I found below an interesting page from an Austrian Doctor, he claims that increased serotonin levels increase survival chances quite dramatically. As far as I have been able to verify this seems to be correct. Pass it on! (Translation by google) from German.

Amyotrophic lateral sclerosis - ALS

Last modified on 29 July 2013 | created on: 25- Jan- 2011 by: Mr Helir

SEROTONIN levels DETERMINES THE SURVIVAL TIME! Antidepressants could shorten time. Causes and poss. Therapies are illustrated with studies.

Due to a colleague with ALS, I have about 1200 studies on "new possibilities in the treatment of amyotrophic lateral sclerosis researched over Christmas holidays 2010/2011 in the PubMed and written together, the results here. Every few months I go through the newly published studies.

Serotonin determines the survival time significantly

Serotonin Survival ALS

At my screening to now 769 studies on amyotrophic lateral sclerosis - I stumbled across an EXTRAORDINARY result. With simple dietary supplement can be the survival time in amyotrophic lateral sclerosis extend to the multiple - and - according to this data results in the following dramatic fact:

SSRI antidepressants shorten the survival time!

Plasma serotonin levels decides whether 30 or 150 months survival time in ALS.

Summary of all studies in a short form - Overview

Therapies my neurological colleagues show that little knowledge of is penetrated from the ivory tower of science to us "exporting doctors" until now, because we are currently using almost only riluzole and antidepressants.

Please read the article carefully, if they are an interested party, this article can influence with - their disease course.

Reference

Because of this website I get requests from all over Europe. However, I am neither a neurologist nor ALS specialist, but a "simple practical doctor" - albeit with a scientific background and extensive additional training (see training Dr. Retzek)

AS A CAUSE

ALS is a scary disease because it affects younger patients and is currently incurable. Intensive research is devoted to the causes of disease and many relationships were revealed in recent years.

A detoxification enzyme - the SOD1 - is disturbed. cause:

Genetically, Overloading of the detoxification

Various toxins, Overload of stim,ulation of nerves too much glutamine in the nerve by aspartame ("Light-drinks")

Heavy metals

Injuries, This process seems to be un - reversible, because by lack of the enzyme is a newly - formed SOD1 also destroyed by the deficient detoxified peroxide in the nerve cell mitochondria.

This process is "contagious" and is slowly spreading throughout the "motor" nerve cell tree and leads to progressive paralysis. Apparently the broken SOD1 acts as a prion (a protein which destroys other proteins) and is transmitted from nerve to nerve!

SOD detoxifies free radicals - superoxide - the nerve. These accumulate now and destroy the nerve.

Inflammatory substance PROSTAGLANDIN E2 is upregulated.

Entirely new data (Feb. 2012) in the ALS mouse model PGE2 - the main Entzüdungsmediator in the body - is just the beginning of the AS - nachweissbar symptoms increased in the affected neurons. Ev . PGE2 acts in neuron -inflammatory and neurodegenerative.

PGE2 is the product of omega -6 fatty acids (sunflower oil, canola , corn , soybean , safflower, oils ...) - the opponent is PGE3 produced from linseed oil and fish oil. PGE2 production can be inhibited by anti-inflammatory drugs (ibuprofen, aspirin, diclofenac .....).

Therapeutic strategies that could result from this

Currently very little "useful" in the pipeline . Most strategies are "ANTI- OXIDATION" high doses , because hereby the damage can be stopped by the enriched peroxide :

Vitamin E, alpha -lipoic acid , vitamin C , melatonin, Q10, Omega 3 , curcumin , resveratrol - the usual antioxidant cocktail that needs to be given always TOGETHER , as a substance itself never sufficient because the antioxidants form a network.

Lithium (Update 2013: disappointed in the studies -> see my page LITHIUM)

Pioglitazone - a diabetes - induced means "peroxisomes " which then propagated reduce peroxide (Update 2013: disappointed) and especially to stretch as the most promising therapeutic approach for the time: SEROTONIN

Serotonin as the main treatment strategy: Summary

Survival time of ALS patients is directly related with the serotonin concentration in the serum together (Study 2010) , Serotonin in platelets and is transported , after coagulation (= serum production) are measured there .

Platelet serotonin is clearly reduced over "anti -depressants " .

