

**Dr. Anthony G. Payne**  
E-mail: [Biotheoretician@gmail.com](mailto:Biotheoretician@gmail.com)

## **ALS Support Regimen**

Methylcobalamin, 20 mgs., 2-3 x daily (Protects against glutamate toxicity i.e, excitotoxic damage to motor neurons)

SAMe, 600-1800 mgs. daily (Take with a B multiple)  
(Ditto)

Coenzyme Q10, 300-450 mgs. 3 x daily (with or after meals)

Vinpocetine, up to 30 mgs. daily (Protects against excessive Calcium release intracellularly)

Octacosanol, Viobin 5 mgs. 3 x daily

Use a branched chain amino acid supplement (But try to avoid taking sulfur-rich aminos such as cysteine or taurine, unless you have familial amyotrophic lateral sclerosis. **If you do have familial ALS, you might want to discuss use of N-acetylcysteine with your primary care physician. See the first abstract posted below for the rationale**).

Krill Oil, 1 softgel 3 x daily with or after meals (Combats free radicals that compromise motor nerve function. Has 300 times the antioxidant power of vitamin E -- ORAC test)

[Remilyn](#) (Nerve growth factors. Use 1 bottle daily for 1-2 weeks, then follow manufacturers suggested dosage regimen)  
*Additional information below.*

**Diet: Avoid sulfur and selenium-rich rich foods like eggs, garlic and onions.** ALS patients have elevated sulfur and selenium levels, both of which contribute to this insidious condition. Calcium is a sulfur antagonist, so calcium-rich foods should be part & parcel of the ALS patient's daily diet (Green vegetables especially. Avoid all dairy products, grains and cereals as these generate compounds that can fuel inflammation).

The Paleodiet is high in calcium and potassium, both of which help lower sulfur and selenium levels.

<http://14ushop.com/wizard/living-longer.html>

**NOTE:** ALS patients appear to defectively process iron. Lactotransferrin (see the abstract below), which is generated to deal with excess iron, is elevated in ALS. This suggests that iron and aluminum levels should be assessed. If they are elevated, a physician can prescribe desferrioxamine -- a drug that chelates these metals out of the human body (Use must be monitored and supervised by an MD or DO).

Also, a subset of ALS patients appear to have defects in terms of their synthesis of SOD (Superoxide Dismutase) to protect neurons from cell damaging free radicals. I am in contact with a firm in Oregon that has a water soluble form of SOD suitable for consumption by consumers and patients. This product is absorbed and does get into various bodily organs and tissues. The website for this firm is [www.advantig.net](http://www.advantig.net). The President of this company, Dr. Jerry Schlessner, is a licensed naturopathic physician who has 25 years experience in R & D, and has been published in numerous peer reviewed medical and scientific journals including the LANCET. This tends to underscore the reliability of what he does and offers.

There are nerve growth factors that appear to retard the progress of ALS, at least in animal models (See abstract below). There is a product on the market OTC that is rich in nerve growth factors -- derived from porcine sources -- called Remilyn (The various nerve growth factors in pigs are almost identical to their human counterpart at the molecular level. As such, the porcine stuff does have a physiological effect. Many MS patients tell me that porcine and bovine-derived nerve growth factors do make a difference in their symptomology).

[Remilyn](#) is manufactured in a lab up in Canada. You can get it through Jim Haverlock online by going to his website: [www.14ushop.com](http://www.14ushop.com) Jim has progressive MS and has benefited greatly from the use of Remilyn (He had stem cell therapy also, which appears to have greatly ameliorated his condition).

Dr. Anthony G. Payne

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Neurobiol Dis. 2003 Aug;13(3):213-21.

[Related Articles, Links](#)

**Mitochondrial dysfunction due to mutant copper/zinc superoxide dismutase associated with amyotrophic lateral sclerosis is reversed by N-acetylcysteine.**

**Beretta S, Sala G, Mattavelli L, Ceresa C, Casciati A, Ferri A, Carri MT, Ferrarese C.**

Department of Neuroscience and Biomedical Technologies,  
University of Milano-Bicocca, San Gerardo Hospital, via  
Donizetti, 106, 20052, Monza (MI), Italy.

