Department of Stomatognathic Physiology and Prosthetic Dentistry
Institute of Dentistry
Faculty of Medicine
University of Helsinki
Finland

SELF-REPORTED BRUXISM

Associated factors among media personnel with or without irregular shift work

Kristiina Ahlberg

Academic dissertation

To be publicly discussed with the permission of the Faculty of Medicine of the University of Helsinki, in the main auditorium of the Institute of Dentistry, Mannerheimintie 172, Helsinki, on August 22, 2008, at 12 noon.

Helsinki 2008

Supervised by:

Professor Mauno Könönen
Department of Stomatognathic Physiology and Prosthetic Dentistry
Institute of Dentistry
University of Helsinki
Helsinki, Finland, and
Helsinki University Central Hospital
Department of Oral and Maxillofacial Diseases
Helsinki, Finland

Docent Jari Ahlberg
Department of Stomatognathic Physiology and Prosthetic Dentistry
Institute of Dentistry
University of Helsinki
Helsinki, Finland

Reviewed by:

Professor Frank Lobbezoo
Department of Oral Function
Academic Centre for Dentistry Amsterdam (ACTA)
Amsterdam, The Netherlands

Professor Pentti Kemppainen
Department of Stomatognathic Physiology
Institute of Dentistry
University of Turku
Turku, Finland

Opponent:

Professor Aune Raustia
Department of Prosthetic Dentistry and Stomatognathic Physiology
Institute of Dentistry
University of Oulu
Oulu, Finland

BRUXISM

One word – a thousand stories

TABLE OF CONTENTS

ABBREVIATIONS

DIS	difficulties initiating sleep
DS	disrupted sleep
ECG	electrocardiogram
EEG	electroencephalogram
EMA	early (morning) awakening
EMG	electromyography
EOG	electro-oculogram
HR	heart rate
ICSD	International Classification of Sleep Disorders
N.A.	not applicable
NREM	non-rapid eye movement (sleep)
NRS	non-restorative sleep
PSG	polysomnography
RDC/TMD	Research Diagnostic Criteria for Temporomandibular Disorders
REM	rapid eye movement (sleep)
RLS	restless legs syndrome
RMMA	rhythmic masticatory muscle activity
SCL-90-R	Symptom Checklist-90-Revised
SLD	sleep deprivation
TMD	temporomandibular disorders
TMJ	temporomandibular joint

ABSTRACT

The present study was performed on media personnel who could be considered to be under sustained pressure at work due to intense on-going technological, organizational and economic changes. The study formed part of a comprehensive investigation of shift work and its sleep/awake consequences.

The general aim was to examine the relationships of self-reported bruxism and sleep quality among employees with or without irregular shift work. The study also focused on the possible associations of bruxism and orofacial pain. Some psychological, neurological and physiological factors known to be detrimental to sleep were also studied.

A questionnaire with several standard questions was mailed to all employees of the Finnish Broadcasting Company with irregular shift work (n=750; 57.0 % men) and to an equal number of randomly selected controls in the same company with regular eight-hour daytime work (42.4 % men). The mean age of invited subjects was 43.0 (SD 10.4) years in irregular shift work and 44.8 (SD 10.2) years in day work. The work duties of the present media personnel included journalism, broadcasting, programme production, technical support and administration. The questionnaire covered perceived bruxism (assessed with a five-point scale) and, among others, the following: demographic items, employment details, general health experience, physical status, pain symptoms, psychosomatic symptoms, psychosocial status, stress experience, work satisfaction and performance, perceptions of sleep and its awake consequences. The overall response rate was 58.3% (53.7% men). The response rate in the irregular shift work group was 82.3% (56.6% men) and in the regular daytime work group 34.3% (46.7% men). The invited subjects and respondents in both shift work and day work groups were similar as regards gender and age (NS).

Frequent self-reported bruxism was found among 10.6 % of subjects overall. The bruxism scores were evenly distributed in the irregular shift work and regular day work groups (NS). Similarly, a total of 43.6 % reported disrupted sleep and 36.2 % perceived their as sleep non-restorative. Current orofacial pain was found overall in 19.6 % of the study population. Among those reporting current pain 88.3 % had experienced it for over six months.

According to the multivariate analyses, self-reported bruxism and dissatisfaction with current work shift schedule were significantly associated with most studied sleep variables. More frequent bruxism (p<0.01) and more severe stress (p<0.001) tended to occur more often among those subjects dissatisfied with their work shift schedule. It was found that dissatisfaction with one's work shift schedule and not merely irregular shift work may aggravate stress and bruxism. In addition, frequent self-reported bruxism was associated with increased numbers of health care visits. The results also revealed significant associations between self-reports of bruxism and anxiety, and bruxism and orofacial pain experience.

Based on the multivariate analyses, it can be concluded that disrupted sleep and bruxism may be concomitantly involved in the development of orofacial pain. It may also be possible that self-reported bruxism indicates sleep problems and their adherent awake consequences in non-patient populations. It was suggested that subjectively conceptualized awareness of bruxism may be linked to stress-related states and behavior which could be useful knowledge for health care professionals.

1. LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original publications, referred to in the text by their Roman numerals. Some additional data are also presented.

I Ahlberg K, Ahlberg J, Könönen M, Partinen M, Lindholm H, Savolainen A. Reported bruxism and stress experience in media personnel with or without irregular shift work. Acta Odontol Scand 2003; 61: 315-18.

II Ahlberg K, Ahlberg J, Könönen M, Alakuijala A, Partinen M, Savolainen A. Perceived orofacial pain and its associations with reported bruxism and insomnia symptoms in media personnel with or without irregular shift work. Acta Odontol Scand 2005; 63: 213-17.

III Ahlberg K, Jahkola A, Savolainen A, Könönen M, Partinen M, Hublin C, Sinisalo J, Lindholm H, Sarna S, Ahlberg J. Associations of self-reported bruxism with insomnia and insufficient sleep in subjects with or without irregular shift work. Head Face Med 2008; 4: 4.

IV Ahlberg K, Ahlberg J, Könönen M, Partinen M, Hublin C, Savolainen A. Reported bruxism and restless legs syndrome in media personnel with or without irregular shift work. Acta Odontol Scand 2005; 63: 94-8.

Original publications are reprinted with the permission of the publisher.

2. INTRODUCTION

The phenomenon of bruxism (grinding or clenching of teeth during sleep or awake) affects millions of people throughout the world. It has been thought that bruxism may be genetic in origin, affected psychosocially or pathophysiologically, as well as caused or perpetuated by occlusal discrepancies. At present, however, bruxism tends to be considered as centrally regulated, with peripheral factors playing only a minor role in its etiology. Sleep bruxism may occur in all sleep stages but is most often detected in non-REM sleep one and two, and towards arousal. Evidence also exists that sleep bruxism events appear concomitantly with transient micro arousals and could thus be an adjunct to disturbed sleep. On the other hand, some studies based on polysomnographic recordings suggest that sleep structure and sleep efficiency are normal in sleep bruxers. Nevertheless, according to current knowledge sleep and awake bruxism differ substantially from each other, and both physiology and pathology of bruxism remain unclear.

Temporomandibular disorders (TMD) are signs and symptoms in temporomandibular joints or masticatory muscles, or both. Of these, the most presented symptom among populations is facial muscle pain. TMD may remain unrecognized in health care despite several consultations and those suffering from TMD may even undergo futile examinations and treatments. Bruxism has been considered as an underlying factor for TMD, but these associations are not fully accepted.

It is well known that irregular shift work may cause insomnia, which when prolonged, leads to fatigue and decreased work performance or hazards. Awake consequences adherent to poor sleep quality may impose a considerable burden on health care utilization. Sleep problems and stress experience also seem to go hand in hand. However, those who for any work or life reason become stressed-out report less ability to control their work and social relations, and may feel their work quantitatively overloaded and unevenly distributed.

Currently in the Finnish media industry the production and delivery of radio and TV programmes are in transition from analogue to digital techniques. Technological changes call for new professions and competence requirements, whereas some existing skills are becoming redundant. The present study involved media personnel, with or without irregular shift work, who could be considered as under sustained pressure at work due to intense on-going technological and organizational changes. The study formed part of a comprehensive investigation on shift work and its possible sleep/awake consequences.

This thesis consists of self-reported cross-sectional data. It was assumed that self-reports of bruxism and sleep quality may associate. The study also focused on the possible associations of self-reported bruxism and orofacial pain, as well as some psychological, neurological and physiological factors known to be detrimental to sleep.

3. REVIEW OF THE LITERATURE

3.1. BRUXISM

3.1.1. PubMed

In a recent review article based on systematic MEDLINE search strategies Lobbezoo et al. (2006) reported a growing interest in bruxism; of the papers available since 1966, about one-fourth were published during the last five years. To update the references pertinent to the present review as regards bruxism, the following PubMed search was performed on 16 October 2007: the MeSH Major Topic '*bruxism*' with the search limits of '*published in the last 5 years*' (the interval since the first paper included in this thesis was in progress), '*humans*', '*english*', and '*all adult: 19+ years*'. It resulted in a total of 92 publications of which 20 could be categorized as case reports or comments, four as reviews, and one as an *in vitro* study. Altogether 68 of the search results were original papers (including the three out of the four papers in the present thesis), which as overviewed might be classified at least in two ways as follows:

1) Studies on sleep bruxism, and sleep/awake bruxism (not always clearly defined):

- sleep bruxism (35 studies), assessed and monitored by overnight recordings
- sleep/awake bruxism (33), which could be further subdivided into

    - self-reports (13)
    - self-reports combined with clinical signs (10)
    - experimental settings (10)

2) Studies based on their design or main interest:

- pain/TMD (15)
- diagnostic/technical (14)
- psychological (12)
- experimental (10)
- treatment (drug, splint, biofeedback) intervention (10)
- other (4)
- tooth wear (3)

3.1.2. Definitions of bruxism

Bruxism as a term is derived from the Greek (brychein), meaning 'to gnash the teeth'. Bruxism as a phenomenon was noted in very early times (approximately 600-200 BC) and as a developing concept it has been described e.g. by Olkinuora (1972), Faulkner (1990a), and Sjöholm (1995). According to the American Academy of Orofacial Pain (Okeson 1996), bruxism is:

> "diurnal or nocturnal parafunctional activity that includes clenching, bracing, gnashing and grinding of teeth"

The International Classification of Sleep Disorders (ICSD) (American Academy of Sleep Medicine 1997) previously grouped the primary sleep disorders under two subgroups: "*dyssomnias*, which included disorders that produce a complaint of insomnia or excessive sleepiness, and *parasomnias*, which included disorders that intrude into or occur during sleep but do not produce a primary complaint of insomnia or excessive sleepiness." The dyssomnias were further subdivided into the intrinsic (i.e. induced primarily by factors inside the body) and extrinsic (i.e. induced primarily by factors outside the body), and circadian rhythm disorders. In that previous edition of the ICSD, bruxism was categorized as a *parasomnia*. However, the recent version of the ICSD (American Academy of Sleep Medicine 2005) does not recognize dyssomnias and defines insomnia somewhat differently (see chapter 3.3.), while bruxism is reclassified in it as "a stereotyped movement disorder characterized by grinding or clenching of the teeth during sleep". According to current ICSD criteria sleep bruxism is involved with:

- reports or awareness of tooth grinding sounds or clenching during sleep with

- one or more of the following present:

  o abnormal wear of the teeth
  o jaw muscle discomfort, fatigue, or pain and jaw lock upon awakening
  o masseter muscle hypertrophy upon voluntary forceful clenching, and

- the jaw muscle activity is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder.

