

Dental, Oral and Maxillofacial Surgery**Fascial involvement in myofascial pain syndrome**Savina Nencheva-Svechtarova¹, Vasil Svechtarov²**Ролята на фасцията в миофасциалния болков синдром**Савина Ненчева-Свещарова¹, Васил Свещаров²**Summary**

Myofascial pain syndrome includes a number of symptoms of the sensory, motor and autonomic nervous systems, which are caused by stimulation of myofascial trigger points. The involvement of the fascia in this syndrome is often neglected. The clinical features of myofascial trigger points, referred pain, fascial components and their contribution to myofascial pain have been described in detail in a number of recent studies, highlighting the clinical significance of this disorder.

This systematic review, based on 50 scientific publications on the subject, complements our knowledge of the etiopathogenesis of myofascial pain, the role of the fascia in the spread of pain, and the disease-specific referred pain. Some modern methods for treating large fascial areas to increase the effectiveness of local methods for the treatment of myofascial pain are also discussed.

Key words: myofascial pain, fascia, red SLD phototherapy

Резюме

Миофасциалният болков синдром включва редица симптоми от сензорната, двигателната и автономната нервни системи, които са причинени от стимулиране на миофасциалните пускови (тригерни) точки. Участието на фасцията в този синдром често се пренебрегва. Клиничните характеристики на миофасциалните пускови точки, на отразената болка, на фасциалните компоненти и техният принос към болката, са подробно описани в редица съвременни проучвания, което подчертава клиничната значимост на това заболяване.

Този систематичен обзор, базиран на 50 научни публикации, допълва познанията ни относно етиопатогенезата на миофасциалната болка, ролята на фасцията в разпространението ѝ и специфичната за това заболяване отразена болка. Дискутирани са някои съвременни методи за третиране на широки фасциални площи – за повишаване на ефективността от локалните методики за лечение на миофасциалната болка.

Ключови думи: миофасциална болка, фасция, червена SLD фототерапия

Introduction

The maxillofacial and in particular the temporomandibular area is a precisely coordinated complex system, a set of interconnected elements with diverse subsystems. These are connected by a richly innervated fascial structure, with functional stability and collagen matrix. [1]

The system itself is subject to the principle of biotensional integrity. According to Donald Ingber [2], the term expresses a modern concept of the pathophysiological mechanisms of myofascial pathology. According to this theory, cell structures follow the principles of tension integrity, changing depending on their position, function, purpose and

¹Doctor of Dental Medicine, PhD, Center for Integrated Dental Medicine, Faculty of Dental Medicine, Medical University – Sofia

²Associate Professor, PhD, Department of Dental, Oral and Maxillofacial Surgery, Faculty of Dental Medicine, Medical University – Sofia

adaptation to environmental factors. The organs and tissues of the human body and in particular of the maxillofacial and temporomandibular areas are integrated by specific fascia, which should be considered not only as supporting but also as functional units, connecting the individual facial elements and supporting them in a structural and functional unity. In this regard, fascia should be the focus of clinicians' attention from a practical therapeutic point of view [9].

Etiology of myofascial trigger points

Myofascial pain syndrome is a chronic pain condition characterized by the presence of myofascial trigger point, a hyper irritable painful spot involving a limited number of muscle fibers. The literature suggest that myofascial trigger points should be considered as peripheral pain generators. The clinical features of myofascial trigger points and their contribution to the patient's pain and disability have been detailed in several recent studies, which support the clinical relevance of the condition. Recent studies reported that manual palpation to identify trigger points has good reliability, although some limitations are intrinsic to the diagnostic criteria. During the last decade, a plethora of treatments have been proposed and positive effects on pain and function demonstrated [9, 19, 27]. Patients with myofascial trigger points will benefit from a multimodal treatment plan including SLD red light photo sessions, occlusal and manual therapy techniques.

Myofascial pain syndrome is described as sensory, motor, and autonomic nervous system symptoms caused by stimulation of myofascial trigger points. Muscle overload is hypothesized to be the result of sustained or repetitive low-level muscle contractions, eccentric muscle contractions, and maximal or sub-maximal concentric muscle contractions. Trigger points may develop during occupational, recreational, or sports activities when muscle use exceeds muscle capacity and normal recovery is disturbed.

