The National Ataxia Foundation has funded important research to better understand what causes ataxia. Once there is an understanding of what is causing a disease, therapeutic treatments and cures can begin to be developed. Through the support of generous donors throughout the years, the NAF is excited to announce that the ataxia research community and pharmaceutical and biotech companies are now ready to pursue clinical research and clinical trials.

What is clinical research?
Let’s step back and better understand how research finds answers to treat diseases. It begins with Basic Science Research. This type of research explores the unknown. It is sometimes referred to as “bench science” because most of the research is done in a laboratory by a scientist at a lab bench. Basic research provides the foundation for discoveries that move the field to Translational Research. Translational research applies the findings from basic research to enhance human health. Its aim is to translate the basic science findings to preclinical studies that will be necessary to develop trials and studies in humans. Clinical Research involves human participants with the goal of finding new treatments that will benefit patients. Clinical research is most often conducted at an academic medical center.

What does ‘clinical trial ready’ mean?
Moving the ataxia research community to being clinical trial ready has been ongoing however, in 2009 the Office of Rare Diseases at the National Institutes of Health awarded two-year funding to Dr. Tetsuo Ashizawa to lead the Clinical Research Consortium of the Spinocerebellar Ataxias. After the two years, Dr. Ashizawa was awarded a Pioneer Translational SCA grant from the National Ataxia Foundation to continue the important work of the consortium. Many in the ataxia community have participated in these research efforts through the National History
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Table of Contents

Annual Ataxia Conference (AAC)
2017 AAC Announcement ......................... 27
2017 AAC Preview .................................. 28
AAC Exhibitors and Sponsors Wanted .......... 28
How the NAF and the AAC Have Helped Me ... 31
Explore San Antonio .............................. 31
Our First Encounter with the NAF and AAC ... 32
Arnie Gruetzmacher Annual Ataxia Conference Travel Grant Fund .......................... 33
2016 Annual Ataxia Conference Photos ...... 34
Articles
Protecting Your Right to Vote ...................... 16
Exciting Announcement from the NAF Board of Directors .............................. 35
Pearls of Wisdom: Holiday Shopping ........... 36
NAF Merchandise .................................... 43
In Memory: Mary Jane Damiano .................. 54
International Ataxia Awareness Day
How Did You Participate in IAAD? .............. 38
Membership Topics
Chapter and Support Group News ............... 39
The deadline to submit materials for the winter issue of Generations is Friday, October 28, 2016.

Membership Topics (cont.)
NAF Directory of Chapters, Support Groups, Social Networks and Ambassadors ........ 44
Calendar of Events .................................. 50
Memorials and In Your Honor .................... 55
Personal Stories & Poems
Walking .............................................. 37
Memory .............................................. 37
The Final Bell Rings ............................... 49
Research/Research Opportunities
Ataxia Research: Moving to Clinical Trials .... 3
$200,000 Matching Grant for the 2016 Research Drive ............................ 5
Research Summaries ................................ 6-26
Cerebellar Ataxia Sleep Study .................... 23
Study of Cardiomyopathy in Friedreich’s Ataxia Patients ............................ 26
Genes in Inherited Neurologic Disorders Study ................................. 40
Patients with MSA-C Needed for an MRI Study .............................. 48
CoRDS Registry ................................... 53
Ataxia Research Prepares for Clinical Trials
Continued from page 1

Study for SCA 1, 2, 3, and 6. It is important that the natural history study continue. Which brings us to the current state of clinical trial ready research.

With a generous donation by the Gordon and Marilyn Macklin Foundation, the National Ataxia Foundation will provide financial support to academic medical centers across the United States so that these sites will have the tools necessary to perform clinical research. Just as basic science needs tools such as test tubes, chemicals, microscopes, clinical research also requires tools. These tools are very different and include rating scales, DNA samples, natural history studies, a research coordinator and most importantly protected time for the clinician researcher to gather data from the human research participants that will provide the knowledge needed for the pharmaceutical or biotech companies to develop treatments and therapies. This is referred to as being “clinical trial ready.”

This is a significant time in ataxia research and with generous support from the Gordon and Marilyn Macklin Foundation and additional donors, the National Ataxia Foundation will continue to support these important endeavors that will find treatments and a cure.

Ataxia Research: Moving to Clinical Trials

Minnesota is known as “The Land of 10,000 Lakes,” however, on August 30-31, Minnesota became “The Land of 10,000 Visions for Ataxia Research”! That’s because on those days, the National Ataxia Foundation hosted 25 individuals who gathered in Minneapolis to pave the way for institutions across the country to be “clinical trial ready.”

To be “clinical trial ready” is essential to bringing treatments to patients. When institutions have the tools in place, clinical trials can then be performed at their sites. Attendees of the meeting in Minneapolis included Ataxia researchers and clinicians, NAF board members and staff, and pharmaceutical representatives. In addition, representatives from non-profit patient groups, that have been successful in helping launch clinical trials, shared their expertise with the meeting attendees.

The discussion included aspects on how to “move the needle” in Ataxia research to bring the Ataxia community closer to treatments. Rating scales, patient registry, secure data base, DNA collection and storage, collaborative research studies, understanding FDA, natural history studies, eye movement measurements, additional funding mechanism, and much more was included in the agenda. Hence, 10,000 Visions!

The Ataxia researchers attending the meeting were energized and provided the following:

“What a great meeting! Such an energetic nucleus of dedicated Ataxia researchers coming together in the united purpose of facilitating Ataxia treatment trials. I am honored to call these folks my colleagues and friends. Work like we did over the last couple of days gives me so much hope that we are on the right path and moving quickly forward towards the goal of treating Ataxia.” – Dr. George (Chip) Wilmot, Emory University, Atlanta, GA

Continued on page 4
“Dr. Santiago Ramon y Cajal, once said ‘Es preciso sacudir enérgicamente el bosque de las neuronas cerebrales adormecidas; es menester hacerlas vibrar con la emoción de lo nuevo e infundirles nobles y elevadas inquietudes’ and with that comprehend that ideas do not last very long and therefore we must act now and do something with them. The NAF embraces exactly what Ramon Y. Cajal said by bringing us researchers and clinicians alike to take the old, implementing it in the present for the future and advancement of the Ataxias.” – Pattie Figueroa, MS, University of Utah, Salt Lake City, UT

“This exciting NAF initiative is really the next logical step in ensuring that basic science discoveries make a tangible impact in the clinical arena. We at Northwestern, are really happy to be part of the Cerebellar Research Consortium. We would especially like our patients and their families to know that we are dedicated to all aspects of this effort.” – Dr. Puneet Opal, Northwestern University, Chicago, IL

“This is a major step forward toward finding efficacious Ataxia treatments.” – Dr. Tetsuo (Tee) Ashizawa, Methodist Hospital Research Institute, Houston, TX

“How did we get here? It has been a long journey for many of us as physicians and researchers caring for Ataxia patients. For decades NAF has been at every step of the way from funding small seed grants to attract young investigators into the field of Ataxia research to igniting the passion and sense of purpose in researchers by connecting them with the wonderful patients with Ataxia and their families. This is a turning point and truly an exciting time.” – Dr. Khalaf Bushara, University of Minnesota, Minneapolis, MN

“This is a very exciting NAF initiative, drawing on the passion, dedication and expertise of the Ataxia investigator community across the country. The MGH Ataxia Unit is completely committed to this new Clinical Trial Readiness Program. We are very hopeful that the collaborative spirit of the Cerebellar Research Consortium, supported by the philanthropic vision of NAF and the generosity of its donors will lead to meaningful advances in treatment, prevention, and eventually cure of the Ataxias and related disorders.” – Dr. Jeremy Schmahmann, Harvard, Boston, MA

“This was an exceptional meeting. I’ve been in the field quite a while, and never before have I heard so many good ideas for possible therapies for Ataxia – either to treat the symptoms of Ataxia and to block the root cause of specific genetic forms of Ataxia. All of the investigators left the meeting charged up to ensure that this Ataxia consortium makes a true difference.” – Dr. Henry Paulson, University of Michigan, Ann Arbor, MI

The National Ataxia Foundation is grateful to the Gordon and Marilyn Macklin Foundation for their generous support of this research initiative. Thank you to those who took time out of busy schedules to attend the meeting in Minneapolis and a special thank you to Dr. Henry Paulson who facilitated the discussions that brought forth “10,000 Visions for Ataxia Research.”
$200,000 Matching Grant for the 2016 Research Drive

From its very beginning in 1957, a mission of the National Ataxia Foundation (NAF) has been to find treatments and ultimately a cure for ataxia. In 2017, NAF will celebrate the 60th Anniversary of the Foundation: “Proud Past ... Focused Future.” We are excited to announce that the celebration begins with a very generous matching research donation of $200,000 from an anonymous donor for the 2016 Annual Ataxia Research Drive. These donations will be used to fund research conducted during 2017, the 60th Anniversary of the National Ataxia Foundation.

During the NAF’s 2016 Annual Ataxia Research Drive from October 15 through December 15, your research donation will be matched dollar for dollar up to $200,000. Don’t miss this opportunity to have your research donation doubled! Many of you have been a part of the NAF’s Proud Past and we invite everyone to become part of the NAF’s Focused Future by making a contribution to the 2016 Annual Ataxia Research Drive. A significant Focus of the NAF’s Future is finding answers to cure ataxia.

The NAF has received more than 100 “Letters of Intent” to apply for a research grant from researchers representing 14 countries, including the USA, Spain, Australia, Italy, Spain, UK, Portugal, Brazil, Canada, France, Cyprus, United Kingdom, Mexico and The Netherlands. These outstanding research proposals focus on all the forms of ataxia and could bring the answers we so desperately need.

Last year the NAF funded 20 promising ataxia research studies because of the generosity of our donors who contributed to the Annual Ataxia Research Drive. Through those studies more knowledge of ataxia is being discovered and contributes to a better understanding of the disease. This is needed to move ataxia research forward to treatments and a cure.

As we kick off the 2016 Ataxia Research Drive, we report back to our generous donors of what research accomplishments were made in 2015 through the lay summaries that you will find in this issue. Thank you for your past support that made these research projects possible.

Look for your 2016 Annual Ataxia Research Drive letter in the mail in mid-October. You will also be able to go online at www.ataxia.org to make an online donation. Remember we are celebrating 60 years of serving ataxia families and invite you to participate by giving a generous research donation between October 15 and December 15. Thank you to the Anonymous Donor who will match research donations dollar for dollar. We all celebrate the NAF’s Proud Past ... Focused Future.

Become an NAF member or renew your membership online today at www.ataxia.org. YOUR MEMBERSHIP MATTERS!
Research Seed Money Grant

Glutamate Decarboxylase in Cerebellar Ataxia

By Christine Hampe, PhD
champe@uw.edu
University of Washington, Seattle, WA

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

Cerebellar ataxias present a group of disorders characterized by symptoms such as gait abnormalities, fine motor incoordination and anxiety. The pathogenesis of cerebellar ataxias is only poorly understood, obstructing the design of effective therapies. Some patients with cerebellar ataxia show markers of autoimmunity, such as antibodies directed against the patient’s own proteins. These antibodies may interfere with the transmission of signals from nerves to muscles. Such interference may cause the disease-associated symptoms. One target of these antibodies is the enzyme glutamate decarboxylase (GAD). This enzyme is critical for neurotransmission and lack of its expression has been linked to neurological symptoms such as epileptic seizures, gait abnormalities and anxiety.

Funding received by the National Ataxia Foundation enabled us to generate genetic constructs to assess the role of GAD in cerebellar ataxias. These constructs will be used to determine which function of GAD is impaired in patients with cerebellar ataxia. In an effort to investigate the autoimmune response to GAD in patients with cerebellar ataxia, we compared the immune response to patients with other neurological disorders, including Stiff Person Syndrome and refractory epilepsy. We also analyzed the immune response in patients with Type 1 diabetes. Type 1 diabetes patients show a high prevalence of antibodies directed to GAD, but do not present neurological symptoms.

We found that all three neurological disorders differed in their GAD-specific immune response from that observed in patients with Type 1 diabetes. This underlines that the immune response in patients with neurological symptoms is significantly different from that in patients without neurological symptoms. This information may be used to guide in the diagnosis of cerebellar ataxia.

Dr. Christine Hampe

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Molecular Pathogenesis Studies of Spinocerebellar Ataxia Type 1

By Janghoo Lim, PhD
janghoo.lim@yale.edu
Yale University, New Haven, CT

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

The human inherited cerebellar ataxias are genetically heterogeneous but clinically similar group of disorders that share many neurological and pathological features, such as loss of balance and coordination, as well as progressive degeneration of neurons, specifically those in the cerebellum and brainstem. We have utilized spinocerebellar ataxia type 1 (SCA1) as a prototype of dominantly inherited cerebellar ataxias. By investigating the fundamental mechanisms of SCA1 pathogenesis, we hope to gain insight into the common key features of this and several other cerebellar ataxias. SCA1 is caused by a poly glutamine expansion in the protein Ataxin-1. With the National Ataxia Foundation support, we have investigated the possibility that SCA1 affects Wnt-β-catenin signaling pathway in the cerebellum. We found that disease-causing mutant Ataxin-1 affects the activity of the Wnt-β-catenin signaling pathway in many cell types, both the Purkinje cells (PCs) and non-PCs, and this may cause and/or modulate the SCA1 disease pathogenesis. We believe that this study will lead us to better understand the pathogenesis mechanisms of SCA1 and other hereditary ataxias, which we hope will open the possibility of future therapies.

Pathological and Therapeutic Roles of Immunoproteasomes in SCA1

By Do-Hyung Kim, PhD
University of Minnesota, Minneapolis, MN

The following is a research summary of a grant funded by the NAF in partnership with the Bob Allison Ataxia Research Center (BAARC) for fiscal year 2015.

Our study has set a central hypothesis that immunoproteasomes, and inducible type of proteasomes, are key components that affect the onset and progression of the diseases. Proteasomes are the major cellular machinery responsible for degradation of misfolded or oxidized proteins. In
Pathological and Therapeutic Roles…
Continued from page 9

conditions where proteasomal functions are highly demanded, the inducible type of proteasomes may become more important. Immunoproteasomes are a specialized type of proteasomes that are induced during inflammation and stress. They have a higher catalytic power in clearing aberrant proteins compared to standard proteasomes. Thus, immunoproteasomes are considered to be more adaptable to roles in preventing accumulation of defective proteins and polyQ proteins that are prone to form aggregates. Despite implicated roles of immunoproteasomes in the protein aggregation diseases, there has not been any investigation to clarify their roles in the polyQ diseases. Our project has revealed a molecular mechanism for induction of immunoproteasomes under conditions of proteotoxic stress accumulation and demonstrated that immunoproteasomes are crucial to attenuate accumulation and of protein aggregates. We found that the regulation of immunoproteasomes is closely linked to cellular nutritional conditions as well as cellular inflammatory conditions when oxidative stress is high. Although the project is still in an early stage, the grant support from the Ataxia Foundation has allowed our group to obtain insightful ideas for future studies on immunoproteasomes for development of therapeutic strategies.

Research Seed Money Grant

Functions of Senataxin, Product of the AOA2 Target Gene SETX, in Autophagy and Lysosome Function

By James L. Manley, PhD
jlm2@columbia.edu
Columbia University, New York, NY

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

We are studying the mechanisms leading to Oculomotor Apraxia type 2 (AOA2), a form of juvenile ataxia caused by mutations in the senataxin (SETX) gene that prevents patients affected by the disease to coordinate their movements. We recently found that SETX plays a role in autophagy, a process used by the cell to eliminate any damaged, unwanted or toxic components, and known to be altered in a broad range of neurological disorders. Using cellular and molecular biology approaches, we found that loss of SETX changes the expression levels of several important players of autophagy, most likely blocking the autophagy process at an early stage. We also found that SETX seems to protect the cells against the accumulation of proteins that carry the degradation mark called ubiquitin. Indeed, cells...
lacking SETX accumulate ubiquitinated proteins that should normally be degraded and/or recycled. We additionally engineered several cell lines using the powerful CRISPR/Cas9 gene editing system to eliminate SETX expression or to express several proteins carrying an AOA2 mutation. We are currently testing those cell lines in order to confirm SETX role in autophagy regulation. Our work provided important insights into the link between autophagy and SETX function that we’ll be further investigating in order to elucidate the detailed mechanism leading to AOA2.

