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# Intralipid blocks the entry of the SARS-Cov2/COVID/19 virus into cells by maintaining receptor a leucine-rich repeat containing 15 (LRRC15), an angiotensin-converting enzyme 2 receptor competitor.

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## Abstract

The effectiveness of Intralipid against SARS-Cov2/COVID/19 with Multiple Organ Dysfunction Syndrome (MODS) prevention or regression is described in the original scientific paper [1]. Intralipid at Oxidative and Nitro-Galogenic stress in patients with SARS-Cov2 / COVID /19, favors the predominance of the membrane-cytoprotective action of Reactive Oxygen Species (ROS) / Reactive Nitrogen Species (ROS/RNS) over the membrane-to-destructive action, restoring the balance between [ROS /AS] / [RNS / ANOS]. Membrane-cytoprotective mechanism Intralipid is due to a decrease in ROS and RNS and an increase in the activity of the Antioxidant System (AS) and Anti Nitro Oxidant System (ANOS), stopping lipid peroxidation (LPO), reducing Electro-Ion Membrane Distress Syndrome (Maria&Irina Vasilieva syndrome) [2], accelerates the regeneration of endothelial and epithelial cells of the alveolar acinus, restoring gas-respiratory metabolism and the predominance of physiological cell apoptosis over necrosis. Intralipid at SARS-Cov 2 / COVID / 19 opposes Microcirculatory Mitochondrial Distress syndrome (MMDS) by Microcirculatory - Mitochondrial Recruitment; as a result of which pCO2 (AVgap) <6 mm Hg, since LPO decreases and at the level of mitochondrial membranes, improving the function of Mitochondrial permeability transition pore-dependent Ca uniporter, mPT pore, support energy metabolism, eliminating energy deficits, restoring Extreme / Abnormal myelopoiesis and impaired autophagy (mitophagy). Thus, Intralipid has been shown in the strategy of targeted treatment of LPO in Oxidative and Nitro-Galogenic stress in patients with SARS-Cov2 /COVID / 19.

Given the successful treatment of SARS-Cov2/COVID/19 patients [3] and the need for in-depth studies [4], further scientific research on using Intralipid in SARS-Cov2/COVID/19 patients made it possible to notice the absence or development minor fibrotic pulmonary changes. The use of Intralipid in the early stages also made it possible to describe the antiviral effect of anti-SARS-Cov2/COVID/19. The content of egg yolk phospholipids rich in leucine in Intralipid predetermined attention to Leucine Rich Repeat Containing 15 (LRRC15) [5]. On the other hand,

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Australian scientists [6] described LC15 as a fibroblast-expressed SARS-CoV-2 spike receptor that controls antiviral and antifibrotic transcriptional programs. LRRC15 is a receptor competitor of Angiotensin-converting enzyme 2 (ACE2) to the spike protein**.** Thus, LRRC15 immobilizes SARS-Cov2/COVID/19 on its spike protein receptors, competing with ACE2, thereby stopping SARS-Cov2/COVID/19 from entering intracellularly. Reduction of pulmonary atelectasis in SARS-Cov2/COVID/19 and volutrauma and barotrauma in SARS-Cov2/COVID/19 in mechanically ventilated patients who were infused intravenously with Intralipid, proves prevention of collapse of the lung alveoli due to the synthesis and presence of surfactant.

Thus, pharmacodynamically demonstrated not only anti-oxidative and anti-Nitro-Galogenic stress in patients with SARS-Cov2 / COVID /19 but also the antiviral effect of intralipid against SARS-Cov2/COVID/19, which prevents its penetration into cells, maintenance of LRRC15 receptors. Stopping the development of MODS caused by cytokine storm, immune dissonance CHAOS, Systemic Inflammatory Response Syndrome (SIRS) or immune paralysis, Compensatory Anti-inflammatory Response Syndrome (CARS), and Persistent Inflammation, immunosuppression and Catabolism Syndrome (PICS) [7-9].

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