

Current concepts on the functional somatic syndromes and temporomandibular disorders

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SUMMARY

Background. The importance of psychosocial factors in the etiopathogenesis of temporomandibular disorders (TMD) has led to the hypothesis that these disorders may be part of a wider group of somatoform disorders, the functional somatic syndromes (FSS).

Types of studies reviewed. The present paper is an overview summarizing the current concepts on the TMD-FSS relationship. A non-systematic search in the Medline database identified peer-reviewed papers on the epidemiological and clinical characteristics of the complex groups of disorders labelled functional somatic syndromes, focusing on the common features to temporomandibular disorders patients.

Results. Literature data suggest that FSS and TMD share many etiopathogenetic and epidemiological features, both groups of disorders having a multifactorial etiopathogenesis and needing a multidisciplinary approach to diagnosis and treatment. Psychosocial characteristics of patients seem to have many similarities and the prevalence of Axis I psychiatric disorders is elevated. The majority of studies focused on the relationship between TMD and fibromyalgia (FM), due to the high rate of orofacial involvement related to FM.

Clinical implications. The presence of common features between TMD and FSS patient may suggest the need for changes in the diagnostic and therapeutic approach to TMD patients, with the introduction of treatment protocols which also address the psychosocial impairment accompanying TMD symptoms, in order to overcome the limits of traditional therapies.

Key words: temporomandibular disorders; functional somatic syndromes; somatoform disorders; epidemiology; fibromyalgia; clinical features.

INTRODUCTION

Temporomandibular disorders (TMD) are a heterogeneous group of pathologies affecting the stomatognathic system and the related structures, whose complex and diversified etiology generate several diagnostic and taxonomic problems (1).

At present, temporomandibular disorders are considered to have a multifactorial aetiology, with a number of local and systemic factors that could co-exist and interact to weaken the tolerance threshold

of stomatognathic system's structures, causing the occurrence of TMD signs and/or symptoms (2-4). Among these factors, psychosocial factors has been investigated in a great number of studies demonstrating an increase in stress, anxiety, depression and somatization in TMD patients (2, 5-8).

In particular, patients affected by chronic and painful temporomandibular disorders share many psychosocial characteristics with subjects presenting other chronic painful syndromes at different body regions. This led to the hypothesis that some types of chronic TMD may be part of an interdisciplinary group of somatoform syndromes defined as Functional Somatic Syndromes (FSS), characterized by similar etiopathogenetic patterns and psychosocial impairment (9,10).

The inclusion of TMD in the group of somatoform disorders should have an impact on the literature on TMD epidemiology and classification,

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but might influence the therapeutic approach to TMD patients as well.

Considering these premises, the present paper is an overview of current concepts on the relation between temporomandibular disorders and functional somatic syndromes. In particular, this review has been focused on the current status of research concerning the clinical, taxonomic and etiopathogenetic aspects of FSS, and their possible correlation with TMD on the basis of the observed clinical and etiopathogenetic overlapping.

To provide and summarize up-to-date knowledge, an electronic search in the Medline database was performed to identify English-language, peer-reviewed articles published before June 2006. The key words "temporomandibular disorders", "functional somatic syndromes", "somatization", "myofascial pain", "somatoform disorders" were used, alone or combined, to perform the search.

LITERATURE REVIEW

Functional Somatic Syndromes

The umbrella term Functional Somatic Syndromes (FSS) refers to several related syndromes characterized by a constellation of disabling symptoms and patients' suffering which are not disease-specific and do not recognize objective structural or functional abnormalities. Physicians in many medical specialties increasingly confronted themselves with patients who have disabling, physically unexplained, somatic symptoms and who were already assigned one or more diagnoses for their illness (10).

Since the proposal of the gate control theory (11), a comprehensive biopsychosocial model has been widely used for the study of pain symptoms. This model suggested that pain perception may originate not only from physical stimuli, but from psychic factors which can stimulate the brain to modulate the gate control mechanism and to generate pain perception as well (12,13).

The acceptance of the gate control theory allowed overcoming the ancient distinction between "real" and "imaginary" pain on the basis of the presence or absence of a physical damage, finally demonstrating that body and mind interact to determine symptoms onset.

