Patients and families dealing with ataxia know well that disorders of the cerebellum produce motor difficulties including wide-based and unsteady, or ataxic, gait; incoordination, or dysmetria, of the arms and legs; articulation impairment, or dysarthria; and eye movement abnormalities that interfere with vision, among other problems. Patients, more than their doctors, have also long suspected that there is more to cerebellar function than motor control issues alone. They have wondered whether problems they experience in the areas of cognition and emotion may be related to the cerebellar damage, and not simply a reaction to chronic disability. This question is at the heart of a new and vibrant area of research, the cognitive neuroscience of the cerebellum, including anatomical and behavioral studies in animal models, functional imaging studies, and clinical investigations in patients. It is now critical that we consider the role of the cerebellum in the realm of behavioral neurology and neuropsychiatry – the study of interactions between the physical brain, and emotions and intellectual abilities.

There are two reasons why it is important to consider these nonmotor aspects of cerebellum function and dysfunction. First, it is important
Generations Staff:
Julie Braun .................................. Financial Director
Sue Hagen................................. Outreach Coordinator
Becky Kowalkowski .............. Patient Services Director
Mike Parent .................................. Executive Director
Lori Shogren .................... Special Projects Coordinator
Donna Gruetzmacher ................... Advisor

Design, Production and Printing............. Leader Printing

Annual Membership Meeting
Ride Ataxia II Reaches Vegas ............... 13
Thank You Athena! .......................... 13
2008 Annual Membership
Meeting (AMM) Review........................ 24
Quotes and Top Three Lists
from the 2008 AMM .......................... 26
2009 AMM Announcement .................. 27
Wanted: Photos ................................ 31
A Special Thank You .......................... 47

Research
Research Subjects Needed .................... 6
NAF Research Grant Summaries .............. 14

Articles (cont.)
Ataxia Center Established at
Johns Hopkins University .................... 11
Caregiver’s Corner ............................ 30
Tissue Donation ............................... 31
News & Notes ................................ 36

Membership Topics
Chapter and Support Group News ............ 32
NAF Merchandise .............................. 37
NAF Chapters, Support Groups
and Ambassadors Listings .................... 38
Calendar of Events ............................ 44
Memorials and In Your Honor ................ 46

Personal Stories and Poems
My Life So Far: 50 Years
of Friedreich’s Ataxia .......................... 22
A Final Act of Selflessness .................... 28
Ataxia Doesn’t Stop Her:
Kayla Prather’s Story ......................... 45

You Could Be Published!
Generations is published quarterly by the National Ataxia Foundation to inform others of the latest research, ataxia chapters and support groups, events and other topics related to all the forms of ataxia. Personal stories from those affected by ataxia are an important part of the publication. 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 susan@ataxia.org to be considered for publication.
to recognize, diagnose and treat conditions that affect the quality of life of patients with ataxic disorders. These problems may arise from damage to the cerebellum itself, from abnormalities in other brain structures that may be affected in conditions such as the spinocerebellar ataxias (SCAs), or from the stress of coping with the disability. Second, if we can understand the wider role of the cerebellum in the nervous system, and the extent to which the cerebellum contributes to neuropsychiatric symptoms even in the absence of ataxia, it may open the way to developing new treatments for many disabling mental health disorders.

Cerebellar highways in the brain

There is an anatomical basis to the cerebellar contribution to nonmotor function. Hard-wired connections link the cerebellum with areas of the cerebral cortex essential for complex thoughts, actions, and emotions. The cerebellum receives information from, and sends signals back to, the prefrontal cortex that is necessary for planning, abstract thinking, multi-tasking, logical reasoning, organizing, and verbal expression; the parietal lobe that is essential for visual-spatial functions, like finding your way and being aware of your body in space; the temporal lobe that is engaged in understanding spoken concepts; and the cingulate gyrus and parahippocampal formation necessary for motivation, mood, emotional processing, and certain forms of memory. In other words, the cerebellum is linked up with the motor cortex that governs movements, and also with cerebral cortical areas that enable the full range of human behaviors. Moreover, different parts of cerebellum are connected to different parts of the cerebral cortex. The anterior lobe of the cerebellum (the part in front and at the top) is mostly connected to motor areas of the cerebral cortex. The posterior lobe of the cerebellum (at the back and underneath) talks to cerebral association areas important for intellect; and the midline of the cerebellum (the vermis), is linked with the brain’s limbic system that is heavily engaged in drive, mood, and feelings.

Experimental studies of the cerebellum

In functional imaging studies using MRI scans, motor actions activate the cerebellum mostly in the anterior lobe (especially lobules IV and V), and in a part near the bottom of the cerebellum called lobule VIII. (There are ten lobules, or subdivisions of the cerebellum, numbered in the Roman numeral style). Thinking tasks, such as language, memory, or planning, activate different parts of the posterior lobe that have expanded massively through evolution, and comprise the major part of the human cerebellum. Functions that have an emotional tone, or that produce anxiety or pain, mostly activate the cerebellar vermis. Thus, there is more to cerebellar activation than movement alone. The activations by motor, cognitive, and limbic studies are located in different places in cerebellum – the anterior lobe, posterior lobe, and vermis respectively.

Behavioral studies in animals provide support for these conclusions derived from anatomical and imaging studies. We produced focal damage in monkeys in parts of the cerebellum thought to be linked with intellect but not with movement. The animals were able to control their limbs normally. But they had trouble with tasks of mental flexibility – the kind of problem that characterizes what is termed, executive dysfunction.

Lessons learned from cerebellar patients

By studying adults and children with damage confined to the cerebellum from stroke, tumor, and antibody-mediated attack, we showed clinical evidence of a relationship between the cerebellum and cognition. We described a cerebellar cognitive affective syndrome (CCAS), including impairments in executive function, visual-spatial analysis, and selected deficits in language skills, as well as changes in personality and behavior.
These impairments resulted from injury to the cerebellar posterior lobe, but not the anterior lobe; and the dysregulation of affect was seen most notably when the cerebellar damage involved the midline (vermis) structures. We have also seen the reverse situation, in adults with cerebellar stroke. We found that when the stroke involves the cerebellar anterior lobe (linked with motor regions of the cerebral cortex), patients have ataxia. But when the stroke avoids the anterior lobe and is confined to the lower parts of the posterior lobe, there is little or no ataxia. So the cerebellar posterior lobe appears not to be engaged in a major way with motor control, but does appear to be important in intellectual and emotional processing.

These findings have immediate clinical relevance. Children who survive resection of cerebellar tumors are challenged in their social relationships and ability to handle the intellectual challenges at school, in addition to any residual motor disability they may experience. Children with an abnormally developed, or underdeveloped, cerebellum have a range of motor deficits. They also have impairments in social-emotional behaviors, and cognitive deficits including language delay, to the point of requiring the use of sign language in the early years. These children require substantial support in the school system and from families.

Depression is frequent in people with ataxia, not only because there is a medical problem that has affected quality of life, but also because cerebellum plays a role in our ability to modulate emotional responses, and influence how we respond to the environment. I have called this dysmetria of thought, similar to the term, dysmetria of movement, used to describe the instability of motor control leading to tremor, over- or under-reaching, and inaccuracy in the coordination of our motor actions.

Almost 40% of patients with multiple system atrophy of the cerebellar type (MSAc) have pathological laughing and crying, also known as pseudobulbar affect. These patients have exaggerated emotional responses, either crying or laughing, to stimuli that would not previously have produced such a reaction, or in a manner inappropriate for the context – laughing at sad situations, or crying when in fact they feel happy. The crying or laughing can be quite protracted, and can be distressing to the patient and family. Rarely, the laughing or giggling can feel like a relief to an MSAc patient, but this is unusual.

I have seen patients with damage to the vermis (the limbic cerebellum) who develop panic disorder. One woman with a stroke became unable to travel in a car because the motion of the vehicle induced vertigo and a sense of overwhelming panic. She recovered, and is back to her hobby of scuba diving. Another patient with atrophy of the vermis has a constellation of incapacitating motion-induced vertigo and panic that has responded somewhat to medications.

Patients with cerebellar disorders frequently notice difficulty with multi-tasking. This problem is similar to that in the monkeys with cerebellar lesions, that have difficulty with mental flexibility, switching from one mental idea to another. Cerebellar patients report they can do one thing at a time, but when they try to multi-task, or keep track of different activities at the same time, they are not successful. The automatic performance of these motor and cognitive skills may be impaired in the cerebellar patient. This may result from loss of the automaticity that relies on cerebellar function, and the ability of cerebellum to smooth out and enhance performance.

Non-cerebellar features in ataxia patients

Long-term memory is not usually lost in patients with purely cerebellar problems. The cognitive impairments we see in the CCAS are different from memory loss that characterizes Alzheimer’s disease. There are some
circumstances in which long-term memory is impaired in patients with cerebellar disorders, however, and this brings us to the consideration of diseases that have a cerebellar component but that also involve other areas of the nervous system. Some of the SCAs are known to have pathological changes in regions of the cerebral cortex involved in memory, so one has to be careful about ascribing all cognitive deficits that arise in these patients to the cerebellum itself. The recently recognized Fragile X associated tremor ataxia syndrome, for example, includes forgetfulness along with spatial disorientation and loss of executive skills. The memory deficit may be too prominent to be a purely cerebellar problem.

**The cerebellum in non-ataxic disorders**

As the understanding of the cerebellar role in behavioral neurology and neuropsychiatry matures, we are in position to pay closer attention to findings suggesting a cerebellar role in dyslexia, attention deficit hyperactivity disorder, autism spectrum disorders, schizophrenia and developmental delay. When the cerebellum is studied with anatomical tools that measure each lobule of the cerebellum carefully, and compare it against healthy control populations, it is apparent that there are differences in the cerebellum between controls and individuals with these behaviorally defined conditions. There is much work to be done in these areas, but now we know that cerebellum is engaged in intellectual and emotional processing; that it seems to be important for automaticity, learning, and modulation of behaviors; that neurobehavioral syndromes result from cerebellar lesions; and that there is sufficient evidence to be interested in the possibility of cerebellar modulation of higher order behaviors in these purely behavioral disorders.

**What this means for you**

This new field of study of the cognitive neuroscience of the cerebellum has direct clinical implications for the ataxia patient population. The first is what I call “the need to know” imperative. It is reassuring to patients and families to know that in the setting of a disorder, particularly a neurodegenerative one in which we have not yet come up with the cure, that the kinds of challenges you face beyond motor control are not in your head, rather, they are in your brain. Second, we can do something about this. There is a long tradition in medicine and the brain sciences of treating symptoms to alleviate distress, even when we cannot cure the underlying disease. That is a central role of the physician, and one that you should expect from your doctor. So without endorsing any off-label indication for any particular product, there is every reason to consider the use of the newer antidepressant medications to treat depression, pathological laughing and crying that responds to these medications, memory enhancing medications that are used in the treatment of Alzheimer’s and other degenerative disorders that affect memory. These need to be studied, perhaps with the help of NAF, in a collaborative manner in patient populations across the country. Cognitive rehabilitation strategies are routinely used in patient with brain trauma and stroke. Physical therapy can be beneficial in patients with ataxia, and cognitive rehabilitation therapy for patients with higher order deficits in the setting of ataxia can also be helpful. Indeed, there is reason to believe that cerebellar disorder in patients may do better than patients with cerebral injury, because one may be able to learn strategies to compensate for the automaticity that is lost when patients with cerebellar dysfunction lose the modulation of mood and intellect. Medications have been used in other patient populations for enhancement of executive function, and studies are needed to evaluate whether this may be beneficial to ataxia patients as well.

If these issues of intellect and emotion seem relevant to your own clinical situation, I urge you to discuss them openly and frankly with your
doctor. If your doctor is not aware of this relationship between cerebellum and intellectual and emotional processing, then suggest some articles they could read to help them understand what you may be going through – including this one, and other select references below. The help of a good counselor can be invaluable to engage in talk therapy. This is not a sign of weakness or failure, but recognition that there are both brain-based issues and real-life social and psychological ramifications of the illness you are experiencing. At the same time, don’t forget the phenomenon we now recognize well in the very different world of Alzheimer’s disease management, in which caregiver burden is dangerous and should be managed. We need to make sure that the healthy partner, parent, or child is also taking care of him/herself during this marathon of dealing with cerebellar disorders.

**Some future directions**

Neurosurgical approaches using deep brain stimulation, and the technique of non-invasive transcranial magnetic stimulation (TMS), have been successfully used in intractable depression, but it remains to be shown whether these are reasonable approaches in the ataxia population. We are currently embarking on a program of TMS of the cerebellum in patients with schizophrenia, based on my dysmetria of thought theory, in the hope that enhancing cerebellar modulation of disordered regulation of emotion and cognition may improve this common and disabling mental health problem.

**References**


Featured Board Member of the NAF: David Zilles

Dave Zilles attended Vanderbilt University where he received a Bachelors of Arts Degree in Mathematics. He had a 33-year career with IBM which included several executive marketing and sales positions. He retired in February 2004, and has been residing in Atlanta, GA for 25 years.

David and his wife Linda have been married for 39 years. They have three children and three grandchildren. Their youngest son, Jon, has Friedreich’s ataxia and was diagnosed at age 11. Jon began using a wheelchair at about age 17. He learned to drive with hand controls, graduated from high school and went on to Auburn University where he graduated with a degree in Psychology. Jon was able to get around Auburn with the assistance of his service dog, Mango, from the Canine Assistants organization in Alpharetta, GA. Jon is currently living independently in his own condo as a result of being on the Medicaid Independent Care Waiver, which provides him with personal assistance in the mornings and evenings.

Dave is one of the founding members of the Greater Atlanta Ataxia Support Group and is currently one of its directors. In addition, he serves on the Board of the State Independent Living Council for Georgia in the position of Treasurer. He has been very active in advocating for young adults with disabilities through the Unlock the Waiting List organization which advocates for the Independent Care Waiver program by meeting with state legislators and providing public testimony. Dave is a member of the 2007-08 Partners in Policymaking class, sponsored by the Georgia Governor’s Council on Developmental Disabilities, and serves on the board of the MDA Seek-a-Miracle Foundation.

Dave joined the National Ataxia Foundation’s Board of Directors two years ago. He has been a very active board member and has brought various initiatives to the Board to help better serve the ataxia community. Dave has a strong feeling that the future of NAF lies within the young adult ataxia community and wants to see more young adults get involved with the Foundation.

Dave stated, “I believe that with all the research available today that sometime in the future a treatment will be found.” He continued, “So much has happened since I first learned about FA in September of 1989. My family did not know anything about ataxia and we felt so alone until we attended our first NAF conference in New Orleans. We had the chance to meet many wonderful people at our first meeting, including Jerry Lawrence, the LeBlanc’s and most importantly Earl McLaughlin and his family.”

Dave has been an outstanding NAF board member and truly cares about the ataxia community. Although his family is affected by Friedreich’s ataxia, Dave is an advocate to families who have been impacted by all forms of ataxia. Dave concluded by stating, “It has always been important to us to know that there is hope and that others have been where you are and can help you along the way. The National Ataxia Foundation and the NAF support groups significantly help in these important areas.”

We are truly grateful for Dave’s leadership, commitment, and dedication in giving ataxia families hope. We are honored to have Dave Zilles on the NAF Board and are thankful for all he does in helping the ataxia community. Thank you, Dave.
The National Ataxia Foundation celebrated its 51st Annual Membership Meeting, “Blazing a Trail in Research,” which was held in Las Vegas on March 28-30, 2008. The meeting was hosted by the Arizona Ataxia Support Group and was attended by the largest gathering of ataxians in the history of the organization. It was wonderful seeing old friends and meeting new friends from across the United States and throughout the world.

Preceding the annual membership meeting, the Foundation hosted its Second International Ataxia Investigators Meeting (see story on page 9). This four-day meeting brought together approximately 120 world leading ataxia scientists, clinicians, and young investigators to share information, encourage collaboration and cooperation, and to help accelerate world-wide research. We are grateful to all these researchers for participating in this most important meeting and to our generous sponsors who made this meeting possible.

