

Huntington's Disease & HBOT

Huntington's disease (HD) is a neurodegenerative genetic disorder that affects muscle coordination and leads to cognitive decline and psychiatric problems. It typically becomes noticeable in mid-adult life. HD is the most common genetic cause of abnormal involuntary writhing movements called chorea, which is why the disease used to be called Huntington's chorea.

It is much more common in people of Western European descent than in those of Asian or African ancestry. The disease can affect both men and women. The disease is caused by an autosomal dominant mutation in either of an individual's two copies of a gene called Huntingtin, which means any child of an affected person typically has a 50% chance of inheriting the disease. Physical symptoms of Huntington's disease can begin at any age from infancy to old age, but usually begin between 35 and 44 years of age. Through genetic anticipation, the disease may develop earlier in life in each successive generation. About 6% of cases start before the age of 21 years with an akinetic-rigid syndrome; they progress faster and vary slightly. The variant is classified as juvenile, akinetic-rigid or Westphal variant HD.

The Huntingtin gene provides the genetic information for a protein that is also called "huntingtin". Expansion of a CAG triplet repeat stretch within the Huntingtin gene results in a different (mutant) form of the protein, which gradually damages cells in the brain, through mechanisms that are not fully understood. The genetic basis of HD was discovered in 1993 by an international collaborative effort spearheaded by the Hereditary Disease Foundation.

Genetic testing can be performed at any stage of development, even before the onset of symptoms. This fact raises several ethical debates: the age at which an individual is considered mature enough to choose testing; whether parents have the right to have their children tested; and managing confidentiality and disclosure of test results. Genetic counseling has developed to inform and aid individuals considering genetic testing and has become a model for other genetically dominant diseases.

Symptoms of the disease can vary between individuals and even among affected members of the same family, but usually progress predictably. The earliest symptoms are often subtle problems with mood or cognition. A general lack of coordination and an unsteady gait often follows. As the disease advances, uncoordinated, jerky body movements become more apparent, along with a decline in mental abilities and behavioral and psychiatric problems. Physical abilities are gradually impeded until coordinated movement becomes very difficult. Mental abilities generally decline into dementia. Complications such as pneumonia, heart disease, and physical injury from falls reduce life expectancy to around twenty years after symptoms begin. There is no cure for HD, and full-time care is required in the later stages of the disease. Existing pharmaceutical and non-drug treatments can relieve many of its symptoms.

Research and support organizations, first founded in the 1960s and increasing in number, work to increase public awareness, to provide support for individuals and their families, and to promote and facilitate research. Many new research discoveries have been made and understanding of the disease is improving. Current research directions include determining the exact mechanism of the disease, improving animal models to expedite research, clinical trials of pharmaceuticals to treat symptoms or slow the progression of the disease, and studying procedures such as stem cell therapy with the goal of repairing damage caused by the disease.

HBOT has successfully been used in the treatment of Multiple Sclerosis, Amyotrophic Lateral Sclerosis, Parkinsons Disease and Alzheimers Disease. All are neuro-degenerative disorders. HBOT does not cure any neuro-degenerative disorders but rather serves to minimize symptoms and maintain the body and brain at a functional level. The disease is not eliminated but merely controlled or maintained. The earlier treatment is initiated in relationship to the early diagnosis or discovery, the greater the benefits will be.

The following are studies and research documentation that is not necessarily Huntington Disease but associated with other disorders which are similar in etiology, signs, symptoms and progression.

Amyotrophic lateral sclerosis (ALS), also known as motor neuron disease or Lou Gehrig's disease, is a rare neuromuscular disease with an incidence rate of about 1 in 100,000. It is characterized by muscular weakness from the degeneration of motor neurons, and like PD, intellect and personality is often unaffected. The National Institute of Neurological Disorders and Stroke reports that only 5-10% of all ALS cases can be traced to genetics, particularly to a mutation related to the superoxide dismutase 1 enzyme. This leaves the vast majority of cases without a known etiology, with the potential for environmental association briefly outlined below.

