Malarial Infection of Female BWF1 Lupus Mice Alters the Redox State in Kidney and Liver Tissues and Confers Protection against Lupus Nephritis.

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Abstract:

Systemic lupus erythematosus (SLE) is a prototypic autoimmune disease characterized by an imbalanced redox state and increased apoptosis. Tropical infections, particularly malaria, may confer protection against SLE. Oxidative stress is a hallmark of SLE. We have measured changes in the levels of nitric oxide (NO), hydrogen peroxide (H2O2), malondialdehyde (MDA), and reduced glutathione (GSH) in both kidney and liver tissues of female BWF1 lupus mice, an experimental model of SLE, after infection with either live or gamma-irradiated malaria. We observed a decrease in NO, H2O2, and MDA levels in kidney tissues after infection of lupus mice with live malaria. Similarly, the levels of NO and H2O2 were significantly decreased in the liver tissues of lupus mice after infection with live malaria. Conversely, GSH levels were obviously increased in both kidney and liver tissues after infection of lupus mice with either live or gamma-irradiated malaria. Liver and kidney functions were significantly altered after infection of lupus mice with live malaria. We further investigated the ultrastructural changes and detected the number of apoptotic cells in kidney and liver tissues in situ by electron microscopy and TUNEL assays. Our data reveal that infection of lupus mice with malaria confers protection against lupus nephritis.

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