At the 2008 NAF Annual Membership Meeting, an overview of the previous year’s activities was presented. Here are some of those highlights:

- In December 2007 NAF made a research funding commitment of more than $850,000 and reviewed a total of 42 research applications. This is the largest number of applications considered in the history of NAF.
- In 2007 NAF awarded funding to 17 promising ataxia research studies, including studies conducted in the United States, Italy, Portugal, Switzerland, Belgium, Austria, Australia and the United Kingdom.
- In 2007 NAF added its fourth research program called the “NAF Friedreich’s Ataxia Special Projects Award.” In late December 2007, NAF awarded its first $200,000 study through this new NAF research program.
- A number of scientists who received support from NAF for fiscal year 2007 announced that through support from NAF they had received additional funding from NIH. Some of these researchers received a five-year NIH grant and one received a hundred-fold increase in support compared to the initial grant NAF awarded.
  - Over the past nine years the National Ataxia Foundation has awarded funding to 115 vital ataxia research studies in 10 countries.
  - From December 2004 to December of 2007, the Foundation made an ataxia research funding commitment of more than $2,400,000.
  - In 2007, NAF, FARA and Ride Ataxia combined forces to fund the first $100,000 Kyle Bryant Translational Research Award. Because of the quality of the applications, three additional studies were co-funded by NAF and FARA.
- Membership support and family fundraisers continued to see increases in 2007. One family alone raised nearly $100,000 for ataxia research.
- In 2007, Charities Navigator, America’s largest independent evaluator of charities, awarded NAF a 4-Star Rating out of a possible four stars for the fourth consecutive year, giving an indication of sound management and solid fiscal responsibility. Only 5% of the charities reviewed by Charities Navigator receive four or more consecutive 4-Star ratings. NAF was also selected as one of the “Top Ten” nonprofits with budgets under two million dollars.
- NAF provided programs and services in all 50 states and in 63 foreign countries.
- In 2007 NAF had 7,690,039 “hits” on its web site with 691,088 visitors. 45% of the visitors came from the United States with the remainder from 114 other countries, including Canada, Mexico, Germany, Spain, Italy, France, Portugal, Greece, Japan, India, Hong Kong, China, Australia, Brazil, Peru, Cuba, South Korea, Pakistan, Russian Federation, Iran, Iraq, and Vietnam.
- In 2007, through the initiative of the Chesapeake Chapter of NAF and funding from the Gordon and Marilyn Macklin Foundation, the National Ataxia Foundation awarded a three-year $450,000 grant to Johns Hopkins for the establishment of the Johns Hopkins Ataxia Center.
- In 2007 NAF continued to update and
Second International Ataxia Investigators’ Meeting (AIM)

By John Day, MD, PhD, Professor of Neurology & Pediatrics at the University of Minnesota

Immediately preceding the 2008 NAF Annual Membership Meeting in Las Vegas, more than 120 ataxia investigators from around the world assembled for a four-day meeting to discuss advances in the ataxias. The meeting detailed relevant aspects of the normal cerebellum, what can go wrong during brain development to cause ataxia, and how aging and brain degeneration can also lead to ataxic disorders. A primary focus of the program was to describe how to characterize ataxias so that meaningful treatments can be brought to patients as quickly as possible.

One of the first day’s highlights was a discussion of normal cerebellar development — there is evidence that abnormal development can underlie some aspects of cerebellar degeneration that occurs later in life. The first day’s program also detailed the increasingly refined understanding of how genetic abnormalities cause nerve cells to sicken and die, which has led to the identification of targets appropriate for drug treatment, most importantly in SCA-1, for which a pilot study trial is now underway at NIH.

The second day of the AIM focused on refining our understanding of disease mechanisms in ataxia, with discussions related to the identification and testing of treatments. Many Friedreich’s ataxia investigators detailed various aspects of the exciting treatment trials now underway. The program on the final day focused on the ataxia develop various ataxia publications, including the 48-page quarterly news publication, Generations. The Winter 2007-08 issue was expanded to 56 pages. Generations now has a readership of more than 30,000 worldwide. In addition, the NAF publication “Evaluation and Management of Ataxic Disorders, An Overview for Physicians,” written by Dr. Susan Perlman, was released in 2007.

• In 2007 the Foundation received nearly 10,000 telephone calls, over 12,000 letters, and more than 42,000 e-mails from persons with ataxia, family members, the medical community, other organizations, and the general public.

• NAF continued in 2007 to update and participate in various conferences including the American Society of Human Genetics and the Society of Neuroscience in San Diego, the American Academy of Neurology Conference in Boston, an NIH conference in Washington, DC, and various Abilities Expos, including one in Minneapolis.

• In 2007 NAF continued to update and add additional sections on its web site to provide current and accurate information. Also, in 2007, NAF expanded its on-line giving efforts and added In Honor Of and In Memory Of sections where donors could write about the person they were honoring and also include a picture of the person honored on NAF’s web site.

• The National Ataxia Foundation celebrated its 50th year in serving ataxia families at the 2007 NAF Annual Membership Meeting in Memphis. Elvis even appeared to help in the celebration! The meeting was hosted by the Mississippi Chapter of NAF, which donated $50,000 during the Saturday night banquet.

These and other program initiatives were made possible because of important support from our generous donors: individuals, corporations, foundations, United Ways, families, Combined Federal Campaigns, chapters/support groups, research and membership drives and others. It is through your gifts that the Foundation is able to continue its important work. Your support truly makes a difference. Thank you.

Continued on page 23
Write Your Letter of Intent NOW!

Planning For the Financial Future of A Person With A Disability

By Arnie Gruetzmacher

This is the second part of a five-part series of articles regarding the Life Plan. Arnie has spent the last 27 years working with families of persons with a disability assisting them in preparing a Comprehensive Life Plan. If you have any questions regarding financial/estate planning, please e-mail or address the Editor and Arnie will reply in our next issue or contact you directly.

Life Planning: The Life Plan

In the last issue I presented the four components necessary to develop a Comprehensive Life Plan to provide a quality of life for your family member with a disability. Those four components are: Life Plan, Legal, Financial and Plan Management. In this issue I will explain in detail why the Life Plan component is important and how to put it together.

The Life Plan component is a written plan. It will provide your successors with all the information necessary to assure that all of your hopes and desires for your family member with a disability will occur even if you are incapacitated or are no longer here. This information should be written in a Letter of Intent. The Letter of Intent will contain the Life Plan.

The Letter of Intent should contain information regarding the future in the following areas: Residence, Education, Employment, Medical Care, Behavior Management, Social Activities and Religious Endeavors. To begin, take a pad of paper and across the top place the title “Concerns for Letter of Intent” and under the title list each of the above areas. Then under each area list four to five choices in the order of preference. As much as possible, the information in the Letter of Intent should be the result of discussions between the primary care provider and the person with a disability.

With these choices made, you now can start writing the Letter of Intent. Don’t worry about sentence structure or correct punctuation because the only persons reading the Letter of Intent will be your successors. Address your Letter of Intent “To Whom it May Concern” followed with basic information regarding the primary caregiver(s) whether they be parents, spouse or other. This should include name, relationship and address. Continue with a history of the person with a disability.

Next use the sheet of “Concerns for Letter of Intent” that you developed and for each area list information regarding the Past, Present and Future for your family member. Give as much detail as possible and be sure to indicate what didn’t work so your successors won’t have to reinvent the wheel. Be as specific as possible. Remember, the sole purpose of this letter is to assist your successors in knowing all there is to know regarding your loved one with a disability and to make your hopes and desires known. The more detail you have provided the less stress there will be for your successors and the person with a disability.

The Letter of Intent should be dated and signed by those person(s) responsible for writing it. Although the Letter of Intent isn’t a legal document, it is meant as a guide to the courts and your successors as to your hopes and desires.

Without the Life Plan as a component of your Comprehensive Life Plan you will create chaos for your successors and the person with a disability. Let these people know you care and write your Letter of Intent NOW!

In the next issue of Generations, I will be explaining the Legal Component.
Ataxia Center Established at Johns Hopkins University

By Carl J. Lauter

It is with great honor and pleasure that the Chesapeake Chapter of the National Ataxia Foundation (CC-NAF) announces the establishment of an Ataxia Center at the Johns Hopkins University School of Medicine (JHUMS) in Baltimore, MD. The center will not be a physical space, but rather a “new way of doing business.”

Why an Ataxia Center?

To coordinate patient care so that comprehensive treatments can be delivered effectively and efficiently. It will be a multidisciplinary center which coordinates patient care with a team of physicians, therapists and nurses who will meet prior to each clinic to review each patient. A clinical case manager dedicated to the center will coordinate appointments on the same day, so that patients may, after first being examined by the physician, be immediately directed to additional specialists (such as for eye movement), therapists (such as for speech, swallowing, physical movement), counseling (such as for psychological, genetic), or for educational materials or contact with other patients through individual counseling or support groups.

To promote collaboration among physicians and researchers across departments. An Ataxia Center research coordinator will be on site to screen and enroll patients in ongoing research trials if the patient indicates willingness to participate, increasing the pace of discovery for new treatments and potential cures for ataxia.

To accelerate research by increasing enrollment of patients in clinical trials, and fostering multidisciplinary relationships among investigators. The research coordinator will screen and enroll patients in appropriate research studies.

To incorporate innovative technologies into patient care and research. This means that as new technologies and treatments are discovered they can be applied to patient care. Also, as patients enter research trials, their findings can be incorporated into the general understanding of ataxia dysfunctions, which can lead to further methods of treatment.

Why an Ataxia Center at JHUMS?

It has been the dream of several of our CC-NAF members to create such a center at nearby JHUMS for a more efficient mode of visiting and coordinated treatment. Also, JHUMS and the Johns Hopkins Medical Institute (JHMI) are...

Continued on page 12
Ataxia Center
Continued from page 11

known world-wide as one of the most reputable medical centers. Having an Ataxia Center at JHUMS will bring ataxia patients from a wide geographical area of the East Coast and worldwide to be diagnosed and treated by renowned neurologists and other neurological specialists.

What will be the role of CC-NAF in relation to the Ataxia Center?

The CC-NAF has been collaborating with JHMI for many years in securing speakers for their meetings and annual Medical Meetings. Many CC-NAF members, in turn, have volunteered their time in participating in research studies of various investigators at JHUMS and the adjoining Kennedy-Krieger Research Institute (KKRI). Thus, in collaboration with the CC-NAF, patients will be referred for additional support services in their community. Individuals from the CC-NAF will also be available to meet with other individuals and families who have recently been diagnosed or who could benefit from additional support as the disease progresses. In addition, the CC-NAF will be instrumental in helping to support the operation of the Ataxia Center through donations to the CC-NAF specifically designated for the Ataxia Center.

Development and Operation of the Center

The initial concept of an Ataxia Center was spearheaded about four years ago, prior to June 2004, when meetings were held in Catonsville, MD, by two neurologists at JHU, Sarah Ying, MD and Elizabeth O’Hearn, MD, in conjunction with members of the CC-NAF Board (A-Team), Libby Labash, Vice President; Carole Connor, Membership Chair and Treasurer; Bill Lee, Secretary; and Carl Lauter, President. Discussions were pursued as to the need for such a Center, the operation of it, and the source of funding. As the years passed, it was noted that one of CC-NAF’s families, Marilyn and Gordon Macklin, were generous benefactors to the CC-NAF and favored the idea of NAF’s research programs. Upon the untimely death of Marilyn with ataxia, later followed by the death of Gordon, it was realized that the Macklin children and Don Dawn, Gordon’s nephew and President of the Gordon and Marilyn Macklin Foundation, were very sympathetic and favorable toward continuing support for NAF’s ataxia research programs through continued gifting to CC-NAF. During 2007, Libby Labash was instrumental in discussing and pursuing a business plan with Mr. Dawn which would help to create an Ataxia Center. The plan of the Gordon and Marilyn Macklin Foundation is to fund the Ataxia Center with gifts of $150,000 per year for three years. Additional funding may be forthcoming when it is observed as to the manner and quality of operations of the Center. The Macklin Foundation will directly fund the NAF with their donation. Credit will be given to the CC-NAF for “assistance and support” toward this project. NAF will pass through 100% of the specified funding to the Ataxia Center with the stipulation that 100% of this gift goes directly to the Center’s operation, with no take-off for JHUMS administrative use. The CC-NAF is eternally grateful to the Macklin family for their past generous support and future commitments toward the goal of helping with the life and comfort of the ataxia patient and for the advancement of research.

The CC-NAF is also grateful for the NAF’s Board of Directors approval earlier in the year of this concept and project and the Johns Hopkins University and Neurology Department administrative staff for their collaborative work with the Macklin Family to negotiate a viable contract between all parties concerned.

Once funded, the Ataxia Center should be operational in early 2008. A contact address and phone number will be provided upon establishment of the Ataxia Center. Come and visit the center, tell your friends, and make a contribution! The doctors are ready and waiting!
Kyle Bryant and 15 other cyclists were welcomed by more than 100 well wishers as they arrived on Thursday, March 27 at the Flamingo Hotel in Las Vegas, NV to help kick off the 2008 National Ataxia Foundation’s 51st Annual Membership Meeting. This is the second year Kyle and others have left from California to finish their journey at the NAF annual membership meeting.

Kyle, who has been diagnosed with Friedreich’s ataxia, and others began the first Ride Ataxia in 2007, beginning in La Jolla, CA and ending in Memphis, TN, the site of the NAF 50th Annual Membership Meeting. The 2,400-mile bike trip helped raise ataxia awareness and raised $40,000 to support promising Friedreich’s ataxia research.

Through funding support from Ride Ataxia I and joint funding from NAF and FARA, a $100,000 Kyle Bryant Translational Research Award was made to Australian researchers and to their British collaborators.

At the 2008 Annual Membership Meeting’s Friday night reception, Kyle gave an extraordinary PowerPoint presentation on the Ride Ataxia II team’s incredible and heroic journey. After the presentation, Arnie Gruetzmacher presented a medal to each rider from Ride Ataxia II to commemorate their incredible journey.

The National Ataxia Foundation applauds the efforts of Kyle and the other riders and sponsors for making Ride Ataxia II so successful. This year’s bike ride continued to raise the level of ataxia awareness and raised more than $110,000 to support vital Friedreich’s ataxia research. NAF, FARA, and Ride Ataxia II will be teaming up again this year in support of the 2008 Kyle Bryant Research Award.

The Ride Ataxia II team celebrates after arriving in Las Vegas.

Thank You Athena!

Athena Diagnostics is a reference laboratory dedicated to the development of diagnostic testing for neurological disorders, including the ataxias. Athena is a leading provider of advanced neurological diagnostic assays, which are performed in their lab in Worcester, MA.

Over the years, Athena Diagnostics has continued to contribute to the National Ataxia Foundation’s annual membership meetings and to the NAF Ataxia Investigators Meetings (AIM). Athena again this year has made a funding commitment to support the 2008 Ataxia Investigators Meeting.

The National Ataxia Foundation is truly grateful to Athena Diagnostics’ continued partnership with the Foundation in serving ataxia families. Thank you Athena!
Friedreich’s ataxia is the consequence of frataxin deficiency. The relevance of the frataxin protein has been assessed in cellular models by showing that the phenotype can be completely reverted by the complementation of the protein. Unfortunately substitutive treatments, as gene therapy approaches, seem to be far away to be readily accessible within the near future. Under this point of view, drugs able to increase the frataxin amount are excellent candidates for a rationale approach to the FRDA therapy. But, up to now, although several drugs have been proposed, there still is no treatment available. It was recently demonstrated that both erythropoietin and HDAC inhibitors can increase the intracellular levels of frataxin in in-vitro models. HDAC inhibitors seem to increase the mRNA transcription while erythropoietin seems to increase only the protein amount. The mechanism by which erythropoietin acts in enhancing frataxin protein amount is not yet elucidated. The relevance of the erythropoietin as potential drug for Friedreich’s ataxia is based mainly on its availability as a commercialized drug. Therefore it could be tested in patients without undergoing the long pharmaceutical and first phase clinical trials. The present project is aimed to test the possible synergy between erythropoietin and a selected HDAC inhibitor, the 4B-BML-210-der. We will perform tests in cellular models, to assess their efficacy alone and in combination, in order to find the best effective dose and administration timing. The results will be useful to plan further clinical trials.

Cláudio M. Gomes, PhD
Instituto Tecnologia Química e Biológica, Universidade Nova de Lisboa, Portugal

Frataxin folding, chaperone role and interactions with iron-sulfur biosynthesis proteins: contributions to understand Friedreich’s ataxia

The neurodegenerative disease Friedreich’s ataxia (FRDA) is the most common of hereditary ataxias with an estimated prevalence of one in 50,000 individuals. This pathology results from a reduction in frataxin levels, a small protein localized in the cellular organelle (mitochondrion). Frataxin function is not yet fully understood but previous data shows a role in the formation of iron-sulfur clusters, the inorganic cofactors that will be incorporated into many different proteins and are essential for life. A more precise understanding of the biological function of frataxin is necessary in order to better understand how FRDA is developed. The present project focuses on the study of the interaction between frataxin and other proteins involved in the biosynthesis of iron–sulfur clusters, using genetic, biochemical and biophysical methodologies.

