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Slowing the Progression of Amyotrophic Lateral Sclerosis (Motor Neuron Disease)

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David Steenblock's Unique Stem Cell Treatment
Program for ALS

8/9/2011

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The article titled “Amyotrophic Lateral Sclerosis: Stem Cell Healing & Amyotrophic Lateral Sclerosis (ALS)*By [David Steenblock, M.S., D.O.](#)” is © 2011 by David A. Steenblock, D.O., Inc. All rights reserved. Used with permission in this compendium of information. The information contained in this article is provided for informational purposes only and should not be construed as medical, sexual or psychological advice or instruction. Readers are advised to consult the appropriate licensed health care or behavioral sciences professional concerning physical or mental health concerns or challenges.

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NOTA BENE: The regimen that follows is informed (in part) by a line of work that culminated in the publication of this paper: [Experimental regimen targeting the ependyma slows disease progression in four patients with amyotrophic lateral sclerosis.](#)

ALSO: Many of the supplement links in this monograph are to offerings from www.iherb.com. Neither Dr. Payne nor David A. Steenblock, D.O., Inc. have any commercial ties to Iherb nor benefits in any way whatsoever from sales of its products. Iherb was chosen because it offers very low prices on all products and superb order fulfillment.

DIET: KETOGENIC OR MCT

A multitude of studies have been published that point to the fact that ketones generated by the [Ketogenic diet](#) & especially the [Medium Chain Triglyceride](#) Diet (respectively) helps keep the dysregulated mitochondria in ALS-stricken neurons working optimally (The ketogenic diet is a high fat, high protein, super low carbohydrate diet that favors production of ketones by the liver. The Atkin's diet produces weight loss through ketogenic means). Given this fact MND patients should seriously consider going on one or the other (Nota bene: The MCT diet generates more ketones than the ketogenic diet). But they should not attempt to do so on their own, as runaway ketosis can create health problems in its own right. As such, patients should always involve an MD or DO and/or a registered dietician in planning their diet and monitoring ketone production status. A link follows below to an on-line, simple-to-use tool for calculating carbohydrate intake so as to encourage ketogenesis.

<http://www.phlaunt.com/lowcarb/DietMakeupCalc.php> - Nutrition calculator for folks on the ketogenic diet

<http://www.biomedcentral.com/content/pdf/1471-2202-7-29.pdf> - **A ketogenic diet as a potential novel therapeutic intervention in amyotrophic lateral sclerosis.**

<http://www.alsforums.com/definitions/ketogenic-diet.html> - Ketogenic diet - ALS forum

[Medium Chain Triglyceride](#) or [Ketogenic diet](#) + drink caffeinate coffee or tea liberally ([Male sALS patients only!](#)) and use [Trehalose](#) to sweeten foods and beverages. There are many reasons the ketogenic or MCT diets might be of benefit to ALS and other MND patients, not the least of which is the fact they tend to increase glutamate transporter gene expression. Trehalose induces autophagy – digestion of abnormal proteins in neurons.

[Liquigen](#)

[Click to access one supplier of MCT powder](#)

[Click to access Ketogenic diet cookbooks on Amazon.com](#)

[Axona](#): Prescription MCT product for inducing ketogenesis.

ALSO: In a [paper from 1940](#) researchers clearly found that orally ingested acetic acid -- Apple Cider Vinegar and such -- increases ketone levels in fasting animals. The basic physiology involved is the same in humans, so it follows that oral ingestion of acetic acid (vinegar) will help increase ketone output from the liver. NOTE: For those readers who find consuming vinegar unpalatable, there is a capsule form available:

[One low cost source of Cider Vinegar capsules](#)

[Trehalose, a novel mTOR-independent autophagy enhancer, accelerates the clearance of mutant huntingtin and alpha-synuclein.](#)

Department of Medical Genetics, Cambridge Institute for Medical Research, University of Cambridge, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2XY, United Kingdom.

Trehalose, a disaccharide present in many non-mammalian species, protects cells against various environmental stresses. Whereas some of the protective effects may be explained by its chemical chaperone properties, its actions are largely unknown. **Here we report a novel function of trehalose as an mTOR-independent autophagy activator.** Trehalose-induced autophagy enhanced the clearance of autophagy substrates like mutant huntingtin and the A30P and A53T mutants of alpha-synuclein, associated with Huntington disease (HD) and Parkinson disease (PD), respectively. Furthermore, trehalose and mTOR inhibition by rapamycin together exerted an additive effect on the clearance of these aggregate-prone proteins because of increased autophagic activity. By inducing autophagy, we showed that trehalose also protects cells against subsequent pro-apoptotic insults via the mitochondrial pathway.