A latent myofascial trigger point is defined as a focus of hyper irritable in a muscle taut

band that is clinically associated with local twitch response and tenderness and/or referred pain upon manual examination. Treating latent trigger points in patients with musculoskeletal pain may not only decrease pain sensitivity and improve motor functions, but also prevent latent trigger points from transforming into active trigger points, and hence, prevent the development of myofascial pain syndrome. There are fundamental differences between the effects produced by the two basic types of trigger points (active and latent). Active trigger points usually produce referred pain and tenderness. In contrast, latent trigger points are foci of hyper irritability in a taut band of muscle, which are clinically associated with a local twitch response, tenderness and/or referred pain upon manual examination. Latent trigger points may be found in many pain-free skeletal muscles and may be "activated" and converted to Active trigger points by continuous detrimental stimuli. They can be inactivated by different treatment strategies; however, they never fully disappear but rather convert to the latent form [3, 4, 5, 6, 7, 8, 13, 14, 15, 16, 18].

Since the 1960s, the clinical literature has included descriptions of myofascial pain syndrome, which is currently diagnosed based on signs and symptoms, including the palpation of tender, indurated nodules termed myofascial "trigger points" [34, 35]. Myofascial pain syndrome is distinguished from fibromyalgia by its more focal and episodic nature and includes both "active" and "latent" phases [36]. The active phase is characterized by spontaneous pain that limits the range of motion, with localized, tender indurated foci within the myofascial tissues. Palpation of these nodules reproduces the patient's pattern of local or radiating pain. During the latent phase, palpable focal nodules are present and may be tender but without spontaneous pain. The latent phase also includes myofascial dysfunction, including soft tissue stiffness and reduced range of motion (not associated with pain) in the affected area. The increased soft tissue stiffness and

movement restriction could reflect focal areas of persistent muscle contraction or connective tissue adhesions that locally restrict shear plane movement or shear strain [1].

The mechanics of TMJ movements require stability of the joints and related structures, as well as a large adaptive capacity. This involves a rapid response coordinated by the central nervous system during masticatory function to achieve occlusal stability. The tension reactivity of TMJ is carried out by the ligaments, the muscles, the connective tissue matrix, the blood vessels and the articulatory disc.

Fascial components and their involvement in myofascial pain syndrome

The participation of fascia in this syndrome has often been neglected. Several manual and physical approaches have been proposed to improve myofascial function after traumatic injuries, but the processes that induce pathological modifications of myofascial tissue after trauma remain unclear.

The fascia as a system is a fundamental structure, providing morphological and functional integrity between the components of the maxillofacial complex. It supports its components: bones, muscle fibers, nerves, lipids, blood vessels and various groups of receptors. The fascia also coordinates biochemical signals, eliminates or mechanically protects tissues from pathogens, plays a role in tissue healing and scar formation, maintains the extracellular matrix, allows full cellular communication. It absorbs and transmits local stimuli to all parts of the body, its sensory network registers thermal and chemical stimuli, pressure, vibration and movement, affects the sensation of pain and plays the role of corrective of the peripheral and central nervous system.

In modern medicine, the fascia is defined as a continuous viscoelastic tissue that forms a functional three-dimensional collagen matrix covering all body structures. Its main characteristics are as follows:

– it is a thin, multi-layered silvery-white reticulated membrane that wraps around muscles and organs without adhering to the underlying tissue. This allows the underlying tissues to have minimal mobility towards it, within certain limits.

– it is made of collagen and elastin fibers, and depending on the collagen / elastin ratio it can be looser or denser. Shows individual differences depending on gender, age, or certain physiological conditions (pregnancy) and more.

– it is a fabric with little elasticity, but with pronounced plasticity - it can lengthen and change its shape as a result of slipping of its own layers. This gives smoothness to body movements.

– it is richly supplied with blood and moistened with a jelly-like, mucous fluid, which allows movement between the underlying tissue / fascia, as well as between the layers of the fascia itself.

– it cannot be visualized by computed tomography or magnetic resonance imaging. It can be examined by a special method with ultrasound for the presence of seals, thickening, reduced mobility, adhesions inside it and for other changes [30, 31, 32, 33, 39].

The fascia is the main communication system in the body as it is saturated with receptors and free nerve endings. It takes an active part in the body's immune system. The fascial network also reflects the psycho-emotional state of the individual – posture, function and facial expression. Connective tissue is a sensitive organ – more than 80% of free nerve endings are concentrated in the fascia. This network structure has not only a strengthening and mobile function, but is also saturated with pain receptors. Isolated fragments of fascial fibers were treated with hormones that are released in the body when it is under stress. In most cases, the tissue reacts by shrinking very slowly. This means that a stressed fascia can reduce its volume, regardless of the condition of the underlying muscles.

