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Review of aetiological concepts of temporomandibular pain disorders: towards a biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors

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Abstract

Several studies have reported that musculoskeletal disorders of the stomatognathic system, commonly known as temporomandibular disorders (TMD) resemble musculoskeletal disorders and chronic pain disorders in general. There is also general consensus that combined biomedical and biopsychosocial methods best support the assessment and management of the cardinal features of TMD, i.e., pain and dysfunction or physical (peripheral) and psychosocial (central) factors. This overview of the aetiology of TMD will outline conceptualizations of past models and present the current view that patients with TMD should be assessed according to both the physical disorder and the psychosocial illness impact factors. The conceptual theories outlined in this review include biomedical models related to temporomandibular joints, muscles of mastication and occlusal factors, psychological models and the biopsychosocial models. An integrated and multidimensional approach concerning physical and psychosocial factors in temporomandibular pain and dysfunction is presented as an example of how the biopsychosocial model and information processing theory may apply in the conceptualization and management of TMD for various health care professionals.

Keywords: Aetiology; Temporomandibular; Pain; Biomedical; Biopsychosocial; Theory

1. Introduction

Several studies have reported that musculoskeletal disorders of the stomatognathic system resemble musculoskeletal disorders and pain disorders in general (Dworkin, 1995b; Suvinen and Reade, 1995; Okeson, 1996; Dworkin and Suvinen, 1998; Sessle and Dubner, 2001; Turk and Melzack, 2001; Walker et al., 2004). There is also general support that biopsychosocial models and methods, which reflect integration of biomedical diagnostic and treatment methods as well as assessment of psychological status and psychosocial level of function, best support their assessment and management

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(Dworkin and LeResche, 1992; Dworkin et al., 1992; Suvinen, 1992; Fricton and Dubner, 1995; Okeson, 1996; Suvinen and Reade, 1995; Dworkin and Suvinen, 1998, Sessle and Dubner, 2001; Turk and Melzack, 2001). At the same time there is acknowledgment that the stomatognathic system is also unique, e.g., in comparison to the spine or the knee as it contains teeth (dentition, occlusion) between bilaterally functioning temporomandibular joints (TMJs) and the associated musculature (Okeson, 1996). TMJs are also unique compared to most synovial joints, as the articular surfaces are lined with dense fibrous connective tissue, not hyaline cartilage, considered to have greater ability for repair (Okeson, 1996).

Several diagnostic names for musculoskeletal disorders of the temporomandibular region have been presented in the literature over the years, reflecting the different theories of aetiology and the different emphasis of the cardinal causative factors thought to be responsible for the various symptoms and signs present in the patient populations (Suvinen, 1992). Currently the term 'temporomandibular disorders' (TMD) has been recommended as a 'collective term embracing a number of clinical problems that involve the masticatory musculature, the TMJs and associated structures, or both' (Griffiths, 1983). TMD are identified as 'a major cause of nondental pain in the orofacial region and are considered to be a subclassification of musculoskeletal disorders' (Okeson, 1996). The Technology Advancement Conference by the National Institutes of Health (NIH) on TMD (NIH, 1997) defined these disorders according to two broad aspects, i.e., pain and psychosocial dysfunction. There now seems to be increasing evidence that these two aspects are the important, if not cardinal, features that make patients seek treatment. At the same time there is uncertainty as to the actual underlying aetiology of TMD (LeResche, 1997). In most cases the diagnosis of TMD is based on careful patient history taking (or anamnesis) and clinical examination, which depends on patient report of levels of pain/discomfort of the TMJs and associated muscles. Often patients with TMD also describe symptoms of pain and dysfunction affecting ears, eyes and/or throat and headaches that involve some or all of the frontal, temporal, parietal, occipital and neck regions. Clinical examination methods include measures of quasi-objective factors that define limitations of mandibular function and tenderness of head and neck muscles. These are currently based on a consensus among leading researchers and clinicians internationally and probably the most widely studied measure of these variables is the Research Diagnostic Criteria for TMD (RDC/TMD) developed at the University of Washington by Dworkin and LeResche (1992). The current perspective regarding TMD is now multidimensional, with an appreciation that a combination of physical, psychological and social factors may contribute to the overall presentation of this disorder – hence the preference for a biopsychosocial integrated approach (Dworkin and LeResche, 1992; Suvinen, 1992; Okeson, 1995; Suvinen and Reade, 1995; Suvinen et al., 1997b, 2005; Dworkin and Suvinen, 1998; Dworkin, 2001; Sessle and Dubner, 2001; Turk and Melzack, 2001).

The aim of this review is to present and critically summarize key concepts and limitations related to the past models, including the biomedical and the psychological models, to outline the earlier integrated multifactorial concepts and to present the new and current biopsychosocial concepts in the aetiology of TMD. An integrated and multidimensional approach of physical (peripheral) and psychosocial (central) factors in temporomandibular pain and dysfunction is presented as an example of how the biopsychosocial model and information processing theory may apply in the therapeutic conceptualization of TMD based on this review for the many health care professionals involved in the management of TMD.

2. Methods to study the aetiology of TMD

The reasons for causation of the many symptoms and signs present in patients with TMD have long eluded both the researchers and clinicians involved in the assessment and management of these conditions (Clark, 1991; Greene, 1995; McNeill, 1997). Based on the general premise, scientific methods available to study causation include animal and human studies. Several recent reviews discuss the interrelatedness of factors involved at the peripheral and central levels and of craniofacial pain and motor function, with implications for more targeted future therapeutic propositions (Browne et al., 1998; Fricton, 1999; Stohler, 1999; Sessle, 2000; Svensson and Graven-Nielsen, 2001; Tenenbaum et al., 2001; Lobbezoo et al., 2002). For example, with the help of sophisticated electrophysiological, neuroanatomical and neuroimaging methods, several investigators have been able to show that peripheral trigeminal pain and inflammation can disturb orofacial motor function (refer to reviews by Stohler, 1999; Svensson and Graven-Nielsen, 2001; Lobbezoo et al., 2002). This disturbance can lead to neuroplastic changes and malfunctions in the central nervous system (refer to reviews by Sessle, 2000; Tenenbaum et al., 2001), which responses are thought to play a role in the aetiology of chronic pain syndromes. Animal studies generally examine the phenomenon of pain at the peripheral and central levels of processes present in pain transmission and perception and morphopathological changes (Sessle and Hu, 1991; Sessle, 2000). The study of chronic pain in humans, however, needs to address much more complex issues of pain appraisal and response, which vary considerably

from patient to patient, including the involvement of the emotional-affective system, cognitions, learning principles, pain behaviour and societal and environmental factors (Suvinen and Reade, 1995; Dworkin and Suvinen, 1998; Melzack, 1999a).

Epidemiological research has contributed to the understanding of the prevalence and incidence of the disorder/s in the general as well as the clinic populations (Carlsson and LeResche, 1995; LeResche, 1997; Stohler, 1997; Drangsholt and LeResche, 1999). Due to differences in definitional criteria and methodology, the prevalence of the symptoms and/or signs of TMD has been reported to vary from 6% to 93%, whilst only 3.6% to 7% of the general populations have been estimated to be in need of treatment (Dworkin et al., 1990; De Kanter et al., 1993; Okeson, 1996; Carlsson, 1999; Macfarlane et al., 2002c). The societal costs of TMD, however, are considered to be significant (Okeson, 1996; White et al., 2001). The majority of patients in clinic samples are female, and this has been explained to be due to an interaction of a variety of factors ranging from biological and hormonal factors to psychological and social factors (Carlsson and LeResche, 1995; LeResche, 1997; Klineberg et al., 1998; Dao and LeResche, 2000; Rollman and Lautenbacher, 2001; Macfarlane et al., 2002a; McGregor et al., 2003). In a review LeResche (1997) outlined how epidemiology can be used in the assessment of community health, use of health services, identification of risk factors, identification of syndromes and the clinical picture as well as indicators of aetiology. LeResche (1997) concluded that further research was needed to study aetiological factors associated with temporomandibular pain and dysfunction; as well as the contribution of biologic, psychological and psychosocial illness impact factors.

In the past, research into the aetiology or conceptualization of TMD, has depended on the available methodology and the ideas and theories that various researchers have formulated to test hypotheses of aetiology (Clark, 1991; Greene, 1995; McNeill, 1997). This research has depended largely on the biomedical model, but in recent decades there has been increasing evidence and support for a more integrated model, known as the biopsychosocial model, that incorporates psychological and psychosocial factors into illness experience (Engel, 1977; Dworkin et al., 1992; Dworkin, 2001; Dworkin and Ohrbach, 2001).

