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The uremic toxin acrolein promotes suicidal erythrocyte death

Mohamed Siyab Eldin Elsadig Ahmed
Central Veterinary Research Laboratory (CVRL), Sudan

Anemia is a major complication of end stage renal disease. It is mainly the result of impaired formation of erythrocytes due to lack of erythropoietin and iron deficiency. However, compelling evidence points to the contribution of accelerated erythrocyte death, which decreases the life span of circulating erythrocytes. Erythrocytes may enter suicidal death or eryptosis, which is characterized by cell shrinkage and by cell membrane scrambling with phosphatidylserine-exposure at the erythrocyte surface. Triggers of eryptosis include increase of cytosolic Ca^{2+}-activity ($[Ca^{2+}]i$). Erythrocytes could be sensitized to cytosolic Ca^{2+} by ceramide. In end stage renal disease, eryptosis may possibly be stimulated by uremic toxins. The present study explored, whether the uremic toxin acrolein could trigger eryptosis.

Cell volume was estimated from forward scatter, phosphatidylserine exposure from annexin-V-binding, hemolysis from hemoglobin release, $[Ca^{2+}]i$ from Fluo3-fluorescence, and ceramide from fluorescent antibodies.

In results a 48 h exposure to acrolein (30 – 50 µM) did not significantly modify $[Ca^{2+}]i$ but significantly decreased forward scatter and increased annexin-V-binding. Acrolein further triggered slight, but significant hemolysis and increased ceramide formation in erythrocytes. Acrolein (50 µM) induced annexin-V-binding was significantly blunted in the nominal absence of extracellular Ca^{2+}. Acrolein augmented the annexin-V-binding following treatment with Ca^{2+} ionophore ionomycin (1 µM).

Finally we concluded that acrolein stimulates suicidal erythrocyte death or eryptosis, an effect at least in part due to stimulation of ceramide formation with subsequent sensitisation of the erythrocytes to cytosolic Ca^{2+}.

Biography

Mohamed Siyab Eldin Elsadig Ahmed has completed his doctorate at the age of 39 years from University of Tuebingen. He is the head department of molecular biology. He has published about 10 papers in reputed journals. Ahmed is also an editorial and advisory board member of JBRC.

m_sea_74@daad-alumni.de

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