Generations

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Genomic Mouse Models of Spinocerebellar Ataxia

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The following is a research summary of a grant funded by NAF for fiscal year 2006.

Mutations in the gene encoding protein kinase C gamma (PRKCG; PKCγ protein) cause Spinocerebellar Ataxia Type-14 (SCA14). To date, 20 different mutations found in the functional domains of PKCγ have been shown to cause highly variable forms of disease (phenotypes).

Our research has focused on generating genomic transgenic (Tg) mouse models of SCA14 that will allow us to investigate the pathogenesis of the disease. It is our belief that a more comprehensive understanding of the pathogenesis of SCA14 may help determine at which stage intervention must be initiated to prevent or ameliorate the neurodegenerative process and that findings from our animal model studies could lead to rational approaches for designing new treatments and therapeutics for SCA14.

The first aim of our study was to genetically manipulate a copy of the human PRKCG gene contained in a bacterial artificial chromosome (BAC: a large piece of human DNA maintained in bacteria) by introducing SCA14-associated PRKCG mutations located in different functional domains of the protein and a genetic tag that would allow us to identify our manipulated human gene/protein in the context of a mouse host.

The second aim of our study was to introduce these modified (mutant) PRKCG "transgenes" into a mouse host, allowing the production of our mutant human PKCγ proteins in the "transgenic" disease model mice. We hypothesized that by using the entire human PRKCG gene we would maintain appropriate (normal) tissue-specific and temporal production of the mutant PKCγ, thus allowing us to complete our third aim of investigating the disease mechanisms in SCA14 over the life-time of our animal models. Because our transgenic mice would have higher levels of PKCγ proteins (over-expression) compared to non-transgenic mice we also sought to create "control" PRKCG transgenic mice that harbored a normal (wild type, WT) non-mutated human PRKCG gene, allowing us to

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discern SCA14 mutation-specific effects from PKCγ protein over-expression effects.

The mutations we introduced were a 101Y mutation in the beginning of the protein and a 643L mutation at the end of the PKCγ protein; not only are the mutations in different functional domains of PKCγ, but they also represent clinically diverse phenotypes found in SCA14.

Comparison of the mutant and WT SCA14 models would allow an evaluation of both region-specific and mutation-specific PRKCG effects in or models of SCA14.

With the generosity and support of the National Ataxia Foundation, in the last year we have generated two different mutant (101Y and 643L) transgenic mouse models of SCA14 and two "normal" PRKCG control transgenic mouse models. The 101Y mouse model and its WT transgenic counterpart have a short V5-His6 genetic tag that will allow identification and purification of our transgenic proteins. The 643L mouse model and its WT transgenic counterpart have a longer EGFP genetic tag that will allow direct fluorescence-based microscopic visualization of the human transgenes.

We have generated one 101Y- RKCG-V5-His6, one WT-PRKCG-V5-His6, seven WT-PRKCG-EGFP, and three 643L-PRKCG-EGFP transgenic founder mice (independent mouse lines). Thus far, four of the WT-PRKCG-EGFP and two of the 643L-PRKCG-EGFP transgenic founders have genetically passed their human transgenes to their offspring, establishing these transgenic "lines."

We have shown in several of these lines that the human PRKCG messenger RNAs (the transitional copy of PRKCG that is then translated into the PKCγ protein) are appropriately expressed in the brain tissues and are not expressed in non-neuronal tissues.

We will continue to establish and expand all of our transgenic lines and behaviorally and pathologically study these mouse models over their lifetime to understand the role of mutant PKCγ in the pathogenesis of brain dysfunction and neuronal cell loss in SCA14. The long-term goals are to study these models throughout their life to aid in the development of treatments for SCA14 and related diseases. ❖

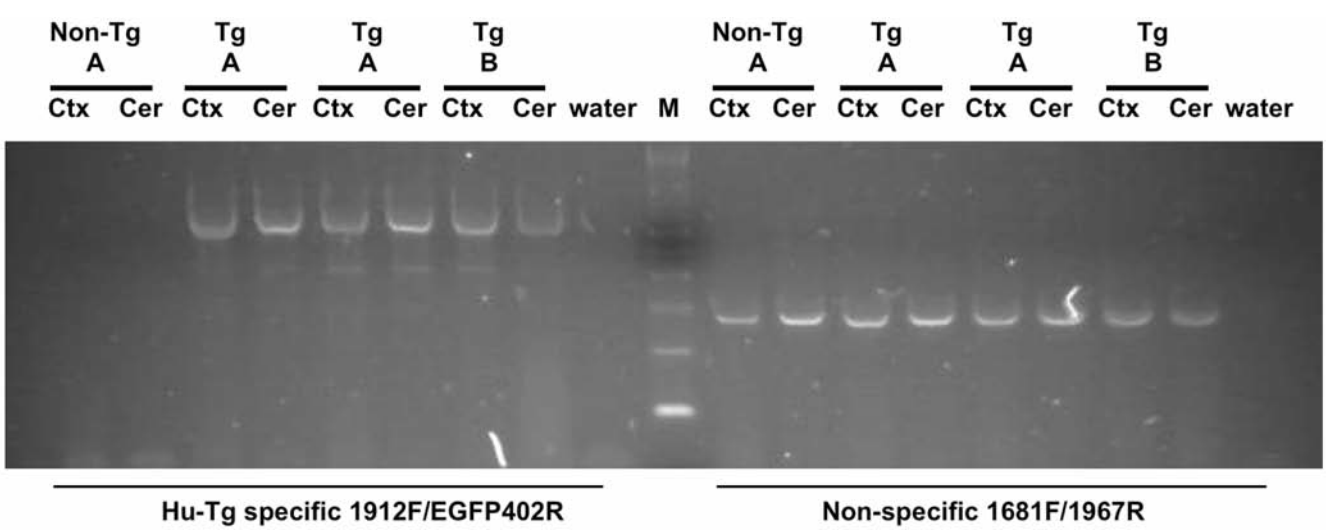

Figure 1. RT-PCR of PKCγ. RNA, isolated from cortex (Ctx) and cerebellum (Cer) tissue of two month-old 643L-EGFP transgenic (Tg) and non-transgenic (Non-Tg) mice, was reverse transcribed and PCR amplified using human transgene specific primers (1912F/EGFP412R) and non-specific PKCγ primers (1681F/1967R). Lines A and B are mutant transgenic lines established from independent founders; two Line A Tg siblings were tested.

The SCAs Go to Washington

By Harry Orr, PhD, National Ataxia Foundation Research Director

Building on the fact that significant advances have been made in understanding the molecular pathogenesis of the spinocerebellar ataxias (SCAs), and that genetic therapeutic approaches have been undertaken in animal models of the SCAs, the National Institute of Neurological Disease and Stroke (NINDS) of the National Institutes of Health (NIH) along with the National Ataxia Foundation sponsored a workshop on these disorders. This two-day workshop entitled "Therapeutic Development for Inherited Spinocerebellar Ataxias" was held in Bethesda, MD on April 10 and 11 of this year and focused on SCA1, SCA2, SCA3, and SCA7.

Dr. Harry Orr

The major goal of this workshop was to address how to translate the basic research findings to a clinical trial for these SCAs. By bringing together leaders in the basic science and clinical manifestations of the SCAs, the workshop aimed to delineate the state of the field and identify what key questions remain to be answered before a trial can be initiated. Together with leaders in the various therapeutic approaches under consideration and experts in other diseases, the participants discussed roadblocks and developed scientific strategies to resolve these issues. In preparation for future trials, the currently available clinical rating scales for assessing severity of disease were critically assessed, and outcome measures for future trials were discussed. Because the objective was to reach consensus on the critical issues and barriers that need to be addressed as we move into clinical trials for these disorders, the workshop was organized with ample time for discussion among participants following each session.

After the welcome by Dr. Story Landis, Director of the National Institute of Neurological Disease and Stroke, the first day was devoted to the clinical and basic science aspects of the SCAs. The genetic and clinical features of the SCAs were updated and summarized by Drs. Alexis Brice and Chris Gomez (University of Chicago), respectively. The next session was devoted to basic research updates on SCA7 and SCA1 from Drs. Al LaSpada (University of Washington) and Huda Zoghbi (Baylor College of Medicine). Both presenters discussed how the research is leading to the elucidation of plausible therapeutic targets.

The afternoon of the first day covered several therapeutic approaches that might be applicable to the SCAs. The presentations included two on the use of screens to identify small molecules with potential for drug development, from Drs. Chris Austin (NIH) and Rob Wilson (University of Pennsylvania). Dr. Beverly Davidson (University of Iowa) then presented her work on the use of small interfering RNAs to silence the expression of mutant polyglutamine proteins in the brains of transgenic mice.

The second day began with two presentations on key issues confronting us as we move the results from basic research to the clinic; so-called translational research. First, how one might deliver therapeutics agents to the brain was summarized by Dr. Andres Lozano from the University of Toronto. Dr. Russell Katz ⏩

from the FDA then reviewed the regulatory issues that must be met in order for a therapy to be approved and the various steps towards final approval.

In the fifth session, specific issues facing clinical trials were discussed. Dr. S.H. Subramony (University of Texas Medical Branch, Galveston) discussed the need for and how one might configure a clinical ataxia rating scale that is easy and readily adaptable to a multi-center trial. This was followed by an elegant presentation from Dr. Thomas Klockgether (University Hospital Bonn, Germany) describing how the Europeans have already addressed many of these issues in their organization EUROSCA. In the last session Dr. Jill Heemskerk from NINDS/NIH reviewed the various resources for translational research available from the NIH. In closing there was a very lively discussion from all participants aimed at reaching a consensus on where things stand and at identifying the roadblocks that must be addressed in order to develop a SCA clinical trial.

Among the key conclusions reached were: 1) There are several potential targets for therapeutic trials for one or more of the SCAs. Two examples that received the most consideration were HDAC inhibitors and lithium. It was strongly felt that in both cases trials with additional models are warranted. 2) Several participants expressed the view that there are compelling reasons to organize a multi-center, possibly international, clinical SCA trial. This requires the presence of more organized, broad-based support for translational research. In addition more work needs to be done to develop a rating scale that will allow clinicians to measure disease progression over time, with a long-term goal of preventive trials in the SCAs on the horizon. 3) To get things started it was suggested that a small scale, phase 1/2 trial of lithium in SCA1 should be developed within the next year.

Over the past few years, substantial progress has been achieved in understanding the SCAs. This fact, together with the discussions fostered by the workshop, indicate that the scientists and clinicians studying the SCAs are well poised to facilitate translation of the basic research findings to a clinical trial for the SCAs.

A manuscript based on the workshop will be drafted in order to summarize the current status of research on SCAs with respect to plausible molecular targets, suitability of in vitro and animal models for therapeutic development, potential modes of therapy and their current status of development, steps required to bring these therapeutic strategies to the clinic, and clinical aspects relevant to conducting a trial including outcome measures.

The workshop catalyzed collaborations to pursue the development of therapeutics, identify gaps in research, and inform the NIH/NINDS about opportunities to expedite the development of therapies for SCAs, and perhaps other disorders caused by similar mutations. The willingness of the NIH/NINDS to hold this workshop is a major milestone for the SCAs. This field is turning the corner towards undertaking clinical trials and development of a treatment.

Congratulations to all of the basic and clinical researchers who are working on this effort. Thanks to the NIH/NINDS and Dr. Wendy Galpern who along with Dr. Katrina Gwinn helped organize the workshop. A special thanks to the NAF and the SCA families without whom this remarkable progress would not have taken place. ❖

New CFC Number

The National Ataxia Foundation's NEW 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.

From the Desk of the Executive Director

The National Ataxia Foundation has been serving ataxia families for 50 years through research, education, and support services. Each of these program areas is essential in the fight against ataxia, but today I would like to focus on the research program.

Currently, there are four ataxia research programs sponsored by the National Ataxia Foundation: NAF Research Program, NAF Research Fellowship Award, NAF Young Investigator Award, and NAF Friedreich's Ataxia Special Projects Award.

During the past eight years the Foundation has awarded funding to 100 promising ataxia research studies in eight countries. Those funds have come from generous donations made by members, foundations, corporations, individuals, family fund raisers, and the annual ataxia research drive.

Michael Parent

Over the years, the majority of these research funds have been made available by generous donors who have contributed to the NAF Annual Ataxia Research Drive. In October, the National Ataxia Foundation will be conducting its 2007 NAF Annual Ataxia Research Drive and we need your help.

Currently, the Foundation is reviewing many promising ataxia research proposals from around the world. These studies will help us find more answers in stopping ataxia. Each year the Foundation receives many more research funding requests than there are funds available. That is why it is so important that each of us support this important research drive.

The process of funding promising ataxia research is in many ways like a three-legged stool. The legs: NAF, researchers, and donations. If one of the legs fails, the stool will fall.

The First Leg: The NAF has a comprehensive system in place to receive applications, review and prioritize research proposals, award research funding, monitor research studies, and report the research results.

The Second Leg: Due, in part, to the long-term efforts of the Foundation, there are many outstanding ataxia scientists throughout the world. These are very exciting times in ataxia research: the Foundation has received many quality ataxia research grant applications which are deserving of funding.

The Third Leg: Donations made through the Annual Ataxia Research Drive makes it possible to fund worldwide, vital ataxia research. Each dollar donated through this drive brings us closer to finding more answers, to developing effective treatments and ultimately to finding a cure.

Your participation in this drive is crucial and we need your help.

Shortly, you will be receiving the 2007 NAF Annual Ataxia Research Drive letter in the mail. Please mail your generous donation in the research drive return envelope or you may donate on-line at *www.ataxia.org*. Donations in the amount of $100 or more can be designated for research in a specific type of ataxia.