DIMETHYLSULFOXIDE (DMSO)

TOPICAL DMSO

One of the players in ALS is compromised functioning in astrocytes in the central nervous system (Astrocytes pump excess excitotoxic glutamate away from motor neurons). The study below indicates that DMSO counters ATP depletion in neurons under certain circumstances (Depletion of ATP - the gasoline in mitochondria and cells -- results in all kinds of cell damaging effects). Mind you, this study was testing a wholly different kind of mechanism that depletes ATP than occurs with ALS -- yet it is conceivable that DMSO will nonetheless address the compromised mitochondrial energetics that prevails in ALS and other MNDs including ATP dysregulation. To try DMSO all a person need do is purchase a gel or liquid and apply it to any part of their body. The DMSO will go through the skin like greased lightning and wind up circulating in the brain in a heartbeat. And it is a relatively safe compound that enjoys FDA approval for IV use in treating interstitial cystitis (A link follows below to a website that sports information on drug interactions and such).

http://www.herbalremedies.com/dmsol.html?source=google&engine=adwords!32&keyword=%28Dimethyl+sulfoxide%29&match_type=&gclid=CKKv8Jv465sCFQ6jagod1nJf5A – Many DMSO product forms

<http://www.drugs.com/cdi/dimethyl-sulfoxide.html> - DMSO drug facts

Eur J Neurosci. 2004 May;19(9):2446-54. Links

[Energy failure in astrocytes increases the vulnerability of neurons to spreading depression.](#)

The neuroprotective effects of dimethyl sulfoxide, deferoxamine and fructose-1,6-bisphosphate suggest that oxidative stress contributes to the neurotoxicity in this situation.

NADH LOZENGES

STABILIZED SUBLINGUAL HIGH-DOSE NADH LOZENGES

There are various studies that have demonstrated that stabilized NADH bolsters neural energetics (The flow of energy within neurons). Here is a link to a commercial website that has assembled some of these studies: <http://www.nadh.com/library.htm#Top>

And here is a link to a high dose form of stabilized sublingual NADH:

<http://www.iherb.com/Co-E1-NADH-Sublingual-20-mg-30-Lozenges/9292?at=0> – Stabilized sublingual NADH lozenges (20 mgs.)

PYRUVATE, CREATINE, MALATE, NICOTINAMIDE

The 3 items below activate a glutamate pump on blood vessel walls that abuts brain tissue. In short, these will help shuttle excess glutamate from CNS tissues into the bloodstream where it will be transported to the liver for processing

Pyruvate - 1 gram tablets – Suggested use: One tablet every 4 hours. Total dose: 4-5 grams daily. If swallowing is a problem, grind up using a mortar & pestle and ingest.

Creatine -- Suggested use: 2-3 grams per day in divided doses. If swallowing is a problem, use a creatine powder: [Creatine Powder](#)

Citrulline malate powder - Suggested use: Take 4 grams daily in divided doses

Slow Release Nicotinamide - 1.5 gram – Suggested use: 1 tablet in the morning and 1 tablet in the afternoon or evening. **Many studies indicate that niacinamide curtails neuroinflammation and is neuroprotective** (See abstracts that follow this section)

http://wis-wander.weizmann.ac.il/site/en/weizman.asp?pi=422&doc_id=4793&interID=4787&sq=4787

<http://www.dana.org/printerfriendly.aspx?id=7376>

This NIH funded study illustrates some of the biochemical "reasoning" behind these supplement recommendations: <http://clinicaltrials.gov/ct2/show/NCT00605930> It is hypothesized that preservation of brain energy homeostasis may allow endogenous neuroprotective mechanisms to reverse or impede free radical injury or other neurotoxic events leading to neurodegeneration in this disease. An emerging literature has described the neuroprotective effects of pyruvate, (as a neuronal energy fuel and free radical scavenger); niacinamide, (which boosts cofactor NAD), and creatine, (which buffers and selectively parcels cellular energy utilization) in various animal models of brain injury or degeneration. Ajay Verma *et al* have further demonstrated a synergistic neuroprotective effect of these three nutrients in various neural injury models.