At present, etiopathogenetic mechanisms at the basis of FSS are only partly known, so their diagnosis is complex and their classification incomplete (10,14,17). Epidemiological data showed a high diffusion of these pathologies; FSS account for 6-36% of medical consultations worldwide, with a prevalence of 20% in the United Kingdom and 35% in USA (10).

These disorders affect mainly women, with a female: male ratio of 5-20:1, and the most common age of onset is under thirty years of age (15).

Many different functional syndromes have been described. Each medical specialty seems to have at least one of them: for rheumatologists, prominent muscle pain and tenderness is fibromyalgia (FM); for gastroenterologists, abdominal pain with altered bowel habit is irritable bowel syndrome (IBS); and for infectious-diseases specialists, chronic fatigue and myalgia is a post-viral or chronic fatigue syndrome (CFS) (9). Many authors claimed that the existence of specific somatic syndromes is largely an artefact of the medical literature (9,10,17,18). That is to say that the differentiation of specific functional syndromes reflects the tendency of specialists to focus only on those symptoms pertinent to their specialty, rather than any real differences between patients.

To support this hypothesis the authors of a recent review underlined common characteristics, such as:

- the published diagnostic criteria for each of the specific functional syndromes overlap in their constituent symptoms;
- patients identified as having one functional somatic syndrome also meet symptoms criteria for others;
- presence of similarities across syndromes in sex prevalence;
- similarities in the proposed aetiology, prognosis, and response to treatment;
- coexistence with psychosocial disorders (9).

Other authors underlined an elevated comorbidity or co-occurrence of two or more syndromes in patients' populations (18). Recently, a review of 53 studies showed a co-occurrence rate from 35% to 70% for fibromyalgia (FM) and chronic fatigue syndrome (CFS), 32-80% for FM and irritable bowel syndrome (IBS), 58-92% for CFS and IBS, 33-55% for FM and Multiple chemical sensitivity (MCS) and 30-67% for CFS and MCS (19).

Functional somatic syndromes are currently described as a group of somatic manifestations which are the expression of a same syndrome generated by central neuroendocrine dysregulation. Such neuroendocrine abnormalities can determine the onset of similar symptoms at different districts, depending on subjective factors (9,20,21). As a consequence pathological precipitation and its somatic expression appear to be influenced by genetic susceptibility, organs' vulnerability, psychosocial factors and alexithymic personality of patients which induce

subjects to react with stress, anxiety, depression or anger (22).

According to most studies, each functional somatic syndrome recognizes a multifactorial aetiology, with an interaction of biological, psychological and interpersonal factors (15). Many authors of different medical fields consider exacerbation and precipitation of many FSS to depend on the emotional response caused by a stressor (10,18,23,24).

This concept was introduced with the theory on homeostasis maintenance, which described the influence of chronic stress on cortisol secretion, determined by the stress-related activation of the hypothalamic-pituitary-adrenal (HPA) axis (22). Widespread somatic manifestations may be the consequence of HPA axis dysfunction; therefore, chronic stress is considered a risk factor for FSS, also due to its influence on anxiety and depression state and, consequently, on patients' disability (6,25,26).

This mechanism has been called into cause for the explanation of the outbreak of some TMD as well. Some authors underlined that TMD symptoms may be related to an unbalanced reaction to stressors and HPA axis dysregulation in some cases (7,27,30).

Neurobiological researches on HPA axis abnormalities, along with the clinical evidences of cortisol deficit in FSS patients, are the basis for the most recent theories on FSS etiopathogenesis.

The association between stress and increased cortisol secretion has been well demonstrated in the literature, but abnormalities of the HPA axis may also induce hypocortisolism in some cases, as described in patients who experienced a traumatic event or have been affected by stress-related pathologies (31). Hypocortisolism has been shown in patients suffering from bodily disorders, such as burnout with physical complaints, chronic fatigue syndrome, fibromyalgia and chronic pelvic pain (18).

Cortisol secretion has a protective role, inducing a number of biological reactions (gluconeogenesis, mobilization of free fatty acids, reduction of protein synthesis) which lead to an increase in energy supplies. Moreover, cortisol secretion controls glucocorticoids action and their effects on immunocellular defensive mechanisms, as described by studies underlining the immunosuppressive action of cortisol to prevent toxic effects of primary defensive mechanism activated as a response to stress (32,33). Thus, a decrease in cortisol availability in traumatized or chronically stressed individuals may determine an increased vulnerability to

bodily disorders, promoting a disinhibition of immune disorders, inflammation, chronic pain syndromes and allergies (20,34).

