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Triggering of suicidal erythrocyte death by uremic toxin indoxyl sulfate

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Introduction: Anemia in end stage renal disease is attributed to impaired erythrocyte formation due to erythropoietin and iron deficiency. On the other hand, end stage renal disease enhances eryptosis, the suicidal erythrocyte death characterized by cell shrinkage and phosphatidyl serine-exposure at the erythrocyte surface. Eryptosis may be triggered by increase of cytosolic Ca^{2+}-activity ($[Ca^{2+}]_i$) and by ceramide, which sensitizes erythrocytes to ($[Ca^{2+}]_i$). Mechanisms triggering eryptosis in end stage renal disease remained enigmatic. The present study explored the effect of indoxyl sulfate, an uremic toxin accumulated in blood of patients with chronic kidney disease.

Methods: In this study, cell volume was estimated from forward scatter, phosphatidylserine-exposure from annexin V binding, ceramide abundance by specific antibodies, hemolysis from hemoglobin release, and $[Ca^{2+}]_i$ from Fluo3-fluorescence.

Results: Our results showed that a 48 hours exposure to indoxyl sulfate significantly increased $[Ca^{2+}]_i$ (≥300 μM), significantly decreased forward scatter (≥300 μM) and significantly increased annexin-V-binding (≥50 μM). Indoxyl sulfate (150 μM) induced annexin-V-binding was virtually abolished in the nominal absence of extracellular Ca^{2+}. Indoxyl sulfate (150 μM) further enhanced ceramide abundance.

Conclusion: Finally we concluded that Indoxyl sulfate stimulates suicidal erythrocyte death or eryptosis, an effect in large part due to stimulation of extracellular Ca^{2+} entry with subsequent stimulation of cell shrinkage and cell membrane scrambling.

Biography

Mohamed Siyab Eldin Elsadig Ahmed has completed his Doctorate from University of Tuebingen. He is the Head of the Department of Molecular Biology. He has published about 10 papers in reputed journals. He is also an Editorial and Advisory Board Member of *JBRC*.

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