Research Seed Money Grant

Generation and Characterization of Spinocerebellar Ataxia 36 Patient Stem Cell-derived Neurons to Study Disease Mechanisms and Develop Novel Therapeutic Strategies

By Wilfried Rossoll, PhD
wrossol@emory.edu
Emory University School of Medicine, Atlanta, GA

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

Spinocerebellar ataxia 36 (SCA36) is an inherited progressive neurodegenerative disorder, which has been diagnosed so far mainly in Japan and Spain. Affected patients develop gait ataxia, eye movement abnormalities, hearing loss and specific motor symptoms similar to amyotrophic lateral sclerosis (ALS) or Lou Gehrig’s disease.

SCA36 is caused by a large DNA repeat expansions in a specific genetic region that affect nerve cells primarily in the cerebellum and the spinal cord. This mutation can cause the expression of abnormal RNA and proteins, but the molecular disease mechanisms are still unknown. This disease is inherited in an autosomal dominant fashion, meaning that each child of an affected parent has a 50% chance to inherit the ataxia-causing mutated gene.

As a first step in our long-term effort to find the cause, treatment, and cure for this ataxia, we have generated “induced pluripotent stem cells” (iPSCs) from skin biopsies of adult SCA36 patients. These stem cells are now being differentiated into nerve cells similar to the cells known to function poorly and ultimately degenerate in SCA36 patients. This new “disease in a dish” cell culture model of SCA36 will allow us to 1) answer important questions about SCA36-specific and general ataxia disease mechanisms, and 2) enable us in the future to test the efficacy of potential therapies for SCA36 and other related ataxias.
Determinants of Neuron-specific Pathogenesis: Study in a C. elegans Model of SCA3

By Andreia Castro-Teixeira, PhD
University of Minho, Braga, Portugal

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

Spinocerebellar ataxia type 3 (SCA3), also known as Machado-Joseph disease (MJD), is caused by mutations in the protein ataxin-3 that make this protein prone to aggregate and toxic to specific groups of neurons. This leads to the progressive neurodegeneration and to the typical symptoms of SCA3: ataxia and severe limitations in eye movements and swallowing among others. Currently there is no effective treatment for this disorder. In this project, we were interested to shed light into why mutant ataxin-3 protein, despite being present nearly in all regions of the human body including the brain, seems to affect and cause death of specific cells of the brains (neurons) and not to all. For this, we used a model of the disease in the worm C. elegans, which in spite of being a very simple animal possesses a very well characterized nervous system of 302 neurons, is quite well characterized at the genetic and molecular level, is transparent – allowing monitorization of events within the neurons in the live animal, and, unlike mouse models, is amenable to large scale studies. This model, which has been generated in our lab, expresses the mutant human protein in its nervous system and replicates important features of the disease, such as the aggregation of ataxin-3 and a neurological impairment of the animals (including abnormal movement). We used it to analyze each one of the 302 neurons and to determine which neuron-subtypes are affected by the expression mutant human ataxin-3 (by identifying neurons containing ataxin-3 aggregates/inclusions). We found that nearly all neurons that use the neurotransmitter 8 serotonin (among others) are highly affected by ataxin-3 aggregation. The function of these serotonergic neurons is also compromised. Importantly, chronic treatment of C. elegans and mouse SCA3 models with FDA-approved drugs which increase serotonin availability in neurons, rescues motor behavior impairment in these animals, as well as protects against mutant ataxin-3-mediated neuronal loss. We are currently analysing the RNA profile of these neurons in order to pinpoint the molecular players underlying these findings. From this work, we hope to better understand the intriguing pattern of neurodegeneration and identify additional new cellular targets for the development of therapies for SCA3.
Research Seed Money Grant

Molecular Mechanism of Autosomal Dominant Sensory Ataxia

By Richard Wojcikiewicz, PhD
WojcikR@upstate.edu
The Research Foundation of SUNY, Syracuse, NY

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

Funding from the National Ataxia Foundation allowed me to pursue a project entitled “Molecular Mechanism of Autosomal Dominant Sensory Ataxia (ADSA).” ADSA is a novel and rare neurodegenerative disease caused by a point mutation in a protein called RNF170. Afflicted individuals exhibit an ataxic gait and loss of sensory perception, particularly in the extremities, and this appears to result from degeneration of neurons in the posterior columns of the spinal cord. However, the molecular mechanism by which the mutation to RNF170 causes the disease is unknown. I was able to apply my laboratory’s expertise in biochemistry and cell biology to the molecular mechanism of ADSA, focusing on how the mutation affects the biochemical and cell biological properties of RNF170. This revealed that the mutation destabilizes RNF170 and disrupts cellular calcium metabolism. This helps us understand the reason why neurodegeneration occurs, and points towards therapeutic strategies for ADSA and perhaps other ataxias.

Dr. Richard Wojcikiewicz

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Young Investigator Award for SCA Research

Structural Insights for Drug Discovery
Targeting SK2/3 Channels for SCA

By Miao Zhang, PhD
zhang@chapman.edu
Chapman University School of Pharmacy, Irvine, CA

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

SK channels play important roles in physiological and pathophysiological conditions. These channels have been linked with the symptom of spinocerebellar ataxia. A tremendous amount of effort has been devoted to developing drugs targeting SK channels. Small molecule positive modulators of SK channels have showed beneficial effects in animal models of ataxia. In this research project supported by National Ataxia Foundation, we investigated the interactions between the small molecule modulators and the SK2/SK3 channels. With this discovery, the computer based drug discovery targeting SK2/SK3 channels has become possible, which can facilitate drug discovery targeting SK2/SK3 channels. We combined computer based approaches and experimental techniques to search for new modulators of SK channels. These new modulators themselves can serve as the candidates in preclinical and clinical researches for new therapeutic approaches towards spinocerebellar ataxia. These compounds can also serve as pharmacological tools for studying of SK channels in Purkinje cells during ataxia development.

Dr. Miao Zhang

Young Investigator Award for SCA Research

Role of Microglia in SCA1 Pathogenesis

By Marija Cvetanovic, PhD
mcvetano@umn.edu
Institute for Translational Neuroscience, University of Minnesota, Minneapolis, MN (in partnership with the Bob Allison Ataxia Research Center)

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

Microglia are brain immune cells that are charged with repairing any injury to the brain. In neurodegenerative disease microglia undergo process of activation that profoundly changes their morphology and function. Many studies have found activated microglia to be harmful and worsen disease outcome in neurodegenerative disease. We have demonstrated that microglia are activated pre-symptomatically in mouse models of SCA1. Main goal of this proposal was to test the therapeutic
potential of an FDA approved drug PLX (PLX3397, Plexxicon Inc.) that deletes microglia from the brain without any adverse effect. We have administered PLX to SCA1 mice in the food starting from pre-symptomatic age of disease to test if we can delay disease onset. PLX treatment successfully reduced number of microglia in SCA1 cerebella. While PLX treated SCA1 mice performed significantly better than untreated SCA1 mice on a balance beam test, a sensitive test of motor behavior, they were indistinguishable from untreated SCA1 mice in rotarod test of motor behavior. Moreover neuronal pathology was not rescued with PLX treatment. Therefore we conclude that PLX treatment does not significantly affect disease onset in SCA1 mice. There are several possible explanations. Activated microglia may exert both protective and toxic effect in SCA1 and removing microglia with PLX will reduce both of these influences and thus in sum does not change the disease. Alternatively, microglia do not contribute to SCA1 during the early stages of disease but they still may play a role during the later stages of disease.

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Young Investigator Award for SCA Research

**Defining Pathways that Regulate Levels of Polyglutamine Protein in MJD/SCA3**

*By Maria do Carmo Pereira da Costa, PhD*

*University of Michigan, Ann Arbor, MI*

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

While many advances have been made toward understanding polyglutamine (polyQ) diseases, no preventive treatment is yet available for this group of fatal neurodegenerative disorders which include Machado-Joseph disease (MJD), also known as Spinocerebellar Ataxia type 3 (SCA3). In MJD and other polyQ diseases the toxic mutant protein accumulates. Thus, a simple therapeutic strategy is to reduce levels of the mutant gene or encoded protein. The goal of this proposal was to identify molecular pathways that modulate levels expanded-polyQ Ataxin-3, the toxic protein causing MJD. I seek to understand Ataxin-3 biology, in particular its cellular stability and clearance, with the long-term objective of manipulating specific cellular pathways to reduce levels of toxic ATXN3 as potential therapy for MJD. I have screened the full human genome (18,110 genes) for genes modulating mutant Ataxin-3 abundance in cells. I selected a group of candidate genes for further validation in an independent cell model of MJD and identified a novel subset of genes involved in the regulation of mutant Ataxin-3. In particular, I observed that one of these genes is involved in a novel mechanism that controls ATXN3 stability/degradation by protein modification. These studies will advance knowledge about how cells handle this important neurodegenerative disease protein and identify novel cellular targets for potential therapy of MJD patients.
Pioneer SCA Translational Research Award

Translating RNAi Therapy for Spinocerebellar Ataxia 1 (SCA1) to the Clinic

By Beverly Davidson, PhD
davidsonbl@email.chop.edu
Children’s Hospital of Philadelphia Research Institute, Philadelphia, PA

The following is a research summary of a grant funded by the NAF for fiscal year 2015 in partnership with the Bob Allison Ataxia Research Center.

This proposal had three aims. The first was to determine the lowest dose of our drug that had benefit in preventing ataxic symptoms from occurring in young pre-symptomatic SCA1 mice. The second aim was to determine the lowest dose of our drug to reverse ataxic symptoms in older SCA1 mice that were already showing symptoms. The third aim was to scale up and test the safety in monkeys.

We completed the first two aims and identified a dose that both prevented symptoms in young SCA1 mice and reversed pre-existing ataxic symptoms in older SCA1 mice.

We are in the process of scaling up these doses to monkeys. This Pioneer Award was instrumental in moving this preclinical program forward to the clinic.

Pioneer SCA Translational Research Award

Identification of Druggable Targets that Modulate the Levels of SCA-causing Proteins in Vivo

By Ismael Al-Ramahi, PhD
ia135819@bcm.edu
Baylor College of Medicine, Houston, TX

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

The ultimate goal of the proposed research is the identification of “druggable” genes able to suppress SCA-related degeneration by decreasing the levels of SCA-causing proteins.

We have carried out a modifier screen using a Drosophila model of Spinocerebellar ataxia type-1 targeting 3048 potentially druggable genes. We identified the Drosophila homologs of 333 genes whose knockdown (using inducible shRNAs) ameliorated the degeneration triggered by ATXN1[82Q] expression in the Drosophila eye.

We next assessed the ability of these genes to decrease ATXN1[82Q] levels in human cells using FACS. We found that viral-shRNA mediated knockdown of 55 of the 333 candidates was...
associated with decreased levels of an ATXN1[82Q] fluorescent reporter in human cells. Of these 55 genes, eight also caused a decrease in ATXN1[82Q] levels in the Drosophila nervous system.

Finally, we assessed the ability of a number of the ATXN1[82Q] modifiers to suppress the degeneration caused by other ataxia-causing proteins in Drosophila. We have identified five genes whose knockdown ameliorates expanded ATXN2 and/or ATXN7 induced degeneration in addition to being ATXN1[82Q] suppressors.

Our effort has uncovered a number of robust therapeutic candidates to be validated in mouse models of SCA1. For those targets where chemical inhibitors exist, we will explore the possibility of identifying tool compounds for further characterization. Finally, a subgroup of the identified hits offers the potential for treating more than one SCA, an added advantage when targeting rare diseases.

Pioneer SCA Translational Research Award

Targeting the Intracellular Localization of Ataxin-3 as a Novel Treatment Strategy for Spinocerebellar Ataxia Type 3 (SCA3)

By Thorsten Schmidt, PhD
University of Tubingen, Tubingen, Germany

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

Spinocerebellar ataxia type 3 (SCA3) or Machado–Joseph disease (MJD) is caused by the expansion of a variable part within the ataxin-3 gene containing tandem repetitions of the three DNA elements C, A and G (CAG CAG CAG … the so called CAG repeat). Everybody usually has two copies of each gene (one inherited by the mother and one by the father). However, due to the nature of the disease, the expansion in already one of our two copies of the ataxin-3 gene is causing SCA3. The ataxin-3 gene serves as blueprint of the ataxin-3 protein and patients with an ataxin-3 gene containing the expanded CAG repeat therefore have an enlarged ataxin-3 protein. This enlarged ataxin-3 protein is prone to aggregate in SCA3 patients and these aggregates typically form inside of the so called cell nucleus in a characteristic group of brain cells. In contrast to this, normal ataxin-3 usually resides in the cellular cytoplasm. This means that in SCA3 patients ataxin-3 somehow translocates from the cytoplasm to the nucleus in order to aggregate there.

During the recent years, we extensively studied this translocation of ataxin-3 within cells and confirmed using our specific animal models that mice develop SCA3-like symptoms only if

Continued on page 16
ataxin-3 indeed translocates to the nucleus. In other words: Keeping the expanded ataxin-3 in the cytoplasm protected our mice from the disease! We therefore anticipated this transport process as a novel treatment target or treatment strategy for SCA3. However, in order to make use of this concept and to convert our observation into a treatment strategy, we needed a test which allows us to measure whether a certain compound or drug indeed keeps ataxin-3 in the cytoplasm. For this reason, we generated specific tests and indeed identified two drugs which kept ataxin-3 in the cytoplasm. Fortunately, these drugs were already approved by the FDA (Food and Drug Administration) but for the treatment of other diseases. However, this means that they are known to be safe in humans. As our initial test could only be performed in the lab and in single cells, we next needed to confirm whether the compounds are also functional in whole organisms i.e. in animal models of SCA3 which was the aim of this project.

We therefore generated a test cohort of our specific SCA3 mouse model and treated the mice with the compounds we identified. Such a treatment study required several mouse cohorts: SCA3 mice treated and untreated with the compounds as well as control mice also treated and untreated as comparison to exclude that the compound have negative side effects in mice. We observed that the compounds are indeed safe and do not induce severe side effects. During the observation and treatment period, we perform specific tests in order to measure the onset and development of SCA3-like symptoms which develop within just a couple of weeks in our mice. Such tests e.g. include specific investigations of the gait of each mouse but also analyses of brain tissue. As anticipated, we indeed observed that one of our compound keeps ataxin-3 in the cytoplasm and that mice treated with this compound show an alleviation of specific SCA3-like symptoms in our SCA mouse model. Studies are currently ongoing to further characterize the mode-of-action of these compounds. One realistically need to state that successful tests in mice do not necessarily mean that these compounds immediately could be applied to human SCA3 patients but our results may be an important contribution towards a development of a treatment approach for SCA3 and maybe other spinocerebellar ataxias.

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Protecting Your Right to Vote

The following was originally posted July 18 on the [www.Disability.gov](http://www.Disability.gov) website in the July Disability Connection Newsletter:

All Americans with disabilities should be able to vote independently and accessibly. The ADA is one of several laws protecting those rights. These laws help ensure polling place accessibility and the availability of alternative voting methods and voting aids for voters with disabilities.

The recently updated “ADA Checklist for Polling Places” has information about making polling places accessible. The U.S. Election Assistance Commission offers resources for voters with disabilities, including helpful tips and a video about polling place accessibility. Learn about the American Association of People with Disabilities’ REV UP voting initiative. The Arc’s voting toolkit includes voting resources and a blog post about how guardianship impacts voting rights. For more information, visit DOJ’s voting section or contact your state’s voting commission or board of elections. Learn how to file a complaint if you feel your voting rights have been violated. Call 1-866-OUR-VOTE (687-8683) to report voting issues or concerns. [www.disability.gov/disability-connection-newsletter-july-2016/](http://www.disability.gov/disability-connection-newsletter-july-2016/)
Young Investigator Award

The Role of mTOR in Friedreich’s Ataxia and Identification of New Pathways for Therapeutic Intervention

By Simona Donatello, PhD
Université Libre de Bruxelles, Brussels, Belgium

The following is a research summary of a grant funded by the NAF, for fiscal year 2015.

