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**Professional paper** 

# A NEW UNDERSTANDING OF FIBROMYALGIA SYNDROME

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Abstract: Fibromyalgia and chronic muscular pain (hereinafter: CMP) are conditions that include the symptom of muscle pain. Pain in fibromyalgia syndrome (hereinafter: FS) is widespread and furthermore, a bilateral muscle pain. For now, there is no therapy for FS, although there are many useful treatments. Patients with FS have more than just muscular pain, which is hypersensitive as many have interstitial edema, as a diffuse distension. Despite rigid diets and diuretics, patients gain weight and have periodic extremity swelling. Neuro muscular electrical stimulators help control edemas, as well as specific lymphatic drainage. Also, chronic fatigue is a common symptom of FS. Patients often suffer from many similar diseases. These medical problems can often be an integral part of FS and these conditions are deteriorating factors of the course of the disease. The key point in treating myofascial pain is to identify these comparative medical problems and treat them accordingly. Also, movement restriction is an integral part of CMP, as FS patients have limited movements within generalized muscular weaknesses. General hypersensitivity to pain has the features of allodynia (condition in which nonpainful stimuli cause pain), which is an attribute of FS. It is often encountered in athletes, as a problem of muscle exhaustion in overly ambitious and overloaded training

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sessions and competitions. This pain has no FS pain characteristics. Thus, it is necessary that doctors and physiotherapists adequately distinguish, understand and treat the state of FS according to the Copenhagen Conference criteria.

**Keywords:** fibromyalgia, chronic muscular pain, baseline, hyaluronic acid, 18 typical pain points.

#### **INTRODUCTION**

The definition of Fibromyalgic Syndrome (FS) was given by the Copenhagen Conference in 1998 in its Declaration. Previously, all painful conditions of the muscular system were defined differently by rheumatologists and physiatrists, either as fibromyositis or rheumatic muscular pain, and even interpreted as conditions of long-term psychological stress. In our country, many medical experts use the expression of fibromyalgic syndrome in all situations of chronic muscular pain (CMP), which are in fact two different clinical states in which muscle pain is only a common symptom.

In this regard, it is desirable to diagnose this clinical condition and to describe the findings as it is now defined by large research centers and clinical studies, as well as to differentiate the comparative difference between FS and CMP. As a matter of fact, a large proportion of the population suffers from both painful states of the muscular system. The basis of recognizing these two different muscular conditions, as well as their diagnosis, is quite confusing. Therefore, therapeutic approaches are insufficiently precise, which leads to the fact that many patients suffer from severe clinical conditions for many years, which in turn results in work disability and various forms of psychological and emotional suffering.

Bearing in mind that most physicians involved in pain treatment in the muscular system today lack sufficient precise and clear training in the pathophysiology of these two different clinical states, according to the principles and knowledge as they are described in modern scientific research centers for this problem, and thereby, there is a need to transfer and unify this knowledge within a modern approach. Because there is insufficient edification, there is no possibility of a clear diagnosis, and therefore there is no entirety of adequate therapy for these two different states.

FS is often encountered in athletes, as a problem of muscle exhaustion in overly ambitious and overloaded training sessions and competitions. This pain has no FS pain characteristics, as it is not widespread in different muscle groups and has no hypersensitivity features. Due to the absence of accompanying systemic diseases, muscular pain in athletes usually responds well to conventional therapeutic procedures, lacks a chronic character and does not result in work disability, which is a feature of FS.

#### The specific character of FS

The main symptom in the manifestation of FS is the emergence of widespread pain in multiple muscle groups in all four quadrants of the body, around the spinal column, neck, upper extremities, shoulders from the front and back, in the lumbar region, around the knees and elbows. In fact, there are 18 pain points in total (Fig. 1). In order to make a diagnosis, 11 painful places, both on the left and right sides, are required in all four quadrants (Davies, 2013). The pain is by its character hyperalgesic, i.e., it provokes various minor irritations, mechanical as well as light and sound. In addition to this painful condition, there are also general symptoms of fatigue during minimum activity. The next symptom is the appearance of unusual edema of soft tissues, often with subfebrile states or temperatures below normal. In addition, there is often unusual skin sensitivity to irritation.