3. The typical profile of patients with TMD

Before we review and discuss the various aetiological models presented in this field, it is important to formulate a picture of the typical patient with TMD as reported in the literature. Fig. 1 is a schematic drawing of some of the various symptoms and signs presented in the literature in relation to pain and dysfunction in the temporomandibular region. As shown, many associated symptoms, varying from head pain, to pain in the ears, jaw and neck, as well as disturbances of function are the general features discussed. For more specific guidelines regarding assessment and management of TMD refer to Dworkin and LeResche (1992), Okeson (1996, 1997), NIH (1997) and Dworkin and Ohrbach (2001). Generally speaking, a typical patient will have two of the following

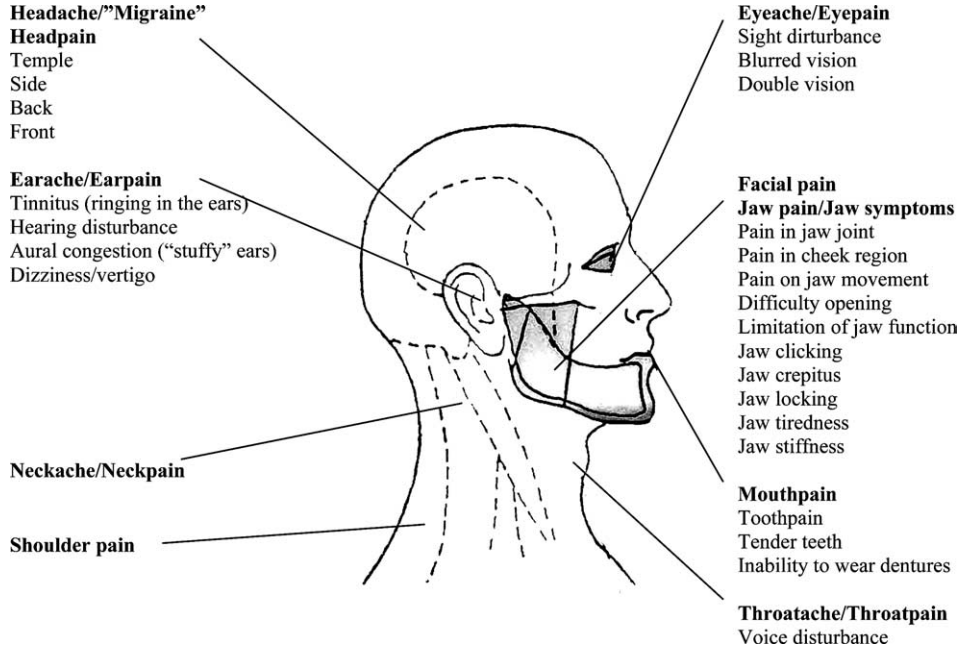

Fig. 1. A schematic drawing of some of the various symptoms and signs causing pain and dysfunction in the temporomandibular region.

to be classified as suffering from TMD, i.e., pain/discomfort in the jaw, mainly in the region of the TMJs and/or muscles of mastication, limitation of mandibular function and/or TMJ sounds (Okeson, 1996). Even though there is evidence that TMJ sounds alone are frequently a natural phenomenon in the general population and fluctuate longitudinally, they are also recognized as a physical diagnostic subcategory of TMD (Dworkin and LeResche, 1992; Okeson, 1996; Kononen et al., 1997).

4. Towards an integrated approach concerning the aetiology of TMD

Based on the NIH Consensus Conference (1997) and several recent studies, it can be hypothesized that the cardinal features of TMD could be viewed from two main domains: a pain domain and a dysfunctional domain, including individual variability (Fig. 2). This conceptualization integrates biomedical models into the biopsychosocial models. Consequently we can, in any given patient, pay attention to how the structural/functional dysfunction and pain are reported as well as what impact these symptoms (dysfunction and pain) are having on the psychological status and psychosocial functioning of patients. The following review will outline biomedical, psychological and biopsychosocial models of aetiology as they relate to the understanding of the cardinal elements of TMD, pain and dysfunction, as well as an integrated conceptual model of aetiology. Fig. 3 presents a synthesis of the various aetiological concepts related to the physical, psychological and psychosocial dimensions of TMD that will be presented in the following sections as part of this review. For other overviews of the historical and biomedical concepts as well as the evolution of these concepts into more multidimensional biopsychosocial conceptualizations refer to

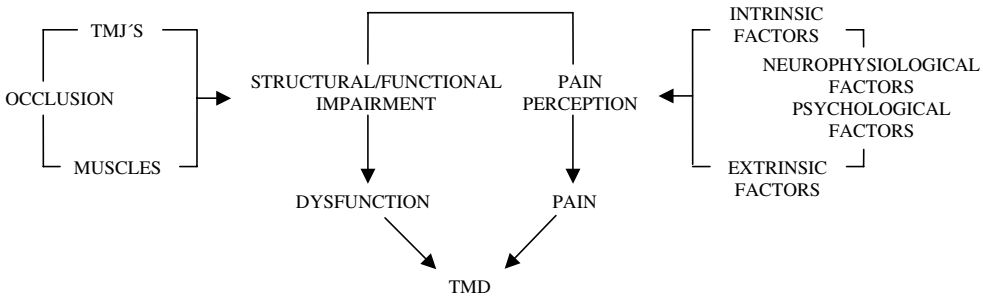

Fig. 2. Cardinal features of temporomandibular pain and dysfunction.

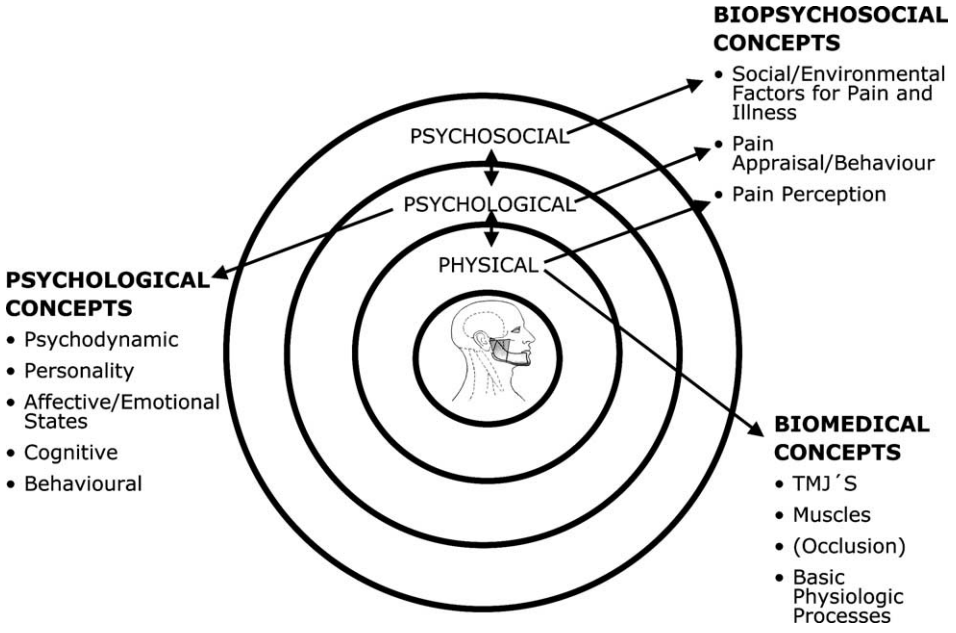

Fig. 3. Summary of the bio-psychosocial concepts reviewed in the aetiology of TMD.

papers by Clark (1991), Dworkin et al. (1992), Greene (1995) and McNeill (1997).

5. Biomedical concepts of the aetiology of TMD

5.1. Aetiological concepts of TMD related to temporomandibular joints

Historically the TMJs and their position in the glenoid fossae were regarded as principal elements causing TMD. The symptoms and signs of TMD were explained by mandibular overclosure after the loss of posterior teeth, which displaced the TMJs distally in their fossae and caused impingement on the auriculotemporal nerve (Prentiss, 1918; Monson, 1921; Goodfriend, 1933; Costen, 1934). Although the anatomical explanation of this theory was proven incorrect, the concept of condylar displacement became the foundation of what was later known as the 'mechanical displacement theory of the temporomandibular articulation' (De Boever and Adrianes, 1983). Later disruptions in the structure and function of the soft tissue structures of the joint complex, including the disc, have been viewed as important causative factors in TMD (Farrar, 1978). These theories have largely depended on the advances in articular imaging, especially in soft tissue imaging, but correlation of data to clinical presentation remains unclear (Chu et al., 2001). Generally speaking, the aetiological concepts related to TMJs can be divided into functional theories and structural or morphopathological theories, including, e.g., such concepts as the trauma theory, the internal derangement theory, the mechanical displacement theory and the osteoarthritic theory.