3.1.3.1. Detecting sleep bruxism: electromyography (EMG) and polysomnography (PSG)

Sleep bruxism is usually detected with EMG by recording masticatory muscle function during sleep. For example, as part of PSG van der Zaag et al. (2005) measured the numbers of bruxism episodes per hour of sleep, bruxism bursts per hour, and calculated a bruxism duration index (percentage of total sleep time spent bruxing). However, three types of episodes based on EMG signals can be scored: 1) *phasic and rhythmic,* when brief bursts (0.25 to 1 second) are separated by inter-burst intervals; 2) *tonic and sustained*, when the EMG burst lasts longer than 2 seconds, and 3) *mixed*, when a combination of these patterns are detected (American Academy of Sleep Medicine 2005). In the diagnosis of sleep bruxism a typical PSG comprises the following methods: bilateral EMG (from masseter, temporal and tibialis muscles), electrocardiogram (ECG), electroencephalogram (EEG), electro-oculogram (EOG), and audio-video recordings (Lavigne et al. 1996, 2001a; van Selms et al. 2004). Lavigne et al. (1996) evaluated the American Sleep Disorders Association criteria (1990) after some adjustment; in their validation study, the clinical diagnosis of sleep bruxism was based on the report of tooth grinding sounds (occurred at least five nights a week in the last six months, as noted by the bed partner) with the presence of at least one of the following secondary criteria: 1) abnormal tooth wear or shiny spots on restorations, 2) jaw muscle discomfort upon awakening, and 3) masseteric muscle hypertrophy upon digital palpation. After evaluating these clinical criteria with PSG among 18 bruxers and 18 asymptomatic subjects, Lavigne et al. (1996) proposed sleep bruxism diagnostic criteria for sleep research purposes. The criteria were derived from EMG and audio-video recognition of jaw muscle activity in relation to: > 4 sleep bruxism episodes/hour of sleep, > 6 bruxism bursts/episode and/or > 25 sleep bruxism bursts/hour of sleep, and the presence of (in each case) > 1 sleep

bruxism episodes with tooth grinding sounds. Their criteria were eventually able to correctly classify over 80 % of bruxers and controls. Lavigne et al. (1996, 2001a) have also shown that nearly 90 % of the EMG events related to sleep bruxism are composed of phasic and mixed type episodes. Based on these findings, they have suggested the term rhythmic masticatory muscle activity (RMMA) for the most typical EMG pattern related to sleep bruxism.

3.1.3.2. Detecting sleep bruxism: ambulatory EMG

Ambulatory EMG recording devices have become easier to operate and are able to measure masticatory muscle activity more minutely, i.e., the number, duration, and magnitude of bruxism events can be evaluated with fair accuracy (Gallo & Palla 1995; Ikeda et al. 1996; Haketa et al. 2003; Harada et al. 2006). Criteria for the detection of sleep bruxism with ambulatory EMG recording system have been suggested (Ikeda et al. 1996), but their validation in a large population is missing. Shochat et al. (2007) recently introduced a novel device for home screening for sleep bruxism; in their validation study the BiteStrip (Scientific Laboratory Products Ltd., Tel Aviv, Israel) was found to be a viable screening tool with acceptable accuracy for identifying sleep bruxism. The BiteStrip is a small (2x7 cm) lightweight (4g) device applied to the cheek, over the masseter muscle. It consists of two electromyographic electrodes, a miniature amplifier, real-time analysis hardware and software, a miniature display unit, and a lithium battery. The BiteStrip registers each electromyographic peak exceeding 30% of maximal voluntary clench (measured at the start of recording) for a period of up to 12 hours. Other devices also have been used for research purposes, e.g. the BruxChecker (N.A.), a simple device for evaluating grinding patterns (Onodera et al. 2006); Bruxcore (N.A.), an analyzing method for quantification of tooth abrasion (Ommerborn et al. 2005); and the Intra-Splint Force Detector (N.A.), a system for sleep bruxism detection as reflected by forceful tooth-to splint contacts (Baba et al. 2003). However, the problem with ambulatory EMG is that with the absence of polysomnography other confounding usual oromotor activities cannot be controlled for.

3.1.3.3. Detecting sleep/awake bruxism: self-reports and clinical guidelines

Most of the criteria to diagnose sleep/awake bruxism include self-reports of tooth clenching or grinding – as perceived by the subject or noted by another person. Several proposed questionnaires exist in the literature (Koyano et al. 2008), but the one by Pintado et al. (1997) appears to serve the purpose; to become classified as a bruxer according to them, at least two of the six following items should be positive with concomitant tooth wear:

- Has anyone heard you grinding your teeth at night?
- Is your jaw ever fatigued or sore on awakening?
- Are your teeth or gums ever sore on awakening?
- Do you ever experience temporal headaches on awakening?
- Are you ever aware of grinding your teeth during the day?
- Are you ever aware of clenching your teeth during the day?

Lavigne et al. (2005) suggest the use of the following clinical features as a guide when diagnosing sleep bruxism in the clinic:

- teeth grinding or tapping noticed by the patient's sleep partner or family member
- complaints of jaw muscle discomfort, fatigue, or stiffness and occasional headaches
- presence of tooth wear or shiny spots on fillings

- tooth sensitivity to hot or cold (one tooth or several teeth)
- muscle hypertrophy
- temporomandibular joint sounds (clicking) or reduction of movement
- tongue indentation

3.1.3.4. Detecting awake bruxism

The diagnosis of awake bruxism is practically based solely on self-reported awareness of the phenomenon. It most likely mainly consists of tooth clenching or bracing of jaw muscles whilst tooth grinding is rarely observed while awake in the absence of certain medication or a neurological disorder. Awake bruxism, however, is neither well understood nor has standardized diagnostic criteria. It is also difficult to differentiate 'true' bruxism-related muscle activity from concomitant normal oromotor activity involved in wakefulness.

3.1.5. Prevalence of bruxism among populations

Because of the broad definitions used, the reported prevalence figures of bruxism vary greatly, between 4-88 % (Faulkner 1990a; Lavigne & Montplaisir 1994, 1995; Hublin et al. 1998; Lavigne et al. 1999; Ahlberg et al. 2002; Carlsson et al. 2003). However, the prevalence of 6-20 % reported by Lavigne & Montplaisir (1994) is often noted in the literature. The prevalence of awake bruxism is about 20 % for the adult population, occurring more often among females. Sleep bruxism, in turn, defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding and/or clenching, is in normal subjects detected in about 8 % of the adult population. Its prevalence decreases with age from 14-18 % in childhood to 3 % in the elderly, with no gender difference (Lavigne & Montplaisir 1994; Laberge et al. 2000; Ohayon et al. 2001; Ng et al. 2005).

3.1.6. Etiological aspects of bruxism

Many theories to explain the controversial character of bruxism have emerged over the years (Reding et al. 1968; Faulkner 1990a, 1990b; Lavigne & Montplaisir 1995; Molin 1999; Lobbezoo & Naeije 2001, Lobbezoo et al. 2006). According to the existing literature, two groups of proposed etiological factors can be distinguished: peripheral (morphological) and central (pathophysiological/physiopathological and psychological). At present, the bruxism is more often thought to be regulated centrally, not peripherally (Lobbezoo & Naeije 2001). Recent reports indicate that genetic effects may have a significant role in the origin of bruxism (Hublin & Kaprio 2003), and that bruxism shares a common genetic background with sleeptalking (Hublin et al. 2001). In addition, bruxism in childhood and adulthood is reportedly highly correlated in both genders (Hublin et al. 1998). Studies have also shown risk factors that may exacerbate sleep bruxism, viz., smoking, caffeine and heavy alcohol consumption (Lavigne et al. 1997; Ohayon et al. 2001); type A personality - anxiety (Pingitore et al. 1991; Pierce et al. 1995; Ohayon et al. 2001; Lavigne et al. 2005); and sleep disorders such as snoring, sleep apnea, or periodic limb movements (Lavigne & Montplaisir 1994; Lavigne et al. 2005; Ohayon et al. 2001; Bader & Lavigne 2000; Sjöholm et al. 2000)

3.1.7. The role of peripheral factors

In the past, peripheral factors were considered as the main cause of bruxism. Ramfjord (1961) suggested that bruxism could be eliminated by occlusal adjustment. Yet, many studies indicate that the relationship between bruxism and occlusal factors is weak or non-existent (Cheng et al. 2004; Demir et al. 2004; Manfredini et al. 2004a, 2004b). In the recent study of Michelotti et al. (2005) experimental supracontacts were indeed associated with significant reductions of the EMG activity when awake. In addition, a series of double-blind randomized controlled studies in Finland have shown that artificial occlusal interferences seem to disturb the oromotor balance in those with previous experience of temporomandibular disorders (Le Bell et al. 2002, 2006; Niemi et al. 2006). According to the review article of Manfredini et al. (2003a), however, there remains a lack of methodologically sound studies to definitively rule out the importance of occlusal factors in the etiopathogenesis of bruxism. On the other hand, the review article by Luther (2007) states that no evidence exists that occlusal interferences could cause bruxism, or that occlusal adjustments could prevent it.

3.1.8. Pathophysiological factors

In controlled polysomnographical studies, Lobbezoo et al. (1997a, 1997b) showed that short-term use of L-dopa (dopamine-precursor) and bromocriptine (D2 receptor agonist) inhibits bruxism, while Lavigne et al. (2001b) found no effect when using bromocriptine. On the other hand, selective serotonine reuptake inhibitors (SSRIs) may have an indirect influence on the central dopaminergic system, as according to Lobbezoo et al. (2001) SSRIs may cause bruxism after long-term usage. Also, severe amphetamine-induced bruxism is described in a case report of See & Tan (2003). Although disturbances in the dopaminergic system (both hyper- and hypo-dopaminergic states) may link to bruxism, other neurochemicals and their possible interactions with dopamine are suggested for further study (Chen et al. 2005; Lobbezoo et al. 2006). However, sound evidence exists that most sleep bruxism appears to be under the transient influence of cardiac sympathetic activity detected, among others, as tachycardia at the onset of RMMA (Sjöholm et al. 1995; Kato et al. 2001, 2003; Marthol et al. 2006). In addition, according to Miyawaki et al. (2003a, 2003b, 2004), there are associations between bruxism activities and a supine sleeping position, gastroesophageal reflux, episodes of decreased esophageal pH, and swallowing. Yet, as recently reviewed (Walters et al. 2007) motor excitability, autonomic nervous system sympathetic activation (dopamine, adrenalin), and stress/anxiety/anger have shown the strongest evidence for both awake and sleep bruxism. Nevertheless, scientific evidence for a putative mechanism that may contribute to explaining the multidimensional physiopathology of bruxism is better overall for sleep bruxism compared to awake bruxism.

3.1.9. Psychological factors

According to the review article of Lobbezoo & Naeije (2001), studies have suggested that stress experience and psychosocial factors may play an important role in the etiology of bruxism. Awake bruxism may be a parafunction associated with life stress or occupational load (Glaros 1981; Kato et al. 2003; Lavigne et al. 2005). Evidence exists that both experienced and anticipated stress associate with awake clenching but seem to be unrelated to sleep-bruxism recorded with ambulatory devices (Watanabe et al. 2003; van Selms et al. 2004). Anxiety disorders have also been associated with bruxism in children (Monaco et al. 2002), and a few such reports exist from adult populations (Olkinuora 1972; Kampe et al.