This will antidepressants to disease accelerators of this disorder and must be avoided.

5HTP serotonin precursor is optimal therapeutic strategy in ALS .

My esteemed colleague neurological exist in each patient on antidepressants , although I have found this in the literature NO disease -related indication, that is, in German: there is no disease-related reason , a possibly occurring depression is also improved reliably with 5HTP .

Read my article detailing to serotonin, there also the research data on the side effects of antidepressants , etc.

Ultimately: MEASURE IT EASY .

The serotonin value is a simple uncomplicated blood test

(Note: following blood clotting must be centrifuged, only the serotonin - value is stable shipping).

To avoid antidepressants necessarily, you may please simply - very practical - measure the serum level of serotonin with antidepressants. I have not found anyone over 30, usually much lower. More on the effects of serotonin deficiency read them on my side "serotonin". The dietary supplement 5HTP is an ingenious way to raise the serotonin levels to over 200 , according to the study shown above, you should raise the Serotoinin levels to maximum values (400).

Attention: "curdle " necessarily to measure blood, then immediately centrifuge , the serum from the low-level disconnect (gel tube) . Otherwise, are the serotonin content " Häusl values " .

Updated May 2012

Creatine and Q10 could be great therapy help: in the mouse model, a 40% extension of life time application , a 50 % improvement in the scores after 1 year in PD patients in clinical study ! See my page: http://www.homeopathy.at/als-updat/

Acetylcysteine

N -acetyl- cysteine can intercept the highly active oxidative stress in neurons and has been shown both in Huntington's disease as well as in ALS animal model, significant functional and life - Lengthening effects.

Now to the actual item

ALS is truly a frightening disease. From the best of health out you get progressive paralysis to the final state.

There are no established dzt. ALS therapy, also not really clear knowledge of causes , however - it's fascinating how intense these "orphan disease" is researched (monthly about 100 studies) - now many clues. On this page I would like everything that falls into my hands about ALS document and link to it. Initial I have about 900 studies (all from 2010-2011) screened and summarize attempts here and network

Warning: everything I write here is supported by numerous studies and therefore absolutely Evidence Based! If you are reading this and cannot find a link, I'm still not to the extent reinzulinken the study because I have picked out about 100 trials for this product. Check it in a few days again or write me an email.

Cause ALS

Here there is actually a variety of studies over the past months. Modern genetic engineering has given us tools into the hand with promptly and fairly comprehensive spezfische questions can be answered. A really clear, transparent image has not yet arisen, but a lot of circumstantial evidence. Helpful is the presence of some "mouse models" of Amyotrophic lateral sclerosis , animals that get just like the people the disease and where basics such therapy studies can be performed.

ALS belongs to a group of "Neurodegenerative Diseases"

Many mechanisms of ALS shares this disease with other neurodegenerative diseases such as Alzheimer's , Huntington's , Parkinson's disease and rarer diseases such as frontal dementia , etc. , in ALS degenerate MOVEMENT NERVE (motor neurons) .

What are the main causes

1) SOD1 mutation

Identified a major cause is an error of a certain protection Enzymes - The Cupfer / zinc superoxide dismutase type 1 (SOD1) . The defective enzyme polymerizes and " poisoned " the motor neurons via multiple mechanisms , especially in any case about the increased oxidative stress - free radicals (ROS) in the energy production can no longer be degraded arise in the mitochondria and damage many other proteins -> leads to increased INFLAMMATION (inflammation) in the area of the nerve

2) GLUTAMATE induced excitation

increased excitatory stress - because of the excitatory (= excitatory) neurotransmitter GLUTAMATE is no longer degraded by the SOD1 disorder and "stimulated to death," the nerves.

3) prostaglandin E2 in the neuron ?

Latest data (February 2012) show a possible involvement of the main inflammatory substance PGE2 . By inflammatory and, inter alia, by change of diet (get away with omega 6 oils , but more Omega3 : fish oil, linseed oil) , this can be influenced.

How come now to the disturbance of SOD1

Depending on the installation - Depending on the installation = genetic = Family. The final expression can be triggered by relatively small TRAUMAS of the spine in the genetic tendency.

Intoxication - Cyanobacteria but a "neurotoxin" from which can induce SOD1 disorder. This toxin can be present in various seafood in high concentration.

