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Mission:

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

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

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UPDATE

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A Special Message From the President

The American Fibromyalgia Syndrome Association (AFSA) is off to a great start this year. We funded a novel treatment trial and anticipate exciting results from previously awarded projects due out in the near future. In addition, six study proposals are under review. None of this progress would have been possible without the generous contributions from fibromyalgia patients and their families. For 14 years, AFSA has been the sole source of non-government research grants in this field. As you read through this *AFSA Update*, I hope you will see why your support is essential for helping to get promising research ideas off the ground so that more effective treatments can become a reality.

Over 90 percent of the contributions AFSA receives go to its research-funding mission. AFSA awards the most relevant patient-focused grant applications that aim to have the greatest impact on relieving the symptoms of fibromyalgia.

Your thoughtful support is appreciated,

Kristin Thorson

Newly Funded Study: Low-Dose Naltrexone for the Treatment of Fibromyalgia

Principal Investigators: *Jarred Younger, Ph.D., and Sean Mackey, M.D., Ph.D.*
Stanford University, Palo Alto, California

Award Amount: \$50,000

Relief from chronic pain is difficult to achieve. Most conventional treatments target neurons; but a second target – microglia – may be as important or more important in some types of pain. There are a few existing drugs that have the ability to modify microglia activity. Naltrexone is one of them.

Under normal circumstances, microglia cells provide support to neurons by cleaning up debris and repairing damaged neural tissue. The microglia are not involved in the normal transmission of pain. However, many substances that activate microglia are

elevated in the spinal fluid of people with fibromyalgia syndrome (FMS). Once activated, microglia produce a surge of inflammatory substances that can irritate the nearby pain-transmitting nerve fibers and lead to painful symptoms.

AFSA has funded Younger and Mackey to test the ability of low-dose naltrexone to relieve the pain of FMS. This represents the first treatment trial in FMS that is designed to target the microglia instead of the neurons. In normal doses (around 50 mg), the drug blocks pain-relieving chemicals such as endorphins (the body's own opioids). This

would certainly not be desirable for people with FMS, but naltrexone also prevents the activation of microglia and its resulting pain-promoting substances. The key is to use a small dose of naltrexone (3 - 4.5 mg) that deactivates the microglia while not appreciably interfering with the body's own opioid pain relievers.

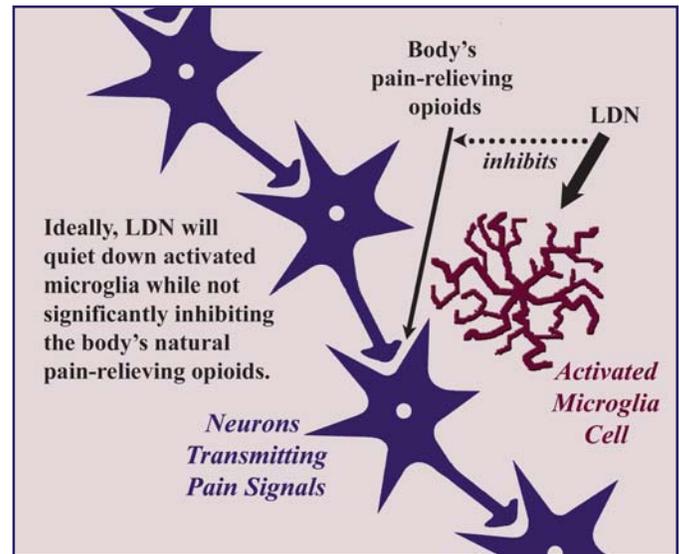
The ability of naltrexone to shut down the pain-promoting activities of the microglia was recently discovered and appears to be safe. It has been tested in small trials for the treatment of Crohn's disease and multiple sclerosis pain. Now it will be determined if the drug can benefit individuals with FMS.

Forty people with FMS will participate in the four-month trial. For part of the study, these individuals will receive low-dose naltrexone (LDN) every night before bedtime. For another part of the study, they will receive an inactive placebo substance. Neither the participants nor the researchers will know what the participants are receiving until the study is completed. In a research plan called a crossover design, each

participant has a 50/50 chance of starting on the drug or placebo, and then is switched to the other compound halfway during the study. This way, participants can find out if the drug works for them individually.

