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October 19, 2017

Dear Rudy and Erin,

Thank you for the cards and letters expressing Christ's love, strength and encouragement. All you've sent is greatly appreciated. Many thanks, too, for your special help to my precious husband.

Through all of this, the power of prayer is most important. Only by the grace of God, do we receive His healing, forgiveness, and mercies. With Christ in us, anything is possible, without Him — nothing.

I have truly enjoyed and learned much from reading the books you've sent. Enclosed is a six-page compilation, plus three pages from Scientific American, that I hope you find interesting. I underlined the parts that appear to be connected to the other info. My hopes and prayers are for this to inspire others to further research, that it may be beneficial to someone.

Hope you're doing okay with the job market being as it is. Our faithful trust in Christ Jesus, will open doors in His timing. May you be blessed in your work for the Lord. Your ministry appears to be His blessing through you.

May God's justice overcome the iniquities of mankind, May His liberty replace tyranny, and His peace, love, and truth reign over all the world. Amen. All in the love, faith, trust, and hope in Christ Jesus.

Peace be with you,
Love from your sister in Christ,
Karen

ALS, ALZHEIMER'S AND LYME'S CONNECTION

Disclaimer: The following information is not to be construed or intended as medical advise. Any person with a medical issue should consult a medical/healthcare professional. The purpose is to encourage others to research and is for educational purposes only.

With that declared, the following information is available for public reading in:

"Green Vanilla Tea" author, Marie Williams (2014)
"Beyond The Rays of Hope" by David Kurt McClain (2016)
"Incurable Me" by Kenneth Paul Stoller, MD (2016)

There seems to be a connection between the devastating diseases suffered by David K. McClain and Dominic Williams, though their worlds were far apart. The main possible common thread appears to be a corkscrew-like bacteria of the Spirochaetaceae family which is found in Lyme disease, amyotrophic lateral sclerosis (ALS), Alzheimer's disease, schizophrenia, and even Parkinson's disease.

Mr. Williams widow writes that her husband was diagnosed, after much denial and mistaken diagnosis, to be ill from ALS plus FTD (frontotemporal degeneration). FTD, one of many neurodegenerative syndromes, results in a wide range of behavorial, cognitive, emotional, and linguistic symptoms, as the frontal brain lobes degenerate.

The Williams originated in South Africa where they enjoyed the outdoor adventures of the land. After the birth of two sons, they relocated to British Columbia, Canada, favor hiking and camping ventures in their local area plus trips to the Oregon Coast and beyond. All these areas pose a possible risk of being bitten by a tick, though none are mentioned in her memoir.

Also, Mr. Williams was never diagnosed with Lyme disease, though due to his work and out-door activities, it may have been present.

Shortly after another relocation, this time to Australia, Dominic began exhibiting unusual cognitive and behavioral symptoms. His work included travel to Sri Lanka and other remote, undeveloped countries. Over a period of four years, the two afore-mentioned diseases ravaged his body, until he passed away at the age of 44 years.

Now, to Mr McClain's story. Born and raised in northern Texas, he grew up a normal, active boy. He migrated to Connecticut in his early twenties, where he met and married the love-of-his-life, Donna. Their family increased with the births of two sons, and a daughter.

David was diagnosed with Lyme disease in 2001, received another tick bite a year later, then in 2003 was diagnosed with ALS, which by the grace of God, he has survived to write his inspiring story, "Beyond The Rays of Hope". He is paralyzed from his shoulders down, requiring the constant loving care of Donna, their family, and friends.

So, what may be the correlation between these two men's illnesses? There appear to be many possibly related causes.

1. Frequent activities in wooded areas and grasslands, favored habitat of ticks.
2. Connecticut and Oregon both states with reported cases of Lyme disease on 1993 graphs by Craven &Dennis.
3. Arkansas, which borders the northeastern tip of Texas, also reported cases of Lyme disease in 1993.
4. Fast-forward to 2017, all U.S. states, Canada, and Australia have reported cases of tick-born Lyme disease
5. In 1991, researchers, Will, & Barry, found that 42% of the ticks evaluated in Australia carry the spirochete

bacteria Borrelia. (Canada to Australia MD's deny this as causation to Lyme disease.)

