AFSA The American Fibromyalgia Syndrome Association, Inc.

UPDATE

Organizational News and Activities

Visit our website at: www.afsafund.org

VOL 15, ISSUE 1 NOVEMBER 2009

In this Issue ...

From the President .. p.1
Electrocardiogram-Based Sleep Spectrogram p.1
Genetic Influences on Pain Modulation Systems in Fibromyalgia p.3
AFSA Donor Recognition p.4
Gene Expression Biomarkers for Fibromyalgia p.6
Are Myofascial Trigger Points Contributing to Your Pain? p.7
Role of Myofascial Trigger Points in FMS - Part 2 ..... p.7

Mission:

AFSA is an all-volunteer nonprofit organization dedicated to funding research that investigates the causes and treatments for fibromyalgia syndrome.

The American Fibromyalgia Syndrome Association, Inc.
PO Box 32698
Tucson, AZ 85751
(520) 733-1570
(520) 290-5550 Fax
kthorson@afsafund.org

AFSA—a 501(c)3 Charitable Nonprofit Organization.
Tax I.D. #77-0355224

A Special Message From the President

In 2008, no one knew for sure if all fibromyalgia syndrome (FMS) patients had myofascial trigger points, and if they did, whether the count was just one or two. Based on two AFSA-funded projects, it seems clear that multiple trigger points significantly contribute to FMS pain. Now a newly funded proposal focuses on the impact of treating these painful knots in the muscles.

The other three projects funded this year all pertain to developing disease severity markers for FMS. One uses a highly scientific yet patient-friendly approach to detecting abnormalities occurring during sleep, the second looks at genetic influences, and the third examines the unusual effects that a half hour of exercise has on the sensory, immune, and neurologic systems. Each one of these projects will greatly build on the science of FMS, and will assist with the prompt diagnosis and effective treatment of people with this condition.

Despite these harsh economic times, AFSA brings promising new research to the table for FMS patients. I hope you will readily see why your thoughtful support is most appreciated!

Kristin Thorson, *president and founder*
kthorson@afsafund.org

Electrocardiogram-Based Sleep Spectrogram
—using sleep as a window into fibromyalgia

Principal Investigator: ***Robert J. Thomas, M.D.***
Harvard & Beth Israel Deaconess Medical Center
Award: $60,000 (September 2009)

Impaired quality of sleep is a dominant clinical feature of fibromyalgia syndrome (FMS) that is not well understood. The standard approach to measure sleep uses the electroencephalogram (EEG) to depict brain wave activity. In general, this approach has not been useful in identifying characteristics specific to people with FMS, nor can it help explain why patients are so unrefreshed following a night of apparently "good" sleep. Even with additional techniques to evaluate breathing (which is helpful for the accurate diagnosis of sleep apnea), an overnight sleep study doesn't readily explain any abnormal physiology that may be occurring during the night.

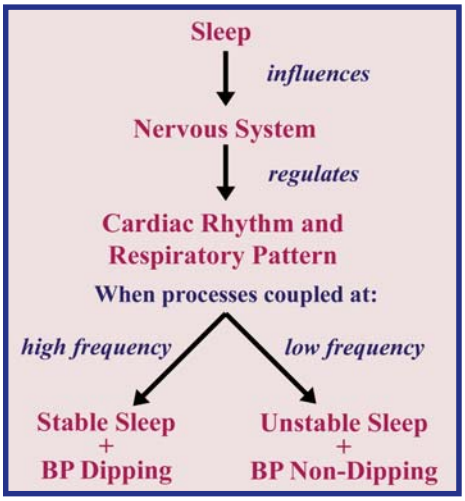
Sleep processes taking place in the brain strongly influence the autonomic nervous system, which in turn, regulates heart rate and breathing. **Robert J. Thomas, M.D.**, will use new technology that evaluates the interactions (or coupling) of the cardiac rhythm and respiratory pattern during sleep using information taken from the electrocardiogram (ECG).

"This technique can easily differentiate stable or effective sleep, which allows normal nighttime functions, and unstable or ineffective sleep, which may not allow normal sleep functions to occur," says Thomas. "Sleep

disrupting conditions reliably decrease stable sleep, while sleep enhancers increase it. This information is obtained without requiring a technician to place numerous electrodes on the skull for an EEG that measures brain wave activity. Instead, the ECG equipment involves a monitor that patients strap around their chest, making it easy to use as often as needed."