Michael D. Hebert, PhD
The University of Mississippi Medical Center, Jackson, MS

Disrupted pre-mRNA splicing in Machado-Joseph disease

The cell nucleus is highly organized and contains distinct domains, territories and bodies. Many of these structures participate in the efficient production and processing of RNA. Disruptions in this process will impede the
flow of information from DNA to RNA to protein and lead to cell death. Several neurodegenerative disorders, such as Huntington’s disease, spinocerebellar ataxia-type 1 and Machado-Joseph disease (MJD), are characterized by mutant proteins that generate large nuclear inclusions in neuronal cells. While these inclusions could be pathogenic, beneficial or incidental, it is clear that various nuclear domains differentially associate with specific types of inclusions. In particular, one nuclear domain known as the Cajal body has been shown to associate with inclusions in MJD patient cells. The mutated protein in MJD is ataxin-3. Since the Cajal body plays a role in making the machinery needed to properly process RNA, it is suspected that Cajal body function may be impaired in MJD. Preliminary data support this suspicion. In this application, we will examine if Cajal body activity is altered in neuronal-like cell lines that can express normal and mutant forms of ataxin-3. By fully understanding the mechanisms by which neuronal cells die in certain neurodegenerative disorders, advances can be made in the development of treatments.

James Maylie, PhD
Oregon Health & Science University,
Portland, OR

Molecular and Cellular Physiology of Episodic Ataxia Type 1, EA1

Episodic ataxia type 1 (EA1) is an autosomal dominant neurological disorder in which affected individuals experience attacks of imbalance and uncontrolled movements, often associated with jerking or tremor of the head and arms and asynchronous twitching of muscle fibers within the facial and hand muscles. Genetic linkage studies and DNA sequence analysis have identified mutations in KCNA1, the gene encoding the voltage-gated potassium channel, Kv1.1, as underlying EA1. Potassium channels are important contributors to the geneses and modulation of electrical activity in every cell particular in the central nervous system. Most affected individuals respond favorably to treatment with acetazolamide, a carbonic anhydrase inhibitor, that reduces the frequency of attacks. The basis for benefit provided by acetazolamide is not understood and one goal of this application is to provide an understanding of the cellular mechanism by which acetazolamide works, as well as provide an animal model to test other potential treatments. The classic neurological concept that the cerebellum is a site of motor control originates from clinical observations that patients with cerebellar damage often exhibit motor-related symptoms of gait ataxia, dysmetria, hypotonia, and tremor. The Kv1.1 gene is expressed in interneurons of cerebellum that release the neurotransmitter GABA, the major inhibitory transmitter in the brain, but not in Purkinje cells, the primary cells that project information out from the cerebellum. It is therefore likely that EA1 symptoms results from altered GABA release. To understand the cellular and behavioral mechanisms that result in EA1, we have constructed a transgenic mouse model for EA1. We will use the EA1 mouse model for behavioral, electrophysiological, and calcium imaging investigations. These experiments will comprise an exemplary use of a diverse technical repertoire and integrated approaches to understand an inherited human disorder in an ion channel gene. The EA1 mice may also suggest and also serve for testing future therapeutic approaches.

Puneet Opal, MD, PhD
Northwestern University, Chicago, IL

Dissecting transcriptional misregulation in SCA1 using laser capture microscopy and transcriptional profiling

Spinocerebellar ataxia type 1 (SCA1) is an inherited disease that causes progressive instability
of gait or ataxia. Unfortunately, there is no treatment for this relentless disease and those afflicted succumb to complications of cerebellar and brainstem dysfunction. This disease is caused by an expansion of glutamines (glutamine is an amino acid) in the disease causing protein, ataxin-1. A major shortcoming in our understanding of SCA1 has been the lack of characterization of pathologic changes in Purkinje cells – the cell type most afflicted in this disease. To address this issue we will use laser capture microscopy (LCM), a method designed to purify specific cell populations from tissue samples from pre-symptomatic SCA1 knockin mice. LCM will then be combined with gene-expression profiling to characterize the earliest transcriptional alterations in SCA1. Apart from providing significant insight into SCA1 pathogenesis, we foresee that these experiments will lead to targeted therapies that might reverse the transcriptional alterations seen in this disease.

David M. Ornitz, MD, PhD  
Washington University School of Medicine, St. Louis, MO

Biological tools to investigate Spinocerebellar Ataxia 27 (SCA27)

Intracellular Fibroblast Growth Factors (iFGFs) are important regulators of the activity of many different neurons in the brain and throughout the body. FGF14 is one of four iFGF genes. When the Ornitz laboratory completely inactivated the FGF14 gene in mice (FGF14 knockout mouse), the mice developed an ataxia syndrome in which they were uncoordinated, showed spontaneous abnormal movements (called paroxysmal dystonia) and occasionally had a tremor. The published phenotype of the FGF14 knockout mouse directly led to the discovery of a mutation in the FGF14 gene in a large human family in which affected individuals have a progressive spinocerebellar ataxia, which is now classified as SCA27. One major difference between the existing mouse model and the human disease is that the knockout mice completely lack a functional FGF14 gene, whereas affected humans have one normal copy of the FGF14 gene and one mutant copy. Thus, although the existing mouse model and human disease phenotypes resemble each other, the underlying molecular mechanism that causes the disease in knockout mice and humans must be different. The Ornitz laboratory, with expertise in making and studying mouse models, is collaborating with the Nerbonne laboratory, with expertise in neurophysiology, to understand the role of FGF14 in regulating the activity (excitability) of neurons and why, in the absence of FGF14, mice develop an ataxia syndrome. A key question that we will answer is how a single mutation in one copy of human FGF14 results in a disease similar to that found in mice that are missing both copies of their FGF14 genes. To fully understand the human SCA27 disease mechanism and to be able to test possible interventional strategies, we will construct a mouse model with a mutation that is identical to the human SCA27 mutation. We will also generate and test antibodies that will allow us to detect the normal and mutant FGF14 protein in mice and humans. With the availability of these essential tools, we will be in an excellent position to apply for NIH funding so that we can expand our research program and investigate detailed molecular mechanisms and therapeutic interventional strategies that could benefit patients with SCA27 and potentially patients with other forms of ataxia and other movement disorders.

Henry Paulson, MD, PhD  
University of Michigan, Ann Arbor, MI

RNAi as therapy for spinocerebellar ataxia type 3 (SCA3)

Spinocerebellar ataxia type 3 is a dominantly inherited form of ataxia for which currently
there is no cure. Because the disorder is caused by a “toxic” disease protein, turning off production of the disease protein is a promising route to therapy. We propose to exploit the power of RNA interference (RNAi) to silence the disease gene (ATXN3) in a mouse model of SCA3. The mouse model we have chosen expresses the full human ATXN3 gene with its normal promoter, making it as close to the human situation as we can get at this time. We and others have already proven that RNAi can suppress the expression of disease-causing genes, as in SCA3. Our goal here is to take advantage of our lab’s expertise in RNAi and in SCA3, our demonstrated commitment to this disease, and our experience with this mouse model to carry out necessary preclinical studies that are desperately needed if RNAi is ever to be brought to the clinic for patients with SCA3. Our studies will create two different RNAi reagents targeting different regions in the disease gene; both will be expressed via engineered viruses that can efficiently be delivered to the brain and result in sustained “knock-down” of the targeted disease gene. If the studies are successful, we will be well-positioned to go forward with second phase preclinical studies which are longer term and beyond the scope of the current one year proposal. Completion of the currently proposed studies will allow us to compete successfully for federal funding to continue the work and, we hope eventually, bring RNAi to the SCA3 clinic.

Parminder J. S. Vig, PhD
University of Mississippi Medical Center, Jackson, MS

Role of Bergmann glia in Purkinje cell development and pathology in SCA1

Spinocerebellar ataxia 1 (SCA1) is a dominantly inherited disease caused by the abnormal functions of ataxin-1 protein. SCA1 is associated with progressive ataxia resulting from the loss of cerebellar Purkinje cells and neurons in the brainstem. A prominent feature of SCA1 pathology is the presence of cytoplasmic vacuoles in Purkinje cells. In SCA1 mice, these vacuoles start appearing early on before the onset of ataxia. It is not understood why vacuoles are formed, however, they may have a role in Purkinje cell pathology. Our recent data show that the process of vacuolar formation is associated with degenerative changes in Purkinje cells. In addition, these vacuoles contain neighboring Bergmann glial specific proteins and are present both in transgenic mice and human patients with SCA1. Bergmann glial cells locate their cell bodies around Purkinje cells, and help Purkinje cell processes to grow and develop. We believe that early and continuous expression of mutant ataxin-1 in Purkinje cells compromises normal signal transduction mechanisms by impairing Purkinje cell Bergmann glial crosstalk. This leads to vacuole formation and Purkinje cell death in SCA1. Therefore, to understand both the role of Bergmann glia in SCA1 pathogenesis and the functional relevance of the presence of glial-derived vacuoles, we will study developmental changes in different kinds of transgenic and wild-type mice. Using cell culture, it will be assessed if an interaction between Bergmann glia and Purkinje cells is required for vacuole formation. Purkinje and Bergmann glial cell cultures will be prepared from zero to one day old mouse pups. To determine if glial protein S100B targets are affected in SCA1 Purkinje cells, S100B inhibitor will be intranasally administered to SCA1 mice to see if this treatment suppresses vacuole formation and SCA1 pathology. The significance of the present study is that an early identification of abnormal communication pathways between Bergmann glia and Purkinje cells could be targets of potential therapies for pre-symptomatic patients with SCA1. The long-term objective of this project is to understand neuron-glial relationship in neuronal degeneration in order to design therapeutic approaches in clinical management of SCA1 or other cerebellar ataxias.

Continued on page 18
RNAi therapeutics for Friedreich’s ataxia

Current therapeutic initiatives for Friedreich’s ataxia comprise the screening and/or testing of conventional chemical compounds – to increase the expression of frataxin, to mitigate or reverse the deleterious effects of decreased frataxin expression on cellular energy production, or to reverse the subcellular iron accumulation that apparently contributes to the signs and symptoms of the disorder. The purpose of the experiments described in this proposal is to identify therapeutics for Friedreich’s ataxia using an alternative, and completely novel, approach. This approach is a selection screen for therapeutics based on what is called RNA interference (RNAi). RNAi is a recently discovered mechanism by which cells regulate gene expression, and which can be exploited for therapeutic purposes. Although delivery issues for RNAi therapeutics are still being worked out (i.e., how to get RNAi constructs into particular cell types), several companies are already involved in clinical trials of RNAi therapeutics, including Acuity (age-related macular degeneration, diabetic retinopathy), Alnylam (respiratory syncytial virus), and Sirna (age-related macular degeneration, hepatitis C). For this proposal, we will put several hundred thousand separate RNAi sequences into several hundred thousand cells (in a tissue culture flask) from an individual with Friedreich’s ataxia. We will then select cells that thrive under conditions normally deleterious to Friedreich’s ataxia cells and retrieve the RNAi sequences that allow the cells to thrive. (In effect, we let the cells tell us which sequences work best.) Through an iterative process of varying the most beneficial sequences, re-screening the variants in cells, and retrieving those that are even more beneficial, we will optimize RNAi sequences as potential therapeutics for Friedreich’s ataxia. We will partner with one or more of the companies involved in RNAi therapeutics to bring the best sequences into clinical development. Although this proposal focuses on Friedreich’s ataxia, the experiments will serve as a proof-of-principal for a similar approach to identifying RNAi therapeutics for the dominant ataxias.

Fragile X tremor ataxia syndrome (FXTAS) is a neurological disorder that causes tremors, balance problems, difficulty in walking, dementia and mental changes in males usually after age of 50. The underlying defect for this neurological disorder is the gene FMR1 responsible for fragile X syndrome, the most common inherited cause of mental retardation. In FXTAS, unlike fragile X syndrome, FMR1 gene produces a toxic messenger RNA because of the abnormally high numbers of the repeating DNA sequences (55-200 CGG units) called premutation. Modeling the FXTAS disease in Drosophila has shown that a particular group of RNA binding proteins in the brain are sequestered by these toxic messenger RNAs, thereby preventing them to perform their normal function. Among the sequestered proteins is Purα that is particular because loss of Purα in mice produces FXTAS like symptoms, suggesting an important role for Purα in the disease mechanism of FXTAS. This project proposal is aimed to understand the cellular and molecular pathways affected due to sequestration of Purα in the brain because of toxic messenger RNA. This proposal is also aimed to utilize Drosophila FXTAS model for screening FDA approved small molecule libraries for potential therapeutic intervention for future use in humans.
Non-progressive congenital ataxias represent a genetically heterogeneous group of neurological disorders. Several of these ataxias are caused by defects of the cerebellum, a region of the brain in charge of balance and coordination of motor functions. Some of these defects may be caused by an embryonic developmental deficit. A key step during cerebellar development is the inward migration of a certain type of neuron, the granule cells, from the external granule cell layer (EGL) to the internal granule cell layer (IGL). This migration is tightly regulated by signaling cues that are known to attract and repel cells and axons. Previous data suggests that a cytoplasmic protein, p130Cas, may be required for some of these signaling cues to be able to instruct cerebellar cells to move. However, little is known about Cas protein function in neuronal migration and cerebellar development. The objective of this proposal is to define and characterize the function of vertebrate Cas proteins in granule cell migration and axon guidance. To achieve this objective we will take different approaches, including the generation of a mouse deficient for p130Cas, which may serve as a model for developmentally caused ataxias.

Spinocerebellar ataxia 2 (SCA2) is an inherited neurological movement disorder caused by mutation in the ataxin-2 gene. The disease is caused by a progressive loss of cells in a region of the brain that controls balance and motor function. Patients have a decrease in fine movement coordination, speech problems and uncontrolled eye movements resulting in an escalating impairment of daily living during the course of the disease. Although the disease gene was identified 10 years ago, the precise mechanisms leading to neuronal dysfunction and nerve cell death have remained elusive. Several years ago, we discovered a protein that binds to atxn2 designated A2BP1 or fox-1. Recently, the function of this protein has been unravelled pointing to a role in processing of messenger RNA and potentially in guiding and stabilizing messenger RNA in specific compartments of the cell. Based on our discovery of interaction of A2BP1 and atxn2, we are now proposing to examine the effects of mutant atxn2 on the processing of messenger RNAs. We hypothesize that mutant atxn2 prevents A2BP1 from its proper function in the nucleus of the cell. We will examine this hypothesis by interaction studies of normal and mutant atxn2 with A2BP1. We will then determine whether mutant atxn2 will cause faulty processing of messenger RNAs. These studies may have general implications for understanding the role of mRNA processing in neurodegeneration, but will also open the door for designing specific therapies for SCA2.

Mutations in the human gene SCA2, which encodes the ataxin-2 protein, can give rise to the neurodegenerative disorders spinocerebellar ataxia type 2 and are also associated with Parkin-
son's disease. The typical biological function of the ataxin-2 protein is currently unknown. Our aim is to uncover this normal function of ataxin-2 in the hope that it will increase our understanding of what may go awry when ataxin-2 is mutated. In order for a gene to be expressed, the DNA of a gene is first transcribed into mRNA molecules. This mRNA can then serve as a template for the synthesis of proteins. Ataxin-2 has been suggested from previous studies (work of several labs including our own) to regulate the expression of mRNA molecules. It is possible that mutations in ataxin-2 may cause neurodegeneration by interfering with the normal regulation of particular mRNAs. We are studying the ataxin-2 protein in the round-worm, C. elegans (homolog, atx-2) to understand precisely how ataxin-2 usually regulates mRNA expression. The main advantage of studying atx-2 in C. elegans is that it is the only model where we already have examples of specific mRNAs whose expression is regulated by ATX-2. Also the ataxin-2 protein shows a high degree of evolutionary conservation making it entirely feasible to study in a model organism such as C.elegans. Future SCA2 therapies will likely be aimed at modulating the function of the ataxin-2 protein. Understanding the normal function of ataxin-2 will greatly aid this process.

Young Investigator Award

Serena Carra, PhD
University Medical Center Groningen, The Netherlands

Role of small heat shock proteins (HspB) in the prevention of mutant ataxin-3 aggregation and toxicity.