Several studies revealed the presence of hypocortisolism in many different populations of patients with stress-related bodily disorders, finding support even in the results produced by some animal models (35). Literature data suggest that hypocortisolism has a prevalence of up to 20-25% in patients with stress-related disorders such as chronic fatigue syndrome, chronic pelvic pain, fibromyalgia, post traumatic stress disorders, irritable bowel syndrome, low back pain and atypical depression (34).

A large variety of hypothesis to explain etiopathogenesis of hypocortisolism were proposed. The most plausible model is the multifactorial one, which suggests that many different factors may interact to determine the development and persistence of cortisol lack, through mechanisms of action which may potentially involve all levels of HPA axis (20).

Clinical aspects of Functional Somatic Syndromes

Each functional somatic syndrome may present some organ-specific and disease-specific manifestations but in general, FSS do not have any characteristic clinical presentations or pathognomonic signs and symptoms complexes that are consistent across cases and that distinguish the syndromes from one another (33,35).

The various FSS have remarkably similar symptoms that share important characteristics like diffuse somatic complaints, often associated with pain and non-specific, ambiguous symptoms, which tend to become chronic, even if fluctuant, and which rarely show natural remission (15).

Symptoms which are common to many FSS include fatigue, weakness, sleep difficulties, headache, muscle aches and joint pain among others. In particular, about 20% of FSS patients present muscle fatigue and about 30% musculoskeletal chronic symptoms (36).

Many studies reported a high comorbidity between FSS characterized by muscle chronic pain, such as fibromyalgia, chronic fatigue syndrome, myofascial pain, and psychiatric disorders (18,37-42). Even though the existence of a cause-effect relation is widely debated, the prevalence of Axis I psychiatric disorders is clearly higher in patients with FSS than in the general population, with a prevalence of 50-70% for mood disorders and 40-50% for anxiety in FSS patients (17,37,40,41).

Relationship between TMD and FSS

Literature data suggest that FSS share many etiopathogenetic and epidemiological characteristics with TMD.

Both groups of disorders have a multifactorial etiopathogenesis and need for a multidisciplinary approach to diagnosis and treatment; in particular, psychosocial characteristics of patients seem to have many similarities (2,3,6-8,40). As in the case of FSS, comorbidity between Axis I psychiatric disorders and TMD is also elevated (1,8,16,43-49), with an estimated prevalence of mood disorders in TMD subjects of about 30-60% (3) among Caucasians, and about 43% in Asiatic patients (8,40). Besides, works investigating the prevalence of mood and anxiety psychopathology in different Research Diagnostic Criteria for TMD (RDC/TMD) groups of patients described a significantly higher prevalence of psychiatric disorders, and in particular depressive symptoms, in patients with painful TMD, regardless of pain location (4,5,48,49).

These observations supported the hypothesis that psychopathological symptoms in TMD patients seem to be related to the presence of a painful condition, and seem to be independent by the origin of pain (i.e.: TMJ or myofascial pain). So, available data suggest the inclusion of TMD within the group of disorders for which the complex pain-depression relation may have a predominant role in symptoms expression and perception.

Similarities between the psychosocial traits of TMD and FSS patients also came from studies adopting Minnesota Multiphasic Personality Inventory (MMPI) test, which showed that chronic pain subjects have a typical profile revealing the tendency to manifest an interior conflict through physic symptoms (50).

In general, different studies seem to suggest that up to 80% of TMD patients show a corollary of psychosocial disorders (1,9,43).

As for the clinical relation between FSS and TMD, two studies reported the presence of TMD muscular symptoms in 42-75% of patients with diverse functional somatic syndromes, such as stress syndrome, fibromyalgia, chronic fatigue syndrome, premenstrual syndrome, irritable bowel syndrome (15,18,51-57).

At present, attention is mostly focused on fibromyalgia, a chronic non inflammatory rheumatic disease affecting soft tissues, whose main features are the presence of generalized musculoskeletal pain and tenderness to palpation. Orofacial involvement and stomatognathic symptomatology, and especially masticatory muscles and temporomandibular joint

pain and tenderness, are a common observation in patients with fibromyalgia (FM).

