Using Social Media in ALS Research

Inside:

FUS gene offers new insights into ALS

‘Gib’s Odyssey’: Confronting ALS and the sea
Research Roundup

Eating and breathing: A possible ‘synergistic’ effect in ALS

Initial results of a multicenter pilot study to evaluate nutritional requirements and early intervention for breathing problems in ALS are helping scientists determine how physical factors such as body mass, diet and activity affect the total daily energy expenditure of people with ALS at different stages of the disease.

Neurologist Edward Kasarskis, professor of neurology, toxicology and nutrition at the University of Kentucky in Lexington, and colleagues, reported preliminary results in the January issue of Amyotrophic Lateral Sclerosis.

The study is designed to determine factors contributing to energy; evaluate nutrition in ALS; and investigate the effects of noninvasive positive pressure ventilation (NIPPV) used for respiratory support in ALS.

Although optimal nutrition and ventilatory function in ALS may appear to be unrelated issues, the investigators noted that each may affect the other and provide “synergistic” benefits overall.

For example, NIPPV used through the night might reduce the number of calories burned during the day. Also, lack of energy translates to muscle weakness and fatigue, and a poor diet can adversely affect the structure and function of the diaphragm (a major respiratory muscle located under the ribcage).

Further analysis of the initial findings will enable the research team to:

• develop two approaches for pinpointing the best time for people with ALS to begin using a feeding tube, based on specific knowledge of each individual’s actual energy needs, rather than on general estimates of indicators such as bulbar function and weight change; and
• design a clinical trial to test NIPPV prior to the onset of respiratory symptoms.

Vitamin E may protect against ALS

Data taken from more than a million people have revealed that those who take vitamin E supplements over a long period of time have a reduced risk of developing ALS.

Among the 1,055,546 trial subjects, whose data was culled from five non-ALS clinical trials conducted between 1976 and 2005, 805 developed ALS. Of those, information on vitamin E use was available for 231.

A research team at the Harvard School of Public Health in Boston published its findings online Feb. 18, 2011, in the American Journal of Epidemiology.

Although a straightforward examination of supplementation with vitamin E itself did not reveal any association with a higher or lower risk of ALS, a relationship between the duration of time for which people take the vitamin and ALS risk was observed.

People who take vitamin E for longer periods of time appear to have a lower relative risk of devel-
An antioxidant, vitamin E helps protect against a cell-damaging process known as oxidative stress. The results from a study led by neurologist Claude Desnuelle at CHU de Nice Hospital in France showed that vitamin E had no effect on survival or on the loss of muscle function in people with ALS who participated in a 2001 trial. Those who were taking it, however, were less likely to progress to severe ALS within the one-year study period.

Analysis of data gleaned from a 1982-1998 questionnaire-based study reported by Albert Ascherio, then an associate professor at the Harvard School of Public Health, and colleagues, also showed a relationship between vitamin E and ALS. Participants who reported taking vitamin E for 10 or more years had only 38 percent the risk of developing ALS than did non-vitamin E users. In those taking vitamin E for fewer than 10 years, the relative risk was 59 percent.

Note: Always consult with a physician before beginning a diet or supplement regimen.

**Naltrexone: Benefits questionable, harm possible**

An investigation has found no data to suggest that low-dose naltrexone might have a therapeutic effect in people with ALS. Furthermore, some data indicate the drug potentially may cause harmful effects, including liver toxicity.

Naltrexone was studied at the request of people with ALS who suggested it via the online forum ALSUntangled (www.alsuntangled.org). The drug is approved by the U.S. Food and Drug Administration (FDA) for the treatment of addictions to alcohol or opiate drugs such as codeine and morphine.

The researchers, all members of the ALSUntangled forum, noted that some of naltrexone’s characteristics suggest that it may prove useful in immune system modification and neuroprotective strategies in ALS. However, “a small pilot study of a drug with similar mechanisms found no objective benefits.”

Additionally, the study team noted, a group of 31 people with ALS who participate in the online forum PatientsLikeMe (www.patientslikeme.com) obtained prescriptions for naltrexone from their physicians. Of the 31 patients who took the drug, 15 completed an evaluation of the treatment, with some reporting benefits that included decreased yawning, better balance, increased energy, improved speech and more effective breathing.

Seven participants (47 percent) said the drug had no effect or that they were unsure about whether it had.

