National Ataxia Foundation

GENERATIONS

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The National Ataxia Foundation (NAF) is pleased to announce that 27 groundbreaking ataxia research studies were awarded funding at the December 2016 NAF Board of Directors for fiscal year 2017. The grantees represent work from the United States, Canada, Germany, the Netherlands, Portugal and Australia. With the funding of these 27 research studies over $1.1 million dollars were committed in 2016 for ataxia research that will take place in 2017.

The results from this year's studies will help forge a pathway for many scientific breakthroughs, including:

- Removing or modifying disease-causing proteins in order to prevent ataxia
- Understanding how abnormalities in particular genes cause ataxia symptoms
- Stopping the death of important motor neurons called Purkinje cells—so ataxia can never develop
- Identifying innovative biological therapies for ataxia
- Expanding and optimizing the National Ataxia Database

Funding for these ataxia research projects was made possible through the generosity of the NAF donors and partners who contributed to the 2016 NAF Annual Ataxia Research Drive. The National Ataxia Foundation extends its heartfelt gratitude to all who support these studies, our organization and our mission. It is through your generosity that enables the NAF to continue to fund cutting-edge research studies that brings us closer to ending ataxia.

Thank you!

Inside This Issue

The National Ataxia Foundation is pleased to announce that our researchers worked with a science writer so that their lay summaries would be better understood by non-scientists. We hope you enjoy reading how your research donations will provide more answers as we seek treatments and a cure for ataxia.

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National Ataxia Foundation
Connecting Ataxia families, researchers, clinicians and the community

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Funded Research for 2017

SEED MONEY GRANTS

Role of VPS13D in Ataxia

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Many different genes can be at play in causing ataxia to develop. To date, more than 70 genes are known to cause ataxia when they are mutated. But in many individuals and families, no mutations in known ataxia genes can be found. This finding suggests that many more genes still need to be identified to help these affected families and to fully understand the causes of their type of ataxia.

My team and I have recruited a large nuclear family of 14 siblings, 5 of whom are affected with an adult onset form of ataxia. The ataxia has caused degeneration of the cerebellum, nerve damage in other parts of the body, and issues with eye movement, but it has not caused cognitive impairment. Because neither the parents of the affected individuals nor any of their children are affected, the inheritance of the ataxia appears to be recessive. As a result, we expect to find ataxia-causing mutations in both copies of the gene—the one inherited from father's side and the one inherited from the mother's side.

Genetic analysis has narrowed the location of the gene to a small interval (stretch of nucleotides) on chromosome 1. Using new genetic sequencing methods, we have identified a likely cause of ataxia in this family in that interval on chromosome 1. All of the individuals affected with ataxia have two mutations in the VPS13D gene, but the individuals without ataxia have no or only one mutation. In this study, we will be conducting experiments in hopes of establishing VPS13D as a new ataxia gene. We already have neurons from affected and unaffected family members, and we also have fruit flies that are missing the fruit-fly equivalent of the VPS13D gene.

We will first determine whether neurons generated from affected and unaffected family members show differences in VPS13D compared with normal cells. In the fruit flies, we will then determine if there are neurological impairments in flies that lack the fly equivalent of VPS13D. (We have previously studied flies to demonstrate how ataxia genes present themselves in the human body.) Finally, by comparing how the normal and the mutant versions of the gene function in the fruit fly system, we plan to show that the specific amino acid change found in the human family is damaging. If we find that the mutant form of VPS13D does cause ataxia in humans, we expect to discover that this mutant form will not be as effective as the normal form of VPS13D in the fly system.

Studying Spinocerebellar Ataxia type 36 (SCA36) with genetically modified fruit flies

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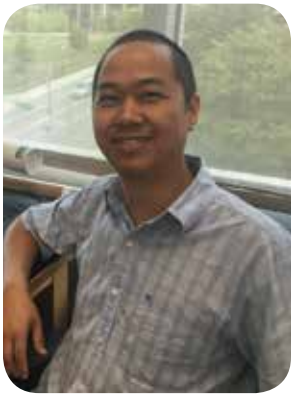

Ataxia of the cerebellum is commonly caused by expanded repeat sequences of DNA—the abnormal over-repetition of DNA sequences. For example, spinocerebellar ataxia type 36 (SCA 36) is a type of cerebellar ataxia caused by a repeat expansion of the six-letter string of nucleotides, GGCCTG. In people without ataxia, only about 3 to 14 repeats of this string of nucleotides appear. But in people with SCA36, the GGCCTG sequence repeats hundreds of times. Although

we know that people with SCA36 have expanded repeats of GGCCTG, no one knows exactly how that repetition damages neurons.

To better determine how expanded repeat sequences lead to SCA36, my team and I will use genetically modified fruit flies (Drosophila melanogastre) that have expressed up to 100 GGCCTG repeats. Through our research, we will learn more about whether these expanded repeats are themselves toxic to the neurons. By learning how these DNA mutations lead to neuron damage, we hope to shed light on how therapies could target and possibly halt the excessive expression of GGCCTG repeats.

Assessing the role of senataxin in cellular inflammation, gene regulation, and innate immunity in Setx -/- mice and a human neuronal model

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Ataxia oculomotor apraxia type 2 (AOA2), a progressive form of cerebellar ataxia, is a neglected rare neurological disorder that develops mostly in late adolescence to early teens. AOA2 occurs from mutations in the SETX gene. The SETX gene encodes the senataxin protein, which plays a key role in the response to DNA damage and regulation of gene expression. Although researchers have described many mutations in the SETX gene, we know very little about the mechanism of disease progression, and no specific treatment exists for AOA2. Currently, the primary way to manage the disease is to provide supportive care (care that does not treat the disease, but instead keeps the patient comfortable).

In 2015, while collaborating with Dr Ivan Marazzi at Columbia University, our research team described an unanticipated role that senataxin plays in controlling innate (natural) immunity, the part of the immune system that responds immediately when a toxin or other foreign substance appears in the body. The involvement of senataxin in innate immunity offers new insight into a possible link between neurodegenerative disorders and inflammation. This finding provides a new framework to explore more fully the possibility that infection and a de-regulated innate immunity may contribute to the development of AOA2—and potentially other neurodegenerative disorders. The purpose of this research project is to assess the role of senataxin in cellular inflammation, gene regulation and innate immunity. Our aim is to gather more knowledge of the how AOA2 begins and then progresses. Specifically, we want to better understand the molecular changes that are involved in AOA2—by (1) further narrowing down the role of senataxin in innate immunity and (2) identifying critical genetic and cellular pathways that are involved in the development of the disease. Advancing an understanding of the cellular function of senataxin and its role in the disease process for AOA2 will be key as researchers help develop effective therapy for patients who have AOA2.

Launching the US-Europe Neuroimaging Partnership in SCA

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A major challenge in developing therapies for spinocerebellar ataxias (SCAs) has been the lack of highly accurate imaging markers for (1) detecting changes due to SCA in the cerebrum and cerebellum and, consequently (2) evaluating the effectiveness of treatments being studied. Although researchers have developed clinical scales that can be used to assess response to treatment, they have several limitations. One important limitation is that results can be highly variable. Therefore, additional non-invasive imaging tests are still needed to measure the direct effects that potential treatments have on the brain. In this project, we will bring together two ongoing efforts in the United States and Europe to validate imaging markers for SCA3.

SCA3 is the most common SCA in the world and affects about 30% of all families with a history of dominant ataxia. Currently, there is no causal treatment for SCA3. However, as we continue to understand the way that SCA3 progresses at the cellular level in the body, several new treatment approaches are being or will be tested in clinical trials. To ensure strong outcomes from these trials, we wish to take advantage of an ongoing, NIH-supported imaging study that focuses on chemical markers of SCA3. We intend to add a magnetic resonance (MR) imaging component to that study so we can get detailed structural and functional MR data, which will also be part of an ongoing European study called the European Spinocerebellar Ataxia Type 3/ MachadoJoseph Disease Initiative (ESMI). Funds from the NAF Research Grant will allow us to initiate a US-Europe imaging collaboration that is long overdue. This collaboration can help validate noninvasive MR biomarkers for upcoming SCA3 trials taking place in many sites throughout the world.

Ataxia team at the Center for Magnetic Resonance Research
Pierre-Gilles Henry, Dinesh Deelchand, Diane Hutter, Gulin Oz, Lynn Eberly, Sarah Larson, Christophe Lenglet, James Joers

Web-based National Ataxia Database

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The National Ataxia Registry, the National Ataxia Database, and the Ataxia Tissue Donation Program were formed to provide the infrastructure for clinical research in the ataxic disorders. They enabled ataxia researchers to notify ataxia patients of upcoming research projects, to store and analyze data from those projects, and to examine tissues from ataxia patients to find out how ataxia develops and how the body responds to it.

The web-based National Ataxia Database is currently housed on the UCLA computer servers, and over the years since its development, has provided natural history database support to the UCLA Ataxia Clinic, as well as to the Ataxia Clinic at Johns Hopkins University. Other "Ataxiologists" in California, Arizona, Nevada, and Colorado have expressed interest in using it as well. It has begun to provide a platform to support and join specialists in clinical care and clinical research of ataxia. It will ultimately assist all members of the Ataxia Clinical Research Consortium in future collaborative endeavors in clinical research and in setting standards for clinical care.

Following the end of funding from the National Institutes of Health for the Rare Disease Network, with of the help of the NAF "bridge" grant, we were able to continue to import the existing data of the natural history study into the National Ataxia Database. This allowed us to continue enrollment and follow-up of participants in this important study of SCA l , 2, 3, and 6. Data collection will begin on participants with SCA 7, 8, and 10. There are now 13 sites contributing to this project and six more will be added. Close to 500 participants have been enrolled and are pursuing natural history examinations and banking of specimens.

The National Ataxia Database will also be open for ataxia researchers to "bank" other clinical data collected either in the individual's private data docks (not accessible to other ataxia researchers) or in data docks shared by several researchers (e.g., for a proposed project to look at coded clinical data on people with sporadic ataxia). Templates will be added for scales to measure fatigue, dizziness, cognition,

and neuropsychiatric symptoms. The National Ataxia Database is an essential tool for the Clinical Research Consortium for the Study of Cerebellar Ataxia.

New therapeutic approaches for Machado-Joseph Disease: Chaperoning protein self assembly

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Spinocerebellar ataxia type 3 (SCA3), also called Machado-Joseph disease, is a rare neurodegenerative disease. SCA3 is caused by an expanded stretch of CAG triplets in the affected gene. The overrepetition of CAG causes a protein called ataxin-3 to form abnormally—and in a way that is toxic to neurons.

For the past 20 years, researchers have made impressive progress in understanding the cellular functions and structural details of ataxin-3, but so far no specific treatments are available for SCA3. We need to better understand the three-dimensional structure of ataxin-3 to fully determine its function and dysfunction at the molecular level. By understanding more clearly how ataxin-3 becomes toxic, we can also learn how to develop targeted therapies. We propose to study a number of synthetic molecules that can bind and reshape ataxin-3 into a non-toxic form. If we can determine how ataxin-3 is shaped with atomic precision, we can hopefully identify which molecules can serve as the best therapy to treat the dysfunction.

Calpain-mediated proteolysis in Machado-Joseph disease

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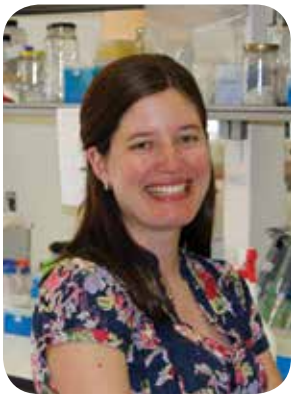

Machado-Joseph disease (MJD), also known as spinocerebellar ataxia type 3 (SCA3), is the most frequent worldwide autosomal dominantly-inherited ataxia, which means that affected individuals have a 50% chance of transmitting the affected gene to their children.

In MJD, a mutation leads to a bigger-than-normal stretch of polyglutamine at the ataxin-3 protein. The ataxin-3 protein is a biological molecule that is important for cellular quality control. Researchers believe that ataxin-3 is cut into smaller fragments. Our research team and others have recently shown that the molecules responsible for the breakdown of ataxin-3 are called calpains. Calpains form toxic fragments, move the ataxin-3 from the cytoplasm to the nucleus, and contribute to degeneration of neurons.

The aim of this project is to understand, at a cellular level, calpains' contribution to the development of MJD. To achieve this aim, we will evaluate how genes encode the calpain system, determine in which places ataxin-3 is cut, and how calpains are activated according to the different brain regions, cell types and timeline for disease progression. Moreover, because no current treatment is available, we will evaluate whether a novel calpain inhibitor can reduce cell injury and alleviate loss of motor coordination. These expected results could be used to develop a therapy for MJD patients in a short-time frame. The results can

DRESS DOWN DAY

The staff at the Joseph Case Junior High School ran a "Dress Down Day" with the help of the Student Leadership Club. This month's "Dress Down Day," was earmarked for NAF and the event raised **more than $150**.

also be used to identify biological measures, or markers, of MJD progression or treatment response. Furthermore, understanding the calpains' role in MJD can inform our understanding of other ataxias that are vulnerable to calcium deregulation.

Whole Exome Sequencing (WES) in the Diagnosis and Management of Atypical Childhood Hereditary Ataxia Conditions

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The National Ataxia Foundation (NAF) was formed by John and Henry Schut in 1957 to promote awareness of the degenerative ataxias, support research into the causes and cure of the disease, and provide support and comfort to ataxia patients and their families. We are resolved to advance these primary objectives through our project "WES in Atypical Childhood Ataxia."