A 3D-looking chart generated by the ECG technique is called a sleep spectrogram and provides information about how well the heart, lungs, and nervous system are coordinating efforts during sleep. When these three systems are coupled at the frequency of respiration (8 to 20 times a minute), a person's sleep is very stable (e.g., good, restorative sleep). When these systems are coupled at a lower frequency, about 2 times a minute, sleep is unstable (e.g., fragmented with lots of arousals and unrefreshing). During sleep, there seems to be little if any overlap between the higher and lower frequencies, making it relatively easy to distinguish between potentially restorative and non-restorative sleep states.

The ECG may be useful for detecting sleep disturbances in FMS patients that are not apparent by looking at the brain waves. In one small study, FMS patients showed a significant reduction in the stable and restorative interactions between heart rate and respirations (in press, Sleep Medicine 2009). At the same time, carefully done conventional sleep studies using an EEG to measure REM and non-REM brain stages did not provide useful information.



Thomas proposes to use "sleep as a window into FMS" and hypothesizes that the sleep spectrogram can be a useful marker of disease for this condition. "It does not matter what causes FMS," says Thomas, adding the analogy that "a thermometer can show fever regardless of cause. The ECG technique can be used as a 'sleep thermometer.' It has been applied to more than 10,000 sleep studies in people of all ages. The patterns of undisturbed sleep are consistent and predictable."

"Development of disease severity markers will greatly assist with monitoring progress in drug trials and the use of other novel therapies."
—Robert J. Thomas, M.D.

Blood pressure (BP) normally drops during restful sleep (called blood pressure "dipping"). Brief awakenings and persistent disruptions from sleep reliably cause BP increases, and the profile is referred to as "non-dipping." Until recently, it was not possible to readily measure BP continuously during sleep because a tight-fitting finger cuff could disturb the natural flow of the nighttime processes. However, recent research in primary insomnia patients shows BP non-dipping.

"A new approach to measuring BP throughout the night makes use of a simple finger probe that is currently in standard clinical use for measuring blood oxygenation levels," says Thomas. "We propose to show that FMS patients are non-dippers—their BP will remain increased during sleep because sleep itself is disturbed. The combination of the ECG-based sleep spectrogram and simultaneously measured continuous BP could provide a useful marker of FMS."

The ECG and BP will be recorded for seven consecutive nights from10 healthy subjects and 20 patients with FMS who are free of medications and without any other painful or psychiatric diseases. "Recording many nights at a time will reduce the impact of changes that can be seen on different nights," says Thomas. "It also allows us to determine if next-day pain and discomfort are related to poor sleep." In addition, Thomas will simultaneously evaluate each participant's sleep with the standard EEG-type of equipment to allow comparisons between the various measures.

Although the small, comfortable devices for measuring the ECG and BP are designed for patients to use at home, all evaluations for this study will take place in the clinical research facilities at Beth Israel Deaconess Medical Center. Performance measures on cognitive tasks, vigilance, and function will be assessed in a consistent, controlled, and quiet environment. As a result, Thomas will be able to explore the relationship between sleep and performance in people with FMS.

"The ECG and BP devices are simple enough to use by patients for monitoring treatment effects, but first we must show that this method provides important information," says Thomas. "Development of disease severity markers will greatly assist with monitoring progress in drug trials and the use of other novel therapies. The absence of reliable markers of abnormal physiology in FMS has presented a major challenge that we hope to overcome. Sleep then becomes a window that allows clinicians and researchers to look into and track the disease state of FMS." **END**

Genetic Influences on Pain Modulation Systems in Fibromyalgia

Principal Investigator: ***Serge Marchand, Ph.D.***
University of Sherbrooke, Quebec, Canada
Award: $55,000 (October 2009)

"There is a genetic predisposition to developing fibromyalgia syndrome (FMS), so why not look at the main neurotransmitters that play a role in pain modulation?" asks **Serge Marchand, Ph.D.** Both dopamine and serotonin are involved in providing pain relief through the diffuse noxious inhibitory control (DNIC) system, while glutamate is known for its role in turning up the volume on the pain amplification system. Various genetic abnormalities that alter the function of dopamine, serotonin, and glutamate in the central nervous system will be evaluated in this study.