Three participants (20 percent) reported “slight” efficacy; and four (27 percent) reported “moderate” benefits.

The full report detailing the new findings, “ALSUntangled No. 8: Low dose naltrexone for ALS,” was published in the January 2011 issue of Amyotrophic Lateral Sclerosis. The paper is available free online; visit http://informahealthcare.com/amlj and put in search terms “ALSUntangled and naltrexone.”

Dozens of researchers worldwide participate in ALSUntangled discussions and investigations of new alternative and off-label therapies. (“Off-label” describes drugs prescribed to treat conditions other than the one[s] for which they were approved.)

The project was organized by the World Federation of Neurology in 2009 and is hosted on the organization’s ALS website (www.wfnals.org).

(See a related article on social media and research on page 10).
Growth factor protects motor neurons in mice

Targeting and confining the experimental treatment G-CSF to the spinal cord improved motor function, delayed disease progression and increased survival time in ALS mice, a research team from Heidelberg, Germany, has reported.

The researchers, who published their findings in the February 2011 issue of Molecular Therapy, orchestrated the intraspinal delivery of G-CSF by encasing it in the emptied-out shell of an adeno-associated virus (AAV) and then injecting the construct directly into the spinal canal.

G-CSF (for Granulocyte-colony stimulating factor) belongs to a family of proteins called neurotrophic growth factors, which support the growth, health and survival of motor neurons.

Although previous studies have shown that the protein confers similar benefits when injected subcutaneously (under the skin) in ALS mice, complications have limited the systemic (whole-body) delivery necessary for the molecule to have more far-reaching effects.

The new findings show that in an ALS research model, G-CSF rescues motor neurons, improves conditions at the neuromuscular junction (the place where nerve cells meet muscle), and enhances regeneration of axons, the long fibers through which nerve cells conduct signals.

‘NurOwn’ stem cell trial under way

Biotechnology company Brainstorm Cell Therapeutics is conducting a phase 1-2 clinical trial of stem cell therapy in adults with ALS.

The trial is designed to test Brainstorm’s experimental therapy NurOwn, and involves transplantation of multipotent mesenchymal stem cells secreting neurotrophic factors (MSC-NTF), into people with ALS. The cells are taken from each participant’s bone marrow and treated with NurOwn stem cell technology to create healthy NTF cells which, when transplanted back into the individual, are expected to produce and secrete neurotrophic factors essential for the survival and outgrowth of neurons.

The trial is being conducted at the Department of Neurology & Laboratory of Neuroimmunology, at the Hadassah Hebrew University Medical Center, Jerusalem, Israel, and is set to include 12 people with early-stage ALS and 12 people in more advanced stages of the disease.

In early-stage ALS trial subjects, MSC-NTF cells will be transplanted into patients’ clinically unaffected (or only mildly affected) upper arm biceps and triceps muscles. Each participant will receive 24 injections (under mild anesthesia) containing a total of 24 million cells.

In those subjects with more advanced ALS, transplantation of MSC-NTF cells will be made intrathecally (into the spinal canal) under mild anesthesia, via lumbar puncture. Each participant in this category will receive a total of 60 million cells.

The study is designed to establish safety of NurOwn first; later, investigators will look for signs of efficacy.

Brainstorm (www.brainstorm-cell.com), with operations in New York and Petach Tikvah, Israel, was granted orphan drug designation for NurOwn by the U.S. Food and Drug Administration in February 2011. (Orphan drug status provides financial incentives for the development of drugs for rare diseases.)

For more information, visit ClinicalTrials.gov and search for “NCT01051882.” Or, contact Dimitrios Karussis at +972-2-6776939, or karus@cc.huji.ac.il; or Adi Vaknin-Dembinsky at +972-2-6776939, or adembinsky@yahoo.com; and refer to the study by its ClinicalTrials.gov identifier.

Cyclosporin-based compound advances toward human trials

Maas Biolab (www.maasbiolab.com) of Albuquerque now has patent protection and orphan drug designation for its cyclosporine-based compound Mitogard.

Cyclosporin (sometimes spelled “cyclosporine”), an immunosuppressant, is believed to have neuroprotective properties.

Mitogard is designed to treat ALS and other neurodegenerative diseases by being infused directly into the cerebrospinal fluid, bypassing natural barriers that typically prevent penetration of the central nervous system.

Maas Biolab currently is conducting animal studies to assess the safety of the compound.
Your Life. Your Ride.

