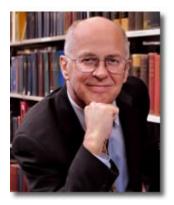
For more than a century, the medical community has been puzzled by amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease). No cause or cure has come forth, according to most researchers.



David Steenblock, DO, says he knows one of the major causes. He hopes to publish a paper with his theory sometime this year.

ALS afflicts perhaps 20,000 people in the US and several hundred thousand people globally. It involves degeneration and death of motor neurons in the brain and spinal cord. It's invariably fatal, often within a few years.

Steenblock, 73, an osteopathic physician and independent researcher based in San Clemente, California, says that ALS is "the most puzzling and insidious human disease there is." He believes that it's generated by a series of events.

## His theory in summary form:

There is an initial trauma to the neck. Following this trauma, over a period of years, degenerative joint disease develops in the neck. Then another injury to the same area introduces a break, an opening, between the blood and the cerebrospinal fluid. Blood carries toxins to the damaged spinal nerve; from there the toxins enter into the cord and cause injury and death of motor neurons.

Steenblock's forthcoming paper will present evidence based on his study of 54 patients, of whom 52 had these changes as revealed by cervical CT (computerized tomography). He believes he has found the most positive biomarker yet for helping to diagnose ALS – a 97 % correlation between (a) spinal nerve injury and reinjury and (b) occurrence of ALS. If his theory holds up, it will represent a major breakthrough and might contribute, in time, to new treatments.

There are two basic forms of the ALS – spontaneous (affecting 85% to 90 % of patients; this is the focus of Steenblock's work) and familial (the remainder).

ALS killed baseball star Lou Gehrig in 1942 (age 37), actor David Niven in 1983 (73), and historian Tony Judt in 2010 (62). Soccer star Patrick Grange had ALS at the time of his death in 2012 at age 29. Physicist Stephen Hawking has a rare slow-progressing form.

Steenblock was born and raised in Iowa farm country. His family raised corn, oats, and soybeans on 160 acres near the town of Buffalo Center in the northern part of the state. The Steenblocks were poor. By age 7, David was doing a full load of chores, getting up at 5 a.m., running a tractor or working in the barn for a couple of hours, going to school, getting home at 4 p.m., and doing chores until dark. Then homework. "It was a little tough," he recalls, "but I learned to work hard, and I've been working hard ever since."

Graduating from Iowa State University in 1964 with degrees in zoology and chemistry, Steenblock enrolled at the College of College of Osteopathic Medicine and Surgery in Des Moines (now known as Des Moines University) and earned a medical degree and a master's degree in biochemistry. He did a rotating internship at a very large medical center in Seattle, spent a couple of years as sole practitioner in

a small town in the Pacific Northwest (with a 32-bed hospital), studied anatomical and clinical pathology for three years at Case Western Reserve University in Cleveland, and did a year of clinical pathology at the University of Oregon in Portland.

In the 1970s, as alternative medicine gained ground in the US, he visited chelation pioneer Garry Gordon MD in Sacramento, California, and came away impressed by the treatment successes. In 1978, in Lake Forest, California, Steenblock opened one of the country's first comprehensive, integrated, holistic medical centers.

Steenblock's current clinic and laboratory in San Clemente covers 14,400 square feet. It holds 60,000-plus medical books, he says, and about 1 million cataloged scientific medical articles. Steenblock spends two or three days a week in the clinic (he and his team treat many other conditions, including multiple sclerosis, stroke, Parkinson's disease, and cerebral palsy) and fills the rest of his working hours with research. He works seven days a week just as he did on the family farm in Iowa.

Steenblock's website is personalized-regenerative-medicine.com. It will include publication details on his ALS paper as soon as they are available. The website for his nonprofit foundation is stemcelltherapies.org.

BF: When did you begin studying ALS, Dr. Steenblock?

DS: In 1977. That's when I had my first case. I developed my theory of primary causation almost immediately, and I've been working ever since to prove it, which I believe I've now done.

BF: No one else has put forth this exact theory?

DS: That's correct.

BF: Let's get to the theory in a moment. First, I want to ask you the burning question that every ALS patient wants answered: What are the chances for a cure in the immediate future, or for really solid treatments that will extend lifespan by many years? I will note here that ALS patients and their families have been disappointed many times over the years by failed approaches to the disease.

DS: In terms of a cure, I don't know when. But in our clinic we already can slow the disease and oftentimes reverse it. Reverse means a regaining of lost functions; this regaining can be long-lived or short-lived depending on how effective we are at ridding the body of all the bad toxins and putting in really good stem cells.