[Homeostasis of glutamate in brain fluids: an accelerated brain-to-blood efflux of excess glutamate is produced by blood glutamate scavenging and offers protection from neuropathologies.](#)

[Brain neuroprotection by scavenging blood glutamate.](#)

[Neuroscience](#). 2009 Jan 12;158(1):301-8. Epub 2008 Mar 18. [Links](#)

[Homeostasis of glutamate in brain fluids: an accelerated brain-to-blood efflux of excess glutamate is produced by blood glutamate scavenging and offers protection from neuropathologies.](#)

[Blood-mediated scavenging of cerebrospinal fluid glutamate.](#)

The maintenance of brain extracellular glutamate (Glu) at levels below its excitotoxic threshold is performed by Glu transporters present on glia and neurons as well as on brain capillary endothelial cells which remove brain Glu into blood.

Results from cerebroventricular perfusions with [3H]Glu, intracerebroventricular injections of [3H]Glu, and measurements of the basal CSF Glu levels point out to the same conclusion that the intravenous administration of pyruvate and oxaloacetate which decreases blood Glu levels accelerates the brain-to-blood Glu efflux. We conclude that the brain extracellular Glu levels can be controlled in part by the blood Glu levels.

Abstracts concerning NIACINAMIDE

Clin Exp Immunol. 2003 Jan;131(1):48-52.

[Nicotinamide is a potent inhibitor of proinflammatory cytokines.](#)

In conclusion, the present study could not only confirm previous reports of a down-regulatory effect on TNFalpha, **but demonstrates that nicotinamide is a potent modulator of several proinflammatory cytokines. These findings demonstrate that nicotinamide has a potent immunomodulatory effect in vitro, and may have great potential for treatment of human inflammatory disease.**

Trends Pharmacol Sci. 2003 May;24(5):228-32.

[Nicotinamide: necessary nutrient emerges as a novel cytoprotectant for the brain.](#)

As both a therapeutic agent and an investigational tool, nicotinamide offers new therapeutic strategies for degenerative disorders of the CNS.

Mol Cell Biochem. 1999 Mar;193(1-2):119-25.

[Newly discovered anti-inflammatory properties of the benzamides and nicotinamides.](#)

Taken together these data strongly support the notion that benzamides and nicotinamides have potent anti-inflammatory and antitumor properties, because their primary mechanism of action is regulated by inhibition at the gene transcription level of NF-kappaB, which in turn inhibits TNFalpha and induces apoptosis.

METHYLCOBALMIN

[Methylcobalamin - Lozenge](#) – (5 mgs.) – Suggested use: **Dissolve 2 lozenges 5 x daily under tongue.** Use 10 lozenges on Monday, 10 lozenges on Thursday, and 10 lozenges on Sunday) Studies done in Japan indicate that injected methylcobalamin slows ALS progression. Injectable methylcobalamin is difficult to obtain in the US, which is why the focus here is on lozenges which release methylcobalamin for absorption by oral cavity tissues.

: [Brain Nerve](#). 2007 Oct;59(10):1141-7. [Links](#)

[Clinical trials of ultra-high-dose methylcobalamin in ALS](#)

A vitamin B12 analog, methylcobalamin, has a protective effect on cultured cortical neurons against glutamate-induced cytotoxicity. We have shown the ultra-high-dose methylcobalamin (25 mg/day i.m.) slows down the progressive reduction of the CMAP (compound muscle action potential) amplitudes in ALS in the short term (4 weeks). The latencies of SSR (sympathetic skin response) were shorter after treatment (50 mg/day i.v., 2 weeks). In the long-term effect of methylcobalamin (50 mg/day i.m., twice a week), the survival time (or the period to become respirator-bound) was significantly longer in the treated group than in the untreated.

MAGNESIUM TAURATE + PREVAGEN®

[Magnesium Taurate \(Magnesium + Taurine\)](#) - Suggested use: 1 to 2 capsules daily. The compounds in this product have a beneficial impact on calcium homeostasis and energy-production in neurons in various studies.

[Prevagen](#) – 20 mgs every 2-3 hours and before bed. Prevagen Pro® 40 mg. capsules are sold to healthcare professionals.

Encourages calcium homeostasis in neurons. In ALS abnormal calcium influx leads to overload and cell death.