New technologies have drastically changed the way we diagnose rare diseases. The human genome contains about 3 billion bases or letters. For over a decade, researchers have had the ability to read a person's entire genome through a process called whole genome sequencing (WGS). However, we were not able to translate the DNA code into information that we could meaningfully use (for example, for treatment or predicting risk of disease). Now, through testing known as whole exome sequencing (WES), we can now focus in on the "coding" portion of the genome, called the exons, that provides instructions for making proteins. WES methods allow the body's entire set of instructions—or exome—to be examined as a single laboratory test—rather than having to individually analyze all 20,000 genes that make up our exome. Through WES, we are able to look for "spelling mistakes" (known as pathogenic mutations) in gene(s) and then determine if these changes are the cause for the person's illness.

With this innovative tool, we aim to enroll and offer WES to 12 children with severe or complex ataxia in order to (1) discover new genes that cause ataxia, (2) expand what we understand about the clinical picture of known human genes that cause ataxia, (3) make sense of these rare genetic ataxic conditions at a microscopic "molecular" level so that we can understand, target, treat and potentially cure these disorders. We plan to achieve these goals by merging our center's unique expertise in combining deep characterization of a patient's clinical picture (combined physical, neurological and metabolic symptoms) with expertise in WES analysis. Our hope is that this focused study of ataxic cases at our center will positively affect the lives of patients and families, forge the discovery of new genetic ataxia disorders, expand descriptions of known conditions and create new treatment opportunities.

Intensive home-based speech rehabilitation for adults with degenerative ataxia

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Loss of ability to speak is a devastating and inevitable outcome of many neurodegenerative diseases. When people lose their ability to speak, they lose their ability to carry out basic tasks. They can be stigmatized and marginalized, and they often have challenges with employment. In time, they experience a decrease in their quality of life. Hereditary ataxias cause a lack of coordination with gait, speech, and eye and hand movement. No therapies are currently available to stop degenerative ataxia from progressing. However, therapy to improve speech is within reach.

My team and I have conducted a successful pilot study that draws on (1) the principles of motor learning and biofeedback and (2) the expertise of a team that specializes in movement therapy and ataxia. We have

designed a home-based, intensive 4-week speech exercise program designed to improve speech in patients with hereditary ataxia. The treatment focuses on improving intelligibility and vocal control. We created exercises and feedback to enhance individuals' ability to monitor their own speech abilities and improvements. The program is suitable for use in a clinical setting so it can readily be brought into clinical practice. It can also potentially be adapted for a range of other progressive neurological disorders. Funding from the National Ataxia Foundation will be used to develop a controlled, well-designed study that can assess the effectiveness of this program.

YOUNG INVESTIGATOR AWARDS

Reduced expression of mitochondrial aldehyde dehydrogenases contributes to metabolic stress in Friedreich's ataxia

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Friedreich's ataxia (FRDA) is a severe form of ataxia caused by decreased production of a protein called frataxin. In patients who have Friedreich's ataxia, the loss of frataxin protein eventually damages multiple organs, including the heart and pancreas. Cells in the heart and brain are the most sensitive to changes in frataxin levels. Even though frataxin is just a single protein, lower levels of it can cause other cell proteins and genes to stop working properly. The goal of this study is to get new information about how and when frataxin causes those cell and gene changes. As we learn more about those changes, we hope to narrow down which particular signs, or biomarkers, show that Friedreich's ataxia may progressing in the body.

Results from previous research my team and I have conducted show that Friedreich's ataxia cells have decreased levels of particular enzymes called mitochondrial aldehyde dehydrogenases. The job of these enzymes is to rid cells of certain toxins that can damage heart and brain cells. In this study, we will be further evaluating the levels and activities of mitochondrial aldehyde dehydrogenases in Friedreich's ataxia cells. In a controlled setting, we will be changing the level and activity of these enzymes to determine how they affect the health and growth of Friedreich's ataxia cells. In doing this work, we hope to better understand what level of mitochondrial aldehyde dehydrogenase activity is needed to prevent or reverse damage in Friedreich's ataxia cells. Our findings could help pave the way for new therapies that deliver the right amounts of these protective enzymes into the Friedreich's ataxia cells—so the ataxia can be treated effectively.

Pumilio1 deficiency: understanding a new ataxia gene and its role in cerebellar dysfunction in mice and humans

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The molecular genetic revolution of the 1990s brought us tremendous knowledge of the genetic mutations that cause many neurological diseases, including many ataxias. Further research into the proteins produced by these genes has revealed that there is another way for a protein to cause havoc in the brain besides being mutated; it might be expressed at levels too low or too high. In the case of several neurodegenerative diseases, including spinocerebellar ataxia type 1 (SCA1) and more common diseases such as Alzheimer's and Parkinson's, we have discovered that too much of even the normal version of the disease-driving protein can cause disease. What if we could find a way to lower the levels of these proteins in the brain? Could we slow disease progression? These questions led me to search for the factors that

regulate the levels of ataxin1, the protein that is involved in SCA1. I discovered that ataxin1 levels are controlled by an RNA-binding protein called Pumilio1 (PUM1). More importantly, we showed that if you take away Pum1 (the mouse version of the protein is written in lower-case letters) in a mouse model of SCA1, and ataxin1 returns to normal levels, the SCA1 mice no longer have ataxia.

We also noticed, however, that mice lacking Pum1 were quite sick: they developed seizures, and they developed ataxia earlier than the SCA1 mice. This led us to suspect that loss of PUM1 function might be the culprit behind some childhood ataxias. In collaboration with medical geneticists around the world, we have identified seven patients with deletion of PUM1 and two patients with mutations in PUM1 who show symptoms similar to the Pum1 mutant mice. This finding confirms our hypothesis that PUM1 deficiency is a genetic cause of an early-onset ataxia syndrome.

In this study, we aim to characterize the phenotype of Pum1 mutant mice as precisely as possible and study the effects of loss of function of PUM1 in patient-derived cell lines. This research is necessary to understand how PUM1 deficiency causes childhood ataxias and neurological dysfunction. Our research will also help us understand all the targets of PUM1's activities in neurons so we can learn whether altering PUM1 levels in the brain could help patients with SCA1 or those with other neurodegenerative diseases.

Transcriptional activation using CRISPR/Cas mutant proteins as a novel therapy for Frataxin gene silencing

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The underlying cause of Friedreich's ataxia is the insufficient production of frataxin. The faulty gene that produces insufficient amounts of frataxin contains a GAA mutation. Interestingly, those who have one copy (carriers) of this faulty gene also show up to a 50% reduction in the amount of frataxin protein, but they do not develop any symptoms of Friedreich's ataxia. Furthermore, the frataxin produced from a faulty copy of the frataxin gene functions as normal.

These facts suggest that by increasing levels of frataxin protein to the amount that carriers produce, we may be able to hinder the progression of the disease.

This project sets out to increase the production of frataxin by addressing the problem at its source. Because of the presence of the GAA mutation, a cell struggles to access and read the information within the gene. This is because the environment (called the epigenome) in which the gene is now found does not permit the "readers" of the DNA to pass along it. This project will investigate the possibility of improving a cell's ability to access and read the gene without affecting other genes.

The project will make use of the new genome-engineering tool CRISPR which allows a protein to be targeted to specific genes, but in a manner in which only the environment surrounding the gene is altered and not the gene sequence itself. We hope that changing the silencing environment around the frataxin gene will provide a radical new approach to treating Friedreich's ataxia.

The National Ataxia Foundation is grateful to our generous year-long sponsors Biohaven Pharmaceuticals and Reata Pharmaceuticals.

Epigenetic Modulation Mediated by RNA Binding Proteins in Neurodegeneration

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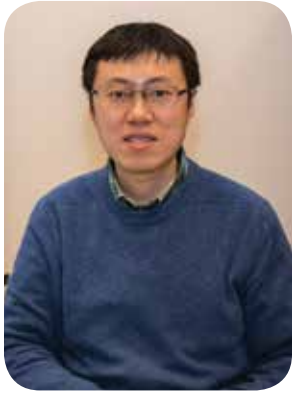

Ribonucleic acid (RNA) molecules, like DNA, are essential information-carriers in all organisms. RNA is particularly important for making proteins in the body. Proteins that bind to RNA play fundamental roles in controlling different aspects of RNA functions which are important for neurons to work properly. Dysfunction with RNA-binding proteins often leads to neurodegenerative disorders that cause ataxia. Fragile X-associated tremor/ataxia syndrome (FXTAS) is a neurodegenerative disorder that usually develops in the late stage of adulthood. It is caused by a 55 to 200 copies of nucleotide "CGG" repeats in the fragile X mental retardation 1 (FMR1) gene. In FXTAS, these extra CGG repeats can restrict the RNA-binding proteins hnRNP A2/B1. This, in turn, leads to the death of critical motor neurons called Purkinje cells which leads to ataxia.

In this NAF Young investigator award application, I hypothesize that RNA-binding protein hnRNP A2/B1 has a novel role to directly bind to DNA and control critical gene expression to influence the life course of Purkinje cells. In the proposed specific aims, I will first study whether and how hnRNP A2/B1 binds to DNA regions of the genome and whether and how restriction of hnRNP A2/B1 leads to Purkinje cell death. I will then use a cutting-edge method called next-generation sequencing to isolate the genuine roles of hnRNP A2/B1 in controlling the expression of genes related to FXTAS. This study will provide novel insights into how RNA-binding proteins help direct the function of normal neuron cells and, conversely, how their restriction can influence the development of ataxia-related disease.

POST-DOC FELLOWSHIP AWARDS

Development and mechanistic study of deep brain stimulation of dentate nucleus for the treatment of degenerative ataxia.

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Degenerative cerebellar ataxias (DCAs), characterized by incoordination of gait, tremor and motor symptoms, affect 1 in 5,000 people worldwide. Many different causes exist, but each combines the progressive death of Purkinje cells, a common neuron in the cerebellum of the brain. Despite the number of people affected by DCAs and decades of research, treatment options are limited. The loss of Purkinje cells is currently irreversible and leads to changes in the way brain cells communicate, so for any treatment to be successful, it will need to partially reverse these communication changes.

Our team believes deep brain stimulation could provide a new form of successful therapy. Using rats that have a loss of Purkinje cells and exhibit tremors and lack of gait coordination, we plan to surgically implant electrodes to repeatedly electrically stimulate targets within the brain and treat DCAs. Deep brain stimulation is frequently used to treat neurological conditions such as Parkinson's disease, essential tremor, and numerous other neurological conditions. The therapy affects the activity of different stimulation targets within the brain in a way that partially restores healthy communication between neurons. The target choice for stimulation in our study will be the dentate nucleus. It is the most important region in the cerebellum for controlling motor activity, and DCAs alter its signaling in a way that leads to motor symptoms. In conjunction with the implantation of stimulating electrodes, we will also implant recording electrodes in the rats to record from numerous neurons

simultaneously. Through these recordings, we hope to determine precisely what elements of neuron signaling directly lead to motor coordination symptoms.

This project represents the opportunity to not only prove the concept of a major treatment opportunity for degenerative cerebellar ataxias, but also to greatly enrich our understanding of the neurological changes that directly lead to associated motor symptoms.

Transcellular regulation of the proteostasis network in Spinocerebellar ataxia type 3

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Spinocerebellar ataxia type 3 (SCA3), also known as Machado-Joseph disease, is an inherited neurodegenerative disorder that is caused by a mutation in ataxin-3. At present, effective treatment is not available for this disease and researchers do not yet know how neuron dysfunction occurs. Ataxin-3 has a role in controlling protein abundance, folding, and transport (called protein homeostasis, or proteostasis) in many tissues, including in cells that are not neurons. Maintaining protein homeostasis in the body is essential because imbalances in any of these processes can pose a danger to cell function and the health of organisms. We are planning to study protein homeostasis in the tissues of worms that have been genetically engineered to have SCA3. Detailed knowledge of cell type-specific events and regulation of protein homeostasis across tissues will not only help to improve our understanding of the disease, but also may lead to new treatments for SCA3—and perhaps other neurodegenerative diseases as well.

Rapid Structure-based lead optimization of a small molecule drug that targets r(CAG)^{exp}

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RNA repeat expansions in spinocerebellar ataxia (SCA) cause degeneration of neurons, which leads to loss of control of body movements. My project will:
(1) Characterize the relationship between the chemical structure of a lead (leading) compound and its ability to target overexpression of "CAG" repeats, which are thought to cause several types of SCA
(2) Optimize dimeric compounds for bioactivity, selectivity and potency.

Dimeric compounds have enhanced affinity for a target RNA over a monomeric compound because binding of one module to the RNA brings a second module within close proximity to the RNA. This increases the chance of the two modules simultaneously binding to the RNA over two separate monomeric compounds.
The compounds investigated in this work will reverse the overexpression of "CAG" repeats associated with these SCAs.

Ataxia with hypogonadotropic hypogonadism due to ubiquitin ligase dysregulation

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Our research team is investigating a disorder called Gordon Holmes Syndrome. This syndrome adversely affects memory, movement and reproduction. Through our team's research, we have identified a gene (RNF216), which, when severely mutated,

appears to cause Gordon Holmes Syndrome. To further explore the biology of RNF 216, we are studying mice who lack this protein. Our team hopes to use this mouse model to understand how abnormalities in this gene affect neurologic and reproductive health. The long-term goal is to develop better therapies to treat this syndrome more effectively.

Identifying Dendro-protective Ion Channels in Cerebellar Ataxia

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Purkinje neurons are important brain cells in the cerebellum, the part of the brain that controls balance and coordination. In cerebellar ataxia, Purkinje neurons often break down and eventually die. This process begins with shriveling of the neurons' dendrites, one of the structures that all neurons use to communicate with each other. Research has shown that the shriveling of Purkinje neuron dendrites play a role in how ataxia symptoms develop. However, researchers do not yet know exactly how and why dendrite shriveling happens.