The DNIC operates on the basis that pain inhibits more pain. When a person is first subjected to a noxious stimulus, the DNIC system releases pain-relieving substances into the spinal cord to reduce the impact of additional discomfort. Marchand has already shown that this system is not working well in FMS patients, but he stresses that there is a lot of variability.[1] "Some healthy subjects have a very strong DNIC and others have such a weak DNIC that if we did not know better, we would think that they were FMS patients," says Marchand. "There are likely certain genetic predispositions that determine the strength of a person's DNIC system."

The DNIC uses serotonin, norepinephrine, and dopamine transmitters that cause the release of opioid-like pain relievers. Given that FMS patients have double the normal concentration of opioids in their spinal fluid, their faulty DNIC is probably not due to a genetic inability to produce opioids. However, genetic abnormalities pertaining to serotonin, norepinephrine, and dopamine have been identified in FMS and will be examined.

Sleep disruption is another factor that can interfere with DNIC function. Yet, it is not known what role this sleep disorder in FMS plays in the lack of DNIC response. As a result, all study subjects (70 FMS patients and 70 healthy controls) will wear a watch-like actigraphy device that monitors movement during the entire week before the DNIC and other pain sensory testing. Not only will this provide a measure for how disturbed a person's sleep is, it will also produce an estimate of physical function.

The role of dopamine in FMS has garnered more attention in recent years, partly due to the efforts of **Patrick Wood, M.D.**, and **Andrew Holman, M.D.**, both in Renton, WA. Wood showed that dopamine activity in the brain was greatly increased in healthy subjects given a painful stimulus, but not in FMS patients.[2] Testing a dopamine-like drug, Mirapex (pramipexole), Holman documented significant FMS symptom relief in a small group of patients.[3] And Marchand has already collected preliminary data that will be expanded upon in this AFSA-funded study to evaluate the influence of the dopamine 3 receptor (D3) in FMS.[4]

A genetic mutation, called the Ser9Gly polymorphism, can alter the sensitivity of the D3 receptor. A less sensitive receptor would require more dopamine to produce the same results, such as the release of opioid-like pain relievers. The preliminary data indicates that the less sensitive (poorer functioning) D3 receptor lowers the heat pain threshold in FMS patients but not healthy control subjects. Why the differing effect? FMS patients already have signs of abnormal pain processing in their spinal cord (e.g., elevated pain transmitters like substance P and glutamate), so having genes that make things worse will have more of an impact on pain thresholds.

"The role of DNIC across the natural menstrual cycle is a good example," says Marchand, who just published a study on this topic.[5] "In healthy women we found that the DNIC was really strong in the mid-phase of the cycle and then drops off. If you have chronic pain, then during your pre-menstrual phase when your DNIC is so weak, it is not blocking pain and could make a significant difference to your pain threshold."

In addition to evaluating the neurotransmitters involved in DNIC, Marchand will look at the pain amplifying system. Increased glutamate levels are believed to play an important role in magnifying FMS pain, and he will measure two enzymes that could help explain glutamate's role in pain.

"With regards to patient relevance, it would be important to show different genetic markers exist between people with FMS and healthy subjects, as well as other diseases like Parkinson's, for example," says Marchand. "If we can show that one neurotransmitter plays a larger role in generating the symptoms of FMS, this can alter our therapeutic strategies." More than likely, he expects to identify different subgroups of patients, some that would respond favorably to dopamine-like drugs, while others that might need a boost in serotonin or could benefit from drugs that reduce glutamate. **END**

1. Julien N, *et al.* PAIN 114:295-302, 2005. **www.afsafund.org/research_2000.html**
2. Wood PB, *et al.* Eur J Neurosci 25:3576-82, 2007.
3. Holman AJ, Myers RR. Arthritis Rheum 52(8):2495-505, 2005.
4. Potvin S, *et al.* J Pain 10(9):969-75, 2009.
5. Tousignant-Laflamme Y, Marchand S. PAIN 146:47-55, 2009.

Donor Recognition
October 1, 2008 - September 30, 2009

The American Fibromyalgia Syndrome Association wishes to thank each and every individual who has made a contribution. In its 15th year of service, AFSA continues as the nation's leading all-volunteer, non-profit organization dedicated to funding research to improve the quality of life for patients with fibromyalgia. While other organizations say they support fibromyalgia, we put your money directly toward research that accelerates the pace of medical discoveries.