Over 200 new and used vehicles in stock.

Great deals on new 2011 models:
All pricing and specials listed online at www.RollxVans.com

Your Pick
Over 200 full-size and minivans in stock ready for immediate delivery, including Honda, Chrysler, Ford, Volkswagen, as well as a large inventory of quality refurbished vehicles.

Factory Direct
With Rollx you buy directly from the manufacturer, which means no middleman and more money in your pocket.

Personal Touch
Rollx is the only manufacturer that features at-home delivery and at-home service.

Service means satisfaction
Rollx Vans holds an “A” rating with the Better Business Bureau and a better than 97% customer satisfaction rating.

VA and State Agencies
We simplify the process by working directly with Veteran Administration, state and county agencies, and other third party groups on your behalf.

Find your ride. Live your life.

Visit www.rollxvans.com for the latest inventory, pricing and rebates.
Or call 1-800-956-6668.
In 2009, it was discovered that genetic mutations in the fused in sarcoma gene — FUS for short — were linked to some cases of ALS (amyotrophic lateral sclerosis, or Lou Gehrig’s disease).

Subsequent studies of the FUS protein have stirred the ALS research pot and raised the idea that disruptions in RNA metabolism may be a crucial part of what’s going awry in the disease.

Mutated FUS is now thought to be the primary cause of 4 to 5 percent of familial (inherited) ALS cases. In addition, abnormal clumps containing FUS protein have been found in the motor neurons of people with the more common sporadic (non-inherited) form of ALS, suggesting a critical role for FUS in the ALS disease process.

Evidence suggests that the only cases of ALS in which FUS doesn’t appear in such protein clumps are familial cases caused by mutations in the SOD1 gene.

Understanding FUS — what it does, and what happens when it functions abnormally — should provide a clearer understanding of the ALS disease process in both the inherited and non-inherited forms of the disease.

**Mutations cause FUS to gather in the wrong place**

FUS normally resides inside the cell nucleus, where it primarily functions as an RNA binding protein. (RNA is the chemical step between DNA and protein synthesis. RNA binding proteins are involved in RNA processing, which is required to prepare it to be efficiently decoded by the cell’s protein-building machinery.)

Studies in cell cultures have shown that ALS-linked mutations in the FUS gene disrupt the nuclear localization signal (NLS), the molecular mechanism that makes sure the FUS protein goes to the cell nucleus.

Without the NLS to guide it, FUS protein improperly locates outside the nucleus in the compartment of the cell called the cytoplasm.

Once in the cytoplasm, FUS recruits other proteins essential for RNA processing into the area, resulting in protein clumps called inclusion bodies or aggregates. These clumps have been observed in ALS-affected motor neurons, and in the glial cells that nourish and support them.

**FUS connected to cellular toxicity in ALS**

In addition to mislocating to the cytoplasm, FUS protein also causes cel-
lular toxicity and cell death in the ALS disease process.

In yeast models, a direct relationship between toxicity and protein levels exists. The higher the levels of mutant FUS protein (or higher-than-average levels of normal FUS protein) the more damaging the toxic effects on the cell.

Research scientists have identified five yeast proteins that suppress toxic human FUS in yeast models. Interestingly, the proteins did this without affecting FUS protein levels, location of FUS in the cell, or FUS-containing aggregates in the cytoplasm.

Additionally, a human version of one of the five toxicity-fighting yeast proteins has been found to reduce toxicity in the yeast model. The human protein, hUPF1, may be a potential target in development of therapies that take aim at FUS-related ALS.

**FUS and TDP43: Alike, but different**

Like FUS, mutated TDP43 also has been identified as a primary cause of some cases of ALS. The two proteins bear a number of common features, beginning with their similar structure.

Both the FUS and TDP43 genes carry coded instructions for RNA-binding proteins which, in ALS, mislocate from the cell nucleus to the cytoplasm, where they form aggregates.

However, the two differ in the ways in which they cause damage to cells. Requirements for FUS and TDP43 aggregation are different, as are the specific ways in which the two proteins affect cells at later stages. While mutations in the FUS gene primarily alter the protein’s location, TDP43 mutations mainly affect the protein’s aggregation and toxicity characteristics.

Screens designed to identify genes that affect the toxicity associated with FUS and TDP43 found two very different sets of modulators.