We started to dissect the role of a protein called mTOR in Friedreich’s ataxia (FA). In particular, we investigated how TORC1, one of the two protein complexes in which mTOR takes part, is activated in FA. TORC1 is a protein kinase complex and it adds phosphates to other proteins in order to activate or switch them off. TORC1 regulation is sensitive to nutrient availability and to the energy status of the cell. It also controls the biogenesis of mitochondria, the organelles that make energy and contain frataxin (FXN). Iron metabolism, which is altered in FA, has also recently been linked to TORC1 regulation through a protein called tristetraprolin (TTP). Our aim is to investigate if and how TORC1 is involved in the pathological processes causing FA, in order to shed new light on the still unclear mechanisms that regulate FXN physiology and identify new targets for FA treatment and cure.

During this first exploratory phase, we obtained data suggesting that TORC1 is differentially regulated in FA according to the tissue context.

In mice, where FXN has been deleted in the liver tissue, and in neurons derived from induced pluripotent stem cells from FA individuals, TORC1 activation seems to be downregulated. Similarly, in cultured cells where the expression of FXN has been reduced, TORC1 activation seems to be transiently reduced, followed by reactivation likely due to compensatory mechanisms.

In a mouse model with moderately reduced FXN levels (KIKO mouse), TORC1 activation varies in the different tissues, probably representing the combination of a direct effect of FXN deficiency and of compensatory responses.

Moreover, preliminary data suggest that TTP is downregulated in FXN-depleted conditions, but it does not seem to correlate with TORC1 activation.

In this exploratory phase, we defined the activation of TORC1 in different in vitro and in vivo models for FA. The next step is to identify in the different contexts, the proteins involved upstream and downstream of TORC1, and interconnect with iron homeostasis and FXN, in order to define novel targets for therapeutic intervention for FA.

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Post-Doc Fellowship Award

Study of Neuropathology of Spinocerebellar Ataxias Using MRI
(Previously Titled: Longitudinal Study of Neuropathology of Spinocerebellar Ataxia Type 7)

By Carlos Roberto Hernandez-Castillo, PhD
crherandezcastillo@gmail.com
Universidad Nacional Autonoma de Mexico, Coyoacan, Mexico

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

Spinocerebellar ataxias (SCAs) are a group of autosomal-dominant cerebellar ataxias caused by degeneration of the cerebellum and its afferent and efferent connections. These are classified according to specific genetic mutation which leads to specific brain degeneration in each subtype. Spinocerebellar Ataxia Type 2 (SCA2) is caused by an expanded CAG trinucleotide repeat in the gene encoding ataxin2. Spinocerebellar ataxia type 7 (SCA7) is considered one of the rarest autosomal dominant cerebellar ataxias and is caused by an expanded CAG trinucleotide repeat in the gene encoding ataxin7. In both cases the mutation causes cerebellar degeneration primarily affecting Purkinje cells, pontine nuclei and inferior olives. Symptoms typically initiate by the third or fourth decade and include several motor and visuomotor disorders, such as ataxia, dysmetria, dysarthria, dyssdiadochokinesia, ophthalmoplegia, and saccade slowing. SCA7 patients also develop macular degeneration which results in permanent blindness. Furthermore, in later stages, both subtypes show an extended brain degeneration in the neocortex which lead to an impairment of the patients’ cognitive skills.

By using the magnetic properities of atoms, Magnetic Resonance Imaging (MRI) technique emerge as a powerful tool that allow us to visualize the entire body in a non-invasive way. This means that it is possible to obtain detailed high resolution images of any body part, included the brain in a relatively easy and absolutely safe manner. In recent years advances in brain imaging techniques have open the possibility of a broad range of analyses to investigate brain structure and function in both healthy and diseased population.

Recent imaging studies carried out in our lab have expanded the knowledge of the pathological changes resulting of the degenerative process in SCA2 and SCA7, including grey matter atrophy, white matter degeneration and functional connectivity abnormalities. It is well known that cerebellar degeneration participates in the ataxia severity in SCAs, however these diseases also affect other brain areas that could also influence the efficiency of the motor coordination affecting both motor planning and action. In order to test this hypothesis, we evaluated a large cohort of SCA7 patients using the scale of assessment and rating of ataxia (SARA). MRI structural images were acquired for the patients group and the volume of each patient’s gray matter was measured using a technique called voxel-based morphometry. Our results showed that the ataxia severity correlates mainly with the volume of gray matter in the cerebellum, but also with...
the parahippocampal gyrus, precentral gyrus, cingulate gyrus, insula, and inferior frontal gyrus volumes. This study is relevant because it shows that the cerebellum is not the only structure related to the ataxia. Furthermore, not all the gray matter atrophy resulting from the SCA7 mutation correlates with the ataxia impairment, but only a specific set of brain areas is critically related to the motor impairment.

Similarly, we explored the integrity of white matter (WM) fibres in a group of SCA2 patients using diffusion tensor imaging (DTI). The ataxia impairment was assessed using the SARA scale. Diffusion imaging allows to observe the water movement in the tissue, and this can be used to do inferences about the integrity of WM tracts in the brain. WM is formed by the neurons axons, that carry the electric pulses that neurons use to communicate with each other. The integrity of these fibres is really important for the correct functioning of our brain. In SCA2 we found WM abnormalities that correlated with the ataxia severity in the cerebellar WM and the middle cerebellar peduncle, but also in the parietal WM and the anterior corona radiata. Furthermore, previous studies have reported cognitive impairments in SCA2 including spatial working memory and attentional performance. We identified WM abnormalities that correlate with cognitive impairment in those patients in the parahippocampal area, inferior frontal and supramarginal gyri and the stria terminalis.

In our study with SCA2 patients we also evaluated the brain functional connectivity. The so-called resting state functional MRI (rsfMRI) is a MRI modality that explore the brain function without the need to ask the volunteer to perform specific tasks during the scanning. During rest, low frequency activity fluctuations reveal an intrinsic brain organization. In this way, several intrinsic networks have been reported including the default mode network which is anti correlated with the default mode network and is more active when a subject is engaged in a cognitive task. In the same way, specific functional connectivity changes can be explored using regions of interest usually called seeds, revealing the activity synchrony among two distant brain areas or between one seed and the whole brain. One advantage of rsfMRI, especially when working with patients with movement disorders, is that it does not rely on any task where performance can be reduced by the patients’ impairment. In our analysis, the SCA2 group showed a decrease in the functional connectivity within the cerebellum and an increase between the cerebellum and superior frontal gyrus and the parietal lobule. These abnormalities correlated with SARA and cognitive scores including learning and reversal and rule acquisition. In general, in degenerative diseases a decrease in functional connectivity indicates brain regions that are not working properly, mainly due to tissue damage such as atrophy. On the other hand, increases might indicate compensatory mechanism that allows the system to work even in the presence of tissue loss.

Overall, our findings contribute to a better understanding of the neural basis of the clinical and cognitive impairment presented by people with SCA7 and SCA2 and can be relevant for future treatments and/or therapies to help patients and their caregivers to have a better quality of life.

Extended information regarding this research can be found in the following scientific articles:


Continued on page 20
Study of Neuropathology…
Continued from page 19

2. Movement Disorders, 30(10), 1391-1399.

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Post-Doc Fellowship Award

**Neural Mechanisms of Cerebellar Function in Ataxia**

*By Marife Arancillo, PhD*

**mvaramcillo@gmail.com**

Baylor College of Medicine, Houston, TX

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

We started to dissect the role of a protein called mTOR in Friedreich’s ataxia (FA). In particular, we investigated how TORC1, one of the two protein complexes in which mTOR takes part, is activated in FA. TORC1 is a protein kinase complex and it adds phosphates to other proteins in order to activate or switch them off. TORC1 regulation is sensitive to nutrient availability and to the energy status of the cell. It also controls the biogenesis of mitochondria, the organelles that make energy and contain frataxin (FXN). Iron metabolism, which is altered in FA, has also recently been linked to TORC1 regulation through a protein called tristetraprolin (TTP). Our aim is to investigate if and how TORC1 is involved in the pathological processes causing FA, in order to shed new light on the still unclear mechanisms that regulate FXN physiology and identify new targets for FA treatment and cure.

During this first exploratory phase, we obtained data suggesting that TORC1 is differentially regulated in FA according to the tissue context.

In mice, where FXN has been deleted in the liver tissue, and in neurons derived from induced pluripotent stem cells from FA individuals, TORC1 activation seems to be downregulated. Similarly, in cultured cells where the expression of FXN has been reduced, TORC1 activation seems to be transiently reduced, followed by reactivation likely due to compensatory mechanisms.

In a mouse model with moderately reduced FXN levels (KIKO mouse), TORC1 activation varies in the different tissues, probably representing the combination of a direct effect of FXN deficiency and of compensatory responses.

Moreover, preliminary data suggest that TTP is downregulated in FXN-depleted conditions,
but it does not seem to correlate with TORC1 activation.  
In this exploratory phase, we defined the activation of TORC1 in different in vitro and in vivo models for FA. The next step is to identify in the different contexts, the proteins involved upstream and downstream of TORC1, and interconnect with iron homeostasis and FXN, in order to define novel targets for therapeutic intervention for FA.

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**Post-Doc Fellowship Award**

**Defining the Role of Mitochondrial DNA Mutations in the Neuronal Degeneration of Friedreich’s Ataxia**

*By Angela Bhalla, PhD*

*University of Alabama, Birmingham, AL*

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

Friedreich’s ataxia (FRDA), the most common inherited ataxia, affects approximately one in 50,000 individuals. FRDA-affected individuals have too little frataxin as a result of an expansion in the frataxin gene of a DNA repeat consisting of three DNA building blocks (nucleotides): guanine-adenine-adenine. The frataxin protein resides in the mitochondria, energy producers of the cell that contain their own DNA which is unique from the DNA located in the nucleus. Frataxin is involved in preparation of iron for use by cellular proteins through the synthesis of iron-sulfur clusters (ISCs). Without frataxin, cells are unable to maintain proper iron levels in their mitochondria, inhibiting the ability of mitochondria to perform normal functions, and leads to excess production of DNA damaging molecules that can mutate the mitochondrial DNA. To date, the extent of mitochondrial DNA damage in FRDA and the potential mechanisms involved are poorly understood. The National Ataxia Foundation Postdoctoral Fellowship I received allowed me to pursue a study defining the role of mitochondrial DNA mutations in FRDA progression. To determine the level of damage in mitochondrial DNA in control and FRDA cells, I measured mitochondrial DNA damage by quantitative polymerase chain reaction. To examine the consequences of low frataxin levels on the number of DNA mutations in the mitochondrial DNA of FRDA-affected cells compared to controls, I employed a next-generation sequencing technique. The results of the study revealed that lack of frataxin leads to an increase in the frequency of mutations in mitochondrial DNA, and demonstrated that FRDA cells have a delayed DNA repair response. Analysis of the level of DNA repair enzymes in FRDA cells provided evidence that the base excision DNA repair pathway may be involved in protecting mitochondrial DNA from acquiring mutations. We propose that over time, accumulation of mtDNA mutations in vulnerable FRDA-affected cells could hinder mitochondrial activity, ultimately leading to cell death.
Post-Doc Fellowship Award

Antisense Oligonucleotide (ASO) Treatment for Spinocerebellar Ataxia 3 (SCA3)

By Gautam Rajpal, PhD
grajpal@umich.edu
University of Michigan, Ann Arbor, MI

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

We identified two compounds referred to as Antisense Oligonucleotides, or ASOs, that were effective in reducing levels of the disease protein in a mouse model of Spinocerebellar Ataxia 3 (SCA3). The research was conducted in collaboration with Ionis Pharmaceuticals, which provided the ASOs. ASOs are currently used to treat certain human diseases and are being tested in clinical trials for several neurodegenerative diseases. When injected into the mouse brain, the ASOs we tested markedly decreased levels of the disease-causing form of the SCA3 protein (known as ATXN3) in many areas of the central nervous system known to be affected in the disease. While this was only a short-term study lasting one month, experiments are underway to assess the long-term effectiveness of these ASOs in SCA3 mouse models. Our study represents a promising first step toward potential disease-modifying therapy for humans affected by SCA3.

Post-Doc Fellowship Award

Developing the Novel microRNA-mediated Therapeutic Approach for Spinocerebellar Ataxia Type 6

By Yu Miyazaki, MD, PhD
yumiyazaki@uchicago.edu
University of Chicago, Chicago, IL

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

Spinocerebellar ataxias (SCAs) are a genetically heterogeneous group of dominantly-inherited neurodegenerative diseases characterized by progressive ataxia and Purkinje cell degeneration. To date more than 30 SCAs have been characterized, each being associated with distinct genes and mutations and therefore requiring individual therapeutic approaches.

SCA type 6 (SCA6) is one of the most common forms of autosomal dominant SCAs, representing 10–20% of patients with dominantly-inherited ataxia, and has an incidence of approximately

Dr. Gautam Rajpal

Yu Miyazaki
5/100,000 persons. Patients with SCA6 develop slowly progressive cerebellar ataxia with extensive selective cerebellar Purkinje cell degeneration, usually beginning at age 40–50 years. SCA6 is caused by mutation in the CACNA1A gene.

We recently discovered that the CACNA1A gene is bicistronic, i.e., it encodes two proteins, α1A subunit of the neuronal Ca2+ channel and a newly recognized transcription factor. We also showed that the mutant α1ACT, not mutant α1A subunit of the neuronal Ca2+ channel, is pathogenic in SCA6.

In our current study, we developed an early onset SCA6 mouse model using a viral gene delivery system to ectopically express mutant α1ACT, in order to test potential therapies. Our SCA6 model mice showed unambiguous disease phenotypes associated with the pathological and clinical features of patients with SCA6 much earlier and more reproducibly than those previously developed. We also identified microRNA-3191-5p, a small endogenous RNA, specifically inhibits the mutant α1ACT expression. Furthermore, viral delivery of microRNA-3191-5p protects from the SCA6 phenotypes in our model mice. We have established the proof of principle that viral delivery of a microRNA can rescue SCA6 phenotypes in a mouse model.

These findings are incorporated in the paper we are submitting to journals and the planned research grant applications. Also, we are now preparing the translation of our small RNA-mediated therapeutic approach for SCA6 into advanced pre-clinical studies.

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**DO YOU HAVE CEREBELLAR ATAXIA?**

**Would you like to participate in a sleep study?**

Massachusetts General Hospital is recruiting individuals with cerebellar ataxia for an in-home sleep study.

The study will span 7 days during which you will wear a watch that will record your activity levels. All procedures will be conducted in your home. On Day 1 of the experiment, we will conduct a sleep apnea assessment, and a brief assessment of your eye movements, speech, gait and mental status. On the evening of Day 6, you will perform a memory game and we will attach electrodes to your scalp, near your eyes, on your chin and leg to assess your sleep. We will then return on the morning of Day 7 to remove the electrodes and to re-rest you on the memory game. Finally, you will complete a set of questionnaires relating to your sleep habits and sleep quality, your mood and quality of life. The total amount of time for the experiment is 5 - 5.5 hours. You are also free to withdraw at any time, as well as to leave any questions unanswered if you choose to do so. Participants must be between 18-79 years of age and have been diagnosed with SCA5, SCA6, SCA8, SCA28, SCA35, ARCA1 or ARCA3, and be willing to temporarily refrain from sleep-affecting medications such as benzodiazepines, antidepressants, stimulants and sleep aids. Additionally, you must not have a history of cerebellar disease caused by isolated cerebellar injury, including cerebellar stroke (ischemic or hemorrhagic) or cerebellar tumor.

If you would like to participate in our research, or have any questions, please call: (617) 726-3216 or email: ataxiasleepstudy@gmail.com

Research conducted by: Dr. Jeremy Schmahmann, Massachusetts General Hospital, Department of Neurology, Charles River Plaza, 175 Cambridge Street, Suite 340, Boston, MA. Approved by Partners IRB (Study # 2015P000744).
Post-Doc Fellowship Award

Understanding Disease Development in Friedreich’s Ataxia in a Time-resolved Way: A New Cellular Model

By Tommaso Vannocci, PhD
King’s College, London, England

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

Friedreich’s Ataxia (FRDA) is a hereditary neurologic disease that is found in one case every 50,000 people. The symptoms are poor coordination and walking problems and the disease usually leads to severe ambulatory deficiencies, heart disease and diabetes. The disease is caused by a change of our genetic code which leads to the insufficient production of a specific protein, called Frataxin (FXN). This is an essential protein, in that its absence is incompatible with life, which lives inside the organelles in our cells that produce the energy necessary for life. Over time, insufficient levels of frataxin cause metabolic changes and severe cellular damages that affect predominately two tissues: the nervous system in the spinal cord and the heart.

