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Effect of Melittin on the Human Erythrocyte and Rat Liver Mitochondrial Membranes

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Melittin as the major toxic component of the bee venom from Apis mellifera has previously been shown to be potent antibacterial agent with application in anticancer therapy. In the present study we analyze the action of melittin on biological systems using human erythrocytes and rat liver mitochondria as test systems. Alterations of lipid bilayer electrical resistance by melittin are probed by electrochemical impedance spectroscopy. We demonstrate that the melittin increases the electrophoretic mobility (EPM), zeta potential and surface electrical charge of erythrocyte membranes, suspended in phosphate buffered saline (PBS, pH 7.4). Melittin reduces the EPM of intact mitochondrial membrane in saline sorbitol suspending buffer (SSB, pH 7.2) and 2 ng/mL but causes an enhancement of negative electrokinetic potential at higher concentrations by 7 mV – 10 mV and net surface charge. The molecular mechanism of action of the cationic peptide on the electrostatics of the membrane is not completely established. Lipid peroxidation of melittin-treated erythrocytes and mitochondria show altered LP products compared to the untreated cell and sub-cellular structures.

Melittin influences transport pumps and increases the permeability of cell membranes to ions. Melittin alters the function of Band 3 of erythrocyte membranes which could accelerate the reaction with peptide. The apparent proton efflux assay is performed and the effectiveness of melittin is discussed. It is established that 0.2 μg/mL and 0.35 μg/mL accelerate the membrane transport of protons outflux the cells. There is a strong reduction of slope of membrane transport in the presence of 0.1, 0.3 and 0.4 μg/mL. We propose that the melittin disrupts the erythrocytes because of electrostatic interactions with membranes and changes in membrane transport via Band 3 alteration.

The results of the present study suggest that melittin treatment and the following enhancement of electrokinetic stability of mitochondrial membranes depends on the existence of high electrochemical membrane gradient ($\Delta\psi$) across the energized membrane. De-energized mitochondria with lack of electrochemical membrane potential and without an electrochemical proton difference across the membrane possess an increase in stability upon melittin action.

The significance of melittin interaction with biological membranes increases its electrokinetic stability and provides a potential explanation of inflammatory diseases and anti-oxidative stress.

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