Only two genes have been identified that are able to modulate the toxicities of both FUS and TDP43, suggesting that while the two proteins cause cell death via different pathways, these pathways may share some common elements.

Studies conducted in a recently developed fruit fly research model have shown that interaction of normal FUS with mutated TDP43 enhances motor neuron death; and likewise, interaction of normal TDP43 with mutated FUS enhances neurodegeneration. This synergistic interaction suggests the two proteins’ pathways do intersect at some point (likely further downstream) in the toxicity process.

**The experts comment**

On the following page are perspectives on FUS from three researchers interested in its relationship to ALS.

The online version of this article includes commentary from additional researchers about the FUS protein and its significance in ALS. These researchers include Edward Kasarskis (University of Kentucky, Lexington), James Shorter (University of Pennsylvania, Philadelphia), Antonio Musaro (University of Rome, Italy) and others.

… continued next page
Benjamin Brooks
Carolinas Medical Center
Charlotte N.C.

How to prevent selective loss of neurons?

Brooks, director of the MDA/ALS Center at Carolinas Medical Center, and colleagues, are interested in the normal and abnormal molecular interactions of FUS, as well as microRNA regulation of FUS and effects on neurotoxicity in cells under stress.

“We find it interesting that proteins with similar functions are involved in the selective loss of motor neurons,” Brooks said, adding that the “million-dollar question” would be, “what molecular mechanisms could be targeted to prevent this selective loss,” and at what stage of human development could mutations or susceptibilities be identified?

Brooks noted that “unknown genes involved in FUS-related pathways may potentially represent drug targets in ALS,” and that “mutated FUS forms may be used in model systems to identify such targets.”

It’s important to study FUS, Brooks said, because ALS is still poorly understood, so any causative mutation, along with its mechanisms and effects, is worth in-depth investigation in the search for treatments and cures.

Teepu Siddique
Northwestern University
Chicago

‘We know very little about FUS’

Siddique, former co-director of the MDA/ALS Center at Northwestern and frequent MDA grantee, is interested in the relationship between FUS and sporadic ALS. With colleagues, he is developing induced pluripotent stem cell (iPSC) lines for use as a model in the identification of the molecular signature of FUS.

Siddique’s group was the first to report the presence of abnormal FUS in the spinal cords of people with sporadic ALS.

Studies of FUS are applicable to both inherited and noninherited forms of ALS, “at least at the pathological level,” said Siddique.

He noted that the normal function of FUS (and, likewise, TDP43) is not known beyond its RNA-binding function. “We know very little about FUS; a lot is still left to learn,” Siddique said, adding that FUS is sometimes associated with cases of ALS in very young people, and — similar to TDP43 — also can cause dementia.

FUS is responsible for about 5 percent of familial ALS, or less than 1 percent of all ALS, Siddique said. Understanding its role “is important in large families, very early onset patients, and in uncovering its downstream or post-translational role in sporadic cases of the disease.”

Aaron Gitler
University of Pennsylvania
Philadelphia

FUS ‘solidifies’ the role of RNA metabolism in ALS

Gitler and colleagues are investigating factors that contribute to FUS aggregation, and also what defines the cellular pathways that are affected when the protein is mislocalized in ALS.

“FUS is now the second RNA-binding protein linked to ALS,” Gitler said. “Since both FUS and TDP43 are very similar proteins, it has been widely assumed that they will contribute to disease by similar mechanisms. However, this hypothesis has not yet been tested.”

Gitler’s team is working to determine the similarities and differences between TDP43 and FUS, and the mechanisms by which they contribute to disease.

Emerging evidence indicates that in addition to mutated FUS contributing to inherited forms of ALS, normal FUS can also contribute to some sporadic cases of other neurodegenerative diseases, Gitler said. The extent to which FUS contributes to sporadic ALS still is uncertain, he said.

The identification of both TDP43 and FUS as causes of ALS “has solidified a role for RNA metabolism” in the disease, Gitler said. (RNA metabolism amounts to the production, processing and degradation of the molecule.)

“Understanding how FUS affects RNA metabolic pathways, normally and in disease, will contribute to our knowledge of cellular pathways with importance to ALS,” noted Gitler.

Ultimately, “FUS and TDP43 are both aggregation-prone RNA binding proteins,” Gitler said, noting that it will be important when developing therapeutic approaches to determine whether loss of normal function or a toxic ‘gain of function’ is at work.