With such painful muscular conditions, there is most often a resistance to conventional therapy, when considering the simultaneous existence of some systemic illnesses and conditions. These clinical-associated conditions are not pain triggers, although they significantly impede pain therapy and the course of 72  $\neg$ 

the disease. In addition to these clinical conditions, pain is the major perpetuum mobile or worsening factor in the course of the disease

# The findings of research centers on the possible causes of hypersensitivity to muscular pain conditions

Research centers, together with large laboratories, which deal with this issue, in their findings state different places of dysfunction as the reason for this type of pain. The dysfunction of the neuromuscular connective tissue or disorder of the center for qualification and sensation of pain sensations is cited as one of the possible places for hyperalgesic pain treatment (Davies, 2013). Other centers report disorders of the secretion of various neurotransmitters that transmit pain sensations at different levels (Starlanyl & Copeland, 2001). It is therefore important to treat different types of analgesics in order to treat these conditions, as well as to make combinations in order to interrupt the transmission of pain signals at different levels. According to other researchers, the disorder is within the system of functioning of the pituitary-hypothalamic-adrenal axis, when the secretion of Arginin and Vazopressin (Starlanyl & Copeland, 2001) is disturbed by long-standing stress. Thus the fight-or-flight response is triggered as often the system of the adrenergic nervous system is automatically activated by small stimuli, and therefore there is also the resistant muscular pain syndrome (Shanks, 1999).

There are no specific diagnostic tests for FS, only specific biochemical and molecular biological methods that show changes in different cellular systems in relation to healthy people.

These studies indicate a disruption in the formation of high-energy phosphates: creatinine phosphokinase (CPK) and adenosine triphosphate (ATP) (Simons, 1999). In the cerebro spinal fluid or lymph system of those with FS, there are increased values of homocysteine and the nerve growth factor (NGF). Other research centers cite mitochondrial dysfunction to metabolize nutrients in high-energy ATP phosphates, which is cited as the reason for the persistence of constant fatigue during minimum activity.

Also, the state of hypometabolism is a cause of pain and fatigue. These are cellular hypothyroidism states, where the conversion of T4 to T3 at cell level is deficient due to the lack of certain enzymes that carry this conversion (Doedhar, 1994; Shanks, 1999). Some centers state that this involves a disorder of insulin-like growth hormone (ILGH) that disrupts the control of metabolizing carbohydrates (Starlanyl & Copeland, 2001).

Research centers provide a more detailed report of myofascia, a thin fibrous membrane that covers all muscles and supports them in all three dimensions. Myofascia is attached to the dermis and when it works well in manipulation techniques, it moves smoothly. In FS, it is fibrotically altered and immobile. At deeper levels, the fascia is formed of denser material, which separates large spaces and groups of muscles. The third level of fascia is subserous membranes that cover the organs and keep the blood vessels and nerves in an adequate position. Contractions and constriction of myofacia at this level compress the various organs and nerves, creating conditions for the occurrence of pain in the body, far from the muscular pain points. In the intracellular spaces of the fascia, support is given to cells, helping them with nutrient flows and eliminating metabolic decay products. This view of myofascia compared to the previous limited understanding, indicates why FS is a much more complex condition than CMP, which is exclusively a pain in the muscular system (Simons, 1999).

An important understanding of research centers is essential regarding the presence of the basic substance (BS) in myofascia. In a state of good health, it is of a liquid gelatinous consistency. As such, it absorbs numerous harmful forces that are created in non-physiological conditions and traumas. In all these situations, it is a significant "shock absorber" of the binding tissue. BS maintains the distance between the fibrous fibers of the tissues and, as such, prevents the formation of micro adhesions in the tissue that give it a subtle elasticity. On the cellular level, BS facilitates the access of nutrients to cellular organelles, as well as an easier elimination of waste metabolic products from the cells.

Researchers state that BS is hardened and becomes like glue or cement in FS, as it collects and contracts and compresses the environment on all levels (Starlanyl & Copeland, 2001).

The failure to maintain a critical distance between fibrous fibers creates conditions for their bonding, with the subsequent creation of transverse fibers. This altered faction is difficult to break with the usual therapeutic procedures. The goal and the aim of the new therapeutic procedures is to break the newly collagen structures in the fascia, so that the BS becomes liquid and gelatinous again. With the return of the BS to the original liquid or gelatinous state, it is possible to efficiently re-eliminate the decay products of the metabolism that pass through the BS to reach the lymph smoothly. Waste products of metabolism are difficult to pass through the cemented structure of the BS, which, as such, accumulate in tissue muscle basins and are triggers for pain and swelling (Starlanyl & Copeland, 2001).

Because of the chronic pain present in muscles with fascia changed thus, patients resort to immovability, which imparts the formation of fibrous thickening and micro-adhesions in fascia.

This dysfunction of myofascia is often referred to as the problem of the irregular functions of the muscular system. This is the reason for the appearance of various pain, both by character and by localization, and the consequence of tissue irritation is an inelastic counterattack of fascia at different levels. Apart from the pain of this altered fascia in the maximum muscle movements, the full capacity of the muscular movement is prevented, which is one of the main reasons for muscular dysfunction (Simons, 1999).