Each leg of a three-legged stool depends upon the stability of one another. We are depending on you for your support. Thank you.

Featured Board Member of the NAF: Robin Smothers

Robin Smothers was elected to the National Ataxia Foundation Board of Directors in 2002 and is also very active in the Foundation's Public Relations Committee. In that capacity, Robin has given countless volunteer hours in bringing ataxia to the forefront.

Robin continues to develop and submit press releases to various media outlets for numerous ataxia events including International Ataxia Awareness Day, NAF's annual membership meetings, support group events, research announcements, and much more. She has developed media kits for the Foundation to be used by chapters, support groups, ambassadors, and members and has given presentations at a number of NAF annual membership meetings on topics relating to the media.

Robin Smothers

Robin is no stranger in working with the media. She has worked in communication positions at nonprofit, corporate, and agency settings for more than 17 years. Robin, who recently received a Certificate in Master of Business Communication from the University of St. Thomas, began her own marketing communications firm in 1999.

As an active community leader, Robin serves on various boards including the Minnesota Women in Communications and the Minnesota School/Public Relations Association. Recently, the Association for Women in Communications awarded Robin the AWC Headliner Award, which recognizes outstanding contributors to the organization.

Robin resides in Golden Valley, MN where she is active in the Golden Valley Little League, church, and her local school district. She is married to John Lawson and has three boys, Tony, Ian, and Will.

As a dedicated NAF Board Member, Robin is committed to "getting the word out about ataxia." Through her technical assistance and hands-on involvement, Robin has made a significant impact on the Foundation and ataxia awareness.

As a support group leader, ambassador, or chapter leader, many of you have met Robin at an NAF Leadership Meeting, where she has given presentations, while others may have met her at an annual membership meeting. For those of you who have not yet met Robin, you will find her to be a caring and compassionate person who cares very deeply about the ataxia community.

"Ataxia awareness is so important in letting people know about ataxia and that there is an organization here to help them." Robin stated. "The National Ataxia Foundation has been serving ataxia families for 50 years through research, education, and support services and we are finding new ataxia families each day through our important awareness efforts."

The National Ataxia Foundation is honored by and truly appreciates Robin's commitment and involvement with the Foundation. She continues to make a significant impact in ataxia awareness and on the ataxia community.

Thank you, Robin. ❖

Potential Role of DNA Instability in the Pathogenesis of Friedreich's Ataxia

By Irene De Biase, MD, PhD

University of Oklahoma Health Sciences Center

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Friedreich's ataxia (FRDA) is characterized by progressive incoordination (ataxia) with onset before 25 years, loss of tendon reflexes, loss of position and vibration senses, and slurred speech. The primary site of pathology is the neurons in the dorsal root ganglia (DRG), which relay sensory information (such as the position of individual joints in space) from the periphery of the body to the spinal cord, and eventually to the brain. Primary degeneration in the DRG is followed by secondary degeneration of several tracks (tracts / columns) in the spinal cord that direct sensory information from the DRG to various parts of the brain (e.g. the spinocerebellar tract, which carries information to the cerebellum, where information is processed in order to maintain balance). Neurodegeneration in FRDA is progressive, and the rate of progression is variable. On average patients are unable to walk independently by 25 years, and often die prematurely.

FRDA is a genetic disease, caused by mutation in both copies of the *FXN* gene. The most common mutation that causes FRDA, accounting for >98% of all mutations, involves a large expansion of a GAA triplet-repeat sequence. So, while most individuals have about eight consecutive "GAA" triplets in their *FXN* genes, patients usually have more than a hundred of these GAA triplets (most patients have around 800 triplets). The *FXN* gene normally codes for a message (RNA) which in turn codes for the protein, frataxin. Frataxin is required for the normal functioning of the mitochondria, the "power generator" of the cell. Expanded tracts of the GAA triplet-repeat sequence in the *FXN* gene interfere with production of the RNA message, which in turn results in the deficiency of frataxin. The absence of frataxin results in a deficiency of energy in some tissues, including neurons in the DRG and cardiac muscle in the heart. This is the reason FRDA patients develop neurodegeneration and heart failure. The deficiency of frataxin is proportional to the length of the expanded repeat, so the length of the expanded GAA triplet-repeat determines the severity of disease. For example, a patient with 250 and 400 repeats in the two copies of the *FXN* gene would develop a milder form of the disease, with later onset and slower progression, compared with a patient who has 800 and 1000 repeats.

It is not entirely clear why the neurons in the DRG specifically succumb to the disease process when the expanded GAA triplet-repeat mutation is present in all neurons of the nervous system. Nor is it clear why the disease is progressive. Our previous results indicated that the expanded GAA triplet-repeat mutation is highly unstable, i.e. it changes in length during parent-to-child transmissions (intergenerational instability), and also among different tissues and cells within the same patient (somatic instability). This is exceptional because most of our DNA remains unchanged in every cell of the body and when transmitted from one generation to the next. Since the extent of frataxin deficiency and the severity of disease are controlled by the length ▸▸

of the repeat tract, we considered the possibility that the DRG in FRDA patients may have longer GAA triplet-repeat tracts than other parts of the nervous system, making the DRG more susceptible to degeneration.

Here we summarize our recent findings which demonstrate that indeed the already expanded GAA triplet-repeat sequence develops further large expansions in the DRG of FRDA patients. We show that somatic instability of the expanded GAA triplet-repeat is progressive throughout life, possibly explaining, at least in part, the progressive nature of the disease process. Finally, we describe a suitable mouse model that mimics the human condition of progressive accumulation of expansions with age in a disease-relevant way. These findings indicate that somatic instability of the expanded GAA triplet-repeat sequence in FRDA is an important determinant of disease pathogenesis, and therefore a target for therapeutic intervention.

Progressive accumulation of large expansions of the already expanded GAA triplet-repeat mutation in DRG of FRDA patients

[This work was published in: De Biase I., Rasmussen A., Endres D., Al-Mahdawi S., Monticelli A., Cocozza S., Pook M., Bidichandani S.I. Progressive GAA expansions in dorsal root ganglia of Friedreich ataxia patients. *Ann. Neurol.* 61:55-60 (2007)]

We used a sensitive method, called small pool PCR analysis, to detect changes in the length of the expanded GAA triplet-repeat mutation in individual cells. We analyzed over 10,000 individual *FXN* genes from multiple tissues, including different parts of the nervous system, derived from autopsies of six FRDA patients. Their onset of disease ranged between 5-11 years, and age of death ranged from 17-47 years. All patients had expanded GAA triplet-repeat mutations, with "baseline" repeat lengths ranging from 350-1030 triplets, and "large expansions" that were >20% longer than the baseline repeat. We found that DRG had the highest frequency of large expansions of all tissues (~7% versus ~1.5% for all other tissues). The frequency of large expansions in DRG showed a strong correlation with the age of the patient; ranging from 0.5% at 17 years to 13.9% at 47 years. This indicates that DRG, the site of progressive pathology in FRDA, is also the site of progressive accumulation of further large expansions of the GAA triplet-repeat mutation.

Somatic instability of the expanded GAA triplet-repeat mutation in FRDA progresses throughout life

[This work was published in: De Biase I., Rasmussen A., Monticelli A., Al-Mahdawi S., Pook M., Cocozza S., Bidichandani S.I. Somatic instability of the expanded GAA triplet-repeat sequence in Friedreich ataxia progresses throughout life. Genomics, in press (2007)]

We wanted to show independent evidence that somatic instability of the expanded GAA triplet-repeat mutation did in fact increase with the age of the patient. We therefore analyzed a panel of DNA from blood samples of nine individuals with a wide age range (2y, 9y, 16y, 17y, 24y, 29y, 36y, 43y, and 49y) who had expanded GAA triplet-repeat mutations ranging in size from 350-1000 triplets. Using the same sensitive assay to detect length changes in the expanded GAA triplet-repeat mutation mentioned above, we analyzed over 6200 individual *FXN* genes. This analysis showed that the amount of somatic instability ranged from 17%-78.7% in these individuals, and there was a statistically significant increase with age. This result supported our previous observations that somatic instability in human tissues is progressive and increases throughout life.

An animal model for somatic instability of the expanded GAA triplet-repeat mutation

[This work was published in: Clark R.M.,

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Potential Role of DNA Instability...
Continued from page 9

De Biase I., Malykhina A., Al-Mahdawi S., Pook M., Bidichandani S.I. The GAA triplet-repeat is unstable in the context of the human *FXN* locus and displays age-dependent expansions in cerebellum and DRG in a transgenic mouse model. *Hum. Genet.* 120:633-40 (2007)]

A potential problem with the identification of progressive somatic instability in humans is the fact that we analyze instability in different individuals over a wide age range rather than by following the same individual over decades. Therefore, the differences in instability could be due to factors other than the age of the individual. Another problem is the limited availability of human tissue samples, especially from comprehensive autopsies that include tissues such as the DRG. To overcome these problems a transgenic mouse was created by inserting into the mouse genome a human *FXN* gene containing an expanded GAA triplet-repeat sequence with 190 triplets, mimicking the *FXN* genes of FRDA patients. Using the sensitive small pool PCR method we identified progressive accumulation of large expansions specifically in the cerebellum and DRG. Careful analysis of mice of different ages revealed low levels of instability at two months, which expanded by about 0.3-0.4 triplets/week, resulting in several large expansions by 12 months (the usual lifespan of mice is two years). Interestingly, the frequency and magnitude of the expansions were similar to those seen in FRDA patients. Since this mouse model has two normal (mouse) frataxin genes of its own, it does not develop FRDA; therefore, the large expansions we observe are not a consequence of the disease process. Because this mouse model recapitulates the disease-specific somatic instability seen in the DRG of FRDA patients, it can be used to study the mechanisms of genetic instability, and to develop specific therapeutic measures aimed at limiting the accumulation of large expansions.

Summary

It is intriguing that patients show progressive accumulation of large expansions of the GAA triplet-repeat mutation specifically in their DRG, the primary site of neurodegeneration in FRDA. This progressive somatic instability may be, at least in part, the reason for the selective and progressive pathology seen in the DRG of FRDA patients. We have developed a mouse model that mimics the disease-specific somatic instability seen in FRDA patients, which will serve as a useful tool to investigate potential therapeutic measures aimed at preventing or limiting the progressive accumulation of large expansions in the DRG. ❖

NAF Video to be Aired on Television

The National Ataxia Foundation is pleased to announce that the new Starfish Television Network will air a 19-minute version of the NAF-produced video "Together There is Understanding" every Saturday afternoon at 3:30 p.m. EST.

Starfish is a nonprofit organization and is providing us this television time at no cost, so we encourage you to let your friends, family, co-workers, and others know about these airings. To watch Starfish on a DISH 1000 system, go to channel 9408. If you do not have DISH 1000, go to *www.starfishtv.org* and click on the "Watch Live Starfish TV" link.

May we also suggest that each of you visit *www.starfishtv.org* and click on the "I Want Starfish TV" button. Here, you can request that your local cable/satellite television provider carry Starfish, helping them get a larger audience and helping assure that more people will see the program and learn about ataxia.

The 50-minute version of the video is available to order at *www.ataxia.org* and in *Generations*.

PolyQ Proteins and Pre-mRNA Splicing

By Michael D. Hebert, PhD

The University of Mississippi Medical Center

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Nuclear inclusions formed by proteins with expanded polyglutamine tracts are found in several neurodegenerative diseases. The effect of nuclear inclusions formed by these disease proteins on the functional organization of the nucleus is only partially understood.

In particular, it is not known if polyglutamine disease proteins disrupt the function of Cajal bodies, which are subnuclear domains that play a role in the biogenesis of small nuclear ribonucleoproteins (snRNPs). Since snRNPs are an integral part of the pre-mRNA splicing machinery, it is possible that mutant proteins that alter Cajal body activity indirectly affect pre-mRNA splicing.

Here, we evaluate three different polyglutamine disease proteins – ataxin-1, ataxin-3 and huntingtin – for their ability to disrupt Cajal body localization and reduce the splicing of an artificial reporter in HeLa cells. Consistent with previous observations, ataxin-1 inclusions do not drastically alter the localization of Cajal bodies. In contrast, ataxin-3 inclusions associate with this structure. Inclusions formed by a fragment of the huntingtin protein do not associate with Cajal bodies or PML bodies, another subnuclear domain. Of the three disease proteins, only ataxin-3 significantly decreases the splicing of an artificial reporter.

These results support the hypothesis that different mutant proteins vary in their ability to disrupt nuclear organization and function. This work also suggests that strategies to increase pre-mRNA splicing may benefit patients with Machado-Joseph disease. ❖

Getting Married?

If you are getting married, you can support National Ataxia Foundation by registering with the I Do Foundation.

From honeymoons to invitations to wedding gifts to charitable wedding favors, the I Do Foundation allows couples and their guests to make wedding-related purchases that generate donations for charity.

The I Do Foundation's Charity Registry service also makes it easy for guests to make donations in lieu of gifts. All of these services are available free of cost at *www.IDoFoundation.org*.

Check it out today, and be sure to select National Ataxia Foundation as the beneficiary of your charitable wedding. ❖

NAF Merchandise

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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

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by G.N. Rangamani, PdD, CCC-SLP
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Mechanisms of Ataxin-1 Mediated Cytotoxicity

Chih-Cheng Tsai, PhD
UMDNJ-Robert Wood Johnson Medical School

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Increasing evidence indicates that several ataxia-causing proteins are functionally connected with gene transcriptional regulation – that is, with turning gene expression on and off. Because gene expression takes place in the nucleus of cells, where our genetic material is stored in the form of DNA, these disease-causing proteins must localize to the nucleus in order to cause toxic effects on selected neurons. Thus far, however, we do not understand in detail how these ataxia proteins cause damaging effects within the nucleus.