We are pleased that in spite of a very difficult economy this past year, you were able to help us continue this important battle. As a reminder, more than 90 percent of your donations go directly to fund scientific studies that have been scrutinized and approved by our Medical Advisory Committee of esteemed professionals. Together, your contributions are making a substantial difference in the lives of millions.

Friends
($25 - $49)

M. Joan Aceves
Nancy Alberino
Marcia Amell
Lucia G. Belsujet
Ellen Berkowitz
IMO Patti Adams
Mary Anne Birchard
Ellis and Robin Blood
Bonnie Bogardus
Mary Bowick
Karen Brakke
Mary M. Bryson
Lee Cali
Anonymous
Luise-Margarete Cashin
James and Glynda Childs
Anonymous
Ruth Crafts
IHO Bruce and Andrea Kameron
Marilyn V. Davidson
Ralph and Diana Delamarter
Dennis and Jeanette Doyle
IMO Dr. Alfred W. Thatcher
Flowayne Drew
Susan W. Durbin
IHO Morgon Forrest
Lowell and Hazel Easter
Richard and M. Clare Fair
Randi Frank
Patricia N. Gaida
Hilda G. Getz
Robert Goldstein
Blanche Groark
Dorothea S. Hanich
Dr. and Mrs. Joseph Henke
IMO Ruth Malling
Barbara Hill
IMO Elisabeth Steckner
Milton and Connie Hinnant
Tina Hopkins
Jerry and Judith L. Horn
Ernestine J.B. Ilcken
David Jacobs
Karla L. Jenkins
Kelley M. Keeley
Alice Jane Kidd
Anonymous
Alan Kwong
Esther Lang
Anonymous
Connie Lipman
Lori Lipman
IHO Connie Lipman
Charles Louther
Gloria H. Lurie
Jennifer P. McDonald
Morris and Gilda Minkin
IHO Dorothy and Bernard Kornheiser
William and Cynthia Mobraaten
Anonymous
Michael Morton
Patrick Murchek
Rodney and Brenda Norris
Rosalyn Olinger
Linda Orr
Loretta Ozimek
Earl and Anita Pakkala
Kathy Pavich
Terry and Linda Peterson
Anonymous
Christine Preuss
Tom and Marcia Price
Donna A. Prisbrey
Linda Provost
Alice Rau
George and Dolores Renner
IHO Linda Renner
Joan Rosenthal
Patricia F. Rundy
Howard Scalone
Pearl R. Sfredda
Kenneth E. Shull
Judith Ann Sibley
Evelyn Sienkiewicz
Laurie E. Simons
Cynthia S. Sokol
Mary Virginia Sommers
Sandra R. Spafford
Reitha Stephens
Michele S. Stern
Thomas Stuckey
Evangeline Swenson
Elizabeth Taylor
Karen Tepper
IHO Lerner-Tepper Family and Friends
Edna M. Unander
Leon J. Valencia
Jayne Volan
Michael Walton
Lauri A. Wegener
Allen and Myrna Woodworth
Paulette D. Wright
Barbara Young

Your Contributions Make a Difference

AFSA is solely supported by generous donations from patients, their families, and other interested individuals. As an independent organization, AFSA does not receive money or support from the government, pharmaceutical, or marketing companies. Projects with the greatest promise to benefit patients are selected without third-party influence.

We encourage you to tell us what types of projects or areas of study you feel we should fund. E-mail Kristin Thorson at kthorson@afsafund.org or write to us. We would be grateful for your input and your support.

Ways to Make a Tax-Deductible Donation:

- Online at **www.afsafund.org**
- Mail your contribution to:
  AFSA
  PO Box 32698
  Tucson, AZ 85751
- Send us the attached envelope with your check, money order, or credit card information.
- Call us at **(520) 733-1570**

Inspirations
($50 - $99)

Misha Backonja, M.D.
Judy Baker
Elizabeth Barge
Edyth J. Bassett
Marilyn B. Bendt
Jens and Christine Braun
Anonymous
Margaret Christmann
Thomas and Julia Davis
Marvin D. Drake, Ph.D.
Dana Ellingson
Suellen Eslinger
Jeanette Faust
Anonymous
Cynthia Gilpin
Charlotte Garavglia
Dorine Greene
Anonymous
Nancy P. Hallman

IMO Elisabeth Steckner
Peter and Kathryn Hanas
Jay and Pamela Hilbert

IMO Patti Adams
Diane A. Huckleberry
Richard and Lynn Hutchinson

IHO Althea Hutchinson
Gail Ito
Jean E. Ives
Charles Kerns
Anonymous
Karrie Kunke
Pamed Le Resche