DISCUSSION

Despite knowledge about etiopathogenesis and epidemiology of functional somatic syndromes has improved over the last years, there are still some diagnostic and taxonomic uncertainties which make standardization of classification difficult and let the debate on which pathologies have to be included within the FSS open.

On the basis of some common aspects in their epidemiology and clinical presentation, the inclusion of chronic TMD within the group of FSS was suggested as well. Such a hypothesis is interesting, even though it is likely that only a small amount of temporomandibular disorders patients is actually affected by a functional somatic syndrome (9,16,18,51,54-56).

The inclusion of TMD within the FSS should lead to changes in the diagnostic and therapeutic approach to TMD patients, and especially to those who achieved limited improvements with traditional therapies. The introduction of treatment protocols which also address the psychosocial impairment accompanying TMD symptoms is fundamental to overcome the limits of traditional therapies (50,58-60).

On this purpose, interesting suggestions came from a series of papers investigating the different response to TMD treatment in function their psychological profile (59).

The use of MPI (Multidimensional Pain Inventory) questionnaire allowed identifying three groups of TMD patients on the basis of symptoms' interferences on life activities.

Dysfunctional (DYS) subjects were those with higher levels of pain, interference of symptoms with life activities, affective distress, and lower levels of activity and feeling of life control; Interpersonally Distressed (ID) patients were similar to those of the dysfunctional group. Anyway, they differ from the first group in their experience of little support from significant people in their environment and in their feeling of receiving a great deal of negative responses and few solicitous responses or assistance related to their pain problem by significant others. Finally, Adaptive Copers (AC) were described as remaining active despite pain, feeling little psychological distress or life interference, and continuing to feel in control of their lives despite the presence of symptoms of TMD.

At the end of a traditional treatment protocol improvements of patients of the AC group were significantly higher than the other groups in both clini-

cal and psychological parameters, and a wide range of improvement was still possible for DYS and ID patients. Given these premises, the need for a different management also addressing psychological factors which interfere with DYS and ID patients daily activities was strongly recommended.

Incomplete management of TMD patients was called into cause as a risk factor for treatment failure (60), and the introduction of alternative methods adopted in the treatment of other chronic pathologies, such as the cognitive behavioral therapy, might be very useful to increase treatment success rate (61).

Considering these suggestions, therapeutic protocols adopted for FSS patients might help TMD management.

A number of proposals for the management of functional somatic syndromes were suggested (10,13,17,18,43,62).

Medical management of FSS symptoms and subjects may be synthesized as a therapeutic protocol resting on six steps:

- 1) Ruling out the presence of diagnosable medical disease,
- 2) Searching for psychiatric disorders,
- 3) Building a collaborative alliance with the patient,
- 4) Making restoration of function the goal of treatment even with palliative pharmacological therapy,
- 5) Providing limited reassurance,
- 6) Prescribing cognitive-behavioural therapy for patients who have not responded to the aforementioned five steps.

At the same time, the physician should discourage the patient from assuming the sick role, should undercut alarming expectations about the clinical course, and should avoid making distressing symptom attributions.

In the presence of DSM-IV Axis I disorders like depression or anxiety, it can be adopted a treatment with antidepressant or other drugs. All these interventions help patients to cope with symptoms

by teaching them how to re-examine their health beliefs and expectations and explore the effects of the sick role, stress and distress on their symptoms. They help patients find alternative explanations for symptoms, restructure faulty disease beliefs, alter expectations, and learn techniques of focused attention and distraction.

Behavioural strategies, such as response prevention, systematic desensitization, graduated exercise regimens, and progressive muscle relaxation, help those with functional somatic syndromes resume normal activities, minimize role impairment, and curtail sick role behaviours.

A recent review of 31 controlled trials on cognitive-behavioral therapy for the treatment of somatic symptoms claimed that available data supported the short-term efficacy of such interventions and that further investigations are needed with longer follow-up periods to confirm promising results from controlled trials with long-term follow-up (63-68). Cognitive-behavioral interventions have shown to be effective in reducing somatic symptoms, generalized distress, and disability, also thanks to the higher compliance of patients with respect to other treatment modalities.