[Amino Acids](#). 2008 Feb;34(2):321-8. Epub 2006 Sep 8. [Links](#)

[Taurine increases mitochondrial buffering of calcium: role in neuroprotection.](#)

The neuroprotective role of taurine was mediated through regulation of cytoplasmic free calcium ($[Ca^{2+}]_i$), and intra-mitochondrial calcium homeostasis, as determined by fluo-3 and $(45)Ca^{2+}$ -uptake. **Furthermore, the overall mitochondrial function was increased in the presence of taurine, as assessed by rhodamine accumulation into mitochondria and total cellular ATP levels.** We specifically tested the hypothesis that taurine reduces glutamate excitotoxicity through both the enhancement of mitochondrial function and the regulation of intracellular (cytoplasmic and intra-mitochondrial) calcium homeostasis. **The role of taurine in modulating mitochondrial calcium homeostasis could be of particular importance under pathological conditions that are characterized by excessive calcium overloads.** Taurine may serve as an endogenous neuroprotective molecule against brain insults.

[J Neurosci](#). 1999 Nov 1;19(21):9459-68. [Links](#)

[Growth factors and taurine protect against excitotoxicity by stabilizing calcium homeostasis and energy metabolism.](#)

We conclude from these data that bFGF and taurine prevent glutamate excitotoxicity through regulation of $[Ca^{2+}]_i$ and mitochondrial energy metabolism. Furthermore, the neuroprotective role of taurine and bFGF was enhanced by their collaboration.

DEPRENYL

[Deprenyl](#) – to 12 mgs daily maximum (Physician must determine dosage). **Liquid forms for sublingual dosing are available.** **Conferred protection against neurotoxic compounds in the cerebral spinal fluid in animal models.**

DEPRENYL: According to a 1994 animal study, “CSF samples from ALS and non-ALS neurological patients were injected into the spinal subarachnoid space of 3-day-old rat pups, followed by a single dose (0.01 mg/kg body weight) of (-)-deprenyl, administered 24 h after CSF injection. After a further period of 24 h, the rats were sacrificed and the spinal cord sections were stained with antibodies against phosphorylated neurofilament (NF, SMI-31 antibody) and glial fibrillary acidic protein (GFAP). Activity of lactate dehydrogenase (LDH) was also measured. The injected (-)-Deprenyl resulted in a significant (61%) decrease in the number of SMI-31 stained neuronal soma in the ventral horn of the spinal cord of the rats exposed to the ALS CSF. This was accompanied by a reduction in the immunoreactivity of astrocytes for GFAP and a significant (35%) decrease in the LDH activity. It is suggested that the (-)-deprenyl confer protection against the neurotoxic compound or compounds present in ALS CSF.” [9]

Dose: Discretionary with each patient’s physician. Use of patches or oral forms (Pills, tablets or liquid). The typical daily dose was 12 mgs/daily.

ACETYL-L-CARNITINE & GBE

[Acetyl-L-Carnitine](#) – Suggested use: 4-8 grams (4000-8000 mgs.) daily in divided doses preferably on an empty stomach. **Powder forms are available for those who have trouble swallowing pills or tablets.**

Mechanism of action: Increases nrf in neurons, a protein in the CNS that is very neuroprotective. Also lowers neuron-damaging ceramide levels.

[Standardized Ginkgo Biloba Extract \(GBE\)](#) – 2-3 capsules 3 x daily.

<http://www.researchgrantdatabase.com/g/1R21NS062302-01A2/NRF2-ARE-PATHWAY-AS-A-THERAPEUTIC-TARGET-FOR-AMYOTROPHIC-LATERAL-SCLEROSIS/> - Cornell University scientists are working on developing Nrf2 pathway inducers (March 2009-2011 project)

University Of Wisconsin-Madison

2005-01-22

<http://www.sciencedaily.com/releases/2005/01/050121102753.htm>

Protective Protein May Hold Key To Halting Progression Of Neurological Diseases

Patients who suffer from neurological diseases such as Huntington's disease, Parkinson's, **Lou Gehrig's disease (ALS)** and Alzheimer's disease have dramatically different symptoms. An Alzheimer's patient, for instance, will lose memory and cognitive function, while an ALS sufferer will gradually lose motor control.

To doctors and researchers, however, how brain cells die in these diseases actually is quite similar. The diseases operate in similar ways and in particular are associated with common events within a cell that lead to cell death.