Research centers which are experts in the structure of myofascia report that in many situations, due to painful syndrome and muscular dysfunction of the musculoskeletal agents, premature surgery on the large joints is undertaken, not so much due to degenerative changes but as a result of painful manifestations (Starlanyl & Copeland, 2001). Degenerative joint changes are evident in the population to the same extent, although there is no joint pain. Thus, the painful syndrome, due to the problems of myofascial conditions in the muscles, is taken as a criterion for surgery, and not treating the condition of altered muscle myofacia that would allow the muscles to have a good complete physiological function, and hence a good painless function of the joint. Reduced volume of joint mobility is a consequence of altered myofascia in important actuator muscles, and not a complete degree of degenerative changes on joint surfaces. Even with the full function of the muscular actuators in the joints, joint surfaces, cartilage and other conditions are kept in order to avoid a state of "splitting and friction," which is the pathophysiological moment of a premature degeneration of the joints. For a long time, the presence of immobility in large muscular systems due to myofascial problems triggered the action of synergistically less conditioned muscles, which, if untrained, are easily overloaded, and create conditions for creating new trigger pain points in these muscles. This is the reason and condition for spreading the painful syndrome to other regions, or the creation of latent pain states (Starlanyl & Copeland, 2001).

#### Features of FS pain

The pain in muscles in these conditions is insidious, as a persistent feeling of inconvenience and lasting indistinct pain, to the feeling of drastic pain as in a discus hernia. Such a muscle has a declining strength. The professional teams that lead many athletes, if unaware of such a seriously changed muscular structure, can set down drastic exercises to strengthen muscle strength and endurance. This in turn provokes the emergence of new latent areas of myofascial alteration, which favors the spread of painful areas, and nothing has been strengthened or removed in regards to pain or in muscle dysfunction.

#### Characteristics of pain in latently modified myofascia

A latent change of myofascia is activated in painful conditions in different life cycles: infections, viruses, inadequate body posture and postural stress, and the like. Patients are usually unaware that they have such an alteration in muscle fascia. These painful latent sites can usually be provoked by acupressure. If the pressure is applied at a force of 4 kg, it incites pain. This altered muscle structure produces greater tension in the muscles, increases their shortening, restricts muscle movements and consumes more energy for their function. Sometimes, in different circumstances, multiple areas with latent muscle states are simultaneously deactivated, which renders dramatic and widespread painful conditions and immobility. Only specialized educated teams from clinical and research centers that deal with the presented issues in detail have knowledge of such pain in latently altered muscular areas, as well as the

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occurrence of distant pain (Bennett, 2000).

#### What FS is not

FS is not a disease of the muscular muscular system. The initial symptoms are the result of a disorder of the secretion of many biochemical substances at different levels. Therefore, this is a syndrome, not a disease. By definition, diseases always have clearly defined pathophysiological changes that cause diseases. Unlike disease, syndromes do not have unique pathophysiological mechanisms, they have only a set of symptoms and signs that usually occur together. In the case of FS, the symptoms are generally present sensations of pain at specific points, left and right, a hypersensitive character of the pain, the presence of unusual fatigue, similar to the state of immunodeficiency. All these conditions, according to unwritten rules, coexist with various systemic conditions and diseases that precipitate symptoms of FS and make them resistant to therapy. As such, it creates confusion in the diagnosis, since myofascia is present anywhere in the body, which triggers a wide array of symptomatology (Bennett, 2000).

#### Clinical conditions commonly found in FS

FS most often coincides with the following clinical conditions, which aggravate the symptomatology of this condition and actual diagnostic confusion, as well as resistance to conventional therapeutic procedures (Starlanyl & Copeland, 2001; Clow, 1997):

- Osteoarthritis
- Hyperactivity in children or Attention Deficit Disorder
- Chronic fatigue syndrome in immunodeficiency
- Depressive states with a feeling of sadness and inability to feel joy
- HIV (which has painful symptoms of both FS and CMP)
- Hypermobile syndrome of the joints and spinal column
- Hypometabolism, either due to hypothyroidism with a defective secretion of thyroid hormones, or cellular hypothyroidism with normal or subnormal thyroid hormone values, but with a clear clinical picture of manifest hypothyroidism
- Interstitial cystitis (a state of chronic inflammation of unknown origin)
- Irritable bowel syndrome
- Multiple sensitivity to numerous substances with the sudden appearance of different types of allergic reactions to hypersensitivity
- Multiple sclerosis
- Hypotension of a neurogenic origin, with a weakness of the autonomic nervous system and frequently present syncope
- Post Traumatic Stress Syndrome (PTS)
- Hypoglycemia caused by excess carbohydrate and initial insulin resistance

- Seasonal affectivity syndrome (winter depression with a cyclic FS expression)\
- The presence of latent saprophytic fungal infections, gastrointestinal and urinary tract
- Reynaud's syndrome
- Lupus (myositis and systemic eritematodes)

According to research by Dr McMakin, FS is divided into several subgroups (Starlanyl & Copeland, 2001):

- A. FS with prolonged psycho-emotional trauma and stress, resulting in the exhaustion of the adrenal gland;
- B. FS due to prolonged exposure to chemicals and pesticides;
- C. FS with frequent tendencies of allergic reactions and a dysfunction of the hepatic metabolism;
- D. FS originating from viral infections and as a result of immunization from different viral states.