Far fewer studies have examined the association of pesticides and ALS than for both AD and PD. McGuire et al. (1997) found that agricultural chemicals have a significant association with the development in ALS, with a stronger association for men than for women.

Metals may play a role in the development of ALS. Some studies have observed an association with occupation in welding or soldering (Armon et al. 1991; Gunnarsson et al. 1992), but not all have found metals to be related to ALS (Gresham et al. 1986; McGuire et al. 1997). More specifically, an association has been observed with exposure to lead (Armon et al. 1991; Chancellor et al. 1993; Felmus et al. 1976; Kamel et al. 2002), but no association was observed between ALS and lead levels in various tissues (Kapaki et al. 1989; Stober et al. 1983) or toenails (Bergomi et al. 2002); however, these studies had limited numbers of study participants. No association was observed between exposure to zinc and ALS (Vinceti et al. 2002), and the evidence from biomarker studies is inconclusive, with an increased (Gellein et al. 2003), decreased (Yasui et al. 1993), and no association observed for levels in brain tissue (Kapaki et al. 1997; Nagata et al. 1985) or toenails (Bergomi et al. 2002) compared with controls. However, these studies may have had limited power based on the size of the study population. Although one epidemiologic study showed no association between exposure to copper and ALS (Vinceti et al. 2002), there was decreased copper concentration observed in both cerebrospinal fluid and blood (Kapaki et al. 1997), and no association in toenails (Bergomi et al. 2002) among patients with ALS versus controls. Mercury was associated with ALS risk (Felmus et al. 1976) but was found in lower concentrations in the blood of ALS patients versus controls (Moriwaka et al. 1993).

Case-control studies examining biomarkers of iron, manganese, selenium, and Al and risk of ALS were found. Increased iron levels have been observed in brain tissue (Kasarskis et al. 1995; Yasui et al. 1993), although not in blood (Nagata et al. 1985) or toenails (Bergomi et al. 2002). An increase of manganese was observed in cervical cords (Miyata et al. 1983), both an increase (Kapaki et al. 1997) and decrease (Nagata et al. 1985) in blood levels, and no difference in toenail concentration (Bergomi et al. 2002) among cases versus controls. Selenium was found to be increased (Nagata et al. 1985) and decreased (Moriwaka et al. 1993) in blood cells, but no association was observed in toenails (Bergomi et al. 2002) of patients with ALS versus controls. An increase was observed in Al in central nervous system tissue (Yasui et al. 1991a, 1991b) and cerebrospinal fluid (Sood et al. 1990), yet others observed no association in spinal cords (Kasarskis et al. 1995) or toenails (Bergomi et al. 2002). However, the latter two studies had small numbers of study participants, possibly limiting the power to detect an association.

A few studies found a relationship between other exposures and ALS. Gunnarsson et al. (1992) found a nonsignificant association with solvents, but the association was stronger and statistically significant for males with family history of neurodegenerative disease or thyroid disease. Others found conflicting results (Chancellor et al. 1993; McGuire et al. 1997). One study found that those with a history of occupation in the manufacturing of plastics have a significant association with the development of ALS (Deapen and Henderson et al. 1986). Occupations in electrical work have been implicated in the development of ALS in a few studies (Deapen and Henderson 1986; Gunnarsson et al. 1992).

Epidemiologic evidence for an association between environmental agents and neurodegenerative disease is inconclusive. The amounts of xenobiotics released into the environment are huge by any measure, and the paucity of information about their effects on various physiologic systems, including neurodevelopmental processes, represents a major gap in knowledge. To close this gap, the following broad areas of research topics need attention: a) better health tracking and monitoring data for chronic diseases, b) more comprehensive and longitudinal biomonitoring of environmental agents that can be linked with specific molecular/biochemical markers of exposure and subsequent health outcome data, and c) more epidemiologic research and testing of environmental agents to better define their effects on the adult and developing brain, as well as other critical organ systems.