Instead of testing 300 subjects, the cross-over design can determine the same drug effect with only 40 people.

Participants will fill out a short questionnaire every night to chart their pain levels, sleep quality, physical activity, mood, and fatigue. These symptom scores will be collected on hand-held computers (personal digital assistants, PDAs) and will allow the researchers to conduct very powerful tests of the drug's effectiveness. Ultimately, the researchers will use the daily data to track symptoms over time, and look at complex relationships



between symptoms. For example, they may see that pain resolves first and then sleep starts to improve a few days later, or vice versa.

Finally, it is well known that not all people will react well to a certain drug. Participants will undergo a variety of tests at the beginning of the study to determine the characteristics of the patients who reap the most relief from the medication. This will help physicians determine which individuals should receive it.

Q & A with Dr. Younger

Why did you decide to study fibromyalgia?

Of all the pain conditions I studied during my postdoctoral training, FMS was the most interesting to me. In other pain conditions, such as rheumatoid arthritis and osteoarthritis, significant advances in understanding the physiological causes have already been made. Therefore, part of my attraction to FMS was based on scientific curiosity. However, it is also important to me that an effective

treatment for FMS would help millions of people. Furthermore, FMS may be connected physiologically to chronic fatigue syndrome, irritable bowel syndrome, and a number of other conditions that we currently do not understand very well. If that is the case, an effective treatment for FMS could also help many others with similar conditions.

Currently, all drug therapies for FMS directly target the function of the neurons (such as increasing serotonin, norepinephrine, dopamine, or slowing down the pain transmission speed). Could you explain how LDN differs?

The important difference with LDN is that it targets microglia cells

and neuropeptides, instead of neurons and neurotransmitters. Neuropeptides can travel longer distances than neurotransmitters, and they have complex interactions with many cell types. They tie together the many systems of the body, bridging the gap between immune, pain, emotional, and other processes. Dysregulations in neuropeptide activity can likely affect virtually every process in the body, including brain function.

The spinal fluid concentration of opioids is almost fourfold greater in FMS patients compared to healthy controls. In addition, the primary opioid receptors in the brain of FMS patients are almost completely occupied. Why?

My guess is that elevated levels of opioids would be the body's natural response to high pain levels. Since pain signals to the brain are being amplified, there is a compensatory rise of opioids, which are produced to help control the pain. The problem may be that in FMS individuals, opioids can cause microglia (immune system cells in the central nervous system) to produce chemicals that actually *increase* pain. If this hypothesis is true, FMS may be a vicious cycle where increased opioid activity creates more pain instead of less, which causes the body to release even more opioids. The end result would be a state in which the body produces opioids at an accelerated rate, but the person still feels sick and in pain.

Why don't you use a higher dose of naltrexone (such as 50 mg) to shut down the microglia activation and bring the opioid levels in the central nervous system back down to normal?

With a high dose of naltrexone, we may solve one problem, but simultaneously create another one. We are trying to suppress receptors on *microglia*, while leaving the opioid receptors on *neurons* alone. The latter receptors are responsible for the body's natural analgesic processes. Therefore, we do not want to interfere with that system. The trick is to find the dosage of naltrexone that blocks the microglia receptors, but leaves the majority of neuronal receptors free to be occupied by helpful neuropeptides such as beta-endorphin. In the future, we may have compounds that specifically target the microglia receptors.

In addition to asking FMS patients about their symptoms, you are measuring pressure pain and heat pain thresholds every two weeks. Why is this important?

The laboratory tests are designed to provide an objective measure of how pain sensitivity changes when

someone takes LDN. Hopefully, the drug will change pain thresholds so that the FMS patients start to look like non-FMS individuals. The real gold-standard of efficacy, however, is still how much the individual's quality of life is improved.

The study participants will use hand-held computers (e.g., PDAs) to chart their symptoms. Could you talk about the benefits of this technology?

Daily measurements are critical to accurately assess drug effects. Many studies have shown that people have poor accuracy when trying to recall their experience over a few weeks. Daily measurements get around this problem and allow us to see how symptoms change over time. While this can be accomplished with paper journals, PDAs are superior because they: 1) are easier to carry around, 2) time- and date-stamp each response to ensure accurate reporting, 3) can ring an alarm when it is time for users to complete a response, 4) can catch user mistakes as they are being made, and 5) remove the problem of inaccuracies that researchers face when typing in all the data from paper journals.