ROS - increased Radical - Oxidative Stress, Free Radicals by stress or lifestyle (smoking, drugs) induced - in lack of protective micronutrients ("antioxidants" - almost always missing in modern " industrial food " = everything is wrapped in plastic) .

Free radicals destroy DNA and possibly lead to misfolding of SOD.

Fehlsplicing - by disturbances of the correct protein formation - presumably again by poisoning of enzymes or mutations of mRNA transport

Unclear environmental factors

Exciting that the brain fluid from diseased animals - injected into healthy embryo - there exactly the same damage and triggers changes as it exists in the diseased animals. This means that the basic damage NOT primarily GENETICALLY but above is maintained or possibly triggered the PRIONS mechanism described below and running - by environmental factors.

PRION -Style Michanismus

Prions are proteins, the "Missing Folded" were in their production and their misfolding is "infectious". A protein is indeed from a long amino acid chain, which must occupy a certain spatial structure (folding) so that it can fulfill its normal function. Misfolding leads to dysfunctional proteins that have to be rapidly removed from the cell. If such non-functional proteins accumulate namely, the degenerate cell (eg, Alzheimer's disease, Creutzfeldt -Jakob , ALS) . Prions are now misfolding that mutually further propagate itself, so transferred the proteins own misfolding to other proteins . There is evidence that the "Mutant SOD1" is disturbed by a prion mechanism.

Why it affects not just a few but all motor neurons?

This part is still unclear: when a motor neuron degenerates, the suffering is propagated throughout the system, from one nerve to the next until the complete degeneration of all nerve and complete paralysis of the organism is. Again, it is significant that a typical aspect of disease can be transmitted to healthy embryo by injected cerebrospinal fluid of diseased animals , ie that changes in the ambient medium of the nerves (" inflammatory factors , cytokines, ... ") ultimately induce a " Selbstpropagierendes destructive patterns ."

Further information on the ALS Cause

Aspartame - Artificial Sweetener -> excitatory neurotransmitter

Oxidative stress , free radicals, peroxides

Interleukin -1 as a mediator of inflammation accelerates ALS

Axonal transport blocked leading to autophagy

Mitochondrial disorder as a mechanism in ALS

Diagnosis of amyotrophic lateral sclerosis

December 2011: discovered new biomarkers , which can diagnose with high AS security

Possible Therapeutic Approaches

The most important point is probably the control of the platelet serotonin content. This can multiply the survival time in ALS!

SEROTONIN

SEROTONIN in platelets determines the course of Erkan Kung

Serotonin Survival ALSDer SEROTONIN CONTENT of blood platelets determines the course and the duration of ALS disease. Niederer plasma levels of serotonin = < 50 months , high plasma levels of serotonin = 150 months !

see : Study Dupuis Sept 2010

Very exciting NEWS! Because of this platelet serotonin is in a very different context prominently on my homepage read - regarding "Tryptophan metabolism / cravings / depression" .

Serotonin as an important co-factor and predictor of ALS progression was described in 2006 in a landmark study of Sandyk. But I find it completely incomprehensible why this information was picked up by 2010 by any other research group!

Am I cynical when I point it, that serotonin is not patentable?

Sandyk described that ALS patients and model animals show strikingly low serotonin levels in motor neurons and exhibited for the first time to potential therapeutic need to raise serotonin levels.

How do we know this is nowadays very easy, using a simple dietary supplement (not patentable) .

In-depth information on serotonin and its Mangelzuständi in the body , especially the harmful effects of antidepressants.

Retzek comment: This is an extraordinary result , incomparable with all other previous study results, I have found and cited!

Affected individuals with ALS among my readers: Correct their plasma serotonin levels, which with a simple and inexpensive dietary supplement is possible to high normal serotonin levels (up to 200ng/ml are quite normal values), they have this "great atmosphere" and perfect appetite control and prolong their life span.