IMO Doris Hughes
Enid Lythgoe
Michael R. MacMillan
Robert and Marlene Malin
Elizabeth G. Marino
Anonymous
Agnes K. Martin
Elizabeth Masi
Jennie McLean
Martha L. Moody
William and Polly Moore
Pam M. Munro

IMO Doris Hughes
Robert and Patricia Myers
Jeune Nicolai
David Nicols
James and Joan Nielson

IMO Helen Landby
James Nugent
Rachel Kisber Obermeier

IMO Patti Adams
Jean Brooks Oliver
Pfizer Foundation (Matching Gift)
Marilyn Rabalais

IMO Craig Rabalais
Mrs. George Reaves
Pauline Rice
Ruth M. Ritters

Every effort has been made to ensure accuracy and completeness of this list. If you find an error, please accept our apology and call (520) 733-1570. Thank you.

Elizabeth R. Schaeffer
Kathleen Smith
John and Celia Synder
Frederick and Susan Stater
Gloria M. Steffens
Patsy W. Stuckey
Tetsuya Takagi
Dreama Thompson Boyd
Carey Toran
Gordon and Dianne Vogt

IMO Irene Wieland
Patricia and Deborah Walters

IMO Doris Hughes
Sheila T. Ward
Kenneth and Susan Weinstein
Stephen and Alana Willens
Keith and Nancy Young

IMO Patti Adams
Zoccoli Family

Motivators
($100 - $499)

Linda Alhadeff
Raffeala J. Allen
Gary and Diane Andresen
Anonymous
Eugene W. Baer
Anonymous
Timothy and Carol Barrett

IHO Katie Barrett
Barbara M. Berry
Sharon Bos-Karnoski
Anonymous
Jennifer Brynn
Renee Burton
Kathryn Checchi

IMO Elisabeth Steckner
Martha and Thomas Churchill

IMO Elisabeth Steckner
Anonymous
Jacqueline G. Cleveland
Sue H. Crawford
Rosalie A. Crispin
Herbert A. Cubberley
Beatrice A. Danko
Terryl A. DeBruin
Diedra A. Defreese
William and Sandra Derman
Alice Dowling
Marvin Drake

IMO Harold Drake
Timothy Ekdom
Hoffman Electric, Inc.
Richard and Joanne Elliott
Patricia Ellsworth
Inge Erickson
Thomas Ervin
Kathleen Esdale
Ben and Peggy Feldman
Hazel Frasca
Robert P. Fruh
Michelyn Gallagher
Herbert and Barbara Ginger
Nancy Glodowski
Eileen B. Goldsmith
Ronald and Pamela Gregg
David and Glynda Hairston
Mr. and Mrs. Michael Hardy
Kathryn Heyner
Kathleen M. Hnath
Sandra Ho
Anonymous
Sandra Joy
Barbara Joyce
Kathleen Kaplan
Anonymous
Jonas Kisber

IMO Patti Adams
Allen and Sharon Krause
Rev. David and Judith Liefeld
Katherine A. Liefeld
Mary E. Loree
Elinor MacDonald
Paul and Caren Masem
Emilie Mason
Corey McCarthy
Melissa and Thomas Monahan
Cornelia and Terry Moore
Janice Myrick
Kent and Jeannette Nicholson
Robert and Terry Nicols
Margaret M. Paul
O.E. Perez
Anonymous
Andrea Pines
Clifford and Robin Plopper
Anonymous
Mary and Don Randa
Robert and Barbara Reynolds
Anonymous
Anonymous
Charmaine Rutkowski

IMO Doris Hughes
Edward Scott
Kathryn Scott
Donald and Caryl Sedushak
Lauren Sheriff
Barbara A. Smith
Miriam Soppeland
Devin J. Starlanyl, M.D.
Sarah Stewart
Meri Thomsen
Marsden and Karen Tielkemeier
Cheryl Toftey
Delton Troyer
Diane E. Warner
Michelle T. Weatherbee
Richard and Lucinda Yeasting

Visionaries
($500 - $999)

Catalyst Foundation
Pat Cannell Coyle
Kristin M. Hartl
Linda Kmetz

IMO Cheter Kmetz
Patricia McDaniel
Diane Phillips
Eric M. and J. Lynn Preston
Carol Selassie
John Tollini
Lois P. Vrhel