Until such time that ethically and scientifically well-designed epidemiologic studies can provide a reasonable certainty that specific environmental agents, either alone or in combination with other agents, cause a given neurodegenerative disease, research on the environmental contribution to neurodegenerative disease needs to continue.

This article is part of the mini-monograph "Early Environmental Origins of Neurodegenerative Disease in Later Life: Research and Risk Assessment."

A Phase I Study of Hyperbaric Oxygen Therapy for Amyotrophic Lateral Sclerosis

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BACKGROUND: Vascular endothelial growth factor and mitochondrial abnormalities have been described in ALS and its animal models. We have reported that hyperbaric oxygen (HBO) treatment delayed the onset of weakness in the wobbler mouse. **OBJECTIVE:** To perform a Phase I safety study of HBO in patients with ALS.

METHODS: Five patients with ALS were treated for 60min with 100% oxygen at 2 atmospheres pressure daily for five days a week for four weeks. The patients reported any deterioration in their condition after each treatment, and their neurological condition was measured serially during the four weeks of the treatment, and for four further weeks.

RESULTS: Four patients reported decreased fatigue, while one patient dropped out at three weeks because of increased fatigue. Maximum isometric voluntary contraction (MVIC) of all muscle groups except right hand grip improved significantly by up to 97%. Most improvement occurred during the four weeks after treatment. It is possible that the improvement in muscle strength was a placebo or a learning effect, though no such effects have been detected in prior therapeutic trials in ALS using MVIC. No change was detected in other measures of neuromuscular function.

CONCLUSIONS: A longer duration, placebo controlled trial in a larger number of patients is needed to determine the safety and efficacy of HBO. Until that is completed, it is not recommended that ALS patients should be treated with HBO.

The improvements seen in the four weeks following treatments are due to the continued and completed growth and repair of nerves and blood vessels. This is typical of all clients, not just neuro-degenerative cases.

Alzheimer's Study links poor blood flow

by MALCOLM RITTER The Associated Press

NEW YORK - Rogue bits of a natural protein may promote Alzheimer's disease by disrupting the flow of blood in tiny vessels of the brain, a study suggests. The study provides more evidence that vitamin E and other antioxidants may fight the disease, and suggests that finding treatments to restore normal blood flow may also pay off. Scientists don't know what causes most cases of Alzheimer's. Many point to overproduction of natural protein fragment called amyloid-beta, which form clumps in the brains of patients. Studies show these fragments can kill brain cells. The new work suggests amyloid-beta, or related fragments, can promote Alzheimer's in a second way: by boosting production of harmful

substances called oxygen radicals, which in turn keep tiny blood vessels from delivering the right amounts of blood to brain cells.

The study is presented in the February issue of the journal *Nature Neuroscience* by neurologist Dr. Constantino Iadecola of the University of Minnesota with colleagues there and elsewhere. Dr. Zaven Khachaturian, senior medical and scientific adviser to the Alzheimer's Association, called the work exciting and said it reveals "a very important part of the story" of what causes the disease. In the brain, amyloid-beta fragments are clipped from long proteins called APP. The researchers studied a strain of mice that overproduce APP, which leads to an overproduction of amyloid-beta. Mice from this strain eventually develop mental problems resembling Alzheimer's.

In the latest study, the mice were studied long before any Alzheimer's-type symptoms appeared. Researchers found that microscopic blood vessels in the mice brains didn't respond to a chemical signal to dilate, which would increase blood flow. In normal life that might mean the vessels can't shunt more blood to brain cells when they need it, Iadecola said. Eventually that could damage those starved cells, or at least make them more vulnerable to damage from other causes, he said. But because the mice produce other fragments of APP in excess, the study can't formally show that amyloid-beta is the cause of the brain troubles in the mice, Iadecola said. The researchers found two bits of evidence that oxygen radicals were involved in the blood vessel problem.