All neurons show electrical activity, which they need to communicate with each other. This electrical activity occurs through the action of a class of proteins called ion channels. In previous studies, my team and I have shown that major changes in Purkinje cell ion channels occur with spinocerebellar ataxia 1 and 2 (SCA 1 and SCA 2). In particular, we have found that the electrical activity in the dendrites of Purkinje neurons changes and that change could be causing dendrite shriveling. In this new study, we hope to find out which ion channels affect dendrite shriveling. The ultimate goal is to discover news therapies that can halt changes in the ion channels with SCA1 and SCA2–thus reducing ataxia symptoms.

The role of astrocytes in SCA1 via NF-kB pathway

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Spinocerebellar ataxia 1 (SCA1) is an incurable and genetically inherited disease that causes progressive impairment in movement and cognition as well as premature death. Researchers believe the impairment in movement is caused by the death of Purkinje neurons in the cerebellum. However, other symptoms may be caused by degeneration of neurons outside the cerebellum, such as in the hippocampus and brainstem. Our previous results in a mouse model of SCA1 showed that astrocytes may play an important role in SCA1. Astrocytes are not neurons; they are cells in the brain required for the normal functions of neurons. In the brains of mice who have SCA1, astrocytes activate the NF-kB pathway, which causes inflammation that is potentially toxic to cells. Blocking the NF-kB pathway in astrocytes improved movement and the function of neurons in the cerebellum of these mice. Based on these results with the cerebellum, we hypothesize that astrocytes also contribute to cognitive impairment and premature death through NF-kB activation in other brain regions. In this study, we will test this hypothesis by blocking NF-kB in astrocytes and examining the effects on cognition and lifespan, as well as the underlying changes in neurons and astrocytes in the associated brain regions.

Matching Gifts

Please ask your employer if there is a Matching Gift Program. If so, your and your co-workers donations may be doubled to support the work of NAF. Thank you.

Unraveling the mechanisms of motor neuron degeneration in Spinocerebellar Ataxia, type1

James Orengo, MD, PhD • jo044504@bcm.edu Baylor College of Medicine, Houston, TX

Spinocerebellar ataxia type 1 (SCA1) is a devastating neurodegenerative disease characterized by progressive loss of coordination of movements and clumsiness. Individuals affected by this disease typically die between 30 and 70 years of age. Although the majority of scientists study loss of coordination in SCA1, individuals with SCA1 actually die from complications related to weak muscles—not the loss of coordination. In particular, the muscles no longer function well enough for the individuals to breathe properly.

My team and I have developed a mouse model for SCA1 that copies the major symptoms of human disease. Based on my clinical background as a neuromuscular disease expert, I made the exciting observation that these mice display signs of muscle weakness as well. In particular, I noted small and sick muscles, breathing abnormalities and muscle stiffness in adult mice. The goals of my project are to examine the molecular changes in these mice that lead to muscle weakness and early death, so that new treatments can be developed.

REMEMBERING NAF IN YOUR WILL

Throughout the years, individuals have named the National Ataxia Foundation as a beneficiary in their wills. Their thoughtfulness and foresight has enabled the NAF to provide more research studies, more services to patients and families and more education and ataxia awareness to the public. We are grateful for the impact that has been made by these compassionate acts. If this is something you would like to consider, please contact Joel Sutherland at **joel@ataxia.org** or call 763-231-2748.

Aberrant Regulation of Voltage-gated Na channels in the Pathophysiology of Spinocerebellar ataxia 27

Manu Ben-Johny, PhD • manu@jhmi.edu Johns Hopkins University, Baltimore, MD

Spinocerebellar ataxia 27 (SCA27) is a recently identified type of ataxia. Patients with SCA27 experience tremors and difficulties with gait and often perform poorly on cognitive tasks. The genetic basis of this disorder has been localized to mutations within a small protein called Fibroblast growth factor homologous factor 4 (FHF4), which is found within neurons. FHF4 interacts with voltage-gated sodium channels—molecules critical for generation of electrical impulses in neurons. Despite FHF4's important role in the development of SCA27, the effect of FHF4 on the function sodium channels is not yet fully understood. This prominent gap in the defining the basic mechanisms that govern FHF4 action on sodium channels has clouded our understanding of SCA27, including how this debilitating disorder develops and progresses.

Curiously, the sodium channel interface that docks FHF4 also harbors calmodulin, a protein that is found within all eukaryotic organisms, ranging from one-celled organisms to human beings. Calmodulin senses changes in the level of intracellular calcium ions to coordinate the activity of numerous proteins. Thus, it allows cells to attain calcium homeostasis. Our recent work has shown that calmodulin also dynamically reduces the activity of sodium channels in response to increases in cellular calcium levels. This feedback control mechanism allows the cell to reduce generation of electrical impulses in neurons after periods of excess activity. Does FHF4 then regulate sodium channels by overriding calmodulin regulation? Could changes in this interaction then cause changes in electrical properties of neurons as observed with SCA27? To answer these questions, our team will study the interplay between FHF and calmodulin in tuning sodium channel activity within cerebellar Purkinje

neurons. We will also explore whether altered dynamics of intracellular calcium ions may play a role in the development of ataxia.
Overall, this project promises to advance our fundamental understanding of the molecular interactions that are essential to fine-tune neuronal function and to ultimately evoke coordination of movement. We also hope that these studies will help identify important molecular targets for the development of new therapies for ataxia.

Role of astrocyte calcium signaling in the pathogenesis of SCA1

Marija Cvetanovic, PhD • mcvetano@umn.edu
University of Minnesota, Minneapolis, MN

Astrocytes are brain cells that neurons need to work normally. Astrocytes help keep neurons alive by giving the neurons nutrients and oxygen and by helping keep the neurons' environment stable and healthy. The level of calcium within astrocytes affects how well they work.

In spinocerebellar ataxia (SCA1), astrocytes undergo a process called astrogliosis, In astrogliosis, astrocytes increase to above-normal levels as a way to protect the nervous system from further damage.

In this study, my team and I will be using mouse models to study how astrogliosis affects the level of calcium in the astrocytes. Using astrogliosis as a guide, we will learn whether calcium in astrocytes is changed in the presence of SCA1. We will also test whether changing the levels of calcium in the astrocytes helps prevent or lessen the severity of SCA1 symptoms. If we discover that calcium in astrocytes plays a significant role in the development of SCA1, scientists could one day develop therapies that can change those levels of calcium—and hopefully delay development of the disease.

Systematic edgotyping of ataxin proteins in cellular systems from yeast to patient neurons.

Vikram Khurana, MD, PhD • vkhurana@bwh.harvard.edu Brigham and Women's Hospital and Harvard Stem Cell Institute, Boston, MA

The identification of gene mutations that cause neurodegeneration offers tremendous hope for understanding and reversing the way neurodegenerative diseases develop. However, researchers have yet to convert genetic insights into real preventive or disease-slowing therapies for patients. Genes code for proteins—the building blocks, signaling molecules and enzymes of our cells. Ultimately, a very significant result of gene mutations is that they lead to abnormal protein changes. We know that gene mutations involved in neurodegenerative diseases lead to some important alterations in protein folding and function, and we know that the mutated proteins collect in affected brain cells. But a global and systematic understanding of these alterations in living cells—and different types of cells—is lacking. To address this lack of base knowledge, we use an unbiased, efficient method, called edgotyping, to systematically look at how gene mutations alter the interactions that take place between affected proteins and the cells they occupy. The edgotyping method can be applied to simple cells (such as yeast) and complex cells (such as

THE DEADLINE FOR SUBMITTING MATERIALS

for the summer issue of Generations is Friday, May 12. Please send articles, your personal story, recaps of ataxia-related events, photos and reports to **joan@ataxia.org**. Thank you.

brain cells, or neurons—even neurons made directly from patients)—and everything in between.

In this proposal, we will be applying edgotyping methods to examine the ataxin protein mutations that lead to spinocerebellar ataxias. We willl systematically define how ataxin mutations (so-called polyglutamine expansions) change the way proteins interact with living cells. The changes in the protein map that result from ataxin mutations have never been examined using edgotyping. But we do know from previous work that some of these changes are important and can be taken advantage of when developing treatments. We have every expectation that the data and platforms we generate in this project will lead to major insights into the biology and treatment of spinocerebellar ataxias, with important implications for neurodegenerative disease in general.

Alleviation of proteasomal inhibition as a therapeutic approach for SCA3

Jana Schmidt, PhD • Jana.Rostock@gmx.de
University of Tuebingen, Tuebingen, Germany

Spinocerebellar ataxia type 3 (SCA3), also known as Machado-Joseph disease (MJD), is caused by the expansion of a repetitive structure within the protein ataxin-3.

Proteins that are unwanted or no longer required for brain function are marked in our cells by a certain label called ubiquitin. The cell then degrades the ubiquitin-labelled proteins in a shredder-like process. In SCA3 patients, this degradation process is disturbed, leading to the accumulation of the expanded ataxin-3 and other proteins. Studies of brain tissue have identified collections of protein as a hallmark of the disease in SCA3 patients and also in other neurological disorders.

In order to identify the mechanisms leading to SCA3 and to identify ways to prevent the onset of symptoms, we generated mouse models of the disease. Our models allow us to study disease processes in a time-lapse fashion. That means that processes needing decades to take place in man can be studied in a mouse model within months. In our research, we recently studied the process by which ataxin-3 degrades and then applied it to specific mouse models. When we prevented a certain type of ubiquitin label from existing in the mice, we observed that these mice had much less aggregated ataxin-3, did not get the disease and did not develop impaired movement. This means that modulating this ubiquitin label may be a promising therapeutic strategy in SCA3. However, such approaches cannot be directly translated from mouse to man.

In our project, we aim to further understand what's happening inside the cell to lessen the disease in our specific mice. We also hope to figure out whether and how our approach could be translated as a therapy to human SCA3 patients. A successful therapy would help remove the toxic, disease-causing ataxin-3—preventing its aggregation and, consequently, the start of the disease and its symptoms.

PIONEER SCA TRANSLATIONAL RESEARCH GRANTS

Testing the therapeutic potential of mesenchymal stem cells and their secretome in an animal model of spinocerebellar ataxia type 3

Patricia Maciel, PhD • pmaciel@med.uminho.pt
University of Minho, Braga, Portugal

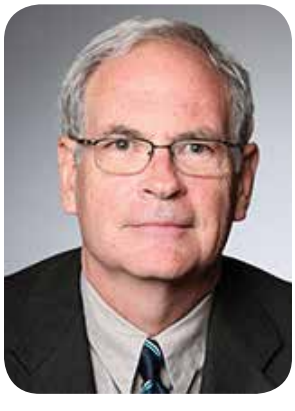
Mesenchymal stem cells are cells that come from several tissues, including bone marrow. Research has revealed that these types of cells have a great potential to regenerate damaged organs and tissues. As a result, they are being tested as biological therapy agents against neurodegenerative diseases. Although the

effect of the cells has been studied in several neurodegenerative diseases—including ataxias—with promising results, no pre-clinical studies have been done for spinocerebellar ataxia type 3 (SCA3), also known as Machado-Joseph disease (MJD).

The goal of this project to study the effectiveness of using either mesenchymal stem cells or biological products derived from these cells to treat SCA3/MJD. For this research, we will evaluate the short-term and long-term effect of these treatments on mice that have SCA3/MJD and have impairments in movement. The tests will measure balance, strength and movement coordination. About 6 months after the stem cells or their biological products have been given to the mice, we will also look at the effectiveness of the treatment in reducing the death of neurons in different areas of the mice's nervous system. Finally, we will compare different protocols, including different injection sites, to determine which one has a higher effect. In total, the findings of this research should provide important proof-of-concept information for future clinical studies of mesenchymal stem cells in patients with SCA3/MJD.

Towards an ASO Therapy for Spinocerebellar Ataxia Type 1

Harry Orr, PhD • orrxx002@umn.edu
University of Minnesota, Minneapolis, MN



Spinocerebellar ataxia type 1 (SCA1) is a fatal inherited form of ataxia that currently has no effective treatment. Studies using mouse models of SCA1 have shown that reducing expression of the SCA1 gene can reduce SCA1-like symptoms, including dysfunction in movement and premature death. Our team's research has shown that SCA1 gene expression can be reduced with a drug called an antisense oligonucleotide (ASO). In mice who have SCA1, treatment with an ASO relieved SCA1-like symptoms. Assessing whether therapies are effective in slowing neurodegeneration is difficult using only clinical scales because SCA1 progresses slowly in the body, and it presents in many different ways. Thus, development of additional ways to follow progress of SCA1 might speed moving a drug to clinical trials. To do this, we will use magnetic resonance spectroscopy neuroimaging to examine the brains of the mice before and after they receive the ASO therapy. Our hope is that our research will inform a future pathway for bringing effective ASO therapy to patients with SCA1.

Advancing the therapeutic potential of exon skipping for Spinocerebellar ataxia type 3

Willeke M.C. van Roon-Mom, PhD • w.vanroon@lumc.nl Leiden University Medical Center, The Netherlands

Spinocerebellar ataxia type 3 (SCA3) is a hereditary form of ataxia where the area of the brain called the cerebellum is most affected. SCA3 is caused by overrepetition of CAG sequences in a specific region of the DNA. In healthy individuals, up to 51 repeats of CAG appear, but in people with SCA3, the CAG repeats increase to 51 times or more. Ataxin-3 is an important protein in the brain but becomes toxic with this overrepetition of repeats.