Our research on ataxin-1 (ATXN1), whose mutant form causes spinocerebellar ataxia type 1 (SCA1), and our characterization of a related protein, called "Brother of Ataxin-1" (BOAT1), revealed that these proteins' functions are connected with a class of transcriptional co-repressors found in the nucleus that includes SMRT (Silencing Mediator of Retinoid and Thyroid hormone receptors) and N-CoR (Nuclear receptor Co-Repressor). Our results appear in two recent papers that rest on research supported by the National Ataxia Foundation: Tsai et al. *PNAS* (2004), and Mizutani et al. *EMBO J.* (2005).

One major function of SMRT and N-CoR is to assist nuclear receptors, such as thyroid hormone receptor (TR), to turn the expression of their target genes off. We therefore focused on the relationships among ATXN1, SMRT/N-CoR, and TR. Indeed, our recent results reveal that ATXN1 binds specifically and directly to the regulatory regions of a known direct target gene of TR in human cells. These new findings establish that ATXN1 is directly involved in gene transcriptional regulation, and suggest that the neurotoxicity caused by mutant ATXN1 may involve the thyroid hormone pathway.

Building on these recent discoveries, we are now investigating whether mutant ATXN1 also affects additional nuclear receptor signaling pathways. By helping us to understand these very detailed events in the nucleus, such as how nuclear receptor signaling is affected by mutant ATXN1, our research may contribute towards finding effective therapeutic approaches to treat SCA1.

We are very grateful for the support from NAF, which has allowed us to venture into new research territory in the study SCA1. The Foundation's generous support made possible the research progress that recently enabled us to secure a R01 grant from National Institutes of Health. Support from the NAF and from the NIH has allowed us to further advance our research on SCA1. We hope that our research will pave the way to find better treatments for SCA1 patients in the not-too-distant future. ❖

Caregiver's Corner

NAF has permission to reprint the following excerpts from the "The Comfort of Home" series.

Practical Aids for Living

Little Things Can Help a Lot

Many people have some physical limitations, whether these involve moving, seeing, hearing, communicating or using everyday tools like knives, forks, spoons, toothbrushes or telephones. Solutions exist for overcoming these limitations. Below are examples of different types of bathroom equipment and other assistive devices

It is important to assess medical, social, and environmental factors to make a good decision on what equipment you need. Before buying any equipment or signing any contract for rental, consult your doctor, physical or occupational therapist, or nurse. Salespeople may not have the training necessary to assist you in making a decision about what you need. Occupational therapists can consult on low-cost substitutes for expensive equipment.

With appropriate doctor's orders and documentation, Medicare or private insurance covers some equipment. For individuals covered by Medicare only (or Medicare and private insurance), you should contact your insurance carrier to check if the equipment is covered. Follow their procedures for pre-authorization.

Eating Aids

- Spoons that swivel for those who have trouble with wrist movement
- Foam that can be fit over utensils to increase the gripping surface so they can be lifted more easily
- Plate guards or dishes with high sides that make it easier to scoop food onto a spoon
- Rocker knives that can cut food with a rocking motion
- Food-warming dishes for slow eaters
- Mugs with two handles, a cover, a spout, and a suction base

Dressing Aids

- Buttonhooks to make buttoning clothes easy
- Dressing sticks that make it possible to dress without bending
- Long-handled shoehorns so a person doesn't have to bend over when putting on shoes
- Lock aids that keep stockings open while they are being put on
- Elastic laces for shoes to allow slip-on

Devices for Summoning Help

- Touch-tone phones with speed dials
- Medical security response systems
- Beepers for the caregiver
- Wireless transmitters for emergency response

Resource for You

- ABLEDATA – (800) 227-0216, (301) 608-8998 or www.abledata.com – Stores information on thousands of assistive devices for home health care, from eating utensils to wheelchairs. Provides prices, names, and addresses of suppliers.

Medicare

Medicare does not help pay for assistive devices, but does pay for durable medical equipment in some cases. To be covered, a doctor must prescribe the equipment and it must be medically necessary. It must be useful only to the sick or injured person and must be reusable. Medicare will pay for the rental of certain items for no more than 15 months. After that time you may buy the equipment from the supplier. If the person in your care has met the deductible, Medicare will pay 80% of the approved charges on the rental, purchase, and service of equipment that the doctor has ordered. ▸▸

Taking Care of Yourself

Outside Activities

Successful caregivers don't give up their own enjoyable activities. Many organizations have respite care programs to provide a break for caregivers. Other family members are often willing – even pleased – to spend time with the person in your care. It may be possible to have respite care on a regular basis. Keep a list of the people you can ask for help once in a while.

If your friends want to know how they can help ease your burden, ask them to:

- Telephone, and be a good listener as you may voice strong feelings
- Offer words of appreciation for your caregiving efforts
- Share a meal or just a cup of coffee with you
- Help you find useful information about community resources
- Show genuine interest
- Stop by or send cards, letters, pictures, or humorous newspaper clippings
- Share the workload or hire a relief caregiver.

Caregivers at Risk

There is increasing evidence that providing care for a chronically ill person can have harmful physical, mental, and emotional consequences for the family caregiver. As families struggle to care for their loved ones, their own health is jeopardized.

Family Caregiver Alliance has a fact sheet called "A Population at Risk" (available online at *http://caregiver.org*) that describes the impact of caregiving on the mental and physical health of the caregiver.

Studies show that caregivers:

- Suffer from high levels of stress and frustration
- Show higher levels of depression
- May exhibit harmful behaviors
- Are in worse physical health than non-caregivers
- May have increased risk of heart disease
- Have lower levels of self-care
- Increased mortality.

Fire Safety Tips for People with Disabilities

People with disabilities should be more cautious because of physical limitations and a decreased ability to react in a fire emergency. In some cases, people with disabilities may need the help of a caregiver to practice proper fire safety precautions.

Be aware of the special fire warning devices that are available, such as smoke alarms with a vibrating pad or flashing light for the deaf and hard of hearing. In case of fire, plan escape around the person's capabilities.

- Know at least two exits from every room and be sure a walker or wheelchair can get through the doorways.
- Contact your local fire department's non-emergency line and explain the special needs. They will probably suggest escape plan ideas, and may perform a home fire safety inspection and offer suggestions about smoke alarm placement and maintenance.
- Ask emergency providers to keep the person's special needs information on file.
- Keep a phone near your bed and be ready to call 911 or your local emergency number if a fire occurs.

Source: USFA, *www.usfa.fema.gov*. ❖

Architecture of the National Ataxia Database Web Application

Susan L. Perlman, MD

David Geffen School of Medicine at UCLA

The following is a research summary of a grant funded by NAF for fiscal year 2006.

We have designed and built a secure database to store and manage the national research repository for ataxia patient data. We have created an easy-to-use web-based interface into the database for study management, data entry, data validation, and data output. The system is highly secure, and fully HIPAA compliant.

Figure 1 illustrates the basic components of the architecture of the data management system. For maximum security, the computer hosting this study's database is not connected to the Internet. The database-serving computer is accessible by only one other computer, namely the computer hosting the web-application, which is online.

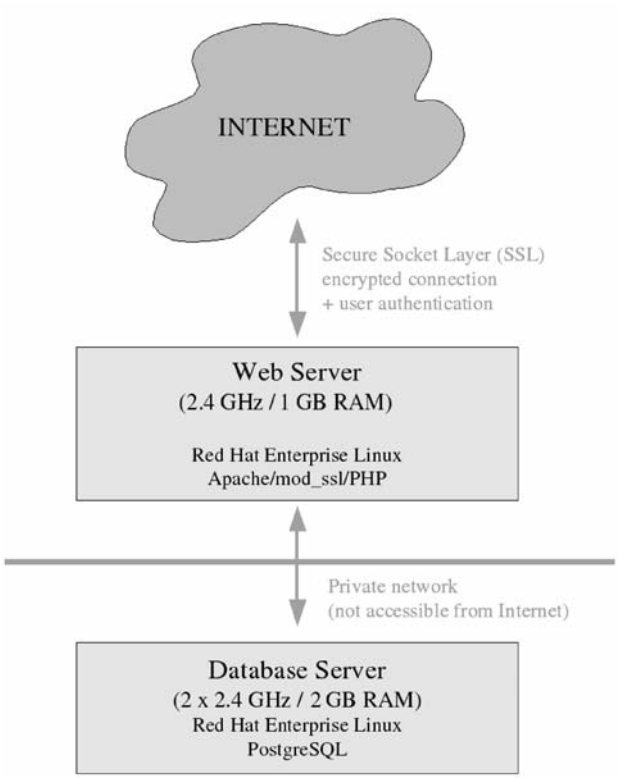

Figure 1

Both of these two Linux-based computers have only a single user account in their operating systems, and only this administrator will ever be able to login to them directly. (Of course, internally the database does have additional password-protected accounts, which will be discussed below.) All system files are updated weekly to their latest versions to be sure that all emerging security holes have been patched. This design secures the base operating systems.

Users of this system will connect to the web-application via the Internet using their preferred web browser. Access is secured in two ways. First, at the infrastructure level, all communication is strongly encrypted using Secure Socket Layer (SSL) technology between the user's web browser and the web-application. As part of this security system, the user's web browser will automatically authenticate our site by verifying the SSL certificate we carry from GeoTrust. Second, at the user level, each user must provide proper authentication in the form of a username and password.

The web-application resides on a server housed at the University of California, Los Angeles. This web server is dedicated solely to hosting security-sensitive applications. There are no public, or general purpose, web sites hosted by this server. There is only a single user account on the system. The operating system employed is Red Hat Enterprise Linux version 3. The Apache Foundation's Apache web server software, configured with the mod_ssl security module, mediates the web connection. The web-application is written in PHP, a widely ⏩

used web-oriented programming language.

Although the web-application resides on an Internet-accessible server, all patient data is stored in the separate database server that is not directly connected to the Internet. This database server resides on a private, non-routable network also running on Red Hat Enterprise Linux version 3. PostgreSQL, an enterprise-level relational database management system, is the software used to store and access data from the study. (PostgreSQL is roughly the open-source analog of Oracle or Microsoft SQL Server. Using PostgreSQL provides an efficient and stable database, with a significant saving on expense of software, hardware and personnel.)

The web server is a Dell model 1750, with a 2.4 GHz Xeon CPU, 1 GB RAM, and 80 GB storage. The database server is a Dell model 2650, with dual 2.4 GHz Xeon CPUs, 2 GB RAM, and 400 GB storage. If needed, there is ample room for expansion of these systems. These computers are secured at UCLA in a locked, off-master room used solely to house computer servers. The room is equipped with both large power converters for clean, uninterruptible power, and a large, dedicated cooling system. The entire system is on a regular tape back-up cycle. Every night an incremental back-up is recorded to a RAID storage system on a separate computing system dedicated to holding back-ups for rapid retrieval. Each weekend a complete image of both computers is recorded to a single tape (each tape can hold 400 GB of uncompressed data). The tape is stored in a fire-proof safe, and the previous week's tape is stored off campus. After four weeks, a tape is erased and put back into the pool of available tapes.

Using the Application

For ease of use, the Application is designed to take advantage of web interface standards as much as possible. Figure 2 shows an example screen of the National Ataxia Database Web Application. When the user first accesses the application, they are presented with a login screen requesting their username and password. Once successfully logged in, their "home" page details any news of relevance to the application and provides a detailed list of links and descriptions of the various forms available to that user.

Every group participating in the national consortium will be able to designate an administrator-level user or users who can create studies; create user accounts; assign user privileges; design and enter forms; create patients and traits; and assign forms, traits, patients, and users to studies. Hard-copy case report forms can be closely reproduced in the online version to streamline data entry. Standard web controls (text inputs, check boxes, radio buttons, etc.) are used; no external requirements are made so that users will not have to install any special software on their computers to use the application.

Figure 2

To help ensure error-free data entry, the system uses multiple levels of data checking. The first level takes place in the user's browser when a form containing data is submitted. Any obvious errors (missing values, out-of-range values, etc.) are flagged and the user is notified of problems in particular fields of the form. Once all of these errors have been rectified, the data is then submitted to the web server, which also validates the data formats. Next, the database server performs its own checks when it receives the data for storage. Finally, we support and encourage double

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Architecture of the National Database
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data entry, in which two or more different users enter data in independent sessions. The database stores all sessions and reports any discrepancies, which must be cleared in a separate session, before the data transaction is finalized.

Each patient data item is assigned to a specific subset of the studies participating in the national consortium. This subset may include all studies, a few studies, or a single study. Entire forms or individual questions and traits collected by one study can easily be shared with other studies and groups. Each user of the database will similarly be assigned to a subset of studies. Only users assigned to a particular study will have access to the data items assigned to that study. Furthermore, access may be limited to specific operations, for example, the ability to read data, write data, edit data, lock data, and generate reports. These levels of access will be specified per user and per data item.

Testing

The application has been in testing by in-house users for six months. A test project of real data has been created. Data entry technicians have entered approximately 92 patients on six forms taken at three time-points. All bugs have been logged and corrected, and enhancements for improving ease-of-use have been identified and added. The application will be ready for initial release for beta testing by ataxia researchers by spring 2007.