1997; Manfredini et al. 2005), although none have recently been performed in non-patients. Already a few decades ago, Olkinuora (1972) suggested that there would be 'non-strain bruxists' and 'strain bruxists', which reminds us of the problem today in separating sleep/awake bruxism. However, a clear-cut association was recently found between self-reported bruxism and stress experience among multiprofessional media personnel (Ahlberg et al. 2002). On the other hand, as regards sleep bruxism, rigorous evidence is lacking to support the notion that it would be an anxiety-related disorder (Pierce et al. 1995; Ohayon et al. 2001). Lack of evidence also exists as regards the effectiveness of cognitive behavioral treatment on sleep bruxism (Ommerborn et al. 2007).

3.2. OROFACIAL PAIN

3.2.1 Pain in the facial area

Orofacial pain may be best defined as follows (Okeson 1996):

"Facial pain that originates from the musculoskeletal structures of the masticatory system is included in the category of pain complaints known as temporomandibular disorders (TMD), which is a collective term embracing a number of clinical problems that involve the masticatory musculature, the temporomandibular joints (TMJs) and associated structures, or both."

Orofacial pain has also been termed as myofascial pain, jaw muscle pain, facial muscle pain, and craniofacial pain, for example, because the expression 'orofacial' has been considered to be influenced by confounding pain from teeth due to a dental problem or, among others, tension headache or migraine (Sipilä 2002), and referral pain from neck (Kuttila et al. 2004).

3.2.2. Assessing orofacial pain

Assessing orofacial pain in research may be based on self-reported symptoms or clinical findings, or both. Both of these are also affected by several intrapersonal factors or states, e.g. somatization or alexithymia. Thus, using methods that could control these underlying factors have been underscored (Suvinen & Reade 1995; Rantala et al. 2004; Suvinen et al. 2005). The Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) (Dworkin & LeResche 1992) is a dual axis system in which Axis I is for the physical disorders (clinical TMD condition) and Axis II for the psychosocial factors. At present, it has been translated into several languages, including Finnish (Rantala et al. 2003). The RDC/TMD has been used in many clinical studies (List & Dworkin 1996; Wahlund et al. 1998; Marcusson et al. 2001; Rantala et al. 2003; Manfredini et al. 2003b) and in epidemiological studies (Dworkin et al 1990; Ohrbach & Dworkin 1998; List et al. 1999).

3.2.3. Orofacial pain among populations and widespread pain

The overall prevalence of TMD among populations is reportedly quite high, at 22–30 % (Von Korff et al. 1988; De Kanter et al. 1993). In adult populations the prevalence of TMD-related pain (of which facial muscle pain is often noted) has been reported to vary between 5 % and

15 % (Von Korff et al. 1998; Lipton et al. 1993; Goulet et al. 1995; Riley & Gilbert 2001). A recently published population study revealed that 14 % of Finnish adults have clinical signs of orofacial muscle pain (Rutkiewicz et al. 2006). TMD may also be part of a generalized pain; clinical studies (Hagberg et al. 1994; Türp et al. 1998) as well as epidemiologic studies (Von Korff et al 1988; Rauhala et al. 2000) have shown associations between facial pain and pain conditions in other parts of the body. A Finnish case-control study (Sipilä et al. 2005) found that subjects with facial pain report more pain and have more muscular tenderness outside the facial area. Moreover, Sipilä et al. (2006) also found that half of the facial pain conditions among young adults were part of a more widespread pain and that those subjects were more often depressed. In addition, comorbidity between facial pain, widespread pain, and depressive mood was more prevalent among women than men.

3.2.4. Bruxism and orofacial pain

Studies have addressed bruxism as an underlying factor for TMD (Molina et al. 1999; Glaros et al. 1998, 2005; Carlsson et al. 2003; Johansson et al. 2006). Some reports (Lobbezoo & Lavigne 1997; Pergamalian et al. 2003) have concluded that the link between bruxism and jaw pain seems weak. However, Huang et al. (2002) reported a significant association between self-reported bruxism and both myofascial pain and TMJ arthralgia. Dao et al. (1994) suggested that pain associated with bruxism and with myofascial pain may be two different entities. In the 20-year follow-up study by Carlsson et al. (2003) bruxism was associated with signs and symptoms of TMD. Patients with persistent sleep bruxism have also been found more prone to craniofacial pain (Camparis & Sigueira 2006). Johansson et al. (2006) found among 12 468 subjects in Sweden that self-reported bruxism was the strongest risk factor for craniofacial pain. Interestingly, a recent study of Rompre et al. (2007) also revealed that mild bruxers may be at greater risk for pain symptoms than moderate or severe bruxers.

3.2.6. Other factors associated with orofacial pain

Besides somatic pain, psychological factors can either contribute to the pain or actually cause the pain disorder. Studies have also shown that stress (Niemi et al. 1993; Kuttila et al. 1997) and depression (Vimpari et al. 1995; Vassend et al. 1995; Korszun et al. 1996; Carlson et al. 1998; Madland et al. 2000; Sipilä et al. 2001; Yatani et al. 2002) associate with the occurrence of TMD symptoms and their severity. Patients with orofacial muscle pain may also have other stress-related disorders like back pain, asthma, dermatological diseases and stomach problems (Lupton 1966; Berry 1969; Wedel & Carlsson 1987). As regards gender differences, in most population studies women report more facial pain than men (Von Korff et al. 1988; Lipton et al. 1993, Goulet et al. 1995; Riley et al. 1998, Riley & Gilbert 2001, Dao & LeResche 2000). In the study of Lipton et al. (1993), women reported facial pain more than twice as often than men and similar clinical findings were reported by Rutkiewicz et al. (2006). On the other hand, there are studies that show no gender differences in the prevalence of orofacial pain (Locker & Grushka 1987; Andersson et al. 1993; MacEntee et al. 1993; Bassols et al. 1999). Studies have also shown a great variability in the prevalence of orofacial pain symptoms in different age groups. In the studies of Locker & Grushka (1987) and Von Korff et al. (1988) facial pain has been suggested to be less prevalent among older persons than in younger subjects. According to Österberg et al. (1992), clinical signs of muscle tenderness tend to decrease with age. In contrast, Rutkiewicz et al. (2006) showed a higher prevalence of clinically detected signs of masticatory muscle pain in older age groups. On the other hand, Lipton et al. (1993) found that the prevalence of facial pain remain relatively constant across age groups.

3.3. SLEEP

3.3.1. Sleep characteristics

By definition (Carskadon & Dement 2005), sleep is:

> "a reversible behavioral state of perceptual disengagement from and unresponsiveness to the environment, and a complex amalgam of physiologic and behavioral processes"

From the physiological perspective, normal human sleep comprises two states: non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, which alternate cyclically during a sleep episode. Sleep begins in NREM and progresses through deeper NREM stages (NREM sleep consists of 4 stages altogether). The first REM episode occurs approximately 80 to 100 minutes after sleep onset. NREM and REM sleep then cycle with a period of approximately 90 minutes. REM sleep episodes lengthen during sleep, forming a total of one-fourth of all sleep. So called slow wave sleep (NREM stages 3 and 4) tends to decline with increasing age. Factors that predictably affect sleep are, among others, previous sleep-wake history (i.e., time spent awake), phase of the circadian timing system, ambient temperature and sounds, drugs (medication), and sleep disorders.

3.3.2. Circadian rhythm

The *master biological clock*, which locates in the bilaterally paired suprachiasmatic nucleus in the anterior hypothalamus, controls the timing of sleep and wake in humans and regulates the 24-hour (i.e., circadian) behavioral, physiologic, and biologic rhythms (Turek et al. 2005).

3.3.3. Insomnia and parasomnia

According to the International Classification of Sleep Disorders (American Academy of Sleep Medicine 2005) insomnia is:

> "a complaint of difficulty initiating sleep, difficulty maintaining sleep, or waking up too early, or sleep that is chronically non-restorative or poor in quality"

The above sleep difficulty occurs despite adequate opportunity and circumstances for sleep. Additionally, at least one of the following awake impairment related to sleep difficulty is reported:

- fatigue or malaise
- attention, concentration, or memory impairment
- social or vocational dysfunction
- mood disturbance or irritability

- daytime sleepiness
- motivation, energy, or initiative reduction
- proneness to errors or accidents at work or while driving
- tensions, headaches, or gastrointestinal symptoms
- concerns or worries about sleep

Although sleep is typically accompanied by postural recumbence, behavioral quiescence, closed eyes, and all the other indicators commonly associated with sleeping, other behaviors may occur. These behaviors are called *parasomnias* and they may include sleepwalking, sleeptalking, bruxism (reclassified as a movement disorder in the ICSD-2005), and other physical activities.

3.3.4. Prevalence of insomnia symptoms among populations

Studies reporting insomnia have used various criteria and comparison of the results may be ambiguous. In western European countries, however, it is estimated that between 10 % and 35 % of the population have insomnia symptoms of various degrees of severity (Ohayon & Partinen 2002). Finland seems to have a unique pattern of insomnia compared with many other European countries; according to recent epidemiological data, the overall prevalence of insomnia symptoms occurring at least three nights per week was 37.6 % and the prevalence of any insomnia disorder diagnosis was 11.7 % (Ohayon & Partinen 2002). Compared with many other European countries studied with the same methodology, including Italy (Ohayon & Smirne 2002), United Kingdom (Ohayon et al. 1997), and Germany (Ohayon & Zulley 2001), the prevalence of insomnia was 1.5 to 2 times higher in Finland. Also, sleep quality was worse in summer and circadian rhythm disorders were at least two times higher than in other European countries. In the study of Hublin et al. (1996), the Finnish twin cohort study, 11 % of females and 7 % of males reported daytime sleepiness on every or almost every day. In Sweden, the prevalence of insomnia symptoms varies from about 10 % to 36 % (Gislason & Almqvist 1987; Liljenberg et al. 1988; Asplund & Åberg 1998; Hetta et al. 1999; Mallon et al. 2000; Janson et al. 2001). In the study of Pallesen et al. (2001) the prevalence of insomnia in Norway was 12 %. The high prevalence of insomnia complaints in the Nordic countries has been explained by the dark period during midwinter, which is thought to influence the circadian rhythm.

3.3.5. Gender, age, and sleep

In the study of Åkerstedt et al. (2002), based on a large representative population sample of 58 115 individuals in Sweden, a major indicator for disturbed sleep and fatigue was female gender. The reason is not clear, but family responsibilities and hormone cycles may contribute to it. Contrary to most other epidemiological reports, a recent study of Ohayon & Partinen (2002) in Finland found that men and women were comparable for all insomnia symptoms and global sleep dissatisfaction. Age has been a predictor of disturbed sleep (Karasek 1979; Ribet & Derriennic 1999; Leger et al. 2000). However, the study of Åkerstedt et al. 2002 showed that although increasing age was indicative for disturbed sleep, waking up was easier and sleep was more often restorative among the older subjects. Interestingly, in the Finnish study of Ohayon & Partinen (2002), although the prevalence of disrupted sleep in those 55 years and older remained comparable with other epidemiological studies, a clearly higher prevalence of disrupted sleep was found among the younger participants.

3.3.6. Shift work and sleep

Shift work is well established as a cause of disturbed sleep or, rather, shortened sleep (Åkerstedt 1998; Härmä et al. 1998). Studies using a sleep diary as well as laboratory studies have shown that the main sleep period at an unusual time is 1-4 h shorter than night sleep (Torsvall et al. 1989; Tepas & Carvalhais 1990). According to Jewett et al. (1999), and Kecklund et al. (1997) shift work has been shown to affect the circadian rhythm and to be connected with work related problems. Moreover, irregular shift work has been implicated as a cause of sleep disorders and tiredness, and it may even expose employees to work hazards (Härmä et al. 2002; Sallinen et al. 2003).