Researchers were able to prevent the abnormality by bathing the brain with an antioxidant, which render oxygen radicals harmless. In addition, mice that were programmed genetically to overproduce an antioxidant in addition to APP didn't show the abnormality in the first place. Iadecola said a 1997 study of Alzheimer patients found that vitamin E, an antioxidant, modestly slowed the course of the disease.

Hyperbaric Oxygenation and Neurological Recovery in Children with Organic Brain Damage

Cordoba-Cabeza-T; Perez-Fonseca-R; Morales-Vargas-D; Lopez-A: Oxigenacion hiperbarica y restauracion neurologica en ninos con dano cerebral organico. *Rev-Neurol*. 1998 Oct; 27(158): 571-4

INTRODUCTION AND OBJECTIVE: In order to determine the effects of hyperbaric oxygenation, 14 Cuban children (8 boys and 6 girls) with affected lesions on the Central Nervous System (CNS), were prospectively studied between September 1994 and September 1995; the patients came from the external consultation of Neuropediatric of the Academic Pediatrics Hospital, Tuna Martyrs, Cuba.

PATIENTS AND METHODS: They were evaluated from the neuropediatric and neurophysiologic point of view and submitted to treatment with hyperbaric oxygenation (HBO). Five patients showed a injury to the CNS by severe asphyxia, seven by generalized infections of CNS, one patient with cerebral damage by craneoencephalic traumatism and other with vasocclusive cerebral crisis of siclemic. The average age of the children was of 4.8 +/- 3.4 years. The symptoms and signs were depending on the type on cerebral damage and its evolution. 100% of the children presented infantile cerebral paralysis (ICP) and epilepsy, most of the children were in treatment with antiepileptic drugs, buy they weren't balanced.

RESULTS AND CONCLUSIONS: Satisfactory response was observed in patients that were oxygenated within the first year of the lesion, with better and faster results.

Inhibition of Apoptosis by Hyperbaric Oxygen in a Rat Focal Cerebral Ischemic Model.

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The hypothesis was tested that hyperbaric oxygen therapy (HBO) reduced brain infarction by preventing apoptotic death in ischemic cortex in a rat model of focal cerebral ischemia. Male Sprague-Dawley rats were subjected to middle cerebral artery occlusion/reperfusion (MCAO/R) and subsequently were exposed to HBO (2.5 atmospheres absolute) for 2 h, at 6 h after reperfusion. Rats were killed and brain samples were collected at 24, 48, 72 h, and 7 days after reperfusion. Neurologic deficits, infarction area, and apoptotic changes were evaluated by clinical scores, 2,3,7-triphenyltetrazolium chloride staining, caspase-3 expression, DNA fragmentation assay, and terminal deoxynucleotidyl transferase-mediated 2'-deoxyuridine 5'-triphosphate-biotin nick end labeling (TUNEL)-hematoxylin and eosin (H&E) costaining. In MCAO/R without HBO treatment animals, DNA fragmentation was observed in injured cortex at 24, 48, and 72 h but not in samples at 7 days after reperfusion. Double labeling of brain slides with NeuN and caspase-3 demonstrated neurons in the injured cortex labeled with caspase-3. TUNEL+H&E costaining revealed morphologic apoptotic changes at 24, 48, and 72 h after reperfusion. Hyperbaric oxygen therapy abolished DNA fragmentation and reduced the number of TUNEL-positive cells.

Hyperbaric oxygen therapy reduced infarct area and improved neurologic scores at 7 days after reperfusion. One of the molecular mechanisms of HBO-induced brain protection is to prevent apoptosis, and this effect of HBO might preserve more brain tissues and promote neurologic functional recovery.

Clinically Observed Reduction of Spasticity in Patients with Neurological Diseases and in Children with Cerebral Palsy from Hyperbaric Oxygen Therapy

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The author presents case studies of 230 children with spastic cerebral palsy who received Hyperbaric oxygen therapy (20 sessions of 1 hour each).

The author observed a significant reduction in Spasticity and improved respiratory function in 94%, and continued reductions in Spasticity, improved motor control and a reduction in convulsions and episodes of bronchitis in 75% of those followed for 6 months.