Many of the most common neurodegenerative disorders including polyglutamine diseases, such as SCA-3/Machado-Joseph disease are characterized by the aggregation and accumulation of misfolded proteins. By interacting with essential cellular components, mutated proteins can entrap these elements into aggregates, thus contributing to the cascade of neurotoxic events which finally leads to neuronal death. A protective role for molecular chaperones, including Hsp and HspB, against mutated protein toxicity has been extensively demonstrated. In the presence of mutated and instable proteins, which are more prone to aggregate, molecular chaperones avoid their accumulation and/or target them for degradation. Recently, a major role for the autophagy has been recently proposed in the degradation of mutated polyglutamine proteins, including mutated ataxin-3, but little evidence exists demonstrating a role for molecular chaperones in modulating the autophagy-mediated degradation of misfolded proteins. We previously reported that HspB8, member of the HspB family (HspB1-HspB10), blocked mutated polyglutamine proteins (huntingtin, androgen receptor and ataxin-3) aggregation. We recently found that HspB8 forms a stable complex with Bag3. Overexpression of the HspB8/Bag3 chaperone complex inhibited the aggregation of mutated huntingtin and, most importantly, stimulated its degradation through the autophagy pathway. In parallel, we observed that Bag3 can function in the absence of HspB8, thus strongly suggesting that it may cooperate with other members of the HspB family. We will identify the chaperone complexes that facilitate the autophagy-mediated clearance of mutated ataxin-3 and we will analyze their expression levels in tissues and cell cultures from patients affected by the Machado-Joseph disease. This will allow us to focus our future studies on the HspB proteins whose profile is altered in the disease. The selective upregulation of the HspB complexes able to facilitate mutated ataxin-3 degradation by autophagy may have relevant therapeutic implications. In fact, mutations in several HspB cause neurodegeneration, suggesting that they may have important neuroprotective functions. Moreover, accumulation of mutated ataxin-3 >>
within the nucleus is required for the manifestation of the symptoms, supporting the hypothesis that facilitating its autophagy-mediated degradation may contribute to slow down the disease progression.

Miriam Cnop, MD, PhD
Universite Libre De Bruxelles, Brussels, Belgium

Pathogenesis of impaired glucose tolerance and diabetes in Friedreich’s ataxia: contribution of insulin resistance and pancreatic beta cell dysfunction

Friedreich’s ataxia is caused by mutations in the frataxin gene. The vast majority of patients have large expansions in both copies of the frataxin gene, and the size of this expansion correlates with age at onset and the severity of neurological symptoms. In addition to the neurological problems, patients with Friedreich’s ataxia are at risk of getting increased blood sugar levels, or glucose intolerance, and around 20% progress to overt diabetes. The cause of diabetes in Friedreich’s ataxia is poorly understood. Glucose intolerance and diabetes can result from a shortage in insulin secretion by the insulin-producing cells in the pancreas, a poor response to insulin in muscle, liver and fat tissues (insulin resistance), or a combination of both. Previous and contradictory studies addressing the cause of diabetes in Friedreich’s ataxia often used inaccurate methods to measure insulin secretion and sensitivity. They were conducted before the frataxin gene mutation was discovered, and before key concepts for the understanding of glucose tolerance were developed. The aim of the present project is to elucidate why patients with Friedreich’s ataxia develop diabetes. Specifically, we will examine the relative role of pancreatic cell failure and insulin resistance in diabetes development. This will be done using state-of-the-art oral and intravenous glucose tolerance tests in Friedreich’s ataxia patients, first degree relatives and control subjects. Insufficient insulin production by the cells and insulin resistance will be correlated with glucose tolerance and with the frataxin gene expansion size. We present preliminary data showing that, while the Friedreich’s ataxia patients in our pilot study are young and lean, they have a high prevalence of impaired glucose tolerance and diabetes. They are insulin resistant but do not increase insulin secretion, suggesting that their pancreatic cells fail to compensate for the increased demand. We expect that this failure underlies progression to diabetes. Based on the understanding of the cause of diabetes in Friedreich’s ataxia, optimal diabetes treatment choices will be defined, and novel approaches to prevent the disorder envisaged. This study will form the basis of subsequent studies to characterize the function of frataxin in cells and/or insulin responsive tissues using animal models.

Friedreich’s Ataxia Special Projects Award

Brigitte Sturm, PhD
Medical University of Vienna, Austria

The role of frataxin in heme synthesis

Friedreich’s ataxia (FRDA) is a severe genetic disorder affecting approximately one in 50,000 people. The most common FRDA symptoms include muscle weakness and loss of coordination, vision impairment, hearing loss, heart muscle abnormalities, scoliosis, and diabetes. The underlying cause of FRDA is a mutation in the FRDA gene which results in reduced levels of an essential protein named frataxin in all cells of the body. The function of frataxin is still unclear but there is some evidence that frataxin plays a role in heme synthesis (the production of hemoglobin which consists of heme and globin proteins and is essential for oxygen transport in the body). In this study we will investigate the
My Life So Far:
50 Years of Friedreich’s Ataxia

By Brenda Dixon

I was born in Montreal, Quebec in 1957, the eldest of three children. When I was nine, my parents noticed that I was having difficulty going down stairs and was sometimes losing my balance. I was seen by a neurologist and, after a stay in the Montreal Neurological Hospital, I was clinically diagnosed with Friedreich’s ataxia at age 10.

I am not affected with diabetes and so far, not cardiomyopathy. Friedreich’s ataxia affects everyone differently. Some people progress faster than others and some progress quite slowly. So far my major physical changes have been skeletal. My speech is not badly affected unless I’m tired. I had scoliosis surgery in 1988. Rather than Harrington Rods, a new type of rod called C-Rods, where the rods are in sections, were inserted. The bottoms of the rods were attached to the back of my pelvis but in 1991 they broke away so I had to have them removed. My spine was straight for awhile, but over the years my scoliosis has slowly returned.

When I was 11 years old my father was transferred to Richmond, British Columbia. My degenerative progression was slow, so I was still walking. As I went through my teen years I would have an unsteady walk and be taunted and accused of being drunk. Once when I was outside with friends, someone called the police thinking I was drunk.

I graduated from high school and began working at a financial institution in Richmond, where I worked for 28 years. I did a variety of banking duties and worked as a programmer for the last 10 years. My peers watched me progress from a walker to a scooter to a wheelchair. In 2004, I was let go and put on long-term disability.

In 1996, a few of us with various types of ataxia including hereditary ataxia founded the BC Ataxia Society, a non-profit registered
Second International AIM
Continued from page 9

clinical research networks that are coalescing in North America and Europe and expanding their reach to include Australia, South America and Asian countries. Cooperative efforts were organized to define useful patient registries and reliable measures for ataxia severity that will be essential in testing the new treatments being developed.

An exciting aspect of the AIM was the involvement of clinical and basic science investigators from many countries, including Australia, Belgium, Brazil, Canada, China, England, Finland, France, Germany, Italy, Japan, and the U.S. As ataxia research matures, a worldwide collaborative effort is essential so that meaningful treatments can be tested and delivered to affected individuals in all countries as rapidly as possible. Furthermore, although the AIM program was principally organized and supported by NAF, important financial and program support was also provided by the National Institute of Neurological Disorders and Stroke, the Friedreich’s Ataxia Research Alliance, the Friedreich’s Ataxia Research Association of Australasia, the Ataxia Telangiectasia Children’s Project, the Muscular Dystrophy Association, the Gordon and Marilyn Macklin Foundation, the Chesapeake Chapter of the NAF, Athena Diagnostics, ApoPharma, and Santhera Pharmaceuticals. Clearly, to attract this much international participation in the meeting and to garner support from this many disease foundations, ataxia investigation is accelerating in a comprehensive and worldwide approach.

One final important aspect of the 2008 AIM is that it included more than 40 young investigators, recruitment of whom is essential so that we can achieve a long-term goal of the NAF — to successfully control ataxia. Members of the NAF can take pride in what their organization accomplished in the meeting, and can be justifiably optimistic about the cutting edge science that was on display for four days in Las Vegas.

In 1997, I attended my first annual meeting. My first encounter with an attendee of the NAF meeting was with NAF Board Member Earl McLaughlin, whom I met on the plane. He said to me, “After going to this conference you will want to come back every year.” And he was right. I have attended every meeting since. As I tell people, “Many people attending the meeting have some form of ataxia or have a family member with ataxia, so no one stares at you or judges you.”

The BC Ataxia Society has been chosen to co-host the 2009 meeting in Seattle with the Seattle Area Support Group. This will be the first time an annual meeting will be hosted with an international ataxia support group. I am very excited about this and feel honored that BC Ataxia Society has been chosen as the first international support group to co-host a NAF annual membership meeting.

(Please see the 2009 NAF Annual Membership Meeting Invitation on page 27.)
The National Ataxia Foundation’s 2008 Annual Membership Meeting was hosted by the Arizona Ataxia Support Group and held in Las Vegas, NV. Many thanks to the Arizona Support Group for all their efforts and congratulations on a highly successful Annual Membership Meeting. Over 600 people attended the 51st meeting from around the world. Thirty-nine U.S. states were represented, along with attendees from Australia, France, United Kingdom, Germany, Switzerland, and Canada.

Thursday, March 27, was the arrival date of the riders of Ride Ataxia II. Over 100 people gathered to welcome the riders! Congratulations to Kyle Bryant, founder of Ride Ataxia and all the other participants of Ride Ataxia II for their courageous efforts. We thank all those that supported Ride Ataxia II to raise ataxia awareness and funds for important ataxia research.

Friday morning started with the breakout sessions, which provided several resourceful topics including Speech and Swallowing, Genetics and Genetic Testing, Dancing with Ataxia, Financial Planning, Selecting a Patient Care Attendant, Social Security and Medicare, Patient Care Techniques for Caregivers, Emotional Impact of Ataxia, Adaptive Sports, and Living with Ataxia. These topics were presented by individuals with either personal or professional experience. We thank all the presenters for their participation.

Friday afternoon attendees were given the opportunity to meet others with the same type of ataxia in smaller groups, get personal questions answered from ataxia investigators, and share experiences with others through the popular “Birds of a Feather” sessions.

Friday evening the Arizona Support Group hosted a western-themed hors d’oeuvres reception that was well-attended. Delicious food and great company was enjoyed by all. At the reception, Dianne Blain Williamson, Northern Alabama and Southern Tennessee NAF Ambassador was recognized for her long-time support of NAF and her efforts to raise ataxia awareness in her area. The Coffey Family was recognized for their long-time efforts in raising funds for important ataxia research programs. Kyle Bryant, founder of Ride Ataxia accepted a plaque dedicated to Ride Ataxia II. Kyle gave a presentation on the highlights of this year’s ride experience. All the riders of Ride Ataxia II were recognized with a medal for their participation in the bike ride, which started in Sacramento and ended in Las Vegas at the membership meeting. Ride Ataxia II raised over $120,000 for ataxia research that will go towards a Friedreich’s ataxia research grant in Kyle Bryant’s honor. NAF and FARA will again partner this year to add funds to increase the amount for this important research award.

Saturday started the General Session program with many new and familiar medical professionals and researchers. Huda Zoghbi, MD (Baylor University) started out the General Sessions by discussing the translational studies being done in spinocerebellar ataxia. Christopher Gomez, MD, PhD (University of Chicago) talked about...
Biomarkers in ataxia. Helene Puccio, MD, PhD (INSERM) reported on what we can learn from Animal Models to further Friedreich’s ataxia research. Laura Ranum, PhD (University of Minnesota) presented a general overview of ataxia followed by George Wilmot, MD, PhD (University of Michigan) who discussed the importance of ataxia patient registries.

Saturday afternoon was busy with more General Session presentations. Nicholas Wood, PhD, FRCP, FMedSci (University of London) presented on SCA11 followed by S.H. Subramony, MD (University of Texas) who presented on Sporadic Ataxia. Jeremy Schmahmann, MD (Boston University) discussed cognition, emotion, and the cerebellum. Harry Orr, PhD (University of Minnesota Medical School) gave a review of all ataxia research. Michael Fahey, MD (Australia) informed us about “what’s up down under.”

Saturday’s banquet was a most enjoyable experience for all who attended. The 50/50 raffle fundraiser was great fun with over 80% of conference attendees participating to raise more than $3,700. Congratulations to the winners of the raffle. Thank you to everyone who donated items for the silent auction and to those that participated in this event which raised over $4,500. At the banquet, Dr. John Day gave us a review of the 2008 Ataxia Investigator’s Meeting (AIM) which was held earlier in the week. Thank you to Dr. Day and all the 2008 AIM organizers for their efforts. Michael Parent, NAF’s Executive Director, presented plaques to Dr. John Day for his organization of the 2008 AIM meeting, his years of involvement in ataxia research and service to the ataxia community, to Dr. Susan Perlman for her years of support to NAF, the ataxia community, and ataxia research, and to Dr. Larry Schut for his lifelong contributions to NAF, the ataxia community, and ataxia research.

On Sunday morning the General Session program continued with Susan Perlman, MD (UCLA) giving an update on FRDA clinical trials. Arnulf Koeppen, MD (Virginia Medical Center, Albany, NY) then presented information on brain tissue repair in hereditary ataxia followed by Broyna Keats, PhD (Louisiana State University Health Sciences Center) who discussed RNAi Research. Armin Alaedini, PhD (Cornell University) talked about gluten sensitivity and the immune response in ataxia. Lawrence Schut, MD (CentraCare Clinic) reviewed what medication can be considered in ataxia. Dr. John Day (University of Minnesota) gave the closing presentation of the conference with his “Top 10” review of what we have learned throughout the weekend.

Each day’s General Session was followed by a question-and-answer session facilitated by Dr. Ranum, Dr. Orr, and Dr. Day which included the presenters of each day. Please watch in future issues of Generations, where presentations will be published so all can learn the important information that was presented. Copies of most of the presentations are available on the NAF website: www.ataxia.org.

This was an excellent and exciting meeting! Thank you again to the outstanding job done by the Arizona Ataxia Support Group!
Here is what was said by meeting attendees...

Quotes and Top Three Lists from the 2008 NAF Annual Membership Meeting

“Las Vegas is a good location for the meeting as there are a lot of things to do after the meeting.”
“I commend NAF on all the hard work, the excellent organization and commitment to a quality experience for everyone.”
“Very informative, presenters were very knowledgeable and knew their subjects, all were good presentations.”
“As always, everyone very helpful and friendly.”
“I don’t know how things could be better – food, staff, transportation – all very good.”
“The speakers are amazing – so dedicated to research.”
“Very well organized. Thank you!”
“NAF did a great job, this was very large.”
“Very helpful – All questions were addressed and answered and helped me understand about the disease process and hope for treatment by the intense research by researchers all over the world.”

On Birds of a Feather
“Was great. Helpful.”
“The Birds of a Feather is one of the most important parts of the conference.”
“It definitely served its purpose of providing those with similar diagnoses to discuss a wide range of topics from how to deal with depression to the latest research.”
“Favorite part of meeting each year.”
“Good to see old friends again.”
“Information discussed very helpful, informative and exciting to have such a support group that was so comfortable and intimate.

Top Three Lists

1) Birds of a Feather
2) Susan Perlman and the other excellent speakers
3) Fabulous socialization opportunities

1) Ride Ataxia II
2) Dr. Perlman
3) Financial Planning

1) Investigator Collaboration
2) General Sessions
3) Meeting old & new friends

1) Breakouts
2) Review at end
3) Question & Answer

1) Having so many outstanding doctors in this field
2) Excellent organization
3) Great staff who were always helpful

1) Networking
2) Research Updates
3) Breakout Session Topics

1) Speakers
2) Organization
3) Accessibility of restrooms

1) Birds of a Feather
2) Dinner
3) Dancing with Ataxia

1) The General Sessions, especially those covering research
2) Friday night reception – good opportunity to meet people
3) “Bookstore” – provided an opportunity to purchase helpful info

1) Ability to interact with doctors informally
2) Sessions from doctors
3) Social Network

1) Hearing from researchers
2) Breakout Groups
3) Meeting Others

1) All the speakers
2) The way everything was organized
3) The banquet

1) Breakout Sessions
2) Networking/Meeting people/Socializing with my support group members
3) General Sessions/Meeting doctors we read about in Generations
The NAF Board of Directors along with the Seattle Ataxia Support Group and the Ataxia Society of Vancouver, Canada would like to invite you to attend the

**National Ataxia Foundation**

**52nd Annual Membership Meeting**

**March 20-22, 2009**
(Leadership Meeting March 19)

**Join us in Seattle for the Annual Membership Meeting!**

The **Doubletree Hotel – Seattle Airport** is pleased to provide the facilities for the 2009 NAF Annual Meeting.

Rooms are available for the **special group rate** of $139 per night.

Please be sure to make your reservations by **February 16, 2009** in order to secure the special group rate. If rooms are available, the special rate will be extended three days before and three days after the meeting dates.