3.3.7. Sleep and health / health and sleep – the vicious circle

Chronic insomnia is considered as a risk factor for the development of depression (Lustberg & Reynolds 2000). According to several epidemiological surveys short sleepers and long sleepers seem to have poorer life expectancy than those sleeping 7-8 hours per night (Hammond 1964; Youngstedt & Kripke 2004; Tamakoshi & Ohno 2004; Hublin et al. 2007). Patients with cardiovascular disease are more likely to have insomnia (Katz & McHorney 1998). Also, in a Swedish study of men aged 30-69 years, hypertension, obstructive pulmonary disease, diabetes, obesity, and rheumatic diseases were found more prevalent among insomniacs (Gislason & Almqvist 1987).

3.3.8. The role of stress

In the study of Katz & McHorney (1998), although medical conditions were associated with an increased risk of insomnia, subjects with depressive disorders were at greater risk than those with chronic somatic illness. The study of Sutton et al. (2001) also showed that a very stressful life, severe pain and dissatisfaction with one's health evinced the highest odds ratios for insomnia. Nervousness and tension were most commonly associated with insomnia in the study of Martikainen et al. (2003) in the middle-aged population and they further concluded that psychosocial factors were more significantly associated with prolonged insomnia than somatic health problems. Elo et al. (2003) introduced a single item five-scale question to measure stress. The method was shown as reliable for measuring perceived stress on the population level as well on the individual level. However, their stress question (‘*Stress means the situation when a person feels tense, restless, nervous or anxious, or is unable to sleep because his/her mind is troubled all the time. Do you feel that kind of stress these days?*’) in fact also includes perceptions of anxiety and insomnia.

3.3.9. Sleep architecture and bruxism

Masticatory muscle contractions may be observed in any stage of sleep but most sleep bruxism (80 %) episodes tend to occur during light NREM sleep stages 1 and 2. Up to 10 % of sleep bruxism episodes may occur during REM sleep. The frequency of episodes peaks during the sleep transition either towards waking or REM sleep (Hyunh et al. 2006). Polysomnographical studies have also revealed that sleep bruxism mainly appears during the transient arousal response and has been reported to associate with both sleep quality and sleep architecture (Macaluso et al.1998; Lobbezoo & Naeije 2001; Ohayon et al. 2001; Kato et al. 2003), and autonomic-cardiac interactions (Sjöholm 1995; Marthol et al. 2006; Lavigne et al.

2007). In accordance with these findings, Hyunh et al. (2006) recently showed that sleep bruxism was associated with micro arousals and that an increased cardiac sympathetic activity preceeds bruxism events. In other words, most of them are associated with arousals and a sequence of autonomic and motor events such as rise in autonomic-cardiac sympathetic activity at least 4 minutes before RMMA, enhancement of alpha EEG activity 4 seconds before the onset of bruxism, increased body movements, and time-restricted acceleration of heart rate or tachycardia (Hyunh et al. 2006). Concomitant to RMMA disorders such as sleep apnea, snoring, and gastroesophageal reflux may also be detected. On the other hand, in some studies perceived and macrostructural sleep quality, and sleep architecture appear to be normal in bruxism patients (Lavigne et al. 2002; Watanabe et al. 2003). Figure 1 schematically illustrates the two states (NREM/REM) that occur periodically during sleep, and change in heart rate (autonomic nervous system activity).
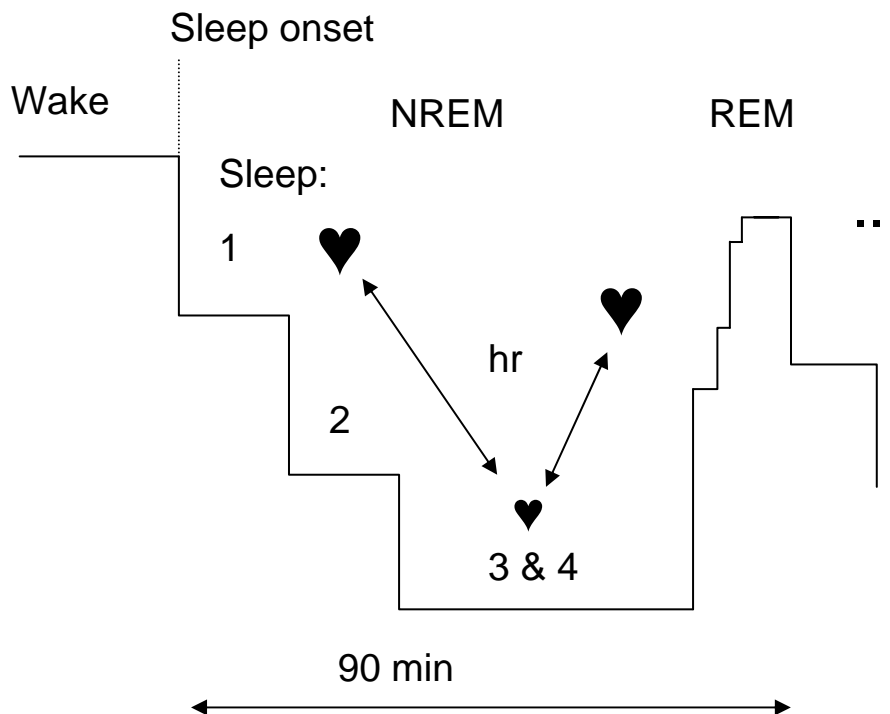
Figure 1. NREM sleep stages 1-4 and REM sleep, and change in heart rate (hr). Sympathetic activity decreases and parasympathetic activity increases towards the slow wave pattern (NREM 3&4), and *vice versa*.

3.3.10. Restless legs syndrome

Restless legs syndrome (RLS) is characterized by discomfort and unpleasant sensations in the legs, which may delay sleep onset or disrupt sleep, and sometimes force the person to move his/her legs or walk to relieve the sensations. RLS does presently have diagnostic criteria; it is regarded as a specific disease entity and, moreover, as one of the commonest neurological disorders (Allen et al. 2003). Periodic limb movement disorder seems clinically close to RLS,

but occurs only during sleep. Interestingly, most recent research suggests that periodic limb movement disorder may be a genetic endophenotype for RLS rather than an independent disease entity (Stefansson et al 2007). The diagnosis of restless legs syndrome is primarily based on the presence of the following four essential diagnostic criteria (Allen et al. 2003):

- an urge to move the legs, usually accompanied or caused by uncomfortable and unpleasant sensations in the legs
- the urge to move or unpleasant sensations begin or worsen during periods of rest or inactivity such as lying or sitting
- the urge to move or unpleasant sensations are partially or totally relieved by movement, such as walking or stretching, at least as long as the activity continues
- the urge to move or unpleasant sensations are worse in the evening or night than during the day or only occur in the evening or night

At present, the ICSD (American Academy of Sleep Medicine 2005) defines RLS similarly to the working group of Allen et al. (2003), but adds that 'the condition is not better explained by another current sleep disorder, medical or neurological disorder, mental disorder, medication use, or substance use disorder'.

Although clinical features of RLS (*anxietas tibiarum*) were first described centuries ago, as cited by Schapira (2004), and 'rediscovered' in recent decades (Ekbom 1945), the general awareness of it remains relatively low. In epidemiological studies utilizing established diagnostic criteria the prevalence of RLS has varied between 3 % and 15 % (Lavigne & Montplaisir 1994; Phillips et al. 2000; Ohayon & Roth 2002, Allen et al. 2005). According to the study of Allen et al. (2005) among 16 202 adults, clinically significant RLS is prevalent in 2.7 % in the general population, seems underdiagnosed, and affects sleep and quality of life. Several studies have found RLS to be more prevalent among females and the elderly (Lavigne & Montplaisir 1994; Rothdach et al. 2000; Ulfberg et al. 2001a, 2001b). Restless legs syndrome can occur as a primary disorder with no apparent cause other than perhaps a genetic predisposition (Desautels et al. 2005). As a secondary condition, it is often related to iron deficiency (O'Keeffe et al. 1994; Sun et al. 1998), pregnancy (McParland & Pearce 1990; Lee et al. 2001;), or end-stage renal disease (Winkelman et al. 1996; Collado-Seidel et al. 1998). Studies show that in idiopathic RLS more than 50 % of patients report having a positive family history of RLS (Lavigne & Montplaisir 1994; Walters et al. 1996; Winkelmann et al. 2000). Also, reduced sleep efficiency reportedly correlates with the severity of RLS (Allen & Earley 2001). A recent Scandinavian study by Bjorvatn et al. (2005) concluded that insomnia is one of the main predictors of RLS. Although RLS and bruxism share certain physiological and neurochemical characteristics and both may be linked to disrupted sleep, according to Lavigne and Montplaisir (1994) there is no strong association between them.

4. AIMS OF THE STUDY

The general aim of the present study was to examine the relationships of self-reported bruxism and sleep quality among employees with or without irregular shift work. The study also focused on the possible associations of bruxism and orofacial pain. Some psychological, neurological and physiological factors known to be detrimental to sleep were studied, especially the somewhat under-recognized restless legs syndrome.

The hypotheses of the study were that self-reported bruxism and:

- irregular shift work would link to each other

- symptoms of orofacial pain and pain severity are associated

- insomnia and its consequences may relate

- restless legs syndrome would share their correlates

5. MATERIALS & METHODS

5.1. Subjects (I-IV)

A standardized questionnaire was mailed to all employees of the Finnish Broadcasting Company with irregular shift work (n=750; 57.0 % men) and to an equal number of randomly selected controls in the same company with regular eight-hour daytime work (42.4 % men). The mean age of invited subjects was 43.0 (SD 10.4) years in irregular shift work and 44.8 (SD 10.2) years in day work. The work duties of the present media personnel included journalism, broadcasting, programme production, technical support and administration.

5.2. Methods

This was a cross-sectional comparative questionnaire study performed among non-patients; in a multiprofessional media population, with or without irregular shift work. The study formed part of a comprehensive investigation on shift work and its sleep/awake consequences, and it focused on irregular shift work. A postal questionnaire with several standard questions was used. Among others, it covered the following: demographic items, employment details, general health experience, physical status, pain symptoms, psychosomatic symptoms, psychosocial status, stress experience, work satisfaction and performance, perceptions of sleep and its consequences. The main variables used in the present series of studies and descriptions of how they were categorized are listed below:

- Demographic data: gender, age in years, weekly working hours, health care visiting.
- Bruxism: self-assessed frequency of tooth clenching or grinding (1 = never, 2 = seldom, 3 = sometimes, 4 = often, and 5 = continually). Those who reported the phenomenon to prevail 'often' or 'continually' were termed as 'frequent bruxers'.
- Level of perceived stress, measured as follows: 'Stress means the situation when a person feels tense, restless, nervous or anxious, or is unable to sleep because his/her mind is troubled all the time. Do you feel that kind of stress these days?' (1 = not at all, 2 = only a little, 3 = to some extent, 4 = rather much, and 5 = very much).
- Anxiety: the 10-item subscale of the Symptom Checklist-90-Revised (Derogatis 1983) score was used as a raw scale. In the logistic regression anxiety was considered as present with a score above the 90th percentile.
- Dissatisfaction with current work shift schedule (irregular shifts vs. day time work) as an expressed urge to change the current work shift.
- Orofacial pain: perceived current orofacial pain (pain in the face, jaw or preauricular area, or in ear) within the past month, and chronic orofacial pain (pain sustained over six months) severity and disablility scores according to the RDC/TMD Axis II for Graded Chronic Pain (Dworkin & LeResche 1992).
- Insomnia symptoms (i.e. difficulties initiating or maintaining sleep): difficulties initiating sleep (DIS), disrupted sleep (DS), and early morning awakenings (EMA). A symptom was considered as present when it occurred at least three nights per week. EMA in the irregular work group means early wakenings, i.e., that subjects with the symptom woke up before they intended, despite the hour, and had difficulties in going back to sleep.
- Perceived consequences of disturbed sleep: non-restorative sleep (NRS) (sustained > 1 month), tiredness (at least 3 days per week), sleep deprivation (SLD) (subjective need for sleep 1h > actual sleep time).