Zarb and Speck (1979) considered micro-/macrotrauma as a principal unifying concept that initiated pathologic processes and functional dysfunction in different parts of the stomatognathic system thus leading to the symptoms of TMD. Others have aimed at unifying the musculoskeletal disorders of the temporomandibular region with biomedical models of musculoskeletal disorders affecting other joints of the body. For example, Reade (1984) proposed a unifying concept of the aetiology of TMD based on a sports-injury paradigm as a form of orthopaedic injury, Stegenga et al. (1989) proposed a unifying concept of osteoarthrosis according to the concepts of rheumatology.

More specifically, it was proposed in the trauma theory according to Zarb and Speck (1979) that the common denominator in all dysfunction of the masticatory system is trauma, either micro- or macro-trauma. According to this theory macro-trauma (resulting in sprain, capsulitis and spasm) could be caused by excessive mandibular opening, such as wide yawning, laughing, biting on large objects, by sudden pressure or a blow, whiplash, long dental appointment, even incorrect occlusal correction. Micro-traumatic lesions (resulting in strain, bruxism and stress syndrome) were explained to be caused by occlusal discrepancies, parafunctions (particularly bruxism), adverse life-event and stress and arthritic and arthrotic changes by systemic conditions such as rheumatoid arthritis and osteoarthritis or repetitive trauma associated with ageing and tooth loss. Consequently, even though the aetiological premise of this theory was related to trauma, it was actually an earlier multidimensional aetiological model, however, no critical appraisal for the multitude of factors listed was given in the causation of TMD.

Similarly, Reade (1984) supported the unifying concept of trauma in explaining the aetiology of TMD. According to Reade's biomedical theory precipitating factors lead to a traumatic incident, a 'sprain' of the soft tissues of the TMJs, as for a 'sprained ankle', which results in pain, muscle guarding and spasm. Depending on the presence of maintaining factors the 'sprain' would either resolve or persist. These maintaining influences according to Reade (1984) were explained to include disrupted occlusion, parafunctional habits (particularly bruxism), and recreational and occupational activities, which would prolong stress on the joint and thus prevent healing, however, it is not clear how, e.g., occlusion would be causally linked to TMD. Apart from factors causing increase or adverse functional loading, psychological elements were recognized as important maintaining influences. According to Reade (1984) 'this theory would explain why similar occlusal interferences do not cause similar symptoms in different individuals and why all individuals with stress do not develop TMD'. The actual biological predictors more recently postulated could include hormonal and structural factors as well as intra- and inter-psychic factors (Carlsson and LeResche, 1995; LeResche, 1997; Dao and LeResche, 2000; Rollman and Lautenbacher, 2001).

The osteoarthritic theory (Stegenga et al., 1989) was based on the premise that osteoarthrosis is the cause of TMD. According to this theory muscular symptoms and internal derangement were secondary to joint pathology. Remodelling was regarded as the normal adaptation to joint loading, and that pathological changes in the TMJs could be induced by absolute or relative overloading. In the former, joint loading is increased through trauma or joint instability (such as hypermobility). In the latter the adaptive capacity of the joint structures is reduced by inflammation and ageing. Stegenga et al. (1989) proposed a sequence of morphopathological changes leading to cartilage breakdown and decreased sliding properties of the joint surfaces. The accompanying symptoms would include disc displacement and limitation of opening movement. Depending on aggravating environmental influences the progression of the disease or attempts to repair would follow. If the cartilage breakdown was progres-

sive, perforation or disruption of the disc and denudation of subchondral bone would follow. This theory may be helpful in explaining some subcategories of TMD, but lacks in its ability to unify, even though several hypotheses are presented to explain the accompanying symptoms and signs observed in patients with TMD.

5.2. Aetiological concepts of TMD related to muscles of mastication

Schwartz (1959) was one of the first investigators to propose the importance of muscle spasm as a primary aetiological factor in initiating pain and dysfunction in the temporomandibular region. He also proposed the importance of the psychological characteristics of the patient. These observations preceded what is now known as the 'psychophysiological theory'.

According to the psychophysiological theory masticatory muscle spasm was responsible for the symptoms of pain and dysfunction (Laskin, 1969). The muscle spasm could be initiated by muscle over-extension, muscle over-contracture or muscle fatigue. The major cause was related to muscle fatigue caused by parafunctional habits used by patients as a method of relieving tension. Occlusal disturbances were explained as aggravating influences. The muscle spasm, it was proposed, leads to pain and limitation of function and may cause occlusal changes. At later stages the functional problem could lead to structural changes, such as degenerative joint changes or muscle contracture, thus rendering the syndrome self-perpetuating. The pain-dysfunction syndrome, also termed as myofascial pain-dysfunction syndrome (MPD) has been explained primarily to be a functional psychophysiological disorder, where occlusal changes and joint changes have been argued to be secondary factors caused by muscle spasm.

Experimental evidence for this theory has been proposed in epidemiological, radiological, psychological, biochemical and physiological studies as follows. According to the epidemiological studies the majority of patients with TMD have shown tenderness in the muscles of mastication (Greene et al., 1969; LeResche, 1997). The radiological studies have shown a low prevalence of observed pathology in the TMJ between cases and controls (Brooks et al., 1992). Psychological studies have shown that patients with functional disorders of the temporomandibular region have similar psychological profiles and psychosocial dysfunction as other chronic musculoskeletal pain disorders such as tension type headache and back or arthritic pain (Suvinen and Reade, 1995; Dworkin and Suvinen, 1998). Studies have reported that patients with MPD showed elevated concentrations of urinary 17-hydroxysteroids and catecholamines, believed to be indicators for increased stress (Evaskus and Laskin, 1972). Others have shown significantly higher cortisol response to experimental stress in TMD patients compared to controls (Jones et al., 1997). Physiological studies have shown that experimental pain in the masticatory muscles can impair masticatory motor functions (Svensson and Graven-Nielsen, 2001) and that muscles of patients with TMD are less fatigue resistance (Direnfeld, 1967 cited in Laskin, 1969). Christensen (1981) showed that fatigue, not pain, caused prolonged muscle activity due to slowed relaxation of muscle fibres. Yemm (1971, 1979) demonstrated that patients with MPD showed increased muscle activity as a result of a stressful task and that it was sustained longer than in the control group.

With regard to the morphopathological explanation 'the muscle theory' emphasized muscles as the primary site of pathological processes. This theory was based on the 'hypokinetic disease' concept of Kraus (1963, 1966). According to Kraus the civilised, mechanised way of life leads to diseases that were due to lack of exercise, overstimulation and tension, and repeated normal response to unreleased fight and flight stimuli. The constant muscle tension caused muscle weakening, stiffening and finally spasm and pain. Pain could be elicited in the muscles or in referred areas of pain by palpation and were referred to as trigger points (Travell, 1960).

Some earlier experimental support for the muscle theory was provided by Schwartz (1959), who reported that patients with TMD showed a generalized muscle response in addition to a specific one related to masticatory muscles. Yemm (1976) supported the proposition that centrally originating muscle hyperactivity, as a response to stress, was responsible for the various signs and symptoms of TMD. More recent studies have indicated the part that stress plays in the aetiology of TMD (Delcanho, 1995; Pierce et al., 1995; Ohrbach and McCall, 1996; Auvenshine, 1997; Jones et al., 1997; Ohrbach et al., 1998) and that patients with TMD can also have generalised fibromyalgia (Klineberg et al., 1998; Raphael and Marbach, 2000; Rhodus et al., 2003). However, the evidence regarding the presence of symptom-specific stress-related psychophysiological responses in chronic pain patients, including TMD, has been found to be inconclusive (Flor and Turk, 1989) and the actual relationship between pain and muscle hyperactivity is not clear (Lund, 1995).

Overall, to date many studies, including epidemiological, neuromuscular and neurophysiological have been inconclusive regarding the theoretical concept of whether muscle symptoms are the cause or the consequence of TMD and what part they play in the clinical manifestation of TMD symptoms.

5.3. Aetiological concepts of TMD related to occlusal factors

The most common theory of occlusal factors in the causation of TMD is the 'neuromuscular theory'.

According to this concept occlusal interferences will induce imbalance in the neuromusculature through proprioceptive feedback, leading to pain and spasm. This imbalance could occur even without teeth due to lack of tooth support. Joint receptors and muscle spindles would then have the primary control over sensitivity and function.