Champions
($1,000 - $9,999)

Susan Cherry
Tulsa Community Foundation
Linda Ferrero-Sheppard
Qualcomm Matching Gift Program
Carl and Henrietta Herrmann

IHO Carol Johnson
Katherine J. Johnson
Ben Pauley
Douglas and Judy Palenschat
Anonymous
George and Margaret Saunders
Susan Stoddart

Gene Expression Biomarkers for Fibromyalgia

Principal Investigator: ***Alan Light, Ph.D.***
University of Utah in Salt Lake City
Award: $50,000 (September 2009)

"One of the critical needs in FMS research is to develop truly objective biomarkers for the disorder," says **Alan Light, Ph.D.** "Blood-based biomarkers are especially valuable because they are consistent with traditional medical models, they are viewed as unbiased, and they are relatively low risk for patients (compared to cerebrospinal fluid or tissue sampling markers)."

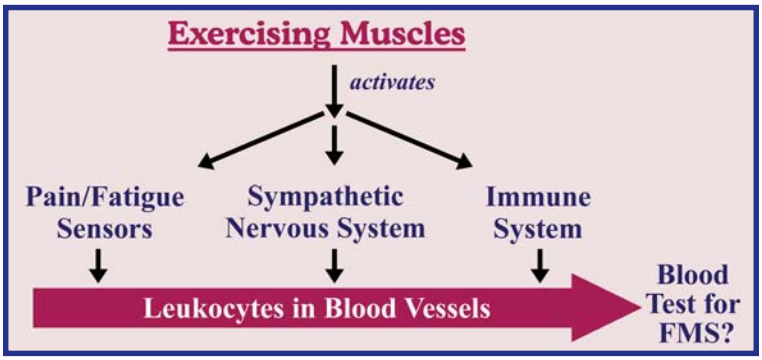
Light has teamed up with his wife **Kathleen Light, Ph.D.**, and together they have overcome the obstacle of the blasé blood work in which everything comes back "normal." A preliminary study shows dramatic differences in many test markers between patients with both chronic fatigue syndrome (CFS) and FMS, compared to healthy controls.[1] All of the subjects were required to perform moderate exercise on a stationary bicycle for 25 minutes.

What does exercise have to do with blood markers and your symptoms? "Muscle pain and both physical and mental fatigue are sensations that a healthy body uses for protection or as a warning signal to prevent overuse of muscles and important areas of the brain," says Light. "However, in FMS and CFS patients these sensations are overwhelming and unrelenting, and interfere with normal life activities."

Based on research in mice, Light identified the specific receptors that are located on the nerve fibers traveling through the muscle, which allow the sensory nerves to detect the chemicals produced by muscle activity.[2] For example, they sense how much energy the muscle has consumed or the amount of lactic acid accumulated. Once a muscle's activity produces a certain level of chemicals, these sensory fibers send a signal to the brain that is interpreted as muscle fatigue. Continued use of the muscle will eventually signal the brain with a message that is perceived as pain.

The sensory nerves in the muscles are alongside the blood vessels. Light found that the white blood cells (or leukocytes) are like magnets picking up information from the sensory fibers, sympathetic nervous system, and the immune system (see below). By measuring the leukocyte messenger RNA (mRNA), which contains the genetic blueprints for making what the cells need, the Lights can look for gene expression test markers.

"The amounts of mRNA did not change in healthy control subjects following moderate exercise, nor did they experience any post-exertional fatigue or pain," says Light. "The picture was different for the patients with CFS, of which 70 percent also had FMS. The mRNA that create receptors increased rapidly and remained high for at least 48 hours after the same exercise task." Certain receptor increases tended to correlate with symptoms of mental and physical fatigue. Other receptors tended to increase in conjunction with the symptom of pain.



What might it be like when the number of receptors on sensory nerve endings increase? Light has a hypothesis: "The small amounts of muscle activity involved in sitting or walking will more readily activate the receptors, causing them to send signals of fatigue and muscle pain to the brain as if the body was always overworking."

Light's work has shown that exercise triggers a substantial increase in mRNA in CFS-FMS patients. Now it's time to look at specific conditions and symptoms. In this AFSA-funded project, 15 people with FMS only and 15 people with regional low back pain will be evaluated by the same exercise protocol that was used in Light's preliminary study. In a separate study funded by the CFIDS Association of America, Light is already testing CFS-only patients and additional healthy controls. All four groups will provide valuable comparisons.