- Neurological and physical confounding factors affecting sleep quality: Restless legs syndrome (presence of the four essential diagnostic criteria according to the NIH diagnosis and epidemiology workshop for RLS), snoring (as perceived or reported by bed partner at least 3 nights per week).
- Smoking: all tobacco use (cigarettes, cigars and pipe) was considered as smoking. Former smokers who had stopped tobacco use less than a year ago were included in the smoker group.

5.3. Statistical methods

Student's t-test and Mann-Whitney U-test were used to compare the group means. The χ^2 test was used to study the associations between categorical variables. The Kruskal-Wallis h-test and Jonckheer-Terpstra test were used to evaluate the anxiety scores in different bruxism categories. Logistic regression models were fitted to analyse the independent effects of the studied background variables on the probability of dependent variables. The forced entry method was used, i.e. all selected independent variables were entered in a single step in each regression model (Afifi & Clark 1996).

6. RESULTS

6.1. Demographic data

The response rate in the irregular shift work group was 82.3% (56.6 % men) and in the regular daytime work group 34.3 % (46.7 % men). The overall response rate was 58.3 % (53.7 % men). The mean age of males in shift work was 45.0 (SD 10.6) years and of females 42.6 (SD 10.7) years (p<0.001); the corresponding figures for daytime workers were 47.4 (SD 9.7) and 45.5 (SD 10.1) years (NS), respectively.

6.2. Bruxism, stress, dissatisfaction, age, and health care visits (Paper I)

Bruxism (p<0.01) and stress experiences (p<0.001) were significantly more occurrent among those dissatisfied with their current work shift schedule. Those with irregular shifts were also more often dissatisfied with their current work shift schedule than those in daytime work (25.1 % vs. 5.1 %, p<0.01). According to logistic regression (Figure 1), the probability of frequent bruxism (reported altogether by 10.6 %) was significantly associated with dissatisfaction (p<0.05), number of dental visits (p<0.05) and visits to a physician (p<0.01), and with younger age (p<0.05).

Figure 1. (data shown in detail in Table 4, Paper I)
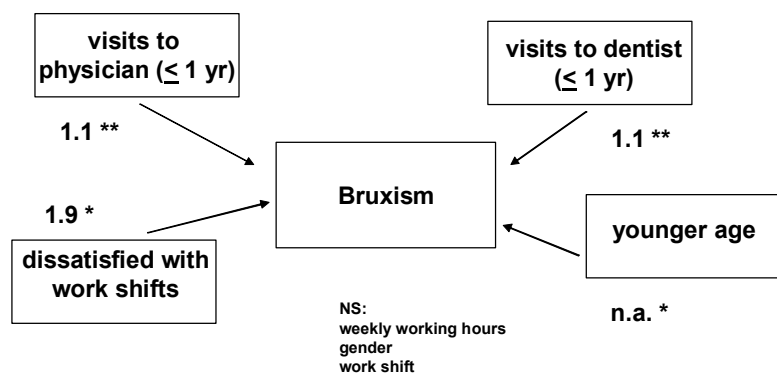

Factors associated with frequent bruxism. Odds ratios and levels of significance.
(*p<0.05, **p<0.01)
n.a. = not applicable; odds ratios not calculated for one additional year of age

6.3. Bruxism: associations with stress and anxiety (unpublished)

Frequent bruxers tended to report significantly more severe anxiety (p<0.001) (Figure 2). According to logistic regression, while the effects of age, gender, work type, and dissatisfaction with work shifts were controlled for, severe stress experience (OR 5.7; 95 % CI 2.9.-11.3) and anxiety (OR 2.6: 95 % CI 1.6-4.2) were significantly associated with the

probability of frequent bruxism. Also, subjects reporting severe stress and anxiety (studied as an interaction term) were found 6.3 (95 % CI 3.1-12.6) times more likely to report frequent bruxism. (unpublished)

Figure 2. (unpublished data)
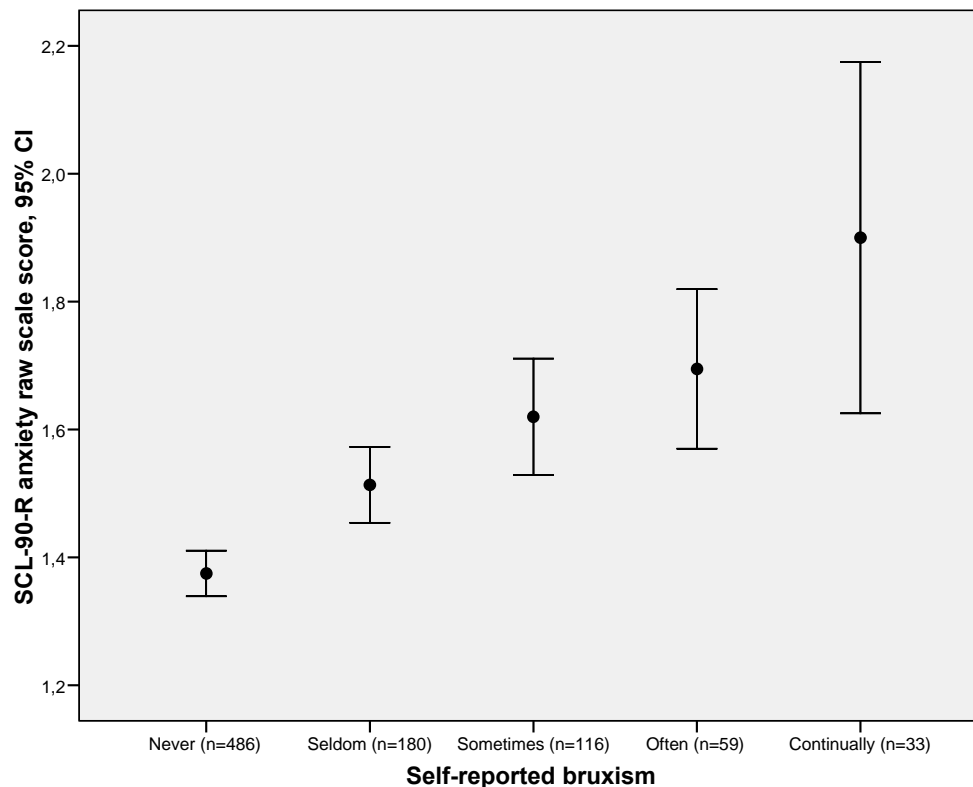
The mean SCL-90-R of 10-item anxiety raw-scale scores and 95% confidence intervals by self-reported bruxism. Statistical evaluation by Kruskal-Wallis (p<0.001) and Jonckheer-Terpstra tests (p<0.001). (N = 874)

6.4. Orofacial pain according to RDC/TMD Axis II, and associations of pain severity with bruxism, insomnia symptoms and dissatisfaction (Paper II)

Current orofacial pain was found overall in 19.6 % of the study population. Among those reporting current pain 88.3 % had experienced it for over six months. They all claimed that their pain characteristically fluctuated; none had had continuous pain. According to the RDC/TMD Axis II scores, none of the subjects with chronic orofacial pain reported their pain as disabling, and grades III and IV were not found. Insomnia symptoms and frequent bruxism were significantly more prevalent in grade II than in lower grades. Similarly, dissatisfaction with one's work shift schedule was more often seen in grade II, and females reported intense pain significantly more often than males. According to logistic regression the probability of current orofacial pain was significantly positively associated with frequent bruxism (p<0.001), female gender (p<0.001) and disrupted sleep (p<0.01), and significantly negatively associated with age $\geq$ 45 years (p<0.01). Frequent bruxers were 6.2 times more likely to perceive current orofacial pain than other subjects. (Figure 3)

Figure 3. (data shown in detail in Table 3, Paper II)
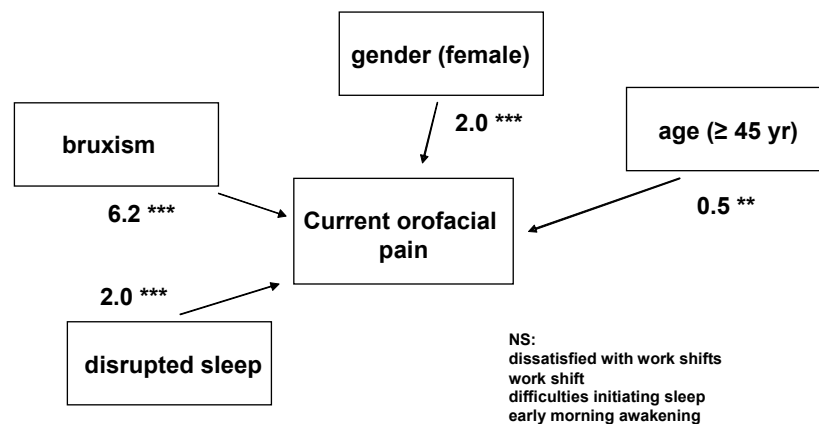
Factors associated with current orofacial pain. Odds ratios and levels of significance.
(*p<0.05, **p<0.01, ***p<0.001)

6.5. Bruxism, perceived sleep quality and consequences of disturbed sleep (Paper III)

Altogether 26.8 % reported difficulties initiating sleep, 43.6 % had experienced disrupted sleep, and 10.3 % suffered from early awakenings. The figures for non-restorative sleep, tiredness, and sleep deprivation were 36.2 %, 26.1%, and 23.7 %, respectively.

Frequent bruxism was significantly associated with difficulties initiating sleep (p<0.05) and disrupted sleep (p<0.05). Dissatisfaction with one's work shifts was significantly associated with difficulties initiating sleep (p<0.01) and early awakenings (p<0.01). Restless legs syndrome was significantly associated with difficulties initiating sleep (p<0.05) as also was snoring with disrupted sleep (p<0.05). Frequent bruxism and dissatisfaction with current work shift schedule were both significant independent factors for all variables describing insufficient sleep consequences. Table 1 shows the found associations of the studied factors with perceived insomnia symptoms and their awake consequences (for more detailed information please see Tables 1-3, Paper III).

6.6. Bruxism and restless legs syndrome (Paper IV)

No statistically significant differences emerged between shift work and day work groups regarding the occurrence of RLS. Females reported RLS (11.4 %) slightly more often than males (7.7 %) (NS). According to logistic regression, self-reported frequent bruxism (p<0.05) and older age (p<0.05) were significantly positively associated with RLS. Dissatisfaction with the current work shift schedule (p<0.05) and RLS (p<0.05) were both significantly positively associated with frequent bruxism, while increasing age (p<0.05) was significantly negatively associated with it. Increasing smoking frequency was modestly associated with both frequent bruxism and RLS (NS).

Table 1.