“An understanding of the similarities and differences between TDP43 and FUS will help guide individual therapies targeting specifically TDP43 or FUS, or perhaps a combination therapy targeting both.”
Expressing emotions shouldn’t be left to chance

- People with pseudobulbar affect (PBA) suffer sudden, involuntary outbursts of crying or laughing throughout their day.
- PBA can occur in people with an underlying neurologic condition—such as Lou Gehrig’s disease (ALS), multiple sclerosis (MS), stroke, or traumatic brain injury.
- Though it may sometimes seem like it, a person with PBA is not alone. More than a million Americans suffer from the condition.

If you or someone you care for shows signs of having PBA, talk to your doctor or visit PBAinfo.org. You can also share your PBA experiences at facebook.com/PBAinfo.
Karen Felzer studies earthquakes, not ALS.

But after her father learned he had the disease, Felzer helped conduct a study that caused a small shake-up in the world of ALS research.

Working with Humberto Macedo, a Brazilian computer analyst who also had ALS, Felzer initiated an online, patient- and caregiver-directed study of the mood drug lithium, which reportedly had been found to dramatically increase survival time in ALS.

“When the news came out that lithium was supposed to be this wonder drug, we were very optimistic,” said Felzer, who has a doctorate in geophysics from Harvard and works for the U.S. Geological Survey in Pasadena, Calif. “And if it was really going to help, we wanted to be able to demonstrate that it did.”

Felzer and Macedo posted messages on online forums to recruit some 200 ALS patients who wanted to take lithium, set up a website (http://alslithium.atspace.com) to track their self-reported data, and created an algorithm to analyze the information.

The pair later joined forces with the website PatientsLikeMe, which provided additional study tools, including matching controls (people of similar ages and stages of ALS progression who weren’t taking lithium) and a more rigorous algorithm.

The results of PatientsLikeMe’s yearlong study confirmed Felzer and Macedo’s earlier, disappointing result: Lithium didn’t work.

But the method used to arrive at that conclusion — online, self-reporting by patients, with matched controls and immediate results — demonstrated that people with ALS (and other conditions) may be able to play a role in advancing research by participating in social media.

**Promise and pitfalls**

ALS experts say the PatientsLikeMe lithium study reveals both the promise and the pitfalls of this trend.

The advantages include:

• speeding up the pace of clinical trials, especially recruitment of participants;

• supplementing information on side effects and adverse events of proposed treatments;

• providing information on off-label, alternative and fraudulent treatments;

• lowering the cost of clinical trials; and

• quickly identifying both promising avenues of inquiry and drugs that are unlikely to work.

But disadvantages are numerous, including:

• unknown source and quality of data (for example, diagnoses have not been verified);

• potential over-, under- or mis-reporting of symptoms;

• possible placebo effect unaccounted for;

• selection bias (for example, younger people are more likely to use social media);

• trial dropouts may be unaccounted for;

• statistical interpretation of results may be incorrect;

• encourages people to self-medicate; and

• confidentiality may be compromised.

“It’s a new field, and we’re still in a learning period,” said neurologist Merit Cudkowicz, director of the MDA/ALS Center at Massachusetts General Hospital in Boston and a member of MDAs Medical Advisory Committee.

Cudkowicz urged caution going forward. She warned that such studies must be well-designed and the results correctly interpreted and validated in order to avoid coming up with wrong answers, or missing safety problems or important treatment effects.

Social media studies may be useful in patient education, information sharing and improving enrollment in standard clinical trials, Cudkowicz said, or for generating hypotheses that then could be tested through more rigorous methods.

A social media component also could be added to traditional clinical trials to make them more efficient, noted MDA Vice President of Re-
search Sanjay Bidichandani.

“Via empowerment of patients, social media can speed up and increase enrollment and ease of reporting symptoms,” Bidichandani said. But he and Cudkowicz strongly emphasized that social media-based studies are not a replacement for traditional clinical trials.

The lithium study

The originators of the online lithium study — Karen Felzer and Humberto Macedo — are examples of the “patient push” driving social media research. Both were professionally educated, computer literate and had a direct emotional connection to the topic: Felzer was helping care for her father Alan, an engineering professor who had ALS, and Macedo, a 42-year-old father of six, had ALS himself.

After working on the project on their own for a while, the pair ultimately turned the study over to PatientsLikeMe (www.patientslikeme.com), a health data-sharing website that already had thousands of people with ALS reporting regularly on what drugs they take, what symptoms they experience, and other information.