Hyperbaric Oxygen Treatment on a Parkinson's Disease Patient: A Case Study

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INTRODUCTION Parkinson's Disease (PD) is a chronic neurodegenerative disorder, which is characterized by the loss of dopaminergic neurons whose cell bodies are located in the substantia nigra pars compacta (SNpc) and project to the striatum. The initiation of this neuronal degeneration is not known, however the process of neuronal loss is suggested to occur via apoptosis rather than by necrosis (1). With the onset of the neurodegeneration of these neurons is the associated loss of the neurotransmitter, dopamine (DA), from its nerve endings and its subsequent release in the striatum. The major symptoms which are observed due to the progressive loss in function of the nigro-striatal dopaminergic neurons may be one or more of the following: resting tremor, rigidity, bradykinesia and/or postural instability. The actual clinical manifestation of the disease in any one patient is highly dependent upon the degree of severity of the neuronal loss, age of the patient and the length of time passed between the onset of the symptoms and the time of diagnosis. Early detection is important in order to institute a therapeutic strategy to relieve the symptoms and/or delay the progression of the disease state.

The major treatment strategy currently used is to affect the function of DA. Because systemically administered DA does not cross the blood-brain barrier; Levodopa (pro drug) is administered, which is taken up into the brain. Since Levodopa

is metabolized both peripherally and centrally to DA by a DOPA decarboxylase, carbidopa an inhibitor of this enzyme is administered in combination with Levodopa to decrease its metabolism peripherally increasing its uptake into the brain. DA agonists and monoamine oxidase-B (MAO-B) inhibitors are also administered as a monotherapy or as an adjunct to Levodopa-carbidopa (Sinemet) therapy, depending upon the clinical condition.

Taking a very different approach in the treatment of PD, Borromei et al. in 1996 showed that hyperbaric oxygen (HBO) therapy appeared to be effective in ameliorating many of the behavioral and motor deficits observed in PD patients (2). The objective of this study was to determine whether HBO therapy might enhance the effects of an antiparkinson treatment in a PD patient as an adjunct therapeutic modality.

METHODS Brief patient history: A 72 year old male was diagnosed with idiopathic PD and placed on Sinemet (10/100) three doses 3 times daily. One year after diagnosis for PD the patient was diagnosed with total occlusion of the right coronary artery. A successful total occlusion angioplasty was performed and he was placed on Lopressor and Lipitor 10 mg daily. There were no complications from this surgical procedure. Eighteen months after being diagnosed as a PD patient he was treated with hyperbaric oxygen (HBO) at 1.9 ATA for 90 min. The patient was treated daily 5 times each week for 5 weeks (25 treatments). During the treatment the patient gradually reduced his dose of Sinemet until he was completely off of this medication between the 3rd and 4th week of HBO treatment. At this point his physician placed him on selegiline 10mg twice daily.

Clinical testing: The patient's voice and speech were evaluated by a speech-language pathologist, and the Jebsen-Taylor hand function test was performed by an occupational therapist prior to and after the end of the HBO therapy. The patient was informed of all aspects of hyperbaric oxygen therapy, including all risks of adverse effects according to the Declaration of Helsinki. The patient also signed an informed consent form detailing the treatment and the rights of the patient.

RESULTS Voice and speech. There was little change in the overall evaluation of voice and speech after HBO therapy. Communication status changed very little. He appeared to be talking more and his rate was somewhat improved. He still had difficulty projecting his voice.

Jebsen-Taylor Hand Function Test. The results of this test are shown in Table 1. In testing the dominant hand there were small increments of improvement after HBO. The total improvement was more than 10%, while the improvement in the non-dominant hand was nearly 32%.

During the treatment period, the patient voluntarily reduced his Sinemet doses until he was completely off the drug after 3-4 weeks of HBO therapy, which was an unexpected result. He has continued to remain off of Sinemet therapy. No complications or adverse side effects such as myopia were observed. The long-term exposure of HBO was tolerated well by the patient.