Associations and levels of significances* (NS in greyish) of the studied independent variables with perceived sleep quality, logistic regression. (Paper III)

DIS	DS	EMA	NRS	Tiredness	SLD
Gender (female) *	Gender (female) *	Gender (female) *	Gender (female) *	Gender (female) *	Gender
Age	Age(≥ 45 yr) *	Age (≥ 45 yr) **	Age (< 45 yr) **	Age	Age (<45 yr) ***
Irregular shift work	Irregular shift work	Irregular shift work	Irregular shift work	Irregular shift work	Irregular shift work
Dissatisfaction **	Dissatisfaction	Dissatisfaction **	Dissatisfaction **	Dissatisfaction ***	Dissatisfaction **
Frequent bruxism *	Frequent bruxism *	Frequent bruxism	Frequent bruxism *	Frequent bruxism **	Frequent bruxism *
Restless legs *	Restless legs	Restless legs	Restless legs	Restless legs	Restless legs
Snoring	Snoring **	Snoring	Snoring	Snoring	Snoring *

DIS = difficulties initiating sleep, DS = disrupted sleep, EMA = early mornig awakening, NRS = non-restorative sleep, SLD = sleep deprivation.

 (* p< 0.05, ** p< 0.01, *** p< 0.001, NS)

7. DISCUSSION

7.1. Subjects

The present study was performed on media personnel who could be considered as under sustained pressure at work due to intense on-going technological, organizational, and economic changes. The study formed part of a comprehensive investigation on shift work and its sleep/awake consequences, and it focused on irregular shift work, which, however, did not emerge as a significant factor in itself. The good response rate in the irregular shift work group (82.3 %) should be noted. Unfortunately, despite several postal reminders, we resulted in a markedly lower response rate in the regular day work group (34.3 %). This was partly expected as the study was transparently targeted to examine the health effects of irregular shift work. Due to these uneven response rates the present study may have failed in detecting the actual differences between these two groups. However, the invited subjects and respondents in the shift work and day work groups were similar as regards gender and age, which possibly suggests that the day work group may also be representative. The multivariate models used in the studies were also tested excluding the work group variable, which did not markedly change the effects of the other independent variables. Thus, the work group variable was not considered to have caused confounding interaction in the models. It should be noted, however, that the study consists of self-reported cross-sectional data only and the results should be interpreted accordingly.

7.2. Methodological aspects

Using questionnaires, as was done in the present study, may cause difficulties in defining the actual prevalence of bruxism: it may be even more common among populations than surveys indicate, but not registered as a behavior by individuals because of its potential subconscious nature. Moreover, reporting of bruxism may be influenced by negative affectivity, and individuals with subjective distress may be more likely to perceive, overreact to and complain about their sensations (Turner et al. 2001). On the other hand, the awareness of a bruxism behavior may be learned from a bed partner or by information provided during dental examinations based on clinical marks in the oral cavity. Although data on bruxism gathered by questionnaires may be difficult to operationalize, numerous surveys have been performed to evaluate possible interactions between bruxism and psychological factors. By definition, however, bruxism events may occur while asleep or awake, although it seems that these two entities do not share their correlates (Lavigne et al. 2005). Awake bruxism is more often tooth clenching or jaw bracing (without tooth contact), while grinding is more rarely noted. Sleep bruxism is more often tooth grinding with phasic (rhythmic) and/or tonic (sustained) or mixed (both types) jaw muscle contractions. Electromyographic recordings definitely reveal masticatory muscle activity, and combined with polysomnography and audio-video recordings sleep bruxism and possible concomitant events can be detected. However, in epidemiological studies data on bruxism are usually gathered from questionnaires or interviews. Thus, awareness of bruxism in epidemiological studies (including the present study) most likely includes of phenomena other than RMMA typical of 'pure' sleep bruxism.

7.3. Bruxism – many questions, few answers

Many of the criteria to define bruxism include reports or awareness of grinding sounds, marks of tooth wear, discomfort/pain, and masseter muscle hypertrophy. It has also been commonly accepted in clinical work that facial muscle pain and tooth wear link to bruxism. However,

orofacial pain may be more often than commonly believed a consequence of brain physiopathology concomitant to bruxism rather than a result of mechanical overload (Svensson et al. 2008). Also, the common belief among dentists that bruxism alone may severely damage teeth may be more of a myth than a fact, as other concomitant factors in the oral cavity may play an even more important role (Johansson et al. 1993, 2008). Bearing in mind this lack of evidence, it is noteworthy that during the past five years only three original papers of the 68 listed in the PubMed with the major topic '*bruxism*' were focused on tooth wear. On the other hand, more than half of the recently published papers about bruxism comprise overnight recordings and many of them include PSG, which at present is often considered as the only method for diagnosing sleep bruxism for research purposes. The PSG studies also tend to have strict inclusion criteria, while they vigorously focus on sleep bruxism (RMMA) and events concomitant to it during sleep (e.g. snoring, sleep apnea, and gastroesophageal reflux), and in such studies subjects with anxiety or stress, for example, are often excluded as confounders. Yet, it may be impossible to isolate the role of anxiety and stress in concomitant autonomic nervous system response and the genesis of bruxism. Thus, it is worth of notion that in the present study dissatisfaction, stress experience, and anxiety were significant factors for the probability of frequent self-reported bruxism. However, an interesting suggestion is that sleep bruxism (RMMA) with protruding movement of the mandible would enhance airway patency in subjects with sleep apnea (Lavigne et al. 2005) and would thus be a protective function. Most recent findings in sleep bruxism studies also propose that with lower cut-off criteria, which may result in more clenching or mixed type bruxists in the analyses, concomitant symptoms (viz., occasional headaches upon awakening, jaw muscle soreness) may emerge (Rompre et al. 2007).

7.4. Bruxism, stress, and anxiety

Some of our main findings were that self-reported frequent bruxism was significantly associated with dissatisfaction, and the frequency of dental and medical visits. Also, that dissatisfaction with the current work shift schedule, and not merely irregular shift work in itself, associated with reported bruxism, and that frequent bruxism was independently associated with severe stress and anxiety. It should be noted that frequent bruxers differed almost totally (95%) as regards their anxiety level compared with those who bruxed seldom or never. Nevertheless, those with irregular shifts reported more stress and were more often dissatisfied with their current work shift schedule than those in daytime work. The dissatisfaction, perhaps, may even explain the different participation rates in the groups. However, perceived bruxism was previously shown in a similar non-patient population to have a coherent relationship with stress and stress-related disorders (Ahlberg et al. 2004), which, together with the present findings, may imply that to some extent it reflects intrapersonal or interpersonal reactivity, or dissatisfaction. In accordance with earlier reports (Könönen & Siirilä 1988; Faulkner 1990a; Lavigne & Montplaisir 1994; Smith et al. 2000), we also found that those of younger age were more often frequent bruxers, and that those in the 35-54-year age group were more stressed than subjects at either end of the age range of the present study. Research indicates, however, that both organizational and personal factors can impact work performance negatively (Burke & Richardson 1996). There is also general consensus that stress problems are multidimensional, i.e. that factors such as coping, psychosocial support and organizational support are involved, along with the availability of effective and biopsychosocially oriented health care (Maslach 1993). These factors have been found important in terms of preventing stress problems becoming chronic and disabling, and of avoiding consequential costs on the individual as well as organizational level.

7.5. Bruxism and orofacial pain

Frequent bruxism was significantly associated with the intensity of chronic orofacial pain, and in the multivariate analysis it was also associated with the overall probability of having orofacial pain while the effects of all other studied variables were simultaneously controlled. In fact, frequent bruxers were over six times more likely to report orofacial pain than those reporting less bruxism. A possible and commonly assumed mechanism for the development of the pain in bruxers may be fatigue in the masticatory muscles caused by overload, but the complex pathways of the pain experience should not be ignored (Svensson et al. 2008). In the present study, however, the RDC/TMD Axis II graded chronic pain scale was used to assess chronic orofacial pain severity. The RDC/TMD permits multiple physical diagnoses for each subject (Axis I) and a dual-axis classification can be applied to psychological issues (Axis II) (Dworkin & LeResche 1992). The Finnish version of the RDC/TMD has recently been introduced and used in non-patients (Rantala et al. 2003). In the present study, the observed prevalence (19.6 %) of acute or current orofacial pain was relatively high but within the range shown in previous surveys (Von Korff et al. 1988; LeResche1997; Macfarlane et al. 2002). It is noteworthy in the present non-patient population, however, that almost all (88.3 %) of those reporting orofacial pain reported awareness of their symptoms over six months, and thus according to the RDC/TMD had a chronic, although fluctuating, pain problem. Congruent with earlier reports, female gender was significantly associated with both current and more intense chronic orofacial pain (Von Korff et al. 1988; Bush et al. 1993; Egermark et al. 2001). In addition, all the studied insomnia symptoms and dissatisfaction with work shift schedule were associated with more intense pain, which may imply that these items are linked to more severe stress experience. On the other hand, nobody reported disabling chronic orofacial pain, which may well be because this was a non-patient population. However, although insomnia symptoms associated with the intensity of orofacial pain in the bivariate analyses, according to logistic regression only disrupted sleep associated with the probability of having current orofacial pain. This may be because RMMA mainly occurs in relation to arousals (Lavigne et al. 2005), i.e., those suffering from disrupted sleep brux more often which, in turn, may cause fatigue in the masticatory muscles and provoke pain. Another explanation could be that those with a sleep disturbance may more often clench their teeth during arousal or awakening and thus would be more aware of it. In addition, they may be more anxious or stressed which may predispose them to presenting pain.

7.6. Bruxism, sleep related problems and their possible consequences

The present findings may indicate that reported bruxism is a sign of dysfunction due to dissatisfaction with stressful working hours. Further, prolonged stress may disturb sleep and lead to enhanced psychological or physical impairment, seen here as significantly more frequent physician visits. The awareness of bruxism may also in part be a sign of an overall increased stress experience and, as bruxing was associated with more frequent dental visits, dentists should be more alert to this symptom complex in treatment planning and in terms of other medical consultations. Studies have suggested that stress experience and psychosocial factors may play an important role in the etiology of bruxism (Lobbezoo & Naeije 2001). In contrast, evidence also exists that both experienced and anticipated stress associate with awake clenching but would be unrelated to sleep-bruxism recorded with ambulatory devices (van Selms et al. 2004; Watanabe et al. 2003). However, difficulties initiating sleep (DIS) and disrupted sleep (DS) were both found to be markedly more common than previously reported in Finland (Ohayon & Partinen 2002). On the other hand, the presence of early awakening (EMA) did not differ from that reported in the general population. Also, female gender was

overall associated with insomnia symptoms, which is in line with previous epidemiologic findings outside Finland (Pallesen et al. 2001; Ohayon & Zulley 2001; Ohayon & Smirne 2002). In the present study, age had diverse effects; those ≥ 45 years more often had DS and EMA but yet the younger subjects were more likely to report insufficient sleep. As regards DS this has not been the case in the general population, but it accords with results found outside Finland. It is noteworthy that DS emerged as a major sleep disturbance affecting nearly half of the subjects in the present non-patient population. In the multivariate analyses, despite the several associations found cross-sectionally, RLS was significantly associated only with DIS. Snoring, in turn, which was bivariately associated only with DS, was multivariately associated with both DS and sleep deprivation. The findings reveal that insomnia symptoms may not necessarily intercorrelate. The results also underscore that neurological or physical factors should be borne in mind when diagnosing and treating insomnia and insufficient sleep problems. In the case of RLS in particular, there may be substantial under-recognition (Allen et al. 2005; Wenning et al. 2005).

