

# Motor Neuron Disease (ALS) - Nutritional Support Protocol Suggested

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- Vitamin B3 (Niacin) (100-300mg)
- Vitamin B6 (50mg twice daily)
- Vitamin B12 (2,000mcg daily)
- Vitamin C (3,000mg daily)
- Alpha Lipoic Acid (250mg twice daily)
- N-Acetyl-Cysteine (500mg twice daily)
- Ginkgo Biloba (250mg daily)
- Vitamin D3 (2,000 IU)
- Co-enzyme Q10 (100-200mg twice daily)
- Phosphatidylserine (100mg per day)
- Acetyl-L-Carnitine (500mg twice daily)
- Creatine (3,000-6,000mg daily)
- Liver detoxification
- Human Growth Hormone (on prescription only)
- Neurodetoxification with intravenous phosphatidylcholine followed by leucovorin, followed by glutathione (given by an experienced doctor).

**Note: Suggested protocol for supplemental support only**

**M**otor Neuron Disease, also known as ALS, is a progressive, degenerative, neuromuscular disease that progresses quickly and destroys nerve cells in the brain and spinal cord. Once these nerve cells (neurons) are destroyed they can no longer send messages to the muscles and control is lost over voluntary muscle movement.

The exact cause of motor neuron cell death is still unknown but the condition is most likely multifactorial and aggravated by a number of underlying health irregularities. Scientists are currently investigating various theories, including free radical damage from oxidative stress, calcium dysregulation, glutamate toxicity, mitochondrial dysfunction and even viral infections as possible causes of MND. It is believed that these factors may work synergistically to cause and advance the disease. Heavy metals, inflammation and environmental agents are also possible factors to consider. <sup>1</sup>

Exposure to neurotoxins occurs every day and contributes to nerve cell and brain damage. Neurotoxins increase

free radical production and promote inflammation. This in turn leads to brain degeneration and accelerated brain aging. The body's attempt to eliminate these toxins is known as the detoxification system, with the liver being primarily responsible for cleaning up the toxic chemicals in the bloodstream. One of the most important antioxidants for the brain is glutathione, and is found in high concentrations in the liver as well. Glutathione binds to toxins and helps excrete them by making them water soluble so that they can be excreted by the kidneys into the urine. Glutathione functions also include the maintenance of protein structure and function, regulation of protein synthesis and degradation and stabilisation of immune function. Glutathione is also the storage and transport form of cysteine, of which the functions include leukotriene and prostaglandin metabolism, microtubule process regulation and bile formation regulation.

Modern day exposure to toxins may exceed the body's ability to detoxify and prevention of toxin exposure needs to be seriously entertained. The body is constantly struggling with these various neurotoxins as found in food such as the mercury in fish and the pesticides sprayed on vegetables and fruits. Excitotoxins such as MSG, aspartame and other food additives add to toxicity. Personal care products contain toxins such as the aluminium in deodorants, shampoo and skincare and the lead in some lipsticks. Home exposure to lead in walls, plumbing, paints and pipes also pose a problem. Modern society is also inundated with radio waves or electronic toxins from excessive television, computer and cell phone exposure. An optimal regimen of nutrients and supplements for individuals with MND may be able to delay the effects of these contributing factors and slow the progression of the disease, therefore reducing symptoms. Conventional medicine, which as yet has been unsuccessful in treating MND, also attempts to lessen symptoms by slowing progression. Currently however,

one pharmaceutical drug approved for MND patients has been shown to extend life span by only two months.<sup>2</sup>

A comprehensive integrative approach should be considered to address the underlying defects of the disease. There is a central role of defective mitochondrial energy production, and the resulting increased levels of free radicals, in the pathogenesis of various neurodegenerative diseases.<sup>3</sup> Defects in energy metabolism may therefore contribute to both excitotoxicity and oxidative damage. Due to this impaired antioxidant activity in MND patients, treatment reducing oxidative stress may slow the course of the disease<sup>4</sup> and should be the first port of call.

A recent study in humans indicated that vitamin E may significantly delay symptom onset and slow disease progression in MND because of its antioxidant properties.<sup>5</sup> Vitamin E helps protect cell membranes against lipid peroxidation.<sup>6</sup> Selenium supplementation may increase the amount of vitamin E in the blood and increase activity of glutathione among MND patients. Ginkgo biloba also has antioxidant properties<sup>7</sup> and may promote healthy mitochondrial function.<sup>8</sup> During an in vitro study, Ginkgo biloba was found to protect against glutamate-induced excitotoxicity.<sup>9</sup>

Zinc is involved in many physiological processes in the body. Changes in zinc metabolism that lead to neurodegeneration can occur during times of oxidative stress.<sup>10</sup> Zinc supplementation has potential benefit due to its integral role in the function of SOD. SOD is an antioxidant enzyme that reduces oxidative stress and can play a key role in assisting MND. Mutations in SOD have decreased ability to bind to zinc and may contribute to MND.<sup>11</sup> It is important to note that large doses of zinc inhibit copper absorption so therefore the dosage in supplementing zinc and the synergistic interaction of copper and zinc needs to be considered in terms of predicting the toxic or neuroprotective effects.<sup>12</sup>