6. Dr. Stoller's research in conjunction with other MD's and scientists, indicates that Lyme disease, of which Dr. Stoller personally suffered, and successfully treated, is caused by the orgnaism Borrelia burghdoferi, which is usually spread by ticks.
7. The Lyme spirochete bacteria infection exhibits as: faux(false) Alzheimer's disease, arthritis, joint pain and swelling.
8. ALS initial symptoms include: joint pain (hips, toes & shoulders), slurred speech, jaw cramping, difficult swallowing, tightness in throat, testicular pain, the weakness in grip of hands (thumbs & fingers), lower abdominal pain, and inability to expell air from lungs.

COPPER & ALS

Recent research indicates that Alzheimers, and Parkinson's diseases, and ALS share common, and/or overlapping pathologic mechanisms. ALS, AD, and PD seem to be the same disease, with slight variations in theme.

THE THEME -- synergistic actions of heavy metals and pesticides.

1. Pesticides accelerale the rate of a-synuclein fibril formation; the synergistic effect results in misfolded a-synuclein fibrils.
2. Neurospirochetosis -- borrelia bacteria thrive and become destructive in the weakened systems of which 90% contain these bacteria.
3. Pesticides affect the <u>microglial</u> cells in the brain. These specialized white blood cells are prevented from cleaning and protecting as designed/created.
4. Help is available for neurodegenerative disorders.
   a. Large amounts of curcumin is helpful.
   b. Add vitamin D3 -- the curcumin deconstructs the amyloid plaques found in Alzheimer's disease (UCLA study found at http://newsroom.ucla.edu./releases/study-finds-vitamin-d-may-97303)

c. copper protects prions (proteins found between nerve cells in the brain).
d. Normal prions bind with copper (Cu) and the copper protects against the conversion of prions to a disease-causing form, which occurs when manganese (Mn) gets too high.
e. Prions normally serve the important roll of neurogenesis (the growth of the nervous system).

Cognitive dissonance -- when you know one thing but try to believe another.

Alzheimer's neuroborreliosis. Pathologist Alan B. MacDonald, MD. reports in Medical Hypotheses (2007) that research indicates Borrelia spirochete bacteria in the amyloid plaques of 100% of the 100 Alzheimer's brains he has autopsied. Amyloid plaques are abnormal tangles of proteins found in neurodegenerative disorders. In a research article written by Judith Miklossy, and published in the "Journal of Neuroinflamation8" (2008), evidenced by Koch's and Hill's Criteria, Alzheimer's disease is termed a Neuro-spirochetosis. Spirochete bacteria are found in 90% of Alzheimer's brains. (2011).

POSSIBLE CONNECTION OF ALS & ENVIRONMENTAL EXPOSURES

DDT (dichlorodiphenytrichloroethane) is widely used to control insects in crops and livestock (food sources), and to combat insect-born diseases from WWII (1940's) to 1972. DDT was allegedly banned in the US in 1972, yet this has proved to be a false assumption, as only a general use moratorium was issued to reduce its use. DDT is still used as a pesticide for which it was originally formulated. DDT does not readily breakdown in the environment, but loses a hydrogen atom (hydrogen chloride HCL).

DDE (dichlorodiphenyldichloroethylene) is another form of DDT. It is just as harmful to humans and animals, actually the whole environment.

DICOFOL is another pesticide similar to DDT and DDE with the same environmental effects. It is DDT with one hydroxyl group added.

NEURODEGENERATIVE DISEASES & EXPOSURES TO PESTICIDES (2004). Research article indicates clearly that occupational exposure increased the incidence of Alzheimer's and Parkinsons disease. Connection between pesticide exposure in both human and animal neurological diseases is on scientific records. Rutgers University scientists directly link a specific chemical compound to Alzheimers disease that is found in DDT & DDE. These pesticides injure and weaken the immune system leaving the body susceptible to incidences of neurospirochetosis. Due to other toxins and heavy metals in food air, and water, one's immune system is already injured, effectively increasing the risks of neurological diseases.