Future Additions

We are in the process of creating a system of simple, on-line versions of the forms that, when appropriate, will allow patients to fill out forms on their own browser and enter them directly into the National Ataxia Database. ❖

Understanding Spinocerebellar Ataxia Type 1

Puneet Opal, MD, PhD
Northwestern University

The following is a research summary of a grant funded by NAF for fiscal year 2006.

A growing number of genetic neurodegenerative conditions share a common pathological feature: the presence of a mutant protein containing an expanded glutamine tract. Spinocerebellar ataxia type-1 (SCA1) is one such disease, in which a glutamine repeat expansion in protein ataxin-1 causes progressive cerebellar and brainstem degeneration. Although enormous progress has been made in understanding SCA1 pathology, two fundamental questions remain unanswered: What are the subcellular pathways involved in toxicity? Why does pathology localize to select populations of neurons, despite widespread expression of the mutant protein? These issues are relevant not only to SCA1, but also to the other polyglutamine diseases and for that matter to other degenerative syndromes, such as Alzheimer's and Parkinson's.

We postulate that a fruitful approach to answering these questions would be to identify and analyze the proteins that interact with disease-causing proteins in the most vulnerable cell populations. We are currently studying one such protein – the leucine-rich acidic nuclear ⏩

protein (LANP) that stands as a compelling mediator of mutant ataxin-1 toxicity: LANP was picked up in a genome-wide screen for identifying proteins that bind ataxin-1; indeed LANP has a greater affinity for mutant ataxin-1 than wild type ataxin-1. Moreover, LANP is expressed at high levels in cerebellar Purkinje cells, the cell most affected in SCA1 and functions as part of a tightly regulated complex that inhibits histone acetylation and silences the transcription of genes. Finally, we have found that ataxin-1 and LANP functionally interact, with the preponderance of preliminary data suggesting that LANP is recruited by ataxin-1 to cause persistent hypoacetylation at promoters of genes resulting in transcriptional aberrations. It is tempting, therefore, to link the ataxin-1/LANP interaction to the aberrations in the gene regulation characteristic of SCA1.

We recently completed a project to probe the neuronal functions of LANP. To study the effects of depleting LANP on the neuronal phenotype, we used the well-characterized PC12 neuronal cell line. This cell line, derived from rat adrenal pheochromocytoma, can be induced to differentiate to take on many of the morphological and functional properties of neurons. These cells have been used in the past by researchers to study neuronal functions because they divided in culture and can be easily maintained. Yet, when Nerve Growth Factor is added they can be induced to differentiate into neurons.

We took advantage of a relatively novel technique called RNA interference to deplete LANP. This is a technique that uses small interfering RNA duplexes to target and shut down expression of genes that these duplexes are targeted against. Using duplexes targeting LANP, we have successfully depleted LANP in PC12 cell lines by more than 75% as ascertained by quantitative densitometry of Western blots (a way to estimate protein expression). We have found that when LANP is knocked down by RNAi duplexes targeting LANP the number of cells expressing neurites approximately doubles. We found that depleting LANP promotes neurite outgrowth. Importantly, we found that LANP depleted PC12 cells could rescue the poor neurite outgrowth induced by mutant ataxin-1. These findings suggest the tantalizing possibility that depleting LANP in the context of SCA1 might prove therapeutic, or at least ameliorate the neuritic pathology characteristic of the disease.

We postulated that LANP regulates the transcription of critical genes involved in neurite outgrowth and maintenance. To test this idea, we performed an analysis of the gene expression pattern of rat PC12 cells when LANP is depleted. We performed a microarray analysis using gene microchips with oligonucleotides to the rat genome spotted on them. We have successfully identified approximately 100 genes that are misregulated only when LANP is depleted. We have also genetically engineered mice that lack LANP. These mice are relatively normal. However, we have found that neurons from these mice, much like PC12 cells seem to have an exaggerated neurite outgrowth compared to those derived from their wild-type littermates. Currently, we are testing if any of these genes are also misregulated in LANP null mice. We are also testing our prediction that genes up-regulated in LANP-depleted cells are up-regulated in SCA1 knock-in mice.

If LANP does prove to be a genetic modulator of the SCA1 phenotype, then these studies immediately open up possibilities for therapeutic intervention, translating our basic science work to the clinical arena. For instance, one could envision screening for small molecules that could modulate LANP function so as to rectify transcriptional aberrations in SCA1, and thus prevent disease onset and/or progression.

Finally, we should like to thank the NAF for their generous support. None of this work would have been possible without these funds. We hope that our work will inspire translational approaches to help patients suffering from ataxia and other degenerative disorders. ❖

'Cerebellar Atrophy' Means I Have Ataxia

By Tiffinay A. Compiano

"I am just one person helping to raise funds here in Iowa to battle this thing called ataxia. In an effort to document my experience with ataxia, I've written a personal memoir." – Tiffinay A. Compiano

Editor's note: Tiffinay's memoir has been edited for this issue of Generations.

Whenever someone asked me to describe my "dizzy" feeling, I would tell them that choosing a greeting card left me with a spinning sensation. Speaking to someone who was seated when I was standing also made me nauseous. I felt like I had a ball filled with liquid inside my head. I had to keep that ball balanced or I'd feel like vomiting. I visited a neurologist in 1987 who diagnosed me with positional vertigo.

Years passed. I had a career, a marriage to a wonderful man, R.J., a home and had given birth to my first son, Ralphy. My second son, Peter, was born one year, 11 months and 29 days later when I was 30 years old.

We received an invitation to a wedding in Kansas City. While there, R.J., Ralphy, and I went to an amusement park. The first ride we encountered was a loop-de-loop roller coaster. After surviving the ride, I had to be helped out of the passenger car. My world was spinning so fast. I wobbled to the nearest umbrella table, vomited violently, and remained sitting on the bench with my head in my arms for the next two hours. Our weekend vacation ended and I returned to work. Still feeling queasy and light-headed I called Mom for advice.

"You'd better get an MRI, honey."

At age 30, an MRI was the piece that would solve the puzzle.

"Hello, Tiffinay. This is your doctor. I'm calling with the results of your MRI."

No "How are you?" or "I'm fine" or any other small talk. Strange, I thought.

"The results showed some cerebellar atrophy."

Pretty sure of what "atrophy" meant. I deduced that my cerebellum must be wasting away. He spoke a little more but I didn't hear anything. I felt the crush of the news on my shoulders.

"Well, Doctor, I know this might sound crazy but, is this *normal*? Like a normal part of aging?" I asked like a small child lost in a forest.

"No. You may want a referral to see a neurologist," he said with calm professionalism.

The phone slipped out of my hand and onto the floor. Twenty years ago the neurologist said I would never have my Dad's disease. Now the doctor wants me to go back? I recovered the phone, said thank you, and hung up.

As if by reflex, I called my mother who'd recognized some of my symptoms. She should know. For more than 30 years she has been my father's caregiver. In the final analysis, she and I assumed that cerebellar atrophy meant that I have ataxia. I made her promise not to tell anyone until I "owned" the idea.

Just as Elisabeth Kubler-Ross defines the stages of death, my first reaction was denial. I told concerned friends that I didn't "own" the idea yet so I didn't want to talk about it. It made me sick to my stomach. With time, I did come to "own" the idea. The knowledge from what I was learning from research made me realize that some of the things that I thought belonged to me, such as the extra sleepiness, my legs tiring quickly and my penmanship getting worse, really belonged to the ataxia. ▸▸

Eventually, I made an appointment with a neurologist at the Mayo Clinic in Rochester. Between the time of my diagnosis and April 30th many changes took place in my life. The biggest was quitting my job. I loved social work. My job gave me a meaning that I will forever miss.

My visit at Mayo consisted of three main components. First, he took my vitals: temperature, blood pressure, and pulse. All normal. Second, he asked me to name the President of the United States. He asked if I knew the year and the day. He handed me a piece of paper and had me sign my name a few times. After this mini-mental exam came the third part, a physical examination of my speech, eyes, reflexes and gait. What a happy birthday!

Then came the diagnosis: I have an undetermined form of spinocerebellar ataxia just like Dad. Tears fell from my eyes. Okay. What in God's name do I do now besides pray? Am I going to be able to raise my children? How am I going to keep working? Will we have to move if I can't earn money? How will R.J. handle this? Will it be too much for him? How will my children make it through this? How will my parents feel?

Research on the internet into ataxia proved insightful and provided some answers. R.J. and I found a national organization, the National Ataxia Foundation, which led us to participating on bulletin boards. This is where I learned of the most powerful tool I have in my box: knowledge about how to better cope and deal with my ataxia. I have since become a member of this organization. I enjoy getting the publication, *Generations*. It is wonderful to know that there is so much positive work happening and many great minds involved. It's comforting to know that you're not alone.

Following our Internet research we decided to tour the nation in order to learn as much as we could. After going to Mayo Clinic, we saw a doctor at the University of Texas in Galveston. Next we had an appointment at UCLA in Westwood, CA. Finally we met a fourth specialist in the field of ataxia at the University of Iowa. All gave us the same diagnosis and same suggestions. "We could do genetic testing to find out which gene is causing the ataxia. However, if we find it, we still can't cure it. Go home, get rest, watch your diet and get exercise."

My brother and two friends and my husband started the Talarico Ataxia Foundation. It would be a community-wide effort at raising funds for this devastating and totally disabling disease. A heaven-sent group of family and friends work to make sure our ataxia fundraising event is a huge success. Each year we host over 100 golfers at an 18-hole tournament. Afterwards, an authentic Italian dinner is served. Following dinner is a live auction hosted by celebrity comedian and Des Moines native, Willie Farrell. Willie continues to donate his time and talent to us in the name of love for our family and promoting research. The money raised, which over the years has totaled nearly $300,000, goes to the National Ataxia Foundation to support promising SCA research. In July 2007 we hosted our sixth annual event to help support the important work of NAF. To make a contribution or to learn more about future golf tournament fundraisers, contact our group at Talarico Ataxia Foundation, 5335 Merle Hay Road, PMB 300, Johnston, IA 50131.

I live a functional and pretty great life; a little slow but wonderful. I trip and stumble constantly. Falling will come next; I know this from watching Dad. My leg pain is working its way into numbness. People ask me to repeat most of what I say because they can't understand me. My handwriting is barely legible on page one and illegible by page two. The disease will continue but I won't put my life on hold until there is a cure.

Although I've recently come to the realization that I may be trapped in this body, I keep my chin up and try to support others in their quest

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Cerebellar Atrophy Means I Have Ataxia
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to live a full life. I believe that what comes around goes around. The symptoms of ataxia seem unsubstantial when I'm at a school recital or a soccer game enjoying my children. My family keeps me going.

A dark curtain falls when I think about what has happened to my Dad. This painful disease has taken away a normal existence and left him in a hospital bed, reliant on others to even move.

I have hope. To date there have been up to 29 different forms of ataxia identified. The National Ataxia Foundation has funded, in part through funds from the Talarico fundraiser, a genetic study at the University of Iowa to help identify the "Talarico" gene for our family and others who are affected by this form of ataxia.

A new friend of mine with ataxia, also hosts a fundraiser for the National Ataxia Foundation in Chicago and "will not stop until there is a cure." In her words, "We will be the last generation to suffer with ataxia."

My fondest wish is to keep the *joie de vivre* and positive attitude my father has shown me. He is truly my hero. I hope to instill this attitude in my children by providing for their needs, modeling a Christian life style, building their faith, encouraging them to dream, and fostering development towards those dreams. ❖

Giving Options

By Kenneth B. Vonderharr, CPA

As 2007 winds down, it may be time to consider some options you still have in regards to your charitable giving before the year is over. As with all financial concerns, you should contact your personal CPA or tax preparer because everyone's situation is different and the various planning items may not apply to your situation or even be available for you.

The first item is only available until the end of 2007 and at this time is not scheduled to be around in 2008. A person over 70-1/2 years old can distribute up to $100,000 directly to a qualified charitable organization and not include the distribution in gross income. The distribution does count towards your required minimum distribution. This provision applies to IRA's but not to SIMPLE IRA's or SEP's. No charitable deduction is allowed for the amounts since the distribution is not included in income. Another side benefit is the amount is then removed from the person's estate, without the usual income in respect to a decedent being reported to heirs.

Another less commonly used provision for donations is the giving of appreciated property, usually stocks. What works the best are stocks that are traded on an exchange. It is very easy to determine market value on a specific date when donating such stocks. The donor gets to itemize the donation at the fair market value on the date of transfer, and does not have to report any gain from what the stock was purchased for to the market value when donated. If an individual is in the habit of substantial giving, this opportunity can maximize the person's donation impact and not require a cash outlay.

You are limited to 50% of your adjusted gross income for charitable donations to qualified charities, but even if you exceed this limit, you are allowed to carry the excess over to the following five years, so you can donate a large block in one year, if you choose, and be able to get the deduction in subsequent tax years.

These are some of your options in addition to the "write a check donation" you are already familiar with and, as stated earlier, may or may not be applicable to you and your situation. ❖

Kyle Bryant Translational Research Award

Given to Australian/UK Collaboration

The $100,000 Kyle Bryant Award was given to Australian researchers and their British collaborators for translational research into the catalytic antioxidant CTMIO as a possible treatment for Friedreich's ataxia (FRDA).

The research proposal was submitted by Dr. Nuri Gueven at Queensland Institute of Medical Research in Australia. The title of the proposal is "Use of a novel catalytic antioxidant, CTMIO, in a GAA repeat mouse model of FRDA." The mouse model and application of CTMIO to the mice will be provided by the London team of Dr. Mark Pook while the analysis will be done in Dr. Gueven's lab in Australia.