To book your stay, call toll-free **1-800-222-TREE** and ask for the **National Ataxia Foundation conference special rate (Group Code: NAF)**.

There are a limited number of ADA rooms available on a first-come, first-serve basis. To reserve an ADA room, you must contact the National Ataxia Foundation at **(763) 553-0020**. NAF will have a limited number of shower chairs, toilet frames and tub bars available on a first-come, first-serve basis at the hotel.

**We look forward to seeing you in Seattle!**
I’m writing this on what would have been my dad’s 63rd birthday. Instead of calling him today to wish him a happy birthday, I will call my mom, to comfort her in his absence. But when I think about it, he’s not really absent. There are pictures scattered around my house and there are plenty of memories. I know the photos will age and some will inevitably be replaced, and the memories will fade or fragment, requiring reconstruction with bits of imagination. But the lessons he taught, almost entirely through quiet example, will accompany me for the rest of my life. This is a story of some of those lessons.

My dad was diagnosed with a form of sporadic ataxia two months after my husband and I moved 2,000 miles away from our families to start our new lives in Northern California. This began seven-and-one-half years of torturous uncertainty, sleepless nights and frequent trans-American flights, but it also offered a priceless opportunity; the opportunity to get to know – and truly appreciate - my dad.

I call the first few years of his illness the “re-learning my dad” phase. I’d known him to be a fantastic athlete, a hopeless workaholic, a borderline neat-freak and a quick-witted practical jokester. But having essentially moved away from home at the age of 18, I had not the chance to observe and appreciate all of the qualities that only post-teenage lenses can see and process. I struggled at first to redefine our relationship, which had largely been based on physical competitions of some sort. The man who’d been the quarterback in high school, pitcher in college and semi-pro ballplayer, accomplished runner (who I couldn’t beat until graduate school), and my frequent coach was now stumbling and needing assistance with basic physical tasks. It was my husband that pointed out the flaw in my attitude. I was attaching so much importance to these trivial physical accomplishments and failing to see the true greatness of my dad … his character.

Describing his character in some ways is difficult. He was simple when it came to his own needs – a minimal wardrobe, a reliable car, a dog on his lap, a bike (or two, or actually four; maybe not so simple there) and an endless supply of chocolate chip cookies. But there was so much more than met the eye. We’d seen examples, and directly benefited, from his generosity and selflessness throughout our lives, but I’m not sure any of us knew the strength of his character until his illness, especially in the final stages.

I was devastated at first when my sister called and told me that he was writing his own memorial service. Was he doing it because he had a lot to say, but was frequently unheard due to the dysarthria that affected his speech? Did he not trust us to do it like he wanted, or did he not want us to have to do it? It could have been any or all of these, but I believe his primary reason was because he didn’t want it to be about him. He’d always been uncomfortable being the center of attention. Regardless of his reasons, what transpired was truly unbelievable and what I refer to as his “final act of selflessness” … that being, he made us laugh … hard!

When I attempt to describe my dad’s memorial service to friends, I tell them it often had the feel of a packed comedy club, in which no one close to my dad was safe. The common theme of “food” ran throughout, and he took special care to thank everyone who’d ever fed him, with special gratitude paid to those who’d designed and orchestrated covert missions to smuggle junk food disguised as vegetable casseroles past my health-obsessed mother. He also took the
opportunity to get the last word in on all sports-related rivalries (Roll Tide!) and then “outed” or made fun of friends and family, in the harmless way that only he could pull off.

Although our family had not been privy to his writings prior to the memorial service, we conspired to produce our own script, summarizing his character in a “Words from the Family” piece, written largely by my sister and read by the two of us at the memorial service. We characterized him as an Educator, Sportsman, Comedian, Non-judgmental/Unselfish Soul, Philosopher, Glutton and Role Model. I’ve selected a few of my favorite parts of this document to share:

**Educator** – “Throughout his life, my father loved teaching, mentoring and educating... wanting little acknowledgement... simply deriving enjoyment from watching others learn and accomplish their goals. His role as an educator lives on, not only in the hearts of those who knew him, but directly through those physicians who will study his brain (donated to Vanderbilt Medical Hospital) in efforts to understand his disease.”

**Sportsman** – “He had a keen love of competition; however, he was equally supportive and encouraging. At least once he wagered a bet at a running race with his youngest sister (the prize, no surprise, being a steak). He had the win all but wrapped up, only to sacrifice it in the end for a ‘tie’ – having already bought the steaks the night before.”

**Comedian** – “Whether appropriate or inappropriate, my father always had a joke. My mother relates that his witty sense of humor was one of the characteristics that first attracted her to him and that remained alive, even in his final days. He never seemed to take himself, or life, too seriously no matter what challenges they encountered.”

**Non-judgmental/Unselfish Soul** – “He was a very unselfish person, who rarely judged others. From a young age, he naturally seemed to think of others before himself, preferring to give rather than receive praise and attention.”

**Philosopher** – “When asked how he maintained such a positive attitude (in light of his disease), he would simply reply, ‘I don’t worry about the things I can’t do, I just focus on the things I can’.”

**Glutton** – “I’ve already alluded to this one, but I can’t resist telling a funny story. Less than 12 hours before my dad died, my sister walked into his room eating a cookie. He lifted his eyebrows, indicating he wanted a bite. As he had difficulty swallowing, it sat on his tongue until we decided it would be best to remove it with a spoon. However, each time my sister tried to retrieve the cookie, he clenched his teeth tightly in protest. We finally gave up and let him enjoy his last cookie.”

**Role Model** – “He showed us how to live, but just as importantly, he showed us how to die – with courage, integrity, humor, and the amazing ability to comfort us, as much, if not more, than we comforted him.”

This was my dad; these were and are his lessons. I feel terribly unlucky to have lost him so prematurely, just as I was re-learning him, but I feel so fortunate to have had him at all.

_Editor’s note: The National Ataxia Foundation is grateful to Kelly McDonald for her contributions to ataxia research._

I race for Touchstone Elite Women’s Cycling Team, based in Berkeley, CA, although our seven members are spread out over all of Northern California. My team was a huge support for

*Continued on page 31*
Safety at Home: Preventing Falls

Falls are serious at any age, but especially for older people who are more likely to break a bone when they fall. Falls are the most common cause of nursing home placement. The risk of falling increases with age.

Falls and accidents seldom “just happen.” Regular exercise as well as regular eye and physical exams may help reduce the risk of falling. Getting rid of tripping hazards in the home and wearing nonskid shoes may also help. Getting enough calcium and vitamin D reduces the chances of breaking a bone if a fall does occur.

If the person in your care has osteoporosis, they are more likely to break a bone if they fall. Osteoporosis is called the “silent disease” because bones become weak with no symptoms. People often find out they have it when a strain, bump, fall, or even a cough causes a bone to break. Get the person in your care tested.

Why Do People Fall?

Some of the reasons people fall are:

- Tripping or slipping due to loss of footing or traction
- Slow reflexes, which make it hard to balance or move out of the way of a hazard
- Balance problems
- Reduced muscle strength
- Poor vision
- Illness

The more medications a person is taking, the higher the chances of falling. Certain medicines increase the risk:

- Blood pressure pills
- Heart medicines
- Diuretics (water pills)
- Muscle relaxants
- Sleeping pills

Drinking alcohol also increases risk because it can:

- Slow reflexes
- Cause dizziness or sleepiness
- Alter balance
- Cause a person to take risks that can lead to falls

Preventing Falls

Here are some ideas for reducing the risk of falls:

Outdoors

- Use a cane or walker
- Wear rubber-soled shoes that don’t slip
- Walk on grass when sidewalks are slick
- Put salt or kitty litter on icy sidewalks

Indoors

- Keep rooms free of clutter, especially on floors and in hallways
- Use plastic or carpet runners
- Wear low-heeled shoes
- Do not walk in socks, stockings, or slippers
- Be sure rugs have skid-proof backs
- Be sure stairs are well lit and have rails on both sides
- Put grab bars on bathroom walls near tub, shower, and toilet
- Use a nonskid bath mat in the shower or tub
- Keep a flashlight next to the bed
- Add more lights in rooms
- Buy a cordless phone so that no one has to rush to answer the phone and so they can call for help if they fall

Preventing Broken Bones if a Fall Occurs

Falling forwards (on your hands) or backwards (on your buttocks) rather than the side can prevent a hip fracture. Falls are responsible for
90 percent of broken hips. Some people wear extra clothes to pad their hips or use special hip pads.

**Exercises to Improve Balance**

- While holding the back of a sturdy chair, sink, or counter:
  - Stand on one leg at a time for a minute and then slowly increase the time. Try to balance with your eyes closed or without holding on.
  - Stand on your toes for a count of 10, and then rock back on your heels for a count of 10.
  - Make a big circle to the left with your hips, and then to the right. Do not move your shoulders or feet. Repeat five times.

*Source: National Institute of Health*

**Assistance Dogs**

Assistance dogs can make life easier for some disabled people. Service dogs help with physical tasks such as pulling wheelchairs, opening doors and offering payment at cash registers. Hearing dogs provide their hard-of-hearing partners with greater independence and security by alerting them to sounds such as sirens and doorbells. Skilled companion dogs work as part of a three-part team with an able-bodied adult, offering a sense of security to people with physical or developmental disabilities such as autism and early-onset Alzheimer’s.

Assistance dogs are chosen for temperament when they are puppies; they then learn basic house training, obedience commands, and socialization skills.

Upon passing the initial screening, the young dogs begin training with professional instructors for six to nine months.

Dogs that complete the training are matched with recipients, who spend two weeks training one-on-one with the dogs at a training facility. Disabled recipients are not charged for their dogs.

For more about assistance dogs, contact Canine Companions for Independence at [www.cci.org](http://www.cci.org) or call 1-800-572-BARK (1-800-572-2275).

---

**Tissue Donation**

If you are interested in helping ataxia research by donation of tissue after death, please contact Dr. Arnulf Koeppen for information.

**Arnulf Koeppen, MD**
Professor of Neurology
VA Medical Center
113 Holland Ave., Albany, NY 12208
Phone: 518.626.6373
Fax: 518.626.6369
E-mail: Arnulf.Koeppen@med.va.gov

---

**Wanted: Photos**

The summer issue of Generations will feature a photo spread of the National Ataxia Foundation 2008 Annual Membership meeting. Send us your photos and they may be published!

Photos should represent a cross section of the meeting and be of interest to all Generations readers. Please burn photos to a CD and mail it to the NAF office, Attn: Generations Editor. Thanks for your help.
Chesapeake Chapter
By Carl J. Lauter

It couldn’t have been a more perfect day – a bright and sunny 45 degrees – to celebrate the 25th Anniversary of the Chesapeake Chapter’s Annual Medical Meetings and to announce the inauguration of the Ataxia Center at Johns Hopkins Medical Institute in Baltimore, MD on February 16 at the Montgomery College in Rockville (see complete story on page 11).

Carl Lauter, President of CC-NAF, welcomed and thanked the 100 people in attendance which included the administrators of Montgomery College, speakers, Johns Hopkins administrators and clinical and research scientists, Macklin family members, JHU Macklin Fellows, and the CC-NAF members, friends, and first time attendees. Carl then presented the financial activities of the CC-NAF for 2007: $35,133 contributed to NAF for several ataxia research funds and $2,000 for administrative and operational functions, plus the release of $18,668 from an accumulating CC-NAF research fund and a $50,000 gift of Gordon Macklin for helping to sponsor the 2008 NAF Ataxia Investigator’s Meeting in March 2008.

Carl then stated, “It is with honor and great pleasure that I can announce, on behalf of the CC-NAF, the establishment of the Ataxia Center at Johns Hopkins University in Baltimore, Maryland.” He then introduced Libby Labash, Vice-President of CC-NAF and Mistress of Ceremonies for the day.

Libby welcomed everybody and proceeded to describe how the Ataxia Center came to be. It had been a long vision of Libby and Norman Labash that such a facility could exist nearby where ataxia patients could be seen and treated efficiently. Through a patient process of waiting and talking and discovering the Macklin family, the dream has come to fruition. Libby introduced Donald Dawn, the nephew of Gordon Macklin and President of the Gordon and Marilyn Macklin Foundation (which is funding the initial phase of the Center), and members of the Macklin family: Stan and Peg Macklin, Lisa and Jim Stargel, Nancy and Dan Hook, and Gary and Linda Macklin, without whom we would not be celebrating this occasion.

Libby then introduced Justin McArthur, MBBS, MPH, Director of the Johns Hopkins Neurology Department, who gave complimentary remarks on the creation of the Ataxia Center. He, among other administrators of JHU, were very understanding and cooperative with the negotiations involved in the creation of the Center, for which we are very thankful.

Sarah H. Ying, MD, Clinical Director of the Ataxia Center, presented a short review of how the center will work explaining the basic structure which they describe as a “CORE” for Clinical, Outreach, Research, and Education. Several of the doctors, connected with the clinical aspects, gave brief presentations of their specialties. Joseph M. Savitt, MD, PhD, Assistant Clinical Director, talked about the importance of education for building a future for ataxia and training of new students in research. David Solomon, MD, PhD, Assistant Professor of Neurology and Otolaryngology, indicated his topic would be genetic counseling and testing. Paul Dash, MD, Clinical Associate, Division of Cognitive Neuroscience, would be involved with cognitive testing. Zoltan Mari, MD, Assistant Professor of Neurology, discussed
the functions of an interdisciplinary team and the technique of deep brain stimulation. Howard S. Ying, MD, PhD, Assistant Professor of Ophthalmology, said his interest would be eye evaluation in the ataxia patient. Laura Marsh, MD, Associate Professor of Psychiatry, remarked on her specialty of psychiatry and the neurological patient and Jenn Millar, MSPT, discussed the importance of rehabilitative therapy for the ataxic patient. She also mentioned a colleague, Jennifer Keller, PT, at adjoining Kennedy-Krieger Institute, also performing physical therapy for patients.

Snacks were graciously provided by local merchants and we returned to hearing Dr. Sarah Ying describe briefly the basic research and education structure of the Clinic, and introduce the research team: Aaron Wong, a PhD candidate in Biomedical Engineering, who brilliantly discussed his work on “Predictions and the Cerebellum.” Jerry Prince, PhD, Professor of Electrical and Computer Engineering, then presented his work with “Imaging the Cerebellum.”

An Ataxia Awareness Campaign was held on the Johns Hopkins campus earlier in the year where questions were posted throughout the campus and the undergrads and graduate students were to respond to them. It went so well and they are so interested and cooperative with CC-NAF, that they may do this activity for IAAD this year. Also, graduate students in Neurology were asked to write a grant proposal telling why they would want to attend the CC-NAF Meeting and what they would expect to learn from it. The winners would be titled “Macklin Fellows.” Ten winners were presented with certificates from JHU and the CC-NAF presented them with a copy of Dr. Susan Perlman’s book: “Evaluation and Management of Ataxic Disorders – An Overview for Physicians.” Letters of congratulations and support were received from the U.S. Senators from Maryland, Benjamin Cardi and Barbara Mikulski, and from NAF President, DeNiece Roach and Michael Parent, NAF’s Executive Director. The Ataxia Center ceremonies ended with a ceremonial ribbon cutting. A wide bright red ribbon strung across the front of the stage, held by members of the Macklin family, JHU and CC-NAF members, was cut by the two Macklin daughters, Lisa and Nancy.

The program continued with a presentation, “The Cerebellum and the Control of Movement,” by Reza Shadmehr, PhD, Associate Professor of Biomedical Engineering, Johns Hopkins.

Libby Labash, having recently lost her faithful and loving husband on December 5, 2007, presented, with broken heart, a loving memorial of Norman, which was followed by touching tributes by Bill Lee, current CC-NAF Secretary and co-founder of CC-NAF, and Dick Sargent, current A-Team member and long-term member.

During lunch break two large birthday cakes were enjoyed celebrating the 25th year anniversary of holding Medical Meetings.

The afternoon lecture titled “Antibodies in Ataxia” by Ejaz Shamim, MD, MS, Clinical Neurology Fellow, Medical Neurology Branch, NINDS, Bethesda, MD summarized some of the work that has been done there for the past several years.

A special attraction of the day was the appearance of Bill Nye, The Science Guy, whose family tree is filled with ataxia members. He gave a joyful talk on “Just carelessness, doorjams, and the Darby glide,” which brought in some of the aspects and problems a family experiences with ataxia. With his enthusiasm, he could well create public awareness of ataxia.