My team's research has shown that we can remove specifically the region of the ataxin-3 protein that contains the overrepetition by using molecules called antisense oligonucleotides (AONs). In doing so, we are able to directly remove the cause of SCA3 without reducing the amount of ataxin-3 protein. Maintaining ataxin-3 protein levels in the body is key because ataxin-3 seems to be an important protein for normal brain function. When testing the treatment in mice, use of the AONs was well tolerated and effective. To bring the treatment closer to use in patients, we will be further testing the AONs to find the ones that best targets the cause of the SCA3. We will also be testing cultured cells to determine that it is safe to use in humans.

Tissue donations for research in Friedreich ataxia

If you have been diagnosed with Friedreich ataxia and wish to contribute to its eradication by helping research, please consider donating your tissues after death. To do so, contact Dr. Arnulf H. Koeppen for detailed information. Tissues affected by Friedreich ataxia are brain, eyes, spinal cord, dorsal root ganglia, sensory peripheral nerves, heart, and the insulin-producing beta-cells of the pancreas.

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Friedreich Ataxia Patient Focused Drug Development (PFDD) Meeting

Tell the FDA what is important to you in finding a treatment for Friedreich Ataxia

An upcoming Friedreich ataxia (FRDA) Patient Focused Drug Development (PFDD) meeting with the U.S. Food and Drug Administration (FDA) is your opportunity to tell FDA and drug developers about challenges and burdens you have experienced with FRDA, and share your thoughts about what is most important to you in evaluating potential new treatments for the disease.

The meeting, co-organized by the National Ataxia Foundation, Friedreich's Ataxia Research Alliance, and Muscular Dystrophy Association, marks the first-time patients and families affected by FRDA will be able to speak directly to the FDA and share their experiences in their own words.

Information captured at the meeting, summarizing input about the patient experience from people with FRDA across the country, will be published in a "Voice of the Patient" report and submitted to the FDA for inclusion in the framework used to evaluate future FRDA therapies.

There are several ways you can get involved:

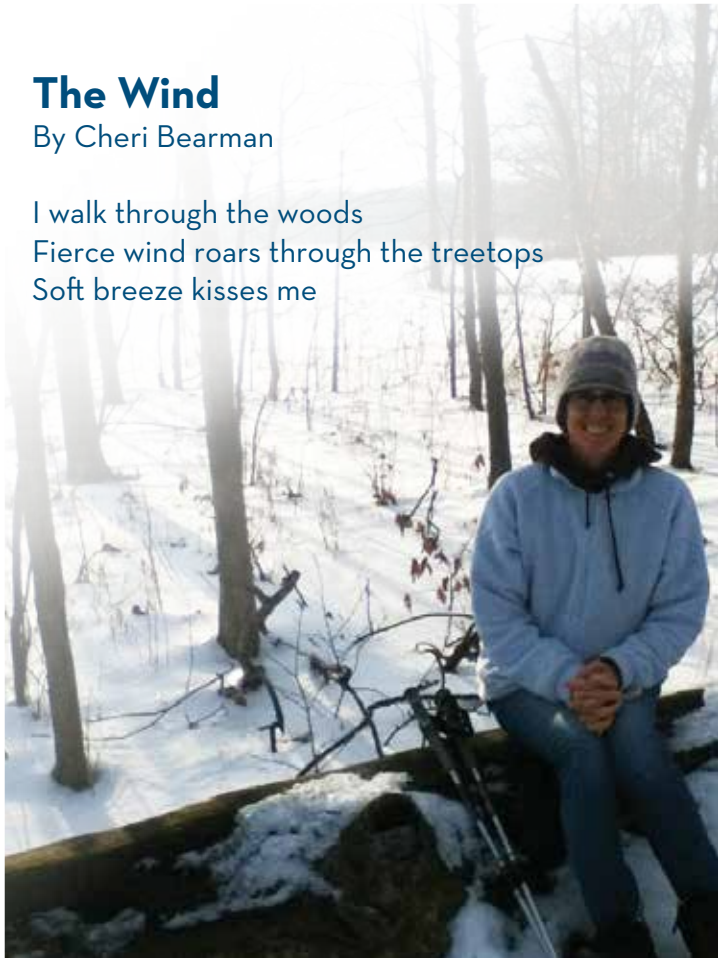
- Attend the PDFF meeting in Bethesda, MD on June 2, from 8 a.m. – 12:30 p.m., at the College Park Marriott and Conference Center.
- If you cannot attend in-person, join online via streaming webcast and share your input on the specific panel questions, as well as demographic questions.
- Keep an eye out for future communications and surveys through which you may be able to contribute your thoughts.

No one can make the voice of the FRDA community heard more than those impacted by the disease. Your participation is critical to making sure our collective voice makes an impact. Don't miss out on the opportunity to make sure your input helps guide the development of successful, effective, meaningful treatments for FRDA. If you have questions, you may contact **Sue Hagen at susan@ataxia.org.**

The Wind

By Cheri Bearman

I walk through the woods
Fierce wind roars through the treetops
Soft breeze kisses me



Pearls of Wisdom

Submitted by Pete Meyerhoff

Aspiration pneumonia

I know two Ataxians who died of it. Aspiration pneumonia is a lung infection that follows a wrong swallow, food going into your lungs instead of your stomach.

Ask your doctor to arrange an in-home visit by a swallowing therapist. You will be introduced to the "dry swallow" a maneuver to help strengthen you.

Bed Time

As the ataxia progresses you may have trouble sitting up in bed. They make a gadget called "bed pull up" to assist you. One end of it loops around a leg of your bed near your feet, the other end is for you to pull on. You don't have to buy it. You can make your own out of a sturdy rope.

Dizziness

Head movements, especially quick ones, can cause me to become dizzy. I can reduce this by blinking (eyes closed) during the head movement. You can experiment with this and see if it helps you. Try this sequence: Focus, close eyes, move head, open eyes, refocus!

Driving

Very hard to give up. Freedom abandoned. At some time, it will have to happen. Don't wait for an accident or a ticket. Opportunities will open-up. Friends, family, ADA buses. Accept it. It is inevitable.

Eye-hand Coordination

What helps me prevent accidents is to look at my hand as it moves to grasp something. I never take my eyes off my hand. I don't assume that I know where my hand is in space.
Move slowly. Remember the Ataxian mantra: "Slower is faster"

iPad

I use it several times a day. It keeps me connected to the world. It is very light weight, has long battery life between charges, has a keyboard large enough for Ataxian fingers, its home base can be any convenient place in the house, its screen is nice size, all apps are available, email and web searches are easy. Just a great gadget. $450 will get you one.

Pain

It certainly is no fun to be afflicted with ataxia: but no pain. How many people do you know with painful conditions? I understand there is not much solace in rejoicing in troubles that others have and you don't. Nevertheless, stop complaining. Count your blessings!

Pills

Put your pill inside some apple sauce it will help it go down. Make sure your chin is tucked. A maneuver you should avoid even though you see it in movies: Chin lifted up toward ceiling, pill in, sip of water, then swallow.
This is intuitive, but it is wrong, especially for Ataxians. Always have your chin tucked when you swallow anything. So: pill in, sip of water, tuck chin, swallow.

Poles

The best gadget for Ataxians is the floor-to-ceiling tension pole. I have two of them, one next to the toilet, the other by my bedside. I recommend that a professional install them. The tension has to be just right: too little and the pole is not stable, too much and you might damage the ceiling. Without the poles I don't think I could transfer to my bed or toilet without falling.

Power, Electric Wheelchairs

I have one. It's ok in the summer, outside. My wife who is over 80 does not have to push. It's also nice for long corridors. I have not had much luck in the house. It's much more difficult to control than a manual wheelchair: Many scratches and dents especially in doorways and small bathrooms.
The manual wheelchair keeps up arm and hand strength.

Preventing Table Accidents

Slide full glasses on smooth table surfaces. It won't work on a table cloth. Avoid lifting the glass. Use a bendable straw. Don't forget the chin tuck. Use a liquid thickener to avoid gagging. Use eating tools with thick handles. Wear a bib. As an Ataxian you can't afford to be fussy.

Regularity

It helps avoid embarrassment. Bran cereal for breakfast. Dried fruit raisins, apricots, prunes. Popcorn. Avoid cheese, ice cream, cookies, Fried food. Check with your Doctor. Lots of water. Pee regularly, every two hours? Establish a written schedule of # 2 events.

Stairs
Go down backwards. If you can still walk, hold onto the railing and go down backwards. If you are in a wheelchair, get out of it and get on your hands and knees in an emergency. Go down the stairs backwards. Hopefully someone will help you at the bottom. If not, keep crawling to safety.

Vanity
Forget it. It will only get you into trouble.

Wheelchair or Transfer Chair in Restaurants
Ask to be seated at the corner of a square table. The corner will bring the table surface closer to you. The arms of your chair will not interfere with the table. Bon appetite!

Generations is always looking for great ideas to share with the Ataxia Community. If you have Pearls of Wisdom or a personal story you would like to share in a future issue of Generations, please submit it to Joan at **joan@ataxia.org**. Please keep your "pearls" short and personal stories to 1000 words or less. Those submitting a personal story are asked to please include a photo or two. Thank you.

Social Security

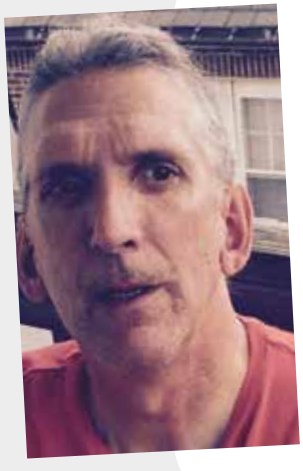
In December, Social Security launched a new service for **my Social Security** account holders where they can check on the status of an application for benefits or an appeal filed with them.

This new addition to the **my Social Security** suite of services will provide detailed information about retirement, disability, survivors, Medicare, and Supplemental Security Income claims and appeals filed either online at socialsecurity.gov or with a Social Security employee.

The service will provide important information about your claim or appeal, including:
- Date of filing;
- Current claim location;
- Scheduled hearing date and time;
- Re-entry numbers for incomplete applications;
- Servicing office location; and
- Claim or appeal decision.

You can still check the status of your benefit applications by phone, by calling 1-800-772-1213, and providing the confirmation number that was received when the claim was filed.
For more information about how to open an account, please visit: **www.socialsecurity.gov/myaccount**.

For the latest Social Security News, check out the latest edition of the Social Security Update.



The National Ataxia Foundation is grateful to Jon Rodis for his volunteer services. Jon has had to navigate the process to receive Social Security Disability. He now helps people all over the United States who are disabled with getting approved for Social Security disability benefits. He is willing to answer any questions you may have about the process. He can also help in areas of Support Groups, Advocacy, State Aid Programs, and Medical Awareness.

His email address is **WSALMGCDJM@aol.com** or you may call him at **617-846-4975**. He does not charge a fee for his service.

Jon is: President-Massachusetts Chapter of The Marfan Foundation • Head-Ehlers-Danlos New England/Massachusetts Support Group • Chair-Physician Awareness Committee(s) for Marfan and Ehlers-Danlos Syndrome(s) • National Disability Advocate for Rare Disorders • Member: Winthrop Disability Commission

Treasure Coast Ataxia Support Group Meeting

Submitted by Linda Farrow and Lisa Cole

On December 3rd, 2016 the Treasure Coast Ataxia Support group had a meeting at Port St. Lucie Community Center, 2195 S.E. Airoso Blvd, Port St Lucie, 34984. Eight people came and shared their experiences regarding some of the Dr's in Gainesville, Florida. Introductions were made and there were positive comments from the group.

One of the guest speakers was Jan Field-Byrne. (field-byrne@comcast.net). She spoke on Pilates, and Micro Movements. Her presentation was very informative and brought about some good questions for discussion. Included with the discussion was a pamphlet which she handed out to everyone.

Our other guest speaker was Dr. Tom Clause. He spoke about reclaiming natural movement. He is knowledgeable in movement rehabilitation and regaining your lost movement skills. He worked with several people in the group.

Our next meeting will be Saturday, February 4th, 2017 at the community center as well. We also are scheduled for a meeting on Saturday April 1st, 2017 with the guest speaker to be Dr. Subramony, MD.

Spoke briefly that some people of the group got to watch the premier of The Ataxian, however the trailer is circulating in the meantime.

Upon close of the meeting, everyone who brought a gift picked one when they left. There were also several great snacks for sharing!

Ataxia Support Group of the Treasure Coast met on Saturday, February 4. We met at the Port St. Lucie Community Center. There were 14 individuals: Ataxians, supporters, and one guest speaker! There was no shortage of fellowship and/or information sharing waiting for the coin toss.

Alabama Ataxia Support Group

Submitted by Becky Donnelly

The Alabama Ataxia Support Group held its first meeting of 2017 on January 28 with 22 members present, including two new members, at Covenant Presbyterian Church in Homewood, AL. The speaker was Kelly Sorrells of Lakeshore Foundation who gave the group suggestions for keeping in shape and detailing sources of availability at Lakeshore Foundation. She also invited the group to have a meeting in 2018 at Lakeshore which would also include a meal.

The Arizona Ataxia Support Group

Submitted by Mary Fuchs

The Arizona Ataxia Support Group meeting was Saturday, February 5th. We had a great time seeing friends and we were so lucky to have wonderful speakers join us! Mary from Maricopa Association of Governments (MAG)and Dolores and Brenda from Valley Metro came to teach us about public transportation options as well as services provided around the valley. Afterwards, a few of us enjoyed a meal and some social time.

Tri-State Ataxia Support Group

Submitted by Kathy Gingerelli

Since this group was started in 2007 we have gone through so many growing pains & it's been a "learn as you go" endeavor but I think over the last few years, we've hit our stride. It's hard to believe how much our group has changed over time.