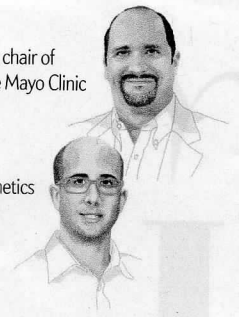
Dr. Stoller, MD. writes specifically in "Incurable Me' that DDT and DDE are directly linked to Alzheimer's disease." These insecticides are classed as organochlorides. They impact the electrical activity in the body, affecting the brain, heart, lungs, and immune system. They are carcinogens, causing cancers. Also they remain in soil, water for decades to come, contaminating food crops as these products are ingested by humans and animals.
Further information from an unclassified paper titled, "Studies of Biologically Active in Cells and Tissue Cultures" from the U.S.Army Medical Research and Development Command, with MIT (Massechuessetts Institute of Technology". "Poliovirus infection --in the polio-virus experiments...it is evident that at the 20 and 40 ug levels of DDT the yield of virus per cell was increased 37% and 90% respectively." Three years later the same article was published (2006) in the "Annals of the New York Academy of Sciences." It specifically stated, "that DDT increased the replication of polio-virus in human cells up to 90%"

The above information is but a tip of the iceberg. It is presented for the reader's interest. This writer hopes to inspire others to research further. "Incurable Me" contains well presented footnotes for verification of information.

Please note: The writer of this paper intends no monetary gain what-so-ever. It seems to this writer that much pain, suffering, expense, and heartache may well be preventable. Once suffering, pain, and fear exceeds your human capacity to bear it, you'll see that all that's left in you to bear it, is God's nature in you. May God bless all.

Leonard Petrucelli is a professor and chair of the department of neuroscience at the Mayo Clinic in Jacksonville, Fla.

Aaron D. Gitler is an associate professor of genetics at the Stanford University School of Medicine.



AMYOTROPHIC LATERAL SCLEROSIS (ALS) STRIKES WITHOUT WARNING. THE condition, which strips nerve cells of their ability to interact with the body's muscles, starts painlessly, with subtle initial symptoms—such as stumbling, increased clumsiness and slurred speech—that are often overlooked. The disease itself attracted little public attention until legendary New York Yankees first baseman Lou Gehrig began dropping balls and collapsing on the field for no apparent reason. Known as the Iron Horse for playing in 2,130 consecutive games over 14 years, Gehrig was diagnosed with ALS in June 1939 and delivered a poignant farewell at Yankee Stadium the next month. Gehrig's loss of muscle control progressed so rapidly that by December he was too weak to attend his National Baseball Hall of Fame induction. Creeping paralysis eventually left him bedridden. He died in June 1941 at the age of 37.

Today more than 6,000 people a year in the U.S. receive a diagnosis of ALS, now commonly known as Lou Gehrig's disease in the States and as motor neuron disease in Europe. It usually afflicts people between the ages of 50 and 60 but can start much earlier or even as late as one's 80s. At its onset, nerve cells in the brain and spinal cord called motor neurons begin to die. Because these cells send signals from the brain through the spinal cord to muscles, their death causes a loss of mobility, dexterity, speech and even swallowing. In most cases, the higher functions of the brain remain undamaged: people stricken with ALS are obliged to watch the demise of their own body as the disease advances unrelentingly. They soon become wheelchair-bound and, eventually, bedridden. Left with no capacity to communicate, eat or breathe on their own, most die from respiratory failure within three to five years. The sole Food and Drug Administration–approved drug for ALS is the glutamate blocker riluzole, which prolongs survival by an average of three months. There is no cure.