The exciting thing is that we now know that an infection of some type is involved in this disease. In the intestine, this infection usually is a combination of yeast and bacteria which forms a biofilm. There are tests we can use to determine what type of infection, and special assays to better know how to treat them.

In addition to the most common form of infection – yeast and bacteria – various other things may be

involved: retroviruses, prions, misfolded proteins, damaged neurofilaments, and other cell-derived aggregates. Dissolving these aggregates as they occur in cerebrospinal fluid is a promising new method of treatment we are working on.

"Cure" means for the disease to go away and never come back. This may be possible, eventually, with the right agents to rid the body of biofilms, heavy metals, retroviruses, prions, amyloid, misfolded proteins, CSF aggregates, hydrocarbons, and other toxins. Identification of each of these toxins, and treating each individual successfully for their specific toxins, and use of stem cells, should give us the cure. As I say, I don't know when this will happen, but I'm optimistic that we have this disease cornered. We just have to beat the bugs before they beat us.

BF: Please elaborate on what, in your opinion, causes this terrible affliction.

DS: I believe that ALS is a complicated combination of unfortunate circum-stances that occur sequentially.

The first occurrence – for most cases – is a neck injury, perhaps caused by a fall, a collision on the sports field, whiplash, some kind of trauma that injures the cervical or neck vertebrae. This injury generally occurs many years before the onset of symptoms – perhaps in high school or shortly thereafter. The injury heals to a certain extent, but it also degenerates from wear and tear, so that, 20 or 30 years later, you see degenerative joint disease such as osteoarthritis, and also something called neuroforaminal stenosis (NFS), a narrowing of the spinal nerve canal, often with calcium deposits around that spinal nerve. It takes many years of chronic irritation for this constriction to occur. So, over time, you're seeing an increase in the amount of extracellular calcium in and around the affected spinal nerve.

There's a reinjury of the same area at some point years later. As a result of this reinjury, a number of toxins are able to penetrate into the spinal cord, including extracellular calcium. Also penetrating are white blood cells – monocytes – which deposit their toxic interior contents in the cord. These toxins lead to the death of motor neuron cells. This is ALS.

There's an almost 100 % correlation between ALS and a breach in the blood/cerebrospinal fluid barrier. Junk from some type of infection in the body is going in and damaging the cord. And that, in a nutshell, is my conclusion about the primary cause of ALS.

There's a lot more to it, of course. ALS is truly a holistic disease, a fact not understood by mainstream researchers.

BF: They overlook the gut?

DS: Yes. In my opinion, the gut is the source for many of the poisons that trigger the ALS disease process. Now, I will note, most patients with ALS do not have diagnosed intestinal problems. Their bowels seem fine in the sense that they don't have pain, diarrhea, or constipation. They really cannot believe, nor do most doctors believe, that the intestinal tract has anything to do with ALS. But it does, in my opinion. When you do lab tests with these patients, you discover they do have gut problems. These problems are

consistent with chronic inflammatory infections seen in colitis and ileitis.

So their guts have infections, especially in the terminal ileum. Generally speaking, the infection is a biofilm. This consists of yeast and bacteria that have taken up residency in the gut wall and have been there for a long time. They sit there and fester, and fester, and fester. So biofilm is a key part of thinking about this disease. It's a very new idea that biofilms are part of ALS.

BF: I'll insert a bit of background here — microbial biofilm consists of "aggregated microorganisms surrounded by a self-produced matrix adhering to surfaces or located in tissues or secretions." The concept was recognized in the 17th century. Biofilm infections started getting serious attention in the 1970s.

The term biofilm was introduced to medicine in the 1980s. Biofilm has been the subject of intense study in recent years; for example, in 2002, the National Institutes of Health issued a major invitation for grant applications for study of the material.

DS: Biofilm protects the yeast and bacteria that make it up. The protection is kind of like a hard-to-penetrate sheet. This prevents the patient's immune system from attacking biofilm; often the patient cannot mount an attack against these bugs.

Biofilm is extremely complicated stuff. It's tough to get it out of there. It's like barnacles on the bottom of a ship that have been there for years and are cemented on there. You pretty much have to take a hammer and chisel to them. White blood cells – the monocytes – do what they can to fight biofilm. There's not a lot for them to attack.

OK, now I want to tell you about superoxide.

The bacteria and yeast on the inside walls of the intestine produce free radical molecules called superoxide. This stuff is poison. The white blood cells try to deal with it, try to protect the body, by secreting an enzyme known as superoxide dismutase (SOD). This is a much-studied enzyme. It has been found to have many mutations in the familial form of ALS. These mutations cause the SOD to not operate correctly, and to form aggregates that poison the cord in the familial form.