Even though, being a genetic disorder, the disease is present since birth, the symptoms often become evident only at an older age, typically around 25 years of age. This is due to the slow but continuous accumulation of damages at the cellular level. Progression of the symptoms are slow but constant. The patients are confined to the wheelchair in 10-15 years from diagnosis and death occurs usually because of heart diseases in early adulthood. At the present, no effective cure for FRDA and only palliative treatments are available.

Although much has been done to understand the function of frataxin and its relation to the FRDA disease, its primary function is still unclear. Understanding of the disease causes has also been complicated by the difficulty of separating causes from secondary effects. Cells derived from FRDA patients have, for instance, been used in several different studies, in which samples have been “exposed” to the disease. However, in most cases the researchers have only looked at the effects after they had accumulated making it difficult to say anything on the early steps of FRDA and thus about its causes. We have instead designed a cellular model system which allows us to switch on/off at will the FXN gene and, therefore, to induce the FRDA symptoms. This means that we can address and describe specifically the very early phases of the disease. This will, in future, let us discriminate between the early stages of the disease and its secondary effects and hopefully also to understand more about the function of frataxin.

During the time span of the NAF project, the cellular model was successfully created using a range of genetic techniques that allow “tailored” gene modifications of desired regions of the genetic code of the cells. This developed molecular tool was used to completely delete the FXN gene and, at the same time, introduce a foreign, artificial FXN gene that can be “switched ON and OFF.”
approach has given us the possibility to maintain cells in a healthy state and to induce the FRDA-like condition at any desired time. The results of this project have been published in the Disease Models and Mechanisms journal. We are now in the process of characterizing the cells and observe the consequences. To create a complete picture of FRDA’s initial phases we have started several international collaborations that will allow us to use new biophysical techniques and instrumentation. This approach will hopefully highlight novel biomarkers that can be used for the early diagnosis of FRDA and help us to design novel treatments that should either completely cure or at least postpone the onset of the disease.

Research Grant Award

Web-based National Ataxia Database

By Susan Perlman, MD
University of California – Los Angeles, CA

The following is a research summary of a grant funded by the NAF for fiscal year 2015.

The Natural History Study of and Genetic Modifiers in Spinocerebellar Ataxias (ClinicalTrials.gov Identifier: NCT01060371), under the direction of Dr. Tetsuo Ashizawa, continues to recruit subjects and monitor changes in their neurologic examinations. Data are entered into the National Ataxia Database, hosted at UCLA under the direction of Professor Jeanette Papp in the Department of Genetics.

The Natural History Study has enrolled over 400 individuals with Spinocerebellar Ataxia types 1, 2, 3, and 6. There are over 15,000 patient forms currently entered in the National Ataxia Database. In 2016 the University of Colorado was added as a participating site, bringing the total current sites to 13.

The Database has served as a valuable repository and analysis tool for data collected in this important collaborative project.

Have You Remembered NAF in Your Estate Planning?

When you make or revise your will or trust, or review your life insurance contracts or retirement funds, please consider naming the National Ataxia Foundation among the charities included as beneficiaries. The use of the following language ensures that your gift is directed appropriately: I bequeath ___% of my estate (or fund) to the National Ataxia Foundation, a 501 (c)(3) non-profit organization located at 2600 Fernbrook Lane No, Suite 119, Minneapolis, MN 55447-4752. Federal Tax ID# 41-0832903

For further information about naming the National Ataxia Foundation as a beneficiary, contact Joel Sutherland at joel@ataxia.org or (763) 553-0020. Thank you for your support of the important work of the Foundation.
Research Grant Award

The Effect of Cerebellar Abnormality on Hearing in Individuals with Spinocerebellar Ataxia (SCA) and Friedreich Ataxia (FRDA)

By Gary Rance, PhD
University of Melbourne, Parkville, Victoria, Australia

The following is a research summary of a grant funded by the NAF for fiscal year 2014.

The findings of this study indicate that timing information in the auditory pathway is disrupted by both Friedreich ataxia (FRDA) and spinocerebellar ataxia (SCA). This deficit (which affects the clarity of sounds rather than whether or not they can be detected) is important for everyday communication and quality of life as many common speech sounds (such as the consonants /p/ & /b/ or /t/ & /d/) can only be differentiated using timing cues.

The mechanisms causing disruption of timing perception appears to be different for the two disease groups. FRDA mainly disrupts neural firing patterns in the auditory nerve/brainstem while SCA affects timing decisions made at the cerebellum. Further teasing out of the auditory changes caused by FRDA & SCA will underpin the development of treatment strategies and may prove useful in measuring the effects of drug therapies or other therapeutic interventions.

Dr. Gary Rance

iSearchiGive Is Good for the NAF

iSearchiGive.com is a search engine powered by Yahoo! Search and iGive.com. It is the Internet’s first online shopping mall where a portion of each purchase is donated to a charity of your choice. When you use iSearchiGive to search the web, your favorite cause receives money for every qualified search.

iSearchiGive.com is totally free, with no hidden fees and provides valuable support for the important work of the National Ataxia Foundation.

Please sign up today and indicate that the National Ataxia Foundation is your favorite cause.

Study of Cardiomyopathy in Friedreich’s Ataxia Patients

A new IRB-approved study at Weill Cornell Medical College on Friedreich’s Ataxia is recruiting patients between 18 to 30 years old. The purpose of the study is to compare different tests and procedures and to evaluate their usefulness in assessing the cardiac manifestations of FRDA. The study requires a two-day, overnight stay in New York City. For more information contact: Michelle Yuan at (646) 962-2672 or miy2006@med.cornell.edu.
THE NAF BOARD OF DIRECTORS ALONG WITH THE NAF SOUTHCENTRAL REGION WOULD LIKE TO INVITE YOU TO ATTEND THE

National Ataxia Foundation
60th Annual Ataxia Conference

March 10-11, 2017

The Grand Hyatt San Antonio is pleased to provide the facilities for the 2017 Annual Ataxia Conference (AAC)

Join us in San Antonio, TX for the Annual Ataxia Conference!

Room Reservations-Begins November 2
Room reservations for all room types at the Grand Hyatt will be made available starting November 2.

Reservations at group rate will be available until February 13, 2017.

The NAF group rate starts at only $169 +tax for Standard Rooms.

ADA room reservations must be reserved through the NAF office.

Reservations for ADA rooms begin on November 2 at noon CST by contacting (763) 553-0020 or lori@ataxia.org.

Calls or e-mails prior to noon CST on November 2 to reserve an ADA room cannot be honored.

Standard room reservations at the Grand Hyatt can be made at https://resweb.passkey.com/go/2017AnnualConference

For guests who prefer to phone in their reservation call Hotel Reservations at 888-421-1442 and ask for the National Ataxia Foundation’s group rate which is under the group name, “2017 Annual Ataxia Conference.”

Meeting Registration-Begins November 2
Registration for the 2017 NAF AAC will open on November 2. You are encouraged to register before January 30, 2017 to receive the early registration discount rate. In addition, members of the NAF pay a lower registration fee to attend the Annual Ataxia Conference. If you are not currently a member of the Foundation go online at www.ataxia.org or call the NAF office at (763) 553-0020 to become a member or renew your membership. For the latest information on conference registration, program schedule, and area information keep checking the NAF’s website www.ataxia.org. Note: The conference will end with the banquet on Saturday night.

2017 NAF Annual Ataxia Conference "Support Our Conference" Campaign
http://ataxia.donorpages.com/2017AACLeterWritingCampaign/

For more information on San Antonio visit http://visitsanantonio.com/.
The National Ataxia Foundation (NAF) Board of Directors and the National Ataxia Foundation Southcentral Region invite you to join us for the NAF’s 60th Anniversary at the NAF 2017 Annual Ataxia Conference (AAC). The AAC is being held at the Grand Hyatt in San Antonio, TX.

The 2017 AAC will bring together the Ataxia community to not only meet and learn from world-leading Ataxia researchers and clinicians, but also to build new friendships and reunite with old friends. Come and be part of the largest Ataxia gathering in the world to learn, share, network, have fun, and observe the NAF’s anniversary!

The complete conference schedule, events and registration forms will be listed in the winter 2016-17 issue of Generations and on the NAF’s website when available. General Sessions and Birds of a Feather Sessions will be held on March 10-11, 2017. The conference will conclude with the Saturday evening banquet celebration in order to address the trending decrease in attendance of the sessions scheduled on Sunday. We hope this change will allow many attendees to depart the conference with more ease and flexibility on Sunday and other attendees more time to visit with other attendees and/or experience the local attractions.

**Featured Speakers**

**Huda Zoghbi, MD**

Dr. Huda Zoghbi enrolled in medical school at American University in Beirut and finished her medical training at Meharry Medical College, in Nashville, TN. She found herself drawn to disorders that affect the brain. With Dr. Harry Orr and her collaborators, Dr. Zoghbi unraveled the genetic underpinnings of a number of devastating neurological disorders, including spinocerebellar Ataxia type 1 (SCA1). Their discoveries have provided new ways of thinking about more common neurological disorders and could lead to better treatments. Internationally renowned scientist, Director of the Jan and Dan Duncan Neurological Research Institute

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**AAC Exhibitors and Sponsors Wanted**

The National Ataxia Foundation is looking for companies or individuals who have products or services that would be helpful for those with Ataxia to submit an exhibitor application to exhibit at the National Ataxia Foundation 60th Anniversary Annual Ataxia Conference. The 2017 conference will be held in San Antonio, TX on March 10-11. Please e-mail joan@ataxia.org for an exhibitor application.

The NAF is grateful to those organizations that have provided generous support of the Annual Ataxia Conference. Please consider being a sponsor of the 2017 Annual Ataxia Conference. For more information on becoming a sponsor please e-mail Joel Sutherland at joel@ataxia.org.

If you are affected by Ataxia or are a caregiver and know of a product or service that has been helpful for you, please let us know by calling (763) 553-0020 or e-mail joan@ataxia.org.
at Texas Children’s Hospital and professor at Baylor College of Medicine, Dr. Huda Zoghbi is a highly sought after speaker at scientific meetings around the world. The National Ataxia Foundation is fortunate and delighted to have Dr. Zoghbi present at the Annual Ataxia Conference in 2017. Don’t miss this opportunity to hear her presentation titled: “Approaches to developing therapies for Ataxias.”

Brent Fogel, MD

Brent Fogel MD, PhD is an Associate Professor in the Departments of Neurology and Human Genetics at the David Geffen School of Medicine at UCLA where he also serves as the Associate Director of the Neurogenetics Program. Additionally, Dr. Fogel is the current Chair of the Neurogenetics Section of the American Academy of Neurology. Dr. Fogel’s research studies basic molecular mechanisms of neuronal function to understand how impairment can lead to neurodegenerative conditions such as spinocerebellar ataxia. Clinically, Dr. Fogel directs the UCLA Neurogenetics Clinic and also treats patients with various disorders of balance and coordination at the UCLA Ataxia Center. In addition, he directs the Ataxia and Neurogenetics Biobank Program where he is using genome-wide methods (including exome sequencing) to identify rare and novel causes of neurodegenerative disease, particularly cerebellar ataxia. He has authored multiple research and clinical articles, reviews, and book chapters on spinocerebellar ataxia, clinical neurogenetics, and neurodevelopmental disease. Dr. Fogel’s work has been funded by the American Academy of Neurology / American Brain Foundation, the National Institutes of Health, and the National Ataxia Foundation. The National Ataxia Foundation is excited to have Dr. Fogel update members on his important work during his presentation, “Advances in the Genetic Diagnosis of the Cerebellar Ataxias.”

Hotel Reservations

All ADA rooms must be reserved through the NAF office starting on November 2 at noon CST by contacting (763) 553-0020 or lori@ataxia.org. ADA rooms cannot be reserved through the hotel. Availability of ADA room types are limited. If you need ADA equipment you are encouraged to bring those items with you or make arrangements to rent equipment locally. The NAF is unable to provide ADA equipment however the Grand Hyatt may have a limited number of shower chairs, grab bars, or detachable shower heads available. Be sure and request these items when making your reservation if needed.

Starting on November 2 standard room reservations at the Grand can be made online at https://resweb.passkey.com/go/2017AnnualConference. For guests who prefer to phone in their reservation, please call Hotel Reservations at 888-421-1442 and ask for the National Ataxia Foundation’s group rate which is under the group name “2017 Annual Ataxia Conference.” Reservations at the group rate will be available until February 13, 2017. The NAF group rate starts at only $169 + tax for Standard Rooms.

Continued on page 30
Conference Registration

The conference registration is available online starting November 2 and in the Winter 2016-2017 issue of Generations. Please fill out the registration form completely, including your travel information, as we need all the information to finalize plans. When registration opens, you are encouraged to register before January 30, 2017 to receive the discounted early registration rate. General registration rates apply after January 30, 2017. Registrations after March 3 will only be accepted on-site at the conference. (Additional “on-site” registration fee will apply.) If you are bringing an attendant, please register together on the same registration form. Each person who is planning on attending daily sessions, the reception, or banquet needs to register. Event entry will be allowed only with properly registered name badges. The conference registration fee includes attendance at all the sessions, light appetizers at the Friday evening Meet & Greet Reception and a plated meal at the Saturday evening Banquet.

Registration Fees

Before January 30:
- NAF Member .................................. $125
- Non Member ................................. $180
January 30–March 3:
- NAF Member ................................. $150
- Non Member ................................. $205
After March 3 – On Site:
- NAF Member ................................. $200
- Non Member ................................. $255

No refunds of registration fees for cancellations received after February 24. Registration at the door is not recommended.

Member Registration Discount

Being a member of the National Ataxia Foundation has its benefits – one benefit is paying a lower registration fee for the Annual Ataxia Conference. If you are not currently a member of the Foundation or if your membership renewal is coming soon visit www.ataxia.org to become a member or renew your membership online. If you are uncertain of your membership status, please inquire by contacting the office at (763) 553-0020 or joan@ataxia.org. This will prevent unnecessary extra fees or errors in your membership status when you register for the 2017 Annual Ataxia Conference. Thank you for taking the time to renew or become a member of the National Ataxia Foundation.

Conference Deductions by Attendees

Medical expenses.

Amounts paid by an individual for expenses of admission and transportation to a medical conference relating to the chronic disease of the individual’s dependent are deductible as medical expenses under section 213 of the Code (subject to the limitations of that section), if the costs are primarily for and essential to the medical care of the dependent. The cost of meals and lodging while attending the conference are not deductible as medical expenses under Code section 213.

Travel Grant Program

Because of the generosity of several donors, the National Ataxia Foundation is able to offer Travel Grants to help with a portion of the travel costs associated with attending the conference. Adults or children with Ataxia are eligible to apply for a travel grant. Individuals interested in the program are required to submit a Travel Grant application. Applications will be accepted until Jan. 6, 2017. Travel Grant applicants will be notified of the status of their application after the application deadline and after all applications have been reviewed. Visit the NAF website, www.ataxia.org, to download the application. If you would like an application sent to you in the mail, contact Lori Shogren at (763) 553-0020 or lori@ataxia.org to request one.

2017 AAC
Continued from page 29
How the National Ataxia Foundation and the Annual Ataxia Conference Have Helped Me

Submitted by Michelle DeCiantis

More than 15 years ago I started to feel like something was different and each year that passes, more questions are added to my story. I have been diagnosed with SCA, an unknown type, with an overlapping of upper motor neuron unknown disease. I’ve seen doctors from UCLA to the University of Michigan. I work hard to get involved in any clinical trials or research studies that I can in order to help them find out about these rare illnesses in order to help the next generation.

The thing I remember the most is when I first went to the doctor and heard the word Ataxia. I had never heard of it before and I was unaware that there were support groups or that there was a National Ataxia Foundation! Since finding those things out, I have made many friends who are really like family. I have built close relationships with them over the years and we stay close. Attending the Annual Ataxia Conference with my newfound family members is what helps me get through each day, to connect through Facebook or pick up the phone to talk to one of my sisters or brothers, that I have adopted through this organization and who I share this diagnosis with, has been a big part of my life line.

No matter how much you educate someone or tell them about what you’re going through, no one really understands except someone who is experiencing it themselves or at least experiencing something close to what you have. It is because of the NAF, allowing me to meet these wonderful people, to attend the conference with the help of a travel grant and discounted hotel rates, I have been able to meet with the doctors and hear the specialists in the field talk about the progress that is being made with scientific research. It helps me fight harder and to keep going to help them find treatments and cures for these diseases.