Pioneering French neurologist Jean-Martin Charcot, who identified the disease in 1869, encapsulated a description of it in its name: "amyotrophic" means no muscle nourishment; "lateral" refers to an area of the spinal cord where portions of the dying motor neuron cells are located; and "sclerosis" is the hardening or scarring that occurs as the process of neural degeneration unfolds. Despite Charcot's straightforward characterization, nearly a century and a half later the complexity of ALS continues to confound researchers. Although the disorder is invariably fatal, for unknown reasons roughly 10 percent of patients survive for more than 10 years, and some do so even longer. That minority includes physicist Stephen Hawking, who has famously lived with ALS for more than five decades. Current research suggests that environmental factors play only a small role in triggering ALS, probably by increasing the vulnerability of individuals who are already genetically susceptible. Most puzzling is that the disorder occurs largely at random. Fewer than 10 percent of cases arise from genetic traits passed down from one generation to the next within a family. The remaining cases are classified as uninherited, or sporadic.

During the past decade sophisticated sequencing technologies have led to exponential growth in our understanding of the disorder's underlying biology. Ongoing research indicates that many different genes, acting alone or in concert, can increase an individual's susceptibility. Specific mutations have been tied to almost 70 percent of familial cases and approximately 10 percent of sporadic ALS. In turn, this wealth of new genetic data is opening up many promising avenues for better therapy. Gene silencing has emerged as a potential treatment for some forms of ALS, with two drugs that target separate rogue genes slated for clinical trials this year. Meanwhile researchers are identifying telltale biomarkers, including measurable substances in bodily fluids or electrical activ-

IN BRIEF

Amyotrophic lateral sclerosis (ALS), a neurodegenerative disorder commonly known as Lou Gehrig's disease, attacks nerve cells that lead from the brain and the spinal cord to muscles throughout the body.

Sophisticated gene-sequencing technologies have led to a flurry of discoveries revealing the genetic underpinnings of ALS. Ongoing research indicates that changes in any of many different genes increase an individual's susceptibility to the disease.

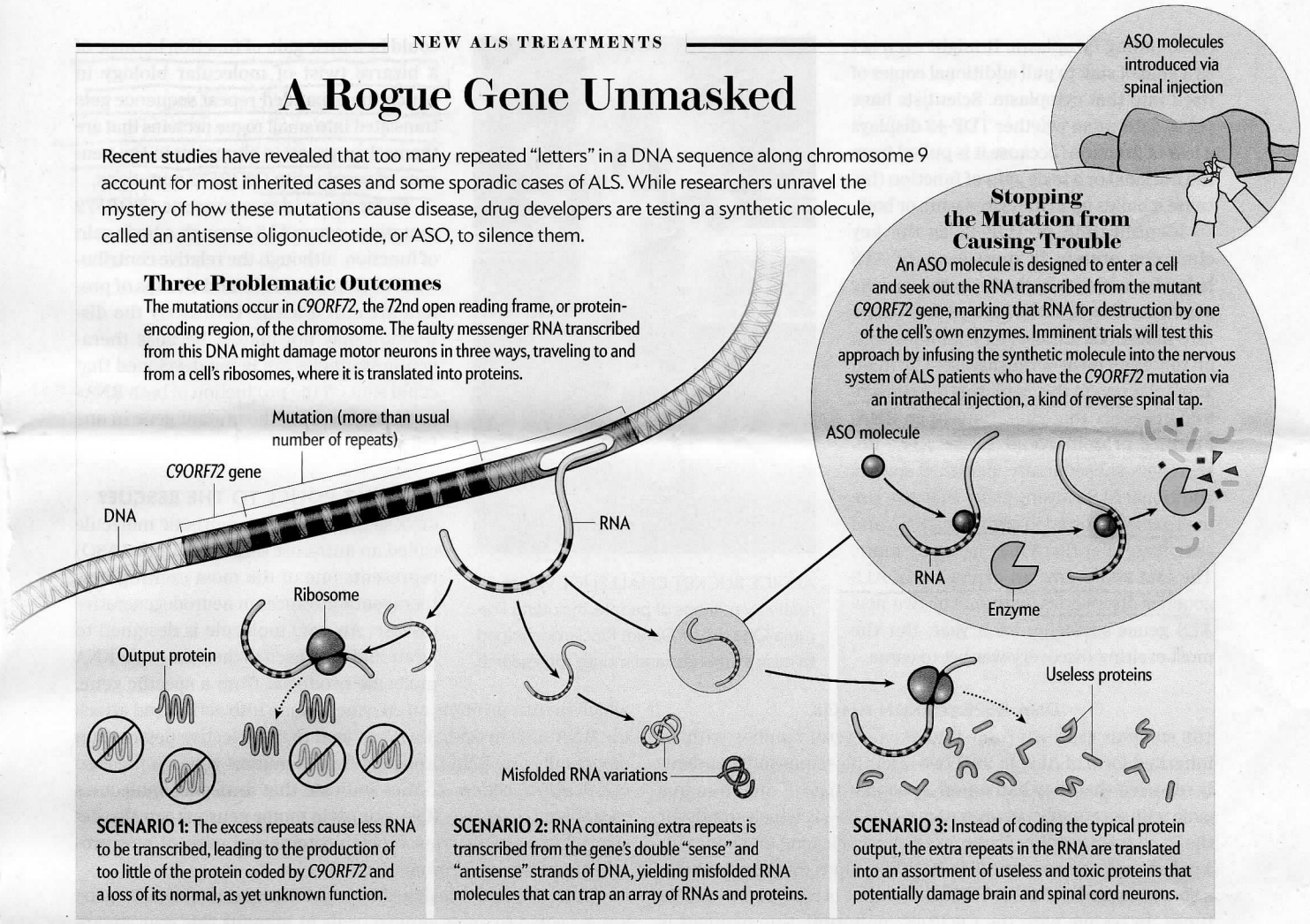
Gene silencing using a synthetic molecule called an antisense oligonucleotide has emerged as a potential treatment for some types of ALS. Researchers are also seeking ways of measuring the disease as it progresses to help with early detection and the development of drug therapies.

