

**MOLECULAR PATHOLOGICAL BIOLOGY OF MITOCHONDRIAL
CALCIUM UNIPORTER AND VOLTAGE-DEPENDENT ANION
CHANNELS IN CONTEXT ELECTRO-ION MEMBRANE DISTRESS
SYNDROME CHRONIC FATIGUE (CFS/ME)**

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Keywords: Molecular pathological biology; mitochondrial permeability pore (mPTP); mitochondrial calcium uniporter (MCU); voltage-dependent anion channels (VDAC) opening/closing functions; mitochondria; mitochondrial membrane potential ($\Delta\Psi_m$); Electro - Ion Membrane Distress Syndrome; membrane pathology; electrochemical membrane potential or proton potential ($\Delta\mu H^+$); membrane potential ($\Delta\Psi$).

Goal of the study: Consider the Molecular pathological biology of the mitochondrial permeability pore (mPTP), mitochondrial calcium uniporter (MCU), and voltage-dependent anion channels (VDAC) opening/closing functions in the context of Electro-Ion Membrane Distress Syndrome.

Introduction: VDACs (s - several channels, plural) are numerous β -barrel proteins in the outer mitochondrial membrane. VDACs maintain electrical and biochemical cellular homeostasis and act as the primary channels for the passage of metabolites such as ATP/ADP, Ca^{2+} ions, and others, as well as signaling molecules, between the mitochondria and the cytosol, making them particularly important in energy production (energy generators) and apoptosis. With optimal support lysosomal mitochondrial clearance of autophagy, mitophagy (selective elimination of mitochondria).

VDACs, these are membrane proteins embedded in the membrane that open/close in response to changes in the electrical charge on the cell membrane. Converting electrochemical membrane potential ($\Delta\mu H^+$) and mitochondrial membrane potential ($\Delta\Psi_m$). The electrochemical potential of a membrane (or proton potential, $\Delta\mu H^+$) is the energy stored in a biological membrane and is composed of the difference in proton concentrations (chemical gradient, ΔpH) and the difference in electrical charges (membrane potential, ($\Delta\Psi$). It serves as the main source of energy for ATP synthesis and the transport of substances. It serves as the main source of energy for ATP synthesis and the transport of substances. Also, acting as "electrical switches" in the body, generating and transmitting nerve impulses. Thus, serving as the basis for generating the action potential, "electrical switches" for the electrical impulse, allowing neurons, muscles and the heart to exchange information and contract.

In this context, a relationship with VDACs in particular of mitochondrial calcium uniporter and voltage-dependent anion channels and the Electro-Ion Membrane Distress Syndrome (syndrome Maria & Irina Vasilieva), chronic fatigue syndrome/myalgic encephalomyelitis (CFS/ME) is demonstrated.

The Electro-Ion Membrane Distress Syndrome, EIMDs (syndrome Maria & Irina Vasilieva) described in 2019 opens up the possibilities of revealing membrane pathology molecular pathological biology. Primary/secondary morphofunctional damage to pores/channels of biomembranes provokes broken to ion-electrogenation, transduction and transmission of the membrane electric potential (MEP). The biomagnetic field and quantum energy /quantum electromagnetic radiation are also destabilized, as a connecting signal of transmission and amplification pathochemical reactions of a pathophysiological cell. We consider this syndrome as electro-ion membrane distress syndrome, whose disorder generates with chronic fatigue syndrome/myalgic encephalomyelitis (CFS/ME). We also called this syndrome Maria & Irina Vasilieva, as a switch that destabilizes the homeostasis of intracellular and extracellular media due to membrane distress. Immuno Compromise(IC) CHAOS dissonance creates a disorder of electro-storm/electroparalysis of MEP due to cytokines

whose disorder install EIMDs and CFS/ME, with diagnostic marker is less deformed red blood cells with a compacted “hard” cell membrane, by determining the permeability of the erythrocyte membrane and the sorption capacity of red blood cells. Standard therapy in surviving critical patients with IC CHAOS dissonance did not provide a stable decrease in CFS/ME, the effect of which was observed after MOST-ELSO, and due to cryo-bio-xeno myelo-timo-spleeno perfusion. This success, of cryotherapy expressed ↓toxic oxygen and nitrogen and heavy water effect of compression and reduction of “synairesis” of proteins, separation of liquid from the gel caused by a reduction in protein due to the release of water from the membrane and the release from of cell membranes and cells deuterium D/²H, “heavy water” which inhibits some cleavage reactions [1-10].