Experimental evidence for and against the neuromuscular theory can be found in epidemiological surveys, autopsy studies, clinical studies, electromyographic studies and case-control studies. Some epidemiological studies have shown a relationship between the number of remaining teeth and the prevalence of TMD (Agerberg and Carlsson, 1973; Helkimo, 1976), whilst others have failed to show any correlation between occlusal factors and TMD, or case control differences in occlusal factors (Seligman and Pullinger, 1991; LeResche, 1997). Similarly many clinical and electromyograhic studies have failed to show any significant differences in patients with TMD and controls in terms of occlusal variables (Seligman and Pullinger, 1989; LeResche, 1997). In one of the few case-control studies, Seligman and Pullinger (1989) compared 196 patients with TMD and 222 controls. They reported a lack of a predictable relationship between occlusal factors and TMD, but postulated that by subdividing the patients into different diagnostic subgroups selective occlusal variables appeared to be associated with some of the subgroups of TMD. The main weakness of this theory was its inability to show differences between patients and controls. Some interpreted this to be caused by avoidance patterns (Storey, 1976) or functional adaptability (Krough-Poulsen, 1969). In a series of several recent long-term follow-up studies no consistent pattern between occlusal variables and TMD was found (Carlsson et al., 2002, 2003; Egermark et al., 2003). The relationship between bruxism, stress and TMD is also not clear (Biondi and Picardi, 1993; Kampe et al., 1997a,b; Pergamalian et al., 2003). Several studies have reported that bruxism is more prevalent than TMD and that not all patients with TMD grind their teeth (Agerberg and Carlsson, 1972; Helkimo, 1974a,b; Glaros and Rao, 1977; Marbach et al., 1990a; Glaros et al., 2000). The review by Marbach et al. (1990a) concluded that clinicians 'aetiological model may influence patients' reporting of grinding'. He emphasized the need to verify tooth grinding in relation to other signs and symptoms and cautioned against experimenter and sampling bias.

To summarize, it seems remarkable that many clinicians consider occlusal factors to be the prime or at least a co-factor in the initiation, aggravation or acceleration of dysfunctional processes, despite substantial evidence that supports a more biomedical or biopsychosocial model (Clark, 1991; Dworkin et al., 1992; Greene, 1995; McNeill, 1997).

5.4. Summary of biomedical aetiological concepts

There is considerable evidence that biomedical or physical factors alone are poor correlates to explain TMD aetiology and presentation (Dworkin and LeResche, 1992; Suvinen, 1992; Suvinen and Reade, 1995; McNeill, 1997). Increasing consensus now, however, exists concerning the type of clinically relevant morphopathological and functional disorders present in patients with TMD (Dworkin and LeResche, 1992; Okeson, 1996). To summarize, scientific literature confirms at least the following general biomedical or physical diagnostic subcategories of TMD as important in the assessment and management of TMD: myofascial or muscle related TMD, articular or TMJ related TMD and/or combined muscle/TMJ TMD. There is general consensus that occlusal variables alone are not considered aetiologic factors in TMD (Pullinger et al., 1993; Okeson, 1996; Pullinger and Seligman, 2000; Seligman and Pullinger, 2000; Carlsson et al., 2003). It is interesting to note also that there is basic science evidence of cortical control of the interrelatedness of articular and muscle factors (Sessle and Hu, 1991). In general, while there is now consensus about the biomedical variables in TMD, their clinical relevance with regard to assessment and management still needs further study, especially longitudinally and in a multicentre international setting. It can be quite comfortably concluded that there is evidence that the temporomandibular region, although unique in terms of occlusal factors (teeth) and bilateral function of the joints and musculature, also shares similarities with other musculoskeletal disorders of the body. In the next section, we will review some of the earlier psychological theories and integrated understanding of biopsychosocial aetiological concepts as they relate to TMD.

6. Aetiological concepts related to psychological theories

There is currently considerable evidence that psychological and psychosocial factors are of importance in the understanding of TMD as with other chronic pain disorders (Greene et al., 1982; Fricton, 1985; Schnurr et al., 1990; Grzesiak, 1991; McCreary et al., 1991; Gamsa, 1994a,b; Greene, 1995; Suvinen and Reade, 1995; Okeson, 1996; LeResche, 1997; McNeill, 1997; Dworkin and Suvinen, 1998; Garafalo et al., 1998; Rollman and Gillespie, 2000; Sipilä et al., 2001; Jones et al., 2003), but there is less evidence that these factors are aetiologic (Beutler, 1986; Brown, 1990; Dworkin, 1994a; Macfarlane et al., 2002d; McBeth et al., 2002). The issue of whether psychological factors cause TMD or reflect the impact of TMD on the person remains, however, unknown, due largely to the absence of longitudinal incidence studies designed to test the relationship of onset

of TMD pain to onset of psychological and psychosocial factors. Nevertheless, these influences are of special importance in the assessment and successful management of individual and subjective dimensions of pain experience associated with TMD (Gale and Funch, 1984; McCreary et al., 1992; Turk et al., 1993; Suvinen and Reade, 1995; Fricton and Olsen, 1996; Krogstad et al., 1996; Suvinen et al., 1997a; Dworkin and Suvinen, 1998, Jensen et al., 2001; Dworkin et al., 2002a,c).

According to the International Association for the Study of Pain (IASP) pain is defined as 'an unpleasant sensory and emotional experience associated with actual and potential tissue damage or described in terms of such damage' (Merskey, 1986). In other words both the sensory and suffering components, including a variety of psychological influences, are important for the overall manifestation of pain perception, appraisal and behaviour. There are several comprehensive reviews available regarding psychological and psychosocial factors in TMD (Dworkin et al., 1990; Schnurr et al., 1990; Dworkin, 1991; Grzesiak, 1991; Dworkin, 1992; Glaros and Glass, 1993; Dworkin, 1994a; Suvinen and Reade, 1995; Turk et al., 1995; LeResche, 1997). In the following sections, we briefly summarize the central conceptualisations regarding psychological theories (Rugh and Solberg, 1979; Grzesiak, 1991; Suvinen and Reade, 1995). The multifactorial perspectives and the biopsychosocial model of the aetiology of pain disorders, as well as TMD are then discussed in more detail. Overall the earlier psychological conceptualizations of TMD implied that the psychological 'make-up' of individual patients was associated with symptoms of a particular organ system such as the temporomandibular region. Now many of these psychological dimensions are understood to be interrelated and form part of the integrated and multifactorial understanding of TMD. In the following, we present an overview of various psychological conceptualizations as they relate to TMD (Fig. 3). As many of the psychological factors are interrelated, there is inherently some overlap between the concepts. The main premise of these psychological concepts is then presented in the summary section.

6.1. Psychodynamic concepts

According to the classical psychoanalytic concept of Freud (1953) a disorder, such as TMD, was viewed as a 'conversion reaction', an outlet for unconscious emotional conflicts. Earlier supporters of this theory have suggested that oral problems are expressions of guilt about sexual conflicts (Moulton, 1955a,b), hysterical conversion (Engel, 1951) or a conversion reaction involving the oral cavity (Lefer, 1966). TMD pain could also be a manifestation or 'a consequence or symptomatic expression of an unconscious psychologic conflict seeking awareness (Grzesiak, 1991).

Psychoanalytic treatment was based on helping the patient to gain insight into their symptoms by focusing on possible unconscious internal emotional conflicts. Generally dynamic psychotherapy with chronic pain patients is recommended in highly selected cases, of brief duration, and with a focus on limited psychological issues not on transformation of the personality structure (Dworkin and Wilson, 1993a). The inherent problem with psychoanalytic concepts has been a difficulty in scientific testing, thus making verification difficult.

6.2. Personality concepts

The personality theories were based on the premise that individuals have consistent personality characteristics or personality traits, which predispose them to specific somatic disorders (Dunbar, 1935; Lupton, 1966). Lupton (1966) supported the concept of personality traits, i.e., that individuals vary in the ways they perceive environmental stimuli. He postulated that TMD may result from 'rigidity' in perceptual style, which was explained to contribute to a persistent state of generalized somatic tension, which may be focused in different organ systems of the body. He stated that personality trait studies of 'hypernormality' support this hypothesis.

Overall the studies in personality types or traits have proven inconclusive. A range of personality characteristics have been reported in the literature over the years, including dependent, narcissistic, obsessive, rigid, domineering, managerial, autocratic, perfectionist, hypernormal, responsible, aggressive, neurotic, emotionally unstable, insecure, hypochondriac and depressed (Rugh and Solberg, 1979). A similar diversity in personality characteristics has been reported in other musculoskeletal disorders of the body (Mendelson et al., 1956; Buck and Hobbs, 1959; Lipowski, 1968; Mongini et al., 2000). Others have disputed the concept of fixed personality characteristics in determining behaviour or that there would be a specific TMD personality (Bandura and Walters, 1963; Mischel, 1968; Kiritz and Moos, 1974; Marbach, 1992). Also personality theories have been unable to explain why some individuals with similar personality characteristics or styles do not have TMD.