CONCLUSIONS

The present overview represents an attempt to describe the complex relation between functional somatic syndromes and temporomandibular disorders on the basis of present knowledge on chronic pain pathologies. Only few studies have been designed to investigate the TMD-FSS relation, and literature data came from too different specialties and settings. Several common epidemiological, clinical and psychosocial characteristics between TMD and some FSS, such as fibromyalgia and chronic fatigue syndrome seem to exist. These considerations have a twofold importance in the field of temporomandibular disorders, since they may contribute to improve knowledge at both diagnostic and therapeutic levels (57, 58, 59).

REFERENCES

1. Lupton DE. Psychologic aspects of temporo-mandibular joint dysfunction. *J Am Dent Assoc* 1969; 79: 131-6.
2. Dworkin SF. Perspectives on the interaction of biological, psychological and social factors in TMD. *J Am Dent Assoc* 1994; 125: 856-63.
3. Ferrando M, Andreu Y, Jose Galdon M, Dura E, Poveda R, Vincente Bagan J. Psychological variables and temporomandibular disorders: Distress, coping, and personality. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2004; 98: 153-60.
4. Fantoni F, Manfredini D, Dell'Osso L, Bosco M. Validity of the General Spectrum Measure-V (GSM-V) questionnaire for psychiatric assessment in temporomandibular disorder patients. Preliminary data. *Minerva Stomatol* 2007. In press.
5. Manfredini D, Bandettini di Poggio A, Cantini E, Dell'Osso L, Bosco M. Mood and anxiety psychopathology and temporomandibular disorder: a spectrum approach. *J Oral Rehabil* 2004; 31: 933-40.
6. Sternbach RA. Psychological dimensions and perceptual