DISCUSSION PD is characterized by the loss of dopaminergic neurons of the nigro-striatal pathway. It is not clear how this neuronal degeneration is initiated, but there appears to be a number of potential ways in which this might occur in any one individual, including genetics, disease, drugs or other chemicals, oxidative stress and/or other environmental factors. However, once it is initiated there seems to be agreement that the degenerative process involves apoptosis and not necrosis.

The results of this study suggest that HBO might be a possible new modality of treatment for PD because it appeared to be able to replace Sinemet as a therapeutic regimen. The mechanism by which the HBO effect might be occurring may be partly due to an anti-apoptotic effect. It has been shown that HBO increased the expression of Bcl-2 protein, a major anti-apoptotic protein, in treating forebrain cerebral ischemia in gerbils (3). The Bcl-2 protein has also been elevated by

repeated HBO treatment in normal gerbils (4). So it is possible that HBO in this study inhibited the apoptotic pathway involved in the progressive neuronal degeneration by stimulating the expression of the Bcl-2 proteins.

Other possible HBO effects should not be discounted such as improved oxygen perfusion due to increased extravascular oxygen diffusion and to possible angiogenesis (5). Axonal repair and regeneration and/or synaptogenesis could occur due to increased expression of neurotrophin(s), since HBO has been shown to increase vascular endothelial growth factor (6) and act synergistically with platelet derived growth factor and transforming growth factor-beta (7).

The results of this case study agree with much of the results observed in the clinical study by Borromei and his coworkers. It is not clear from their study whether some of their patients were concurrently being treated with anti-parkinson drugs. In our study, HBO replaced the Sinemet therapy and appeared to improve the clinical condition. Thus, results from this case study suggest that HBO therapy might be a potential therapeutic modality in treating patients suffering from PD without causing untoward side effects such as dyskinesia observed in long-term Sinemet therapy.

In conclusion, we suggest that HBO therapy might be neuroprotective in nature to the nigro-striatal neurons by acting as an antiapoptotic process. This could stabilize neuronal function, thereby potentially decreasing the progression of the neurodegeneration observed in Parkinson’s Disease.

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Table 1. Jebsen-Taylor Hand Function Test.

Clinical Testing : Time (in sec)	Pre-HBO	Post-HBO
Dominant Hand		
Writing	14	12
Card Turning	7	6
Manipulating Small Objects	11	11
Simulated Feeding	11	10
Stacking Small Objects	8	5

Lifting Large Light Objects	11	10
Lifting Large Heavy Objects	8	6
Total	70	60
Non Dominant Hand		
Writing	41	28
Card Turning	8	4
Manipulating Small Objects	11	7
Simulated Feeding	12	12
Stacking Small Objects	13	8
Lifting Large Light Objects	9	6
Lifting Large Heavy Objects	7	5
Total	101	70

These are tidbits of conversation gleaned from a Huntington Disease members' website:

HDFighter

Re: Hyperbaric Oxygen Chamber Therapy

My wife has used HBOT. She has a 42 cag count, is 60 years old, and her chorea symptoms began to really be noticeable about 4 years ago. We've used HBOT, but I've never found anybody who has really nailed down what pressure is best for helping neuronc stem cell rejuvenation. Some things I read said 4.5 psi was best for neurological problems (such as Parkinson's), but other sources recommended much higher pressures as helping even HD.

Even if HBOT does increase a person's stem cell production, without using things that dissolve the amyloid plaque (like Neotine, Trehalose, and lots of other supplements all together), HBOT alone won't take care of HD.