The afternoon finished with 45 minutes of an open forum discussion with questions to the speakers from the attendees. This is one of the more valuable times of the medical meetings, as

Continued on page 34
**Chapter and Support Group News**

**Continued from page 33**

questions can be answered and the medical staff is so pleased to participate. In turn, we of CC-NAF have volunteers to participate in the various research studies being carried out at Johns Hopkins and the NIH.

As a summary, in respect to the generosity of our members and in a very significant way to the philanthropy of the Macklin family, in memory of two members, Gordon and Marilyn Macklin, approximately $49,214 has been given over the last nine years in the form of direct gifts to CC-NAF or from their Challenge Grant for research, in addition to the current $150,000 for the establishment of the Johns Hopkins Ataxia Center, the only Ataxia Center on the East Coast. In addition, the Macklin Family has provided $50,000 toward sponsorship of the AIM meeting held concurrently with the 2008 NAF annual membership meeting.

We are dedicated to finding significant funding for continued research on the causes and potential cures for ataxia and for the betterment of the Ataxia Clinic.

**Los Angeles Ataxia Support Group**

*By Sid Luther*

The Los Angeles Ataxia Support Group meets bi-monthly at the Westside Center for Independent Living (WCIL). We meet on the second Saturday of the month at 12901 Venice Blvd. between the hours of 2 to 4 p.m.

At our last meeting on March 8 we watched the NAF Video “Together there is Understanding.”

We also discussed Section 8 Housing. A few of our members are trying to find affordable housing for disabled.

At our May 10 meeting we are planning to have a speech and swallowing specialist from UCLA Medical Center in attendance. Last November Dr. Joanna Jen from UCLA came and gave us about updates in ataxia research and answered questions. We also had pizza.

Most recently we have taken several group outings with Casa Colina Rehabilitation Center, including kayaking, sailing and whalewatching. Great fun for everyone involved. We also did a trip to the Aquarium of the Pacific followed by a late lunch at a local restaurant. This spring we are planning on going to some concerts in the park. During the month I received several phone calls and e-mails regarding ataxia questions.

**Denver Area Ataxia Support Group**

*By Tom Sathre*

The Denver Ataxia Support Group met at Swedish Hospital on March 8 for its quarterly meeting. We enjoyed a potluck lunch, massages from four trainees at Heritage College, and a presentation by a local non-medical home care agency. The presentation took about two hours and was frequently interrupted by questions, which shows the interest the audience had in the topic! About 20 people were there at 1 p.m., the announced time of the meeting – about half were care-givers and half were ataxians – and more came in later. In the business part of the meeting, notes were taken and the address list was updated. On Sunday, June 8, from 6-8 p.m. in the Tivoli Turnhalle on the Auraria campus in downtown Denver, there will be a NAF fundraiser with a musician, a vocalist and a dance group performing, a silent auction and delicious hors d’oeuvre-sized desserts for sale.
We are looking forward to the NAF Annual Membership Meeting and the upcoming MED life trade show to be held in May at the Long Beach Convention Center and the Abilities Expo in June to be held at the Anaheim Convention Center. For more information visit www.ataxia.org/chapters/LosAngeles/default.aspx or www.geocities.com/HotSprings/Falls/6629.

Northern California Support Group
By Deborah Omicitin

Dr. David Schaffer of the Helen Wills Neuroscience Institute at UC Berkeley was our guest speaker in October 2007. His timely topic was “Human Embryonic Stem Cells: Fact and Fiction.” Our next meeting will be on April 12 at Our Savior’s Lutheran Church at 1035 Carol Lane, Lafayette at 11:30 a.m.

Tampa Bay Area Support Group
By Chris Frohna

We had a good turnout at our Seventh Annual TBASG Picnic on March 8 despite the unusually cool and windy weather. Craig Baker, our Vice President, was our chef, cooking burgers and chicken, and everyone brought a dish to share. The picnic was held at Phillippe Park in Safety Harbor.

After lunch we were privileged to have Professor Jeannie Stephenson, PT, MS, NCS of the USF School of Physical Therapy and Rehabilitation Sciences give us her input on what physical therapy can do for us as ataxia patients in helping us with strengthening our bodies and loosening tight muscles. One of her students was also present to help Professor Stephenson demonstrate some of the helpful stretching exercises. She also answered questions from the group. Professor Stephenson has an ataxia clinic as part of her curriculum each year that the group participates in and USF has a full-time Physical Therapy Clinic.

The rest of the afternoon we were treated to a session with Dr. Tom Clouse of “Dancing with Ataxia” fame. Dr. Clouse went into great detail explaining that part of our problem with walking as ataxians is our mind set and our posture. He had several volunteers come up to re-learn how to walk and in a short time two of them made remarkable progress. I even joined in the fun and found I have a lot of work to do! Dr. Clouse explained we need to re-center ourselves at our “belt” and walk from our knees and stop looking at our feet, because if we lift our knees, of course, the feet naturally follow. Afterwards we were privileged to have Dr. Clouse and his partner dance for us and were very impressed! It was such a thrill to have Dr. Clouse there with us! We’re hoping to have him back another time for an indoor and hopefully warmer session.

Thanks to all the brave group members who shivered along with me. It’s not supposed to be cold and windy in Tampa in March!

Utah Support Group
By Julia Kleinschmidt

The February 27 meeting, with speech pathologists presenting “Ataxia and Speech Therapy: Can It Help and How?” was very informative. On May 7 many in the group will attend the Utah Opera's production of Don Giovanni. Each year since 1998, the Community Education

Continued on page 36
Chapter and Support Group News

Continued from page 35

Director of the opera company has arranged for special accommodations for people with visual impairment and other special needs. So our group enjoys a pre-opera talk in which, in addition to information on the production, props are brought down from the stage to be enjoyed close up. During the production, those with visual impairment wear headphones in which the stage action is described. It’s a wonderful evening.

On May 14, our guest speaker will be Dr. Stefan Pulst, Chair of the University of Utah’s Neurology Department and SCA researcher.

..........................................................

Tri-State Ataxia Support Group

By Denise Mitchell

On April 2, the Tri-State Ataxia Support Group was formed at Beth Israel Hospital in New York City. There was a great turnout and everyone enjoyed getting to know each other. The group will meet the first Wednesday of each month. Mark and Denise Mitchell are going to be the leaders of the group.

The next meeting is May 7 at 6:30 p.m. in the Friedman Conference Center (Room 1) on the second floor of the Phillips Ambulatory Care Center (PACC) 10 Union Square East.

Please RSVP to Jeannie Soto-Valencia at (212) 844-8711. Desserts will be served.

..........................................................

News & Notes

Your E-mail Address and Address Changes Are Requested

Help NAF keep its records current and complete and reduce postage expenses. Your e-mail address is especially important to update NAF’s current database.

Please e-mail your name, mailing address, phone number and e-mail address to Julie Braun at julie@ataxia.org at your earliest convenience.

Combined Federal Campaign Number

The National Ataxia Foundation’s Combined Federal Campaign (CFC) number is 10752. This program provides a convenient way to donate to the Foundation and provides great benefit to those with ataxia.

Please give as generously as you can and please ask your co-workers to also give to the National Ataxia Foundation.

Using GoodSearch and iGive?

Are you using the two great websites that support the National Ataxia Foundation?

Use GoodSearch.com and iGive.com and each time you search or make a purchase, NAF receives a donation. Please bookmark these sites today!

Correction

The photo caption on page 34 of the Winter 2007-08 issue of Generations incorrectly identified June Wood as Jane Wood. Our apologies to June.
NAF Merchandise

**BOOKS**

**Ten Years to Live** by Henry Schut
The story of the Schut family’s struggle with hereditary ataxia and the impact it had on this extended family. Paperback, photos. $8.75

**Living with Ataxia** by Martha Nance, MD
A compassionate, easy to understand explanation and ideas on how to live with ataxia. Paperback. $14

**Healing Wounded Doctor-Patient Relationships** by Linda Hanner and contributor John J. Witek, MD
Offers demonstrations of how effective dialog can help move patients and doctors to productive relationships. Paperback. $10

**Friedreich’s Ataxia Research Cookbook**
Julie Karjalahti of Savage, Minnesota has published this cookbook to raise money for FA research. Includes recipes from around the U.S. $12

**Recipes and Recollections** by Kathryn Hoefer Smith
Full of delicious recipes and recollections, this book is perfect for fund raisers. Proceeds go towards FA research. Paperback. $10

**Managing Speech & Swallowing Problems** by G.N. Rangamani, PdD, CCC-SLP
A basic guide to understanding and managing speech and/or swallowing problems. $7.50

**Evaluation and Management of Ataxic Disorders, an Overview for Physicians** by Susan L. Perlman, MD
A guide for physicians treating ataxia patients. Paperback. $5

**VIDEO / CD**

**Ballads of a Family Man CD**
10 songs in memory of Billa Ballard. $5 of purchase price goes to support the work of the NAF. $13

**“Together there is Understanding” VHS or DVD**
Continuation and expansion of “Together There is Hope.” 50-minute in-depth look at ataxia and ataxia research. VHS $20 or DVD $25

**SHIRTS / MISCELLANEOUS**

**2008 Annual Membership Meeting T-Shirt**
Dark blue with “Blazing a Trail in Research” logo. Various sizes. $10

**NAF Shoulder Bag**
Blue with white NAF logo. 11x15x2 inches. $10

**NAF Polo Shirt**
Royal blue w/ white embroidered NAF logo. $27.50

**NAF Denim Shirt**
Denim with white embroidered NAF logo. $27.50

**“Ataxia is not a foreign cab” T-Shirt**
White. New design. Sizes small to XXX-large. $10

**“Ataxia is not a foreign cab” Sweatshirt**
Ash colored. Sizes small to XXX-large. $20

**NAF Baseball Caps**
White w/ blue embroidered NAF logo or blue w/ white embroidered logo. Velcro strap for sizing. $10

**Window Cling or Bumper Sticker**
$1 each or 6 for $5

**NAF Ataxia Awareness Band**
Blue. One size fits all. $2

**NAF Ataxia Awareness Ribbon Magnet**
Blue with white lettering/logo. $4

To order, call (763) 553-0020, fax (763) 553-0167 or mail this completed form to National Ataxia Foundation, 2600 Fernbrook Lane, Suite 119, Minneapolis, MN 55447

<table>
<thead>
<tr>
<th>Description</th>
<th>Qty.</th>
<th>Size</th>
<th>Each</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**SUBTOTAL:**

**Shipping:** (Add) $5.00

(Outside U.S. add additional $4) 

**ORDER TOTAL:**

**PLEASE ALLOW 4-6 WEEKS FOR DELIVERY**

NAME: ________________________________
ADDRESS: ________________________________
CITY ___________________ STATE: ____ ZIP: _______
PHONE: ________________________________

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

CIRCLE ONE: Visa Mastercard
NAME ON CARD: ________________________________
CARD #: ________________________________
EXP DATE: ________________________________
SIGNATURE: ________________________________
Chapters, Support Groups and Ambassadors

The following is a list of National Ataxia Foundation chapters, support groups and ambassadors. The use of these names, addresses and phone numbers for any purpose other than requesting information regarding NAF or joining a chapter or support group is strictly prohibited. We encourage you to contact the chapter or group nearest you.

Chapters

Chesapeake Chapter
Carl J. Lauter, President
3200 Baker Circle, I-117
Adamstown, MD 21710-9666
(301) 644-1836
E-mail: carljlauter@erols.com
Web: www.geocities.com/HotSprings/Oasis/4988/
www.ataxia.org/chapters/Chesapeake/default.aspx

Louisiana Chapter
Carla Hagler, President
PMB 51056
2250 Gause Blvd.
Slidell, LA 70461
(985) 643-0783
E-mail: ataxia1@earthlink.net
Web: www.angelfire.com/la/ataxichapter
www.ataxia.org/chapters/Louisiana/default.aspx

Mississippi Chapter
Camille Daglio, President
P.O. Box 17005
Hattiesburg, MS 39404
E-mail: daglio1@bellsouth.net
www.ataxia.org/chapters/Mississippi/default.aspx

Support Groups

Alabama
BIRMINGHAM S.G.
Becky Donnelly
16 The Oaks Circle
Hoover, AL 35244
(205) 987-2883
E-mail: donnellyb6732@aol.com
www.ataxia.org/chapters/Birmingham/default.aspx

Arizona
PHOENIX AREA S.G.
Rita Garcia
2322 W. Sagebrush Dr.
Chandler, AZ 85224-2155
(480) 726-3579
E-mail: rtg22@cox.net
www.ataxia.org/chapters/Phoenix/default.aspx

California
LOS ANGELES ATAXIA S.G.
Sid Luther
339 W. Palmer, Apt. A
Glendale, CA 91204
(818) 246-5758
E-mail: harryluther@sbcglobal.net
Web: www.geocities.com/HotSprings/Falls/6629/
www.ataxia.org/chapters/LosAngeles/default.aspx

Jim Fritz
(310) 397-5208
E-mail: ondefritz@aol.com

Northern California S.G.
Deborah Omictin
26840 Edridge Ave.
Hayward, CA 94544
(510) 783-3190
E-mail: rsisbig@aol.com
Web: www.geocities.com/casupport/
www.ataxia.org/chapters/NorthernCalifornia/default.aspx

Orange County S.G.
Daniel Navar
829 W. Gary Ave.
Montebello, CA 90640
(323) 788-771
E-mail: dnavar@ucla.edu
Web: www.geocities.com/ocasgg/
www.ataxia.org/chapters/OrangeCounty/default.aspx

San Diego S.G.
Earl McLaughlin
2087 Granite Hills Dr.
El Cajon, CA 92019
(619) 447-3753
S.G. e-mail: sdasg@cox.net
Earl’s e-mail: emclaugh@cox.net
Web: www.geocities.com/ataxia_sdasg
www.ataxia.org/chapters/SanDiego/default.aspx
Colorado
DENVER AREA ATAXIA S.G.
Donna & Tom Sathre
5902 W. Maplewood Dr.
Littleton, CO 80123
(303) 794-6351  Fax: (801) 640-8602
E-mail: tom_sathre@acm.org
www.ataxia.org/chapters/Denver/default.aspx

Connecticut
See Tri-State Ataxia S.G. under New York

Florida
NORTHEAST FLORIDA S.G.
June McGrane
54 Troon Terrace
Ponte Vedra, FL 32082-3321
(904) 273-4644
E-mail: jmcranepvb@bellsouth.net
www.ataxia.org/chapters/NortheastFlorida/default.aspx

ORLANDO ATAXIA S.G.
Jim Ellson
E-mail: jellson@juno.com or jellson@comcast.net
www.ataxia.org/chapters/Orlando/default.aspx

TAMPA BAY S.G.
Crystal (Chris) Frohna
9753 Elm Way
Tampa, FL 33635
(813) 453-1084
E-mail: chrisfrohna@yahoo.com
Web: www.flataxia.org
www.ataxia.org/chapters/TampaBay/default.aspx

Georgia
GREATER ATLANTA AREA S.G.
Greg Rooks
320 Peters St., Unit 12
Atlanta, GA 30313
(404) 822-7451
E-mail: rooksgj@yahoo.com

Dave Zilles
2400 Kimbrough Ct.
Atlanta, GA 30350
(770) 399-6710
E-mail: dzilles@earthlink.net

Lynn Robinette
1971 Sumter Court
Lawrenceville, GA 30044
(770) 982-0275
E-mail: lynn.robinette@comcast.net
www.ataxia.org/chapters/Atlanta/default.aspx

Illinois
GREATER CHICAGO AREA ATAXIA S.G.
Craig Lisack
410 W. Mahogany Ct., Unit 505
Palatine, IL 60067
(847) 496-7544
E-mail: caasg2@aol.com

Richard Carr
120 South Elm
Mount Prospect, IL 60056
(847) 253-2920
E-mail: caasg@comcast.net
www.ataxia.org/chapters/Chicago/default.aspx

METRO AREA CHICAGO ATAXIA S.G.
Christopher Marsh
5633 N. Kenmore, Apt. 059
Chicago, IL 60660
(773) 334-1667
E-mail: cmarsh34@ameritech.net
http://health.groups.yahoo.com/group/u-r-notalone/
www.ataxia.org/chapters/ChrisMarsh/default.aspx

Indiana
LOUISVILLE AREA ATAXIA S.G.
Monica Smith
1102 Ridgedwood Dr. Apt. 4
Huntingburg, IN 47542
(812) 630-4783
E-mail: monicasfaith@insightbb.com
www.ataxia.org/chapters/Louisville/default.aspx