Explore San Antonio

Request a free “Visit San Antonio” guide book, provided by the San Antonio Convention and Visitor’s Bureau, to help you plan your trip to the 2017 NAF Annual Ataxia Conference.

This complementary book is filled with information about the city, food, arts and major events for 2016-2017. This free guide can be ordered by calling 1-800-447-3372, or may be requested or downloaded instantly by following this link: http://visitsanantonio.com/english/Leisure-Guide-Request-Form. Please allow up to four weeks for your guide book to arrive.

To find out more about the 2017 NAF Annual Ataxia Conference, please visit the NAF’s website, www.ataxia.org.
Our First Encounter with the National Ataxia Foundation and the Annual Ataxia Conference

Submitted by Cathy & Joe DeCrescenzo

Ten years ago my husband Joe was diagnosed with hereditary Spinocerebellar Ataxia, SCA2. That marked the day our lives changed and would never again be the same. Since then, our youngest daughter tested positive for SCA2 as well.

We had time to process the diagnosis and vowed not to let it change who we are or alter our daily lives. We found a neurologist who specializes in movement disorders and Ataxia.

We also joined two support groups, one at Johns Hopkins Medical Center, and the other with the Chesapeake Chapter NAF. We met so many wonderful, inspiring people who were struggling with Ataxia but always stayed positive, which we admired.

Our next step was to join the National Ataxia Foundation (NAF). We are thankful we took that step. Our first encounter with the NAF was attending the Annual Ataxia Conference (AAC) five years ago in San Antonio, TX. I must admit, it threw me for a loop emotionally and I did spend the first couple days inconsolable at times. The first conference had a huge impact on our lives. It made us realize we are not alone and it gave us strength to move forward. It allowed us to witness how various age groups deal with Ataxia. So many young people in wheelchairs and with rollators were having the time of their lives, socializing with old friends and making new friends. They were laughing, smiling, and, yes, even dancing at the banquet ... it was incredible! We have become advocates, started our own support group in Delaware, and organized many fundraisers for the NAF. We attend the annual Ataxia conferences, where a wealth of information and updates on current research towards a treatment/cure for this rare disorder is available. Also, we volunteer at the conference each year and Joe is now serving on various committees. Research is essential to find a cure; therefore, Joe has participated in numerous research studies over the years.

Our message to the newly diagnosed, and those who feel alone and overwhelmed, is to do everything in your power to stay positive. Some days will be difficult, though if you learn to accept the hand you’ve been dealt with dignity and to adapt your life style as the Ataxia progresses, it will be easier to get through the more challenging times. Please DO NOT loathe in self-pity nor complain ... we are all in the same boat trying to stay afloat, and negativity is exhausting and will NOT change anything. Control your own destiny by continuing your daily life as normal and safely as possible. Join a local support group – get involved! Attend the Annual Ataxia Conference ... it’s an awesome experience, one you will never forget. Hopefully, what you take away from the conference will help you move forward on your Ataxia journey, knowing you are never
alone. The NAF has undoubtedly helped us immensely to accept the Ataxia in our family, and with hope in our hearts, we pray a cure will be found in our daughter’s lifetime. PLEASE become a member of the NAF today!

We have met so many amazing people through the NAF who we can truly call our friends, and each year we are excited to welcome new friends to the NAF family. Throughout the year, we keep in touch with our friends between conferences, from across the pond to across the country. We count down the time until we meet again.

Hope to see you at the next NAF AAC! In the meantime, stay strong.

You can also view a video by Cathy here: [www.youtube.com/watch?v=7PbC7lMnlYI](http://www.youtube.com/watch?v=7PbC7lMnlYI).

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**Arnie Gruetzmacher Annual Ataxia Conference Travel Grant Fund**

Arnie Gruetzmacher’s passion to help the Ataxia community never wavered, particularly when it came to the NAF Annual Ataxia Conference. He would spend countless hours every year helping to create a venue to bring Ataxia families together to share, learn and network.

In Honoring the Memory of Arnie, the “Arnie Gruetzmacher Annual Ataxia Conference Travel Grant Fund” has been established. This Fund will help provide travel grants to those with Ataxia who would otherwise not be able to afford to attend the Annual Ataxia Conference.

To support the Arnie Gruetzmacher Annual Ataxia Conference Travel Grant Fund, please visit the NAF’s web site at [www.ataxia.org](http://www.ataxia.org).

Thank you for furthering Arnie’s vision and fulfilling his legacy.

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**NAF’s Travel Grant Program Needs Your Support**

“Being around other people with ataxia at the meeting helps me feel less alone.”

The National Ataxia Foundation’s Annual Ataxia Conference (AAC) connects the ataxia community. The meeting program is designed to foster learning and understanding by providing informative presentations about ataxia research and on living with ataxia. Connecting with those who understand and face the same challenges is also an important component in which individuals with ataxia, their family members, and caregivers have the opportunity to share and network.

You can help someone with ataxia attend the AAC by making a donation to the NAF AAC Travel Grant Program. Your gift to the AAC Travel Grant Fund will make an immense difference in someone’s life. Thank you for your support and for making the ACC experience possible for an individual affected by ataxia who would not have been able to attend without your help.
2016 Annual Ataxia Conference Photos

Barbara Ofenstein and Charlotte DePew

Emily Phillips and Susan Penn, sisters from Banning, CA

Lisa Cole and Ronnie Orlandi

Chris & Lyn Tippett and son Guy from the UK

Laura Ranum, PhD; Dave & Linda Zilles

David Henry, Kevin Halbert and Mike Haydon

AAC speakers

Tetsuo Ahsizawa, MD; Larry Schut, MD; and Gülin Öz, PhD

John Mauro

Amanda Hernandez and Aunt Martha Maya Castillo
Exciting Announcement from the NAF Board of Directors

The NAF Board of Directors is pleased to announce that after reviewing many resumes and interviewing several candidates, Joel Sutherland has been named the new Executive Director of the National Ataxia Foundation. As announced in the Summer issue of Generations, Joel was hired earlier this year as the National Director of Development. Joel brings an abundance of energy, along with a depth of experience in marketing and fundraising and will serve in both positions during the transition after Mike Parent’s retirement. We welcome Joel and look forward to working with him in expanding our development efforts and managing the day-to-day operations of the National Ataxia Foundation. He joins the NAF Board in his commitment to continue Ataxia research and outreach to Ataxia families.

The Board had received notice from Mike Parent that he would retire as Executive Director of the National Ataxia Foundation at the end of July. Mike’s tenure at NAF was both lengthy and productive. A lunch was held for Mike during his last week on the job when several numbers were mentioned that offered reflection on Mike’s time at NAF: 34 … 12 … 4 … 4.

Mike was associated with NAF for 34 years ... and served as Executive Director for the past 12 years. In June of this year, NAF was awarded a 4 Star rating by Charity Navigator ... the 4th consecutive year that NAF has received the highest of Charity Navigator’s ratings – a record shared with just a handful of other non-profits. This accomplishment is a reflection of Mike’s contributions over the past several decades. We wish Mike well in his retirement.

Change can be a challenge ... but also an opportunity. Advances in research have heightened the interest of pharmaceutical companies in searching for treatments and a cure for Ataxia. To ensure that NAF will be ready to assist in finding those treatments and to position our organization to be a key partner in this search, the Board approved a five-year Strategic Plan establishing four broad goals:

1. **Increasing revenues** to fuel organizational growth to build on our history to become a stronger, more vital organization.

2. **Increasing awareness and recognition** of Ataxia and NAF to strengthen and grow support for NAF’s mission.

3. **Becoming “clinical trial research ready”** to help move us towards effective treatments and a cure for all Ataxias.

4. **Building internal capacity through technology** to increase organizational efficiency and effectiveness.

We thank all of you, the Ataxia community, for your past involvement and support. We look to the future with optimism, while recognizing the challenges Ataxia families face on a daily basis. We have a long road ahead, but we know that long journeys are easier when taken together.

Board of Directors
National Ataxia Foundation

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**Deadline**

*Generations* is published quarterly by the National Ataxia Foundation. The deadline to submit materials for the Winter edition is Friday, October 28.

Please submit content by e-mail to *joan@ataxia.org*, or by mail to the National Ataxia Foundation, 2600 Fernbrook Lane, Suite 119, Minneapolis, MN 55447-4752.
Pearls of Wisdom: Holiday Shopping

Submitted by Martha Elliott

I’ve just finished my holiday shopping, all in one day and with no help. Through the year I collect mail order catalogs. All it takes is a single order to one catalog to get going. They sell their mailing lists to each other, so very quickly one is receiving catalogs from places you’ve never heard of. I collect them, skim them, and pass along those I’m uninterested in to my cleaning lady. Information on everything from fancy foods to cleaning aids comes right to my door, and their products are delivered by mail as well.

Here’s how I use them:

• Since I have no use for some (children’s, educational, duplicates) they’re given away at once.
• I mark pages with items of interest by turning down the page corner, then stack and collect them.
• As I go through them, I comparison shop, not only for the item, but also for shipping costs and return policies. Prices per item vary by as much as double, and shipping costs can add up depending on how they’re figured.
• All catalogs I’m not using are discarded (they’ll be back in a few months).
• Phoning is free to 800 numbers, so I call in orders and get info on specials and back orders.

Here’s my technique for preparing orders:

• Choose a relaxed time. Many companies have 24-hour ordering (it says on the order form).
• Choose a catalog and tear out the order form.
• Decide which credit card to use. It’s easier to use the same card for all orders.
• List all pages of desired items in order on the form. My writing is now illegible, so I just read off the printed stock numbers of items I’m buying, turning the pages as I go. The order taker will respond with the name of the item, so I can verify that it’s correct.
• At the outset I tell the order taker that I have a speech impediment, and that they should ask for any clarification they need. I also listen carefully to be sure they have correct address, credit card, etc., numbers.

The order taker will have questions, so answer slowly and courteously. If they’re impatient or otherwise give you a hard time, speak with the customer service representative (an 8 – 5 job), don’t try to correct them yourself. I’ve never once had a problem. Most companies have good service as their goal and will bend over backwards to make things right, so if they’re not right, just give them a call.

Back to those order forms. As purchases come to you, check them against the orders to be sure quantities, sizes, colors, and personalization is right. Only when you’re completely satisfied should you discard the original forms. I keep the forms for items I may wish to reorder – saves time and effort.

Use the catalogs to shop for things you want/need year round, and be inventive. If it fills a need for you, even if it’s not being advertised for that purpose, go for it. Some items would be handy, but consider how often you will use them, or could you make do nearly as well with another item you already have. I recently saved 300% on an item by considering if I really needed the more expensive one or if the cheaper one would suit as well.

Starting late in December many of these catalogs will come around again, this time with winter sales. Just don’t stock up on perishables for next year, but there will be some real bargains in there.

If I can help, e-mail me at docelliott268@gmail.com.
Walking west, watching my feet move me forward, one step at a time ... I chase the shadows of my trekking poles, as the morning sun rises behind me.

Always moving, Ataxia will not stop me, always walking.

Listening to my inner voice ... move, move, move, don’t stop.

Keep going, keep on keeping on, keep walking, keep moving.

Inspiration comes from above, urging me on.

Now going back, east into the rising sun, sweat on my brow, leg strength waning, my shadows are chasing me home.

God is with me, all around me ... my constant companion ... walking with me, with me and my Ataxia.

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Memory
By Allida “Ami Angel” Aplanalp, 9-2011

Folded deep within my mind
I never thought I’d come to find
Memories so fresh and clear
of all my dreams of yesteryear...
“Just be positive!”, they’ve said
The words thrash throughout my head
“Thanks, now I can feel guilty, longer,
for not being emotionally stronger.”

If it were that simple I’d be there in that mindset
And guess what, I am, when I’m not full of regret.

Never gave much thought of this position
Never even really knew of this condition
I still hold the mental pictures of me
And each of the things that I wanted to be.

If you would like to contact Ami please e-mail her at oracle333@comcast.net.

Above: Ami with shooting instructor Vaughn.
International Ataxia Awareness Day
— Friday, September 25, 2016 —

How Did You Participate in IAAD?
Tell us how you recognized International Ataxia Awareness Day (IAAD) this year. Share a photo with us to be included in a future issue of Generations. Please e-mail your story/photo to joan@ataxia.org or mail to the National Ataxia Foundation, Attn: Generations Editor, 2600 Fernbrook Lane, Suite 119, Minneapolis, MN 55447-4752.

Sharing your stories on how the day was recognized could live on in a future issue of Generations. Please send us your articles, photos and proclamations.

Thank you to all of you that have organized, participated and promoted awareness of Ataxia. Together we can make a difference.

Share Your Story
Personal stories from those affected by ataxia are an important part of Generations. Stories submitted should be no longer than 1,200 words. If possible, tell how NAF has made an impact in your life or situation. Submit stories to joan@ataxia.org to be considered for publication.

E-mail Blasts
E-mail blasts from the National Ataxia Foundation are sent out periodically on ataxia research, events and other timely issues of interest. Please email your e-mail address to joan@ataxia.org so you don’t miss out on important information.
The CNY Ataxia Support Group will be present at the walk for Dave on Saturday, August 6 in Liverpool, New York.

Treasure Coast Ataxia Support Group
Submitted by Roberta Santa Croce & Lisa Cole

We had 11 people come to our meeting and had one guest speaker. We had a resource table at the side of the room with various “must know” Ataxia-related materials.

The meeting got off to a bit of a late start due to all the Treasure Coast Ataxians having to travel an hour-and-a-half or more to get to the meeting. Once kickoff started, the group was a force to be reckoned with!

Rose is Lisa Cole’s Personal Trainer. She works with Rose rather than a Physical Therapist. There was a very animated discussion about diet and exercise as it related to our symptoms. Also, Lisa volunteered to forward any e-mail questions to Rose. Hank introduced the group to ataxia.org. He used the projector to navigate the site for everyone and go through the “What is Ataxia” PowerPoint presentation.

There was so much energy in the room at the conclusion of the meeting it was difficult to draw to a close, with everyone realizing we
wouldn’t resume for another two months. Suggestions were passed about sharing e-mails.

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**Chi-Town Ataxia Friendship Support Group**
Submitted by Shannon Dunphy Lazo

On June 13, members and friends of the Chi-Town Ataxia Friendship Support Group, in Chicago, met at the Lincoln Park Zoo near Lake Michigan. It is a great venue with wide, paved paths and wheelchair rentals. Eighteen of us enjoyed seeing the animals, visiting with each other and picnicking. The zoo outing was a great chance to get out of the house and socialize. Lots of fun! Also, the weather was great.

We were glad to host the Chicago Support Group too! We plan to have more of these outings in Chicagoland.

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**Central PA Ataxia Support Group**
Submitted by Mike Cammer

We held our last meeting on July 30 with a good group of 10. Our speaker, Jordan Taylor PhD, a professor and ataxia researcher from Princeton University, gave an outstanding presentation. He presented “Discovering new strategies for motor control: implications for...”

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**Genes in Inherited Neurologic Disorders Study #HUM00041414**

Dr. Burmeister at the University of Michigan is recruiting individuals with ataxia for the research study Genes in Inherited Neurologic Disorders. This study is designed to find what and how changes in the genetic material (DNA) cause inherited neurologic disorders, such as ataxia. We are recruiting individuals with inherited ataxia, their affected relatives (such as a brother or sister, a cousin, or a parent), and their unaffected family members, where possible. We are currently recruiting persons with an unknown form of ataxia, so at least one affected in your family should first be tested for the most common known causes of ataxia and found to be negative. We are recruiting both subjects with or without other affected family members.

In this study, you will be asked to provide information about your symptoms and diagnosis, if other relatives are similarly affected, and about your ethnic background. You will also be asked to donate a blood sample (up to 8 teaspoons of blood) for DNA testing and related experiments. The blood sample can be drawn by your local physician; you will not need to travel to the University of Michigan.

The lab has already identified several novel ataxia genes, and additional cases with newer known ataxia genes as well as mutations in genes causing other diseases involving ataxia and other, seemingly unrelated, symptoms such as tooth problems, although most subjects in our study have ataxia as main symptom.

More detailed information about this study is available in the consent forms: Affected Subjects Consent, Unaffected Relatives Consent.

