A Special Message From the President

Since the June 2008 Update, the American Fibromyalgia Syndrome Association (AFSA) has funded three important projects that could impact what we believe will lead to a better understanding of painful symptoms associated with fibromyalgia syndrome (FMS). The first two studies delve into the role of myofascial trigger points. Leaders in the field agree that this area of research has long been neglected, but understanding trigger points has the potential to alter diagnostic criteria and lead to better treatments. A third study will focus on patients’ premature loss of gray matter in several areas of the brain and how this relates to pain, cognitive abilities, fatigue, and physical function. Finally, a recently published study funded by AFSA could possibly unravel the genetic underpinnings of FMS. I’m truly excited as great strides are being made toward understanding this illness and credit the generous contributions from fibromyalgia patients and their families for making all these differences.

Your support is valued,

Kristin Thorson, president and founder
kthorson@afsafund.org

Myofascial Trigger Points: Are they the source of your pain?

Tender points define fibromyalgia syndrome (FMS). But what exactly are these areas that hurt when pressed? Although the American College of Rheumatology’s diagnostic criteria requires that FMS patients report pain in at least 11 of the 18 specified tender points, their locations in the muscles are somewhat vague. And when treatment trials are performed in FMS patients, the tender point count seldom budge even though other validated measures indicate that the patients do feel much better.

On the other hand, myofascial trigger points (MTPs) also cause pain, a sense of muscle weakness or tightness, and restricted range of motion. MTPs typically occur in the belly or mid-area of the affected muscle, but they can also produce severe pain where the muscle attaches to the bone (i.e., the muscle tendon junction). The reason for the pain and other symptoms is that the affected muscle has become shortened due to the sustained contraction of some of its fibers. Patients will sense that the muscle is tense or tight, and they may also feel a firm nodule, knot, or bulge where the fibers are contracted. Pressing on this knot causes extreme pain that radiates to other areas.

It used to be that tender points just hurt in their specific area when pressed and could be distinguished from MTPs that not only hurt when pressed but also referred pain. However, researchers in the field are scrutinizing this simplistic separation of the two types of “points” of FMS and related regional pain syndromes.

“In reality, virtually every patient with fibromyalgia has numerous tender areas that generally occur in the vicinity of the muscle tendon junction,” writes Robert...
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Bennett, M.D., of Oregon Health Sciences University in Portland. “From a purely clinical point of view, these other tender areas usually fulfill the criteria for a myofascial trigger point.”

Bennett goes on to state, “The artificial distinction between tender points and trigger points could be eliminated and the current locations could be redefined as myofascial trigger points on the basis of clinical and electromyographic findings.” Electromyography, or EMG, uses a tiny needle probe, that when inserted into a muscle, records the electrical activity in the specific area. When the EMG probe is inserted into the knot or “eye” of the MTP, it will pick up enhanced electrical activity that helps identify the precise location of MTPs. Clinically, if tender spots elicit a referred pain distant from the spot where they are pressed, they will be MTPs and not only tender points.

But what does it matter if some or all of the 18 diagnostic tender points or any of the other regions that cause pain in FMS patients are actually MTPs? It could mean a world of difference. If MTPs rather than tender points are the driving force of muscle pain and dysfunction in FMS, it could lead to a re-evaluation of the diagnostic criteria. More importantly, it would have a dramatic impact on the way FMS is treated.

The eye or center of an active MTP is known to contain high concentrations of nasty chemicals such as substance P, bradykinin, calcitonin gene-related peptide, tumor necrosis factor, and norepinephrine. Even latent MTPs, which do not cause any spontaneous pain when a person is at rest and not moving, contain slightly elevated concentrations of these awful substances.3 Latent MTPs occur in tight, ropy-like muscles that are “primed” to become active MTPs if the muscle is overworked. Almost everyone has latent MTPs. They inhibit muscle movement and can cause a person to substitute the use of other muscles to compensate for the presence of a latent MTP. However, most people will be unaware of latent MTPs because they do not produce any pain until pressure is applied directly to them. But unlike tender points, once the nodule of the MTP is deactivated, the chemicals inside dissipate along with the pain, muscle tightness, and weakness.