The fascia is composed of layers that smoothly pass into each other without a definite border. In most cases, the morphological component of fascial pain cannot be visualized because it be-

comes the sum of fine cleavages in the fascia at a microscopic level, which create micro defects, e.g. in case of injuries or chronic overexertion. Connective tissue can be damaged not only by overload (for example, in intense sports), but also by lack of physical activity. After surgery, denser areas are usually created that narrow the space around the nerve fibers that pass through the fascia.

The main cellular building blocks of the fascia are fibroblasts and fibrocytes, which can move relatively freely and actively communicate with surrounding tissues, hormones and enzymes. They can react according to the situation, restoring balance in the system. When lesions occur, they concentrate on the damaged area, stabilizing the tissue, “gluing” the micro ruptures. By unclear mechanism, in some cases fibroblasts produce more collagen and “adhesive” than necessary and thus turn a loose fascia into a dense, fused, thickened structure (adhesions). This is the typical picture in post-traumatic and postoperative conditions. In many conditions – prolonged immobilization, overload, tissue injuries, low local temperature, increased acidity, dehydration, stress, insomnia and depression, the fascia reacts with narrowing, thickening, loss of viscosity and adhesion to the underlying tissues. Modern research shows that fascia can be repaired, which is important for the treatment of myofascial type of damage [18,19,20,22].

Fasciae are composed of irregularly arranged but tightly woven connective tissue that can bear high tensile loads [37, 40, 41]. All connective tissue consists of both cellular and extracellular components, and it is the varying molecular and architectural characteristics of these extracellular constituents that determine their mechanical properties [12, 24, 26, 29]. Muscular fasciae and aponeuroses are known to be richly innervated with small-diameter afferent fibers that can transmit nociceptive signals, especially in the presence of inflammation. [17]. At a cellular level, several

types of ion channels are activated by mechanical stimuli [22].

Fascia mobility, is critical for musculoskeletal function and needs to be better integrated into bio mechanical models of musculoskeletal pain. Biological tissues are viscoelastic, which means that their mechanical behavior can be described as that of both solid (elastic) and liquid (viscous) [25]. A fundamental concept of the elastic component is the relationship among stress (applied force), strain (deformation resulting from an applied force), and stiffness [28].

Viscosity is also important for determining the effect of normal and shear forces and is related to the “liquid-like,” rate-dependent component of strain; that is, connective tissue will deform less when a force is applied quickly than when applied slowly, which allows the tissue structure to reorganize in a liquid-like manner as the force is applied [21]. The application of mild mechanical effects - massage, affects fibroblasts and regulates collagen production. In the presence of a lesion or overexertion in the fascia, fibroblasts react by compensatory accumulation of collagen to thicken and stabilize the affected area. In recent years, the application of red light phototherapy by means of various cluster devices, through which large areas are treated, is particularly promising and therefore they are most suitable for myofascial injuries and related pain conditions.

According to Langevin [10, 11, 12] 20 minutes of over voltage is enough to activate the tissue remodeling mechanism. If the process is prolonged (days to months), long-term tissue contractures are formed due to the accumulation of excessive amounts of collagen and loss of elasticity. Thus, muscle dysfunction and stiffness depends not only on the electromyographic activity and mechanical properties of muscle sacromers, but is greatly influenced by the derived fascial elements (membranes, aponeuroses, tendons, ligaments).

Many different nerve endings and proprioceptors are “intertwined” in the fascia, which allows it to respond adequately to different types,

nature and intensity of stimuli. Proprioception is mediated by mechanosensory neurons located in muscles, tendons and joints. [42, 43, 44] There are many types of proprioceptors that are activated during different behaviors and encode different types of information: speed and movement, organ load and more. This not only leads to a mechanical reaction (change in its tension), but can also cause changes in breathing, heart rate, blood pressure and more vegetative reactions. Other important nerve endings with which the fascia is richly “populated” are the pain nociceptors. In 2002 Langevin [12] published a study on the coincidence of the acupuncture points from the energy meridians of Chinese medicine with the points where the fascia branches or connects. The results of this study (80% coincidence of the fascial points with the acupuncture points) brings the understanding of the body’s energy system in Chinese medicine closer to the point of view of Western medicine, which recognizes the fascial system in this phenomenon [23, 45].