As for my wife, we're fairly sure that if we had not tried all these things her chorea would probably be much worse than it is. If anything, we can say she has not gotten terribly worse and her rate of deterioration has lessened since we started combining all the things I've mentioned.

huntingtons research foundation

A Little Info & Clearing of the Air

We put 5 people in the chamber for 20 sessions an hour long each over the course of two weeks at 2 and 3 atmospheres. This was done by Richmond Hyperbarics here in B.C. We found that after a few treatments that there was a definite improvement. The individuals who went in the tank all loved it. Said it made them feel better. I would not say that it was significant but it was most certainly worth it. We found that motor control was improved and that overall cognitive abilities had improved. As an observer I definitely noticed a difference in all the people who participated. These individuals had varying conditions 2 H.D and 2 P.D 1 M.S. The M.S patient showed almost unbelievable progress. When she arrived in Vancouver she could hardly walk when she was finished she was able to throw away the walker and cane, she was very active. We have had demands for another round from all involved which we will provide as soon as we are able to obtain the funding.

There is no question that the therapy is expensive. We spent \$8,000 on the project and we were getting it done at cost because the institution that we did it at was gathering information to provide to Health Canada and they considered it a

study group. If you all were in B.C I would be happy to get you all in if we could and if accommodations can be arranged we would be happy to pay for the therapy. My opinion is to find someone who is sympathetic to your cause and ask them for a discount we found one center on Vancouver Island who is willing to do it for \$50 a session. Given the current state of our respective economies you may find someone who is willing to help. Other than that it will provide a benefit but do not expect any miracles. I think its something that should be done annually and looked at as a progressive way of prolonging or enhancing your quality of life.

We will be posting before and after videos of our participants on our site next week. Along with those, testimonials that barb says is only motivated by profit. I find this amusing if we did not collect testimonials how could we begin to represent any therapy to interested parties?

Watch out for Barb, she has an agenda and she is trying to stop people from making their own conclusions. It seems more misguided than malicious but if you look at my other post you will see that she is not interested in an exchange of info and opinion.

We are working with natural doctors, who are real doctors, ones with just as much training and experience as any M.D. Just because someone decided to not go with chemical drugs doesn't mean that they are not a doctor. We also have an M.D coming on board he sought us out because he believes in what we are doing and in natural therapies. This line that is drawn between pharma and natural is weird to me. You should always take what is safest and most effective regardless of what it is. This conflict is counter productive and most of these drugs are synthesized from plants anyways so whats the big deal? She also keeps telling people that we work with herbalists that is not true at all. We do not have a single herbalist on our team although we did consult with one on adding Ginko Biloba to our mixtures.

One more thing Barb has stated that we are interested in marketing a product and that in the end we will only use our health study as a tool to make some money. You need to know that we are literally spending all of our money on providing these therapies for free. To date not a single penny has been charged and there has been a real demand here in Canada, everyone is feeling the benefits. This makes costs for a small nonprofit formidable. If I wanted to market a product I could have done it without this health study. Even if we do have a retail product which is the only way we could do this in the states it would be at an affordable price and for those who are truly impoverished we would provide it at cost or less in order to deliver the benefits. I really do not get what she is thinking. Is she saying that... lets pick one, say say ACR 16, if it works well do you think that it will be free and no one will profit off of it? Last time I checked pharmaceutical companies are more or less solely motivated by profit and some of the largest businesses in the world. As Americans you should have a really good idea about how much they tax people for drugs and medical supervision that may or may not work and all without exception have potential side effects. In our organization no one makes over \$30,000 per year and our top hourly earner is \$10 per hour. I made \$9,000 last year and operated a moving business to make ends meet. I have a feeling that is not the case with the other "trusted" groups. These guys make millions and have six figure salaries if there are real questions about motivation and profiteering I suggest you start there. If you do your diligence on that keep in mind that these people work very hard and running a foundation is a full time job. People have to earn a living and is it not better to earn one helping people instead of say slaving away for an oil company that destroys the earth and charges you up the you know what? These people should be applauded for choosing to work in an industry that helps people not ostracized because they make a pay cheque off of helping people like us.

I will leave you all with this: trust no one, do your own research. Opinions are motivated by all sorts of things and some of them may be preventing you from getting all the facts. Make all decisions regarding your health with a trusted health professional and your loved ones. I also recommend that you all take trehalose no matter where from. Its cheap, effective and proven to work.