If you are affected in this regard and seek advice - or even when you are a journalist and have the opportunity to spread this in a newspaper or magazine, the disease is so "disastrous" that this news should get around quickly : please call me at : 0664-2642765 or 07672-23700 (private -11)

More therapy. Evidence from the research

Vitamin D and amyotrophic lateral sclerosis - In this article is presented that many of the ALS underlying causes may be affected by vitamin D in a positive way and manner. Since vitamin D is now identifiizert as all-encompassing health preserver (Evidence Level 1A) is an optimized setting on good physiological Vit D levels conducive ANY EVENT . (Study May 2011)

N-acetylcysteine - In an animal model of Huntington's disease and ALS shows N- acetylcysteine nervenzellprotektive effects and prolongs life. Study AS 2000 - see my page for Huntington's disease

Problema Grande : NAC costs in the weekly pack € 3, - in the pharmacy (ACC) , so it will probably never be allowed to come to a clinical trial.

Melatonin - Review 2009 - Melatonin affects neuroprotective in ALS by inhibition of apoptosis. Very exciting because melatonin in tumor therapy has exactly the opposite effect , namely to trigger apoptosis of malignant cells .

Vitamin E intake reduces risk of ALS significantly - A variety of studies have examined VE - taking and associated with diseases, especially "Cancer" . In a post- analysis of more than 100,000 participants and > 800 ALS cases - has been found that VE SIGNIFICANTLY to the risk of developing ALS decreases u.zw. dependent in a period style & fashion. If the study participants had taken vitamin E for 1 year reduced the risk of ALS, but if VE was taken 2-4 years if taken by nearly 25 %, 5y , then ALS risk was reduced by 30%. Study March 2011

In a 2005 study , there was a risk reduction of 60% in subjects who VitE more than 10 years had taken , wärhend for VitC no effect could be detected : Study 2005

Cannabis and neurodegenerative diseases such as ALS,

Cannabinoid receptors are the most widespread in the brain and strongly influence the membrane fluidity of neurons. Disorders of this system (eg by excessive cannabis consumption or by lipid disorders) could have a major impact on outbreak / course of these diseases. (Feb 2011) .... plays important functions in neurodegenerative and neuroinflammatory disorders like Alzheimer 's disease , Parkinson 's disease , amyotrophic lateral sclerosis and multiple sclerosis ..... There is evidence that influenced by CANNBINOIDE (cannabis) , this system positively neurodegenerative diseases and will be influenced in a positive sense .

Note Retzek : In my practice, I can several cases of long-lasting "Fatigue" and complete loss of appetite / nausea induced by chemotherapy report, which was disbanded permanently in a few minutes after a single administration of a cannabinoid .

Creatine as a therapeutic agent in neurodegenerative KH - Studies show that Creatine - a cofactor for energy production and transfer of energy in muscle and brain (therefore often used in body- builders) - may improve the survival of nerve and can cause a lifetime extension of almost 10% even in healthy mice (2008). Studies on the therapeutic use of creatine in ALS, Parkinson's, Huntington's disease are currently under way. March 2011.

Coenzyme Q10 as a mitochondria - protective therapy Neurodegenerative KH already on my Q10 page you can read many studies on the Q10 and neuroprotection. Subsequent study shows significant effect of Q10 together with creatine on neurodegenerative mouse model (June 2009).

So creatine and Q10 could be great therapy help: in the mouse model, a 40% extension of life time application , a 50 % improvement in the scores after 1 year in PD patients in clinical study!

I have swapped out the exact studies on a separate page :: http://www.homeopathy.at/als-updat/

Prostaglandin E2 as a contributor? COX -2 anti-inflammatory drug could help

Abnormal activation of the prostaglandin E2 production in the area of nerve - Cox -2 inhibitors can slow down this. study 2010

Abnormal prostaglandins allow for the first time definite diagnosis of ALS : Study February 2012

Prostaglandin E2 - receptor is a pacemaker for the degenerative inflammatory process - Study 2008

Long-term users of anti-inflammatory drugs have .... to get a significantly lower risk neuroinflammatory disease such as ALS , Parkinson's. PGE2 may be important therapeutic approach: Full Text Review 2008

I do not understand is that we have so little "Impact Research ". Well on that account because represent all Entuzündungshemmmer unpatented expired medications and corresponding Forshcung therefore is priceless?