Contemporary treatment modalities for myofascial pain relief

Phototherapy with super luminescent diodes is a physical method in which photons are generated mainly in the visible red spectrum (approx. 630 nm) and to a lesser extent in the infrared. They penetrate to a depth of 2 cm into the tissues, which makes them particularly suitable for wide irradiation of the facial and cervical fascia and lead to efficient absorption of light energy at a cellular level. As a result, a number of photochemical reactions are initiated, expressed in increase of tissue metabolism, pain reduction, stimulation of the vascular, immune and lymphatic systems with a cumulative effect in the cells. A particularly important effect of this type of phototherapy is the stimulation of collagen synthesis, which supports the reparative processes in the fascia [46, 47, 48].

This process can be potentiated by introducing external light energy. The mechanisms of ac-

tion of near-infrared radiation are related to the activation of electron transfer along the respiratory chain of mitochondria. At the same time, the activation of tissue respiration, together with an increase in the intensity of metabolism all lead to an extremely high accumulation of reactive oxygen and radical intermediates. Photosensitive chromophores and other elements in the cell absorb light energy and initiate a series of important photochemical reactions, such as increased production of nitric oxide, singlet oxygen and adenosine triphosphate (ATP), as well as changes in cell membrane permeability. Electron transfer is of paramount importance for the respiratory chain in mitochondria, where the major chromophores involved in photochemical reactions are located. Stimulation of the Krebs cycle initiates the production of ATP, which provides additional energy and speeds up metabolism. Light energy and the biochemical reactions associated with its assimilation lead to the normalization of cellular functions [49, 50].

Conclusion

This systematic review presents a more in-depth interpretation of the etiopathogenesis of myofascial pain syndrome and current scientific understanding of the role of the fascia in the spread of disease-specific referred pain.

This review completes our knowledge of the role of the fascial components in clinical features of myofascial pain syndrome and their involvement in pain distribution.

Red SLD phototherapy is also described as an effective modern method for the treatment of large fascial areas, which increases the effectiveness of local treatment of myofascial pain syndrome.

References:

1. Stecco A, Gesi M, Stecco C, Stern R. Fascial components of the myofascial pain syndrome. *Curr Pain Headache Rep*, 2013, Aug; 17(8):352.
2. Brownell L, The “architecture of life” described by computer modeling. New multi-scale modeling approach re-