page 46-51
As to Dovids Story.

G
RY!

A Rogue Gene Unmasked

Recent studies have revealed that too many repeated "letters" in a DNA sequence along chromosome 9 account for most inherited cases and some sporadic cases of ALS. While researchers unravel the mystery of how these mutations cause disease, drug developers are testing a synthetic molecule, called an antisense oligonucleotide, or ASO, to silence them.



SCENARIO 1: The excess repeats cause less RNA to be transcribed, leading to the production of too little of the protein coded by *C9ORF72* and a loss of its normal, as yet unknown function.

SCENARIO 2: RNA containing extra repeats is transcribed from the gene's double "sense" and "antisense" strands of DNA, yielding misfolded RNA molecules that can trap an array of RNAs and proteins.

SCENARIO 3: Instead of coding the typical protein output, the extra repeats in the RNA are translated into an assortment of useless and toxic proteins that potentially damage brain and spinal cord neurons.

ity in the brain, that could help clinicians make early diagnoses and better gauge the progress of the disease. Such biomarkers may also be useful in the development of other drug treatments.

EARLY GENETIC CLUES

ALTHOUGH PEOPLE WITH FAMILIAL ALS, most of whom have a 50 percent chance of passing it to the next generation, make up a small portion of ALS sufferers, they have played an outsize role in helping to unravel the genetic underpinnings of the disease. The first genetic link to ALS came in 1993 from studies that identified a mutation in a gene called *SOD1* in approximately 20 percent of familial ALS cases. *SOD1* codes for the antioxidant enzyme superoxide dismutase, which converts the highly reactive molecule superoxide—an oxygen free radical—into less damaging forms.

Researchers initially theorized that the mutation in *SOD1* might weaken the enzyme's antioxidizing capabilities and thus allow oxygen free radicals to wreak havoc on motor neurons. A quarter of a century later we have learned with near certainty that that is not the case. Rather it seems that this mutation triggers what scientists call a toxic gain of function, in which the enzyme does something beyond what it is normally supposed to do.