"We want to determine if patients with focal or low back pain show a regulation of the sensory receptor pathways that is different than patients with the widespread muscle pain of FMS," says Light. "Our long-term goal is to create a blood test that can be used to objectively diagnose FMS and CFS, as well as distinguish these disorders from other painful or fatiguing conditions. The biomarkers could also be used for early interventions, new therapies, and could shed light on the causes of the primary symptoms of FMS and CFS." **END**

1. Light AR, *et al.* J Pain 10:1099-112, 2009.
2. Light AR, *et al.* J Neurophysiol 100:1184-201, 2008.

Are Myofascial Trigger Points Contributing to Your Pain?

Two AFSA-funded researchers, **Hong-You Ge, M.D., Ph.D.**, of Denmark, and **César Fernández de las Pénas, P.T., Ph.D.**, of Spain, addressed the question of myofascial trigger points (MTPs) and pain in studies this past year.

Fernández de las Pénas' group at the University of Rey Juan Carlos in Madrid, already presented preliminary data at the September European Federation for the Study of Pain in Lisbon, Portugal, and the first of many reports was just published by Ge and his team at Aalborg University. [1] Based on the findings of both investigators, active myofascial trigger points (MTPs) are present in all fibromyalgia syndrome (FMS) patients. In addition, these firm nodules that can be found in tight, ropy muscles significantly contribute to your pain.

Eighteen tender points are used to diagnose FMS, but the presence of 12 or more signify a lowered pain threshold and an inability of the central nervous system to regulate pain. MTPs also cause a lowering of pain thresholds and are most predominantly identified in the two large triangular trapezius muscles that span from the base of the skull down to the mid-back and out to the shoulder joint. In the 22 FMS patients, Ge found an average of seven active MTPs on each side in just the upper portion of the trapezius muscle and none in the 22 healthy control subjects.

"The aim of this study was to learn how many MTPs exist in the upper half of the trapezius muscle," says Ge. Until this carefully designed study was done, no one knew how many active MTPs might exist for a single muscle in FMS patients, although this particular one takes a real beating from daily activities.

The most common areas for the active MTPs were along the upper ridge of the trapezius on each side of the neck. These areas also corresponded to the lowest pressure pain thresholds. If you place your hand on top of your collar bone, your fingers will extend to detect the upper ridge and belly of the trapezius midway

Continued on back cover ...

Role of Myofascial Trigger Points in FMS - Part 2

Principal Investigator: ***Hong-You Ge, M.D., Ph.D.***
Aalborg University, Denmark
Award: $20,000 (October 2009)

Now that **Hong-You Ge, M.D., Ph.D.**, and his colleagues have demonstrated that myofascial trigger points (MTPs) are an important contributor to the painful symptoms of FMS, Part 2 of this project focuses on treatment. Ge will recruit 60 FMS patients who will be divided into two therapy groups: active and placebo. The active treatment will consist of using acupuncture needles to inactivate the MTPs during twice weekly sessions for four to five consecutive weeks. The insertion of tiny acupuncture needles into the heart of the MTP forces the area of sustained contraction to relax. This treatment approach, called dry needling, releases the nasty chemicals that are held within the muscle knot and allows for improved circulation.

The placebo group will meet for the same number of therapy sessions as those in the active group, except instead of inserting the needle into the MTP to release it, the needles will only penetrate the top skin layer. All patients will be evaluated before and after treatments by a skilled examiner. Neither the patients nor the examiner will know who has been assigned to the active therapy group. To assess how robust the treatment protocol is for the inactivation of the MTPs, the examiner will assess patients in the "active" group at three, six and twelve months after the end of the therapy sessions.

In previous studies involving healthy subjects, Ge has shown that painful stimulation of latent MTPs (injecting them with a chemical irritant) causes an increase in sympathetic nervous system activity that leads to constriction of nearby blood vessels.[2] Latent MTPs do not produce spontaneous pain so people are not aware of them unless they are pressed, or in the case of Ge's experiments, injected with a chemical irritant. Based on these studies, one might ask if the sympathetic nervous system's hyperactivity that is well-documented in people with FMS might be reduced upon inactivation of MTPs.