6.3. Concepts regarding emotional and affective states

The most frequent affective concomitants of pain include anxiety and depression, but they may include anger, aggression, guilt and subservience (Craig, 1989). While it has been common over the past several decades to assume that depression was the major concomitant associated with TMD and other chronic pain conditions, recent evidence suggests that anxiety may be an equally potent factor (Tversky et al., 1991; Suvinen and Reade, 1995; Madland et al., 2000). Although the

specific mechanisms of action by which anxiety might modulate chronic pain, e.g., through autonomic nervous system regulated psycho-neural-humoral processes, for example, have not yet been elaborated, it is expected that anxiety, as an important affective component of TMD, will continue to receive more attention than it has in the recent past (Madland et al., 2000; Mandfredini et al., 2004; Wright et al., 2004). In summary, there is relatively strong evidence that some patients with TMD are more anxious and/or depressed compared to asymptomatic controls (for a more detailed review of motivational-affective factors in TMD and their assessment refer to, e.g., Grzesiak (1991), Suvinen and Reade (1995), Dworkin and Suvinen (1998), Madland et al. (2000), Dworkin et al. (2002b) and Turk and Melzack (2001)). In the following, we will review the earlier as well as the central conceptualizations related to affective states in TMD.

In the 1950's Moulton (1955a,b) postulated that anxiety may produce subjective symptoms of pain without actual tissue damage. She also proposed that anxiety may cause alterations in the autonomic nervous system, which may result in structural damage. It has been reported also that anxiety may provoke muscle tension or oral habits, which if prolonged, may result in muscle fatigue, tissue damage and pain. This latter view was supported widely in the past (Schwartz, 1955; Franks, 1965; Laskin, 1969; Lupton, 1969; Evaskus and Laskin, 1972; Molin et al., 1973). The more recent theoretical approaches to explain the relationship between emotions and pain include biologic, psychodynamic, cognitive, and behavioural models (Suvinen and Reade, 1995). Biologic theories refer to key neurotransmitters that have been shown to mediate neuro-anatomic pathways in control of both pain and emotion (Fields, 1988). According to the psychodynamic conceptualization the inability to modulate and express intense, acceptable feelings, e.g., anger and guilt, may underlie this relationship. In cognitive terms helplessness and lack of control and in the behavioural terms, e.g., severe reduction in activity, may explain the relationship between affective dysfunction and chronic pain. Research findings have supported a relationship between anxiety, muscular tension and TMD symptoms (Mercuri et al., 1979; Rugh and Solberg, 1979; Scott, 1980; Carlson et al., 1993; Ohrbach and McCall, 1996; Fricton, 1999).

As stated earlier, despite ample support concerning the relevance of emotional and affective factors in TMD, it is still not clear whether they are the cause or the consequence of pain (Beutler, 1986; Brown, 1990; Dworkin, 1994a; Suvinen and Reade, 1995; Dworkin and Suvinen, 1998). There is, however, a consensus that affective factors are important in clinical assessment and management of TMD as well as in determining treatment outcome (Fricton and Olsen, 1996; Okeson, 1996; Suvinen et al., 1997a; Dworkin and Suvinen, 1998).

6.4. Cognitive concepts

Early supporters of pain having a cognitive aspect suggested that psychosomatic disorders were the result of 'attitudes', i.e., what the individuals felt was happening to them and what they wanted to do about it (Grace and Graham, 1952). Each attitude was explained to be connected to a specific physiological reaction, which if prolonged led to pain and organic damage.

More recently, the 'evaluative components of pain' have been widely studied in terms of cognitions and cognitive coping. Coping includes appraisal and efforts to alleviate symptoms. For a more detailed review of various studies regarding cognitive factors in pain and TMD refer to, e.g., Grzesiak (1991), Jensen et al. (1991), Dworkin and Wilson (1993b), Suvinen and Reade (1995); Turk and Melzack (2001) and Turner et al. (2001). Studies have shown that at least two aspects of cognitive dimensions emerge that may be relevant therapeutically and in terms of understanding the variable nature of pain experience in TMD. These include the perceived control or adjustment over pain and the type of cognitive coping strategies, especially the use of maladaptive coping strategies, e.g., catastrophizing, to control pain (Rudy et al., 1989; Jensen et al., 1991; Suvinen and Reade, 1995; Suvinen et al., 1997a; Jensen et al., 2001; Turk and Melzack, 2001; Jones et al., 2003). It has been proposed from the results of some studies that 'adaptive' coping is an important variable in a subgroup of patients with TMD (Rudy et al., 1989; Suvinen et al., 1997b, 2005; Rudy et al., 1995; Jensen et al., 2001).

6.5. Behavioural concepts

Acquisition of new behaviours, beliefs and attitudes in humans are based on learning principles. Both normal and abnormal behaviour patterns can be seen as ways of adaptation to external and internal environment. Fordyce (1974) has explained that non-organic, chronic pain symptoms may be a result of external contingencies, i.e., desirable consequences to report pain in terms of attention, escape from stressful work, or monetary compensation. Learning principles may be used to explain oral habits through modelling or through non-verbal communication in case of strong facial expression and clenching of teeth (LeResche, 1997).

Important dimensions regarding behavioural aspects in TMD pain include 'illness behaviour' or 'sick role', i.e., the ways in which given symptoms may be differentially perceived, evaluated and acted upon by different kinds of persons' (Mechanic, 1962) or 'the ways in which individuals react to aspects of their own functioning, which they evaluate in terms of health and illness' (Pilowsky, 1978). The definition of illness behaviour encompasses not only behaviour, but also thoughts (cognitive aspects) and emotions (affective aspects) (Dworkin,

1991; Suvinen and Reade, 1995). The behavioural aspects can be further influenced by ethnocultural factors, social factors and environment (modelling) (Suvinen and Reade, 1995). Of importance is the recognition of 'somatization' in the assessment and management of TMD, i.e., 'preoccupation with physical symptoms disproportionate to actual physical disturbance' (Dworkin and LeResche, 1992; Macfarlane et al., 2002b). It is important to distinguish somatization as a psychiatric disorder from somatization as 'a personal functioning characterized by the tendency to experience and/or report numerous physical symptoms' (Dworkin, 1994b; Wilson et al., 1994). Somatization has been linked to a range of behaviours including frequent use of health care services and seeking a biomedical explanation and treatment for physical symptoms. A more detailed description of the important aspects related to illness behaviour and somatization in TMD have been presented previously in several publications by Dworkin (Dworkin, 1991, 1992, 1993, 1994b, 1995a,b, 1996; Dworkin and LeResche, 1992; Dworkin and Wilson, 1993a; Dworkin et al., 1994, 2003; Yap et al., 2003). There is now support for the proposition that cognitive-behavioural forms of therapy are an integral part of any 'state of the art' management program of TMD and several studies and reviews are available in support of these therapies (Turk and Rudy, 1990; Flor and Birbaumer, 1993; Oakley et al., 1993; Rudy et al., 1995; Suvinen and Reade, 1995; Dworkin, 1997; Dworkin and Suvinen, 1998; Dworkin et al., 2002a,b).

6.6. Summary of psychological aetiological concepts

Psychological concepts and theories have validated the importance of intra- and inter-psychic factors and their inter-relatedness in TMD disorders and illness impact. As a detailed presentation of all of these factors and past literature is beyond the scope of this review, we have referred to previous studies. To summarize, scientific literature confirms at least the following psychological and psychosocial dimensions as important in the assessment and management of TMD: affective disturbance (depression and/or anxiety), somatization and psychosocial dysfunction. Also poor correspondence between objective signs (peripheral dysfunctional aspects) and subjective symptoms (intrinsic and extrinsic central aspects of pain perception), maladaptive coping resources and excessive use of the health care system are considered important. There is now general agreement that all patients with TMD should be screened for psychological and psychosocial dysfunction (Turner and Dworkin, 2004), e.g., by utilizing such methods as the RDC/TMD to tailor TMD treatment regarding patient's psychosocial adaptation (Dworkin et al., 2002a,c) and regarding the need to use multidisciplinary approaches.

There is need for further research using prospective designs to clarify the possible roles of, e.g., stress, depression and somatic distress in the onset and as risk factors for TMD (Von Korff et al., 1993; LeResche, 1997; Huang et al., 2002; John et al., 2003; Rammelsbeg et al., 2003). Studies in psychoneuroimmunology have implied the importance of emotional stress and its effect on neuroendocrine function and TMD (Marbach et al., 1990b; Maier et al., 1994; Auvenshine, 1997; Biondi and Picardi, 1999). All of these factors are commonly accepted to be of relevance in the presentation, management and resolution of TMD. It is also generally accepted that individual variation exists and that instead of looking for one common psychological denominator, a better way, based on recent evidence, is to assess individual status and possibly subtypes of psychosocial functioning and their correlation with biomedical variables.

7. Towards an integrated biopsychosocial concept of the aetiology of TMD based on an interplay of peripheral and central factors

7.1. Earlier integrated multifactorial aetiological concepts of TMD

The review of biomedical and psychological and psychosocial concepts of TMD provide no conclusive evidence to support a single cause for TMD and consequently in the recent decade multifactorial concepts of the aetiology of TMD have been emphasized. Bell (1990) presented a view that multiple factors, from the constitutional make-up of the individual, and from psychological differences to structural factors can influence the shift from physiological response to pathologic response. He stated that seldom if ever 'should one look for a single isolated aetiologic agent. In a general way one should think in terms of predisposing conditions, activating factors and perpetuating influences.' According to Bell (1990) multiple aetiologies could affect different parts of the masticatory system. As an example, muscle disorders could result from altered neural input, delayed muscle soreness, deep pain input, inflammatory conditions, emotional tension, head and body position and some medications; whilst TMJ disorders could result from traumatic events, functional overloading and systemic conditions or structural aberrations.