We had our First Annual Potluck on January 12 and our night started out with a lot of laughs and smiles greeting old friends and welcoming new ones. I gave a quick overview of 2016 and everything the group had accomplished with the highlight being the First Tri-Sate Walk n' Roll to Cure Ataxia which we held on August 27 and what a huge success it was! We are so proud to know that we surpassed the NAF record for a group hosting their first event by raising more than $42K and also by having more than 200 people attend from all over from the New Jersey and New York area.

We are looking forward to some of our upcoming events this year which will include the NY Abilities Expo in May, the NYC Disability Pride Parade in July and our Second Annual Tri-State Walk n' Roll to Cure Ataxia on August 26.

The evening ended with answering some questions and great food. Thanks to Joe Bruno for taking such a great group photo.

Happy New Year!

I hope everyone is ready to get involved for a very busy 2017...it's hard to believe how much our group has changed over time. Since this group was started in 2007 we have gone through so many growing pains & it's been a "learn as you go" endeavor but I think over the last few years, we've hit our stride. Part of that is our annual potluck for our 1st meeting of the New Year which we had at our 1/12/17 meeting.

Our night started with a lot of laughs & smiles, greeting old friends & welcoming new ones. I gave a quick overview of 2016 and everything the group accomplished with the hi-light being the 1st Tri State Walk 'N' Roll To Cure Ataxia held on 8/27/2016 @ Liberty State Park & what a huge success it was. Not only did we surpass the NAF record for a group hosting a 1st time event by raising over $42K but there were over 200 people attending from all over NJ & NY. I quickly went on to talk about some things we're doing as a group in 2017.....they will be:

1. NY Abilities Expo—May 5-7 @ the NJ Convention & Expo Center in Edison, NJ. Fri-11am-5pm, Sat 10am-6pm & Sun 11am-4pm For nearly 40 years the Abilities Expo has been a great source of information for the entire Community of people with disabilities & their families. Learn all about what the Expo has to offer by checking out the website @ **www.abilities.com** Our group will be in charge of the NAF Booth.....I am looking for volunteers to hang @ the booth & give out info (provided by NAF) The hours are listed above so please think about it and be ready to sign up soon. I will have a sheet at the next meeting and after I have all the names of everyone attending, I can register you under our group name so you'll have a badge waiting for you.
2. NYC Disability Pride Parade—Sunday, July 9, 2017; this is the 3rd year for this event & we've been lucky enough to have been present since the beginning. The mission of the parade is to promote inclusion, awareness & visibility of people with disabilities and redefine public perception of disability. Learn more at **www.disabilitypridenyc.org**
3. 2nd Tri-State Walk 'N' Roll To Cure Ataxia—August 26, 2017 @ Liberty State Park, Jersey City, NJ. We will be using all the contacts we made last year plus using everyone willing to volunteer to make this day unforgettable & more even more amazing than last year.

After answering some questions, we got down to the important part of the night.....the food!! After loading plates with everything from homemade sushi, pulled pork sliders, boneless bbq ribs & potato croquettes plus more; we all sat and enjoyed great food, conversation & friendship. Before the night ended, our go-to photographer (member Joe Bruno) took a great picture of the group.

Thanks to everyone who helped make our annual potluck meeting a huge success & extremely fun night & here's to 2017!

Next meeting is Thursday, March 9, 2017

The Ultimate Finish Line.... a Cure for Ataxia

What is Walk n' Roll to Cure Ataxia?

The Walk n' Roll to Ataxia program is the National Ataxia Foundation's largest national grassroots fundraising event held in recognition of International Ataxia Awareness Day (IAAD).

Walk n' Roll, which began in 2007, is held in cities across the U.S. Walk n' Roll for Ataxia has raised more than $2,258,000 thanks to the support and tireless commitment from walkers, rollers, runners, volunteers, donors, and sponsors.

Why Walk or Roll?

Thousands of families, friends, co-workers, neighbors, and communities come together each year to support NAF's fight to improve the lives of people affected by ataxia and their families.

How Can I Participate?

For more information, or to start a Walk n' Roll in your community, please contact Lori Shogren, NAF Special Projects Coordinator at **763-231-2743** or **lori@ataxia.org**.

New Hampshire Ataxia Support Group

Submitted by Jill Porter

The New Hampshire Support Group held their Sixth Annual Luncheon on February 4 at Fratellos Italian Grille in Manchester, NH.

Western PA Ataxia Support Group

Submitted by Ed Schwartz

The Western PA held their Support Group Meeting on February 7 with ten in attendance. They have changed their meeting day to the first Wednesday of each month,

The group has set the date for their 2017 Walk n' Roll for September 23 at South Park. They have also set-up a couple of fundraisers.

Family Planning with Ataxia Gene

By Amy Lowe

It had been a year since my husband and I got married. We always longed to have children of our own. As we began to think more seriously about this next stage in our lives, my dad's Ataxia diagnosis loomed in our minds. After years of reduced balance, slurring of his speech, and a few falls, my dad was diagnosed in his late 50's with spinocerebellar Ataxia type 2 (SCA2). I learned through my research that this type of Ataxia is passed down in families in an autosomal dominant pattern. This means that if one parent has SCA2, each of their children has a 50%, or 1 in 2 chance of also having SCA2.

What did this mean to me and my family? Part of me kept thinking just live your life and either you have it or you don't and hopefully my kids won't inherit it. But I didn't want the future of my children to lie with chance. This first decision was an easy one for me – I wanted to get tested. After an initial consult with a Genetic Counselor at Johns Hopkins, I was reassured in my decision.

On November 10, 2008, my husband and I met with the Genetic Counselor again to learn that I

had a positive genetic test for SCA2. We were told that this meant that, over time, I will develop symptoms of SCA2 even though I wasn't showing any significant signs of the condition at that time. The information did not, however, give me any indication of when the onset of symptoms may occur, their severity, the speed of progression, or which symptoms I would develop. I must admit that one of the hardest things was trying to plan my family's future when I wasn't showing any symptoms and felt a feeling of denial about the whole thing.

I left Johns Hopkins that day with more questions than answers about what my future may look like; however, I had a much bigger decision to make moving forward. Our children. I couldn't accept the feeling I would have if I knowingly passed the gene on when I could have prevented it. However, I also struggled with the idea of what the process would entail. It was called preimplantation genetic diagnosis, or PGD. The counselor explained that the purpose of PGD is to identify embryos that do not have the SCA2 gene change and implant only those embryos in me. The PGD process requires in vitro fertilization (IVF), which involves preparing the body to release multiple eggs that can be extracted and tested and then implanting the unaffected embryos to develop.

That was a lot to digest. Was I mentally, emotionally, and physically ready for this? Could I go through with it knowing that it challenged aspects of my faith and beliefs? Was this really God's plan for me and my family? Not how I imagined it, to be honest. With the support of my husband and my family, as well as the PGD research indicating that the process significantly reduces the chance of having a child with SCA2, I decided to go for it. Appointment after appointment, test after test, injection after injection. Months of questioning my decision and praying for strength and for this to work.

My prayers were answered. I vividly remember the day I received the call. I was helping my middle school students and staff deal with a tragedy. That morning, a student decided to take a short cut to school, which involved crossing the train tracks. Sadly, he didn't make it over the tracks. As I dug within myself to find strength to help others in a time of need, I remember reflecting on how precious a child's life is and how I had no words for the young man's dad sitting at the table with me. I then received a call that brought light to the overwhelming darkness of the day.
The PGD process was successful and I was pregnant....with TWINS!!!!! Our lives changed forever that day and each new day has brought more joy and happiness to our family. Natalie and Claire were born November 25, 2009, almost a year after I got tested. Although born premature at 35 -weeks, they couldn't have been healthier (and Ataxia-free). And we couldn't have been happier.

Yes, the procedure is expensive and no, unfortunately most insurance companies don't cover the expense. Yes, it can take more than one round of IVF (usually 8 weeks of injections) for the pregnancy to take, but I was very fortunate that it worked after the first round. If you're wondering what happened to the other embryo (for me 2 were Ataxia-free, and 1 was questionable if it was affected), the non-implanted embryo was donated for Ataxia research.

I am hopeful that there will be a cure for Ataxia sooner rather than later. But, in the meantime, I'm enjoying every moment with my two beautiful daughters and my family. My dad fights every day through his symptoms, yet he never gives up. He is on the National Ataxia Foundation's Board of Directors, advocating for Ataxia awareness and research and attending the national conferences each year. He and my mom run the Delaware Ataxia support group to support our local Ataxia community. They are models of strength in the face of adversity.

If you'd like to hear more about my story or have any questions, please don't hesitate to email me at **amylowe626@gmail.com**. This is the path I chose to go down, but please know that there are other reproductive options and our genetic counselor was amazing at explaining everything and reassuring us that whatever decision we made would be best for our family. I recommend that you do your research and take that first step. It is SO worth it!

"Making of" video for my next CD, Absence

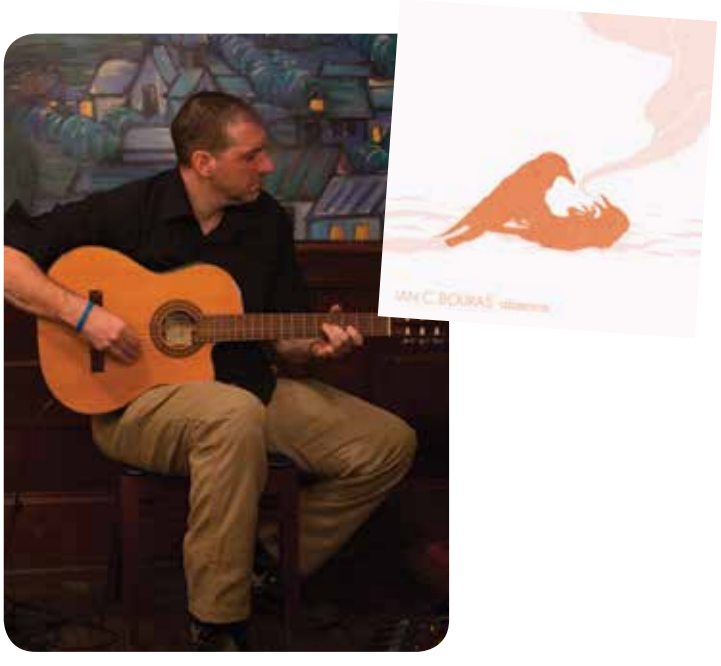
Submitted by Ian Bouras

I was diagnosed with Ataxia. Fortunately for me, mine is progressing slowly. Basically, all my coordination is starting to deteriorate, and sadly that includes my ability to play guitar the way I want. However, I have started to experiment with live looping. I have attached a guitar synth to my guitar, and play bass, piano, synth, guitar, etc... live from the guitar synth and then loop them to create a full band. I run everything through a delay pedal, a pitch shifter, and I have at least one other pedal, and I manipulate the sounds live, so I get to be a composer, musician, and audio engineer all at the same time, and not a lot of people get to do that, so I am lucky.

When I started listening to music, it gave me a wonderful feeling, and I wanted to be able to give others that same feeling. I started performing, and enjoyed the connection I felt, with the audience. Now I am moved to create based on that, plus, Ataxia gives me inspiration, because I like to show that people with any "issue" can create like everyone else, and often create new things, in order to find a way around their "roadblock". I am not thrilled about having Ataxia, but I am happy to be forging my own path, and am the first person, that I know of, to be doing what I'm doing. People do live looping, but not like this.

I am now creating like never before: https://www.youtube.com/watch?v=AuiS2SRc410 and Ataxia has led me to do that. I am also inspired by the confidence I gain by performing. People don't judge based on anything else but the music, and that lets me know they can see beyond any "issue" and focus on the music, so I don't need to be so self-conscious. The other evening I was on a podcast: https://soundcloud.com/arbofcourse/podcast-4-ian-bouras and I'm starting to see that people accept me the way I am.

In a "Making of my next CD, Absence" video, it explains why and how I am doing what I'm doing. You can view it here: https://www.youtube.com/watch?v=iGCwHDR5alA



Welcome Stephanie Lucas!

The National Ataxia Foundation is delighted to report that Stephanie Lucas joined the NAF staff as our Communications Manager on Monday, March 6th. Stephanie came to NAF after 12 years with RS Eden in St. Paul. Simultaneously she became an event manager with Ingster Marketing in Bloomington all while finishing her BA degree at Metropolitan State University with a major in Organizational Communications and a minor in English. Stephanie has a 12-year-old son Marquess and they live in South St. Paul. Stephanie is working with Joel Sutherland and others to create a social media strategy for NAF and be responsible for its tactical implementation. She serves as the project manager for the NAF website redesign. In addition, she facilitates NAF's overall communications. Join us in welcoming Stephanie to the National Ataxia Foundation.

Stephanie@ataxia.org

Announcing the

61st NAF Annual Ataxia Conference

April 5-6, 2018 at the

Marriott Philadelphia Downtown Philadelphia, PA

Reservation information will be announced soon

Join us in Philadelphia!

www.discoverphl.com

For the latest information on conference registration, program schedule, and area information keep checking NAF's website – www.ataxia.org

Kozy Nook Fundraiser

Submitted by James Coyne, Jr.

My wife Janet had SCA3 for many years, as did her mom, Ruth and my daughter.

Janet died on September 21, 2008 and as IAAD was fast approaching, my family thought we should do something special in memory of Janet. We reached out to a local family restaurant that Janet and I would visit. They offered an "Eat In or Take Out" from 4-closing and donated 15% of the proceeds to the NAF. The evening brought customers from area towns and all were pleased with the food and service.

I would like to that all those that made the fundraiser a HUGE success.

The Coyne family will be looking forward to more fundraisers in 2017. The event raised just under $900 for the National Ataxia Foundation.