If you would like further information or are interested in participating, please contact:

Dr. Margit Burmeister, PhD or Dr. Erin Sandford
Molecular & Behavioral Neuroscience Institute, University of Michigan
5063 BSRB, 109 Zina Pitcher Place, Ann Arbor MI 48109-2200
Telephone: (734) 6472186; (734) 615-3359
E-mail: margit@umich.edu or esandfor@umich.edu
neurorehabilitation.” In a nutshell and in layman terms: Why do we have brains? We really value movement and movement is really complex.

The first question, “Why do we have brains?” It is the only way to interact with the world! Seemed simple enough, until Jordan went into a little more detail on how complex just moving your arm can be. He then talked about how “We really value movement.” What he means by that is the millions we pay professional athletes and the billions we spend on stadiums and arenas to watch them effortlessly perform their respective sports.

But do we really value movement? Most people take activities of daily living for granted. Those activities are priceless to those of us with Ataxia and other movement disorders. Just think back to how simple and effortless life seemed before Ataxia. I never gave walking, eating or reaching for my toothbrush a second thought, much less going to the bathroom in the middle of the night!

Next Jordan turned “movement is really complex” into a really complex mathematical equation! Seems simple enough – see your toothbrush, reach for your toothbrush and brush your teeth – not! It’s more like Is(q)q+Gs(q,q) q+E(q,q)=C – see how simple it is! We saw a short video clip of engineers using robots to replicate human movement. Some were able to perform basic functions, but all failed at achieving the graceful movements of the human body. We moved on to the three main areas of the brain and what they do to control movement, the cerebellum, basal ganglia and motor cortex. It was very interesting to see the disproportionate amount of your motor cortex is used to control just your hands and mouth. How your cerebellum is used for coordination, but not necessarily needed for movement and how the basal ganglia is used for selecting the proper movement to complete a task. Truly amazing how the brain (human computer) generates data and puts it all together to create movement. And if that wasn’t enough, he discussed the hippocampus and its importance for the brain in remembering and replicating movement.

I have participated in some of Professor Taylor’s research at Princeton, if you have ataxia and would like to participate, you can contact him at jordanat@princeton.edu.

We would like to wish “fair winds and following seas” to Mike Parent as he starts his journey into retirement. It was a pleasure and I hope our paths cross again!

Western Washington Ataxia Support Group

Submitted by Sherry McLaughlin

Our support group met for the first annual Summer picnic in the beautiful city of Auburn, WA. We got right down to business with eating, then moved on to some informal conversation about our group. As always, friendships were established and the laughter was plentiful. According to new member Sunny Prom, “It’s such a breath of fresh air to spend time with folks whom you don’t feel judged by.”

Western Washington Ataxia Support Group

We ended our “name the group” contest with an award to Linda Murrell for her winning nominee, “Western Washington Ataxia Support Group.” Linda won some NAF merchandise. We also gave a big round of applause to Pat Boyles for finding and booking this great location.

Continued on page 42
Future events will include a wheelchair accessible tethered hot air balloon ride (25 feet up and fully attached to the ground) and a Seahawks football viewing party (TBD).

Our little start-up group has grown to 30 members in our six months of existence. If you live in Western Washington, come on out and join us. And watch for our new website and Facebook page coming soon.

Denver Area Ataxia Support Group

Submitted by Charlotte DePew

Among our quarterly meetings, July often has a low attendance, but not so this year. It was great to see over 35 people in the room buzzing with energy to share, learn and be among friends/other who understand and share mutual frustrations, joys, triumphs, etc. Or maybe, attendance was up because we had not met since January due to a severe blizzard that forced the cancellation of our April meeting.

Dr. Terry Chase came from Grand Junction as our guest speaker. Her up-beat style had us getting involved in savoring the present moment through one or more of our five senses as a means to relax, meditate or have a “Zen” moment. The idea is that anyone can do this, plus improve physical and mental health.

Charlotte shared news from the NAF, the great AAC Orlando Conference, her award, and plans for the Sixth Annual Denver Run, Walk ‘n Roll in Honor of Anne Killan. Anne succumbed to heart failure due to her Friedreich’s Ataxia about a year ago.

Arizona Ataxia Support Group

Submitted by Angela Li

On Saturday, August 13, the Arizona Ataxia Support Group met in Phoenix. We learned so much from Dr. Brian Rice of Red Mountain Chiropractic, who came to give a health presentation and talk about exercise and genetics.

We gave updates on our upcoming Arizona Ataxia Extravaganza and collected baskets for the silent auction. After the meeting, we went to a restaurant for additional socializing and to enjoy some delicious food.

The Arizona Ataxia Support Group

Attention Support Group Leaders

The end of the year is almost here. Don’t forget to submit your 2017 planned meetings and activities by October 28 to lori@ataxia.org.

Thank you for your time and effort on behalf of those with Ataxia and their families!

Ask About Matching Gifts

Many employers will match your gift to the National Ataxia Foundation through a Matching Gifts Program. This valuable benefit will allow you to have twice the impact on the lives of families touched by Ataxia.

Please ask your employer if they have a matching gifts program. If they do, your gift and the gifts of your co-workers will double in value. Thank you for your support.
**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"Wx8"Hx1"D $5
- Original NAF IAAD T-Shirt S & XXXL only $10
- NAF Baseball Cap (White or Blue) $10
- NAF 50th Anniversary Coffee Mug $3
- 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
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- 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

<table>
<thead>
<tr>
<th>Description</th>
<th>Qty.</th>
<th>Size</th>
<th>Each</th>
<th>Total</th>
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<tr>
<td>IAAD T-Shirt</td>
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<tr>
<td>IAAD Sweatshirt</td>
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<tr>
<td>NAF Polo Shirts</td>
<td>$10</td>
<td>SALE ... Limited supply!</td>
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<tr>
<td>NAF Denim Shirt</td>
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<tr>
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<td>$1</td>
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<tr>
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<td>$1 ea. or 6 for $5</td>
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<td></td>
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<tr>
<td>NAF Ataxia Awareness Band, Reflex Blue</td>
<td>One size $1 ea. or 3 for $2</td>
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<td>NAF Ataxia Awareness Ribbon Magnet</td>
<td>$4</td>
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<tr>
<td>&quot;Know Ataxia&quot; Backpack</td>
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<td>NAF Lapel Pin</td>
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<td>Magnetic Power Clip</td>
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<tr>
<td>NAF Shoulder Bag</td>
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If you or a family member or friend 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 at lori@ataxia.org or (763) 553-0020.

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

— 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
Bart Beck – Tucson, AZ
(520) 885-8326
E-mail: bbeck15@cox.net

www.ataxia.org/chapters/Tucson/default.aspx

— ARKANSAS —

AMBASSADORS
Judy and David King – Hot Springs Village, AR
E-mail: drkingpd@suddenlink.net

www.ataxia.org/chapters/JudyKing/default.aspx

— CALIFORNIA —

LOS ANGELES AREA SUPPORT GROUP LEADERS
Lora Morn – Santa Monica, CA
E-mail: loramorn@gmail.com

Harvey Kahn – Whittier, CA
(562) 789-5776
E-mail: jkhk@aol.com

www.ataxia.org/chapters/LosAngeles/default.aspx

N. CALIFORNIA AREA SUPPORT GROUP LEADER
Jen Buehler – Fremont, CA
(510) 468-6474
E-mail: jenbuehler@aol.com

Sacramento Area Location Representatives
Darrell Owens – Davis, CA
E-mail: droopydog36@hotmail.com

Donna Hoag – Lincoln, CA

www.ataxia.org/chapters/JudyKing/default.aspx
E-mail: donna.hoag@icloud.com
Teresa Bredberg – Sacramento, CA
E-mail: tbredberg@sbglobal.net
S.G. Website: norcalataxia.org
www.ataxia.org/chapters/NorthernCalifornia/default.aspx

ORANGE COUNTY AREA SUPPORT GROUP LEADER
Cindy DeMint – Yorba Linda, CA
(714) 970-1191
E-mail: cindyocataxia@gmail.com
www.ataxia.org/chapters/orangeCounty/default.aspx

PALO ALTO SUPPORT GROUP LEADERS
Victoria Tanoury, RN, CNRN – Stanford, CA
(650) 736-1399
E-mail: vtanoury@stanfordhealthcare.org
Sarah Kahn – Stanford, CA
E-mail: skahn@stanfordhealth.org
www.ataxia.org/chapters/PaloAlto/default.aspx

AMBASSADORS
Barbara Bynum – Merced, CA
(209) 383-1275
E-mail: bjb@vtlnet.com
www.ataxia.org/chapters/BarbaraBynum/default.aspx
Deborah Levi – Morro Bay, CA
(805) 407-0437
E-mail: Debblelevi213@yahoo.com
www.ataxia.org/chapters/CentralCA/default.aspx
Deborah Omictin – Hayward, CA
(510) 783-3190
E-mail: rsisbig@aol.com
www.ataxia.org/chapters/DeborahO/default.aspx
Martha Elliott – Camarillo, CA
(805) 987-2490
E-mail: DOCElliot268@gmail.com
www.ataxia.org/chapters/Camarillo/default.aspx

— COLORADO —
GREATER DENVER AREA SUPPORT GROUP LEADER
Charlotte DePew – Aurora, CO
(720) 379-6887
E-mail: cldpew77@comcast.net
Facebook Group: www.facebook.com/groups/denverataxia2011/
www.ataxia.org/chapters/Denver/default.aspx

— CONNECTICUT —
TRI-STATE SUPPORT GROUP LEADERS
Kathy Gingerelli – Parsippany, NY
(973) 334-2242
E-mail: kgingerelli@msn.com
Denise Mitchell – Bronxville, NY
E-mail: markmghan@aol.com
www.ataxia.org/chapters/Tri-State/default.aspx

— DELAWARE —
DELAWARE SUPPORT GROUP LEADER
Joe & Cathy DeCrescenzo – Bear, DE
(302) 369-9287
E-mail: jdec26@verizon.net
E-mail: cdesccres@verizon.net
www.ataxia.org/chapters/DeCrescenzo/default.aspx

— FLORIDA —
TAMPA BAY SUPPORT GROUP LEADER
Nygel Lenz – Clearwater, FL
(727) 451-9165
E-mail: nygellenz@gmail.com
www.ataxia.org/chapters/TampaBay/default.aspx
TREASURE COAST SUPPORT GROUP LEADER
Lisa Cole – Port St. Lucie, FL
(772) 370-3041
E-mail: lcoie2234@gmail.com
www.ataxia.org/chapters/PortStLucie/default.aspx

AMBASSADORS
Carol Neff – Edgewater, FL
(386) 424-4192
E-mail: cmneff@yahoo.com
www.ataxia.org/chapters/Edgewater/default.aspx
Meghan McBrearty – Tallahassee, FL
(850) 524-9060
E-mail: megan10@hotmail.com
www.ataxia.org/chapters/McBrearty/default.aspx

— GEORGIA —
GREATER ATLANTA SUPPORT GROUP LEADERS
Dave Zilles – Atlanta, GA
(678) 596-6751
E-mail: djzilles@earthlink.net
Greg Rooks – Atlanta, GA
(404) 822-7451
E-mail: rooksgj@yahoo.com
Lealan Sims – Hilton Head Island, SC
(678) 234-6600
E-mail: lealan@mac.com
S.G. Email: atlantaataxia@yahoo.com
Facebook Group: www.facebook.com/groups/317380459539/
www.ataxia.org/chapters/Atlanta/default.aspx

AMBASSADOR
Kristie Adams – Savannah, GA
E-mail: opal1011@comcast.net
www.ataxia.org/chapters/KristieAdams/default.aspx

— ILLINOIS —
CHI-TOWN FRIENDSHIP GROUP LEADER
Jonas Cepkauskas – Matteson, IL
(708) 381-5555
E-mail: jnaps@chitownataxia.org
www.ataxia.org/chapters/Chicago/default.aspx
CHICAGO METRO FRIENDSHIP GROUP LEADER
Christopher (Topher) Marsh – Chicago, IL
(312) 662-1127
E-mail: cmash34@ameritech.net
www.ataxia.org/chapters/ChicagoMetro/default.aspx

AMBASSADOR
Elaine Darte – Coffeen, IL
(618) 397-3259
E-mail: elainedarte@yahoo.com
www.ataxia.org/chapters/SouthernIllinois/default.aspx

Continued on page 46
NAF Directory
Continued from page 45

— INDIANA —

AMBASSADOR
Cheryl (Cheri) Bearman – Hoagland, IN
(260) 452-6231
E-mail: cheribearman@gmail.com
www.ataxia.org/chapters/Indiana/default.aspx

— IOWA —

AMBASSADOR
Emily Medina – West Des Moines, IA
(515) 727-8713
E-mail: emily061578@yahoo.com
Facebook Group:
www.facebook.com/groups/107944351294/
www.ataxia.org/chapters/EmilyMedina/default.aspx

— KANSAS —

AMBASSADOR
Jalean Retzlaff – Park City, KS
(316) 303-2351
E-mail: jlrrolls@yahoo.com
www.ataxia.org/chapters/Retzlaff/default.aspx

— KENTUCKY —

AMBASSADOR
Janice Johnson – Brownsville, KY
(270) 597-3854
www.ataxia.org/chapters/JaniceJohnson/default.aspx
Jennifer Mueller – Lexington, KY
(859) 554-5939
E-mail: jenmu@yahoo.com
www.ataxia.org/chapters/Jen妮/default.aspx

— MAINE —

MAINE SUPPORT GROUP LEADER
Alan and Paula Nadeau – Belgrade, ME
E-mail: psn92871@roadrunner.com
www.ataxia.org/chapters/Maine/default.aspx

— MARYLAND —

CHESAPEAKE CHAPTER PRESIDENT
Carolyn Davis – Vienna, VA
(703) 759-2008
E-mail: ccanpres@gmail.com
www.ataxia.org/chapters/Cheasapeake/default.aspx

MID-ATLANTIC SOCIAL SUPPORT GROUP LEADER
Carrie Berlett, Ataxia Clinic Coordinator
Timonium, MD
(410) 616-2816
E-mail: cmokar1@jhmi.edu
www.ataxia.org/chapters/JHASG/default.aspx

AMBASSADOR
Karen DeVito – Frederick, MD
(301) 682-5386
E-mail: karen.devito@yahoo.com
www.ataxia.org/chapters/KarenRosenberger/default.aspx

— MASSACHUSETTS —

BOSTON AREA SUPPORT GROUP LEADERS
Denise Mindle – South Dartmouth, MA
(508) 369-7925
John Mauro – Auburn, MA
(508) 736-6084
E-mail: john@ataxia.org
S.G. E-mail: ngataxia@outlook.com
www.ataxia.org/chapters/Boston/default.aspx

CENTRAL MA SUPPORT GROUP LEADER
John and Dana Mauro – Auburn, MA
(508) 736-6084
E-mail: john@ataxia.org
E-mail: danamauro63@msn.com
Facebook Group:
www.facebook.com/ataxiadidyouknow?ref=hl
www.ataxia.org/chapters/CentralMA/default.aspx

— MICHIGAN —

DETROIT AREA SUPPORT GROUP LEADER
Tanya Tunstull-Marshall – Detroit, MI
(313) 736-2827
E-mail: tinyt48221@gmail.com
www.ataxia.org/chapters/Detroit/default.aspx

WESTERN MICHIGAN SUPPORT GROUP LEADER
Lynn K. Ball – Grand Rapids, MI
(616) 735-2303
E-mail: lynnkball@aol.com
www.ataxia.org/chapters/LynnBall/default.aspx

— MINNESOTA —

CENTRAL MN SUPPORT GROUP LEADER
Marsha Binnebose – St. Cloud, MN
(320) 248-9851
E-mail: mbinnebose@hotmail.com
www.ataxia.org/chapters/StCloud/default.aspx

TWIN CITIES SOCIAL GROUP
Lenore Healey Schultz – Minneapolis, MN
(612) 724-3784
E-mail: schultz.lenore@yahoo.com
Maryann Sweeney – Minneapolis, MN
(612) 924-4947
E-mail: maryann.sweeney@gmail.com
www.ataxia.org/chapters/TwinCities/default.aspx

AMBASSADORS
Julie Schuur – Luverne, MN
(507) 283-2555
E-mail: jschuur@vastbb.net
www.ataxia.org/chapters/JulieSchuur/default.aspx
Lori Goetzman – Rochester, MN
(507) 282-7127
E-mail: logoetz@gmail.com
www.ataxia.org/chapters/LoriGoetzman/default.aspx