Kansas
KANSAS CITY S.G.
Lois Goodman
729 S. Clark St.
Fort Scot, KS 66701
(620) 223-1996
www.ataxia.org/chapters/KansasCity/default.aspx

Louisiana
See Louisiana Chapter

Maine
MAINE SUPPORT GROUP
Kelley Rollins
P.O. Box 113
Bowdoinham, ME 04008
E-mail: rollins@gwi.net

Monique Godbout
56 King Road
Lisbon, ME 04250
E-mail: mrgodbout@excite.com
Web: www.ataxiaME.com
www.ataxia.org/chapters/Maine/default.aspx

Maryland
HOWARD COUNTY S.G.
Kathy van’t Hoff
(301) 854-2650
E-mail: kakimatt@msn.com

Tim Daly
(410) 715-1241
Chapters, Support Groups and Ambassadors
Continued from page 39

Web: www.geocities.com/hcasg/
www.ataxia.org/chapters/HowardCounty/default.aspx

Massachusetts
NEW ENGLAND S.G.
Donna & Richard Gorzela
45 Juliette St.
Andover, MA 01810
(978) 475-8072
www.ataxia.org/chapters/NewEngland/default.aspx

Minneapolis
TWIN CITIES AREA S.G.
Lenore Healey Schultz
2549 32nd Ave. S.
Minneapolis, MN 55406
(612) 724-3784
E-mail: lschultz@bitstream.net
www.ataxia.org/chapters/TwinCities/default.aspx

Mississippi
See Mississippi Chapter

Missouri
KANSAS CITY S.G.
Jim Clark
6605 N. Holmes
Gladstone, MO 64118
(816) 468-7260
E-mail: clarkstone9348@sbcglobal.net
www.ataxia.org/chapters/KansasCity/default.aspx

MID-MISSOURI ATAXIA S.G.
Roger Cooley
1609 Cocoa Court
Columbia, MO 65202
(573) 474-7232 before noon
www.ataxia.org/chapters/RogerCooley/default.aspx

New Jersey
See Tri-State Ataxia S.G. under New York

New York
CENTRAL NEW YORK ATAXIA S.G.
Linda Johnson
2849 Bingley Rd.
Cazenovia, NY 13035
E-mail: johnsons@summitsolutions.net
www.ataxia.org/chapters/CentralNewYork/default.aspx

TRI-STATE ATAXIA S.G.
Jeannie Soto-Valencia
Beth Israel Dept. of Neurology, Suite 2R
10 Union Square East
New York, NY 10003
(212) 844-8711

Denise Mitchell
E-mail: markmeghan@aol.com
www.ataxia.org/chapters/Tri-State/default.aspx

North Carolina
See South/North Carolina

Ohio
CENTRAL OHIO S.G.
Cecelia Urbanski
7852 Country Court
Mentor, OH 44060
(440) 255-8284
E-mail: iksnbru@earthlink.net
www.ataxia.org/chapters/CentralOhio/default.aspx

Oregon
WILLAMETTE VALLEY ATAXIA S.G.
Malinda Moore, CCC-SLP
Albany General Hospital
1046 Sixth Ave. S.W.
Albany, OR 97321
(541) 812-4162 Fax: (541) 812-4614
E-mail: malindam@samhealth.org
www.ataxia.org/chapters/Willamette/default.aspx

Pennsylvania
SE PENNSYLVANIA S.G.
Liz Nussear
(610) 277-7722
E-mail: lizout@aol.com
www.ataxia.org/chapters/SEPennsylvania/default.aspx

South/North Carolina
CAROLINAS S.G.
Cece Russell
1305 Cely Rd.
Easley, SC 29642
(864) 220-3395
E-mail: cecerussell@hotmail.com
www.ataxia.org/chapters/Carolinas/default.aspx

Texas
GOLDEN TRIANGLE AREA S.G.
Dana LeBlanc
2801 W. Sunset #59H
Orange, TX 77630
(409) 883-5570
E-mail: tilessal@yahoo.com
www.ataxia.org/chapters/GoldenTriangle/default.aspx

HOUSTON AREA S.G.
Angela Cloud
9405 Hwy 6 South
Houston, TX 77083
(281) 693-1826
E-mail: angelahcloud@aol.com
www.ataxia.org/chapters/Houston/default.aspx
NORTH TEXAS S.G.
David Henry Jr.
7 Wentworth Ct.
Trophy Club, TX 76262
E-mail: cheve11e@sbcglobal.net
www.ataxia.org/chapters/NorthTexas/default.aspx

Utah
Dr. Julia Kleinschmidt
Moran Eye Center, U of Utah
50 N. Medical Dr.
Salt Lake City, UT 84132
(801) 585-2213
E-Mail: julia.kleinschmidt@hsc.utah.edu
www.ataxia.org/chapters/Utah/default.aspx

Virginia
See Chesapeake Chapter

Washington
SEATTLE AREA
Milly Lewendon
14104 107th Ave. NE
Kirkland, WA 98037
(425) 823-6239
Milly's e-mail: mmlewendon@comcast.net
S.G. e-mail: ataxiaseattle@comcast.net
www.ataxia.org/chapters/Seattle/default.aspx

Electronic Support Groups
E-NAF (ELECTRONIC NAF) S.G.
Jim Kardos
1283 Westfield SW
North Canton, OH 44720
(330) 499-4060
E-mail: jkardos@juno.com
www.ataxia.org/chapters/E-NAF/default.aspx

International Support Groups
Canada — British Columbia
ATAXIA SOCIETY VANCOUVER
Brenda Dixon
206-8611 Ackroyd Rd.
Richmond, B.C. V6X 3P4
(604) 273-2789
E-mail: bdixon57@telus.net or info@bcataxia.org
Web: www.bcataxia.org
www.ataxia.org/chapters/Vancouver/default.aspx

India
SAMAG (INDIA ATAXIA S.G.)
Chandu Prasad George.CH,
H-No:5-9520, Sri Laxmi Nagar Colony, Old Alwal
Secunderabad, 500 010 India
Phone: 0091-040-27971043
Mobile: 0091-9949019410 Fax: 091-040-27971043
E-mail: sam_ataxiaindia@yahoo.com
E-mail: george@samataxiaigroup.org
www.ataxia.org/chapters/Chandu/default.aspx

Ambassador Listing

Alabama
Dianne Blain Williamson
123 Leigh Ann Rd.
Hazel Green, AL 35750
(256) 828-4858
E-mail: diennebw@aol.com
www.ataxia.org/chapters/DianneWilliamson/default.aspx

Arkansas
Judy and David King
17 Sanchez Point
Hot Springs Village, AR 71909
E-mail: drkingpd@suddenlink.net
www.ataxia.org/chapters/JudyKing/default.aspx

California
Barbara Bynum
3801 W. Bailey
Merced, CA 95340
(209) 383-1275
www.ataxia.org/chapters/BarbaraBynum/default.aspx

Mike Fernandes
7251 Brentwood Blvd. #114
Brentwood, CA 94513
(925) 516-6906
E-mail: fernandesml@comcast.net
www.ataxia.org/chapters/MikeFernandes/default.aspx

Mike Betchel
315 W. Alamos, Apt. 141
Clovis, CA 93612
(559) 281-9188
E-mail: mike_betchel@yahoo.com
www.ataxia.org/chapters/mike/default.aspx

Connecticut
Terre Di Placito
107 Barton St.
Torrington, CT 06790
(860) 489-5092
www.ataxia.org/chapters/TerreDiPlacito/default.aspx

Florida
Christina Sugars
302 Beach Dr.
Destin, FL 30541
(850) 654-2817

Continued on page 42
Chapters, Support Groups and Ambassadors
Continued from page 41

E-mail: csugars@cox.net
www.ataxia.org/chapters/ChristinaSugars/default.aspx

Jim Henderson
3212 Lee Shore Loop
Orlando, FL 32820
(407) 568-9092
E-mail: jamesone24@aol.com
www.ataxia.org/chapters/JimHenderson/default.aspx

Thomas Clouse, MD
1604 Cheshire Circle S.
Lehigh Acres, FL 33936
(239) 989-5150
E-mail: tcdoc@comcast.net
www.ataxia.org/chapters/ThomasClouse/default.aspx

Georgia
Kristie Adams
258 Beaufort Rd.
Savannah, GA 31419
E-mail: opal1011@comcast.net
www.ataxia.org/chapters/KristieAdams/default.aspx

Diana Kimmel
3607 Shepherds Ln
Loganville, GA 30052
E-mail: dk00602@aol.com
www.ataxia.org/chapters/DianaK/default.aspx

Illinois
Elaine Darte
36 Lindorf Dr.
Belleville, IL 62223
(618) 397-3259
www.ataxia.org/chapters/SouthernIllinois/default.aspx

Kevin Donnelli
6525 Thomas Parkway
Rockford, IL 61114
(815) 633-8620
www.ataxia.org/chapters/KevinDonnelli/default.aspx

Indiana
Jenney Roemke
4522 Shenandoah Circle W.
Ft. Wayne, IN 46835
(219) 485-0965
www.ataxia.org/chapters/JenneyRoemke/default.aspx

Iowa
Emily Medina
3720 Patricia Dr. #1
Urbandale, IA 50322
(515) 727-8713
E-mail: 061578@yahoo.com

Kentucky
Janice Johnson

8555 Brownsville Rd.
Brownsville, KY 42210
(270) 597-3854
www.ataxia.org/chapters/JaniceJohnson/default.aspx

Albin Douglas Johnson
10602 Tarrytowne Dr.
Louisville, KY 40272
(502) 751-4585
www.ataxia.org/chapters/AlbinJohnson/default.aspx

Maryland
Karen Rosenberger
6411 Spring Forest Rd.
Frederick, MD 21701
(301) 682-5386
E-mail: kdrosenberger@comcast.net
www.ataxia.org/chapters/KarenRosenberger/default.aspx

Michigan
Lynn K. Ball
35015 Riverview Dr.
Paw Paw, MI 49079
(269) 657-5191
E-mail: lynnkball@aol.com
www.ataxia.org/chapters/LynnBall/default.aspx

Minnesota
Lori Goetzman
5179 Meadow Dr.
Rochester, MN 55904
(507) 282-7127
E-mail: logoetz@gmail.com
www.ataxia.org/chapters/LoriGoetzman/default.aspx

Debbie Kelly
310 Fern St. #7
Big Lake, MN 55309
(763) 263-1812
www.ataxia.org/chapters/DebbieKelly/default.aspx

Julie Schuur
218 Cashin Dr.
Luverne, MN 56156
(507) 283-2555
E-mail: jschuur@iw.net
www.ataxia.org/chapters/JulieSchuur/default.aspx

Missouri
Susan L. Strode, PhD
12 Jackson #811B
Jefferson City, MO 65101
(573) 659-4759
E-mail: dursusie@socket.net
Web: www.dr-susie.com

New York
Valerie Ruggiero
36 West Redoubt Rd.
Fishkill, NY 12524
(845) 897-5632
E-mail: vrabsolutely@aol.com
www.ataxia.org/chapters/ValerieRuggiero/default.aspx

Diane P. Hall
210 E. Utica St.
Buffalo, NY 14208
(716) 881-0677
www.ataxia.org/chapters/DianeHall/default.aspx

Mary Ann Costa
460 Brielle Ave.
Staten Island, NY 10314
(718) 317-3802
www.ataxia.org/chapters/MaryAnn/default.aspx

Ohio
James Kardos
1283 Westfield S.W.
North Canton, OH 44720
(330) 499-4060
E-mail: jkardos@juno.com
www.ataxia.org/chapters/E-NAF/default.aspx

Joe Miller
Box 148
Mesopotamia, OH 44439
(440) 693-4454
E-mail: kakah@windstream.net
www.ataxia.org/chapters/JoeMiller/default.aspx

Oklahoma
Mark Dvorak
915 Thistlewood
Norman, OK 73072
(405) 447-6085
E-mail: czechmarkmh@yahoo.com
www.ataxia.org/chapters/Ambassador/default.aspx

Darrell Owens
5700 S.E. Hazel Rd.
Bartlesville, OK 74006
(918) 331-9530
E-mail: droopydog36@hotmail.com
www.ataxia.org/chapters/DarrellOwens/default.aspx

Texas
Jose Julio Vela
6702 Long Meadow
Corpus Christi, TX 78405
(361) 993-9006
www.ataxia.org/chapters/JoseJulioVela/default.aspx

Barbara Pluta
356 Las Brisas Blvd.,
Seguin, TX 78155-0193
(830) 557-6050
E-mail: acemom65@att.net
www.ataxia.org/chapters/BarbaraPluta/default.aspx

Virginia
Dick Sargent
(703) 321-9143
E-mail: dcksgnt9@aol.com
www.ataxia.org/chapters/DickSargent/default.aspx

Washington
Linda Jacoy
PO Box 19045
Spokane, WA 99217
(509) 482-8501
www.ataxia.org/chapters/Spokane/default.aspx

International Ambassadors

American Samoa
Bob Coulter
P.O. Box 9062
American Samoa 96799
(684) 688-2437

Australia
Renee Moore (Nee McCallum)
44 Lotherton Way
Hocking, W. Australia 6065
61-8-9404-7052
E-mail: moorear@bigpond.com
www.ataxia.org/chapters/ReneeMoore/default.aspx

Canada
Susan M. Duncan
#401-1330 Richmond Rd.
Ottawa, Ontario K2B 8J6
(613) 820-7900
E-mail: smduncan1@sympatico.ca
www.ataxia.org/chapters/SusanDuncan/default.aspx

Cathy Chamberlain
551 Vermilyea Rd.
Belleville, Ontario K8N 4Z5
(613) 962-9623
www.ataxia.org/chapters/CathyCamberlain/default.aspx

Prentis Clairmont
299 Somerset West, Apt. 402
Ottawa, Ontario K2P 2L3
(613) 864-8545
E-mail: prentis.clairmont@gmail.com
www.ataxia.org/chapters/PrentisClairmont/default.aspx

Terry Greenwood
37 Ericsson Bay
Winnipeg, Manitoba R3K 0T8
(204) 885-3955
E-mail: tgreenwood6@shaw.ca
www.ataxia.org/chapters/TerryGreenwood/default.aspx

India
Abhinav Kedia
A9/7A Gomti Apartments, Kalkaji Extension
New Delhi-19
Phone: 0091-011-29960809/29962759/41861809
Mobile: 0091-9466355238
E-mail: abhinav_kedia_2000@yahoo.com
www.ataxia.org/chapters/AbhinavKedia/default.aspx

International Ambassadors
Calendar of Events

Wednesday, May 7, 2008
Tri-State Ataxia Support Group Meeting
6:30 p.m. at the Friedman Conference Center (Conference Room One) second floor of Phillips Ambulatory Care Center (PACC), 10 Union Square East, New York, NY. Desserts will be served. RSVP to Jeannie Soto-Valencia (212) 844-8711.

Saturday, May 10, 2008
Kansas City Area Ataxia Support Group
2-4 p.m. at the Northeast Library, 65 Wilson Ave, Kansas City, MO. Contact Lois Goodman (620) 223-1996 or Jim Clark at clarkstone9348@sbcglobal.net. www.ataxia.org/chapters/KansasCity/default.aspx.

Los Angeles Area Ataxia Support Group
2-4 p.m. The Westside Center for Independent Living, 12901 Venice Beach, CA. Contact Sid Luther for more information (818) 246-5758. www.ataxia.org/chapters/LosAngeles/default.aspx.

North Texas Ataxia Support Group Meeting
10 a.m.-noon at Los Colinas Medical Center, 6800 Mac Arthur Blvd at Hwy 161, Irving, TX. David Henry, Jr. www.ataxia.org/chapters/NorthTexas/default.aspx.

SE Pennsylvania Ataxia Support Group Meeting
10-11:30 a.m. at Mercy Suburban Hospital, 2701 Dekalb Pike Norristown, PA. Contact Liz Nussear (610) 277-7722 or lizout@aol.com. www.ataxia.org/chapters/SEPennsylvania/default.aspx.

Tampa Bay Ataxia Support Group Meeting
1-3 p.m. at Feathersound Community Church, Clearwater, FL. Contact Chris Frohna at (813) 453-1084 or chrisfrohna@yahoo.com. www.ataxia.org/chapters/TampaBay/default.aspx.

Sunday, May 11, 2008
Seattle Area Ataxia Support Group Meeting
2-4 p.m. at Madison House Retirement Community, 12215 NE 128th St, Kirkland, WA. Speaker Dr. Kristie Spencer, Professor, Speech-Language Pathologist at University of Washington. Contact Milly Lewendon at mllewendon@comcast.net. www.ataxia.org/chapters/Seattle/default.aspx.