Parker (1990) presented a dynamic model of the aetiology of TMD. He proposed that interactions between adaptive responses (orthofunction) and destructive processes (pathofunction) operate dynamically. This concept of orthofunction was an extension of the concept presented earlier by Krough-Poulsen (1969). According to this model several factors, including trauma, health, nutrition, structure, coping and gender could affect an

individual's adaptive potential on the structural, functional and psychosocial basis thus tilting the balance towards pathofunction. Apart from increased and decreased adaptive responses, disorders of the masticatory system could be explained by examining those factors that increased hyperactivity thus directly leading to pathofunction. Such factors, according to this theory, were explained to include adverse posture, occlusion, pain, depression, sleep disorders, and life stressors. However, as shown by the extensive set of studies by Pullinger and Seligman (Seligman and Pullinger, 1991; Pullinger et al., 1993; Pullinger and Seligman, 2000; Seligman and Pullinger, 2000), neither occlusion nor posture have been shown to have strong supporting data for the causation of TMD. This model allowed the assessment of multiple aetiological factors, which could be used in decision making for treatment, however, no clear causal explanation for the various factors in TMD was given.

As pain is a cardinal feature that makes patients with TMD seek treatment, the evolution of pain theories has influenced thinking concerning the aetiology of TMD (Melzack and Wall, 1965; Melzack and Casey, 1968; Rugh, 1987; Suvinen and Reade, 1995). The multifactorial concept of aetiology in relation to pain perception was summarized by Rugh (1987), when he described the multidimensional model of pain applied to TMD. This seven-component model was based on the 'gate-control theory of pain' by Melzack and Casey (1968) and illustrated the complex phenomena of pain experience. At the basic neurophysiological level the receptive system detects, modulates and transmits the pain stimulus to the perceptive system, which recognizes pain depending on the pain threshold and tolerance and thus leads to pain experience. This basic pathway is, however, influenced and interacted upon by a complex interplay of emotional affective system, cognitions and learning principles, including pain behaviour and societal and environmental factors. This theory provided an insight into the understanding and measuring of the complex nature of individual pain experience. Melzack (2001) has expanded the gate-control theory to reflect recent advances in methods to observe brain functioning in awake patients experiencing chronic pain. His neuromatrix theory, while not fully validated, nevertheless seems of tremendous heuristic value, because of its attempt to integrate how peripheral events are reflected, integrated and then acted upon in a dynamic fashion as the ongoing resolution of peripheral pathology with central information processing that incorporates into perception, appraisal and response to pain, such higher order functions as attention, memory, emotions and organized behavioural response patterns (Melzack, 1999b, 2001).

For a more recent and detailed presentation and discussion on the nature of TMD pain, theories of pain perception and/or the multidimensional pain assessment and management refer to, e.g., previous reviews by Dahlström (1993), Rugh et al. (1993), Suvinen and Reade (1995), Dworkin and Suvinen (1998), Dworkin (2001) and Sessle and Dubner (2001). The appreciation and understanding of the multidimensional aspects of pain perception has led to an improved conceptualization, assessment and treatment of patients with TMD, often incorporating a variety of disciplines; medicine, dentistry, physiotherapy, psychology in multidisciplinary chronic pain management teams.

7.2. Summary of earlier multifactorial aetiological concepts

The assessment of presumptive aetiological factors is of importance for the development of adequate and effective treatment modalities and for the evaluation of risk factors. They are also important for the formation of preventive guidelines and methods. Several multifactorial aetiological theories have been proposed for chronic musculoskeletal pain disorders in general as well as for those affecting the temporomandibular region. In many of the earlier theories a multitude of factors have been proposed as possible causative factors, however it is not clear or substantiated whether the proposed factors are aetiologic or contributing or as Bell (1990) proposed predisposing, activating or perpetuating in nature. Since Melzack and Casey (1968) and Engel (1977) first called for the need to broaden the biomedical (physical disorder) model, the biopsychosocial (physical illness impact) model has become central to an understanding of chronic pain, including TMD pain (Dworkin et al., 1992). In the following, we will review concepts and studies related to the biopsychosocial model of TMD.

8. Biopsychosocial model of the aetiology of TMD

The biopsychosocial model for understanding disease and illness grew from the more narrowly focused biomedical model, which has characterized much of medical diagnosis, assessment and management. The biomedical model emphasized the greater importance of assessing physical disorder factors, while the biopsychosocial model attempts to integrate both the physical disorder factors, i.e., *biological factors* as well as the illness impact factors, i.e., *psychological and social factors* (Fig. 3).

More specifically, according to the biomedical model (Engel, 1977), individual complaints and disorders are understood as a result of underlying pathophysiology or pathobiology as the presumptive and sole aetiologic factor for the biological disorder present, with treatment aimed at curing the physical pathology and disorder/s present and correcting the morphopathological and functional processes. The limitations of this model in-

clude inadequacies in understanding the poor relationship between abnormal or even normal biological variation and the total clinical presentation of the patient, including reliance on biological variation, problems with current, exclusively biologically based, diagnostic systems, while not considering, for example, the potent role in, and complications created by differing patient–doctor explanatory models (Massoth, 1992; Massoth et al., 1994). Engel (1977) identified the major limitations of the biomedical model to be its failure to identify psychological and psychosocial variables in health and disease and their dynamic interaction with pathophysiological processes. The consequence of this approach has been that it yielded treatment approaches that focused on permanent cures – an unrealistic expectation for many chronic pain conditions, instead of encouraging a rehabilitation model for managing chronic pain and chronic illness. In other words biomedical orientation is a cure instead of a rehabilitation model, while more generally the biopsychosocial model implicitly encourages a rehabilitation model for conceptualizing management of chronic illness. Engel (1977) proposed that a bio-psycho-social model, which allowed for the integrated assessment between biological, psychological, and social factors, was needed to conceptualize and treat not just the 'disorder' domain, but also the 'illness', which per force, includes illness impact on the patient, to reduce suffering and dependence on health care.

The biopsychosocial model is closely related to the multidimensional model of pain in that it recognizes biological disorder in the context of illness experience, i.e., personal, interpersonal and cultural reactions through perception, labelling, explanation and appraisal and valuation and behaviour of the disease experience.

In 1992, Dworkin and his colleagues reviewed epidemiologic and relevant clinical studies in TMD and presented a comprehensive biopsychosocial model of chronic pain development and experience that was especially applicable to TMD research and an understanding of TMD pain. This model followed the influence of Melzack and Wall (1965), Fordyce (1976), Melzack and Casey (1968) and Loeser (1980) and others and helped explain the variability in the individual expression of subjective pain experience and overt pain behaviours. It integrated dynamic and multilevel (physiologic, psychologic and social) factors at different stages in the development of pain and pain dysfunction thus reflecting for the first time comprehensive biopsychosocial perspective (multidimensional aspects) of TMD. More specifically, this model showed the dynamic nature of intrinsic intrapersonal factors (such as nociception, pain perception, pain appraisal) and extrinsic interpersonal factors (behaviour responses to pain, social roles for the person in pain within the context of the family, the health care delivery system, the workplace, and the social welfare system) in chronic pain, including TMD. The model showed how these factors could be intensified or minimized and how augmentation of pain perception, appraisal and pain behaviours can lead to chronic TMD pain dysfunction. By including the construct of time (temporal aspects) this model allowed for an understanding of the order, timing and course of chronic pain and pain dysfunction and gave insights to causal mechanisms. For the complete presentation of this model, considered fundamental in the way the biopsychosocial conceptualization was first introduced with respect to pain in this field, refer to the original publication and illustrations (Dworkin et al., 1992).

Following the presentation of this model Dworkin and his colleagues have in the last decade provided several reviews and studies in support of this model and approach to understanding, assessment and management of TMD pain. In the following, a brief summary is provided of this substantial contribution to the field of TMD research and an understanding of TMD. For a more detailed review refer to the original papers. Especially important has been the development of the RDC/TMD for the systematic assessment of TMD in different centers internationally, which will be presented together with other related studies in the following paragraph (Dworkin and LeResche, 1992).