CTMIO is a catalytic nitroxide antioxidant that has been found to correct some neurobehavioral function in a mouse model of ataxia-telangiectasia. The overall goal of this project is to determine if CTMIO can alleviate pathological changes in the GAA repeat mouse model of FRDA. If so, this class of compounds could become a promising therapeutic approach for FRDA. This type of antioxidant is different structurally and mechanistically from the other antioxidants (Idebenone, CoQ10, and Vitamin E) that are being studied in FRDA clinical trials.

Dr. Nuri Gueven

Dr. Nuri Gueven studied at and received a PhD from University of Konstanz in Germany. His fields of study were Cell Culture and Molecular Biology and Signal Transduction and Radiation Biology. He is currently a Senior Research Officer at the Radiation Biology and Oncology Laboratory at The Queensland Institute of Medical Research.

Dr. Mark Pook

Dr. Mark Pook received a BSc degree from the University of Leeds and a PhD from the University of Manchester. He is currently a lecturer and the Head of the Ataxia Research Group at Brunel University in Uxbridge, United Kingdom.

This research project was selected from 10 excellent applications that were peer-reviewed and then ranked by Friedreich's Ataxia Research Alliance and National Ataxia Foundation scientific advisors. The award was established in honor of Kyle Bryant, the courageous young man who has Friedreich's ataxia and pedaled his cycle from La Jolla, CA to the annual NAF meeting in Memphis earlier this year, increasing awareness of Friedreich's ataxia and raising funds to support the research into treatments and a cure. Bryant raised $40,000 on his cross-country "Ride Ataxia," visiting FRDA researchers and patient families along his route. NAF and FARA announced at the end of his ride that the two organizations would add sufficient funds to bring the total of the Kyle Bryant Research Award to $100,000.

Executive Director Michael Parent commented, "The National Ataxia Foundation is truly grateful to Kyle Bryant for his courageous journey to help raise ataxia awareness and needed funds to support promising Friedreich's ataxia research. Kyle's initiative has made it possible to fund this exciting research study." ❖

Deranged Calcium Signaling in SCA3 Neurons

Ilya Bezprozvanny, PhD

University of Texas Southwestern Medical Center at Dallas

The following is a research summary of a grant funded by NAF for fiscal year 2006.

During the last two years, with the support of an NAF grant, we focused on the analysis of connections between calcium signaling and degeneration of SCA3 neurons. This project is based on our discovery of association between mutated ataxin-3 and type 1 inositol 1, 4, 5 trisphosphate receptor (InsP3R1), which is an intracellular calcium (Ca^{2+}) release channel. In biochemical experiments we demonstrated that mutant ataxin-3 binds to InsP3R1. In functional experiments we demonstrated that mutant ataxin-3 makes InsP3R1 more active. Importantly, wild type ataxin-3 (without CAG expansion) does not bind to InsP3R1 and does not activate it. Obtained results provided strong support to our hypothesis that excessive Ca^{2+} signaling may be a cause of neuronal death in SCA3 neurons.

To test this idea, we cultured neurons from either pontine nuclei or substantia nigra, which are the most affected brain parts of SCA3/MJD patients, using SCA3-YAC-84Q transgenic mouse model. Since the culture density of pontine neurons was too low and neurons from substantia nigra were not homogenously dopaminergic, we could not further set up the "in vitro model," which is to measure the deranged calcium release by calcium imaging technique and to quantify the apoptosis of neurons by TUNEL staining. To avoid this issue, we performed the experiments at whole animal level. We fed SCA3-YAC-84Q mice with dantrolene, a clinically relevant stabilizer of intracellular calcium signaling at the dosage of 5mg/kg twice a week. By the behavioral assays of beam walking and footprint, we found that dantrolene significantly improved the performance of SCA3-YAC-84Q mice. The neuro-anatomical analysis revealed that SCA3- YAC-84Q mice fed with dantrolene had higher brain weights than control SCA3 and wild type mice. Our results indicate that deranged calcium signaling may play an important role in SCA3 pathology and calcium stabilizers such as dantrolene can prevent or slow-down the progress of SCA3, thus may be considered as potential therapeutic drugs for treatment of SCA3/MJD1 patients.

At present, we are doing the stereologically neuronal countings of pontine nuclei and substantia nigra to evaluate the neuroprotective effect of dantrolene. These studies are supported by a renewed grant from NAF. We were able to obtain additional support for SCA3 project from Ataxia MJD research project. In addition, the R01 grant has been submitted to National Institute of Health to provide further support for our work on SCA3.

I am truly thankful to the Foundation for continuous support of our research program on causes and potential treatments of SCA3. ❖

Thank You

The National Ataxia Foundation wishes to express our sincerest thank you to the NAF chapter presidents and officers, support group leaders, and ambassadors for their continued hard work and commitment in helping local ataxia families.

Your tireless efforts bring help, hope, and community to local ataxia families. Thank you!

Function of the Multifunctional Protein BAG1 in SCA3 Pathology

By Pawel Kermer, MD

University of Goettingen, Germany

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Our recent data identified BAG1 as highly neuroprotective gene accelerating neuronal differentiation. BAG1 over-expression leads to an induction of neuronal chaperone foldase activity in situ, provided e.g. by heat shock protein 70 (Hsp70), with neuroprotective effects depending on BAG1/Hsp70 interaction. Hsp70 or Hsc70 over-expression has also been shown to suppress neuropathology in mouse models of polyglutamine repeat (polyQ) diseases, including spinocerebellar ataxia.

Hence, with the funding provided by the National Ataxia Foundation, we started to further characterize neuronal BAG1 function in vitro by transfer of our findings to models of spinocerebellar ataxia 3 (SCA3) with a focus on (1) cell death, (2) proteasomal/ chaperone function, and (3) inclusion body formation.

To this end, we employed rat and human neuronal cell lines (CSM 14.1 and SHSY-5Y) for transient and stable transfection assays with tagged SCA3 and polyQ-SCA3. While polyQ-SCA3 decreased cell viability within 48 hours by more than 30 percent when compared to wild-type SCA3 in our study, it did not increase cellular susceptibility to other toxic stimuli like staurosporine. In line with the latter finding, mitochondrial fission and fusion as well as mobility presenting a hallmark of mitochondria-dependent neuronal apoptosis remained unchanged in the presence of wild-type or mutant SCA3. With regard to proteasomal function in our cellular model, polyQ-SCA3 inhibited substrate degradation activity, but only for cleavage next to hydrophobic amino acids (chymotrypsin-like activity), while the other two peptidase activities (caspase-like activity and trypsin-like activity) remained unchanged. As reported before, polyQ-SCA3 forms cytosolic and nuclear inclusions. To study and quantify inclusion body formation, we not only relied on immunocytochemical methods but also established a filter assay for cellular polyQ-SCA3 aggregates allowing blotting and densitometric quantification.

Having established the tools, we are currently investigating the effects of our co-chaperone BAG1. Unfortunately, our preliminary results indicate that SCA3 toxicity could not be prevented by the presence of BAG1 in our cells. Moreover, BAG1 seems to increase the amount of cellular SCA3 aggregates. Experiments aiming to uncover the activities of cellular detoxification mechanisms, namely the chaperone and proteasome system, employing fluorescent biosensors and FLIM techniques are on their way.

The results summarized here have in part been published at the German Neuroscience Conference (Liman et al., Abstr.-No. T4-2A) and will be presented as poster contribution on the Annual Meeting of the Society for Neuroscience 2007 in San Diego.

Based on these results and with the NAF support we successfully obtained a two-year research grant including personnel (PhD position) and consumables by the Fritz Thyssen-Foundation. ❖

Matching Gifts

Many employers will match your gift to NAF. Please ask your employer if they have a Matching Gifts Program.

National Ataxia Foundation
51st Annual Membership Meeting

"Blazing a Trail in Research"

The Flamingo Las Vegas Hotel — March 28-30, 2008

The National Ataxia Foundation Board of Directors and the National Ataxia Foundation Phoenix and Tucson Arizona Ataxia Support Groups would like to welcome you to the 51st Annual Membership Meeting. Please join us in exciting Las Vegas, NV to learn, share, network, have fun, and enjoy the sights.

The 2008 NAF Annual Membership Meeting will continue to focus on bringing together NAF members and their families to meet and learn from world leading ataxia researchers and neurologists but also to build new friendships and reunite with old friends.

Whether this is your first meeting or your 51st, the 2008 Annual Membership Meeting will be filled with education, celebration, sharing, and caring!

Registration forms will be available in the 2007/2008 Winter Issue of *Generations* and on our website at the end of December 2007. You may also view the latest information available about the Annual Membership Meeting on our website at *www.ataxia.org*. The Annual Membership Meeting Program that you will receive at the meeting will include the most updated information. Please use your Meeting Program for meeting room assignments and times.

Program Overview

Thursday, March 27

Leadership Meeting – Current Chapter Presidents, Support Group Leaders and Ambassadors are encouraged to attend the Leadership Meeting. Due to limited space, only those who are Chapter Presidents, Support Group Leaders, or Ambassadors will be permitted to attend unless one representative has been indicated prior to the meeting to attend in place of the Leader who is unable to attend. This meeting will address questions and concerns that are unique to those in these appreciated positions as well as offer the opportunity to meet one another and learn some tips from both peers and professionals. This meeting will run from 1-5:00 p.m. If you are not currently a group leader, but are interested in becoming one, please inquire ahead of time by contacting Lori at *lori@ataxia.org*.

Internet Group – This is your opportunity to meet some of the internet friends you have met on the NAF chat room, NAF Bulletin Board, Internaf, Tricks of the Trade, Ataxia Forum, Ataxia Chat 2002, FAPG, and u_r_notalone.

Friday, March 28

Teen Program and Activities – Teens ages 13-20 are invited to meet in the Reno II Room. This room is reserved for the teens to get to know each other, play games, watch movies and relax. In addition there will be scheduled group activities and guest speakers. Please make sure to mark "TEEN" on your registration form so we can plan accordingly. A full schedule will be available in the Meeting Program. Don't forget, teens are also welcome to attend the regular meeting program.

Breakout Sessions – Friday morning 45-minute sessions will be available on various topics. ▸▸

Birds of a Feather – Friday from 2-5 p.m., groups will be sectioned off in individual or divided rooms based on your type of ataxia, whether you are a caregiver or parent, etc. This is a tremendous opportunity for you to meet others with your type of ataxia or who share in a similar situation and make friends that will last a lifetime. Medical professionals will also be on hand, circulating between groups, in case you have any questions.

Friday Night Reception – Please join us for a casual hors d'oeuvres reception in the Sunset Ballroom. All registered meeting attendees are welcome to attend and admittance to this event is included with your registration.

Saturday, March 29

General Sessions – On Saturday morning and afternoon there will be General Sessions in the Sunset Ballroom. General Sessions are large group presentations, typically with a medical or research focus. Many of the world's leading ataxia researchers and clinicians, along with other ataxia experts, will be presenting the latest research and additional information. A half-hour Question and Answer session will follow the morning and afternoon General Sessions with a panel of the doctors and researchers who presented.

Church Services – Both Catholic and non-denominational church services will be held on Saturday at 6:00 p.m.

Silent Auction – As a tradition at our annual gathering, the silent auction will be begin on Saturday afternoon and conclude at 7:30 p.m. We hope you will bring an auction item with you to the meeting and get the winning bid on an item you choose. Auction items range from something that represents your state or country, such as a painting, spices, statues, sports memorabilia or basket of knickknacks, to a hotel stay or weekend getaway. Good luck everyone!

Saturday Evening Banquet – The Saturday Evening Banquet will begin at 7:00 p.m. in the Sunset Ballroom. Please get your tickets – which are included in your registration fee – ahead of time. You must reserve seating for the banquet in advance. The banquet will include a plated dinner.

Sunday, March 30

General Sessions – Sunday morning wraps up the 2008 Annual Membership Meeting with the final round of General Sessions at 8:30am in the Sunset Ballroom followed by a Question and Answer Session.

About Las Vegas

Las Vegas, an ever-changing fantasy land of a city, has seen unbelievable expansion since it emerged from the desert more than 100 years ago. The sights and sounds of Las Vegas are enjoyed by over 38.9 million visitors every year.

Please visit *www.visitlasvegas.com* for a complete list of attractions and planning information.

Continued on page 28

WANTED: Exhibitors for the NAF Annual Membership Meeting

Have you used a product or service that has been helpful as it relates to ataxia? NAF is currently exploring various vendors to be exhibitors at the Annual Membership Meeting in Las Vegas in March 2008.

If you have found a product or service beneficial in your daily life, please forward the name of the company to the NAF office and we will contact them with the details to become a possible exhibitor. Please call (763) 553-0020 or e-mail *susan@ataxia.org*.

Thank you for your help.

Blazing a Trail in Research
Continued from page 27

About Flamingo Las Vegas

The Flamingo Las Vegas, located on the Vegas Strip and minutes from the McCarran International Airport, is the official conference hotel of the 2008 NAF Annual Membership Meeting.

Guests of the Flamingo can enjoy 15 acres of Caribbean-style foliage which includes an extravagant Wildlife Habitat and five separate pools, with cascading waterfalls for swimming and sunbathing. Guests can also shop at the more than 10 shops, relax at the Flamingo Spa & Salon and dine at the eight various food venues available. Guests of the Flamingo always have a full house when it comes to entertainment, from superstar Toni Braxton to George Wallace.

The Flamingo Hotel is a very large hotel and casino. Please allow yourself time to familiarize yourself with the layout of the hotel. Valet parking is available at the hotel's east entrance. Self-parking is available in the South Garage, located immediately east of the main entrance. Both are complimentary. Please let NAF know if you are driving a van with a lift.