7.7. Restless legs syndrome and self-reported bruxism

The prevalence figures for self-reported frequent bruxism and RLS, and the finding that RLS was more prevalent among women, accord with earlier reports (Könönen & Siirilä 1988; Faulkner 1990a; Lavigne & Montplaisir 1994; Lavigne & Montplaisir 1995; Hublin et al. 1998; Lavigne et al. 1999; Phillips et al. 2000; Ohayon & Roth 2002). The main result here was a significant association between bruxism and RLS, which is contradictory to previous findings (Lavigne & Montplaisir 1994). In addition, the slightly positive, although statistically non-significant, effect of smoking behaviour on both bruxism and RLS found in the present nonpatient population is also noteworthy. Logistic regression enabled us to study the independent effect of each background variable on the probability of the studied item while simultaneously controlling the effects of other background variables. Although the use of cumulative smoking as a categorical independent variable in the models resulted in only weak associations, they may imply, as also suggested earlier (Lavigne et al. 1997), that when examining the effects of smoking on both bruxism and RLS, nicotine dose response, duration of habit and degree of dependence need to be more specifically controlled. Also, the significant associations between bruxism and RLS may indicate some comorbidity between them. However, age had diverse effects on bruxism and RLS in the models and, in addition, dissatisfaction with one's work shift schedule - possibly signifying a stressful situation - was significantly associated with frequent bruxism but not with RLS. Thus, despite the co-occurrence of bruxism and RLS in the present study, and although both were slightly associated with smoking behaviour, they are probably distinct entities. Moreover, a possible explanation for their co-occurrence is that both of them can be linked to sleep problems.

8. CONCLUSIONS

Based on the results of the present cross-sectional questionnaire study, performed on healthy subjects with or without irregular shift work, it can be concluded that self-reported bruxism may have a relationship with:

- insomnia and insufficient sleep
- presence of orofacial pain and pain severity
- dissatisfaction with work shifts (not with the type of work in itself)
- perceived stress and anxiety
- younger age
- numbers of dental and medical consultations,

and that bruxism may have comorbidity with the restless legs syndrome.

It is also suggested that subjectively conceptualized awareness of bruxism may be linked to stress-related states and behavior which could be useful knowledge for health care professionals.

ACKNOWLEDGEMENTS

I am privileged to be surrounded by a family who has always believed in me. My father Heikki has encouraged me throughout my life; he and his wife Elina were not only interested in my work but always willing to offer a helping hand. Thank you for your love. My sisters Susanna and Elina, and brother Heikki, thank you for being always there for me.

Finally, my deepest love goes to my husband Jari and to our beloved children Aino, Ella, Oskar, and Otto. Without you, nothing would matter.

Helsinki, June 2008

Kristiina Ahlberg

REFERENCES

Afifi AA, Clark V. Computer-aided multivariate analysis. Texts in statistical science. London: Chapman & Hall; 1996.

Ahlberg J, Rantala M, Savolainen A, Suvinen T, Nissinen M, Sarna S. et al. Reported bruxism and stress experience. Community Dent Oral Epidemiol 2002; 30: 405-8.

Ahlberg J, Savolainen A, Rantala M, Lindholm H, Könönen M. Reported bruxism and biopsychosocial symptoms: a longitudinal study. Community Dent Oral Epidemiol 2004; 32: 307-11.

Åkerstedt T. Shift work and disturbed sleep/wakefulness. Sleep Med Rev 1998; 2: 117-28.

Åkerstedt T, Fredlund P, Gillberg M, Jansson B. Work load and work hours in relation to disturbed sleep and fatique in a large representative sample. J Psychosom Res 2002; 53: 585-8.

Allen RP, Earley CJ. Restless legs syndrome: a review of clinical and pathophysiological features. J Clin Neurophysiol 2001; 18: 128-47.

Allen RP, Walters AS, Montplaisir J, Hening W, Myers A, Bell TJ, Ferini-Strambi L. Restless legs syndrome prevalence and impact: REST general population study. Arch Intern Med 2005; 165: 1286-92.

Allen RP, Picchietti D, Hening WA, Trenkwalder C, Walters AS, Montplaisir J, Restless Legs Syndrome Diagnosis and Epidemiology workshop at the National Institutes of Health; International Restless Legs Syndrome Study Group. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. Sleep Med 2003; 4: 101-19.

American Academy of Sleep Medicine (AASM). The international classification of sleep disorders revised: diagnostic and coding manual (ICSD) Rochester, MN: American Academy of Sleep Medicine; 1997.

American Academy of Sleep Medicine (AASM). The international classification of sleep disorders revised: diagnostic and coding manual (ICSD) 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005.

American Sleep Disorders Association. Parasomnia: International classification of sleep disorders: Diagnostic and coding manual. Thorpy, MJ. editor: Rochester, Minnesota: American Sleep Disorders Association ASDA, 1990. pp. 142-85.

Andersson HI, Ejlertsson G, Leden I, Rosenberg C. Chronic pain in a geographically defined general population: studies of differences in age, gender, social class, and pain localization. Clin J Pain 1993; 9: 174-82.

Asplund R, Åberg H. Sleep and cardiac symptoms amongst women aged 40-64 years. J Intern Med 1998; 243: 209-13.

Baba K, Clark GT, Watanabe T, Ohyama T. Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain 2003; 17: 58-64.

Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. Review Article. Sleep Med Rev 2000; 4: 27-43.

Bassols A, Bosch F, Campillo M, Cañellas M, Baños JE. An epidemiological comparasion of pain complaints in the general population of Catalonia (Spain). Pain 1999; 83: 9-16.

Berry DC. Mandibular dysfunction pain and chronic minor illness. Br Dent J 1969; 127: 170-5.

Bjorvatn B, Leissner L, Ulfberg J, Gyring J, Karlsborg M, Regeur L, et al. Prevalence, severity and risk factors of restless legs syndrome in the general adult population in two Scandinavian countries. Sleep Med 2005; 6: 307-12.

Burke RJ, Richardson AM. Stress, burnout and health. In: Cooper CL, Ed. Handbook of stress, medicine and health. Boca Raton: CRC Press; 1996.

Bush FM, Harkins SW, Harrington WG, Price DD. Analysis of gender effects on pain perception and symptom presentation in temporomandibular pain. Pain 1993; 53: 73-8.

Camparis CM, Siqueira JT. Sleep bruxism: clinical aspects and characteristics in patients with and without chronic orofacial pain. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006; 101: 188-93.

Carlson CR, Reid KI, Curran SL, Studts J, Okeson JP, Falcate D, et al. Psychological and physiological parameters of masticatory muscle pain. Pain 1998; 76: 297-307.

Carlsson GE, Egermark I, Magnusson T. Predictors of bruxism, other parafunctions, and tooth wear over a 20-year follow-up period. J Orofac Pain 2003; 17: 50-7.

Carskadon A, Dement W. Normal human sleep: An overview. In: Kryger MH, Roth T, Dement WC. (Eds). Principles and practice of sleep medicine. 4th ed., Philadelphia, PA: Elsevier Saunders; 2005. pp. 13-23.

Chen WH, Lu YC, Lui CC, Liu JS. A proposed mechanism for diurnal/nocturnal bruxism: hypersensitivity of presynaptic dopamine receptors in the frontal lobe. J Clin Neurosci 2005; 12: 161-3.

Cheng HJ, Chen YQ, Yu CH, Shen YQ. The influence of occlusion on the incidence of bruxism in 779 children in Shanghai. Shanghai Kou Qiang Yi Xue 2004; 13: 98-9.

Collado-Seidel V, Kohnen R, Samtleben W, Hillebrand GF, Oertel WH, Trenkwalder C. Clinical and biochemical findings in uremic patiens with and without restless legs syndrome. Am J Kidney Dis 1998; 31: 324-8.

Dao TT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J Orofac Pain 1994; 8: 350-6.

Dao TT, LeResche L. Gender differences in pain. J Orofac Pain 2000; 14: 169-84.

De Kanter RJ, Truin GJ, Burgersdijk RC, Van't Hof MA, Battistuzzi PG, Kalsbeek H, Käyser AF. Prevalence in the Dutch adult population and meta-analysis of signs and symptoms of temporomandibular disorders. J Dent Res 1993; 72: 1509-18.

Demir A, Uysal T, Guray E, Basciftci FA. The relationship between bruxism and occlusal factors among seven- to 19-year-old Turkish children. Angle Orthod 2004; 74: 672-6.

Derogatis LR. The SCL-90-R. Administration, scoring, and procedures manual II. Baltimore, MD: Clinical Psychometric Research; 1983.

Desautels A, Turecki G, Montplaisir J, Xiong L, Walters AS, Ehrenberg BL, et al. Restless legs syndrome: confirmation of linkage to chromosome 12q, genetic heterogeneity, and evidence of complexity. Arch Neurol 2005; 62: 591-6.

Dworkin SF, LeResche L. Research diagnostic criteria for temporomandibular disorders: review, criteria, examinations and specifications, critique. J Craniomandib Disord 1992; 6: 301-55.

Dworkin SF, Huggins KH, LeResche L, Von Korff M, Howard J, Truelove E, Sommers E. Epidemiology of signs and sympoms in temporomandibular disorders: clinical signs in cases and controls. J Am Dent Assoc 1990; 120: 273-81.

Egermark I, Carlsson GE, Magnusson T. A 20-year longitudinal study of subjective symptoms of temporomandibular disorders from childhood to adulthood. Acta Odontol Scand 2001; 59: 40-8.

Ekbom KA. Restless legs syndrome : A clinical study of a hitherto overlooked disease in the legs characterized by peculiar paresthesia (>>anxietas tibiarum>>), pain and weakness and occurring in two main forms, asthenia crurum paraesthetica and asthenia crurum dolorosa [dissertation]. Stockholm: Karoliniska Institutet; 1945.

Elo A-L, Leppänen A, Jahkola A. Validity of a single-item measure of stress symptoms. Scand J Work Environ Health 2003; 29: 444-51.

Faulkner KD. Bruxism: A review of the literature. Part I. Aust Dent J 1990a; 35: 266-76.

Faulkner KD. Bruxism: A review of the literature. Part II. Aust Dent J 1990b 35: 355-61.

Gallo LM, Palla S. Activity recognition in long-term electromyograms. J Oral Rehabil 1995, 22: 455-62.

Glaros AG. Incidence of diurnal and nocturnal bruxism. J Prosthet Dent 1981; 45: 545-9.

Glaros AG, Tabacchi K, Glass EG. Effect of parafunctional clenching on TMD pain. J Orofac Pain 1998; 12: 145-52.

Glaros A, Williams K, Lausten L. The role of parafunctions, emotions and stress in predicting facial pain. J Am Dent Assoc 2005; 136: 451-8.

Gislason T, Almqvist M. Somatic diseases and sleep complaints: an epidemiological study of 3201 Swedish men. Acta Med Scand 1987; 221: 475-81.

Goulet JP, Lavigne GJ, Lund JP. Jaw pain prevalence among French speaking Canadians in Québeck and related symptoms of temporomandibular disorders. J Dent Res 1995; 74: 1738-44.

Hagberg C, Hagberg M, Kopp S. Musculoskeletal symptoms and psychosocial factors among patients with craniomandibular disorders. Acta Odontol Scand 1994; 52: 170-7.

Haketa T, Baba K, Akishige S, Fueki K, Kino K, Ohyama T. Utility and validity of a new EMG-based bruxism detection system. Int J Prosthodont 2003; 16: 422-8.

Hammond EC. Some preliminary findings on physical complaints from a prospective study of 1,064,004 men and women. Am J Public Health Nations Health 1964; 54: 11-23.

Harada T, Ichiki R, Tsukiyama Y, Koyano K. The effect of oral splint devices on sleep bruxism: a 6-week observation with an ambulatory electromyographic recording device. J Oral Rehabil 2006; 33: 482-8.

Härmä M, Tenkanen L, Sjöblom T, Alikoski T, Heinsalmi P. Combined effects of shift work and life-style on the prevalence of insomnia, sleep deprivation and daytime sleepiness. Scand J Work Environ Health 1998; 24: 300-7.

