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Editorial

Editorial Note on Lipid Therapy

Bindu Madhavi

Department of Biotechnology, Vignan's University, Vadlamudi, Andhra Pradesh, India

EDITORIAL NOTE

Lipid therapy, also known as fat therapy or therapeutic lipovenous injections, is a controversial medical procedure that involves the injection and expulsion of fats and lipids to enhance cognitive and memory function, according to proponents. Important fatty acids, such as linoleic acid (LA), an omega-6 fatty acid, and alpha-linoleic acid (ALA), an omega-3 fatty acid, are present in intralipid and other healthy lipid emulsions. The emulsion is used as part of intravenous nutrition for people who are unable to consume food through their mouths.

Experimental models of extreme cardio toxicity caused by intravenous overdoses of local anesthetic drugs including bupivacaine react well to lipid emulsions. They've helped people who haven't responded to traditional resuscitation approaches. They've also been used off-label to treat overdoses caused by other fat-soluble drugs. Intralipid is being researched as a possible cardio protective agent, primarily as a treatment for ischemic reperfusion injury. Although it is important to restore myocardial blood flow rapidly in order to save the ischemic heart, it also has the potential to cause damage due to oxidative stress and calcium overload. Calcium overload and increased reactive oxygen species with reperfusion open the mitochondrial permeability transition pore (mPTP), allowing hydrogen ions to migrate from the mitochondrial matrix into the cytosol. The hydrogen flux causes mitochondrial swelling and outer membrane rupture by disrupting the membrane potential and the release of pro-apoptotic factors.

In vivo rat models, intralipid given five minutes before reperfusion delays the opening of mPTP, making it a possible cardio protective agent. Researchers discovered that the cardio protective aspect of Intralipid is triggered by the accumulation of acylcarnitine in the mitochondria and includes inhibition of the electron transport chain, as well as an increase in ROS output during early reperfusion and activation of the reperfusion injury salvage kinase pathway. The accumulation of acylcarnitines in mitochondria inhibits the electron transport chain at complex IV, resulting in protective reactive oxygen species (ROS). The effects of ROS are both "location" and "time" sensitive, which means that both factors can eventually decide whether ROS are beneficial or harmful.

The created ROS, which are produced from electrons leaking from the mitochondria's electron transport chain, act directly on mPTP to prevent it from opening. ROS then stimulate signalling pathways in the mitochondria, causing mPTP opening to decrease and defence to be mediated. The Danger pathway is activated by ROS, which increases the phosphorylation of other pathways including phosphatidylinositol 3-kinase/Akt and extracellular-regulated kinase (ERK), which are both present in mitochondrial pools. Glycogen synthase kinase-3 beta (GSK-3) function is altered when the Akt and ERK pathways converge. Akt and ERK phosphorylate GSK-3, which inactivates the enzyme and prevents mPTP from opening. The mechanism by which GSK-3 prevents the mPTP from opening is unknown.

Correspondence to: Bindu Madhavi, Department of Biotechnology, Vignan's University, Vadlamudi, Andhra Pradesh, India.

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