Spinal cord constriction may be more important in triggering pacemaker of ALS and MS

A case report of 3 patients shows that a herniated disc with narrowing of the spinal canal as possible triggers of neurodegenerative diseases (such as ALS and MS) could be. Feb 2011

Lithium as a therapeutic agent in ALS - Lithium is used for decades in psychiatry, it is a very effective and safe salt. Reports regarding the efficacy of lithium in ALS and Huntington's disease are "complementary" repeatedly cited : 2010th lithium users were significantly protected against ALS mechanisms and versus neg glutamate effect (2003). .... Lithium 's neuroprotective abilities did imply it could be used to treat or prevent prevention braindamage Following traumatic injury , : such as stroke, and neurodegenerative diseases: such as Huntington 's and Alzheimer 's diseases ..... Meanwhile, also shows a clear molecular mechanism lithium may be the "autophagy " - the self-dissolution of nerve cells - block and also many other mechanisms of neuronal cell death (2004). A variety of application studies are currently under way. . Nov. 2010 Incidentally, lithium has other positive effects on the brain : it protects against the negative effects of alcohol on the brain (" death of neurons through the influence of alcohol : 2010)

PIOGLITAZONE and rosiglitazone in ALS

The glitazones were designed as a modern anti - antidiabetics . In fact, it is extremely potent inducers genetic - they stimulate the cell's production of peroxisomes

For the first time, I found a study from 2005 on the neuro- protective effect in ALS mice : study 2005

Also, another study from 2005 which the survival time of mice models by 13 % increase with pioglitazone

A study showing that the neuroprotective effect is not due to improvement of glucose metabolism in the brain appear in 2006.

2007 will be a study showing that in spinal injuries caused by pioglitazone to achieve significant improvement in the long-term outcomes is through a rundown of the injury following nerve degeneration . In another study of 2007 super show effects in spinal injuries.

From an exciting review work of 2008: (important only the text in bold characters)

We have tested the neuroprotective effect of pioglitazone in G93A SOD1 transgenic mouse model of ALS and Showed did pioglitazone treatment improved motor performance , delayed weight loss , attenuated motor neuron loss , and Significantly Increased survival by delaying the onset of ALS [38] . Our results therefore show did pioglitazone treatment Reduced microglial activation and gliosis in the spinal cord as Assessed by immunohistochemical staining for CD40 (microglia marker) and GFAP (astrocyte marker) , respectively. Further More, we did pioglitazone treatment Showed Reduced iNOS , NF- B , and 3-nitotyrosine immunoreactivity in the spinal cord of G93A transgenic mice . Our findings were confirmed .... so by another study on the effect of pioglitazone treatment in G93A SOD1 transgenic mouse model of ALS [39] . In this study, PPAR - ? agonist treatment improved survival , muscle strength , and weight loss in ALSmice . Quantification of motor neuron loss what nach EN 90 days of age where approximately 30 % of motor neurons were lost in G93A mice spinal cord. Pioglitazone treatment completely Call Prevented this motor neuron loss in the spinal cord of G93A mice. They also Showed significant reduction in microglial activation as well as reduction in the expression of COX -2 and iNOS [39] .

In 2010, this effect can be further confirmed. Pioglitazone is a serious therapeutic approach for ALS!

Exciting, a recent study shows that induced spinal cord injury (in rat) Pioglitazone have significantly less permanent damage than in the control animals. Feb 2007

Pioglitazone (Actos (r)) take 1 x daily, with or without food , always at the same time currently is taking " off-label" , but clinical trials are ongoing.

<u>To avoid</u>

Glutamate and ALS - Glutamate is a neurotransmitter for activating motor neurons (the nerves that degenerate in ALS) . Once glutamate has exerted its stimulatory effect, it should be removed as rapidly as possible , so by the nerve cell protection (GLIA) cells. If it is not taken "poisoned" the glutamate motor neurons and ultimately leads to their "over -stimulatory degeneration" . Inhibition of glutamate secretion can influence positively in the rat model of ALS disease. Study March 2011. The implication of this study lies in my opinion in regard FOOD GLUTAMATE to minimize (glutamate -free condiments) , but without having a clear evidence that this influence on the brain glutamate metabolism increases .

Aspartate = Nutrasweet = "LIGHT products"

Aspartame -free report1 - Aspartate is not as fast as eliminated glutamate and leads to the "excitation" to the excessive duration of stimulation of neurons , which favors their death. Sept 2010.

Mercola - my personal idol as "medical science" - has a big section with information collection to the beneficial effects of the artificial sweetening substance ASPARTAME (Nutrasweet) , and a section with acute ellen original studies on aspartame. This molecule acts in the brain "excitatory" - ie permanent cause for certain nerve cells , especially in the motor neurons .