PatientsLikeMe charted and tracked the data, established the control group, conducted the analysis and, earlier this year, published the results in the scholarly journal Nature Biotechnology. The study ended up being more about the utility of social media in research than whether or not lithium works. (Other, more traditional studies were already confirming that it didn’t.)

Tom Masters, 54, of Anaheim,
Calif., who received a diagnosis of ALS in 2008, was one of the participants in the lithium study. He said he entered his data — including scores from his self-administration of the Revised ALS Functional Rating Scale — into the PatientsLikeMe site about every two weeks. It took about 10 minutes each time.

Even though lithium didn’t work for him, Masters said he would definitely participate in such a study again. “The advantage is time to results,” he said in an email. “Instead of waiting years, results can be had in months.”

Nevertheless, questions remain about the quality of the self-reported data in the study. How do we know these people even have ALS? Or that they’re reporting their symptoms accurately? According to PatientsLikeMe, about 70 percent of trial participants gave the name of their diagnosing physician, but the site was unable to independently confirm participants’ diagnoses.

Felzer, who is trained in statistical analysis, believes that the large size of the final study group (149 people taking lithium and 447 who weren’t) should “wash out” the effects of people who might not be reporting their symptoms accurately.

She also noted that, when studying a drug as inexpensive and widely available as lithium, people have little incentive, financial or otherwise, to fake data. But if the drug or treatment wasn’t easy to obtain and/or profits were at stake, the situation might be different.

Social media research boom

PatientsLikeMe, which now has more than 100,000 people entering data on some 500 different medical conditions, may be the most comprehensive, but it is only one of a growing number of health data-sharing websites.

Some of these sites are disease-specific. The Life Raft Group, for example, focuses on a rare form of intestinal cancer and limits its membership to patients and family caregivers. It functions as an online support community that also conducts research on drugs and treatments its members are taking.

Another site, 23andMe, which focuses on genetics, just published its first peer-reviewed, scientific study utilizing self-reported data from its members. The site hosts a research arm, called 23andWe, that collects patient-entered information on various conditions.

Researchers have found that sites like these, and networking tools like Facebook and Twitter, are useful for disseminating information and for identifying potential study subjects. But the open channels of communication can cut both ways.

In the last few years investigators in Canada have found themselves inundated with requests from people using social media who want them to study an unproven treatment for multiple sclerosis that involves widening patients’ veins.

“The case indicates the unprecedented pressure scientists … worldwide now face to alter research priorities even in the absence of credible scientific evidence,” the Canadian physicians recently wrote in the journal Nature.
ALSUntangled

One research website that is open to social media-based inquiries is ALSUntangled (http://alsuntangled.com). The site was founded in 2009 by a group of respected physicians from the World Federation of Neurology — some MDA affiliated — who wanted to provide scientific answers to the many questions posed by ALS patients and their families about alternative, off-label and experimental treatments.

Using Twitter, ALSUntangled takes suggestions for treatments and therapies to be studied and then conducts quick reviews of available research on the most popular topics. It also sometimes conducts its own investigations (for example, visiting stem cell clinics or interviewing people who claim to have found “cures” for ALS).

ALSUntangled so far has published on its site the results of 10 reviews of alternative and off-label therapies (see “Naltrexone: benefits questionable, harm possible,” page 3). It is collaborating with PatientsLikeMe and other websites to provide accurate and comprehensive reviews of purported ALS treatments and to warn people about frauds and rip-offs.

Going forward

In the three years since Karen Felzer and Humberto Macedo began their innovative project, both Felzer’s father and Macedo have passed away from ALS. Neither benefited from the study they helped pioneer.

But the work they inspired lives on, providing new tools and methods to researchers hoping to speed the search for ALS treatments.

For more information

- Numerous news articles also are available on this subject under the search terms “social media and research.”

Change your view of the world!

Join the thousands of people world-wide who now have their lives back thanks to the Airpulse PK™—people like Wayne King who travels to new sites with those he loves most.

“ ’We take on the toughest seating cases when other cushion options are no longer effective at preventing pressure sores.’