You are first looking for improvements in pain, but you are also looking to see if LDN might improve sleep. Is this because you theorize that if you calm down the microglia, you will reduce the sleep-disrupting cytokines that these cells produce? Don't you also have to worry about blocking the sleep-enhancing cytokines, or is this less likely to happen?

Proper sleep is a critical component of combating FMS, as we know that pain disrupts sleep, and poor sleep in turn exacerbates pain. We do believe that reducing microglia activity may prevent sleep-disrupting cytokines from being produced. Most of the cytokines produced by microglia seem

to be pro-inflammatory, and generally produce unwanted effects in humans. While other cytokines may enhance sleep, LDN should not affect those systems. Despite occasional anecdotal reports about mild sleep disturbances due to LDN, we have had no reports of those problems in our study.

So many drugs on the market that are being used for FMS cause significant weight gain and other unwanted side effects, such as nausea, drowsiness, dizziness, dry mouth, GI dysfunction, etc. What can you say about the side effect profile for LDN?

One of the most exciting aspects of LDN is its reported low side-effect profile. In our study, we ask about side

One of the most exciting aspects of LDN is its reported low side-effect profile.

effects every day, and I explore potential side effects more closely at every laboratory visit (every two weeks). The complaints typically associated with other drugs appear to be non-existent with LDN. So far, the only side effect that participants have is more vivid dreams.

Although you were just funded, roughly, how many patients have you already recruited for this trial?

We currently have 17 people who are enrolled in the study. Our recruitment efforts were greatly helped by the Fibromyalgia Network, who sent out a call for volunteers to its members in our area. We still have some people waiting to start the study, but we are always looking for more individuals. We want to spend the proper amount of time on each participant, so we have capped the number of people who can be enrolled at any given time to 20. As those participants complete the study, we will have new people begin.

END

Update: Fibromyalgia Tissue Donor Program

AFSA was excited to award a groundbreaking grant in 2006 that was pivotal in establishing the nation's first fibromyalgia syndrome (FMS) tissue donor program. After its first year, **Dianne Lorton, Ph.D.**, a neuro-immunologist, had already enrolled 50 FMS patients into the study at Sun Health Research Institute (SHRI, a nonprofit research foundation in Sun City, AZ).

Now, with the additional support of a \$1.4 million grant from the National Institutes of Health (NIH), her goal is to recruit 350 to 500 patients plus age- and gender-matched healthy control subjects within a five-year period.

"The NIH grant goes a long way toward securing the future of the FMS clinical and tissue bank program," says Lorton. "But AFSA, with its foresight to see how beneficial such a program could be in moving our understanding of fibromyalgia forward and developing new treatments, was the first to supply financial support."

Brain, spinal cord, and other tissues/fluids will be collected from FMS donors whose clinical symptoms are evaluated and charted each year. "The SHRI tissue bank is world renown for its quality of post-mortem tissue," says Lorton. She adds that tissues are collected within hours after a donor passes on so they closely resemble how the cells functioned when the person was alive. "This makes it possible to conduct research

projects that would not be feasible with tissue from other banks."

Lorton believes the post-mortem tissue bank program will be able to change the image and the research landscape for fibromyalgia. "It is my firm belief that the program will do for FMS what it has done for Alzheimer's disease."

Much of the earliest and even current research in Alzheimer's disease relied on human studies for developing criteria for diagnosis, pathology, and causes of the illness. As with FMS today, there were no animal models available that truly mirrored Alzheimer's meaning the underlying mechanisms of neurodegeneration and its causes could not be accurately researched, Lorton says.

"Post-mortem tissue studies were critical for moving the study of Alzheimer's disease to where it is today. Now there are good animal models, the cause of the disease is fairly well understood, diagnostics are close to being available to predict early on who will get Alzheimer's, and rational therapeutic approaches are being investigated. Without the availability of post-mortem tissue for this disease, this progress would not have been possible.

"The FMS tissue program will be an invaluable resource for determining the mechanisms underlying the disease and will aid in the development of

therapeutics specific for this illness," says Lorton. "It is my belief that an understanding of the causes, diagnostic tests, development of animal models, and discoveries of potential treatments will not be possible unless central nervous system tissue becomes available for researchers to readily study FMS. This tissue donation program will help to fulfill these needs. Also, making this tissue resource available to other investigators will draw a variety of talented scientists into the field, providing a larger fibromyalgia research community."