Within the cell, the mitochondria function as a sort of power plant, directing and regulating energy to fight off natural toxicities and keep cells functioning normally. As a person ages, the "power plant" begins to slow down, and the cell's natural defense mechanisms begin to fail. Eventually, cell toxicities overwhelm the defense mechanisms, causing neurons in the brain to begin dying. In certain individuals, this is the beginning of the long, slow path into neurodegenerative diseases such as Huntington's and Parkinson's.

That's where a substance called Nrf2 comes in.

"Nrf2 is a protein that, when you put it into cells, it brings up all the defense mechanisms simultaneously. Not only do you increase the cell's endogenous antioxidants, but you're also increasing the enzymes that remove toxicities from the cell," says Johnson. **"The protein also has a global effect - it doesn't just protect that which is inside, but also the normal cells in and around its environment."**

To test their hypothesis, Johnson and his research team transplanted cells with a high expression of Nrf2 into the brains of mice. After about five weeks, the team began exposing mice to toxins that kill neurons, the same neurons that are lost in Huntington's disease.

The result? **"The Nrf2 seems to completely protect the mice from toxicity,"** says Johnson.

Editor's Note: The original news release can be found [here](#).

Click to access [NFR2 pathway chart](#)

<http://www.pnas.org/content/103/3/768.full.pdf+html> - Activation of the Keap1/Nrf2 pathway for neuroprotection by electrophilic phase II inducers

[J Neurosci](#). 2008 Dec 10;28(50):13574-81. [Links](#)

[Nrf2 activation in astrocytes protects against neurodegeneration in mouse models of familial amyotrophic lateral sclerosis.](#)

Comment -True in sporadic & familial ALS → Activation of the transcription factor Nrf2 in astrocytes coordinates the upregulation of antioxidant defenses and confers protection to neighboring neurons.

Over-expression of Nrf2 in astrocytes significantly delayed onset and extended survival. These findings demonstrate that Nrf2 activation in astrocytes is a viable therapeutic target to prevent chronic neurodegeneration.

<http://www.jneurosci.org/cgi/content/abstract/28/50/13574>

J Neurosci Res. 2005 Feb 15;79(4):509-21. [Links](#)

[Acetylcarnitine induces heme oxygenase in rat astrocytes and protects against oxidative stress: involvement of the transcription factor Nrf2.](#)

Accordingly, we report here that treatment of astrocytes with acetyl-L-carnitine induces heme oxygenase-1 in a dose- and time-dependent manner and that this effect was associated with up-regulation of heat shock protein 60 as well as high expression of the redox-sensitive transcription factor **Nrf2** in the nuclear fraction of treated cells. In addition, we show that addition of acetyl-L-carnitine to astrocytes, prior to proinflammatory lipopolysaccharide- and interferon-gamma-induced nitrosative stress, prevents changes in mitochondrial respiratory chain complex activity, protein nitrosation and antioxidant status induced by inflammatory cytokine insult. Given the broad cytoprotective properties of the heat shock response, molecules inducing this defense mechanism appear to be possible candidates

for novel cytoprotective strategies. Particularly, manipulation of endogenous cellular defense mechanisms via acetyl-L-carnitine may represent an innovative approach to therapeutic intervention in diseases causing tissue damage, such as neurodegeneration.

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<http://sageke.sciencemag.org/cgi/content/abstract/2002/35/nw124> - role of ceramide in motor neuron die-off

[Use of basic amino acids derivatives for lowering ceramide levels](#)

“It has in fact been found that administration of high doses of basic aminoacids, low molecular weight basic compounds or acyl derivatives thereof and pharmacologically acceptable salts thereof **reduces ceramide levels** and such compounds can thus be used for the treatment of diseases characterized by high levels of ceramide.

In particular, it has been found that basic aminoacids such as arginine, lysine, histidine, ornithine, and **carnitine or acyl derivatives** thereof and pharmacologically acceptable salts thereof can be used for the treatment of diseases characterized by high levels of ceramide.”

One difficulty with respect to activating Nfr2 synthesis and accumulation in neurons and astrocytes and such lies in the fact KEAP1 <http://en.wikipedia.org/wiki/KEAP1> suppresses it. What's needed is a KEAP1 inhibitor. I found one naturally occurring one (The rest are experimental drugs that are mainly in the animal study phase right now): **Ginkgo Biloba Extract** (GBE)

Here's an abstract of a study that disclosed GBE as a KLEAP1 inhibitor (Again, the trick to protect motor neurons is to raise Nfr2 levels, but this build-up ---using the compounds and dietary agents sent in my last e-mail -- is undone by KEAP1. Now, by pairing up GBE with the Nrf2 synthesis promoting compounds and such -- Nfr2 will accumulate in neurons & etc. and thus confer neuroprotection against glutamate spawned excitotoxicity)

[Extract of *Ginkgo biloba* induces phase 2 genes through Keap1-Nrf2-ARE signaling pathway](#)

Abstract

The standard extract of *Ginkgo biloba* (EGb) has been demonstrated to possess remarkable antioxidant activity in both cell lines and animals. However, the molecular mechanism underlying this effect is not fully understood.