James Coyne, Jr,(R) & Jeff Abrams, owner of the Kozy Nook in Westport, MA

Sarah Elizabeth Hale

This is a tribute to my sister, Sarah Elizabeth Hale, who died of complications due to Ataxia on December 23, 2016, age 67. It is also a tribute to the National Ataxia Foundation and to Wilderness Inquiry, both of which made a significant contribution to the quality of her life in her last eight years.

We're six kids, but Sarah is my only sister, six years younger than me. We were raised with a love for camping, nature, and a genuine interest in other people. Sarah took on all this and more. She had a zest for life and a love of adventure. She enjoyed people and was just plain fun to be around. Friendships were many. She was certainly the favorite aunt. She was also a determined person, and could be very stubborn – which served her well in the end. Maybe being fifth in the pecking order added to her feistiness.

If she wanted to do something, she did it. She played horn in the high school band, and wanted to be the majorette. "Not possible" said the band teacher, "we don't have any uniforms for that." So she made a majorette uniform. Although she joined our mother and me when I tried on wedding dresses, she wouldn't let me buy one: she made it – a beautiful dress, and her bridesmaid's dress to match. I was finishing my Masters and a package arrived: a doll with my wedding dress on, right down to small, matching lace – "to inspire me to finish". A year earlier I had received another package: a carved pumpkin for Halloween. She figured grad students didn't have time to carve such things. You had to open her packages with caution.

She loved wearing costumes for any occasion that might call for it, and two years in a row, won the best costume at the Anchorage Fur Rendezvous: the first time holding a lantern and wearing an airplane contraption that said, "Fly by night" on the side, the second time wearing white long-johns under a red negligée, and a platinum blond wig, with a sash that read "North Slope Recreation Director." Even her friends didn't know who she was.

She loved sewing and made everything from backpacks, to a lot of her clothes, and she also did beautiful embroidery, crewel, and knitting, and made moccasins with all the bead work. A friend of our grandmothers had knit us all Christmas stockings as we were born. By the time we were married and had our own kids, she was no longer with us, so Sarah took up the tradition, and the next two generations also have knit Christmas stockings.

She was a Girl Scout from grade school all through high school, with a troop that still gets together. After graduating from Southwest High in Minneapolis, she began college at Hamline, as a math major, but decided to major in outdoor recreation, so transferred to the University of Oregon. She married Jim Satterwhite (but didn't wear her waffle stompers for the ceremony as she threatened), and they eventually moved to Alaska. Sadly, Jim died a few years later, but she stayed in Alaska. She worked for the Girl Scouts. She worked for Wien Air Alaska, in the office and as a stewardess. "Coffee, tea or milk" – "No, it's too early for beer. Coffee, tea, or milk" and they accepted, she was so charming. Eventually she went to work for Arco on the North Slope, first as a stewardess for the crew flights; then as an engineering assistant, and finally as a well-head engineer, not jobs usually filled by women. She was a volunteer with the Kuparuk Fire Department. Over the years, she raised and raced sled dogs; learned to play the bagpipes, a dream from when she was a little girl, and joined the Crow Creek Pipes and Drums. She curled, hiked, camped, skied and sailed.

She bought an RV and when Arco went to two weeks on and two off, instead of one and one, she travelled around the lower 48, parking it somewhere to fly back to work. She was always accompanied by Elliott, a rather quiet companion, but very loyal. When she left Arco, she continued to live in her RV, travelling the country. She became a member of an RV group and made many friends over the years. Looking for other work, she took the training to repair RVs and

set up her own shop, summers in Minnesota, winters in Nevada. With our parents aging, she moved permanently to Minneapolis and became a letter carrier with the U.S. Postal Service transferring to an office job when she became inexplicably unsteady on her feet.

She often went camping, with a brother if one was available, otherwise with friends or on her own. In about 2008, she started having trouble keeping her balance and her handwriting wasn't as neat as it usually was. This was of concern, but a first visit to a neurologist was not helpful. A friend at work suggested she look at the National Ataxia Foundation (NAF) website. She read up on Ataxia and decided that was what she had. She also discovered one of two major research centers is at the University of Minnesota, so she made an appointment. I happened to be visiting her that week. On the way to the appointment, she said she wasn't going to do anything about her situation – such as getting out Mother's canes, which she still had – until she knew what it was. She was thoroughly examined by three people and when the doctor announced that, "Yes, you have Ataxia", I'm sure it's the only time a patient reacted by chuckling. But Sarah's diagnosis was confirmed,

and we were simply glad to know. As soon as we got home, she got out the cane. The next thing we did was go up to the North Shore to go snow shoeing.

Sarah became a member of the NAF and read everything she could find on Ataxia. She joined an Ataxia support group, which proved to be very helpful and made a number of friends through them. For the next several years she attended the NAF Annual Ataxia Conference, which is both highly scientific and very helpful for those with, or helping those with, Ataxia. She always had a team at the annual "Walk, Stroll n' Roll" in St. Louis Park, even when she could no longer go herself. She followed the research, and participated when she could, believing that though it would be too late to help her, it might help others in the future.

She had a rare Ataxia that doesn't get much attention. All this she would write in detailed e-mails. Since we are all scattered, that's how we got news of the disease, the progress, and expectations, and how she was coping. "I can't do it that way anymore, but I can do it this way." Or, "I can't do that anymore at all, but I can still do this, and this, and this." Slowly, however, her e-mails got shorter and shorter, until they stopped altogether when she could no longer hit the right keys.

A week after the diagnosis the pre-service program at church was on "The importance of inclusivity". In a church, that could be a lot of things, but in this case it was someone from Wilderness Inquiry, a Minneapolis-based canoe and camping outfitter that specializes in taking handicapped people – people challenged in all kinds of ways. They even had Ataxia on their list. "Good, says Sarah, "I don't have to give up camping." Over the next few years she went on day trips on the Mississippi, week-long canoe trips around Apostle Islands on Lake Superior and in the Boundary Waters; dog sledding in the winter. She organized a special trip with them around Apostle Islands for our extended family, while she could still pass the tip test, which is one of the best things we've done together. Being able to make these trips meant a great deal to her, and was also another source of deep friendships.

Sarah never let the Ataxia slow her down more than she needed to. She continued to manage things far longer than you might have thought possible. "I know what I want to do, my body just doesn't obey me," she used to say. As she went from cane to walker to wheel chair to motorized wheel chair. She continued to be more mobile than you would have thought possible. When she had her motorized wheel chair she even drove 5 miles across Minneapolis and back to visit a friend. Already on feeding tubes, with our brother, Peter, weakening from melanoma, but none of us able to get there, her caregiver drove her to Bozeman and back for a two-day visit. Her caregivers over the years made a huge difference to her life, and they loved working with her because she was so fun and so determined. She was a great story teller, knew more family history than anyone, and had a wonderful sense of humor. Eventually her speech went from being slightly slurred and "drunken", to less and less articulate and softer and softer. Our cousin made a board with the alphabet and she would spell out what she wanted to say. Whole stories and descriptions. As time went on, it became harder and harder to point to the letter she wanted, so there was increasing guesswork on the part of the listener. I was never sure who was more frustrated, us or her. She had so much to offer and to say. Her mind never went. But she couldn't articulate or, in the end, express her thoughts and knowledge in any way. That was probably the most difficult aspect of this disease – the inability to communicate when her mind was fully functional.

But through it all, she never lost her determination, her zest for life, or her wonderful sense of humor – all of which was infectious, and many of us are the richer for it.

The National Ataxia Foundation and Wilderness Inquiry, and friends she met through them, added a lot to her last years of life.

Member Stories

Share your story as an NAF member to increase awareness and support for the NAF mission. Member stories can be read on NAF's website at **ataxia.org/about/member_stories.aspx**.

JOHNS HOPKINS MEDICINE

Approved October 24, 2016

JOHNS HOPKINS MEDICINE

Kennedy Krieger Institute

Individuals with SCA6 and SCA8 are Needed to Participate in Research Studying Ataxic Movements at the Kennedy Krieger Institute

Participation will involve 2-4 hours of behavioral testing and a neurological exam, with multiple visits to our lab possible.

Behavioral testing may involve having small sticky markers placed on your arms and legs so that a computer can detect your movement as you stand, balance or walk on a treadmill.
It may also involve reaching with your arms while sitting at our KinArm robot.
We may also ask you to do non-invasive, very low intensity brain stimulation.

Through our studies we hope to gain a better understanding of why cerebellar disease makes movements ataxic and whether different behavioral therapies can help rehabilitate ataxia symptoms.

There are no significant risks associated with our studies.
Participants will be paid $20 per hour of study time and lunch will be provided.

Contact **The Center for Movement Studies** by email at **ataxiastudies@kennedykrieger.org** for more information.

Principle Investigator: Amy J. Bastian, PhD
Kennedy Krieger Institute
Funded by the National Institutes of Health
JHM IRB Application #: NA_00043851

Memorials and In Your Honor

The National Ataxia Foundation is grateful to those who have made contributions in memory of or in honor of their friends and families whose names are listed below. This list reflects contributions made in November 2016 through January 2017. We are sorry that we cannot separate the memorial contributions from those made in honor of someone, as sometimes the person making the contribution does not always let us know if the contribution is a memorial or in honor of their friend or family member.

Chandler Abel
Jason Aiello
Crystal Allsopp
Jack Archual
Michael Athey
Sharon Baggett
Vickie Balogh
Nancy Banning
Brandon Barker
Elle Barnhart
Mary Barros
Betty Beck
Clair Beck
Deborah Blaes
Stephanie Blake
Myrl Branscom
Jaime Brooks
David Brown
Peggy Brunnert
Timothy Buckley
Sandra Burke
Katherine Campbell
Katie Campbell
Keene Campbell
Panet Campbell
Marlene Canfield
Grant Carew
James Carr
Richard Carr
Robert Carr
Kai Chau
Robert Clausen
Lou Colletti
Roger Cooley
Debra Covington
Heather Cox
Janet Coyne
Karen Crawford
Kevin Curry
Frances D'Amato
Jeannette Davis
Richard Davis
Joseph DeCrescenzo
De Mint Family
Charlotte Depew
Connie DiVincentis
Koby Dotch
Deena Driskill
Florence Durrant
Richard Edwards
Andrew Egeressy
Louise Estabrook
Knolen Face
Barton FerrisJr
Fred Flory
Willard Forman
Ron Fraiser
Becky Frasier
Peggy Frasier
Shantay Frasier
Ruth Furniss
Gregson Gann
Steven Geye
Beverly Giles
Lynda Gillam
Penny Golminas
Steve Golomski
Pansy Gooch
Katherine Gorman
George Guffin
Thomas Gunnigle
Sally Hale
Sarah Hale
Andrew Haluska
Evelyn Hankins
Jim Hankins
Jimmy Hankins
Janet Hannaford
Stephen Harkulich
Darlene Helgren
Karl Hoff Sr
Bryce Hollis
Jacob Hollis
Robert Hooker Jr
Robert Hooker Sr
Jenny Hovey
Louis Howe
Sydney Hubbard
Jordan Hubbard
Meghan Huffman
Krista Humes
Lisa Jaffe
Roger Jaffe
Marianne Jones
Lisa Kelso
Mac Kelso
Martin "Marty" Keniley
Marty Keniley
John Kent
Anne Killan
Wayne Kist
Donna Klot Family
Donna Klotz
Susan Knopp
Dr Arnulf Koeppen
Robert Koetz
Jordan Kohl
Rich Korosa
Normand LaBarre
Diane Laufman
Jennifer Law
Jennifer Leader

Wilson Lee
Amy Legault
Johna Leidholt
Richard Lewis
Mr & Mrs Ronald Ley
Cindy Linari
Joanne Loveland
Bill Lowry
Robert Lowry
Lowry Family
Michael Lundquist
Sidney Luther
Brad Machado
Katherine Manolopoulos
Olivia Mantovani
Anne Marshall
John Marten
Page Martin
Jack Mason
Susan Mason
Brent Masserant
Dana Mauro
Diana Mauro
John Mauro
Lois McCamy
Melba McCarthy
Maury McDonald
Bettylou McIntosh
Earl McLaughlin
Jose Medeiros
Kevin Merk
Amy Messigian
Kim Michael
Kimberly Michael
Deacon Mickens
James Mickens
Jim Mickens
Lora Minichillo
Vrouke Moore
Margaret Morris
Theresa Morrissette
Olivia Mueck
Mae Mustian
Audrey Naylor
Patricia O`Brien
Jeffrey Oram
Greg Ostrom
Julie Peng
Yao Peng
Stephanie Peterman
Cody Peterson
Eric Peterson
Jane Petticrew
Nina Piatetsky
Renee Poli
Ted Poli
Zexia "Zax" Pourney
Julie Quinlivan
Redman Family
Gerard Reidy
Jennifer Reintjes
Elizabeth Riley
Florence Rinaldi
Ernest Rogers
Kim Rolleri
Vincent Rolleri
Mary Rotolo
Kerstin Safari
Sol Santos
Mary Schlickbernd
Bruce Schneider
John Schodey
Lenore Schultz
Heidi Schuman
Larry Schut
Loretta Schut
Carol Segura
Marlene Sequeira
Sienna Shank
Dr James Skok
Doyle Smith
Ronald Smith
Kathry Smithers
Terry Snider
Abbie Spellman
Jenny Spiller
Ellen Stamelos
Donald Stanosheck
David Stein
Pearl Straub
John Surabian
Kelly Swier
Kyle Swier
Vernon Swier
Linda Swinkola
Kelly Tambourine
Barbara Tinari
Delores Tise
Pat Tobias
Isaac Todman
Mark Tokarz
Thomas Tschida
Margaret Tseng
Buck Turnbull
Phil Turnbull
Margaret Uhland
Antoinette Varron
Marcia Vaughey
James Vingo
Robert Vozar
Shirley Wagner
Barry Washburn
Olive Westhoff-Derrington
David Westrick
Charles Williams
Lorranie Wilson
Berta Wofford
David Yingling
Yingling Family
Allan Yousten
Hitomi "Patsy" Zeller
Jack the Cat

biohaven pharmaceuticals

RESEARCH STUDY IS CURRENTLY BEING CONDUCTED TO EVALUATE AN INVESTIGATIONAL MEDICATION FOR SPINOCEREBELLAR ATAXIA
YOU MAY QUALIFY TO PARTICIPATE IF YOU:

