the ratios were seen to decline steadily, at variable rates in different subjects, up to day 70. The long-term cumulative effects of these opposing transport-mediated processes are a progressive dissipation of the sodium and potassium gradients, and a balanced volume control with a marginal, not functionally compromising increase in cell density for most of the lifespan of the cells.

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Dense ISCs could be formed without added calcium, implying that entry of external calcium is not an essential requirement for cellular dehydration; ISCs formed without calcium tended to be less rigid ie, to have lower static and dynamic rigidities than those formed with calcium.

Rheologic Impairment of Sickle RBCs Induced by Repetitive Cycles of Deoxygenation

However, Ca 2+ extrusion and Gardos channel-mediated K + transport are hardly affected because of the huge native spare capacities of these transporters ; , ; , ;. Thus, cellular dehydration contributes to the observed changes of viscoelasticity, although an irreversible alteration of membrane structure also appears to be involved. After a brief description of basic hemorheology, the present review focuses on the role of the hemorheological abnormalities in the causation of several SCD complications, mainly in sickle cell anemia and hemoglobin Hb SC disease.

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