....., we found that Keap1 content was inhibited by EGb and then more Nrf2 would be released to translocate into nucleus. Thus, EGb was testified for the first time to induce the phase 2 genes through the Keap1-Nrf2-ARE signaling pathway, which is (or part of) the antioxidant mechanism of EGb.

METHYLENE BLUE NOSE DROPS

Very dilute methylene blue may slow ALS progression:

<http://www.sciencedaily.com/releases/2008/08/080818101335.htm>

A newly published (June 25 2009) Japanese study underscores the potential of dilute methylene blue in ALS (plus Alzheimer's!) – see abstract below. This study makes reference to TDP-43, which readers can delve into by perusing this article: [The Dana Foundation - ALS Researchers Focus on Mystery Protein TDP-43](#)

Rather than ingest a dilute MB solution and wind up with much of it being dumped in one's urine, there is a way to better insure delivery to the Central Nervous System (CNS): Namely, nose drops.

To create a dilute MB nasal spray: Put 1 drop of MB in a gallon of distilled water. Shake well, then take a teaspoon of that and add it to a 6 oz. bottle of saline nasal spray (These are sold as generics at all pharmacies). Store the gallon you made up in a fridge. Then spray 1 time into each nostril three times daily (morning, noon, evening) and before retiring at night. This should provide a-diluted but effective dose that will enter the CNS quickly through the nasal mucosa.

Other Measures to Mull Over with A Physician

The items below are part of: [Experimental regimen targeting the ependyma slows disease progression in four patients with amyotrophic lateral sclerosis](#)

[CoQ10](#) (Ubiquinol) – Suggested use: 200 mg every 2 hours (1200 mgs total daily). Rationale for use – CoQ10 extended the survival time of transgenic murine models of ALS versus a control group in a Massachusetts General Hospital lab study [5]. Dose: 200 mgs. every 2 hours during the day (1200 mgs daily). CoQ10 is well tolerated and produced no adverse effects in dose of 3g/daily over an eight month period [6].

[Noni capsules](#) – Suggested use: 1 capsule every 2 hours or drink the juice liberally. Rationale for use: Contains a potent quinone reductase inducer [7]. QR reduces glutamate toxicity in cells.

[Turmeric](#) (standardized to 15% bisdemethoxycurcumin) – 500 mgs. Suggested use: Every 2 hours during waking hours. Rationale for use: Quinone reductase inducer in astrocytes (Lowers glutamate)

[Lithium carbonate](#) – Dosage: High dose lithium carbonate has bombed in various studies. It may well be that low dose lithium orotate works best. Say 50 mgs or so daily.

[IV Glutathione](#): Rationale – Glutathione is depleted in many ALS patients or at risk of becoming so over time. The intravenous (IV) dose is determined by each patient's physician.

Amyotrophic Lateral Sclerosis: Stem Cell Healing & Amyotrophic Lateral Sclerosis (ALS)*

By

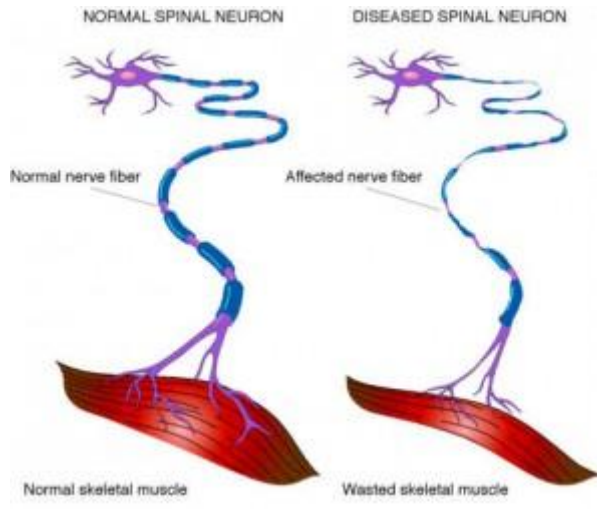
[David Steenblock, M.S., D.O.](#)

WARNING – Patients who have ALS (Lou Gehrig’s disease) should read this before having stem cells anywhere!