Reservations – Guest rooms are available for a special group rate of $124 per night. Please be sure to make your reservations by February 22, 2008 in order to secure the special group rate. To book your stay online go to: *www.harrahs.com/CheckGroupAvailability.do?propCode=FLV&groupCode=SFNAT8* or if you prefer to make your reservation by phone, please call toll-free 1-800-732-2111 and ask for the National Ataxia Foundation group rate.

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

Transportation

NAF is not responsible for transportation to and from the hotel. The following may be used as a helpful guide for your convenience.

Lift-Equipped Shuttles – Lift-equipped shuttles are available at the McCarran International Airport to the Flamingo Hotel. The cost is approximately $7 per person one way and $14 per person round trip. Pick-up areas are located on the north and west sides of the baggage claim area outside exit doors 8-14. The shuttle is not owned or operated by the Flamingo Hotel or Harrah's Properties.

Taxis – All taxi companies in Las Vegas have lift-equipped vans accommodating one wheelchair. Ask in advance for an accessible taxi van.

Strip Trolley – The trolley operates on the Strip and pulls up to the entrance of each hotel on its route. Most are lift-equipped. For details call (702) 382-1404.

CAT (Citizens Area Transit) – Bus service is fully accessible, including buses that are lift-equipped. Reduced fares are available for persons with disabilities. Call (702) 228-7433.

Paratransit – If you are certified to ride paratransit, bring your certification and you will be allowed to ride for up to 21 days without a Nevada certification. Call (702) 455-0997 one to three days in advance to schedule rides.

Parking – If you have a parking permit from your home city for your car's dashboard and you will be renting a vehicle in Las Vegas, bring ▸▸

Going Once, Going Twice, Sold!

Let's make the 2008 National Ataxia Foundation Annual Membership Meeting's Silent Auction the biggest yet!

Proceeds from the silent auction benefit those with ataxia and their families, so get your items ready to go to Vegas!

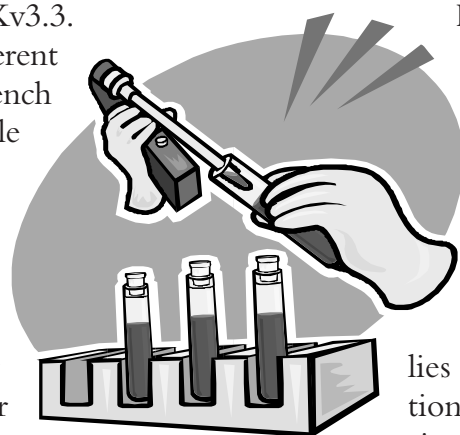
Mutation Analysis of the KCNC3 Voltage Gate Potassium Channel in Sporadic and Familial Ataxias

Stefan M. Pulst, MD

Cedars-Sinai Medical Center

The following is a research summary of a grant funded by NAF for fiscal year 2006.

The gene causing SCA13 was recently discovered. It codes for a voltage-gated potassium channel designated KCNC3 or Kv3.3. We initially discovered two different mutations in a Filipino and a French family, each changing a single amino acid in the channel protein.



With funding from NAF, we examined a large number of DNA samples from sporadic and familial ataxia patients for mutations in this channel. After screening more than 500 families, we identified that the mutation initially identified in the Filipino family is recurrent and was observed in three additional families of French and German origin. We also discovered a new mutation in two French pedigrees. Although all SCA13 patients have ataxia, the age of onset and rate of progression vary considerably depending on the mutation. We are now examining the effect of these mutations on channel function in cultured cells.

The importance of these studies lies in the identification of additional genetic causes of ataxia and point to the importance of voltage-gated channels for functioning and survival of Purkinje neurons. ❖

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it with you. You may also make advance arrangements for a free 90-day temporary disabled parking permit through the City of Las Vegas. Write to City Hall, Parking Permit Office, 400 E. Stewart, Las Vegas, NV 89101, (702) 229-6431. Send a doctor's letter explaining the condition and the duration of the condition, and allow two to three weeks for a response. Valet parking is also an option at nearly every hotel.

Vegas Strip – With all of these transportation options, you will still cover a lot of the city without transportation. That said, the length of the Strip can be deceptive... it takes more time than you would think to get from one end to the other. Be sure to wear comfortable shoes and carry a bottle of water. See you in Las Vegas! ❖

Volunteers Needed

Volunteers donating their time contribute greatly to the success of each NAF Annual Membership Meeting.

To volunteer at the 2008 AMM, please contact Rita Garcia, Phoenix Area Support Group Leader, at (480) 726-3579 or e-mail *rtg22@cox.net*.

Nurse(s) Needed

One or more registered nurses are needed onsite for the duration of the NAF 2008 Annual Membership Meeting in Las Vegas, NV, March 27-30, 2008.

To volunteer or for more information, please contact Lori at *lori@ataxia.org* or call (763) 553-0020.

LAS VEGAS

SERVICES & RESOURCES

Personal Care Attendants

Alliant Healthcare Service
1050 E. Flamingo Rd.
Suite W-253
Las Vegas, NV 89119
(702) 733-1599
Fax: (702) 733-9190

Comfort Keepers
701 N. Green Valley Pkwy., Suite 200
Henderson, NV 89074
(702) 360-7475
Fax: (702) 990-3156

Nurse Core
4423 W. Flamingo Rd.
Las Vegas, NV 89103
(702) 458-1137

Wheelchair & Scooter Rentals

Ability Center
6001 S. Decatur Blvd, Suite N
Las Vegas, NV 89118
(702) 434-3030
1-800-546-7622

Active Mobility
4625 S. Procyon St., Suite 202
Las Vegas, NV 89103
(702) 736-4399
(800) 877-6106
Fax: (702) 736-5900

Desert Medical Equipment
5030 S. Decatur Blvd.
Las Vegas, NV 89118
(702) 876-9171
Fax: (702) 876-908

Encore Productions
(in-house supplier)
Located in the Las Vegas Convention Center
3150 Paradise Rd.
Las Vegas, NV 89109
(702) 943-6780

Freedom Medical Supply & Equipment
1725 E. Warm Springs Rd. #10
Las Vegas, NV 89119
(702) 386-9997
Fax: (702) 228-9996

Mesa Medical Equipment
5225 S. Valley View Blvd. #10
Las Vegas, NV 89118
(702) 367-0737
1-800-852-3006
Fax: (702) 895-9347

Scootaround Inc.
3904 Vanessa Dr.
Las Vegas, NV 89103
(888) 441-7575
Fax: (204) 478-1172

Scooters4less Inc.
224 Bailey Island Dr.
Henderson, NV 89074
(702) 436-2240
Fax: (702) 436-2241

Total Home Care
5321 Cameron
Las Vegas, NV 89118
(702) 796-1016

Village East Drugs
(wheelchairs only)
5025 S. Eastern
Las Vegas, NV 89119
(702) 736-7018

ADA Assistance Offices

ADA Business Connection
Governor's Committee on the Employment of People with Disabilities
Suzanne Thomas
Voice: (702) 486-4318
TTY: (702) 486-4393

Las Vegas Convention and Visitors Authority ADA Coordinator
(702) 892-0711
Nevada Relay Service
Voice: (800) 326-6888
TTY: (800) 326-6868

Funding for Travel Grants Needed

Each year the National Ataxia Foundation offers a limited number of travel grants to help persons with ataxia who have financial restraints attend the NAF Annual Membership Meeting. These travel grants provide persons with ataxia the opportunity to attend these important conferences to learn the latest information on ataxia research, attend various break-out sessions on topics relating to ataxia, participate in "Birds of a Feather" sessions, and perhaps most importantly, meet people who share the same issues and concerns.

Over the years these travel grants have been made possible through the generosity of individual donors and families who know the value of these meetings. Please help others attend the 2008 NAF Annual Membership Meeting by making a tax-deductible donation today. You may go on-line to make your gift or you can mail your donations to NAF and write on your check memo, "Travel Grant."

We are deeply grateful to those who have given in the past for this fund, their gifts have truly touched the lives of persons with ataxia. Please help others and give to the 2008 NAF Travel Grant Fund today. Thank you.

Late Onset Ataxia Due to a CGG Repeat Expansion in the FMR1 Gene

By Greg Mayeur, PhD
University of California, Davis

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Fragile X Associated Tremor/Ataxia Syndrome (FXTAS) is a recently reported form of clinical involvement in older carriers of the fragile X premutation. The major clinical features include gait ataxia, progressive intention tremor, parkinsonism and peripheral neuropathy. Gait ataxia begins with balance problems and progresses to the point where use of a cane, walker, and/or wheelchair is successively required. The average age of onset is approximately 60 years old with patients rarely reporting symptoms before 50 years of age.

The fragile X gene contains a trinucleotide repeat region in a part of the gene that does not encode the protein. This repeat expands into a premutation leading to FXTAS in older patients; further expansion into the full-mutation leads to fragile X syndrome in children. These two diseases are distinct in their origin, but interrelated in the search for a treatment.

Treatments for FXTAS will potentially require a two-pronged approach. The first element that must be addressed is the elevated levels of fragile X mRNA that are a direct result of the premutation repeat expansion. This expanded and elevated mRNA is the direct cause of FXTAS. Therefore we must look to lowering the mRNA levels as a treatment. However the premutation mRNA is also less efficient at making the fragile X protein, which is involved in normal learning and memory formation. Reduction of fragile X mRNA would also result in reduction of fragile X protein. The second element that must be addressed is a mechanism for increasing the efficiency of protein production from the premutation mRNA.

To gain a better understanding of how the premutation leads to the elevated mRNA levels we have created a series of DNA constructs to evaluate the role different regions of the gene play in mRNA production. We are specifically looking at modifications of the chromosome structure, and of the DNA itself to understand what may be happening.

To evaluate the role of the premutation in protein production we are looking at two mechanisms: 1) the role the fragile X mRNA structure plays in protein production, and 2) the role other proteins play in regulating fragile X protein production. We have identified several proteins that directly and specifically interact with the fragile X mRNA. One of these proteins, nucleophosmin, is involved in the regulation of fragile X protein production potentially by inhibiting a negative regulator of protein production. We wish to further examine nucleophosmin's role in regulating fragile X protein production in hopes of developing a treatment that could increase protein production efficiency.

An added benefit of this research is that all of it is applicable to finding a treatment for fragile X syndrome is children. ❖

The deadline for the Winter issue of Generations is November 9

Disease-Regulated RNA Interference for Spinocerebellar Ataxia Type-1

Gumei Liu, PhD
University of Iowa

The following is a research summary of a grant funded by NAF for fiscal year 2006.

Spinocerebellar ataxia type-1 (SCA1) is a dominant neurodegenerative disorder caused by polyQ expansion in ataxin-1 gene. SCA1 primarily affects Purkinje cells in the cerebellar cortex. The loss of Purkinje cell function results in cerebellar atrophy and degeneration of downstream pathways.

MicroRNAs are a group of small non-coding RNAs that participate in many biological processes such as cell proliferation, differentiation and apoptosis. Changes in microRNA expression have been indicated in several neurological disorders such as Tourette's syndrome, Fragile X syndrome, and DiGeorge syndrome.

In this study, we investigated the role of microRNAs in SCA1 pathogenesis and its potential application for RNA interference therapy. During the period of this grant, we have (1) compared microRNA expression profiles between SCA1 and wildtype cerebella; (2) identified candidate microRNAs that are differentially expressed in SCA1 cerebellum and enriched in Purkinje cells; (3) developed anti-mir as a tool to regulate microRNA expression in vivo and to access the function of candidate microRNAs; (4) demonstrated that candidate microRNAs are actively involved in cerebellar function supporting their role in SCA1 pathogenesis.

In conclusion, our study reveals, for the first time, that microRNAs actively participate in adult cerebellar function and are differentially expressed in SCA1, suggesting its potential application in future therapy for SCA1 and other neurodegenerative disorders. We are currently investigating how the candidate microRNAs affect SCA1 pathogenesis. Two papers entitled "Differential expression of microRNAs in spinocerebellar ataxia type 1" and "Functional knockdown of microRNAs in the cerebellum using LNA antisense oligonucleotide" are in preparation. ❖

Remembering NAF in Your Will

There have been a number of true heroes over the years that have quietly made a significant impact on the National Ataxia Foundation and the ataxia families it serves. These are people who have named NAF as a beneficiary in their will.

Most of the time the Foundation is unaware of the kind acts of these champions until after they are gone, but each time we are deeply touched and honored by their selfless commitment in helping others.

Over the years these individuals, who have chosen NAF as a beneficiary, have given anywhere from a few thousand dollars to nearly one million dollars. Their forethought and benevolence has enabled the Foundation to support promising ataxia research and provide meaningful programs and services to ataxia families. It is because of these quiet heroes that many research studies and programs have been funded. Their kindness impacts ataxia families today and will be felt for years to come.

We are truly thankful for their humanitarian and compassionate acts and we will be eternally grateful for the impact they have made in helping ataxia families. Their legacy lives on in the hope they have given ataxia families.

Perhaps this is the time to consider adding the National Ataxia Foundation in your will.

How Did You Celebrate IAAD?

The Eighth Annual International Ataxia Awareness Day on September 25, 2007 is now history, but your stories on how that day was celebrated and recognized could live on in a future issue of *Generations*.

Please send your articles, photos and proclamations so the entire NAF family can relive a day set aside for the important mission of bringing ataxia to the forefront.