- Are between the ages of 18 - 75
- Have a known or suspected diagnosis of the following hereditary ataxias: SCA1, SCA2, SCA6, SCA7, SCA 8, or SCA10
- Are able to walk 8 meters (26 feet) without assistance (canes and other devices are allowed)

For more up-to-date information about this study and how to contact specific sites, refer to https://clinicaltrials.gov and search on the identifier number: NCT02960893

PARTICIPATING RESEARCH SITES INCLUDE:
CNS Trial, Long Beach, California *
University of California, Los Angeles, California *
University of California, San Francisco, California *
University of Colorado Denver, Denver, Colorado *
University of Florida, Gainesville, Florida *
University of South Florida, Tampa, Florida *
Emory University, Atlanta, Georgia *
Northwestern University, Chicago, Illinois
University of Chicago, Chicago, Illinois
Johns Hopkins University, Baltimore, Maryland
Harvard University (Massachusetts General Hospital), Boston, Massachusetts *
Harvard University (Beth Israel Deaconess Medical Center), Boston, Massachusetts
University of Michigan, Ann Arbor, Michigan
Columbia University, New York, New York *
University of Texas Southwestern, Dallas, Texas
Houston Methodist Research Center, Houston, Texas

*Currently recruiting

This content is a summary from information that is publically available on Clinicaltrials.gov

Connect and engage with the ataxia community through the new National Ataxia Foundation App!

ANDROID APP ON Google play

Download on the App Store

http://tinyurl.com/NAF-Google-App
http://tinyurl.com/NAF-Itunes-App

Desktop version –
ataxia.echurchapps.com/#!section=14600426

The MOXIe Study

A study of omaveloxolone (RTA 408) in Friedreich's ataxia

MOXIe is a double blind, placebo-controlled, multi-center Phase 2 study of the safety and efficacy of omaveloxolone (RTA 408) in Friedreich's ataxia

About the Study

- Treatment: Omaveloxolone or placebo capsules taken by mouth once daily
- Approximately 8 visits over 16 weeks
- Primary endpoint: Change in peak workload, measured on a recumbent bicycle
- Cost of travel may be reimbursed

Criteria for Participation

- Between ages 16 and 40
- Genetically confirmed Friedreich's ataxia
- Willing to discontinue taking some medications
- Not pregnant, planning a pregnancy, or breastfeeding

Recruiting Study Center Locations

- **Los Angeles, California: UCLA**
  Susan Perlman, MD
- **Gainesville, Florida: University of Florida**
  S.H. Subramony, MD
- **Tampa, Florida: University of South Florida**
  Theresa Zesiewicz, MD
- **Atlanta, Georgia: Emory University Hospital**
  George Wilmot, MD
- **Columbus, Ohio: Ohio State University**
  Chad Hoyle, MD
- **Philadelphia, Pennsylvania: CHOP**
  David Lynch, MD
- **Innsbruck, Austria: Medical University Innsbruck**
  Sylvia Boesch, MD
- **Parkville, Victoria, Australia: Murdoch Children's Research Institute**
  Martin Delatycki, MD

MOXIe

Contact information for participating study centers can be found on the clinicaltrials.gov listing

REATA PHARMACEUTICALS

Go to www.clinicaltrials.gov/ct2/show/NCT02255435 for more information

Version 1; September 2016

NAF Staff Directory, Directory of Chapters, Support Groups, Social Networks and Ambassadors

The National Ataxia Foundation has a large network of volunteers who serve as support group leaders, chapter presidents, and ambassadors for our organization. These volunteers help identify important local resources and professional care for people with ataxia and their families.

If you or a loved one has been newly diagnosed with ataxia, please contact the NAF leader nearest you. If there is not a group in your area, we encourage you to visit our online social networks. You may also consider starting a support group in your area or becoming an NAF ambassador. If you are interested in these volunteer positions please contact Lori Shogren of the NAF staff at **(763) 231-2743 or lori@ataxia.org.**

The use of these names and contact information for any purpose other than requesting information regarding the NAF, joining a chapter or support group without the NAF's written permission is strictly prohibited.

NAF Staff Directory:

Julie Braun, Financial Director	(763) 231-2745
Sue Hagen, Patient Services and Research Director	(763) 231-2742
Joan Jensen, Outreach Coordinator & Generations Editor	(763) 231-2741
Stephanie Lucas, Communications Manager	(763) 231-2744
Mary Ann Peterson, Office Assistant	(763) 231-2747
Lori Shogren, Special Projects Coordinator	(763) 231-2743
Jan Stewart, Office Assistant	(763) 231-2746
Joel Sutherland, Executive Director	(763) 231-2748

Social Networks

NAF Bulletin Board
Moderator – Atilla and Bear
www.ataxia.org/forum/toast.asp

NAF Chatroom
Moderator – Della (ddpokernut@yahoo.com)
www.ataxia.org/connect/chat-rooms.aspx

NAF Facebook Group
www.facebook.com/groups/NAFmail/

NAF Facebook
www.facebook.com/ataxiafoundation/

NAF YouTube Channel
www.youtube.com/user/NatlAtaxiaFound?feature=mhum

NAF Twitter
https://twitter.com/NAF_Ataxia

Chapters, Support Groups and Ambassadors

Please note: Hometown of each Support Group Leader or Ambassador is noted below. For group meeting locations please refer to the Calendar of Events & Support Group Meetings

Alabama

Alabama Support Group Leader
Becky Donnelly-Hover, AL
(205) 987-2883
E-mail: donnelly6132b@aol.com
www.ataxia.org/chapters/Birmingham/default.aspx

Ambassador
Dianne Blain Williamson-Huntsville-AL
(256) 429-9092 or (256) 520-4858
E-mail: diannebw@aol.com
www.ataxia.org/chapters/DianneWilliamson/default.aspx

Arizona

Phoenix Area Support Group Leaders
Angela Li-Peoria, AZ
(847) 505-4325
E-mail: angelali1010@gmail.com

Mary Fuchs- Casa Grande, AZ
(480) 212-6425
E-mail: mary11115@msn.com
Facebook Group:
www.facebook.com/groups/arizonaataxia/
www.ataxia.org/chapters/Phoenix/default.aspx

Ambassador
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Friedreich's Ataxia Clinical Trial at Mayo Clinic with Medication Epicatechin.

Study # NCT02660112

Dr. Gavrilova and team at Mayo Clinic Rochester Minnesota are recruiting individuals with Friedreich's ataxia to participate in a drug clinical trial. This clinical trial is designed to determine if the study medication Epicatechin will improve the neurological or heart functions of patients with Friedreich's ataxia. Epicatechin is an organic based medication with a good safety record. We are recruiting children and adults (age 10-50 years) with a confirmed diagnosis of Friedreich's ataxia and neurological and heart related symptoms. The participants' disease duration should be of 7 years or less.

In this clinical trial, you will be evaluated at Mayo Clinic Rochester during three separate visits over a 6 month period. After the first visit, if you qualify for participation and if you are willing to take part in the study, you will be asked to take an oral medication three times per day for six months.

At each visit you will be evaluated by neurology, cardiology, endocrinology, genetic and physical medicine specialists. You will also have an MRI and an echocardiogram. Blood and urine samples will be obtained at each visit to the clinic.

More detailed information about this clinical trial is available in the consent form and on the website clinical trials.gov, Mayo, NAF and FARA websites.

If you would like further information or are interested in participating, please refer to this study by its ClinicalTrials.gov identifier: **NCT02660112**

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International Ataxia Awareness Day (IAAD)

Get Involved in IAAD • September 25, 2017

International Ataxia Awareness Day (IAAD) is an international effort from ataxia organizations around the world to recognize September 25 as International Ataxia Awareness Day. To find out how you can get involved, please download the IAAD Kit on the National Ataxia Foundation's website, **www.ataxia.org**, on the IAAD page under the Event Section. Please let the Foundation know about your IAAD event by contacting Lori Shogren at **lori@ataxia.org**.

2017 Calendar of Events

Wednesday, April 5, 2017
Western PA Ataxia Support Group Meeting
7pm at the Bethel Park Community Center, 5151 Park Avenue, Pittsburgh, PA. For more information contact Ed Schwartz at eds@ataxia.org or 724-941-2210.

Saturday, April 8, 2017
Central Minnesota Ataxia Support Group Meeting
10am - 12pm at 1038 Sunset Ridge Rd, St. Cloud, MN 56303. For more information contact Marsha Binnebose at 320-248-9851 or mbinnebose@hotmail.com.

Saturday, April 8, 2017
Greater Atlanta Ataxia Support Group Meeting
1-3pm at Emory Rehabilitation Hospital, 1441 Clifton Road, NE Room 101, Atlanta, GA 30322. For more information contact atlantaataxia@gmail.com or 404-822-7451.

Saturday, April 8, 2017
Kansas City Ataxia Support Group Meeting
2-4pm at the Northeast Library, 600 Wilson Rd, Kansas City, MO. For more information contact Stephanie Wilkins at 816-623-3318 or sfwilkins@yahoo.com.

Saturday, April 8, 2017
North Texas Area Ataxia Support Group Meeting
10am - Noon at the Ben Washington Baptist Church - Rev Jr Sheppard Educational Center, 615 Davis St, Irving, TX 75061. There is lots of parking and it is handicap accessible. The meeting room is in a separate bldg from the church. For more information contact David Henry at cheve11e@sbcglobal.net.

Saturday, April 8, 2017
Northern California Ataxia Support Group Meeting
11am - 3pm at Our Savior's Lutheran Church, 1035 Carol Ln, Lafayette, CA 94549. You can RSVP to the meeting directly from the group website below and then clicking on the meetings and entering your name and the number in your party.
www.norcalataxia.org

Saturday, April 8, 2017
Running For Sophie
I'm Stephanie Yi and on April 8th I'll be running the Hollywood Half Marathon in Hollywood California in honor of our daughter Sophie. Many of you know that our daughter Sophie has a rare "disorder" called Ataxia. Ataxia makes it incredibly difficult for her to walk & sometimes she needs to use her wheelchair. As a parent, it's so hard to watch your baby struggle. Please visit our website & consider making a donation to The National Ataxia Foundation in Sophie's honor. Thanks in advance from the bottom of my heart. Stephanie
https://ataxia.donorpages.com/2017RunningForSophie/

Wednesday, April 12, 2017
Willamette Valley Ataxia Support Group Meeting - Albany Location
11:30am-1pm at Albany Hospital – 4th Floor Conference Room, 1046 6th Ave SW, Albany, OR 97321. For more information contact Jason Wolfer at wolfer.jason@gmail.com or 503-502-2633.

Saturday, April 15, 2017
NCASG - Sacramento Area Ataxia Support Group Meeting
1-4pm at Sutter Roseville Medical Center - Meeting Room 8, 1 Medical Plaza Dr, Roseville, CA 95661. We meet on the third Saturday of each month. For more information or to be added to this group's mailing list contact Teresa Bredberg at 916-215-2686 or tbredberg@sbcglobal.net.

Saturday, April 15, 2017
Orange County Ataxia Support Group Meeting
2-4pm at Orange Coast Memorial Medical Center Hospital, Breast Cancer Center-Pacific Coast Room, 9900 Talbert Ave, Fountain Valley, CA 92708.
For more information contact Cindy DeMint at cindyocataxia@gmail.com.
http://orangecountyataxia.org/

Saturday, April 15, 2017
Rhode Island Ataxia Support Group Meeting
11am - 2pm at the Bristol Community Center, 101 Asylum Road, Bristol, RI. For more information contact Anabela Azevedo at azevedo70anabela@gmail.com or 401-297-8627.

Saturday, April 15, 2017
Tampa Bay Ataxia Support Group Annual Picnic
11am at Lake Seminole Park, 10015 Park Blvd, Seminole, FL 33777. Shelter 7 TBA. For more information contact Darlene Harris msdee004@yahoo.com 813-431-2859 or Linda Farrow at lndfrrw2@gmail.com.

Saturday, April 15, 2017
Twin Cities Ataxia Social Group
The Twin Cities Ataxia Support Group meets once a month. Family and friends of an afflicted individual are always welcome! We meet on the third Saturday of every month at 10:00am for 2 hours in a meeting room at Langton Place which is located on the south side of the road on County Road D roughly four tenths of a mile east of I35W in Roseville at 1910 W. CTY. RD. D., ROSEVILLE, MN 55112. We wanted to provide a central location that it easy to access which is why we picked this place. Please join us, and make new connections! For more information contact Lenore Healey Schultz at 612- 724-3784 or schultz.lenore@yahoo.com.

Friday, April 21, 2017
Brain Health Fair
10am - 4pm at the Boston Convention and Exhibition Center, Boston, MA. The Brain Health Fair is a unique, free, one-day-only event connecting hundreds of patients, caregivers, families, and kids who are affected by brain disease and who are interested in the wonders of the brain. http://patients.aan.com/go/activities/brainhealthfair

Saturday, April 22, 2017
Denver Area Ataxia Support Group Party
1-4pm at the Swedish Medical Center, 501 E Hampden Ave, Englewood, CO 80113. Potluck lunch at 1pm. Speaker at 2:15pm. Drinks, cups, utensils, and plates provided. For more information or to be added to this group's mailing list contact Charlotte DePew at 720-379-6887 or cldepew77@comcast.net.