In particular, the new function leads to changes in the shape of certain proteins in neurons. Most autopsies of people with ALS reveal a typical pattern of brain pathology: clumps of proteins accumulated within motor neurons. For these neurons to function optimally, the protein building blocks inside the cells must be recycled efficiently; with ALS, that recycling system breaks down. All proteins, including enzymes, need to adopt precise three-dimensional shapes as they are synthesized in cells if they are to work properly. Researchers eventually discovered that mutations seem to cause individual proteins to fold improperly and then clump together. Cells tag these misshapen proteins with ubiquitin, a molecular marker, which signals that they need to be removed. When this cellular disposal system becomes overwhelmed, the trash builds up. In people with certain types of familial ALS, motor neurons are littered with clumps of aberrant SOD1 proteins tagged with ubiquitin.

A major breakthrough in ALS research occurred in 2006, when scientists looked at cases of ALS without *SOD1* mutations. In virtually every one, they discovered that another protein, called TDP-43, also clumps within motor neurons. TDP-43 belongs to a class of proteins that regulate the activity of messenger RNAs—mobile copies of DNA that serve as templates for making the proteins encoded by a gene's DNA "letters." TDP-43 binds to a messenger RNA, guides its processing in the nucleus, transports it to where it needs to go in the cell and performs other functions important for "translating" the RNA into a protein. Somehow in ALS, the TDP-43 protein gets pulled out of the nucleus and starts accumulating in the

Illustration by Jen Christiansen

surrounding cytoplasm. It might even act as a kind of sink to pull additional copies of itself into that cytoplasm. Scientists have yet to determine whether TDP-43 displays a loss of function (because it is pulled from the nucleus) or a toxic gain of function (because it builds up in the cytoplasm), or both.

Identification of TDP-43 as the key clumping protein in most cases of ALS helped geneticists home in on the specific gene encoding it, *TARDBP,* and they found rare mutations among some families with an inherited form of the disease. The main game changer in this work was the conceptual discovery that alterations in an RNA-binding protein could cause ALS. Researchers subsequently identified several additional ALS-causing genes that give rise to proteins involved in regulating RNA and anticipate that there may be many more. The late 2000s saw an explosion in ALS genetics discoveries, with one or two new ALS genes surfacing each year. But the most exciting discovery was yet to come.

ALS ICE BUCKET CHALLENGE videos made by millions of people, including Formula One driver Daniel Ricciardo, helped to raise awareness and money for research.

DNA REPEATS RUN AMOK

THE FINDINGS EMERGED from studies of several families with an inherited form of ALS. In 2011 two scientific teams independently reported that they had found a peculiar type of mutation in a gene with an equally peculiar name—*C9ORF72,* which stands for the 72nd open reading frame, or the part of a gene that codes for a protein, on chromosome 9. In healthy people, this gene includes a short sequence of DNA—GGGGCC—that is repeated two to 23 times. In people with the *C9ORF72* mutation, this segment is repeated hundreds or sometimes thousands of times.

Subsequent research revealed that these excessive repeats could explain 40 to 50 percent of familial ALS cases and 5 to 10 percent of seemingly sporadic cases. Intriguingly, the discovery of the mutations provided a genetic connection between ALS and another disease, a form of dementia called frontotemporal degeneration (FTD). FTD is marked by changes in personality and decision making. *C9ORF72* mutations can cause ALS or FTD, or even a combination of both called ALS-FTD. And clumps of that ever present TDP-43 protein build up in the neurons of people with *C9ORF72* mutations, providing yet another connection between the two disorders. This association implies that ALS and FTD might be part of a spectrum of related conditions, although how mutations in the same gene would lead to such divergent symptoms is unclear.