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MISSISSIPPI CHAPTER PRESIDENT
Camille Daglio – Hattiesburg, MS
E-mail: daglio1@bellsouth.net
www.ataxia.org/chapters/Mississippi/default.aspx

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Jim Clark – Oak Grove, MO
(816) 898-6872
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(816) 257-2428
www.ataxia.org/chapters/KansasCity/default.aspx

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Sarah “Janeen” Rheinecker – St. Louis, MO
(417) 379-3799
Email: stlataxia@gmail.com
www.ataxia.org/chapters/StLouis/default.aspx

AMBASSADOR
Roger Cooley – Columbia, MO
(573) 474-7232 before noon
E-mail: rogercooley@mediacombb.net
www.ataxia.org/chapters/RogerCooley/default.aspx

— NEBRASKA —
NEBRASKA ATAXIA SUPPORT GROUP LEADER
Linda Snider – Omaha, NE
(402) 212-3060
E-mail: lindasnider@cox.net
www.ataxia.org/chapters/Omaha/default.aspx

— NEW HAMPSHIRE —
NEW HAMPSHIRE SUPPORT GROUP LEADER
Jill Porter – Manchester, NH
(603) 626-0129
E-mail: jillporter@comcast.net
www.ataxia.org/chapters/Bedford/default.aspx

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NEW JERSEY SUPPORT GROUP LEADER
Priya Mansukhani – Bridgewater, NJ
(908) 685-8805
E-mail: priyamans@gmail.com
www.ataxia.org/chapters/NewJersey/default.aspx

TRI-STATE SUPPORT GROUP LEADERS
Kathy Gingerelli – Parsippany, NJ
(973) 334-2242
E-mail: kgingerelli@msn.com
Denise Mitchell – Bronxville, NY
E-mail: markmegan2@gmail.com
www.ataxia.org/chapters/Tri-State/default.aspx

— NEW YORK —
TRI-STATE SUPPORT GROUP LEADERS
Kathy Gingerelli – Parsippany, NJ
(973) 334-2242
E-mail: kgingerelli@msn.com
Denise Mitchell – Bronxville, NY
E-mail: markmegan2@gmail.com
www.ataxia.org/chapters/Tri-State/default.aspx

— NORTH CAROLINA —
TARHEEL SUPPORT GROUP LEADERS
Ron and Donna Smith – Garner, NC
(919) 779-0414
E-mail: rsmith@sacherokee.com
dsmith@sa-pr.com

AMBASSADOR
Jodie Kawa – Brevard, NC
(828) 384-8414
E-mail: jodiekawa@citcom.net
www.ataxia.org/chapters/Tarheel/default.aspx

— OHIO —
AMBASSADOR
Julia Soriano – Cincinnati, OH
(513) 899-1195
E-mail: julia@epivision.com
www.ataxia.org/chapters/Cincinnati/default.aspx

CLEVELAND AREA SUPPORT GROUP LEADER
Carmen Pieragastini – Willough, OH
(216) 272-5588
E-mail: willowpier@roadrunner.com
www.ataxia.org/chapters/Cleveland/default.aspx

— OREGON —
WILLAMETTE VALLEY SUPPORT GROUP LEADER
Jason Wolfer – Gervais, OR
(503) 502-2633
E-mail: wolfer.jason@gmail.com
Facebook Group: www.facebook.com/groups/388993597939205/
www.ataxia.org/chapters/Willamette/default.aspx

— PENNSYLVANIA —
CENTRAL PA SUPPORT GROUP LEADER
Michael Cammer – Downingtown, PA
(610) 873-1852
E-mail: michael.cammer62@hotmail.com
Facebook Group: www.facebook.com/groups/1475283086068548/
www.ataxia.org/chapters/CentralPA/default.aspx

WESTERN PA SUPPORT GROUP LEADER
Ed Schwartz – Venetia, PA
(724) 941-2210
E-mail: eds@ataxia.org
Donna Eiben – South Park, PA
(412) 655-4091
dawn.eiben@verizon.net
Facebook Group: www.facebook.com/wpaataxia
www.nafwesternpasupportchapter.weebly.com/
www.ataxia.org/chapters/SouthPark/default.aspx

— RHODE ISLAND —
RHODE ISLAND SUPPORT GROUP LEADER
Anabela Azevedo – Bristol, RI
(401) 297-8627
E-mail: azevedo7@anabela@gmail.com
www.ataxia.org/chapters/Rhodelsland/default.aspx

— TENNESSEE —
MIDDLE TN AREA SUPPORT GROUP LEADER
Alex Cohn – Nashville, TN
(256) 504-0240
E-mail: alex.j.cohn@us.pwc.com
www.ataxia.org/chapters/TN/default.aspx

— TEXAS —
GREATER HOUSTON AREA SUPPORT GROUP LEADER
Ashley Grayson – Houston, TX
(832) 530-0866
E-mail: ashleygrayson90@gmail.com
Facebook Group: www.facebook.com/groups/atxia.houston/
www.ataxia.org/chapters/Houston/default.aspx

Continued on page 48
NORTHERN TEXAS AREA SUPPORT GROUP LEADER
David Henry, Jr. – Trophy Club, TX
(817) 739-2886 (contact by e-mail preferred)
E-mail: cheve11e@sbcglobal.net
Facebook Group:
www.facebook.com/Ataxiasupport
www.ataxia.org/chapters/NorthTexas/default.aspx

AMBASSADORS
Dana LeBlanc – Orange, TX
(409) 883-5570
E-mail: tilessal@yahoo.com
www.ataxia.org/chapters/GoldenTriangle/default.aspx
Debra Whitcomb – El Paso, TX
(915) 329-0721
E-mail: debrawhitcomb@hotmail.com
www.ataxia.org/chapters/Whitcomb/default.aspx

— UTAH —

UTAH SUPPORT GROUP LEADERS
Grant Beutler – Salt Lake City, UT
E-mail: grant.beutler@gmail.com
Jenny Durrant – Pleasant View, UT
E-mail: jenny@utahataxia.org
Lisa Ord, PhD, LCSW – Salt Lake City, UT
(801) 587-3020
E-mail: lisa.ord@hsc.utah.edu
Facebook Page: www.facebook.com/utahataxia
S.G. Website: www.ataxia.org/chapters/Utah/default.aspx

— VIRGINIA —

CHESAPEAKE CHAPTER PRESIDENT
Carolyn Davis – Vienna, VA
(703) 759-2008
E-mail: ccnafpres@gmail.com
www.ataxia.org/chapters/Chesapeake/default.aspx

— WASHINGTON —

WESTERN WASHINGTON SUPPORT GROUP LEADER
Sherry McLaughlin
(360) 344-2445
E-mail: ccherilynmc@yahoo.com
www.ataxia.org/chapters/WWA/default.aspx

AMBASSADOR
Linda Jacoy – Spokane, WA
(509) 482-8501
E-mail: linda4727@hotmail.com
www.ataxia.org/chapters/Spokane/default.aspx

— WISCONSIN —

WISCONSIN SUPPORT GROUP LEADER
Kory Macy – Madison, WI
(608) 237-6090
E-mail: kstab77@yahoo.com
www.ataxia.org/chapters/Wisconsin/default.aspx

International
Support Groups & Ambassadors

— CANADA —

OTTAWA SUPPORT GROUP LEADER
Prentis Clairmont – Ottawa, Ontario
(613) 864-8545
E-mail: prentis.clairmont@gmail.com
Facebook Group:
www.facebook.com/groups/1468963499991380/
www.ataxia.org/chapters/Ottawa/default.aspx

— INDIA —

INDIA SUPPORT GROUP LEADER
“Seek a Miracle Ataxia Group” (SAMAG)
Chandu Prasad George
Hyderabad, Secunderabad, India
Mobile: 0091-9989899919, 0091-9885199918
E-mail: sam.ataxiaindia@yahoo.com
S.G. E-mail: india.ataxiagroup@gmail.com
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— PAKISTAN —

AMBASSADOR
Sajjad Haider – Karachi, Pakistan
0092-(300) 828-1784
E-mail: sajjadhaiderb@hotmail.com

PATIENTS with
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at the University of Minnesota,
Minneapolis

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Contact: Diane Hutter
(612) 625-2350
hutte019@umn.edu
In June 2005, I packed my briefcase, left the room in order and locked the elementary classroom door for the final time. I took the memories of a 20-year teaching career with me. For the past several years, I knew my ataxia had progressed. During this last school year, I was absent too many days due to constant illness. Weakness and fear of falling occupied my thoughts and took over my body. It became difficult to walk across the schoolyard or make my way to the office. I could no longer take advantage of my social time. Even the lunchroom was off limits. I retired at age 55 with a disability. Regardless of how hard I tried to hide my physical condition, I could no longer deny the truth.

Normally, the last day of any school year is a time for celebration but that day was different. It marked the end of an era and the beginning of new unchartered territory. The thought of not having to get up early, or do nightly paperwork, or commute a long distance to work thrilled me. However, my admission of being disabled terrified me. Was this an ending or a beginning?

The time had come to retire and re-think. During the past years, many students came and went. During my career as a teacher, I always taught my students to understand and locate the beginning, middle, and end of a story. I explained that there will be new developments along the way. There will always be turns along the paths. Now the time arrived to learn this firsthand as a student. My lessons had just begun. In all that time of instruction, I now believe the most important student was me. The teacher happened to be my body, and my body now instructed me to slow down.

Until this time, I had lived a pre-planned existence and measured my days and thoughts by how efficiently things went. The clock and the calendar were central to my decisions. I calculated every lesson and activity to the minute. I took pride in my organizational abilities. When that final bell rang, I entered a new “time zone” based on a much deeper clock, an internal clock, a clock that can’t be measured, seen or heard.

I began to read and contemplate the stages of loss and possible alternative methods of healing my inner turmoil. Was I alone? Is there anyone else who feels this way? Since there is no definite cure for cerebellar ataxia, I felt it necessary to investigate other ways to find relief from the trials of body breakdown. The term victim kept reappearing. I wanted to learn more...

Gradually I realized how “victim consciousness” limited my thoughts, experiences and kept me away from people. The idea that I had no control over the events that happened to me became false. I knew that type of thinking could be changed. Living with cerebellar ataxia is both difficult and demanding. It requires an enormous amount of patience and tremendous self-love and self-care. There is no time for “victim consciousness.” However, there needs to be a distinction between physical and emotional healing and I needed to find my inner warrior. I needed time to listen, time to be receptive, time to re-define and re-adjust my thoughts, and time to think beyond myself. I hope this time and awareness will come to you, and come before the final bell rings.

To contact Deborah Levi about this story or to join the new support group she is starting in Central CA, e-mail debbielevi213@yahoo.com.
Calendar of Events

The most current event information is available on the NAF website, www.ataxia.org.

Why Attend an Ataxia Support/Social Group?

Many of you may ask, “Why should I attend a support group meeting?” Support groups can remind us that we are not alone and that while each individual may experience Ataxia in a different way, together we have many things in common. A benefit of attending a support group is simply to have a chance to talk with others and learn how different people deal with the same disease.

Attending a support group meeting may give you a glimpse into the many different stages and types of the disease. This can help by using some of the strategies that have been beneficial to others in order to avoid and/or plan for some of the same challenges that others have faced in the progression of their Ataxia. Hopefully attending a support group meeting will leave you with a sense of hope and inspiration, knowing that if others can cope, so can you.

Come. Learn. Share. But most of all, know that you are NOT alone.

SUPPORT GROUP MEETINGS

— Tuesday, October 4, 2016 —

Cleveland Ataxia Support Group Meeting
Time: 6:30 p.m.
Location: Garfield Heights Public Library, 5400 Transportation Blvd., Garfield Heights, OH
Details: For additional information contact Carmen Piaragastini at (216) 272-5588 or willowpier@roadrunner.com.

Western PA Ataxia Support Group Meeting
Time: 7 p.m.
Location: Bethel Park Community Center, Park Ave, Bethel Park, PA 15102
Details: For additional information contact Ed Schwartz at (724) 941-2210 or eds@ataxia.org.

— Saturday, October 8, 2016 —

Central Minnesota Ataxia Support Group Meeting
Time: 9:45 – 11:45 a.m.
Location: Harvest Bank Branch, 24952 County Road 7, St. Augusta, MN 56301
Details: For additional information contact Marsha Binnebose at (320) 248-9851 or mbinnebose@hotmail.com.

Kansas City Ataxia Support Group Meeting
Time: 2 – 4 p.m.
Location: Northeast Library, 6000 Wilson Rd., Kansas City, MO
Details: For more information contact Lois Goodman at (816) 257-2428 or Jim Clark at (816) 468-7260

North Texas Ataxia Support Group Meeting
Time: 10 a.m. – noon
Location: Ben Washington Baptist Church, 615 Davis St., Irving, TX 75061
Details: The meeting room is in a separate building from the church. For more information contact David Henry at cheve11e@sbcglobal.net.

Northern California Ataxia Support Group Meeting
Time: 11:30 a.m. – 2 p.m.
Location: Our Savior’s Lutheran Church, 1035 Carol Ln., Lafayette, CA
Details: For additional information or to RSVP contact Jen Buehler at (510) 468-6474 or jenbuehler@aol.com.

Rhode Island Ataxia Support Group Meeting
Time: 11 a.m. – 2 p.m.
Location: Bristol Parks Recreation, 101 Asylum Rd., Bristol, RI 02809
Details: For additional information contact Anabela Azevedo at (401) 297-8627 or azevedo70anabela@gmail.com.

— Wednesday, October 12, 2016 —

Willamette Valley Ataxia Support Group Meeting – Albany
Time: 11:30 a.m. – 1 p.m. on the second Wednesday of every month
Location: 400 NW Hickory, Albany, OR 97321
Details: For more information contact Jason Wolfer at (503) 502-2633 or wolfer.jason@gmail.com.

— Thursday, October 13, 2016 —

St. Louis Area Ataxia Support Group Meeting
Time: 5:30 – 7:30 p.m. Meetings will be held at on the second Thursday of every month.
Location: Washington University Medical Center, 4444 Forest Park Ave., Rm. 509, St. Louis, MO 63108
Details: For additional information contact Janeen Rheinecker at (417) 379-3799 or slataxia@gmail.com.

— Saturday, October 15, 2016 —

Denver Area Ataxia Support Group Meeting
Time: 1 – 4 p.m.
Location: Swedish Medical Center, Second Floor Conference Center (meeting room TBD), 501 E. Hampden Ave., Englewood, CO 80113
Details: For more information contact Charlotte DePew at (720) 379-6887 or cdepew77@comcast.net.

NCASG-Sacramento Area Location Ataxia Support Group Meeting
Time: 1 – 4 p.m.
Location: Sutter Roseville Medical Center, 1 Medical Plaza Dr., Roseville, CA 95661, Conference Room B
Details: For additional information contact Teresa Bredberg (916) 421-2173 or tredberg@sbcglobal.net.

Orange County Ataxia Support Group Meeting
Time: 2 – 4 p.m.
Location: Orange Coast Memorial Medical Center Hospital, Breast Cancer Center Conference Room A, 9900 Talbert Ave., Fountain Valley, CA 92708
Details: For more information contact Cindy DeMint at cindyocataxia@gmail.com.

Twin Cities Ataxia Social Group Meeting
Time: 10 a.m. on the third Saturday of every month (approximately two hours)
Location: Langton Place in Roseville at 1910 W. County Rd. D, Roseville, MN 55122
Details: For additional information contact Lenore Healey Schultz at (612) 724-3784 or schultz.lenore@yahoo.com.

— Saturday, October 22, 2016 —

New Hampshire Ataxia Support Group Meeting
Time: 10 a.m. – noon
Location: Villa Crest Nursing and Retirement Home, 1276 Hanover St., Manchester, NH
Details: For more information contact Jill Porter at (603) 626-0129 or jilleporter@comcast.net.

Tarheel Ataxia Support Group Meeting
Time: 1 – 3 p.m.
Location: Wake Forest Baptist Health Davie Medical Center, 313 NC Hwy. 801, Bermuda Run, NC
Details: Julie Applewhite, who is trained by Motion Therapeutics, will be our guest speaker. For more information contact Ron Smith at (919) 779-0401 or rsmith@sacherokee.com.