Saturday, April 22, 2017
Tarheel Ataxia Support Group Picnic
In the Fellowship Hall at Aversboro Road Baptist Church, Garner, NC. Our guest speaker will be Howie Shareff of the You Call This Yoga studio. Howie specializes in how an adaptation of yoga – not the type you visualize – can help people with disabilities. For more information contact Rom Smith at rsmith@sacherokee.com or 919-779-0414.

Saturday, April 22, 2017
Wisconsin Ataxia Support Group Meeting
10am - 12pm at Lakeview Library located at 2845 N Sherman Ave. Madison, WI 53704. Dr. Christopher Gomez will be there to share his extensive knowledge about Ataxia and answer and questions.**Please note the earlier time and change in location** For more information contact Kory Tabor at kstab77@yahoo.com and 608-237-6090.

Saturday, April 29, 2017
Alabama Ataxia Support Group Meeting
10am - 2pm at Covenant Presbyterian Church, Homewood, AL. For more information contact Beck Donnelly at donnelly6132b@aol.com or 205-987-2883.

Saturday, April 29, 2017
Mid-Atlantic Ataxia Wellness Day
10am-3pm at BWI Airport Marriott in Baltimore, MD. People with Ataxia and Care Partners are welcome to attend and participate in seated massages, yoga, facilitated group discussions, and lunch. Please register for this event by April 5th. For more information or to register contact 410-616-2811 or ddeleno1@jhmi.edu.

Saturday, April 29, 2017
Treasure Coast Ataxia Support Group Meeting
1-4:30pm at the Port St. Lucie Community Center, 2195 SE Airoso Blvd, Port St. Lucie, FL 39484. There will be water, fruit and veggies. Come and make new connections, share your experiences, and learn from others. For more information or to be added to this group's mailing list contact Lisa Cole at lcole2234@gmail.com or 772-370-3041.

Sunday, April 30, 2017
Willamette Valley Ataxia Support Group Meeting - Portland Location
3-4:30pm at the Capitol Hill Library in Portland. For more information contact Jason Wolfer at wolfer.jason@gmail.com or 503-502-2633.

Sunday, April 30, 2017
Ottawa Ataxia Support Group Meeting
1pm at Hazeldean Branch of the Ottawa Public Library. For more information contact Prentis Clairmont at 613-864-8545 or prentis.clairmont@gmail.com.

Wednesday, May 3, 2017
Western PA Ataxia Support Group Meeting
7pm at the Bethel Park Community Center, 5151 Park Avenue, Pittsburgh, PA. For more information contact Ed Schwartz at eds@ataxia.org or 724-941-2210.

Friday, May 5, 2017
New York Metro Abilities Expo
May 5-7, 2017 at the New Jersey Convention & Expo Center, 97 Sunfield Ave, Edison, NJ 08837 http://www.abilities.com/newyork/

Saturday, May 6, 2017
Arizona Ataxia Support Group Meeting
1pm at Ability 360, 5025 E Washington Street, Phoenix, AZ 85034. For more information contact Mary Fuchs at 480-212-6425 or mary11115@msn.com.
Wednesday, May 10, 2017
Willamette Valley Ataxia Support Group Meeting - Albany Location
11:30am-1pm at Albany Hospital – 4th Floor Conference Room, 1046 6th Ave SW, Albany, OR 97321. For more information contact Jason Wolfer at wolferjason@gmail.com or 503-502-2633.

Thursday, May 11, 2017
Tri State Ataxia Support Group Meeting
6:30pm - 8:30pm at Beth Israel Medical Center - Phillips Ambulatory Care Center - 2nd Floor, Conference Room, 10 Union Square East, New York, NY. Please join us! We look forward to seeing you! For more information, please contact Kathleen Gingerelli at kgingerelli@msn.com.

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Saturday, May 13, 2017
Alabama Ataxia Support Group Outing
Group visit to the Birmingham Zoo in Birmingham, AL. For more information contact Becky Donnelly6132b@aol.com or 205-987-2883.

Saturday, May 13, 2017
Central Minnesota Ataxia Support Group Meeting
10am - 12pm at 1038 Sunset Ridge Rd, St. Cloud, MN 56303. For more information contact Marsha Binnebose at 320-248-9851 or mbinnebose@hotmail.com.

Saturday, May 13, 2017
North Texas Area Ataxia Support Group Meeting
10am - Noon at the Ben Washington Baptist Church - Rev Jr Sheppard Educational Center, 615 Davis St, Irving, TX 75061. There is lots of parking and it is handicap accessible. The meeting room is in a separate bldg from the church. For more information contact David Henry at cheve11e@sbcglobal.net.

Saturday, May 20, 2017
Manny, Mike, and Mary Ride to Cure Ataxia
Come and learn about ataxia while having fun raising funds for NAF. The event is a 6 mile fun ride followed by a picnic. The event is followed by refreshments, ataxia information, and team recognition for the most dollars raised. All proceeds benefit the National Ataxia Foundation. For more information please contact Mike Cammer at 610-996-5814 or michael.cammer62@hotmail.com.
https://ataxia.donorpages.com/2017MannyMikeandMaryRide/

Saturday, May 20, 2017
NCASG - Sacramento Area Ataxia Support Group Meeting
1-4pm at Sutter Roseville Medical Center - Meeting Room 8, 1 Medical Plaza Dr, Roseville, CA 95661. We meet on the third Saturday of each month. For more information or to be added to this group's mailing list contact Teresa Bredberg at 916-215-2686 or tbredberg@sbcglobal.net.

Saturday, May 20, 2017
Twin Cities Ataxia Social Group
The Twin Cities Ataxia Support Group meets once a month. Family and friends of an afflicted individual are always welcome! We meet on the third Saturday of every month at 10:00am for 2 hours in a meeting room at Langton Place which is located on the south side of the road on County Road D roughly four tenths of a mile east of I35W in Roseville at 1910 W. CTY. RD. D., ROSEVILLE, MN 55112. We wanted to provide a central location that it easy to access which is why we picked this place. Please join us, and make new connections! For more information contact Lenore Healey Schultz at 612- 724-3784 or schultz.lenore@yahoo.com.

Saturday, May 27, 2017
Los Angeles Ataxia Support Group Meeting
2-4pm at the Veterans Memorial Complex, 4117 Overland Ave, Culver City, CA 90230. For more information contact Lora Morn at loramorn@gmail.com or Harvey Kahn at 562-686-9720.

Saturday, June 3, 2017
Greater Atlanta Ataxia Support Group Picnic
1pm at Lake Lanier, 1360 Buford Dam Rd, Buford, GA 30518. For more information contact atlantaataxia@gmail.com or 404-822-7451.

Saturday, June 3, 2017
Rhode Island Ataxia Support Group Meeting
11am - 2pm at the Bristol Community Center, 101 Asylum Road, Bristol, RI. For more information contact Anabela Azevedo at azevedo70anabela@gmail.com or 401-297-8627.

Wednesday, June 7, 2017
Western PA Ataxia Support Group Meeting
7pm at the Bethel Park Community Center, 5151 Park Avenue, Pittsburgh, PA. For more information contact Ed Schwartz at eds@ataxia.org or 724-941-2210.

Email the dates of meetings and events to lori@ataxia.org

Letter from our Executive Director

Hi Everyone,

I just returned from the Annual Ataxia Conference in San Antonio rejuvenated by the many people I met and the conversations we shared. What a fantastic conference! While the next issue of "Generations" will center on the conference I want to share with you the process of creating our new logo that you see atop of this issue. It signals the beginning of a new NAF and we hope you like it.

Last October the Board of Directors decided it was time for a change. With a new website also in the mix, it only made sense to design the new logo and branding first. From that point, several lengthy call-in sessions took place between the Executive Committee of the NAF and S&A Communications from Cary, NC. Those calls focused on what the committee felt about NAF. Words like compassionate, strong, leader, hopeful, family, research, and connect were just some of the words that were mentioned.

From there eight designs were created and shared with the entire Board of Directors. Those eight were narrowed down to three "finalists" with the entire board, staff and roughly two dozen people from around the country voting for the final new NAF logo.

The outcome is a logo reflective of the words used to describe the Foundation. Dark blue letters of NAF in a font illustrating strength; a circular icon to show the connectivity between researchers, clinicians, patients and families; a gold sphere on top to depict the hope we all have for finding a treatment and light blue lettering illustrating the compassion that is felt throughout for our entire community.

Also under the heading of new news, I would like to announce the addition of Stephanie Lucas to our team here in Minneapolis. Stephanie is the first Communications Manager hired at the NAF. Among many things to do Stephanie will oversee the development of our new website, serve as our in-house web administrator and oversee our social media strategy and tactics. It is a position that has been needed for quite some time and we are sincerely excited to have Steph on-board here at the NAF. Thank you.

Gift — Honor — Memorial

A contribution given in memory of a friend or relative is a thoughtful and lasting tribute, as are gifts to honor your friends or family. A Gift Membership is a wonderful gift to a friend or relative for special occasions like birthdays, graduations, anniversaries, and holidays. NAF will acknowledge your gift without reference to the amount. Simply fill out this form and mail with your check or credit card information to the National Ataxia Foundation. Honor/Memorial envelopes are available free of charge by writing or calling NAF.

My contribution is: ☐ In Memory ☐ In Honor ☐ Gift Membership
Name _____
Occasion _____
Send Acknowledgment Card to:
Name _____
Address _____
City/State/Zip _____
From:
Name _____
Address _____
City/State/Zip _____

Membership

Yes, I want to help fight Ataxia! Enclosed is my membership donation.
(Gifts in U.S. Dollars)
☐ Lifetime membership – $500

Annual Memberships:
☐ Patron membership – $100-$499 ☐ Professional membership – $65
☐ Individual – $40 Household – $60 ☐ Addresses outside the U.S. please add $15

Recurring Gift Membership Program:
If you wish to contribute monthly or quarterly, please consider the Recurring Gift Membership Program. For more information contact the NAF office or visit
www.ataxia.org/giving/default.aspx.
Name _____
Address _____
City/State/Zip _____
Phone _____
E-Mail _____

☐ Yes, sign me up for NAF e-mails

PAYMENT INFORMATION

Gifts are tax deductible under the fullest extent of the law.
☐ Check. Please make payable to the NAF.
Total Amount Enclosed $ _____
Card: ☐ Visa ☐ MasterCard
☐ Discover ☐ AMEX

Name on Card _____
Card # _____
Exp. Date _____ CVV # _____
Signature _____
Phone Number _____

NAF Merchandise

BOOKS

Healing Wounded Doctor-Patient Relationships by Linda Hanner with contributions by John J. Witek, MD $10

Living with Ataxia: An Information and Resource Guide by Martha Nance, MD (2nd ed. 2003) $5

Managing Speech and Swallowing Problems: A Guidebook for People with Ataxia by G.N. Rangamani, PhD with contributions from Douglas E. Fox, MS (2nd ed. updated 2006) $5

Ten Years to Live by Henry J. Schut $9

There's Nothing Wrong with Asking for a Little Help ... and Other Myths by Dave Lewis $10

Evaluation and Management of Ataxic Disorders: An Overview for Physicians, 2nd Ed. – Updated 2016 by Susan L. Perlman $5

VIDEO/CD

Together There is Understanding VHS $5 DVD$5

SHIRTS/MISCELLANEOUS

NAF Wheelchair/Walker Pouch 9.5"Wx5"Hx1"D $5

Original NAF IAAD T-Shirt S & XXXL only $10

NAF Baseball Cap (White or Blue) $10
Limited supply!

IAAD T-Shirt Sizes S to XL $12

IAAD Sweatshirt Sizes S to XXXL $25

SHIRTS/MISCELLANEOUS cont.

NAF Polo Shirts $10 SALE ... Limited supply!
Mens – Royal blue w/white NAF logo S, M & XXXL
Light blue w/royal blue NAF logo in L to XXXL
Womens – Light blue w/navy NAF logo in S to XXL

NAF Denim Shirt w/white NAF logo S, M, L, XXXL $10 SALE ... Limited supply!

"Ataxia is Not a Foreign Cab" T-Shirt $10
White sizes S to XXXL

"Ataxia is Not a Foreign Cab" Long-Sleeve T-Shirt
Light blue sizes S to XXXL $15

"Ataxia is Not a Foreign Cab" Sweatshirt
White sizes S to XXXL $20

Ataxia Necklace, 20" Chain $20

"Ataxia is Not a Foreign Cab" Magnet $1

Window Cling or Bumper Sticker $1 ea.or 6 for $5

NAF Ataxia Awareness Band, Reflex Blue
One size $1 ea. or 3 for $2

NAF Ataxia Awareness Ribbon Magnet $4

"Know Ataxia" Backpack 20"x16" $5

NAF Grip n' Sip Water Mug $5 NAF Lapel Pin $5

Magnetic Power Clip $3

NAF Shoulder Bag $5 SALE ... Limited supply!

To place your order, call (763) 553-0020, fax (763) 553-0167, mail a copy of this form to National Ataxia Foundation, 2600 Fernbrook Lane, Suite 119, Minneapolis, MN 55447 or visit www.ataxia.org

Order Form

Description	Qty.	Size	Each	Total

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Shipping within U.S. Add $6.50

Shipping outside U.S. Add $25.00

Order Total: _____

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Address _____

City _____ State _____ Zip _____

Phone _____

E-mail _____

For credit card orders, please fill out the following information (you MUST include phone number and signature):

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