Researchers are investigating three cellular mechanisms that might explain how the mutations in this mysterious gene cause ALS. The repeating DNA segment could interfere with the way the genetic code is normally copied into messenger RNA and then translated into C9ORF72 protein, decreasing the amount of protein synthesized. This decrease could diminish the protein's effects, although its exact function is still unknown. Alternatively, there could be a toxic gain of function: perhaps the repeating sequence causes the RNA itself to form clumps that build up in the nuclei of neurons and act like a sink, trapping RNA-binding proteins and preventing them from going about their usual business. Or there could be a toxic gain of function because of a bizarre twist of molecular biology in which the expanded repeat sequence gets translated into small rogue proteins that are themselves prone to clumping in the neurons of people with *C9ORF72* mutations.

So far the evidence suggests *C9ORF72* mutations cause ALS through a toxic gain of function, although the relative contributions of clumps of RNA and clumps of protein are still unclear. Ultimately the distinction may not matter, because therapeutic strategies are being developed that could shut off the production of both RNAs and proteins from the mutant gene in one fell swoop.

REPEAT POLICE TO THE RESCUE?

GENE SILENCING using a synthetic molecule called an antisense oligonucleotide (ASO) represents one of the most exciting new therapeutic advances in neurodegenerative disease. An ASO molecule is designed to locate and bind itself to the messenger RNA molecule produced from a specific gene, which in turn prompts an enzyme to snap into action and attack the RNA-ASO hybrid. ASOs can lead to the selective destruction of virtually any RNA produced from a mutant gene. In the case of *C9ORF72,* rodent studies indicate that antisense molecules engineered to destroy RNA clumps in motor neurons can also destroy clumps of aberrant repeat proteins and prevent new protein clumps from forming.

Antisense drugs designed to target the mutant *C9ORF72* gene are expected to enter clinical trials in humans this year. Meanwhile researchers have also designed an antisense agent for the familial form of ALS caused by *SOD1,* and results of an initial clinical trial indicate it is safe to inject into the fluid-filled space of the spinal column, a site chosen to allow the drug to travel through the cerebrospinal fluid that flows around the brain and to find its way into motor neurons.

The success of an ASO developed for another neurodegenerative disease, called spinal muscular atrophy, gives researchers cause for cautious optimism. This genetic motor neuron disease in infants is similar to ALS. Very few children who suffer from it live past their third birthday. In two recent clinical trials of an antisense drug designed to correct a gene defect that leads to abnormal messenger RNA, children with spinal muscular atrophy showed such dramatic improvement in their motor skills that the FDA fast-tracked those trials and gave formal approval for the drug in late December 2016.

SOLVING SPORADIC ALS

STUDIES OF RARE FORMS of ALS with a clear familial inheritance pattern have paved the way for a better understanding of the underlying biology of the disease. The biggest challenge going forward is to identify mutations in the genomes of individuals with sporadic ALS that make them susceptible to the disease. Efforts are under way around the world to collect DNA samples from people with ALS and to scour their genomes for data.

MARK THOMPSON Getty Images

SCIENTIFIC AMERICAN ONLINE Read about how Stephen Hawking lives with ALS at ScientificAmerican.com/jun2017/als-genes

To expedite this task, geneticists have developed a microchip that lets them conduct so-called genome-wide association studies (GWASs) to readily compare the genomes of people with ALS with those of healthy people. The chip focuses on genome regions known to have variants called single nucleotide polymorphisms—places where a DNA letter, or nucleotide, can vary from one person to another. GWASs are correlational and thus cannot reveal whether something is causing ALS, but they can identify suspect discrepancies that warrant closer examination. Several recent international efforts to perform GWASs of more than 10,000 people with ALS and more than 20,000 healthy people uncovered a number of genomic differences that are now under investigation. New technologies have also simplified the process of collecting genetic data, making it possible to sequence an individual's entire genome in one day for less than $1,000. It takes even less time and money if you sequence only the exome, the part of the genome that codes proteins.

Once researchers have assembled a comprehensive catalog of genetic variants associated with a predisposition for ALS, they will attempt to decipher the complex ways in which ALS-related genetic mutations increase the risk of disease. That attempt will include studying how various genes interact and investigating whether multiple mutant genes might be involved in some forms of ALS, as well as considering how environmental factors might help trigger the disease in some people. Some new studies suggest that ALS may even result in part from the reawakening of a dormant retrovirus—a viral DNA sequence that long ago inserted itself into the genome and normally would have sat quietly. It may be that a retrovirus in some people with ALS jumps from neuron to neuron in the brain, potentially causing damage and initiating the disorder in its wake.