If multiple MTPs were found to be present in people with FMS, then health care providers would need to be trained on how to identify and treat them. Also, future FMS studies, especially treatment trials, would need to take into consideration the presence of MTPs when evaluating the results. “The lack of consideration of MTPs in FMS research has resulted in a whole body of literature highly contaminated by unrecognized MTP effects.” — David Simons, M.D.

The role of MTPs in FMS has long been neglected in research, even though studies have shown MTPs do lower the pressure pain thresholds in the affected muscle (e.g., making the muscle more sensitive to pain).5 In addition, the sympathetic nervous system hyperactivity (which is present in FMS patients) causes enhanced pain and an increased intensity of the referred pain from the MTPs.6 In recent years, head pain (which is present in over 70 percent of FMS patients) has been shown to be caused by MTPs in the nearby muscles.7 It’s about time that the presence of both active and latent MTP “pain generators” are documented in more than one FMS patient population, which is why AFSA recently funded two projects involving MTPs and people with fibromyalgia.

END

Role of Myofascial Trigger Points in FMS - Part 1

Principal Investigator: Hong-You Ge, M.D., Ph.D.
Aalborg University, Denmark
Award Amount (September 2008): $30,000

Hong-You Ge, M.D., Ph.D., and his colleagues at Aalborg University in Denmark, plan to evaluate 30 FMS patients and 30 healthy age-matched control subjects for the presence of active and latent MTPs. Initially the MTPs and their referred pain patterns will be identified by careful palpation of the muscles in the neck, shoulders, low back, and extremities. The pressure pain thresholds at all MTPs will also be measured. An anatomical map of the latent and active MTPs, as well as the areas of referred pain will be generated for each subject. Ge’s team also will confirm the presence of each MTP by measuring its electrical activity with electromyography (EMG).

In a second session one week later, subjects will be evaluated at the 18 diagnostic tender areas for FMS. Ge will look for the presence of latent or active MTPs at or near the 18 tender points. He will also determine if any of the tender points are located in an area of referred pain generated by an MTP. In addition, the MTPs will be confirmed by EMG. Since a tender point exam is done to identify areas of lowered pain threshold and MTPs have been documented to cause a lowering of pressure pain thresholds, it’s plausible that the 18 tender points might possibly be MTPs, but this has never been explored.

To fully assess the extent to which MTPs contribute to the generalized pain of FMS, Ge proposes to look at the impact of treating the MTPs in “Part 2” of his study. Upon successful completion of the evaluation phase, AFSA has already pre-approved the funding of Ge’s treatment phase.

Obviously, if effective treatment of the MTPs does lead to significant pain and symptom relief, this would provide important evidence of the role of MTPs in FMS and will prompt increased education on the treatment of MTPs.

Myofascial Trigger Points and Central Sensitization in People with FMS

Principal Investigator: César Fernández de las Peñas, P.T., Ph.D.
Universidad Rey Juan Carlos, Alcorcón, Madrid, Spain
Award Amount (September 2008): $25,000

César Fernández de las Peñas, P.T., Ph.D., and his colleagues in Spain, propose to evaluate the role of MTPs in generating FMS symptoms using a different approach from that of Ge’s team. Fifty FMS patients and 50 healthy age-matched controls will be carefully examined for the presence of active and latent MTPs. The referred pain patterns of each active MTP will also be charted. Muscles in the head, neck, shoulders, back, buttocks, legs, and forearms will be physically assessed for the presence of firm nodules and pain tenderness to identify the MTPs and their associated referral pain patterns. This will provide insight about each person’s peripheral pain generators in their muscles.