Knowing the cause of an illness can in many cases lead to more effective rational treatments.

In time, enough tissue will be available for multiple large-scale projects, but in the meantime, patient recruits may wish to participate in clinical studies. Ten years ago, SHRI established a center for the clinical studies of Alzheimer's and Parkinson's disease, and it is being expanded to include FMS. Aside from testing therapeutic interventions for the key symptoms, such as pain and sleep disruption, a registry of volunteer FMS patients and matched healthy controls opens up the door for other research opportunities. For example, Lorton states that many neuro-endocrine-immune studies could be conducted on the blood and urine samples being gathered. Blood cells collected could be used for genetic studies that may be linked to specific symptoms or risk factors for the development of FMS.

Linking the results of sleep evaluations, brain imaging, and neuro-endocrine-immune studies of patients enrolled, to the results obtained on autopsy is likely to answer many more

Your Contributions Make a Difference

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

Please tell us what types of projects or areas of study you feel should be funded. E-mail kthorson@afsafund.org or write to us. We would be grateful for your input and your support.

critical questions, says Lorton. In fact, greater scientific strides can be made at an accelerated rate with a program that combines clinical studies with post-mortem evaluations than what could be accomplished using clinical data or studies with tissue alone.

“In the case of FMS and many other chronic pain syndromes, the

disorders uniquely afflict human beings,” says Lorton. “We seldom can take tissue samples from affected organs of living people in order to gain progress toward finding causes or cures. Evaluating central nervous system abnormalities is essential for developing treatments that really work to alleviate the debilitating pain and

other symptoms associated with FMS. Progress toward finding a cure critically depends on the ability of scientists to examine brain and spinal cord samples from the deceased patients of this disorder. In addition, comparisons must be made to healthy people of the same age to understand what is normal.”



You have given talks to the community, support groups, and physicians in the Phoenix area. How have they responded to this project?

They are excited about the program. There is a sense that it has the potential to provide answers to a lot of important questions about what causes FMS. Knowing the cause of an illness can in many cases lead to more effective rational treatments. The program will also establish a large pool of FMS patients who have been appropriately assessed and medically charted. These patients will have the opportunity to participate in clinical trials for promising new treatments as they become available.

Could you comment on how important it is to have an animal model for FMS and how your tissue bank may play a role in its development?

Animal models are crucial for testing the proposed causes of diseases. Many types of experiments that can truly test and clearly demonstrate cause and effect can only be completed in animals. Further, if potential drug treatments become available, normal animals and animals that have a disorder are tested for potential toxic side effects long before they are considered for use in human clinical

trials. Thus, it is extremely beneficial to have animal models that closely mimic the disease pathology to identify disease causes and to develop effective therapies. For FMS, no good animal models exist. One of the reasons for this is that FMS is so poorly understood and characterized that researchers don't know what criteria even would define a "good" animal model. It is my hope that once tissue is available through our tissue bank, the pathological features that define FMS will be determined. Then researchers can begin to develop an adequate animal model that will be required for testing the pathology of FMS and for preclinical trials of potential treatments.

Now that you have succeeded in procuring the large four-year NIH grant, what does this mean and what are your future goals?

The NIH grant goes a long way toward securing the future of the FMS clinical and tissue bank program. Not only does it provide financial support, but also provides the required increased credibility that will help recruit patients. My intention is to show the NIH that our organization can successfully develop this tissue bank into an incredible resource for FMS research, and that the program deserves to receive continued funding. It is also my intention to establish connections with other FMS researchers in hopes of developing collaborative efforts that can lead to grant submission to NIH with the SHRI clinical

center and tissue bank being a core facility on the application. It would be ideal to recruit FMS-focused scientists to a center that not only supplies the clinical data and tissue, but also plays an active role in the research. **END**

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Update: Are Cytokines Causing Your Sleep Problems?