We report that the expression of mutant G93A copper/zinc superoxide dismutase (SOD1), associated with familial amyotrophic lateral sclerosis, specifically causes a decrease in MTT reduction rate and ATP levels and an increase in both cytosolic and mitochondrial reactive oxygen species (ROS)

production in human neuroblastoma SH-SY5Y cells compared to cells overexpressing wild-type SOD1 and untransfected cells. Exposure to N-acetylcysteine lowers ROS production and returns mitochondrial functional assays to control levels. No large aggregates of human SOD1 are detectable under basal growth conditions in any of the investigated cell lines. After proteasome activity inhibition, SOD1 aggregates can be detected exclusively in G93A-SOD1 cells, even though they do not per se enhance cell death compared to control cell lines. Our findings indicate that mitochondrial homeostasis is affected by mutant SOD1-generated ROS independently from the formation of aggregates and that this alteration is reversed by antioxidants.

PMID: 12901835 [PubMed - indexed for MEDLINE]

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Science. 2003 Aug 8;301(5634):839-42.

### **Retrograde viral delivery of IGF-1 prolongs survival in a mouse ALS model.**

**Kaspar BK, Llado J, Sherkat N, Rothstein JD, Gage FH.**

Laboratory of Genetics, The Salk Institute for Biological Studies, La Jolla, CA 92037, USA.

Amyotrophic lateral sclerosis (ALS) is a progressive, lethal neuromuscular disease that is associated with the degeneration of spinal and brainstem motor neurons, leading to atrophy of limb, axial, and respiratory muscles. The cause of ALS is unknown, and there is no effective therapy. Neurotrophic factors are candidates for therapeutic evaluation in ALS. Although chronic delivery of molecules to the central nervous system has proven difficult, we recently discovered that adeno-associated virus can be retrogradely transported efficiently from muscle to motor neurons of the spinal cord. We report that insulin-like growth factor 1 prolongs life and delays disease progression, even when delivered at the time of overt disease symptoms.

PMID: 12907804 [PubMed - indexed for MEDLINE]

You might also want to check out this website:

[www.newhopediscoveries.com](http://www.newhopediscoveries.com) (Lorene O'Bryne, Director)

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Brain Res. 1994 Jul 4;650(1):20-31.

**The iron-binding protein lactotransferrin is present in pathologic lesions in a variety of neurodegenerative disorders: a comparative immunohistochemical analysis.**

**Leveugle B, Spik G, Perl DP, Bouras C, Fillit HM, Hof PR.**

Department of Geriatrics and Adult Development, Mount Sinai School of Medicine, New York, NY 10029.

Lactotransferrin is a glycoprotein that specifically binds and transports iron. This protein is also believed to transport other metals such as aluminum. Several lines of evidence indicate that iron and aluminum are involved in the pathogenesis of many dementing diseases. In this context, the analysis of the iron-binding protein distribution in the brains of patients affected by neurodegenerative disorders is of particular interest. In the present study, the distribution of lactotransferrin was analyzed by immunohistochemistry in the cerebral cortex from patients presenting with Alzheimer's disease, Down syndrome, amyotrophic lateral sclerosis/parkinsonism-dementia complex of Guam, sporadic amyotrophic lateral sclerosis, or Pick's disease. The results show that lactotransferrin accumulates in the characteristic lesions of the different pathologic conditions investigated. For instance, in Alzheimer's disease and Guamanian cases, a subpopulation of neurofibrillary tangles was intensely labeled in the hippocampal formation and inferior temporal cortex. Senile plaques and Pick bodies were also consistently labeled. These staining patterns were comparable to those obtained with antibodies to the microtubule-associated protein tau and the amyloid beta A4 protein, although generally fewer neurofibrillary tangles were positive for lactotransferrin than for tau protein. Neuronal cytoplasmic staining with lactotransferrin antibodies, was observed in a subpopulation of pyramidal neurons in normal aging, and was more pronounced in Alzheimer's disease, Guamanian cases, Pick's disease, and particularly in Down syndrome. Lactotransferrin was also

strongly associated with Betz cells and other motoneurons in the primary motor cortex of control, Alzheimer's disease, Down syndrome, Guamanian and Pick's disease cases. These same lactotransferrin-immunoreactive motoneurons were severely affected in the cases with amyotrophic lateral sclerosis. It is possible that in these neurodegenerative disorders affected neurons either take up or synthesize lactotransferrin to an abnormally elevated rate. An excessive accumulation of lactotransferrin, as well as transported iron and aluminum, may lead to a cytotoxic effect resulting in the formation of intracellular lesions and neuronal death.

PMID: 7953673

Other compounds of possible merit in ameliorating ALS can be found in [\*\*Dr. Payne's MS Support Regimen\*\*](#)

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