If you or a loved one are misfortunate enough to get ALS (or any other devastating neurological disease) you are probably considering the use of stem cells (and rightfully so). Unfortunately, no doctor, or person for that matter, has all of the answers yet, and even stem cells, in my experience, are only one of many treatments that can and should be used to slow the progression of and remediate ALS. Indeed, prior to getting stem cells, there are issues to be addressed that have to do with why a person has ALS in the first place. Doesn’t this make sense? After all, you cannot douse a fire while simultaneously fanning the flames.

What is needed in ALS in my professional opinion is body cleansing as well as more natural methods to treat the disease, something that sets the stage for a better response to stem cell-based therapy. One very insightful and compelling collection of natural body cleansing methods is contained in books penned by an ALS patient of mine, [Eric Edney](#) (who was diagnosed with ALS over twenty years ago. Typically, ALS patients live two to five years after being diagnosed.) Two of his books, “[ERIC IS WINNING !! Beating a Terminal Illness with Nutrition, Avoiding Toxins and Common Sense](#)” and “[SURVIVING WITHOUT YOUR MD: DO PRESCRIPTION DRUGS EVER CURE?](#)” are both available for purchase online. Another great book about the reasons behind diseases like ALS, as well as how to cleanse your body, is “[The Truth About Migraines To Multiple Sclerosis And More: What Your Doctor Isn’t Telling You But Science Has Proven](#)” by Barbara J. Tancredi BSc, CN available on Amazon. These books should be read immediately and carefully in my opinion and experience. Why? Because I have found no clinic, hospital, clinical trial or doctor who is currently addressing the fundamental problems that are contributing to the progression of ALS prior to the administration of stem cells. I admit that at various times I have recommended the immediate use of stem cells when a patient was losing ground quickly. However, in most cases ALS sufferers do have time to obtain a careful evaluation, and to have their body cleansed of the poisons that are either causing or contributing to this terrible condition.

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Amyotrophic Lateral Sclerosis

As is true of all diseases, variations in lifestyle, environment, and genetics influence disease progression. As such all ALS patients need to be thoroughly evaluated. There are at least 100 known players with respect to ALS including many different types of mutations of the SOD (superoxide dismutase) enzyme. One common factor in ALS and similar diseases is the contribution of free radicals which lead to the production of superoxide anion which, in turn, reacts with nitric oxide producing a very toxic substance called peroxynitrite. There are a multitude of reasons why one person produces excess nitric oxide, and these include the presence of infections, consumption of and/or inability to handle glutamate and consuming too much arginine, an amino acid which is extraordinarily high in nuts, seeds and chocolate. Peroxynitrite damages and even destroys the supporting cells of the motor neurons as well as the spinal cord motor neurons themselves. By the way, one of the best natural substances to help stop this destruction immediately is to take a powerful flavonoid complex called **LutiMax™** that can be ordered from my office or directly from www.luteolin.com.

So where to start? The tests that I recommend for patients with ALS will help determine what needs to be cleansed from the body, especially prior to considering stem cell therapy. These tests include: 1) DMPS Challenge tests 2) CDSA and parasite test 3) urine organic acid test 4) immunological tests and 5) hormonal tests to determine which hormones and growth factors are missing. STEM CELL-BASED TREATMENTS AVAILABLE in either my Mission Viejo, CA clinic or at a facility I consult for in Tijuana, Mexico:

1) Simple bone marrow transplant. This is the least expensive and also least effective route (\$4K USD.)

2) [Neupogen®](#) pretreatments and then bone marrow transplantation which is 5 times more effective than #1 above in my experience and estimation (\$7K USD.)

3) Bone marrow stem cell isolation, culture and injection into cerebrospinal fluid (50-75 million cells are given per CSF injection.) This is by far more effective than #1 or #2 above and runs \$12K USD. This is done in Mexico.