Benjamin Natelson, M.D., is nearing the end of his four-year National Institutes of Health (NIH) project on the role of sleep disturbances, exercise, and cytokine production in chronic fatigue syndrome (CFS) and fibromyalgia syndrome (FMS) patients. At the beginning of 2006, AFSA funded a tag-on study to this NIH grant to evaluate two additional cytokines that are known to be elevated in FMS patients: interleukin-6 (IL-6) and interleukin-8 (IL-8). Cytokines are substances secreted by immune cells throughout the body, including the central nervous system where the glial cells reside. The box

below indicates how IL-6 and IL-8, in addition to the many other cytokines that Natelson is measuring, might be involved in the symptoms of pain, fatigue, and disturbed sleep.

All cytokine studies to date involving CFS or FMS patients have only looked at the level of these substances in the blood at one time during the day (i.e., just a single blood sample). While this approach is a good place to start, it does not reveal whether specific cytokines might be contributing to disturbed sleep. Natelson collected blood samples at bedtime, three times during the night, and then upon awakening. He did this for three nights (not

necessarily in a row) while the participants (patients and healthy controls) were put through various daytime tests, including an exercise protocol. Natelson's project design represents a giant leap forward to detect an altered rhythm of cytokine production that may be associated with disturbed sleep, daytime fatigue, and pain.

Natelson's entire NIH study—including the small portion funded by AFSA—provides an innovative way to look at sleep and the many complexities of CFS and FMS. He answers several questions about his project to keep contributors to AFSA informed about his progress.



How far along are you on this project?

Our sleep studies are done and we've just finished doing all our cytokine assays. A postdoctoral fellow just joined our research team, and his primary task will be to look at the various interactions or associations among the data. This part will take some time because of the tremendous amount of information and the complexity of the data we've collected.

Your hypothesis is that cytokines disrupt sleep and not the other way around. What do you suspect might be causing the alterations in cytokine production?

That's the 64 million dollar question. There are so many possible answers that have not yet been explored, and any response I provide

would just be speculation at this point. Perhaps a virus or the existence of a brain malfunction induces an alteration of the immune system, causing changes in cytokine production and producing chronic sleep disruption. These are just possibilities.

AFSA's role was to fund the analysis of two additional cytokines (IL-6 and IL-8). Could you explain the importance of this part of your project?

I particularly wanted to add IL-8 because there are data in the scientific literature showing that this cytokine is affected in FMS. IL-6 is the most common cytokine studied in a number of disorders involving physiological challenge conditions. For example, it is elevated in people with impaired sleep quality. Of interest is a recent paper reporting elevations in both IL-6 and IL-8 in patients with irritable bowel syndrome.

When do you expect the cytokine results to be published, and do you anticipate that it might generate several different papers?

With partial support from AFSA, we did look at cytokines in FMS patients over the 24-hour time period. Our goal in doing these studies was to look across time at a number of cytokines to determine if these differed in FMS compared to healthy controls. Those data have been extremely complex, but after a year's worth of work we are just about done with the analyses. We are in the process of writing up the study, and this will represent the first sleep and cytokine data to come out of my lab. As for the rest of our study data, I am confident that they will generate several more research papers. **END**

Actions of Cytokines

IL-6: causes fatigue, pain, and cognitive dysfunction

IL-8: causes pain

IL-1, TNF: sleep enhancers/pain promoters

IL-4, IL-10: sleep disruptors/pain relievers

AFSA Acknowledges Dr. Mary Lou “Whitey” White and Family



The American Fibromyalgia Syndrome Association (AFSA) wishes to recognize **Dr. Mary Lou “Whitey” White, Ed.D.**, of Cayucos and Morro Bay, CA, for her inspiring life achievements and generous contribution toward fibromyalgia research. Dr. White became a member of AFSA in 1994 (its first year of operation) and passed away on April 9, 2008 after a brief illness. She was 84.

Dr. White was a pioneering, vocal advocate for women’s collegiate athletics at Cal Poly San Luis Obispo where she leaves an enduring legacy.

Born in 1922, the middle of three sisters, she was raised in Corvallis, OR. After graduating from Oregon State College in 1946 she taught girl’s physical education in St. Helens, OR, before accepting a position instructing and coaching at Clark College in Vancouver, WA, where she stayed for 13 years.

In 1961 she moved to Cayucos, CA, and joined the professorial coaching staff at Cal Poly. Receiving a doctorate in education from the University of Oregon in 1973, her thesis involved the then newfangled technology of video taping for the purpose of instructional improvement in sport fencing.