Aspartame -containing LIGHT products must be absolutely avoided!

Dr. Dietrich Klinghardt & ALS

Note: Dr. Dietrich Klinghardt claims in his lectures and videos that no patient with ALS more it died for about 10 years. According to him, the ALS is a multifactorial combination of several factors:

1) BIOFILM infection in the brain and of the afferent nerves in the brain infection by borrelia and babesia and

2) exposure to toxins (pesticides) and heavy metals (especially mercury from the amalgam here) .

3) subtle specific micronutrient deficiency , vitamin deficiency

Klinghardt could by decades of research a "cocktail" design with which he treated many neurodegenerative disorders and which includes everything "eclectic ", which eliminates the aforementioned causes. Klinghardt used to be very comprehensive therapeutic Sprektrum (orthomolecular , phytotherapy , physical therapy , homeopathy, detoxification , psychotherapeutic techniques , Systemic Therapy , ...) demand-driven individually and according to kinesiology testing of.

Therefore, his system cannot be "just" Accept Reproducing , you have to go to a good and long -trained therapists. Klinghardt - I always recommend here Ulrike Grosch in Vienna , which is then distributed to their students.

Heavy metals as a cause

These studies are thinly sown and also very critical. Actually, yes probably no vested interest exist here to give clear instructions as this serious under some circumstances political and V: would entail economic changes by itself, so here at this level only "silent revolution" instead - as the removal of : A thimerosal (mercury) from vaccines after since the introduction of Monster inoculation of all children (in the U.S. up to 50 vaccinations to school). AUTISM rate has increased enormously within a few years . Because of potential damage claims , it is of course impossible at present only some amalgam critical study in established "peer - reviewed " journals accommodate whose possession is the established pharmaceutical community.

Systematic Review on many studies: shows an undeniable Ri increase by pesticides (Study 2009)

Mercury accumulates over the years in the nerve cells of mice, but solves NOT the typical ALS protein changes from (Study 2010). Now, this study is concerning because so interesting because it is shown here

how dramatically Mercury intercalates permanently in motor neurons. While no ALS proteins are produced, but the study aimed not to other, caused by QS changes which would be easy to imagine . So far, so any correlation between QS and neurological diseases has been denied!

Case of 81j difficult with QS intoxicated patient with ALS (2009 publication)

Literature review shows overview moderate what is published in various studies on context, heavy metal and various types of ALS. V.A. LEAD is suspected as a potential candidate. V.A. Here the notice that certain GENETIC CONSTELLATIONS talk and act " toxic " heavy metals in the brain can (Study 2011), which Klinghardt always preaches that ApoE - sulfur groups brain detoxify and ApoE4 the heavy metals leaves without sulfur group in the brain and these pave the way for a systematic infection with borrelia and babesia .

In 414 ALS patients in the cases of borrelia was only 4% - antibody titer increases . Therefore Borrelliose is excluded as ALS -timer (Study 2009)

CYSTIC form of borrelia could be the trigger of a variety of neurodegenerative diseases. This form is ignored by many academic researchers and tracked adverse, other researchers are convinced . Med Hypothesis 2006

I would in any event optimize serotonin levels , taking possibly one a Cox - 2 inhibitor, reduced glutamine ingestion, highly dosed antioxidants for the brain to take (melatonin , ALA as amphiphilic also penetrate in the oxidized state, the cell membrane) and extra iron and Cupfer from the brain rausholen (eg by EDTA infusion series) . Dr. Retzek , according to research from 1400 PubMed - studies

Finally, a separate case from the practice:

55j patient comes with ascending weakness of the legs , can only walk with help.

Duration dizziness - falls not held to when.

Bulbar weakness: cannot speak intelligible. Choking, biting his tongue / cheek

Severe depression. Suicidal, completely Perspektivenlos

For 2 years, several hospital stays (Neurology) with no result.

Homeopathic initially hardly a therapeutic appeal to me - of course: in such a neurodegenerative disease, we are actually just happy if we can slow down the progression, a "standstill" is already "a miracle ".

Gruber - Institute mineral medicine - only own mineral and heavy metal measuring point in Austria