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Microbiology - Infectious diseases

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ACTIVITY OF HUMAN ERYTHROCYTE MEMBRANE-BOUND GLYCOHYDROLASES IS RELATED TO PLASMODIUM FALCIPARUM PRODUCTS

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Background: Plasmodium falciparum (Pf) malaria causes about 600,000 deaths each year. Pathogenesis of anemia is due to several factors including parasite induced hemolysis, diserythropoiesis and reduced deformability, accelerated senescence and removal of uninfected red blood cells (RBC). Modifications of the physicochemical properties of RBC plasma membranes and of their enzyme content can be ascribed to increased oxidative stress (OS) caused by parasite heme products such as Fe(III)-protoporphyrin IX (hematin) or hemozoin, present at high concentration in the plasma of malaria patients. Due to their role in signalling early membrane alterations in OS related pathologies, several plasma membrane glycohydrolases of human RBC have been proposed as new markers of cellular OS. However, an association between RBC glycohydrolases alteration and malaria infection has not been previously described.
The present work was aimed to investigate the ability of malaria parasite products to alter RBC membrane by affecting glycohydrolases activity.
Methods: RBC from human donors were incubated for 24 hours in the presence of supernatants, derived from Pf cultures, containing heme products released during parasite growth. RBC were then treated with different concentrations of hematin (20-10-5 μg/ml) or hemozoin (10-5-2.5 μg/ml) purified from Pf cultures. Hexosaminidase, β-D-Glucuronidase, α-D-Glucosidase and acidic Sialidase activities were evaluated by fluorimetric assays; membrane fluidity by fluorescence anisotropy method.
Results: A decrease in the enzymatic activity was observed after incubation of RBC in the presence of culture supernatants from different Pf strains. A significant dose dependent inhibition was observed after treatment with either hematin or hemozoin. At the highest dose, hematin induced a decrease of enzymatic activity between 65% and 55%. Likewise a decrease of RBC membrane fluidity was observed following treatment with hematin or hemozoin.
Conclusions: Our results are in agreement with accelerated RBC senescence observed in malaria infection and the parasite heme products seems to be the main contributors to these membranes damages. Glycohydrolases alterations could be indeed promising candidates as early markers of RBC damage in malaria infections.