- analyses, including pathologies of pain. In: Carterett ED, Friedman MD, eds. *Handbook of Perception*. New York: Academic Press; 1978. p 231-61.
7. Fearon CG. Stress: a common denominator for non-organic TMJ pain dysfunction. *J Prosthet Dent* 1983; 49: 805-8.
 8. Yap AUJ, Chua EK, Tan KBC. Depression and somatization in patients with temporomandibular disorders. *J Prosthet Dent* 2002; 88: 479-84.
 9. Wessely S, Nimnuam C. Functional Somatic Syndromes: one or many? *Lancet* 1999; 354: 936-9.
 10. Barsky AS, Borus JF. Functional Somatic Syndromes. *Ann Intern Med* 1999; 130: 910-21.
 11. Melzack R, Wall PD. Pain mechanism: a new theory. *Science* 1965; 150: 971-9.
 12. Fillingim RB. Individual differences in pain responses. *Curr Rheumatol Rep* 2005; 7: 342-7.
 13. Philips HC. The psychological management of chronic pain. *Behav Res Ther* 1989; 27: 460-8.
 14. Schappert SM. National Ambulatory Medical Care Survey: 1989 summary. *Vital Health Stat* 1992; 13: 1-80.
 15. Henningsen P, Zimmermann T, Sattel H. Medically unexplained physical symptoms, anxiety, and depression: a meta-analytic review. *Psychosom Med* 2003; 65: 528-33.
 16. Korszun A, Papadopoulos BS, Demitrack M, Engleberg C, Crofford L. The relationship between temporomandibular disorders and stress-associated syndrome. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 86: 416-20.
 17. Mayou R, Farmer A. ABC of psychological medicine: Functional somatic symptoms and syndromes. *Br Med J* 2002; 325: 265-8.
 18. Kroenke K. Patients presenting with somatic complaints: epidemiology, psychiatric comorbidity and management. *Int J Methods Psychiatr Res* 2003; 12: 34-43.
 19. Aaron LA, Buchwald D. A review of the evidence for overlap among unexplained clinical conditions. *Ann Intern Med* 2001; 134: 868-81.
 20. Heim C, Ehler U, Hellhammer DH. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. *Psychoneuroendocrinology* 2000; 25: 1-35.
 21. Muhammad B, Yunus MD. Functional Somatic Syndromes. Best Practice of Medicine. 2000.
 22. Cassano GB. *Manuale di Psichiatria*. UTET; 2002.
 23. Ford CV. *The Somatizing Disorders: Illness as a Way of Life*. New York: Elsevier Biomedical; 1983.
 24. Hall EM, Johnson JV. A case study of stress and mass psychogenic illness in industrial workers. *J Occup Med* 1989; 31: 243-50.
 25. Kirmayer LJ, Robbins JM, Paris J. Somatoform disorders: personality and the social matrix of somatic distress. *J Abnorm Psychol* 1994; 103: 125-36.
 26. Salovey P, Birnbaum D. Influence of mood on health-relevant cognitions. *J Pers Soc Psychol* 1989; 57: 539-51.
 27. Pierce CJ, Chrisman K, Bennett ME, Cluse JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. *J Orofac Pain* 1995; 9: 51-6.
 28. Auvenshine RC. Psychoneuroimmunology and its relationship to the differential diagnosis of temporomandibular disorders. *Dent Clin North Am* 1997; 41: 279-96.
 29. Landi N, Lombardi I, Manfredini D, Casarosa E, Biondi K, Gabbanini M, et al. Sexual hormone serum levels and temporomandibular disorders. A preliminary study. *Gynecol Endocrinol* 2005; 20: 99-103.
 30. Landi N, Manfredini D, Lombardi I, Casarosa E, Bosco M. 17-beta-estradiol and progesterone serum levels in temporomandibular disorder patients. *Minerva Stomatol* 2004; 53: 651-60.
 31. Yehuda R, Southwick SM, Nussbaum G, Wahby V, Giller EL Jr, Mason JW. Low urinary cortisol excretion in patients with posttraumatic stress disorder. *J Nerv Ment Dis* 1990; 178: 366-9.
 32. Yehuda R, Teicher MH, Trestman RL, Levengood RA, Siever LJ. Cortisol regulation in posttraumatic stress disorder and major depression: a chronobiological analysis. *Biol Psychiatry* 1996; 40: 79-88.
 33. Kelso A, Munck A. Glucocorticoid inhibition of lymphokine secretion by alloreactive T lymphocyte clones. *J Immunol* 1984; 133: 784-91.
 34. Bateman A, Singh A, Kral T, Solomon S. The immune hypothalamic pituitary adrenal axis. *Endocr Rev* 1989; 10: 92-112.
 35. Fries E, Hesse J, Hellhammer J, Hellhammer DH. A new view on hypocortisolism. *Psychoneuroendocrinology* 2005; 30: 1010-6.
 36. Cunningham LS, Kelsey JL. *Epidemiology of musculoskeletal impairments*. Elsevier Biomedical; 1983.
 37. Terr AI. Environmental illness. A clinical review of 50 cases. *Arch Intern Med* 1986; 146: 145-9.
 38. Hyams KC. Lessons derived from evaluating Gulf War syndrome: suggested guidelines for investigating possible outbreaks of new diseases. *Psychosom Med* 1998; 60: 137-9.
 39. Fiedler N, Kipen HM, DeLuca J, Kelly-McNeil K, Natelson B. A controlled comparison of multiple chemical sensitivities and chronic fatigue syndrome. *Psychosom Med* 1996; 58: 38-49.
 40. Yap AUJ, Chua EK, Tan KBC. Depression symptoms in Asian TMD patients and their association with non-specific physical symptoms reporting. *J Oral Path Med* 2004; 33: 305-310.
 41. Simon GE, VonKorff M, Piccinelli M, Fullerton C, Ormel J. An international study of the relation between somatic symptoms and depression. *N Engl J Med* 1999; 341: 1329-35.
 42. Ekstrand J, O'Malley PG, La butta R, Jackson JL. Mental disorders in a neurology clinic setting. *J Gen Med* 2000; 15 (Suppl 1): 112.
 43. Harris MB, Fillingim RB, Sigurdsson A, Kincaid S, Maixner W. Pain sensitivity in patients with temporomandibular disorders: relationship to clinical and psychosocial aspects. *Clin J Pain* 1996; 12: 260-9.
 44. Gatchel RJ, Garofalo JP, Ellis E, Holt C. Major psychological disorders in acute and chronic TMD: an initial examination. *J Am Dent Assoc* 1996; 127: 1365-74.
 45. List T, Dworkin SF. Comparing TMD diagnoses and clinical findings at Swedish and US TMD centers using research diagnostic criteria for temporomandibular disorders. *J Orofac Pain* 1996; 10: 240-53.
 46. Kight M, Gatchel RJ, Wesley L. Temporomandibular disorders: evidence for significant overlap with psychopathology. *Health Psychol* 1999; 18: 177-82.
 47. Kino K, Sugisaki M, Ishikawa T, Shibuya T, Amagasa T, Miyaoka H. Preliminary psychological survey of orofacial outpatients. Part 1: Predictors of anxiety or depression. *J Orofac Pain* 2001; 15: 235-44.
 48. Manfredini D, Bandettini di Poggio A, Romagnoli M, Dell'Osso L, Bosco M. A spectrum approach for the assessment of manic-depressive symptoms accompanying temporomandibular disorders. *Minerva Stomatol* 2003; 52: 231-6.
 49. Manfredini D, Bandettini di Poggio A, Romagnoli M, Dell'Osso L, Bosco M. Mood spectrum in patients with different painful temporomandibular disorders. *Cranio* 2004; 22: 234-240.
 50. McCreary CP. The usefulness of the Minnesota Multiphasic Personality Inventory in the clinical management of patients with temporomandibular disorders. *Ann Acad Med Singapore* 1995; 24: 38-42.
 51. Plesh O, Wolfe F, Lane N. The relationship between fibromyalgia and temporomandibular disorders: prevalence and symptom severity. *J Rheumatol* 1996; 23: 1948-52.
 52. Hedemberg-Magnusson B, Ernberg M, Kopp S. Symptoms