Please send information by mail to National Ataxia Foundation, 2600 Fernbrook Lane, Suite 119, Minneapolis, MN 55447-4752 or e-mail *susan@ataxia.org*.

June 25, 2007

To Whom It May Concern:

I am a Grade 6 teacher at Barrhead Elementary School in Barrhead, Alberta, Canada. Recently, my class was the recipient of a $100.00 prize at a local Music Festival.

After we won the money, as a class we discussed what would be the appropriate way to use the money – there were "no strings attached" – we could use it for whatever we chose. The consensus was that half of it should go to a charity. After much discussion, we decided the charity should be one that has a personal connection. Many suggestions were made but the one that became the unanimous class choice was your foundation. Why? The mom of one of my students suffers from Ataxia. This student shared with the class how the disease has affected his mom and how her condition has deteriorated over the past few years. He also shared how the disease affects him on a daily basis and the grim future that they, together, face. After he spoke, the classroom was silent and then one student quietly said, "I think that we should give the money to his charity." All students wholeheartedly agreed.

So, on behalf of Grade 6B of Barrhead Elementary School, and in support of my student and his mom, please accept this donation for your Foundation.

Sincerely,

Grade 6 Teacher

Editor's note:

It was a delight for the National Ataxia Foundation staff the day this letter and donation arrived at the office. We are very grateful to these students for their gift.

Chapter and Support Group News from Around the Country

Greater Atlanta Ataxia Support Group

By Dave Zilles

The Greater Atlanta Ataxia Support Group has had a busy summer. On June 30 we held a get-together at Stone Mountain just outside Atlanta. Stone Mountain is the world's largest free-standing piece of exposed granite and is home to the world's largest Laser Show Spectacular. The group gathered in front of the Confederate Memorial for a picnic dinner and then stayed to watch the Laser Show.

On August 11, we held our Support Group meeting at Emory University. We were very excited to have several new members join our group. Our speaker was Jeff Hoder, a physical therapist who works for Dr. Wilmot at Emory University. He brought several devices to show the group and also talked about some of the things that could be done to help improve mobility.

Greater Atlanta Ataxia Support Group

We also discussed our upcoming picnic at Lake Lanier on Sunday, September 23 to celebrate International Ataxia Awareness Day (September 25). The Annual International Ataxia Awareness Day Proclamation signing with Governor Perdue at the capitol building will be on September 6. We will have a Holiday Party the first week in December. Last year we each brought a gift and played a game to exchange the gifts and had plenty of food.

We lost two members of our group recently and our sympathy goes out to the families of Connie Farmer and Jennifer Stewart-Page.

Our next support group meeting will be on November 3.

Alabama Ataxia Support Group

By Becky Donnelly

The Alabama Ataxia Support Group held a social outing in July at Whole Foods in Mountain Brook, AL. Members took a tour of the facility and enjoyed a lunch followed by wonderful fellowship. A guest told of his musical dreams for the future and entertained the group by singing.

A sad note for our group during this quarter was the loss of members Maury McDonald and Lucille Tucker. Our sympathy continues to be with their families.

Denver Area Ataxia Support Group

By Tom Sathre

The Denver Area Ataxia Support Group met June 9 at Swedish Hospital. Nineteen people were present, about half were ataxians and half were caretakers. We shared a meal and listened ▸▸

to two excellent presentations. One presentation was from the three people from our support group who attended the National Ataxia Foundation's Annual Membership Meeting in Memphis. They had photos, a listing of speakers and a doctors' address list with them for us. The other speaker was a local, licensed, experienced, educated acupuncturist and dietician. He displayed a needle set for the group to see. There were several questions from the audience, which shows how engaged they were.

The program "committee" is hard at work planning for the next Denver Area Ataxia Support Group meeting on September 10. It will be hard to top this meeting.

Houston Area Support Group

By Angela Cloud

The Houston Area Ataxia Support Group is a new group. We held our second meeting at the Auditorium of Texas A&M Institute of Bioscience & Technology (IBT) with an attendance of 60 or more people.

We had four excellent speakers. Dr. Tetsuo Ashizawa, John Sealy Professor and Chairman of Neurology at The University of Texas Medical Branch (UTMB) Galveston, discussed types of cerebellum ataxia and what kind of treatments are on the horizon. He also mentioned the new effort of a U.S. clinical study group, the Cooperative Ataxia Group (CAG) along with similar organizations in other countries, including Euro SCA in Europe and others in Japan, China, Australia and South America. Dr. Subramony, Professor of Neurology, also from UTMB Galveston, spoke about efforts to create a National Registry of patients with ataxia. He also mentioned clinical research at UTMB (awaiting committee approval) to look at walking patterns using an electronic monitor and vision problems with special vision tests. Albino Bacolla, PhD, Institute of Bioscience and Technology, reported on the presence in human chromosomes of DNA sequences similar to the expanded GAA repeat that causes Friedreich's ataxia. He explained and demonstrated with a slide show how GAA-type DNA is common in the human genome and in genes that are involved in cognitive functions in the brain. GAA-type DNA is also common on the sex chromosomes particularly in a region named PAR 1 that is essential for sperm maturation. He explained that comparisons with other primate species indicate that GAA-type DNA has been evolving much faster than other parts of chromosomes. Taken together, this data indicate that although GAA-type DNA causes Friedreich's ataxia, it plays other important roles in human cells. Marek Napierala, Assistant Research Scientist and Center for Genome Research (IBT), who

Dr. S.H. Subramony, Dr. Tetsua Ashizawa, Angela Cloud (Houston Area Support Group Leader), Dr. Marek Napierala, and Dr. Albino Bacolla

received NAF's Young Investigator Award, gave a presentation about high throughput screening systems designed to design possible new treatments for ataxia. His and the staffs' focus at IBT is mainly on Friedreich's ataxia. The meeting was very informative.

Our very first meeting in November at Baylor College of Medicine included guest speaker Dr. Huda Zoghbi, who gave an excellent demonstration of the human brain. She took a model of a brain apart and explained how ataxia affects the brain and in return affects the body.

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Chapter and Support Group News
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The primary goal for the last two meetings was to give the group a clear understanding on what ataxia is, how it affects the body and what future treatments and studies will be made available to people with ataxia. The guest speakers did a great job in explaining that to those present.

Los Angeles Ataxia Support Group

By Sherry McLaughlin

The Los Angeles Ataxia Support Group met for our annual barbecue on July 14 at the home of Jim Fritz. With nine people in attendance, we roasted hot dogs, steak and a few fingers. We were pleased to be joined by several Orange County support group members.

The highlight of the meeting was a fantastic presentation by Casa Colina. Their program of outdoor adventures includes everything from simple boat cruises to sky diving and camping trips at a very reasonable cost. President Sid Luther will investigate the possibility of a joint adventure with other support groups. Since we will soon be scheduling one of these adventures, this is a great time for new members to join us.

We also had a brief review of the Long Beach Abilities Expo and were reminded of the upcoming 6th Annual All California Research Meeting (ACARM) on Sunday, October 14. See you at the next meeting!

Northern California Ataxia Support Group (NCASG)

By Deborah Taylor Omictin

The April 14 meeting of the Northern California Ataxia Support Group (NCASG) did not have a formal guest speaker, but instead heard reports from those who attended the 50th Anniversary NAF Annual Membership Meeting, "The Bridge to Hope," in Memphis, TN March 22-25, 2007. After a delicious lunch we broke into two groups for discussion for the remainder of the meeting.

Renate Olaisen, Aquatic Rehabilitative Director at Betty Wright Swim Center (BWSC) at CAR (Community Association for Rehabilitation) in Palo Alto, was the guest speaker at our July 21 NCASG meeting. She spoke about the benefits of an aquatic therapy program, especially a warm-fdwater temperature pool which helps relax and tone. In addition it allows persons to exercise using the hydrostatic pressure, buoyancy, and resistance that only aquatic therapy can provide. Although the facility at BWSC offers warm water, fully accessible aquatic therapy, adapted fitness, and recreation, which are ideal, Renate advised us to use *www.findapool.com* to find a pool near where we live.

She explained in detail the benefits that water exercise allows: a fuller range of motion and strengthening with less stress than using a land exercise program. She showed examples of its effectiveness despite any physical limitations and its adjustability for specific needs. Another benefit is the support and encouragement you receive from your classmates and instructors. Additional benefits are shown if you find a warm water pool, which provides therapeutic comfort and has pool ramps, stairs and aquatic wheelchairs to accommodate those with disabilities. I hope to work with Renate to develop a program to address our group's specific needs.

To have our meeting flyers mailed directly to your home you must become a member of NAF because NAF mails our flyers. You can sign up to become a member online at *www.ataxia.org*, by phoning the NAF office and using a credit card, or by mail using the form on the back of a *Generations* magazine. To subscribe to our on-line group, send an e-mail to *NoCalAtaxiasubscribe@yahoogroups.com*. To sign up for the Cooperative Ataxia Group Registry online, for ataxians interested in participating in ataxia studies or clinical trials, go to *www.cooperative-ataxia-group.org/participate.htm*. ▸▸

Upcoming Events:

• *Sunday, October 14, 2007* – ACARM6-All California Ataxia Research Meeting will be held at the Hilton Irvine/Orange County Airport.

• *March 28-30, 2008* – NAF Annual Membership Meeting hosted by the Arizona Support Group will be held in Las Vegas, NV.

Contact me to borrow a VHS or DVD copy of the "Together There is Understanding" video, which our group has purchased.

Our next (and last meeting for 2007) has been changed to October 27 at the wonderfully accessible Our Savior's Lutheran Church in Lafayette at 11:30 a.m. Our guest speaker will be David Schaffer, PhD, Associate Professor at University of California at Berkeley.

For further information on our group, please contact Deborah Omictin, NCASG Leader, at (510) 783-3190 by e-mail at *rsisbig@ aol.com* or by visiting *www.geocities.com/casupport/*.

Central New York Ataxia Support Group

By Andy and Linda Johnson

Tai Chi and exercise were the topics at the July meeting. Dr. Ferrell talked about the origin of Tai Chi, along with the benefits, which include better balance. He shared a video showing some of the movements and then led a demonstration.

Dr. Carol Krehel then demonstrated exercises that could be done in a chair as well as standing. She said that everyone should be doing some form of exercise and stretching to help them maintain or improve their quality of life. Exercising and stretching can start out with as little as 10 minutes a day with no purchased equipment.

Mary Jane and Emma (her service dog) were on the MDA Telethon. They gave a demonstration of how Emma helps around the house.

Our next meetings will be on:

September 22 at 2 p.m. – International Ataxia Awareness Day Ceremony at North Syracuse Community Center in North Syracuse.

November 17 from 2-4 p.m. (social time 1:30-2 p.m.) – Topic will be nutrition. ❖

News & Notes

Information Requested

The National Ataxia Foundation is presently updating its database to include current e-mail addresses of our members and we need your help. The information you provide will help us with our mailings and "E-mail Blasts."

Please e-mail the following information to Julie Braun at *julie@ataxia.org*: Your name, address, phone number, and e-mail address. Please also include the type of ataxia which affects you or your family member.

Thank you in advance for your help with this important project.

Shopping on the Web

Shop on-line at *www.iGive.com* and for each purchase you make a donation will be made to the National Ataxia Foundation.

Attention NAF Leaders

It's that time of year again to submit your 2008 Schedule of Events and Support Group Meetings. Even if your schedule will remain the same, please contact Lori with NAF at *lori@ataxia.org* or (763) 553-0020. Thank you.

Donate Your Vehicle to NAF

Donating your vehicle to NAF is fast, easy, and tax deductible. For more information on how to donate your vehicle, please call the Foundation office at (763) 553-0020.

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.

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Phone: 0091-040-27971043
Fax: 091-040-27971043
E-mail: sam_ataxiaindia@yahoo.com
E-mail: george@samataxiagroup.org
www.ataxia.org/chapters/Chandu/default.aspx ❖

Calendar of Events

October

Monday, October 1, 2007

Spokane Area Support Group Meeting
5:30-7 p.m. at Sacred Heart Hospital, Spokane, WA in the Mary Bead Room. Contact Linda Jacoy at *jacoyL00@usintouch.com*.

Wednesday, October 10, 2007

Willamette Valley Ataxia Support Group Meeting
11 a.m. - 12:30 p.m. at Albany General Hospital, Albany, OR. Contact Malinda Moore, CCC-SLP at (541) 812-4162 or *malindam@samhealth.org.*

Saturday, October 13, 2007

Ataxia Society of Vancouver, B.C. S.G. Meeting
1-3:00 p.m. at GF Strong Rehab Centre Room 109. Contact Brenda Dixon at (604) 273-2789, *bdixon57@telus.net*, or *bcataxia.org/events.htm*.

Kansas City Ataxia Support Group Meeting
2-4 p.m. at NE Library, 65 Wilson Avenue, Kansas City, MO. Contact Lois Goodman at (620) 223-1996 or Jim Clark at *clarckstone9348@sbcglobal.net*.

North Texas Ataxia Support Group Meeting
10 a.m. at Las Colinas Medical Center, 6800 MacArthur Blvd, Irving TX.

San Diego Ataxia Support Group Meeting
1-3 p.m. at Sharp Rehabilitation Center, 2999 Health Center Drive (behind Sharp Memorial Hospital). Contact Earl McLaughlin at (619) 447-3753, *sdasg@cox.net* or visit *www.geocities.com/ataxia_sdasg/*.

SE Pennsylvania Ataxia Support Group Meeting
10-11:30 a.m. at Mercy Suburban Hospital, DeKalb Pike, Norristown PA, 2nd floor Gerber Room. Contact Liz Nussear at (610) 277-7722 or *lizout@aol.com*.