- affirms that tensegrity is a fundamental determinant of living systems at all size scales. Wyss Institute at Harvard University, 2018, <https://wyss.harvard.edu/>
3. Carel Bron 1, Jan D Dommerholt Etiology of myofascial trigger points *Curr Pain Headache Rep.* 2012 Oct;16(5):439-44.
 4. Ge HY, Arendt-Nielsen L. Latent myofascial trigger points. *Curr Pain Headache Rep.* 2011 Oct;15 (5):386-92.
 5. Celik D, Mutlu EK. Clinical implication of latent myofascial trigger point. *Curr Pain Headache Rep.* 2013 Aug;17(8):353
 6. Bron C, Dommerholt JD. Etiology of myofascial trigger points. *Curr Pain Headache Rep.* 2012 Oct;16 (5):439-44.
 7. Dommerholt J, Chou LW, Finnegan M, Hooks T. A critical overview of the current myofascial pain literature - February 2019. *J Bodyw Mov Ther.* 2019 Apr;23 (2):295-305.
 8. Barbero M, Schneebeli A, Koetsier E, Maino P. Myofascial pain syndrome and trigger points: evaluation and treatment in patients with musculoskeletal pain. *Curr Opin Support Palliat Care.* 2019 Sep;13(3):270-276.
 9. Stecco C., Porzionato A., Lancerotto L., Stecco A., Macchi V., Day J.A., De Caro R. Histological study of the deep fasciae of the limbs. *J. Bodyw. Mov. Ther.* 2008; 12:225–230.
 10. Langevin H.M., Huijing P.A. Communicating about fascia: History, pitfalls, and recommendations. *Int. J. Ther. Massage Bodyw.* 2009;2:3-8.
 11. Langevin H.M., Fox J.R., Koptiuch C., Badger G.J., Greenan-Naumann A.C., Bouffard N.A., Konofagou E.E., Lee W.-N., Triano J.J., Henry S.M. Reduced thoracolumbar fascia shear strain in human chronic low back pain. *BMC Musculoskelet. Disord.* 2011;12:203.
 12. Langevin H.M., Churchill D.L., Wu J., Badger G.J., Yandow J.A., Fox J.R., Krag M.H. Evidence of connective tissue involvement in acupuncture. *FASEB J.* 2002;16:872-874.
 13. Skootsky S.A., Jaeger B., Oye R.K. Prevalence of myofascial pain in general internal medicine practice. *West. J. Med.* 1989;151:157-160.
 14. Friction J. Myofascial pain: Mechanisms to management. *Oral. Maxillofac. Surg. Clin. N. Am.* 2016;28:289-311.
 15. National Institutes of Health. NIH HEAL Initiative Workshop on Myofascial Pain. 2020; <https://www.nccih.nih.gov/news/events/nih-heal-initiative-workshop-on-myofascial-pain>.
 16. National Institutes of Health. The Science of Interoception and Its Roles in Nervous System Disorders. 2019; <https://www.nccih.nih.gov/news/events/the-science-of-interoception-and-its-roles-in-nervous-system-disorders>.
 17. Hoheisel U., Rosner J., Mense S. Innervation changes induced by inflammation of the rat thoracolumbar fascia. *Neuroscience.* 2015;300:351-359.
 18. Mense S. Innervation of the thoracolumbar fascia. *Eur. J. Transl. Myol.* 2019;29:8297.
 19. Stecco C., Pirri C., Fede C., Fan C., Giordani F., Stecco L., Foti C., De Caro R. Dermatome and fasciatome. *Clin. Anat.* 2019;32:896-902.
 20. Chen W.G., Schloesser D., Arensdorf A.M., Simmons J.M., Cui C., Valentino R., Gnadt J.W., Nielsen L., St Hillaire-Clarke C., Spruance V., et al. The emerging science of interoception: Sensing, integrating, interpreting, and regulating signals within the self. *Trends Neurosci.* 2021;44:3-16.
 21. Proske U., Gandevia S.C. The proprioceptive senses: Their roles in signaling body shape, body position and movement, and muscle force. *Physiol. Rev.* 2012;92:1651-1697.
 22. Chesler A.T., Szczot M., Bharucha-Goebel D., Cěko M., Donkervoort S., Laubacher C., Hayes L.H., Alter K., Zampieri C., Stanley C., et al. The role of PIEZO2 in human mechanosensation. *N. Engl. J. Med.* 2016;375:1355-1364.
 23. Wang X., Chan S.-T., Fang J., Nixon E.E., Liu J., Kwong K.K., Rosen B.R., Hui K.K.S. Neural encoding of acupuncture needling sensations: Evidence from a fMRI study. *Evid. Based Complement. Altern. Med.* 2013;2013:483105.
 24. Klingberg F., Hinz B., White E.S. The myofibroblast matrix: Implications for tissue repair and fibrosis. *J. Pathol.* 2013;229:298-309.
 25. Elosegui-Artola A. The extracellular matrix viscoelasticity as a regulator of cell and tissue dynamics. *Curr. Opin. Cell Biol.* 2021;72:10-18.
 26. Chaudhuri O., Cooper-White J., Janmey P.A., Mooney D.J., Shenoy V.B. Effects of extracellular matrix viscoelasticity on cellular behaviour. *Nature.* 2020;584:535–546.
 27. Stecco C., Pirri C., Fede C., Yuceosy C.A., De Caro R., Stecco A. Fascial or muscle Stretching? A narrative review. *Appl. Sci.* 2021;11:307.
 28. Fung Y.-C. *Biomechanics: Mechanical Properties of Living Tissues.* 2nd ed. Springer; New York, NY, USA: 1993.
 29. Pavan P.G., Stecco A., Stern R., Stecco C. Painful connections: Densification versus fibrosis of fascia. *Curr. Pain Headache Rep.* 2014;18:441.
 30. Wu P.-H., Aroush D.R.-B., Asnacios A., Chen W.-C., Dokukin M.E., Doss B.L., Durand-Smet P., Ekpenyong A., Guck J., Guz N.V., et al. A comparison of methods to assess cell mechanical properties. *Nat. Methods.* 2018;15:491-498.