There is now considerable evidence that TMD can be viewed primarily as a chronic pain condition that shares many features in common with other common chronic pain conditions and hence should be studied and managed from the biopsychosocial perspective (Dworkin and Massoth, 1994; Dworkin, 1995b). Based on data from longitudinal epidemiologic and intervention studies Dworkin (1995b) summarized general characteristics of patients with TMD as follows: TMD is a chronic, recurrent and to some degree self-limiting condition, associated with 'appreciably distressful, but typically non-specific clinical features'. Even though studies support the proposition that the majority of patients with TMD cope adequately with their symptoms there is also evidence of a psychologically dysfunctional segment of patients (Dworkin, 1994b, 1995b; Suvinen et al., 1997a,b; Dworkin and Suvinen, 1998). This patient group appears to be unable to cope, and shows higher rates of depression, somatization, and health care utilization (Dworkin, 1994a, 1995b; Yap et al., 2002b). Several reviews and studies have provided insight and guidelines for assessment and management of these more complex patients with TMD (Dworkin, 1992, 1995a,b; Dworkin and LeResche, 1992; Suvinen and Reade, 1995; Dworkin and Suvinen, 1998; Dworkin and Ohrbach, 2001; Dworkin et al., 2002a,b,c).

Several studies support the use of the biopsychosocial model in the understanding and assessment of TMD as a prevalent chronic pain condition. One of the most widely studied instruments in this orientation is the

RDC/TMD (Dworkin and LeResche, 1992), which conceptualizes TMD according to a two-axis system, one for the physical disorder factors (Axis I) and the other for the psychosocial illness impact factors (Axis II). The RDC/TMD has been accepted in the scientific community worldwide, including the establishment of an international consortium of RDC/TMD-based researchers, to ensure the use of uniform, standardized clinical research data gathering methods and non-English translated versions of the RDC/TMD with consistent back-translation and consensual validation processes. The RDC/TMD has been used as a classification and diagnostic system in several study populations, including epidemiological studies (Dworkin et al., 1990; Ohrbach and Dworkin, 1998; List et al., 1999; Rammelsbeg et al., 2003), clinical studies (List and Dworkin, 1996; Wahlund et al., 1998; Marcusson et al., 2001; Yap et al., 2002a,b, 2003; Manfredini et al., 2003; Rantala et al., 2003) and intervention studies (Dworkin et al., 2002a,b; Wahlund et al., 2003).

Others have examined various integrated psychological or psychophysiological subtypes of TMD and reported that patients can be subdivided into groups based on ternary models, i.e., interplay between physiological and/or psychosocial variables. Rudy et al. (1989) used k-means cluster analysis to derive three subtypes of TMD based on the West Haven Multidimensional Pain Inventory (MPI, Kerns et al., 1985) of psychosocial dysfunction and proposed a psychological taxonomy with TMD including the following subtypes: adaptive copers, interpersonally distressed and dysfunctional. The robustness of the MPI has since been shown in other studies (Turk and Rudy, 1990; Rudy et al., 1995; Dahlstrom et al., 1997). Butterworth and Deardorff (1987) had earlier presented a similar taxonomy of TMD patients using the craniomandibular questionnaire, which included the SCL-90R of psychological dysfunction and proposed the following TMD subtypes: psychologically normal, moderately distressed, severely distressed. Suvinen et al. (1997b, 2005) used the Temporomandibular Pain Dysfunction Questionnaire, which includes measures of both physical disorder and illness impact factors, such as coping style, affective disturbance, disease conviction as well as illness behaviour and life impact factor and proposed the following subtypes: simple, intermediate and complex TMD. All of these models emphasize the importance of Axis II or psychosocial variables and future studies will show whether it is more effective to assess the relationship between Axis I and II individually instead of assessing ternary group differences. Currently only the RDC/TMD has established reliability for diagnostic assessment (Dworkin and Ohrbach, 2001) and has been validated with regard to psychosocial assessment (Dworkin et al., 2002b). The RDC/TMD is used internationally for the systematic assessment of TMD and the Bulletin of the American Pain Society reproduced the RDC/TMD protocol in its entirety, citing it as a model system applicable to the assessment of all chronic pain conditions (Garofalo and Wesley, 1997).

9. The adaptation of biopsychosocial model and information processing theory to the management of temporomandibular pain and dysfunction

The following conceptual model is presented as an example of how the biopsychosocial approach may be conceptualized in terms of the cardinal features of TMD. This hypothetical therapeutic model is intended as an overview and summary of the biopsychosocial orientation. The therapeutic regimens are not intended to represent an in-depth review and evaluation of past studies regarding the management of TMD, as it is beyond the scope and main emphasis of this review, but rather they are intended to be heuristic and possibly as an aid to seeking directions for future research.

According to the biopsychosocial conceptualization presented in Fig. 4 it is hypothesized that some form of 'peripheral event' referred to here simply as an 'injury' to the temporomandibular region disrupts part of the masticatory system in a susceptible individual. The acceptance that some form of 'injury' or trauma could be a common 'peripheral' denominator in the aetiology of TMD is supported by some authors (Zarb and Speck, 1979; Reade, 1984; Pullinger and Seligman, 1991). Pullinger and Seligman (1991) reported that as many as 44–79% of patients with TMD could recollect a traumatic event, but it is also recognized that not all patients with TMD recall a 'traumatic' incident. Other 'injuries' could include, for example, various forms of macro- or micro-trauma, i.e., motor vehicle accident, overload or repetitive strain injury or muscle fatigue. It is further hypothesized that both structural or morphopathological and/or functional factors as well as factors involved in pain perception underlie the process for which patients with TMD seek treatment. It is well-documented that a significant proportion of patients with TMD has suffered fluctuating and/or chronic symptoms (Dworkin, 1995b, 1999; Ohrbach and Dworkin, 1998; Magnusson et al., 2000; Egermark et al., 2001). Several factors, such as adaptive processes, coping style and illness behaviour may influence the degree of disability following the 'peripheral event/s' or 'injury/injuries'. All of these dimensions should be carefully assessed in the initial history taking and examinations of patients presenting for treatment of TMD (Suvinen and Reade, 1995; Dworkin and Suvinen, 1998; Dworkin and Ohrbach, 2001). As there is now support that TMD are to be conceptualized as musculoskeletal biopsychosocial disorder with considerable chronicity, careful initial history taking is a key to successful assessment and management.

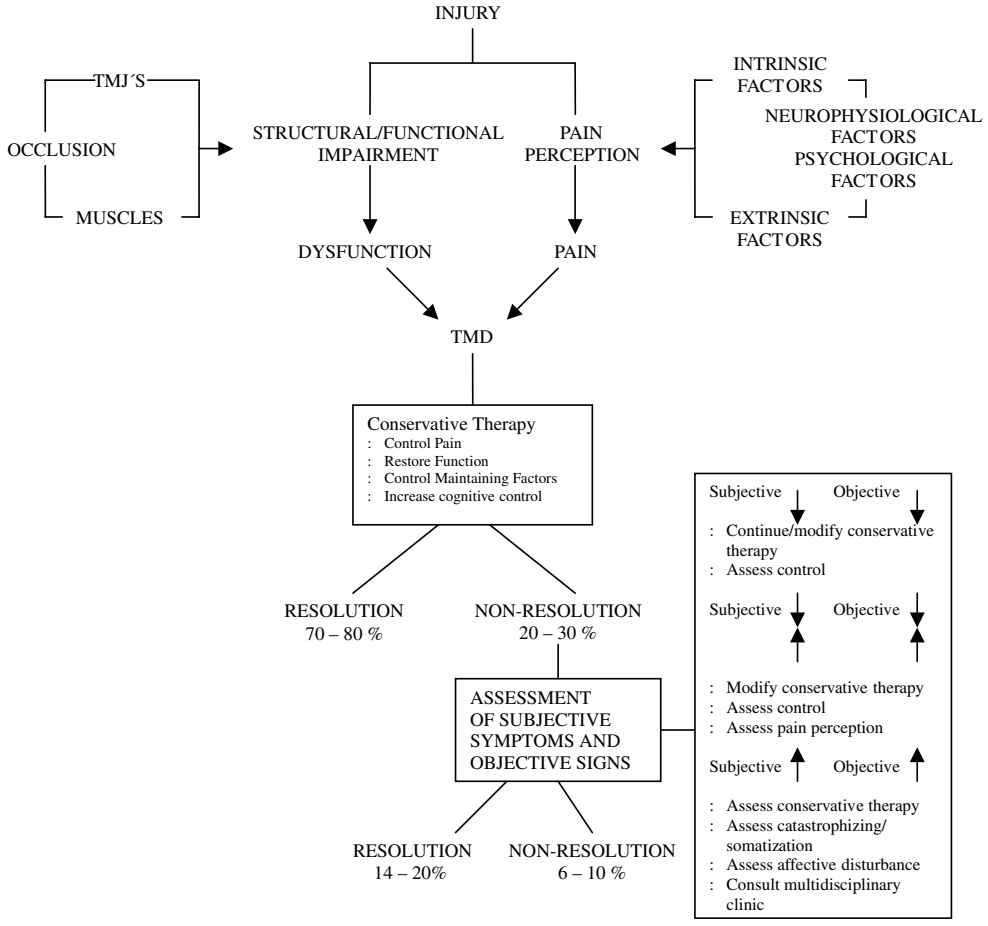

Fig. 4. Conceptual therapeutic model of psychophysiological aspects of temporomandibular pain and dysfunction.