Tampa Bay Ataxia Support Group Meeting
Noon - 3 p.m. at Feather Sound Community Church, 13880 Feather Sound Drive, Clearwater, FL. Contact Charlie Kirchner at *charlie@flataxia1.org* or visit *www.flataxia1.org.*

Sunday, October 14, 2007

6th Annual All California Research Meeting
Contact Earl McLaughlin for more information at (619) 447-3753 or *emclaugh@cox.net*. A reservation form can be found at *home.comcast.net/~fernandesml/ACARM6.pdf.*

Tuesday, October 16, 2007

Twin Cities Ataxia Support Group
Meetings on the third Tuesday of the month at 7 p.m. at Presbyterian Home in Roseville (located off 35W on County Rd D). Contact Lenore H Schultz at (612) 724-3784 or *lschultz@bitstream.net*.

Saturday, October 20, 2007

Orange County Ataxia Support Group Meeting
2-5 p.m. at Orange Coast Memorial Medical Center, 9920 Talbert Avenue, Fountain Valley, CA. Contact Peggy Hyatt at *mahyatt@social.rr.com* or visit *www.geocities.com/ocasgg/.*

Sunday, October 21, 2007

Seattle Area Ataxia Support Group Meeting
Federal Way 320th Library, 848 S. 320th St. Contact Milly Lewendon at (425) 830-7299 or *mmlewendon@comcast.net*.

Saturday, October 27, 2007

Alabama Ataxia Support Group Meeting/Luncheon
Covenant Presbyterian Church, Birmingham, AL. Contact Becky Donnelly at (205) 987-2883 or *donnelly6132B@aol.com*.

Northern California Ataxia Support Group Meeting
Our Savior's Lutheran Church in Lafayette, CA. Contact Deborah Omictin at (510) 783-3190 or *risbig@aol.com*.

November

Saturday, November 3, 2007

Ataxia Society of Vancouver, B.C. S.G. Meeting
1-3 p.m. GF Strong Rehab Centre Room 109. Contact Brenda Dixon at (604) 273-2789, *bdixon57@telus.net*, or *bcataxia.org/events.htm*.

Greater Atlanta Area Ataxia S.G. Meeting
Emory Center for Rehabilitation Medicine. Contact Greg Rooks at (404) 822-7451 or visit *www.geocities. com/atlantaataxia.*

Monday, November 5, 2007

Spokane Area Support Group Meeting
5:30-7 p.m. at Sacred Heart Hospital, Spokane, WA in the Mary Bead Room. Contact Linda Jacoy ⏩

at *jacoyL00@usintouch.com*.

Saturday, November 10, 2007

Kansas City Ataxia Support Group Meeting
2-4 p.m. at NE Library, 65 Wilson Avenue, Kansas City, MO. Contact Lois Goodman at (620) 223-1996 or Jim Clark at *clarckstone9348@sbcglobal.net*.

Los Angeles Ataxia Support Group Potluck
2-4 p.m. at The Westside Center for Independent Living, 12901 Venice Beach, California. Contact Sid Luther for more information at (818) 246-5758 or visit *www.geocities.com/HotSprings/Falls/6629/.*

North Texas Ataxia Support Group Meeting
10 a.m. at Las Colinas Medical Center, 6800 MacArthur Blvd, Irving TX.

SE Pennsylvania Ataxia Support Group Meeting
10-11:30 a.m. at Mercy Suburban Hospital, DeKalb Pike, Norristown PA, 2nd floor Gerber Room. Contact Liz Nussear at (610) 277-7722 or *lizout@aol.com*.

Tampa Bay Ataxia Support Group Meeting
Noon - 3 p.m. at Feather Sound Community Church, 13880 Feather Sound Drive, Clearwater, FL. Contact Nygel Lenz at *nygellenz@gmail.com* or Chris Frohna at (813) 453-1084 or *chrisfrohna@yahoo.com* or visit *www.flataxia1.org.*

Wednesday, November 14, 2007

Utah Ataxia Support Group Meeting and Tour of the Moran Eye Clinic Building
Contact Dr. Julia Kleinschmidt at (801) 585-2213 or *julia.kleinschmidt@hsc.utah.edu.*

Willamette Valley Ataxia Support Group Meeting
11 a.m. - 12:30 p.m. at Albany General Hospital, Albany, OR. Contact Malinda Moore, CCC-SLP at (541) 812-4162 or *malindam@samhealth.org.*

Saturday, November 17, 2007

Central New York Ataxia Support Group Meeting
2-4 p.m. Topic: Nutrition. Contact Linda Johnson or visit *www.ataxia.org/chapters/CentralNewYork/default.aspx.*

Orange County Ataxia Support Group Meeting
2-5 p.m. at Orange Coast Memorial Medical Center, 9920 Talbert Avenue, Fountain Valley, CA. Contact Peggy Hyatt at *mahyatt@social.rr.com* or visit *www.geocities.com/ocasgg/.*

Sunday, November 18, 2007

Chicago Area Ataxia Support Group Meeting
1 p.m. at Good Samaritian Hospital–White Oak Room, 3815 Highland Ave., Downers Grove IL. Contact Craig Lisack at (847) 496-7544 or *caasg2@aol.com.*

Tuesday, November 20, 2007

Twin Cities Ataxia Support Group
Meetings on the third Tuesday of the month at 7 p.m. at Presbyterian Home in Roseville (located off 35W on County Rd D). Contact Lenore H. Schultz at (612) 724-3784 or *lschultz@bitstream.net*.

December

Monday, December 3, 2007

Spokane Area Support Group Meeting
5:30-7:00 p.m. Sacred Heart Hospital, Spokane, WA in the Mary Bead Room. Contact Linda Jacoy at *jacoyL00@usintouch.com*.

Saturday, December 8, 2007

Ataxia Society of Vancouver, B.C. S.G. Meeting
1-3 p.m. GF Strong Rehab Centre Room 109. Contact Brenda Dixon at (604) 273-2789, *bdixon57@telus.net*, or *bcataxia.org/events.htm*.

Kansas City Ataxia Support Group Meeting
2-4 p.m. at NE Library, 65 Wilson Avenue, Kansas City, MO. Contact Lois Goodman at (620) 223-1996 or Jim Clark at *clarckstone9348@sbcglobal.net*.

SE Pennsylvania Ataxia S.G. Winter Luncheon
Location TBD. Contact Liz Nussear at (610) 277-7722 or *lizout@aol.com.*

Wednesday, December 12, 2007

Willamette Valley Ataxia Support Group Meeting
11 AM - 12:30 p.m. - Albany General Hospital, Albany, OR Contact Malinda Moore, CCC-SLP (541) 812-4162 *malindam@samhealth.org.*

Saturday, December 15, 2007

Orange County Ataxia S.G. Meeting
2-5 p.m. at Orange Coast Memorial Medical Center, 9920 Talbert Avenue, Fountain Valley, CA. Contact Peggy Hyatt at *mahyatt@social.rr.com or visit www.geocities.com/ocasgg/.*

Tuesday, December 18, 2007

Twin Cities Ataxia Support Group
Meetings on the third Tuesday of the month at 7 p.m. at Presbyterian Home in Roseville (located off 35W on County Rd D). Contact Lenore H. Schultz at (612) 724-3784 or *lschultz@bitstream.net*. ❖

"Generations"

From December 1982 to January 1988, John B. Gallant, Jr. was a regular contributor to *Generations*. In his writings, he shared the challenges and opportunities that were his due to hereditary ataxia. In one of his articles he described his daughter, "A girl of 15 who is starting senior high school this year. She is still full of wonder and love." That daughter is Tracey Gallant Balis who, like her dad, has ataxia as well as a talent and desire to continue writing the story that her father began. Look for her stories in future issues of *Generations*. At right is a poem she wrote:

Dreams

By Tracey Gallant Balis

In my dreams I am free
In my dreams I have no disability
I can run through the rain
and I feel no pain
I can jump over rocks
Even put on my own socks
In my dreams it all can happen
I can do this even while napping
Never let your dreams go
Just lay back and let them flow.

Letter to the Editor

By Theresa Gonzales

Editor's note: This letter is in response to Dr. Thomas L. Clouse's article "Dancing with Ataxia," published in the Spring 2007 issue of *Generations*.

To the Editor of *Generations*:

My name is Theresa Gonzales and I have wanted to write you for some time. I woke up this morning feeling rested, but at the same time, feeling tired and worn out; somehow I think you understand. The type of ataxia I have is undiagnosed. I am blessed with a wonderful doctor. I can still walk with a walker, cane or by just holding onto others or objects for help.

When I read your article, "Dancing with Ataxia" and visited your website, I found such encouragement! I cried when I realized how much you had to give up – being a surgeon. I felt so encouraged when you said that ataxia affects self- esteem. That was the first time I heard or thought about that. I am a person who believes in the power of positive thinking and how your attitude affects your own personal journey and take on life. I didn't realize that the progression of ataxia really shakes one's confidence, self-esteem, and the very core of your being.

Thank you for those very encouraging words. I am grateful that there is a professional out there, (and I don't mean this in a disrespectful or negative way) who shares our daily experiences and struggles in a personal way.

Thanks again and God bless,

Theresa Gonzales

Want to Share Your Story?

Letters to the Editor, personal stories, and poems are all an important part of *Generations*, as they give inspiration and encouragement to our readers. We welcome your letters, stories and poems. Please send materials care of "Editor" using the address on page two, or e-mail *susan@ataxia.org* by November 9.

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 June 2007 through July 2007. 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.

Frank Aldridge, Jr.
Florence Allen
Laura Anthony
Sharon Baggett
Bob Barbeau
Esther Bender
Joan Bott
Linda Bowen
Hilda Braswell
Ernest Brede Family
Jane Brewer
Kyle Bryant
Chris Buechel
Bob Burdett
Donald Burdett
Donna Burdett
Ellen Burdett
Keith Burdett
Kyle Bussas
Darrin McCarty
Eugene Clark
Joe Coffey
Edith Cooley
Mike Craft
Dot Crawford
Kevin Crowley
Archie Curtit
Marcia Cyganowski
Mary Danson
Jeannette Davis
Patrick Dewey
Anita Dillaha
Mr. & Mrs. Ernest DiMonte, Jr.
Carlo DiSilvestro
Andrew Dolan
Kevin Donnelli
Frederick Donnelly, Jr.
Diane Dusbiber
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Andrew Egeressy
Sandra English-Martin
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Ginette Friedman
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Rachael Gammill
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Alfred Gruen
Paschal Guercio
Ricardo Guerrero
Teresita Guerrero
Annabel Guffin
George Guffin
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Carmela Herman
Johnny Hogan
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Penny Hurst-Kirkham
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Rinda Janney
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Maureen Jurek
Robert Keithly
Colleen Kosieracki
Jamie Kosieracki
John Lane, Sr.
Rodger Larsen
Clarke Lauchmen
Linda Lee
Amy Lenahan
Harriet Lewis
Richard Lewis
Peggy Littlejohn
June Liverman
Mendall Long
Virginia Lutz
Gordon Macklin
Marilyn Macklin
Carly Magnuson
Deborah Markham
Sandra Marlon
Sandra Martin
Brent Masserant
Betty McAdam
Robert McAllister
Maurice McDonald, Jr.
Mac McGrath
Ira McLain
Charley McLaughlin
Earl McLaughlin
Marylaine McLaughlin
Reggie Mellon
Margaret Millsap
Alfred Moline
Patrick Moore
Dolores Morello
Mandy Morse
Valerie Morse
Carol Mullen
Grace Mutschler
Bruce Nanninga
Eileen Natches
Gretl Neuwald
Christina Newell
Jennifer Page
Patti Page
Michael Parent
Corey Pemberton
Sean Pemberton
Janet Pepitone
Robert Picou
Patrick Pisano
Rita Powell-Lobascio
David Price
Mrs. Ravinsky
Dawn Rissell
Valerie Ruggiero
Jeremey Ryan
Mathew Salles
Mark Salvani
Vince Sanfilippo
Harley Scheffler, Sr.
Tony Schlickbernd
Dr. Jeremy Schmahmann
John Schreiber
Rogene Schreiber
Derek Semler
Carolyn Shamblin
Sherry Sharp
Zena Sims
Kimberly Skinas
Barbara Smilow
Jules Smilow
Kathleen Smith
Kenneth Smith
Abbie Spellman
Roxanne Stafford
Joey Staiger
Thomas Stanley
Jerome Stewart
Dick Strong
Tiffinay Talarico-Compiano
Robert Taylor
Therese Thalmann
Jeanette Thaman
Fayne Thiel
David Thomas
Dr. Aymee Torres Michels
Gertrude Tougas
Bonnie Tucker
Bruce Turner
Jay Underwood
Rudolph Van't Hoff
Mary Viscido
Jo Voecks
Lois Welter
Jean Wentz
Walter Wentz
Betty White
John Wilcox
Charles Williams
Ethan Williams
Barbara Williamson
Jerry Williamson
James Wisniewski
Betty Worfel
Joyce Woudenberg
Jenny Young
Mark Young
Nathan Young
Ryan Young ❖

NEW: On the NAF Website

For those who choose to make an on-line donation of $100 or more In Honor Of or In Memory Of, you now have the option of creating your own Honor or Memorial page (including a photograph) on our web site.

Go to *www.ataxia.org* and click on *Donate Now*.

National Ataxia Foundation
2600 Fernbrook Lane, Suite 119
Minneapolis, MN 55447-4752
(763) 553-0020

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 _____

From:

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 _____

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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 _____

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