Within the monocyte is the endoplasmic reticulum, which is like a little river flowing to the outside of the cell. On the banks of the river are ribosomes that make SOD. The SOD molecules, when they're released into the endoplasmic reticulum, are not in a completed form. They're in an unfolded form. They need to get folded in order to be effective against superoxide.

They travel along this river, from the inside of the monocyte cell to the outside, and exit. As they travel along the river they're escorted by chaperones, little tugboats. These chaperones get the superoxide dismutase to fold properly, so that by the time the SOD exits the monocyte, it's folded and fully functional.

In ALS, the SOD doesn't get properly folded. The chaperones aren't working, they're rusted out, they're

falling apart from the toxins the cells are trying to deal with. So the misfolded SOD is trapped inside the monocyte, it can't get secreted unless it's folded properly. There's a gate at the monocyte's surface that stops it from getting out. The misfolded SOD accumulates in the monocyte's endoplasmic reticulum.

The monocyte is attracted to the reinjured area of spine. It deposits its contents in the constricted damaged spinal nerve. These contents include the misfolded SOD and other toxins. These toxins are joined by the extracellular calcium we discussed previously.

The toxins lead to the formation of aggregates which are engulfed by microglia and astrocytes within the cord. This in turn leads to the production of free radicals and peroxynitrates and so on; and these cause damage to the motor neurons, which is a defining part of the ALS disease process.

Various other materials are also involved in this – endotoxins, serotonin, arginine, tumor necrosis factor, gamma interferon, certain cytokines. And, very importantly, high mobility group box 1, which I think is a key trigger for the disease process, and is a hot topic these days in the study of ALS and all the autoimmune diseases. It's found in the cytoplasm, the nucleus, and so on. It's triggered by inflammation, infection, ischemia, a lot of things.

Heavy metals are a part of this too, especially mercury and lead. In 1968, we saw the first four cases linking lead toxicity to ALS, and since then we have seen a lot more cases, but to this day, the standard conventional doctor doesn't think that heavy metals are a contributing factor. These heavy metals damage cell membranes and allow calcium to enter into the cells; this excess calcium damages and will even kill the cells. Calcium influx is the "final common pathway for cell death." Most alternative doctors – comprehensive, holistic, integrative – understand this concept. They're much better doctors when it comes to handling complicated diseases such as ALS which have holistic or "many systems" origins.

I'll add here that the poisons that cause ALS can come from places other than the gut. Sinus infections can be involved. Also Helicobacter pylori, Lyme disease, mouth spirochetes, osteonecrosis of the jaw, dental abscesses. Treatment of these can result in significant improvement. But probably 90% to 95 % of all ALS cases originate in the gut.

BF: What's your treatment protocol for ALS?

DS: We use a variety of agents, especially stem cells, to seal the injured area in the spine where the poisons are entering the cord. We inject stem cells directly into the injured cervical spinal areas to stop the leak and the progression of the disease. Intravenous and intranasal stem cells are helpful in this regard and also for overall treatment.

I do a lot of testing to identify the biofilm bugs and then try to treat them appropriately. The biofilm is usually the number one enemy, and to treat that effectively, one must use a variety of treatments all at the same time. In our clinic we throw everything we can at biofilms including antifungals and antibiotics plus electrical stimulation to open up cell membranes. Ultrasound. Photodynamic therapy, laser therapy, hyperthermia, intravenous vitamins and minerals. Ozone – IV and topical. Autohemotherapy. Oxidizing agents. I have a microbiology laboratory to do some really fancy testing. Our stem cells are from our own laboratory.

We have a few patients in remission. We're the first clinic in the world, as of the autumn of 2015, that can do T cell therapy against ALS. This involves taking blood from someone with ALS and converting their T cells into immunologically stimulating cells that work well against yeasts or whatever infections they have in their system.

We believe that we are on the cusp, here in our clinic, of getting people truly well. Now that we've identified one of the principle causes, it's just a question of identifying the best therapy. I think each patient will be different in terms of the bugs involved. I'm open to suggestions about therapies, by the way. I solicit ideas.

BF: Could you provide some nutritional advice that might be helpful for an ALS patient?