31. Ophir J., Alam S.K., Garra B.S., Kallel F., Konofagou E.E., Krouskop T., Merritt C.R.B., Righetti R., Souchon R., Srinivasan S., et al. Elastography: Imaging the elastic properties of soft tissues with ultrasound. *J. Med. Ultrason.* 2002;29:155.
32. Bishop J.H., Fox J.R., Maple R., Loretan C., Badger G.J., Henry S.M., Vizzard M.A., Langevin H.M. Ultrasound evaluation of the combined effects of thoracolumbar fascia injury and movement restriction in a porcine model. *PLoS ONE.* 2016;11:e0147393.
33. Yin Z., Lu X., Cohen S.C., Sui Y., Manduca A., Van Gompel J.J., Ehman R.L., Huston J., 3rd A new method for quantification and 3D visualization of brain tumor adhesion using slip interface imaging in patients with meningiomas. *Eur. Radiol.* 2021;2021
34. Simons D.G. New views of myofascial trigger points: Etiology and diagnosis. *Arch. Phys. Med. Rehabil.* 2008;89:157-159.
35. Shah J.P., Thaker N., Heimur J., Aredo J.V., Sikdar S., Gerber L. Myofascial trigger points then and now: A historical and scientific perspective. *PM R.* 2015;7:746-761.
36. Weller J.L., Comeau D., Otis J.A.D. Myofascial pain. *Semin. Neurol.* 2018;38:640-643.
37. Barbe M.F., Gallagher S., Massicotte V.S., Tytell M., Popoff S.N., Barr-Gillespie A.E. The interaction of force and repetition on musculoskeletal and neural tissue responses and sensorimotor behavior in a rat model of work-related musculoskeletal disorders. *BMC Musculoskelet. Disord.* 2013;14:303.
38. Langevin H.M. Reconnecting the brain with the rest of the body in musculoskeletal pain research. *J. Pain.* 2021;22:1-8.
39. Mazza D.F., Boutin R.D., Chaudhari A.J. Assessment of myofascial trigger points via imaging: A systematic review. *Am. J. Phys. Med. Rehabil.* 2021
40. Stanley S., Balic Z., Hubmacher D. Acromelic dysplasias: How rare musculoskeletal disorders reveal biological functions of extracellular matrix proteins. *Ann. N. Y. Acad. Sci.* 2021;1490:57-76.
41. Xiong X., Berrueta L., Urso K., Olenich S., Muskaj I., Badger G.J., Aliprantis A., Lafyatis R., Langevin H.M. Stretching reduces skin thickness and improves subcutaneous tissue mobility in a murine model of systemic sclerosis. *Front. Immunol.* 2017;8:124.
42. Yucesoy C.A., Koopman B.H.F.J.M., Baan G.C., Grootenboer H.J., Huijting P.A. Extramuscular myofascial force transmission: Experiments and finite element modeling. *Arch. Physiol. Biochem.* 2003;111:377-388.
43. Wilke J., Schleip R., Yucesoy C.A., Banzer W. Not merely a protective packing organ? A review of fascia and its force transmission capacity. *J. Appl. Physiol.* 2018;124:234-244.
44. Smith T.O., Jerman E., Easton V., Bacon H., Armon K., Poland F., Macgregor A.J. Do people with benign joint hypermobility syndrome (BJHS) have reduced joint proprioception? A systematic review and meta-analysis. *Rheumatol. Int.* 2013;33:2709-2716.
45. Cramer H., Quinker D., Schumann D., Wardle J., Dobos G., Lauche R. Adverse effects of yoga: A national cross-sectional survey. *BMC Complement. Altern. Med.* 2019;19:190.
46. Sveshtarov V, Nencheva-Sveshtarova S, Grozdanova R, Prodanova K. Superluminous devices versus low-level laser for temporomandibular disorders. *Acta Medica Bulgarica*, Vol. XLV, 2018,1, 11-15
47. Maia M, Bonjardin L, Quintans S, et al. Effect of low-level laser therapy on pain levels in patients with temporomandibular disorders: a systematic review. *J Appl Oral Sci*, 20, 2012,6, 594-602.
48. Carrasco T, LD Gnerisol, D Gnerisol, Mazzeto M. Evaluation of low intensity laser therapy in myofascial pain syndrome - *Cranio*, 27, 2009,4, 243-247.
49. Karu T. Primary and secondary mechanisms of action of visible to near-IR radiation on cells. *Journal of Photochemistry and Photobiology B: Biology.* 49, 1999,1, 1-17.
50. Baxter D. Low intensity laser therapy. In: Kitchen S, Bazin S. *Electrotherapy: Evidence-Based Practice.* London: WB Saunders; 2003.

Address for correspondence:

Dr. Savina Nencheva-Svechtarova
Center for Integrated Dental Medicine
Faculty of Dental Medicine
Str. „G.Sofiiski“ 1
1431, Sofia
e-mail: s.nencheva@fdm.mu-sofia.bg