After coaching women’s volleyball, basketball, softball, track, and men’s and women’s fencing, she retired in 1979 as the associate dean of the School of Human Development and Education. In 1990, Professor

Emeritus White was inducted into the Cal Poly Athletic Hall of Fame and recognized for being “The individual most responsible for the development of Women’s Athletics at Cal Poly.”

Apparently, few people knew that Dr. White lived with fibromyalgia much of her life. White’s nephew Dean Koehler said, “She once described her condition as ‘pouring a bucket of pain’ over her head, yet she was able to lead a very active life. Many people were unaware of what she suffered.”

Vibrant and active, to the best of her abilities till the end, she was still a regular at the gym. She was well known in motoring circles for her beautiful, one owner 1955 MG which won many awards. No fish was safe along the coast if she pulled out one of her several rods. A lifelong lover and savior of wayward cats and dogs, her home was never without a furry companion.

She remained active in the community, volunteering with the San Luis Obispo County Sheriff’s Patrol and playing weekly at the Morro Bay Vets Hall as a percussionist in the “Something Very Special Band.” She filled the same role with an RV club that played music and traveled as far east as Oklahoma. Those that knew her will remember her remarkable, humble, yet accomplished life and her upbeat spirit in the face of physical challenge.

More About AFSA

Updated Website

AFSA has updated its website to reflect its newly funded studies, provide more information, and offer easier access to make your tax-deductible donations today. Visit www.afsafund.org.

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You can receive 3 free informational brochures by sending your self-addressed, stamped envelope (business size #10 with 42 cents postage) to AFSA, PO Box 32698, Tucson, AZ 85751.

Update: Comparing Healthy vs. FMS Cell Function

As donor tissue from patients becomes available to the nation’s first fibromyalgia syndrome (FMS) tissue bank, it will be compared to age- and gender-matched healthy control tissue to better understand how glial cells function in this chronic illness. This is the first time glial cells are being thoroughly evaluated in humans. Thus far, most of the information on these cells have come from studying tissues taken during brain surgery and from animals.

Researchers believe data from this

project can be used to encourage some drug companies to bypass the animal testing phase and include people with FMS in their clinical trials. Pharmaceutical companies are already developing medications that target glial cells as a novel approach to controlling chronic pain. In fact, the first drug in this class (AV11) has shown promising results in the treatment of neuropathic pain.

Most chronic pain conditions, such as low back and neuropathic pain,

Continued on back page

AMERICAN FIBROMYALGIA SYNDROME ASSOCIATION

PO BOX 32698
TUCSON, AZ 85751-2698

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... *Continued from page 7*

have an animal model that closely represents or mimics the human condition (typically a rat or mouse is used). These models enable scientists to study tissues that cannot be safely extracted from humans (such as spinal cord or brain cells). In each of these animal models, the glial cells have been shown to play a vital role in the production and maintenance of pain. Unfortunately, an animal model for FMS is nowhere in sight, and because glial cells reside in the brain and spinal cord, taking samples from live donors with this condition is not an option.

Rather than wait for an animal model, **Linda Watkins, Ph.D.**, of the

University of Colorado in Boulder, and **Dianne Lorton, Ph.D.**, of Sun Health Research Institute in Arizona, are collaborating on an AFSA-funded study to examine the function of glial

Chemical markers that represent glial cell activation, pro-inflammatory cytokines, and other glial cell substances known to enhance pain will be measured.

cells in human tissues taken from deceased patients with FMS. The project involves taking brain and spinal cord tissues from healthy controls who have died and comparing them to individuals with FMS who have died—all from the tissue donor program (see pages 4-5).

So far, Watkins says several samples of human spinal cord tissue

from the lumbar (low back), thoracic (mid-back) and cervical (neck) region have been taken from healthy donors for evaluation. Chemical markers that represent glial cell activation, pro-inflammatory cytokines, and other glial cell substances known to enhance pain will be measured.

“The importance of the samples collected from the deceased healthy donors at this point,” says Watkins, “is to try to get a feeling for how variable the concentration of chemical markers are (a) across spinal cord regions and (b) across pain-free controls.”

This will provide essential information about which substances are typically produced by glial cells and the concentration range that would be considered normal. Commenting on the uniqueness of this initial part of the study, Watkins says, “I think we may well be able to publish these data by themselves.”

END

AFSA Donation Form

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