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Infusion Materials for the IV PK Protocol

Essentiale N 5 ampoules per box 1 ampoule 5 ml	Average 2.5 boxes per treatment
Sodium Phenylbutyrate 10 grams per vial	2 to 5 grams needed per treatment
Leucovorin 5 to 10 infusions per vial	0.5 cc to 3 cc needed per treatment
Fusilev (LevoLeucovorin) 50 mg per vial	0.25 cc to 1.5 cc needed per treatment Use half the dose normally used, this is the pure isomer
Glutathione *USA* 10 & 100 mL per vial	3 cc to 20 cc needed per treatment
Glutathione *Europe* Tationil 600 mg per 4 mL vial	3 four mL vials needed per treatment
Methylcobalamin 10 mg per mL	10 mg per subq injection following IV treatment

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MEMBRANE MEDICINE

NeuroMetabolic Lipid Intervention as a Treatment for Encephalopathy 'Brain on Fire' following Mold, Lyme and Viral Exposure

Lipid Rescue Therapy for Biotoxic Illness with the use of a Membrane Stabilizing Protocol with Phenylbutyrate, Phospholipids, Leucovorin and Glutathione

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Keywords
Butyrate/Phenylbutyrate, Phosphatidylcholine, Essential Fatty Acids-SR3 oil, Cardiolipin, Mitochondria, Peroxisome, Phospholipase A2 (PLA2), Very Long Chain Fatty Acids (VLCFAs)

Abstract
Maintaining the appropriate balance and content of lipids in cellular membranes is critical for normal neural processes. Accumulating evidence suggests that even subtle perturbations in the lipid content of neurons and myelin can disrupt their function. The membrane and organelles within the cell are the primary focus of electrical discharge within the central nervous system and can be stabilized with phospholipid therapy and a Phospholipase A2 (PLA2) suppressive diet. Dietary intervention includes a targeted, essential fatty acid and phospholipid nutrient dense diet with low carbohydrate and moderate protein. The brain is 60% lipid, containing phospholipids, which comprise the plasma and organelle membranes, along with cardiolipin, located exclusively in the inner lipid membrane of mitochondria and myelin, and is a target for toxic exposure. Stabilization of cardiolipin is a primary therapeutic target in neurotoxic disorders which may be addressed using lipid therapy. Recent research has revealed that in the brain myelin acts as one enormous mitochondrion. Examination of d cell lipids at Johns Hopkins Peroxisomal Diseases Laboratory in subjects with Epilepsy, Autism, Alzheimer's, inborn errors in metabolism, Post Stroke, PDD, NeuroLyme, Mold toxicity, Multiple

Sclerosis, Motor Neuron and Parkinson's Disease over the past 17 years in 9000 analyses has revealed a characteristic accumulation of very long chain fatty acids (VLCFAs), which comprise lipid rafts, or ceramides, revealing cell membrane derangement per disturbance in peroxisomal respiration, which has a vital role in cell membrane integrity and function. Membrane phospholipid abnormalities with elevation of VLCFAs may be indicative of exposure to neurotoxins resulting in suppressed peroxisomal beta oxidation of VLCFAs. Identification of nuclear and mitochondrial DNA adducts (toxins) has revealed a link between toxic exposure and the development of cell membrane derangement / dysfunction. In our current studies we have captured visual images of distorted phospholipid membranes and have linked the impact of the DNA adducts (toxins) altering gene expression to aberrations in lipid metabolism, cellular dysfunction and alteration of the structure of phospholipids in the cell membrane characteristic to the presenting diagnosis and symptoms. Our treatment protocol includes an oral targeted lipid therapy, dietary with supplemental protocol, and in some cases an intravenous infusion of phenylbutyrate, phosphatidylcholine, Leucovorin, and glutathione to clear bioaccumulation of toxins impacting gene expression and to stabilize cellular architecture. Stabilization of cardiolipin, therefore myelin, and the cell/organelle membranes are new therapeutic targets in neurotoxic disease, which may be addressed in a clinical setting using targeted lipid therapy with emphasis on linoleic acid, phosphatidylcholine and phenylbutyrate with a balanced lipid modified ketogenic diet to optimize cellular function. While further work remains and the microscopic significance of these correlations needs further clarification, initial biochemical and clinical results appear promising. We have documented significant clinical neurological improvement in our patients, along with marked normalization of cellular function (via laboratory analysis) following three months of an oral and intravenous lipid regime. The administration of our lipid protocol may offer a new therapeutic strategy for patients with neurological disorders arising from infectious and toxic exposures.