— Tuesday, November 1, 2016 —

Western PA Ataxia Support Group Meeting
Time: 7 p.m.
Location: Bethel Park Community Center, Park Ave, Bethel Park, PA 15102
Details: For additional information contact Ed Schwartz at (724) 941-2210 or eds@ataxia.org.

— Saturday, November 5, 2016 —

Greater Atlanta Ataxia Support Group Meeting
Time: 1 p.m.
Location: Emory Rehab Hospital, Rm. 101, 1441 Clifton Rd., Atlanta, GA.
Details: For additional information contact the group at (404) 822-7451 or atlantaataxia@gmail.com.

— Tuesday, November 8, 2016 —

Utah Ataxia Support Group Meeting
Time: 7 p.m.
Location: John A. Moran Eye Center, 65 Mario Capecchi Dr., Salt Lake City, UT 84132
Details: For more information contact Dr. Lisa Ord, PhD, LCW at (801) 585-6635 or lisa.ord@hsc.utah.edu.

— Wednesday, November 9, 2016 —

Willamette Valley Ataxia Support Group Meeting – Albany
Time: 11:30 a.m. – 1 p.m. on the second Wednesday of every month
Location: 400 NW Hickory, Albany, OR 97321
Details: For more information contact Jason Wolfer at (503) 502-2633 or wolfer.jason@gmail.com.

— Thursday, November 10, 2016 —

St. Louis Area Ataxia Support Group Meeting
Time: 5:30 – 7:30 p.m. Meetings will be held at on the second Thursday of every month.
Location: Washington University Medical Center, 4444 Forest Park Ave., Rm. 509, St. Louis, MO 63108
Details: For additional information contact Janeen Rheinecker at (417) 379-3799 or slataxia@gmail.com.
Calendar of Events
Continued from page 51

Rheinecker at (417) 379-3799 or stlataxia@gmail.com.

Tri-State Ataxia Support Group Meeting
Time: 6:30 – 8:30 p.m.
Location: Beth Israel Medical Center, Phillips Ambulatory Care Center (PACC), Second Floor Conference Room, 10 Union Square East, New York, NY
Details: For more information contact Kathy Gingerelli at kgingerelli@msn.com or Denise Mitchell at markmegan2@gmail.com.

— Saturday, November 12, 2016 —

Arizona Ataxia Support Group Meeting
Time: 1 p.m.
Location: Ability 360, 5025 E Washington St., Phoenix, AZ 85034
Details: For more information contact Mary Fuchs at (480) 212-6425 or mary11115@msn.com.

Central Minnesota Ataxia Support Group Meeting
Time: 9:45 – 11:45 a.m.
Location: Harvest Bank Branch, 24952 County Road 7, St. Augusta, MN 56301
Details: For additional information contact Marsha Binnebose at (320) 248-9851 or mbinnebose@hotmail.com.

North Texas Ataxia Support Group Meeting
Time: 10 a.m. – noon
Location: Ben Washington Baptist Church, 615 Davis St., Irving, TX 75061
Details: The meeting room is in a separate building from the church. For more information contact David Henry at cheve11e@sbcglobal.net.

— Saturday, November 19, 2016 —

NCASG-Sacramento Area Location Ataxia Support Group Meeting
Time: 1 – 4 p.m.
Location: Sutter Roseville Medical Center, 1 Medical Plaza Dr., Roseville, CA 95661, Conference Rm. B
Details: For additional information contact Teresa Bredberg (916) 421-2173 or tredberg@sbcglobal.net.

Tampa Bay Ataxia Support Group Meeting
Time: 12:30 – 3 p.m.
Location: The University of South Florida Ataxia Research Center, Morsani Center, 13330 USF Laurel Dr., Ste. 1013, Tampa, FL 33612
Details: For more information contact Nygel Lenz at (727) 451-9165 or nygellenz@gmail.com.

Twin Cities Ataxia Social Group Meeting
Time: 10 a.m. on the third Saturday of every month (approximately two hours)
Location: Langton Place in Roseville at 1910 W. County Rd. D, Roseville, MN 55112
Details: For additional information contact Lenore Healey Schultz at (612) 724-3784 or schultz.lenore@yahoo.com.

— Sunday, November 20, 2016 —

Chitown Ataxia Friendship Group Meeting & Potluck
Time: 1 p.m.
Location: Advocate Good Samaritan Hospital, 3815 Highland Ave., Downers Grove, IL 60515
Details: For more information contact Jonas Cepkauskas at (708) 381-5555 or jonas@chitownataxia.org.

— Saturday, December 3, 2016 —

Mid-Atlantic Ataxia Social Group Holiday Party
Time: 11 a.m. – 2 p.m.
Location: TBD
Details: For more information contact Nicola Menucci at (410) 616-2816 or nmennic1@jhmi.edu.

New Hampshire Ataxia Support Group Meeting
Time: 10 a.m. - 12 p.m.
Location: Villa Crest Nursing and Retirement Home, 1276 Hanover St., Manchester, NH
Details: For more information contact Jill Porter at (603) 626-0129 or jilleporter@comcast.net.

Orange County Ataxia Support Group Holiday Party
Location: TBD
Details: For more information contact Cindy DeMint at cindyocataxia@gmail.com.

Treasure Coast Ataxia Support Group Meeting
Time: 1 – 3:30 p.m.
Location: Port St. Lucie Community Center, 2195 SE Airoso Blvd., Port St. Lucie, FL 34983
Details: Guest speaker: Jan Field-Byrne. For more information, RSVP or to be added to the group’s mailing list contact Lisa Cole at (772) 370-3041 or lcole2234@gmail.com.

— Tuesday, December 6, 2016 —

Western PA Ataxia Support Group Meeting
Time: 7 p.m.
Location: Bethel Park Community Center, Park Ave, Bethel Park, PA 15102
Details: For additional information contact Ed Schwartz at (724) 941-2210 or eds@ataxia.org.
— Thursday, December 8, 2016 —
St. Louis Area Ataxia Support Group Meeting
Time: 5:30 – 7:30 p.m. Meetings will be held at on the second Thursday of every month.
Location: Washington University Medical Center, 4444 Forest Park Ave., Rm. 509, St. Louis, MO 63108
Details: For additional information contact Janeen Rheinecker at (417) 379-3799 or stlataxia@gmail.com.

— Saturday, December 10, 2016 —
Atlanta Ataxia Social Group Christmas Party
Time: 1 p.m.
Location: TBD
Details: For more information contact the group at (404) 822-7451 or atlantaataxia@gmail.com.

Central Minnesota Ataxia Support Group Meeting
Time: 9:45 – 11:45 a.m.
Location: Harvest Bank Branch, 24952 County Road 7, St. Augusta, MN 56301
Details: For additional information contact Marsha Binnebose at (320) 248-9851 or mbinnebose@hotmail.com.

Kansas City Ataxia Support Group Meeting
Time: 2 – 4 p.m.
Location: Northeast Library, 6000 Wilson Rd., Kansas City, MO
Details: For more information contact Lois Goodman at (816) 257-2428 or Jim Clark at (816) 468-7260

North Texas Ataxia Support Group Meeting
Time: 10 a.m. – noon
Location: Ben Washington Baptist Church, 615 Davis St., Irving, TX 75061
Details: The meeting room is in a separate building from the church. For more information contact David Henry at cheve11e@sbcglobal.net.

— Wednesday, December 14, 2016 —
Willamette Valley Ataxia Support Group Meeting – Albany
Time: 11:30 a.m. – 1 p.m. on the second Wednesday of every month
Location: 400 NW Hickory, Albany, OR 97321
Details: For more information contact Jason Wolfer at (503) 502-2633 or wolfer.jason@gmail.com.

— Saturday, December 17, 2016 —
NCASG-Sacramento Area Location Ataxia Support Group Meeting
Time: 1 – 4 p.m.
Location: Sutter Roseville Medical Center, 1 Medical Plaza Dr., Roseville, CA 95661, Conference Rm. B
Details: For additional information contact Teresa Bredberg (916) 421-2173 or tredberg@sbcglobal.net.

Twin Cities Ataxia Social Group Meeting
Time: 10 a.m. on the third Saturday of every month (approximately two hours)
Location: Langton Place in Roseville at 1910 W. County Rd. D, Roseville, MN 55112
Details: For additional information contact Lenore Healey Schultz at (612) 724-3784 or schultz.lenore@yahoo.com.

Continued on page 54

Everyone who has any form of Ataxia or who is at risk for Ataxia is encouraged to enroll in the CoRDS/NAF Ataxia patient registry.

To register in the CoRDS Ataxia patient registry, go to www.ataxia.org and click on “Ataxia Patient Registry.” If you prefer to enroll by postal mail, please contact CoRDS personnel.

For more information on CoRDS and/or enrollment, visit www.sanfordresearch.org/cords or call (605) 312-6413. Thank you for participating in this important research tool.

This link takes you to a page that has a video on why and how to enroll in the CoRDS Registry: www.sanfordresearch.org/cords/aboutcords.
Calendar of Events
Continued from page 53

INFORMATIONAL, AWARENESS, AND IAAD EVENTS AND FUNDRAISERS

— Saturday, October 1, 2016 —
Utah Walk n’ Roll
IAAD Event and Fundraiser
Time: 11 a.m. – 2 p.m.
Location: Layton Commons Park, Layton, UT
Details: All proceeds benefit the National Ataxia Foundation. For more information contact Lisa Ord at (801) 585-6635 or lisa.ord@utah.edu. www.ataxia.org/walk/utah

— Tuesday, October 18, 2016 —
Boscov’s Friends Helping Friends
Details: Discount shopping coupons for $5 each that can be used at Boscov’s stores on October 18th. All proceeds benefit the National Ataxia Foundation. For shopping coupons or more information contact Mike Cammer at (610) 996-5814 or michael.cammer62@hotmail.com.

— Friday-Sunday, November 18-20, 2016 —
San Jose Abilities Expo
Time: Friday and Saturday 11 a.m. – 5 p.m., Sunday 11 a.m. – 4 p.m.
Location: San Jose McEnery Convention Center, 150 W. San Carlos St., San Jose, CA 95110
Details: Admission is free.
www.abilitiesexpo.com/bayarea/

— Friday, December 2-4, 2016 —
DC Metro Abilities Expo
Time: Friday and Saturday 11 a.m. – 5 p.m., Sunday 11 a.m. – 4 p.m.
Location: Dulles Expo Center, 4320 Chantilly Shopping Center, Chantilly, VA 20151
Details: Admission is free.
www.abilitiesexpo.com/dcmetro/

— Friday, January 20-22, 2017 —
Toronto Abilities Expo
Time: Friday and Saturday 11 a.m. – 5 p.m., Sunday 11 a.m. – 4 p.m.
Location: International Centre, 6900 Airport Rd., Mississauga, ON L4V 1E8, Canada
Details: Admission is free.
www.abilitiesexpo.com/toronto/

In Memory:
Mary Jane Damiano

The National Ataxia Foundation (NAF) is saddened by the recent passing of Mary Jane Damiano of North Syracuse, NY, on Aug. 31. She was 59.

Mary Jane was the Central New York Ataxia Support Group Leader. Despite her increasingly debilitating condition, she coordinated many public relations and fund raising efforts to support the NAF throughout most of her adult life. Mary Jane felt she had a mission, and the “Proclamation Wall” in her home was a testament to getting local politicians to recognize her cause of fighting Ataxia. The wall has dozens of proclamations on display that had been presented annually for years. She was truly an ambassador for the NAF and will be missed greatly by the Foundation and Central NY Ataxia Community. Mary Jane’s determination to stay as independent as possible and be such a strong advocate for awareness of Ataxia is truly inspiring.
Memorials and In Your Honor

The National Ataxia Foundation is grateful to those who have made contributions in memory or in honor of their friends and families whose names are listed below. This list reflects contributions made in June 2016 through July 2016. 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 let us know if the contribution is a memorial or in honor of their friend or family member.

Dave Alessi  Chie Franklin  Austasia Manlupig  Steven Ofenstein  John Surabian
David Ashley  Gregson Gann  Carolyn Marceda  Laura Osplanik  Joe Sweeney
Mike Athey  Kathy Gardner  David Marcy  Santos Perez (son)  Mike Sweeney
Sharon Baggett  Tanya Goldman  Christopher Marsh  Carmen Pieragastini  Tom Sweeney
Linda Bamford  Penny Golminas  Greg Marshall  John Prola  Janet Sweetser
Jean Barnett  Nick Grace  Jeannette Martinho  Ron Randol  Jeff Sweetser
Kitty Barrett  Brenda Graner  Chip Masamitsu  Jim Richards  Barbara Tinari
Jay Bartle  Duane Graner  The Masserant Family  Elizabeth Riley  Josephine Tredent
Cheri Bearman  Lawerence Graner  David Matley  Janet Riley  Tiny Tucker
Mike Beer  Arnie  Gruetzmacher  Michael McCoy  Lynda Rocheford  Jacob Van Buren
Antonio Bermudez  Aymee  Guerrero-Torres  Charlie McLaughlin  Armando Rodriguez
Edward Brand  Peg Harris  Dennis Hanold  Robert McMurtry  Greg Rooks
James Braswell  Bernice Hartley  Susan Hamer  Denise Mindel  The Schnobrich Family
Audrey Breaux  Helen Henry  Delores  Denise Mindle  Edward Schwartz
Delores Broussard-Tise  Dale Hines  Susan Hamer  Ellen Moetsch  Derek Semler
Angela Brown  Bernice Hartley  Heather Hawkins  Eileen Monteleone  Cheryl Serge
Joe Bruno  William Huber  Joseph Stammer  Sherry Moody  Diane Simpson-Dement
Benjamin Cappelli Jr.  Krista Humes  William Huber  Mandy Morse  Janette St. George
Paul Capriolo  Alva Jeffers  Patricia Hogan  Valerie Morse-Sims  Carmen Viveiros
Chip Carroll  James Jeffers  Patricia Hogan  Pacifica Grace  Antone Villa
Stella Cheung  Kerry Johnson  Patricia Hogan  Carmen Viveiros  Jeannette Viveiros
Christine Church  Terry Johnson  Irma Holland  Carol Mullen  Barry Washburn
Jolanta Chyla  Mary Holleman  William Huber  John Noonan  Olive Westhoff-Derrington
Judith Cifolelli  Krista Humes  William Huber  Joseph Novalany  David Westrick
Arlen Crawford  Alva Jeffers  Dale Hines  Patricia Hogan  Marty Willen
Sergio Damasio  James Jeffers  Dale Hines  Carol Mullen  Mike Williams
Mary Danson  Kerry Johnson  Terry Johnson  John Noonan  Virgie Wince
Cindy de Mint  Barry Karas  Wayne Kist  Joseph Stammer  Richard Worster
Joe DeCrescenzo  Wayne Kist  Barry Karas  Josepaph Novalany  Susan Wuchner
Carlos Disilvestro  Ngan Koo  William Huber  Patricia Hogan  Ken Yousten
Connie DiVincentis  Jamie Kosieracki  William Huber  John Noonan  Glenn Zoller
Fred Donnelly  Susan Kresnye  Rocelle Krone  Carol Mullen  GoodSearch
Rick Donnelly  Barb Kucera  Jesse Kuenzi  John Noonan  GoodSearch Is Good for NAF
Olivia Douglass  James Langreder  Diane Laufman  Joseph Novalany  Did you know that donating money to the National Ataxia Foundation can be as easy as changing your Internet search engine?
Charlotte Ennen  James Langreder  Diane Laufman  Joseph Novalany  GoodSearch.com
Louise Estabrook  Diane Laufman  Carl Lauffer  Joseph Novalany  donates 50 percent of its revenue to the charities designated by its users. To get started, simply go to the site and follow the easy steps to make NAF your choice of charity. Then use GoodSearch as you would any other search engine.
Daniel Eustache  Diane Laufman  Carl Lauffer  Joseph Novalany  This simple change will make a difference in the lives of those with ataxia!
Betty Face  Carl Lauffer  Joseph Novalany  Betty Face  GoodSearch Is Good for NAF
Mary Farley  Sammy Lawson Jr.  Jola Li  GoodSearch Is Good for NAF
Skip Fauver  Jola Li  Michael Lundquist  GoodSearch Is Good for NAF
Don Folger  Michael Lundquist  GoodSearch Is Good for NAF
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 ____________________________

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 ____________________