# Clinical differences between FS and CMP

Both conditions have muscle pain as a clinical symptom.

- CMP has no systemic illnesses or any evident diseases mentioned above;
- In patients with FS, in addition to muscle pain, there is an unusual fatigue similar to that of patients with immunodeficiency (Focuda, 1994);
- At the current level, there is no therapeutic procedure for curing FS, although there are procedures that can alter the symptomatology and the course of the disease;
- CMP pain is conditioned by the presence of trigger point painful nodules and tight strips in muscle fibers. Everything is conditioned by altered contractions of non-elicited actin and myosin fibers, which remain locked and as such create nodules and relaxation problems (Figure 2).

**Figure 2**. A schematic representation of painful nodules and tight strips in muscle fibers (Davies, 2013).



- The treatment of this kind of pain in CMP is carried out by local therapeutic procedures.
- In addition to local therapeutic procedures, FS should be treated in parallel with systematic disease, because the pathophysiology of these conditions aggravates and precipitates the symptomology of FS, and makes it resistant to conventional therapies.
- The FS examination does not include painful nodules in the muscles, nor tight strips that are palpated like strips (a CMP characteristic).
- Restriction of muscle movement is not a feature of FS, although sometimes due to the general weakness of this syndrome, there is inadequate muscular function and movement in all joints.
- The next important feature in the manifestation of FS in the muscles is the emergence of an intracellular and extracellular edema. Retention of water in muscle tissue occurs due to an excess of hyaluronic acid, which is otherwise an integral part of FS (Liu, 1998).

#### Clinical characteristics of retention of water in FS

With all the FS symptomology, patients often have testicular edema and weight gain, despite regular diets and taking diuretics. There are no common measures to give relief to the patients in these conditions of rigorous edema of the lower extremities. Diuretic administration even aggravates their condition, because the ionic changes deteriorate the redistribution of fluid, and hence the symptomatology of pain and muscle weakness (Doedhar, 1994).

Israeli researchers have found that in FS there is a mass of gelatinous changes that contain excess hyaluronic acid. These "microblots" of gelatinous masses in myofascia are the reason of a resistance to the usual therapeutic procedures (Doedhar, 1994). Furthermore, Simon and David have found that the main content of these microblots of gelatinous masses are large molecules of glucosamine glycans. In addition to the hyaluronic acid that is present in FS, the glucosamine glycan molecules potentiate the unusual binding of extracellular and intracellular water, which is the reason for resistant edema (Simons, 1999).

The therapy of these conditions is by way of neuromuscular stimulants, using electronic galvanic stimulating gloves, with special forms of massage and requirements for lymphatic drainage (Valand manual lymphatic drainage) (Simons, 1999).

#### The problem of myofascia and the dysfunction of mitochondria

Dr. Paul St. Amond finds that mitochondrial dysfunction and excess ion phosphate and calcium in mitochondria cause edemas in mitochondria. In studies, muscle biopsies have shown the presence of excess ion phosphate in mitochondria. Ion phosphate behaves like acidic ion, while ion calcium acts as the base and performs the buffering of excess acidic ions. Quamescina, an old drug previously used for the treatment of rheumatic pains is starting to be used in therapeutic procedures. It has a great impact on increased urine phosphate excretion - by 60% and 30% oxalate after taking oral tablets. This treatment results in a FS symptom reversal. In addition, there is the elimination of ion phosphate and the elimination of the excess of captured toxins. This situation causes symptomology similar to colds: an excess of stagnated mucus from the tracheobronchial tree is emitted, there is sweating, headaches, and increased urination with irritation (Wrutniak Cabello, 2001).

# CONCLUSION

In regards to Serbia, it is clear that there is a high representation of the aforementioned symptomatology, which, as a rule, appears along with the above criteria. In fact, patients are not diagnosed in such a complex way, and they do not receive the qualification and diagnosis of FS as defined in the Copenhagen Declaration today. Patients with a wide range of symptomatology go from one team to another, their conditions become chronic and as such they fail to receive adequate therapeutic treatment. Over time, they lose their full working capacity due to widespread pain and other symptoms and undergo psychological and emotional suffering. Given that these patients possess different clinical diseases and conditions that are the cause of resistance to the usual therapeutic procedures of muscular pain, the necessary importance of FS training in all the above criteria is emphasized. The question is whether inadequate training regarding FS by modern criteria is ethically justified as patients fail to receive full therapeutic treatment, which is otherwise multidimensional and complex.

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