Material and methods: There were analyzed articles from Google Scholar, Scite -AI, PubMed database from the newest resources mentioned such words as: Molecular pathological biology; mitochondrial permeability pore (mPTP); mitochondrial calcium uniporter; voltage-dependent anion channels opening/closing functions; mitochondria; mitochondrial membrane potential ($\Delta\Psi_m$); Electro - Ion Membrane Distress Syndrome; membrane pathology; electrochemical membrane potential or proton potential ($\Delta\mu H^+$); membrane potential ($\Delta\Psi$).

Results: The transition from physiological apoptosis to pathological molecular biology of cell necrosis is caused by disruption of lysosomal mitochondrial clearance of autophagy, mitophagy, and the occurrence of abnormal/extreme myelopoiesis.

VDACs represent a consequence of multiple destructive factors of molecular biological pathology associated with impaired mitochondrial Ca²⁺ homeostasis. Ca²⁺ levels and damage the open-close function of the mPTP, the MCU, and VDAC. MCU is an inner mitochondrial membrane protein that acts as the primary pore-forming channel for calcium ions (Ca²⁺) to enter the mitochondrial matrix. By regulating mitochondrial Ca²⁺ levels, it plays a critical role in cellular metabolism, energy (ATP) production, and apoptosis. The mPTP is a large, non-selective protein channel in the inner mitochondrial membrane. Normally closed, it opens under stress (like calcium overload or oxidative stress) to cause membrane depolarization, mitochondrial swelling, and cell death via apoptosis or necrosis. mPTP and MCU are two different protein structures located in the inner membrane of mitochondria, which control calcium homeostasis and cell death. Immunocompromised dissonance (IC) CHAOS-[C]ardiovascular Compromise: shock; [H]omeostasis; [A]poptosis; [O]rgan dysfunction; [S]uppression of the immune system ICCHAOS provoking Endothelial Disorders and Endotheliosis as predictors of Acute Vascular Distress Syndrome and the development of Multisystem Inflammatory Syndrome confirmed and microcirculatory-mitochondrial distress syndromes. Where the increased rigidity of cell membranes plays a special role during less deformed red blood cells (erythrocyte) maintained and Abnormal/extreme myelopoiesis, developing acute/chronic systemic cellular hypoxia. Is devoid of flow deformation with a violation of the sigma effect, the Phareus-Lindquist phenomenon, which provides a high degree of erythrocyte deformation in a flow the destabilization of which gives rise to tissue ischemia, Electro-Ion Membrane Distress Syndrome (syndrome Maria & Irina Vasilieva), and microcirculatory-mitochondrial distress syndromes [11-64].

Conclusion: We demonstrated the molecular pathological biology of the membrane mitochondrial calcium uniporter and voltage-dependent anion channels, directly

linking them to the electroion membrane stress syndrome (Maria and Irina Vasilieva syndrome). Dysfunctions of the opening/closing functions of the mPTP, MCU, and VDAC.

This disorder, at the level of the molecular pathological biology of the mitochondrial calcium uniporter and voltage-dependent anion channels, is considered a serious disturbance of electrochemical membrane potential or proton potential ($\Delta\mu\text{H}^+$); membrane potential ($\Delta\Psi$); and mitochondrial membrane potential ($\Delta\Psi\text{m}$), and therefore biomagnetic field and quantum energy /quantum electromagnetic radiation leading to disruption of electrochemical cellular homeostasis. As a result, disorders and connecting signal of transmission and amplification pathochemical reactions of a pathophysiological cel. Energy deficiencies contributing to the development of chronic fatigue syndrome (CFS/ME).

Scientific data obtained through the development of pathological molecular biology, a medical-biological pathological discipline that needs to be introduced into medical universities

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