PROMISING NEW LEADS

A GROWING BODY of research suggests that ALS is not merely a disease of dying motor neurons. So-called glial cells, which are even more abundant in the brain and the central nervous system than neurons, may also play an important role. Glial cells perform a variety of functions: some provide physical support for neurons; others regulate the internal environment of the brain, especially the fluid surrounding neurons and their synapses. Recent studies of mice with the *SOD1* gene mutation produced a surprise. Shutting off synthesis of the mutant gene in glial cells prolonged life despite the continued presence of toxic SOD1 protein in the animals' motor neurons. It appears that ALS may originate in the motor neurons but that communication with glial cells helps to drive the progression of the disease. Glial cells might also contribute to ALS by producing a toxic factor, although scientists are not exactly sure of what that factor is or how it works. Once the factor (or factors) is identified, ways to block its production or hinder its ability to transmit its bad signal to motor neurons could be developed to slow or halt ALS.

Amid the quest to unravel the myriad causes of ALS, researchers have also been scrambling to identify biomarkers that can help doctors assess the progress of the disease. For example, ongoing efforts aim to detect the abnormal repeat proteins made from that *C9ORF72* DNA expansion in easily accessible body fluids, such as the blood or spinal fluid. In March one of us (Petrucelli) reported that he had detected these proteins in the cerebrospinal fluid of people with ALS and ALS-FTD—as well as in asymptomatic carriers of the mutated gene. Such measurements could potentially aid in early diagnosis. Other biomarker research is focusing on developing imaging techniques to help detect the TDP-43 protein clumps that build up in the brains of people with ALS before these aggregates start to kill motor neurons. All these biomarkers could also serve as useful benchmarks to judge the success of possible therapies in clinical trials.

The rapid advances taking place in genetics and genomics, as well as the development of new and improved biomarkers, will usher in an era of precision medicine for ALS. In the near future, patients will be grouped together based on the type of ALS they have and will then receive a treatment or preventive tailored to them.

THE POWER OF SOCIAL MEDIA

MUCH OF THE PROGRESS in ALS research during the past decade can be attributed to the willingness of large numbers of individuals afflicted with the disease to volunteer both their time and their DNA to participate in large-scale genomics studies. People with ALS and their families have also helped increase public awareness and canvas funds to support ongoing research and patient services through the power of social media.

The "ALS Ice Bucket Challenge" took the Internet by storm in 2014. Pete Frates, a former captain of Boston College's baseball team who was diagnosed with ALS two years earlier, at age 27, helped to get things rolling when he posted a video on Facebook challenging his friends to dump buckets of ice water over their heads to raise money for the ALS Association. The campaign quickly went viral as a host of celebrities, including Mark Zuckerberg, Bill Gates, Oprah Winfrey, Leonardo DiCaprio and LeBron James, took the challenge. During an eight-week period, Facebook users posted more than 17 million videos of themselves getting drenched for the cause. Supporters ended up raising more than $115 million, of which 67 percent went to research, 20 percent went to patient and community services, and 9 percent went to public and professional education.

ALS is a relentlessly cruel disease. Before Gehrig's stirring retirement speech at Yankee Stadium—in which he famously referred to himself as "the luckiest man on the face of the earth"—and news of his diagnosis spread, most people who contracted the disease suffered in silence. But now public awareness continues to grow, in part because of people like Frates. The social media campaign he helped to spark revitalized the ALS Association, which has since tripled its annual budget for research. Scientists are optimistic that the explosive growth in our understanding of ALS biology will continue and that casting an ever widening dragnet for rogue genes will lead to better therapies for holding this stealth killer at bay. SA

MORE TO EXPLORE

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FROM OUR ARCHIVES

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