"Sympathetic activation is one of the perpetuating factors that leads to the formation of MTPs," says Ge. An important question to answer is: Which came first, the MTPs or the sympathetic hyperactivity in people with FMS? Ge will be evaluating the level of sympathetic nervous system activity in all subjects. "If deactivating trigger points leads to decreased sympathetic activity, then it will prove that MTPs are one of the causes of sympathetic hyperactivity in FMS."

AMERICAN FIBROMYALGIA SYNDROME ASSOCIATION
PO BOX 32698
TUCSON, AZ 85751-2698

NONPROFIT ORG.
U.S. POSTAGE PAID
TUCSON, AZ
PERMIT NO. 1315

ADDRESS SERVICE REQUESTED

... continued from page 7

between your shoulder joint and neck. A diagnostic tender point for FMS is also located in this area, along with one at the base of the neck.

Active MTPs cause spontaneous pain in these areas as well as referred pain to other regions. People are very aware of active MTPs because they really hurt and restrict movement. On the other hand, latent MTPs do not cause spontaneous pain and only hurt when pressed. Usually people are unaware of latent MTPs, so it should be no surprise that the pain-free control group had several latent MTPs in their upper trapezius. In fact, the FMS patients also had many latent MTPs.

Prior to evaluating the FMS patients, Ge's group asked them to shade in the areas of a body diagram (front and back) that reflected their spontaneous pain pattern. Most all areas of the body were shaded, with some more densely filled in than others because of the body-wide nature of FMS. After each MTP (latent or active) was identified, all subjects shaded the body diagram to show the local and referred pain patterns produced by pressing on the MTP in the upper portion of the trapezius muscles. The area of the pain induced by the MTPs was about ten times greater in the patients than the control subjects. This pain extended to the head, face, neck, shoulder blades, mid and lower back, upper chest, and down the arms all the way to the fingers.

"This study confirms our hypothesis that active MTPs contribute to fibromyalgia pain."
—Hong-You Ge, M.D., Ph.D.

Additional findings from this project will be published soon, but for now, Ge says, "This study confirms our hypothesis that active MTPs contribute to fibromyalgia pain." Naturally, it's essential to determine what impact deactivation of the MTPs have on the pain and other symptoms in people with FMS, *which is the focus of Part 2 in Ge's project that was recently funded* (see text box on page 7). **END**

1. Ge HY, *et al.* PAIN Oct. 8 [Pub. ahead of print] 2009.
2. Zhang Y, Ge HY, *et al.* Arch Phys Med Rehabil 90:325-32, 2009.

AFSA Donation Form

The American Fibromyalgia Syndrome Association, Inc.

PO Box 32698 • Tucson, AZ 85751-2698

Phone: (520) 733-1570 • Fax: (520) 290-5550 • www.afsafund.org

Name: _____ ID (if available): _____

Address: _____

City: _____ State: _____ Zipcode: _____

Country: _____ Phone: (_____) _____
(required for credit card donations)

E-mail: _____

Annual Contribution Categories

- ❑ Friends $25
- ❑ Inspirations $50
- ❑ Motivators $100
- ❑ Visionaries $500
- ❑ Champions $1,000
- ❑ Heros ..... $10,000+
- ❑ Any size contribution is welcome! ..... $_____

❑ *Check here if you would like your donations of $50 or more to be anonymous (i.e., no mention in the Update or Web site).*

❑ *Check here if you would like to be mailed instructions on how to make a donation of stock/securities.*

Memorials

❑ This donation is made **in memory of:** _____

Please tell us who we should notify that you have made this donation:

Name: _____

Address: _____

City: _____ State: _____ Zipcode: _____

This person being notified is the _____ of the deceased.
(Relationship, i.e., mother, sister, daughter, etc.)

AFSA Note Cards

❑ 12-card pack w/envelopes ($10 each) Quantity: _____ x $10 Total: $_____

❑ 24-card pack w/envelopes ($18 each) Quantity: _____ x $18 Total: $_____

Visit our website at **www.afsafund.org** to learn more about our beautiful awareness-raising note cards.

Payment Method

Total Donation Amount: $ _____

(Please make checks payable to AFSA)

❑ Visa ❑ MasterCard ❑ American Express ❑ Check / Money Order Enclosed

Card Number: __ __ __ __ __ __ __ __ __ __ __ __ __ __ __ __ Expiration Date: __ __ / __ __

Authorized Signature: _____ Date: _____

AFSA — a 501(c)3 Nonprofit Charitable Organization (Tax ID Number: 77-0355224)