There is promise in using co-enzyme Q10 for treating MND. Co-enzyme Q10 is a critical component of the electron transport chain for proper mitochondrial function and acts as an antioxidant.<sup>13</sup> A study found that oral supplementation significantly reduces weight loss, delays motor deficits, and extends life span in MND.<sup>14</sup>

Acetyl-l-carnitine has been shown to inhibit mitochondrial damage and apoptosis.<sup>15</sup> It could also have neurotrophic activity and increase glutathione concentrations.<sup>16</sup> Early oral administration of acetyl-l-carnitine may significantly delay symptom onset, prolong motor function and extend survival rates. The effects of acetyl-l-carnitine are increased when administered in conjunction with alpha-lipoic acid.<sup>17</sup>

Alpha-lipoic acid, also an antioxidant may increase intracellular levels of glutathione<sup>18</sup> and chelate metals such as iron and copper.<sup>19</sup> Furthermore, alpha-lipoic acid has been shown to protect cells against glutamate-induced excitotoxicity.<sup>20</sup> Alpha-lipoic acid may therefore significantly delay impaired motor performance as well as increase survival.<sup>21</sup>

Dietary supplementation with amino acids may have some beneficial effects on the course of the disease.<sup>22</sup> The use of creatine has also been encouraging. Creatine aids in the formation of ATP, which is the primary source of cellular energy. In multiple studies, creatine was shown to provide protective mechanisms against neurodegenerative diseases by helping to stabilise mitochondrial membranes and mitochondrial energy-transfer complexes.<sup>23</sup> Creatine may also reduce oxidative stress, increase glutamate uptake and improve motor performance.<sup>24</sup> In addition, a small preliminary study found that creatine supplementation helps reduce the loss of muscle strength in MND patients.<sup>25</sup> Ultra high doses of vitamin B12 may also improve or slow muscle wasting, which is common during the later stages of the

disease.<sup>26</sup> The observation that patients with MND may have elevated plasma homocysteine levels has encouraged the use of the B vitamins, particularly folic acid and vitamin B12.

More recent data has suggested that curcumin (an extract from turmeric) may help improve calcium status in muscle tissue and reduce inflammatory processes.<sup>27,28</sup> While human studies are needed to confirm these results, preclinical evidence suggests that curcumin could be useful in MND. Vinpocetine and Resveratrol may also inhibit the flow of calcium into cells, which is associated with glutamate-induced cell toxicity.<sup>29,37</sup> This is similar to the mechanism of action of riluzole, the only FDA-approved drug used to treat MND.

### **Due to the impaired antioxidant activity in MND patients, treatment reducing oxidative stress may slow the course of the disease<sup>4</sup> and should be the first port of call.**

Evidence suggests that MND associated nerve cell death is partly due to low levels of the antioxidant glutathione, which protects cells from toxins and free radicals.<sup>30</sup> Lower glutathione peroxidase activity has been shown in plasma and cerebrospinal fluid of MND patients.<sup>31</sup> Glutathione peroxidase, catalase, and superoxide dismutase (SOD) are all antioxidants synthesised by the body that counteract reactive oxygen species (ROS) damage. Toxicity resulting from mutated SOD is implicated in the pathophysiology in familial MND. Increasing glutathione levels could therefore help prevent free radical damage to cells.<sup>32</sup> The glutathione precursor N-acetyl cysteine (NAC) boosts blood levels of glutathione and oral administration decreases motor neuron loss, improves muscle mass, and increases survival time and motor performance.<sup>33</sup>

Epigallocatechin gallate (EGCG) is a major catechin found in green tea. It displays antioxidant, anti-inflammatory<sup>34</sup> and mild metal chelating properties.<sup>35</sup> EGCG also significantly delays symptom onset and prolongs life span. Pycnogenol also has antioxidant properties,<sup>36</sup> as well as protective effects against glutamate excitotoxicity.<sup>9</sup> Procyanidins, which can be found in grape seeds, cranberries, blueberries, almonds, and peanuts, demonstrate potent antioxidant properties.<sup>36</sup> Pycnogenol is a common complementary therapy option among MND patients.<sup>6</sup>

Dehydroepiandrosterone (DHEA) is a naturally occurring steroid hormone and in several in vitro studies, DHEA has been shown to protect against glutamate-induced toxicity<sup>38</sup> although only a few studies have examined DHEA in MND patients.

The benefits of aggressive nutritional support in affecting the course of disease and survival are well documented with glutathione being the major defence against toxic oxygen products. Intravenous phosphatidylcholine, leucovorin and glutathione IV push over 3-5 minutes relieves the body's burden of metal toxicity and neurotoxins.

**Suggested dose:** 1400mg glutathione in 9cc sterile water, initial push 1800mg-2400mg twice weekly for 20 weeks

**Paediatric dose:** 200mg-800mg 2 x weekly for 20 weeks

Please consult Dr Golding or email at [craigeg@mweb.co.za](mailto:craigeg@mweb.co.za) for these protocols.

Nutritional supplements that prevent free radical damage, stabilise mitochondrial membranes, or stimulate electron transport chain complexes may all be of tremendous value in MND patients.

**References on request.**