4) Continued culture of bone marrow stem cells (see #3) and then repeat cerebrospinal fluid stem cell administration of 50-70 million cells. This may be done up to 6 times after one bone marrow collection with the cost per lumbar puncture (CSF administration) running \$3K USD. Example: If you want to come every month for a CSF injection of stem cells the cost for 6 months of therapy this would be \$12K USD for the first and \$3K USD for each subsequent injection X 5= \$15K USD. Total for the original bone marrow procedure, cultivation of cells and delivery of 50 million cells into the CSF is \$12K USD plus 5 more CSF injections at \$15K USD would be \$25K USD for the entire 6 month course of stem cell therapy. The total number of stem cells you would be receiving during this time would be over 300 million stem cells (again, directly into your CSF) on a monthly basis. If you decide to do it only once than the cost would be \$12K USD. For another \$3K USD you can come back and have the same treatment that you just paid \$12K USD for. In other words, it is much cheaper and better for you to commit to 6 months of therapy than doing only one CSF injection since you will get almost 5 times more for your money.

ALS RESEARCH being done at Steenblock Research Institute, Inc., my namesake nonprofit research institution and R & D (Research & Development) laboratory located in southern California: Recent preliminary results from some very sophisticated blood tests are showing deficiencies of various interferons and interleukins in the serum of patients with ALS. These changes are now being compared with the same tests done on the patient's cerebrospinal fluid in hopes of finding more clues to the cause and treatment of this terrible condition.

Another area of exciting ongoing research in our lab is taking ALS patients' bone marrow stem cells, culturing them and then testing them by growing them in the presence of approximately 800 natural substances found in the body or nature to see which are inhibitory and which are growth-promoting to the stem cells. This is being done because research is proving that there are defects in the functionality of the bone marrow stem cells of ALS patients. The goal is to discern what is helpful to your stem cells which tells us what to prescribe in terms of supplements rich in these helpful agents. This approach should give your stem cells the greatest opportunity to grow and, in turn, repair your diseased nervous tissue. Also, after we determine what factors are best for your stem cells, we can better grow them in tissue culture. This provides more healthy stem cells that can then be given back to you directly into your cerebrospinal fluid (Mexico) which allows the stem cells to get into contact immediately with the diseased spinal cord tissues.

Finally, my crack medical team and I are beginning to try minimally manipulated stem cell-rich fat tissue injected into the CSF in hopes of getting better results than from the stem cells derived from bone

marrow used in Mexico. The only problem so far with this method is obtaining enough fat from the ALS patients who have little fat present. Even with little fat, we can process the cells but it may take longer than usual to get the required number for a good treatment. We are not the only doctors and researchers who have found that stem cells can aid in treating ALS. As you may know, the reason this is not front page news is because there is little to zero acknowledgement among mainstream researchers and doctors of the many toxins that trigger and contribute to the disease, much less the need to properly test and do a thorough body detoxification prior to stem cell therapy.

To learn more call toll free **1-800-300-1063** or send me (Dr. Steenblock) a message using his on-line contact form which can be readily accessed by [clicking this link](#). In addition, my www.StemCellMD.org group works with clinics in Mexico that have been successfully doing adult (non-embryonic) stem cell treatments for ALS and other neurodegenerative diseases for many years now. In Mexico, umbilical cord stem cells can be mixed with your fat and bone marrow stem cells for the ultimate combination stem cell treatment. The cost for umbilical cord stem cells per vial is \$6K USD (1.5-1.8 million CD34+CD133+ pure stem cells.)

You can get more information on this program by calling **1-800-288-7016** or e-mailing me at info@stemcell.md. Additional information on umbilical cord stem cells is available at www.stemcelltherapies.org or by purchasing the book "[Umbilical Cord Stem Cell Therapy: The Gift of Healing From Healthy Newborns](#)" by David Steenblock, M.S., D.O. and Anthony Payne, Ph.D. from my office at **1-800-300-1063**.

For patients who cannot afford any of these treatments I would recommend you go on my ALS diet http://stemcell.md/diets/als_diet.pdf and use my all natural stem cell support supplement **Stemgevity™**. This product contains 4 mg of lithium per capsule which has demonstrated certain positive effects in patients with ALS in various studies. Begin with one or two per day and gradually build up to the level at which you feel the best and then stay on that dose. If you feel that your disease is progressing or you are experiencing any side effects then stop taking these pills for one to two weeks and start over and bring your dose back to the number per day where you were feeling the best. Taking them at night is the usual method since they can make you feel tired. **Stemgevity™** can be ordered from my office toll free at **1-800-300-1063**. For more information, visit www.stemgevity.com.

[ALS: You have to cover all the bases! \(Blog entry on DavidSteenblock.com\)](#)