DS: I recommend a ketogenic diet, which is high fat, adequate protein, and low carb. Stay off all sugars – fruits, fruit juices, candy, cakes, ice cream, alcohol. Avoid acidic foods such as tomato sauce, processed foods, dairy products. Avoid salt. Eat small, frequent, bland meals. Wear a neck collar; avoid injuries to the neck, including twisting. Take as many natural antifungal agents as you can, such as caprylic acid and garlic. Take a level teaspoon of freshly crushed raw garlic 3 times a day. Mix a quarter teaspoon of calcium EDTA, a chelating agent, with this, if you tolerate it. Coconut oil is very good – it's the cheapest and best antiyeast product we have – 4 tablespoons a day is great, up to as much as 8 to 10 tablespoons a day. Scale up slowly. So, definitely, the natural things are good, but as I say, you also need the antifungals and antibiotics, and stool tests and so forth.

Interestingly, arginine, which is found in abundance in nuts and seeds, may well help the immune system fight biofilms. But at the same time, arginine stimulates the growth of yeast, so when arginine is consumed, one needs to treat yeast with antifungals and a no-sugar diet. Nuts and seeds should not be taken intact by ALS patients, since nuts can irritate the gut and worsen the condition. Ground nuts like almond butter or peanut butter should be used instead of whole nuts. Arginine in the form of a supplement is OK if used with antifungals and a no-sugar diet. Arginine alpha-ketoglutarate (AAKG) is especially helpful.

BF: What is your view of mainstream ALS research?

DS: For the most part, ALS researchers are very academically oriented; they're located at big universities and big research centers; they talk about the mutations that cause the familial form of the disease, and how those mutations might help us figure out the spontaneous form, how we can extrapolate from the one to the other. Some lessons from familial can be applied to spontaneous, but really, in terms of the spontaneous, I think I've pretty well pinned it down in terms of primary causation.

BF: Is there anything new in the mainstream that's interesting to you?

DS: There's a new technique called CRISPR whereby it will be possible to cut out bad genes and maybe put good ones in, but this has a ways to go before we can use it to treat the familial form. We're starting to look at it in our clinic.

BF: Some ethical concerns about that, yes?

DS: That's right.

BF: With regard to research priorities – I'm not clear why most research focuses on the familial form of ALS, since it represents just a small fraction of cases.

DS: This focus happens because you can use a mouse model in dealing with the familial. The PhDs doing the research can look at mouse genes and say, "Oh! Here's an interesting gene! You get the misfolded SOD with this!" They've found about 170 different mutations of familial ALS. What does that have to do with spontaneous? Not much.

The researcher can take a mouse or rat and tear it apart and look at every little detail and run it through a lot of high-powered technology, and write papers, and boy, that's great! Writing papers and getting published – then you get more money from the government, and you can keep running on your happy little treadmill. They work 20 years and they say, "We have a lot of really interesting discoveries, but we need at least another 20 years." You say, "What have you come up with that's practical?" They say, "Well, we haven't really come up with much that's practical, but we sure know a lot about the disease now! And one of these days we're going to have the answer! This has a lot of potential!" And nothing ever happens because there's nothing there.

The lack of practical knowledge on the part of the PhDs is a real problem. They don't see patients, generally; they're in laboratories and attending research conferences. They talk about things that are very academic, very complicated, and very detailed, and they need to spend all their time doing that if they're going to get ahead. They study the most minutely detailed material such as genes and proteins, RNA, DNA. Yet they really don't have a clue about the spontaneous form of the disease.

BF: Can you elaborate on how your training in alternative medicine, and de-cades of experience in the field, helped you arrive at your causation theory?

DS: I would never have solved this if I had not been looking holistically at the microbiome and seeing that yeast infections are a problem; if I had not known about certain metabolites found in the urine; if I had not known about serotonin problems associated with ALS and why they are occurring. And so on. My training and experience in alternative medicine have actually been more important to me than anything else for understanding and better defining this disease.

BF: You have established the Steenblock Research Institute, a nonprofit 501(c)(3) that accepts donations that are tax deductible. Suppose someone donates \$5 million for ALS research. How would you spend it?

DS: First of all I'd buy a couple of very special machines such as a MALDI-TOF spectrophotometer, and some new molecular-weight mass spectrometers, to look at the aggregates in the cerebrospinal fluid, and try to better identify what they're made of, and what we can do to dissolve them. I think the thing that can lead to a cure is solving these aggregates, and superoxide dismutase, and the prions, and the

neurofilaments that are involved. Mass spectrometry allows us to look at the yeast and bacterial metabolites in the blood and intestinal tract and cerebrospinal fluid. So that's close to \$2 million right there. Then you need a laboratory to fit around these machines, and qualified personnel. So \$5 million would get me up and running for about 3 to 5 years and then I'd be out of money. But by that time I would probably pretty much know what was going on, and what to do, and how to do it.