All subjects will undergo sensory pain testing to determine thresholds to pressure, cold, and heat pain. Muscles throughout the body will be used in these tests, which are designed to reflect impairments in the central nervous system’s ability to process pain. In addition, participants will fill out validated questionnaires to assess quality of life, physical functioning, and pain severity.

Statistical analyses will be performed to determine how the number of active and latent MTPs influence the sensory processing system, quality of life, functional ability, and overall pain severity.

The 18 diagnostic tender points will also be assessed to determine if any of the tender points are nearby active or latent MTPs, or in an area of referred pain that is produced by an active MTP. The goal of this part of the study is the same as that of Ge’s, because it will take more than one research team or medical journal report to draw attention to the possible overlap between the diagnostic tender points of FMS and the presence of MTPs.

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AFSA has created new donation categories to better reflect our appreciation for your role in improving the quality of life for patients with fibromyalgia syndrome. 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.

We would like to express our sincere gratitude to Sholom Dinsky, of Lord, Abbett & Co. LLC, who instead of having a grand retirement party, chose to have the money donated to AFSA. His sentiments and contribution will carry us further on our mission than a gold watch ever could. All the best.

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Several memory and mental processing tests show that fibromyalgia syndrome (FMS) patients have good reason to be complaining about their cognitive function, or what is called “fibro fog.” Putting your thought processes into perspective, one study showed that you are functioning at a level that is 20 years older than your actual age.1

Not surprisingly, last year M. Catherine Bushnell, Ph.D., of McGill University in Montreal, Canada, reported a premature loss of gray matter in several areas of the brain involved in pain and memory processing.2 The study was small and compared ten FMS patients to ten healthy pain-free control subjects (all women and age-matched to reduce variability).

As a person with FMS, you most likely want to know what the loss in gray matter means. Is it linked to your symptoms of pain? Does it correspond to your cognitive dysfunction? Or, perhaps loss of gray matter is tied to many symptoms. The initial study showed that gray matter loss increased with duration of symptoms, so a study involving a larger number of subjects is certainly warranted.

“It is well documented that cognitive functions, such as speed of information processing, working memory, and long-term memory, decline continuously across the adult life span beginning in the second decade of life,” says Bushnell. “So an obvious question arising from our preliminary findings is: ‘What is the relationship between accelerated brain aging in FMS patients and cognitive function?’ To answer this question, our study will examine the relationship between brain anatomy and different measures of cognitive functioning in 30 non-depressed, medication-free FMS patients. We will test the hypothesis that FMS patients have an increased age-related decline in cognitive functioning. We will determine the degree of dysfunction and the amount of gray matter loss relative to 30 age-matched control subjects.”

Other important goals of the study will be to determine if loss of gray matter correlates with:

1. pain threshold and other experimental measures of pain
2. level of physical functioning, and
3. fatigue scores

It’s essential that fatigue or alertness be excluded as the cause of the cognitive difficulties in FMS patients. Given that widespread pain is present in all patients, how is this symptom related to the loss of gray matter and cognitive dysfunction? Could pain be distracting or interfering with a person’s ability to process information? Also, how does the gray matter loss relate to a patient’s functional ability (or disease severity) and duration of symptoms?

Answers to these questions are needed to better characterize the significance of the gray matter loss, as well as the interrelationships between pain, fatigue, physical function, and cognitive abilities. If accelerated gray matter loss is found to correlate with diminished cognitive function, then efforts must be placed on early identification of FMS as well as testing therapies that can reduce or reverse the structural changes in the brain.

We will test the hypothesis that FMS patients have an increased age-related decline in cognitive functioning.

— M. Catherine Bushnell, Ph.D.
What could be the possible genetic underpinnings of fibromyalgia syndrome (FMS)? Manuel Martinez-Lavin, M.D., of the National Institute of Cardiology in Mexico City, believes that the sympathetic nervous system (the body’s “fight-or-flight” stress response) is the source of your many symptoms. In addition, he has found that FMS is related to genetic factors that alter the function of the sympathetic branch of the autonomic nervous system, which consists of the sympathetic and parasympathetic systems.