- and signs of temporomandibular disorders in patients with fibromyalgia and local myalgia of the temporomandibular system: a comparative study. *Acta Odontol Scand* 1997; 55: 344-9.
53. Raphael KG, Marbach JL, Klausner J. Clinical characteristics of those with regional vs widespread pain. *J Am Dent Assoc* 2000; 131:161-71.
 54. Manfredini D, Salvetti G, Fantoni F, Bosco M. Relationship between fibromyalgia and temporomandibular disorders: a review of current understandings. *J Chin Clin Med* 2006; 1: 336-41.
 55. Salvetti G, Manfredini D, Bazzichi L, Bosco M. Clinical features of the stomatognathic involvement in course of fibromyalgia syndrome. A comparison with temporomandibular disorders patients. *Cranio* 2007. In press.
 56. Manfredini D, Tognini F, Montagnani G, Bazzichi L, Bombardieri S, Bosco M. Comparison of masticatory dysfunction in temporomandibular disorders and fibromyalgia. *Minerva Stomatol* 2004; 53: 641-50.
 57. Rhodus NL, Friction J, Carlson P, Messner R. Oral symptoms associated with fibromyalgia syndrome. *J Rheumatol* 2003; 30: 1841-5.
 58. Kaplan AJ, Assael LA. Temporomandibular disorders. Diagnosis and treatment. W.B. Sanders Co. ; 1990.
 59. Turk DC. Psychological assessment of patients with persistent pain II. Alternative views. *Pain Manag* 1990; 3: 227-37.
 60. Turk DC. Psychosocial and behavioral assessment of patients with temporomandibular disorders: diagnostic and treatment implications. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 83: 65-71.
 61. Dworkin SF. The case for incorporating biobehavioral treatment into TMD management. *J Am Dent Assoc* 1996; 127: 1607-10.
 62. Kroenke K, Swidle R. Cognitive-behavioral therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. *Psychother Psychosom* 2000; 69: 205-15.
 63. Black DW, Rathe A, Goldstein RB. Environmental illness. A controlled study of 26 subjects with "20th century disease." *J Am Med Assoc* 1990; 264: 3166-70.
 64. Brodsky CM. "Allergic to everything": a medical subculture. *Psychosomatics* 1983; 24:731-2, 734-6, 740-2.
 65. Clark DM, Salkovskis PM, Hackmann A, Wells A, Fennell M, Ludgate J. Two psychological treatments for hypochondriasis. A randomized controlled trial. *Br J Psychiatry* 1998; 173: 218-25.
 66. Sharpe M. Cognitive behavioural therapies in the treatment of functional somatic symptoms. In: Mayou R, Bass C, Sharpe M, eds. *Treatment of Functional Somatic Symptoms*. New York, Oxford Univ Press; 1995. p.122-43.
 67. Mayou RA, Bryant BM, Sanders D, Bass C, Klimes I, Forfar C. A controlled trial of cognitive behavioural therapy for non-cardiac chest pain. *Psychol Med* 1997; 27: 1021-31.
 68. Lidbeck J. Group therapy for somatization disorders in general practice: effectiveness of a short cognitive-behavioural treatment model. *Acta Psychiatr Scand* 1997; 96: 14-24.

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