Wayne King, world traveler

AirPulse PK
Powered Cushion System

• Alternate air cells inflate and deflate automatically to the desired firmness on an adjustable timed cycle
• Relieves pressure and stimulates circulation to facilitate the prevention and healing of pressure sores
• Customized to address each user’s unique needs

HSPCS E2689 • VA FSS V797P-3200M

“Gib’s Odyssey” tells of a single-handed journey from the Florida Keys up the Intercoastal Waterway to New York and back, completed in 2004 by Gib Peters, a 68-year-old financial consultant, newspaper columnist, weekend sailor and person with ALS. Published after his death, it was pieced together from Gib’s emails from sea by his doctor, Walter Bradley, former director of the Kessenich Family MDA/ALS Center, University of Miami School of Medicine.

The details of the seven-month trip are hair-raising. Due to bulbar-onset ALS, Gib could not speak by radio with other boats or the Coast Guard while piloting his 29-foot Wellcraft, the Ka-Ching. As his arm and neck strength waned, he resorted to steering with his feet while leaning back in a chair to keep his head balanced.

“I see it as a challenge to defeat my ALS by inventing ‘workarounds’ and innovations,” he painstakingly typed in one of his lively emails, often written at the end of the day after a jigger of rum had gone down his feeding tube. “Small victories in a war that won’t be won.”

The emails share insights and stories about close calls, mechanical failures, botched rescue attempts and the antics of Ka-Ching’s two cats, Faith and Hope.

Bradley, who has written nearly 30 books and is lead editor of the textbook “Neurology in Clinical Practice,” rounds out the book with tidbits of history, geography and seamanship, as well as inside information about ALS based on his many years as a physician and researcher.

Why did he take on this project? “I think it’s something ALS patients ought to know about,” says Bradley. “Gib had the best insights I’ve ever heard about life, death and facing the consequences of ALS.”

Even so, Bradley at first tried to talk Gib out of the trip. “I thought he was mad!” he says, noting he suspected Gib might be experiencing the impaired judgment that sometimes occurs in ALS.

“No sensible person, even able-bodied, would really think that would be an easy trip to do single-handedly — a 29-foot boat is not easy to steer around the Intercoastal Waterway. Anyone who had common sense would want to take somebody with them for that.”

Some of Gib’s friends and family also were critical of his decision to go off alone, but after 40 years together, Gib’s wife Marcia knew this voyage was Gib’s way of fighting the disease.

Now semiretired, Bradley says he often used to advise his ALS patients that the disease is not always quickly fatal — and in very rare instances even can “burn itself out — so don’t kill yourself because there’s a possibility this disease won’t kill you!”

Nonetheless, he is supportive of people with ALS who, like Gib, want to follow a crazy — even dangerous — dream. “It all depends on your character and what you want to do with your life.”

Book Review: Sorting Out ALS at Sea

I Reach Over: Spiritual Correspondences on ALS, Death and Living, Scott B. Shappell. This collection of poetry written by a physician-scientist combines science and spirituality as a way of coming to terms with ALS.

ALS, Yet Healthy & Healed, Dee Jordan. This short, self-published volume takes a Christian perspective on living with ALS.

Patrick and the Giant by David McNelis; Dancing the ABCs by Kimberly Kim; and Your Ema Loves You by Eloise Lovelace all are children’s books written in response to their authors’ ALS.

More reviews online

These additional book reviews can be found in the online version of this article at alsn.mda.org (July-August 2011 issue):

MDA/ALS Newsmagazine • July/August 2011
The National Amyotrophic Lateral Sclerosis (ALS) Registry

It's Here!

www.cdc.gov/als
(800)232-4636

Josephine of Wisconsin worked at a major newspaper in Milwaukee until her retirement in 2002, despite being challenged by the effects of muscular dystrophy. Throughout her career, she purchased company stock to supplement her income and enhance her future.

At the end of 2007, Josephine, like many individuals, took time to assess her financial strategies. By donating a portion of her securities to MDA, she found she could receive a significant capital gains deduction, while helping to fuel MDA’s fight against muscular dystrophy and related diseases.

This year, please review your charitable-giving priorities with your financial advisers, and consider how a gift of stocks, bonds or mutual fund shares can help to assure maximum benefits for you — and for the important work of MDA.

Please contact MDA’s Special Gifts & Philanthropy Department for more information by calling (800) 223-6011 or emailing MDA’s Rick Brown, Ed.D., CFRE, at rbrown@mdausa.org.

Important Tip: Don’t sell the shares before contacting MDA, as the IRS will impose a capital gains tax on your sale.