It should parallel the medical model and include the chief complaint/s, associated symptoms/signs, history of present illness, medical history, dental history and personal history (Okeson, 1996; Greene and Laskin, 2000). All precipitating, initiating, alleviating, aggravating, contributing and maintaining factors should be carefully assessed, including psychosocial status and illness impact factors, as well as any previous treatment and their outcome. There are now general guidelines available for the screening purposes, for comprehensive clinical assessment and for research purposes, as well as for psychosocial illness impact assessment (Dworkin and LeResche, 1992; Okeson, 1996, 1997; NIH, 1997).

It is generally accepted that conservative reversible therapies are the treatment of choice in the majority of patients with TMD, with a reported long-term efficacy in approximately 75–80% of cases (Greene and Laskin, 1983; Okeson, 1996; McNeill, 1997). Irreversible forms of therapy that permanently alter the jaw relationships or orthodontic and/or prosthetic rehabilitation are not recommended and especially should not be given until conservative reversible forms of therapy have been completed to alleviate painful symptoms (Okeson, 1996; De Boever et al., 2000a,b), although the provision of adequate dentures may in some cases be indicated as part of TMD treatment. Surgical interventions should be targeted for highly selected cases only, as non-surgical management has proven efficacy in most patients with TMD (Okeson, 1996; NIH, 1997; Fricton et al., 2002). It is noteworthy that 'even in the absence of perfect understanding of aetiology, we still can provide good conservative care and should avoid aggressive and irreversible treatments, especially when they are based on flawed concepts of aetiology' (Greene, 2001).

The choice and the effectiveness of different therapies in clinical practice should be based on evidence-based care (NIH, 1997; Raphael and Marbach, 1997). Conservative treatments generally incorporate both biomedical as well biobehavioural approaches (Dworkin, 1997; Dworkin and Suvinen, 1998; Sherman and Turk, 2001; Dworkin et al., 2002a,b,c). The conservative reversible forms of therapy are generally aimed at reducing pain, restoring and rehabilitating normal function and decreasing aggravating and/or contributing factors. Such methods as education and counseling and physical and appropriate interocclusal appliance therapies have been supported in the literature (NIH, 1997; Dworkin and Suvinen, 1998). Education and counseling should

be aimed at explanation of the cause, description of the type of disorder and its anticipated treatment and progression and reassurance about its benign nature (Suvinen, 1992; Dworkin and Massoth, 1994; Dworkin, 1997; Dworkin et al., 2002a). Emphasis should be given to the success of conservative treatments, and physical and interocclusal appliance therapies should be aimed at providing the patient with advice and self-management skills to avoid further 'injury', to restore function and to allow adaptive processes to remodel the morphopathological or functional 'injury'. Interestingly, in two recent randomized clinical trials it was shown that a short-term cognitive-behavioural self-care management program together with usual TMD treatment significantly reduced pain intensity, even in patients with significant pain-related dysfunction (Dworkin et al., 2002a,b,c). The relationship of physical disorder variables as well as the psychological status and psychosocial dysfunction should be assessed in an integrated fashion during initial assessment and examination and during the first phase of conservative therapy generally reported to last on average from four to six months (Suvinen, 1992; NIH, 1997; Suvinen et al., 1997a).

During the observation and review period of the initial response to conservative methods there is a need to assess both *objective* and *subjective* variables. The objective variables include changes in functional improvement (e.g., co-ordination and stabilization of masticatory function). The subjective variables include changes in psychological (affective state and coping style), as well as psychosocial illness impact factors and effect of TMD on patients' lives (time lost from work, inactivity, sleep disturbance, high use of health care, generalized somatic distress). The balance between changes in both of these domains (physical disorder versus psychosocial illness impact domain) could be used as a guide to adjust therapeutic measures, e.g., as shown in Fig. 4. For example, if patients show a decrease in suffering, but little functional improvement the degree of functional and/or morphopathological 'injury' should be a guide to using additional physical conservative treatment methods. In the principal author's experience in the treatment of TMD the use of therapies such as physiotherapy, various designs of interocclusal appliances or bio-behavioural management strategies to reduce overload through parafunctional habits, could be some of the measures of choice. On the other hand, if patients show functional improvement, but continue to complain of severe pain, factors in pain perception should be addressed, such as perceived control (maladaptive coping such as catastrophizing and lack of symptom control), affective disturbance (depression, anxiety) or environmental and societal influences (ability to work, life satisfaction, inactivity, health care use) as well as patient compliance to avoid chronicity as a results of failed conservative management. In a small proportion of patients who can be considered 'complex' the interplay between psychological and psychosocial illness impact factors is best managed by an interdisciplinary team, incorporating chronic pain management clinicians from dentistry, physiotherapy, psychology, social work and psychiatry, and in some cases various medical specialties, such as neurology.

To summarize, therapeutically it is important to avoid excessive or irreversible forms of therapies and to avoid chronicity as a result of inappropriate or failed therapies or as a consequence of inappropriate assessment of overall biopsychosocial dimensions of TMD. It is noteworthy that if patients fail to respond appropriately or in a predicted pattern, it does not necessarily mean that the symptoms are 'in the patients' mind'. Generally combined therapies are best suited to assess aspects in the domains of pain perception as well as jaw dysfunction (Sherman and Turk, 2001). There may be a need to develop more targeted and tested therapies regarding the dysfunctional domain (Stohler, 1999; Sessle, 2000; Greene, 2001; Svensson and Graven-Nielsen, 2001; Tenenbaum et al., 2001).

Finally, we can now hypothesize on the application of the integrated model to the typical patient suffering from chronic TMD. If we accept the general information processing theory the following scenario could be used to explain TMD symptom formation and processing. This model could then be applied to the rehabilitation of the patient with TMD.

In most cases it can be argued that a 'peripheral event' precipitates the central phenomenon. This could be in the form of macro- or micro-trauma, i.e., motor vehicle accident, overload or repetitive strain injury or muscle fatigue. When this initial peripheral event fails to heal, because of a variety of possible underlying factors such as the general vulnerability of the patient, which could be based on genetic predisposition, hormonal factors, behavioural habits or failed therapy, a prolonged phase, i.e., a chronic condition, ensues. Based on the neuromatrix and information processing theory the central events that follow will lead to the spread of pain in the neuromatrix so conditioning the patient to negative event processing (Jerome, 1993; Melzack, 2001). The general feature that sets in motion in response to the peripheral signal is negative, i.e., 'I don't like this sensation'. The predictable pattern is a change in physiology, negative affect, negative cognitions and general behaviours associated with pain behaviours. It is noteworthy that central neural networks are capable of generating pain experiences independently of peripheral sensory inputs (Melzack, 1999a,b; Melzack et al., 2001). This general disregulation can present as physical symptoms and/or negative central factors such as depression, social isolation, loss of interest, inactivity, excessive health care use, chronicity and somatic distress with poor response to biomedical treatment approaches.

By driving this model backwards a basic rehabilitation model can be produced. The main emphasis of this model is to firstly 'do no harm' followed by approaches that 'facilitate healing/rehabilitation', including the restoration of the person by bio-behavioural methods that can increase optimism, positive emotions and increased activity. This attitudinal state can change physiology, break habits, restore physical health, break conditioned, learned response, in general alter the pain state and the physical state, thoughts, emotions and behaviour thus stopping the possible spiraling into the victim state of long term chronicity by altering susceptibility and specificity.

10. Conclusions

In conclusion, this review was aimed to examine biomedical and biopsychosocial aetiological concepts as they apply to TMD. Specific evidence was presented in support of each theory and its conceptualization as well as a hypothetical biopsychosocial therapeutic model as an example of how this evidence may be integrated in physical disorder as well as psychosocial illness impact domains.

The theory of an information processing system, together with advances in basic and clinical sciences, especially psychoneuroimmunology and cognitive neuroscience has helped with our understanding of the complexity of chronic pain. The scientific integration between biological and psychological processes together with evidence based treatments and assessment of their effectiveness by meta-analyses and randomized placebo-controlled clinical trials will allow the clarification of empirical and scientific paradigms, current practices, and the future of pain research. Clinical applications of a biopsychosocial model for understanding and managing chronic pain disorders, in particular TMD, will be assisted by this approach.

The biopsychosocial model outlined in this review is considered to provide an inclusive approach to the management of TMD based on information processing and an integration between physical and psychological processes. There is a clear need for continuing assessment to provide for evidence-based management of chronic pain disorders and in particular TMD.

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