Wednesday, May 14, 2008
Utah Ataxia Support Group
7 p.m. at the University of Utah John A. Moran Eye Center. Special Guest speaker Dr. Stefan Pulst, Chairman of the Dept. of Neurology, Univ. of Utah School of Medicine. Contact Julia Kleinschmidt at julia.kleinschmidt@hsc.utah.edu for more information. www.ataxia.org/chapters/Utah/default.aspx.

Saturday, May 17, 2008
Greater Atlanta Ataxia Support Group Meeting
1 p.m. at Emory Center for Rehabilitation Medicine, 1441 Clifton Rd, Room 101, Atlanta, GA. www.ataxia.org/chapters/Atlanta/default.aspx.

Orange County Ataxia Support Group Meeting
1:30-4 p.m. Orange Coast Memorial Medical Center (in the basement, next to the cafeteria), 9920 Talbert Ave., Fountain Valley, CA. Contact Daniel Navar at dnavar@ucla.edu. www.ataxia.org/chapters/OrangeCounty/default.aspx.

Monday, May 19, 2008
Mid-Missouri Ataxia Support Group Meeting
10 a.m. at the Daniel Boone Regional Library, Columbus, MO. Contact Roger Cooley at (573) 474-7232 before noon. www.ataxia.org/chapters/RogerCooley/default.aspx.

Tuesday, May 20, 2008
Twin Cities Ataxia Support Group
7 p.m. at Roseville Presbyterian Home (located off 35W on County Rd D). Contact Lenore H. Schultz at (612) 724-3784 or lshultz@bitstream.net. www.ataxia.org/chapters/RogerCooley/default.aspx.

Saturday, June 14, 2008
Denver Area Ataxia Support Group Meeting
1-4 p.m. at the Swedish Hospital & Medical Conference Center (Room Spruce A), 501 East Hampden Ave., Englewood, CO. Contact Tom Sathre at tom_sathre@acm.org or (303) 794-6351. www.ataxia.org/chapters/Denver/default.aspx.

Kansas City Area Ataxia Support Group
2-4 p.m. at the Northeast Library, 65 Wilson Ave., Kansas City, MO. Contact Lois Goodman

The deadline for the Summer issue of Generations is May 16
Ataxia Doesn’t Stop Her: Kayla Prather’s Story

By Dianne Thigpen, Kayla’s grandmother, who when asked about Kayla, says “She is my hero!”

Kayla is a typical 15-year old. She likes to shop, read the latest teen magazines, talk on the phone, go to movies, and all the other carefree fun things that teenage girls do. The one thing that sets Kayla apart from her friends and other teenagers is that she has Friedreich’s ataxia (FRDA) and uses a wheelchair. But she doesn’t let that stop her. She lives by a motto that states, “I can do anything that others do – I just have to do it differently!”

Kayla, an honor roll student, is a member of the Jaguar Journal, the yearbook staff and recently joined the BETA Club. She volunteers to create awareness about FRDA. She has secured signed proclamations from local city, county and state leaders for International Ataxia Awareness Day, been featured in several newspaper articles, and appeared in a local public service announcement for the Make-A-Wish Foundation. Kayla has been a spokesperson and helped raise funds for various ataxia organizations including NAF’s Greater Atlanta Ataxia Support Group.

Prior to being diagnosed with FRDA, Kayla was a member of a competitive dance team which won several first place trophies. She still has that competitive spirit today and has participated in her school pageant. Kayla has been the only girl in the pageant with a physical disability.

As a sixth-grader Kayla was named Most Photogenic. The next year she placed second runner-up. Kayla enjoys the pageants because they allow her to participate and compete with girls her age, while building new friendships. It also affords her the opportunity to create awareness about FRDA. She uses the time that she “walks” for the judges to share information about FRDA.

This year – her last year in middle school and to her surprise – she won the People’s Choice award and was crowned Miss Jaguar. Words cannot express the joy and pride she felt.

Kayla has a dynamic personality and a zest for life that inspires those who know her. Even with her daily challenges, Kayla stays positive and is always willing to help others. She has been thinking about career choices which include becoming a professional fundraiser or a lawyer working with the physically challenged population and helping to enforce ADA requirements.

“Unless you have a disability, you don’t realize how difficult seemingly simple things are for those with disabilities,” says Kayla, “or even how difficult it is to just get down to the football field to support your team.” Kayla wants to make a difference and is determined to make her dreams become realities.

Kayla shares with other young people who may face challenges, “Never give up – your ambitions can take you places allowing you to change your life or the lives of others.”

Kayla Prather

Photo by TCPICS

North Texas Ataxia Support Group Meeting
10 a.m.-noon at Los Colinas Medical Center, 6800 MacArthur Blvd. at Hwy. 161, Irving, TX. www.ataxia.org/chapters/NorthTexas/default.aspx.

Tuesday, June 17, 2008

Twin Cities Ataxia Support Group
7 p.m. at Roseville Presbyterian Home (located off 35W on County Rd D). Contact Lenore H. Schultz at (612) 724-3784 or lschultz@bitstream.net. www.ataxia.org/chapters/OrangeCounty/default.aspx.

Saturday, June 21, 2008

Orange County Ataxia Support Group Meeting
1:30-4 p.m. Orange Coast Memorial Medical Center (in the basement, next to the cafeteria), 9920 Talbert Ave., Fountain Valley, CA. Contact Daniel Navar at dnavar@ucla.edu. www.ataxia.org/chapters/OrangeCounty/default.aspx.
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 from November 2007 through March 2008. 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.

Heidi & Peter
Timothy Adkins
Ralph Aiello
Alexander Family
M/M A. Alibrio
Michele Aioto
Crystal Allsopp
Jackie Anderson
Kathy Anderson
Linda Anderson
Constance Ashton
Olga Azurdia
Vicki Balogh
Helen Barkhouse
Lenda Barth
Maureen
Bartlett-Carter
Dianne Bates
Dr. Luke Baxley
Betty Beck
Clair Beck
Kay Bell
Jennifer Bellini
Patricia Benyo
Eva Birdsong
Kim Bishop
Stephanie Blake
Fred Blasberg
Gary Blasius
Anetha Borup
Matthew Bousha
Linda Bowen
John Boyd
Hilda Braswell
Muriel Brelan
Jane Brewer
Jamie Brooks
Nancy Brooks
Lynda Brother
Kyle Bryant
Theodore Burdyl
Barbara Cali
M/M Edward Calisi
Evelyn Camacho
Sharon Cameron
Jimmy Campbell
Kenny Canter
James Carr
Richard Carr
Terry Carroll
Jeremy Carroll
Christopher Casey
Peter Castaneda
Gerarda Cellucci
Charles Charleston
Lucille Charleston
Mary Charlton
Stephanie
Chartrand
Douglas Chin
Quock Chin
William Chwee
Jim Cieciernski
JoAnn Cieciernski
Eugene Clark
Krista Clarke
Michael Clementz
Patricia Clementz
Janice Cohen
James Collins
Les Cooley
Verla Corby
Emma Cornwell
Joan Costello
Lucile Covey
Shirley Cox
Jack Cunningham
Carol Curtis
M/M D. Bagwell
Marty Dail
Charlene Danielson
Jeannette Davis
Kennon Davis
Page Davis
Raymond Davis
Pamela Dawn
Diane Deniger
Marlene Dietrich
Constance
DiVincentis
Fred Donnelly
Joe Drake
Monte Drew
Jerome Drew
Shari Dresselhaus
Sandy Dudzic
Ronald Eakins
Buzz Earnhart
Phil Earnhart
Mary Erickson
Daniel Eustace
Trinity Falk
Connie Farmer
Matt Farrow
Vera Faulds
Betty Fears-Jones
Charlie Fisher
M/M Fred Flory
Mark Frykman
Mary Fuchs
Ann Gallagher
Gregson Gann
Rita Garcia
Bryan Gervais
William Gill
Garima Goel
Tanya Goldman
Penny Golfinas
Donna Gorzela
John Goshert
Joan Gowan
Brenda Graner
Lawrence Graner
Jacqueline Gray
Dick Gregory
Larsen Gregory
Richard Gregory
Alan Groben
Brian Groben
David Groben
Nancy Groben
Philip Groben
Jackie Grecio
Paschal Guerio
Shikha Guha
Nicholas Guilde
Patty Gutierrez
Bryan Hackett
Wilbur Hackett
Gordon Hagan
June Hagan
Evelyn Hankins
Jimmy Hankins
Mary Hansen
Anthony Hanson
George Happell
Jean Happell
Donald Hareid
Mary Hartmann
Dyna Hauert
James Hay
Shirley Hayworth
Lois Henrich
David Henry, Jr.
Martha Herbold
Carmela Herman
Ginger Hertfeldt
Greg Hess
Denise Higdon
Candace
Hintermister
Johnny Hogan
Bette Holmes
Jim Home Hanksins
Lois Hough
Louis Howe
Sammy Hubbard
James Hughes
Vera Hughes
Conrad Humphries
Lisa Jaffe
Rinda Janney
Kerry Johnson
Terry Johnson
R. Jurasek
Maureen Jurek
Keiko Kain
Marvin Kamen
Auray Kave
William Keaveney
Robert Keithly
Amy Keller
Pauline Kelly
Regina Kelly
Thomas Kennedy
M/M W. Kern
Maj. (Ret.) Young
D. Kim
John Kiney
Robert Kirchner
Grace Kirkwood
Jamie Kosieracki
M/M J. Kreizner
Norman Labash
M/M J. Laird
Dan Lane
Irene Lanzendorfer
Max Lanzendorfer
Peter Lanzendorfer
Rodger Larsen
Gerald Laukhuf
Lorrie Laukhuf
Denise Laundy
Chui Lee
Linda Lee
Christine Leslie
Bootsie Levick
Harriet Lewis
Richard Lewis
June Liveman
Jacqueline Lombardo
Stephanie Lovelock
Homer Mace
James Mace
M/M R. Macedonia
Gordon Macklin
Marylin Macklin
Carly Magnuson
Gregg Malkin
Kerry Manchester
Rebecca
Mandernach
David Marcy
Deborah Markham
John Marten
Sandra Martin
Bruno
Mastroprimiano
Marco
Mastroprimiano
Rhonda
Mastroprimiano
Helen Mays
Darrin McCartney
A Special Thank You

The National Ataxia Foundation would like to extend a special thank you to all the attendees, speakers, facilitators, exhibitors and numerous volunteers of the NAF 2008 “Blazing A Trail In Research” Annual Membership Meeting held in Las Vegas, NV.

The NAF would like to especially thank the Arizona Ataxia Support Group for their efforts. It was a pleasure working with Rita Garcia, Bart Beck, Mary Fuchs, and Mary-Lisa Orth on this meeting.

Many thanks to Charlotte DePew, who volunteered as our on-site nurse this year; your services are so much appreciated.

We would also like to thank this year’s sponsors. Thank you Special Needs Vehicles for your donation of the name badges. Thank you to Bulk Nutrition for your donation of tote bags for this year’s meeting materials.

The “Blazing A Trail In Research” meeting had over 645 attendees! We appreciate your participation in making this conference so successful. Thank you so much for the wealth of information and knowledge that was brought to the conference by all the speakers, facilitators and exhibitors. The information and skills taken away from this conference by the attendees is invaluable and worth more than any words can say.

Thank you to the Flamingo Hotel and the Las Vegas CVB for their service and hospitality throughout this event. A special thank you is also extended to NBC Channel 8 News–Las Vegas for their media coverage.

Michaela McAlpine
Maury McDonald
M/M J. McDonough
Charley McLaughlin
Earl McLaughlin
Emily Medina
Suzanne Merrill
John Miller
Refiy Miller
Linda Mitchell-May
Diane Modaff
Carl Moles
Minnie Molini
Rose Montalbano
Dolores Morello
Gary Morris
Charles Murphy
Rhett Myers
Bruce Nanninga
Craig Nielsen
Chip Niles
John Norton
Iris Nusbaum
Leta O’Brien
Patricia O’Brien
M/M W. O’Connell
Kathie O’Day
Mary-Lisa Orth
Josh Ostby
Darrell Owens
Hanna Parce
Irvin Parce
Katherine Parnham
Paula Partilla
Julie Passarelli
Tyrell Pavelec
Linda Perkowski
Jo Ann Peter
Michael Peter
Jane Petticrew
Rita Powell-Lobasco
David Price
Denise
Price-Dudley
Jan Primeaux
Julie Quinlivan
Scott Quinn
John Rakshys
Charity Ranger
Florence Rinaldi
Jennifer Robinette
Janice Robinson
Nathan Robinson
M/M R. Rocca
Don Roemke
Ken Roemke
Walter Roemke
Rodney Rydeen
Mark Salvani
Santa Croce Family
Donald Santa Croce
N. Santa Croce
Marilyn Saunders
Josephina
Schembre-McCabe
Marcella Schifrin
Jay Schlueter
David Schon
Marvin Schoon
Yvette Scimone
Derek Semler
Sarah Shabaker
Col. Alexander
Shafer
Hunter Shankle
Sherry Sharp
Breah Shepherd
Phyllis Siegel
Dianne Simao
Col. D. A.
Slingerland
Kelly Smillie
Robin Smith
Windy Smith
Miriam Sommese
Marlene Stadille
Joey Staiger
Joseph Staiger
Gert Stein
Cathy Steward
Pearl Straub
Mathew Strop
Elizabeth Stueber
Jody Stutchbury-Raposo
Dee Sweeney
Wes Sweeney
Ernie Talarico
Tiffinay Talarico-Compiano
Roger Teske
Bernice Thierfelder
Melva Tillett
Garrett Timble
Mark Tokarz
Mark Torvinen
Donna Triebes
Dennis Trietsch
Eva Tischler
Margie Tseng
Bonnie Tucker
Phil Turnbull
Jay Underwood
John Underwood
Bob Vande Brake
M/M R. Van Horn
Leslie
Van Iderstine
Derrick Veder
Maria Vergili
Mary Vida
Marlea Waddell
Dean Walker
Donald Walker
Vinitha Weera-Sooriya
Susan Weiler
Albert Wester
David Westrick
Michael Wheeler
Pat Whiton
Charles Williams
Jeanette Wilson
Linn Wilson
Connie Wolff
Wolfson Family
Alena Wolfson
Michael Wolfson
Donald Woods
Nicholas Woods
Thomas Wooten
Arthur Workley
Clarence Workley
Harry Workley
Joanne Workley
Larry Workley
William Workley
Leona Yates
Jack Yobs
Nathan Young
Ryan Young
Pete Zamakoupis
Hitomi Zeller
Is your address correct? Are you receiving more than one issue of *Generations*? If there are any changes that need to be made, please call NAF at (763) 553-0020 or e-mail naf@ataxia.org. Thank you!

**GIFT – HONOR – MEMORIAL**

A contribution given in memory of a friend or relative is a thoughtful and lasting tribute, as are gifts to honor your friends or family. A Gift Membership is a wonderful gift to a friend or relative for special occasions like birthdays, graduations, anniversaries, and holidays. NAF will acknowledge your gift without reference to the amount.

Simply fill out this form and mail with your check or credit card information to the National Ataxia Foundation.

Honor/Memorial envelopes are available free of charge by writing or calling NAF.

My contribution is:
- ✐ In Memory
- ✐ In Honor
- ✐ Gift Membership

Name ________________________________
Occasion _____________________________

Send Acknowledgment Card to:
Name ________________________________
Address ______________________________
City/State/Zip __________________________

**MEMBERSHIP**

Yes, I want to help fight ataxia! Enclosed is my membership donation, which enables NAF to continue to provide meaningful programs and services for ataxia families. (Gifts in US Dollars)

- ✐ Lifetime membership $500 +
- ✐ Annual memberships:
  - ✐ Patron membership $100-$499
  - ✐ Professional membership $45 +
  - ✐ Individual $25 +
  - ✐ Household $45 +
- ✐ Addresses outside the U.S. please add $15

Your Name ________________________________
Address ________________________________
City/State/Zip ____________________________
E-Mail _________________________________

**PAYMENT INFORMATION**

Gifts are tax deductible under the fullest extent of the law.
- ✐ Check. Please make payable to the National Ataxia Foundation.

Total Amount Enclosed $ _________________

Credit Card: ✐ Visa ✐ Master Card
Name on Card ____________________________
Card # __________________________________
Exp. Date ________________________________
Signature ________________________________
Phone Number ____________________________

Is your address correct? Are you receiving more than one issue of *Generations*? If there are any changes that need to be made, please call NAF at (763) 553-0020 or e-mail naf@ataxia.org. Thank you!