Martinez-Lavin’s published, AFSA-funded study evaluates variations in the genes that regulate the speed of the catechol-O-methyltransferase (COMT) enzyme. Dopamine, norepinephrine, and epinephrine are all broken down by the COMT enzyme so they can be eliminated by the body. Genetic abnormalities that lead to an excessively slow or “sluggish” COMT enzyme will cause accumulation of norepinephrine and epinephrine, the two transmitting substances used by the sympathetic nervous system. As a result, FMS could be due to sympathetic hyperactivity with a genetic component.

Referring to a study of 202 healthy women, Martinez-Lavin comments, “Women who slowly degraded the catecholamines are more sensitive to pain.” He adds that there are six known variations in the COMT gene that can lead to a slow-working enzyme and make a person more susceptible to developing a facial-jaw pain condition called temporomandibular dysfunction or TMD. Different ethnic factors may also influence the variations in the COMT gene, so Martinez-Lavin looked at two populations: women from Mexico and women from Spain. He found a higher incidence of three alterations in the COMT gene in the Spanish FMS patients that distinguished them from healthy controls (80 FMS patients versus 80 control subjects).

“In the Spanish people, there was a clear relationship between the number of defects in the COMT gene and FMS severity, according to the Fibromyalgia Impact Questionnaire (FIQ),” says Martinez-Lavin. Whereas, in the Mexican population, there was only a slight correlation with two sub-scales of the FIQ.” In other words, the Spanish patients with FMS had a sluggish COMT enzyme related to genetic glitches, while the Mexican FMS patients were not that different from the control subjects.

Both the Spanish and Mexican patients had very similar FIQ scores, meaning that they were equally impacted by their FMS. This leads Martinez-Lavin to go one step further to look at the adrenergic receptors that are activated by norepinephrine and epinephrine (the two chemical transmitters that build up when the COMT is slow). He hypothesizes that genetic alterations may cause the adrenergic receptors to become highly sensitized in the Spanish and Mexican FMS patients—similar to findings in women with TMD. He is currently testing this possibility.

As a patient, what does this mean for you? Regardless of whether you have genetic defects in your COMT enzyme or adrenergic receptors, the end result is a malfunctioning sympathetic nervous system that causes pain and many other symptoms. However, “Genetic make-up, partially influenced by ethnicity will play a role in therapy,” says Martinez-Lavin. “This is what pharmacogenetics is all about. As an example, a person with a COMT enzyme that slowly degrades norepinephrine may have important side effects from antidepressant medications that increase norepinephrine levels.” Overall, therapies that calm down the sympathetic system should lead to symptom improvements, such as adrenergic receptor blockers, many anti-epileptic medications, benzodiazepines, biofeedback/relaxation, yoga, tai chi, etc.

Regardless of whether you have genetic defects in your COMT or adrenergic receptors, the end result is a malfunctioning sympathetic nervous system that causes pain and many other symptoms.”

— Manuel Martinez-Lavin, M.D.

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The criteria for fibromyalgia syndrome (FMS) does not convey how severely impacted a person is with this condition. In fact, the diagnostic criteria are not completely objective and two patients with all 18 tender points may be differently affected by their FMS. Having a marker that reflects illness severity would be beneficial.

A chemical referred to as NAA appears to correlate with disease severity and can be detected by magnetic resonance spectroscopy (MRS) in the hippocampus region of the brain, according to Patrick Wood, M.D. (see box on page 6).1 “I suspect that eventually NAA may become a biomarker that allows us to track and demonstrate changes in the clinical profile” of a person with fibromyalgia, says Wood.

As another part of his AFSA-funded study, Wood also demonstrated significant gray matter loss in several brain regions.2 Given the somewhat scary nature of these findings, it is important to have a disease marker that shows objective changes in brain function. With more research, low NAA levels or some other substance is bound to show up as a marker.

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