Härmä M, Sallinen M, Ranta R, Mutanen P, Müller K. The effect of an irregular shift system on sleepiness at work in train drivers and railway traffic controllers. J Sleep Res 2002; 11: 141-51.

Hetta J, Broman JE, Mallon L. Evaluation of severe insomnia in the general population-implications for the management of insomnia: insomnia, quality of life and healthcare consumption in Sweden. J Psychopharmacol 1999; 13(4Suppl 1): 35-6.

Huang GJ, LeResche L, Critchlow CW, Martin MD, Drangsholt MT. Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). J Dent Res 2002; 81: 284-8.

Hublin C, Kaprio J, Partinen M, Heikkilä K, Koskenvuo M. Daytime sleepiness in adult, Finnish population. J Intern Med 1996; 239: 417-23.

Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998; 7: 61-7.

Hublin C, Kaprio J, Partinen M, Koskenvuo M. Parasomnias: co-occurrence and genetics. Psychiatr Genet 2001; 11: 65-70.

Hublin C, Kaprio J. Genetic aspects and genetic epiedemiology of parasomnias. Sleep Med Rev 2003; 7: 413-21.

Hublin C, Partinen M, Koskenvuo M, Kaprio J. Sleep and mortality: a population-based 22-year follow-up study. Sleep 2007; 30: 1245-53.

Huynh N, Kato T, Rompré PH, Okura K, Saber M, Lanfranchi PA, et al. Sleep bruxism is associated to micro-arousals and an increase in cardiac sympathetic activity. J Sleep Res 2006; 15: 339-46.

Ikeda T, Nishigawa K, Kondo K, Takeuchi H, Clark GT. Criteria for the detection of sleep-associated bruxism in humans. J Orofac Pain 1996; 10: 270-82.

Janson C, Lindberg E, Gislason T, Elmasry A, Boman G. Insomnia in men- a 10-year prospective population based study. Sleep 2001; 24: 425-30.

Jewett ME, Dijk D-J, Kronauer RE, Dinges DF. Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. Sleep 1999; 22: 171-9.

Johansson A, Omar R, Fareed K, Haraldson T, Kiliaridis S, Carlsson GE. Comparison of the prevalence, severity and possible causes of occlusal tooth wear in two young adult populations. J Oral Rehabil 1993; 20: 463-71.

Johansson A, Unell L, Carlsson GE, Söderfeldt B, Halling A. Risk factors associated with symptoms of temporomandibular disorders in a population of 50- and 60-year-old subjects. J Oral Rehabil 2006; 33: 473-81.

Johansson A, Johansson A-K, Omar R, Carlsson GE. Rehabilitation of the worn dentition. J Oral Rehabil 2008; 35: 548-66.

Kampe T, Edman G, Bader G, Tagdae T, Karlsson S. Personality traits in a group of subjects with long-standing bruxing behaviour. J Oral Rehabil 1997; 24: 588-93.

Karasek RA. Job demands, job decision latitude and mental strain. Implication for job redesign. Adm Sci Q 1979; 24: 285-308.

Kato T, Rompré P, Montplaisir JY, Sessle BJ, Lavigne GJ. Sleep bruxism: an oromotor activity secodary to micro-arousal. J Dent Res 2001; 80: 1940-4.

Kato T, Montplaisir JY, Guitard F, Sessle BJ, Lund JP, Lavigne GJ. Evidence that experimentally induced sleep bruxism is a consequence of transient arousal. J Dent Res 2003; 82: 284-8.

Katz D, McHorney C. Clinical correlates of insomnia in patients with chronic illness. Arch Intern Med 1998; 158: 1099-107.

Kecklund G, Åkerstedt T, Lowden A. Morning work: effects of early rising on sleep and alertness. Sleep 1997; 20: 215-23.

Korszun A, Hinderstein B, Wong M. Comorbidity of depression with chronic facial pain and temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1996; 82: 496-500.

Koyano K, Tsukiyama Y, Ichiki R, Kuwata T. Assessment of bruxism in the clinic. J Oral Rehabil 2008; 35: 495-508.

Kuttila M, Kuttila S, Niemi PM, Alanen P, LeBell Y. Fluctuation of treatment need for temporomandibular disorders and age, gender, stress, and diagnostic subgroups. Acta Odontol Scand 1997; 55: 350-5.

Kuttila S, Kuttila M, Le Bell Y, Alanen P, Suonpää J. Characteristics of subjects with secondary otalgia. J Orofac Pain 2004; 18: 226-34.

Könönen M, Siirilä H. Prevalence of nocturnal and diurnal bruxism in patients with psoriasis. J Prosthet Dent 1988; 60: 238-41.

Laberge L, Tremblay RE, Vitaro F, Montplaisir J. Development of parasomnias from childhood to early adolescence. Pediatrics 2000; 106: 67-74.

Lavigne GJ, Montplaisir J. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep 1994; 17: 739-43.

Lavigne GJ, Montplaisir J. Bruxism: epidemiology, diagnosis, pathophysiology, and pharmacology. In: Fricton JR, Dubner RB, editors. Orofacial pain and temporomandibular disorders. New York: Raven Press; 1995. pp. 387-404.

Lavigne GJ, Rompré PH, Montplaisir JY. Sleep bruxism: validity of clinical research diagnostic criteria in a controlled polysomnographic study. J Dent Res 1996; 75: 546-52.

Lavigne G, Lobbezoo F, Rompré PH, Nielsen TA, Montplaisir J. Cigarette smoking as a risk factor or an exacerbating factor for restless legs syndrome and sleep bruxism. Sleep 1997; 20: 290-3.

Lavigne GJ, Goulet JP, Zuconni M, Morrison F, Lobbezoo F. Sleep disorders and the dental patient: an overview. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999; 88: 257-72.

Lavigne GJ, Guitard F, Rompré PH, Montplaisir JY. Variability in sleep bruxism activity over time. J Sleep Res 2001a; 10: 237-44.

Lavigne GJ, Soucy JP, Lobbezoo F, Manzini C, Blanchet PJ, Montplaisir JY. Double-blind, crossover, placebo-controlled trial of bromocriptine in patients with sleep bruxism. Clin Neuropharmacol 2001b; 24: 145-9.

Lavigne GJ, Rompré PH, Guitard F, Sessle BJ, Kato T, Montplaisir JY. Lower number of K-complexes and K-alphas in sleep bruxism: a controlled quantitative study. Clin Neurophysiol 2002; 113: 686-93.

Lavigne GJ, Manzini C, Kato T. Sleep bruxism. In: Kryger MH, Roth T, Dement WC, editors. Principles and practice of sleep medicine. 4th ed. Philadelphia, PA: Elsevier & Saunders; 2005. pp. 946-59.

Lavigne GJ, Huynh N, Kato T, Okura K, Adachi K, Yao D, Sessle B. Genesis of sleep bruxism: motor and autonomic-cardiac interactions. Arch of Oral Biol 2007; 52: 381-4.

Le Bell Y, Jämsä T, Korri S, Niemi PM, Alanen P. Effect of artificial interferences depends on previous experience of temporomandibular disorders. Acta Odontol Scand 2002; 60: 219-22.

Le Bell Y, Niemi PM, Jämsä T, Kylmälä M, Alanen P. Subjective reactions to intervention with artificial interferences in subjects with and without a history of temporomandibular disorders. Acta Odontol Scand 2006; 64: 59-63.

Lee KA, Zaffke ME, Baratte-Beebe K. Restless legs syndrome and sleep disturbance during pregnancy: the role of folate and iron. J Womens Health Gend Based Med 2001; 10: 335-41.

Leger D, Guilleminault C, Dreyfus JP, Delahaye C, Paillard M. Prevalence of insomnia in a survey of 12,778 adults in France. J Sleep Res 2000; 9: 35-42.

LeResche L. Epidemiology of temporomandibular disorders: implications for the investigation of the etiologic factors. Crit Rev Oral Biol Med 1997; 8: 291-305.

Liljenberg B, Almqvist M, Hetta J, Roos BE, Agren H. The prevalence of insomnia: the importance of operationally defined criteria. Ann Clin Res 1988; 20: 393-98.

Lipton JA, Ship JA, Larach-Robinson D. Estimated prevalence and distribution of reported orofacial pain in the United States. J Am Dent Assoc 1993; 124: 115-21.

List T, Wahlund K, Wenneberg B, Dworkin SF. TMD in children and adolescents: prevalence of pain, gender differences, and perceived treatment need. J Orofac Pain 1999; 13: 9-20.

List T, Dworkin SF. Comparing TMD diagnoses and clinical findings at Swedish and US TMD centers using research diagnostic criteria for tempomandibular disorders. J Orofac Pain 1996; 10: 240-53.

Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997; 11: 15-23.

Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: a controlled clinical trial. Mov Disord 1997a; 12: 73-8.

Lobbezoo F, Soucy JP, Hartman NG, Montplaisir JY, Lavigne GJ. Effects of the dopamine D2 receptor agonist bromocriptine on sleep bruxism: report of two single-patient clinical trials. J Dent Res 1997b; 76: 1610-14.

Lobbezoo F, Naeije M. Bruxism is mainly regulated centrally, not peripherally. J Oral Rehabil 2001; 28: 1085-91.

Lobbezoo F, van Denderen RJ, Verheij JG, Naeije M. Reports of SSRI-associated bruxism in the family physician´s office. J Orofac Pain. 2001; 15: 340-46.

Lobbezoo F, van Der Zaag J, Naeije M. Bruxism: its multiple causes and its effects on dental implants - an updated review. J Oral Rehabil 2006; 33: 293-300.

Locker D, Grushka M. Prevalence of oral and facial pain and discomfort: preliminary results of a mail survey. Community Dent Oral Epidemiol 1987; 15: 169-72.

Lupton DE. A preliminary investigation of the personality of female temporomandibular joint dysfunction patients. Psychother Psychosom 1966; 14: 199-216.

Lustberg L, Reynolds CF. Depression and insomnia: question of cause and effect. Sleep Med Rev 2000; 4: 253-62.

Luther F. TMD and occlusion part II. Damned if we don´t? Functional occlusal problems: TMD epidemiology in a wider context. Br Dent J 2007 Jan 13; 202(1): 1-6. E3; discussion 38-9.

Macaluso GM, Guerra P, Di Giovanni G, Boselli M, Parrino L, Terzano MG. Sleep bruxism is a disorder related to periodic arousals during sleep. J Dent Res 1998; 77: 565-73.

MacEntee MI, Stolar E, Glick N. Influence of age and gender on oral health and related behaviour in an independent elderly population. Community Dent Oral Epidemiol 1993; 21: 234-9.

Macfarlane TV, Blinkhorn AS, Davies RM, Kincey J, Worthington HV. Oro-facial pain in the community: prevalence and associated impact. Community Dent Oral Epidemiol 2002; 30: 52-60.

Madland G, Feinmann C, Newman S. Factors associated with anxiety and depression in facial arthromyalgia. Pain 2000; 84: 225-32.

Mallon L, Broman JE, Hetta J. Relationship between insomnia, depression, and mortality: a 12-year follow-up of older adults in the community. Int Psychogeriatr 2000; 12: 295-306.

Manfredini D, Landi N, Romagnoli M, Cantini E, Bosco M. Etiopathogenesis of parafunctional habits of the stomatognathic system. Minerva Stomatol. 2003a; 52: 339-45, 345-9.

Manfredini D, Cantini E, Romagnoli M, Bosco M. Prevalence of bruxism in patients with different research diagnostic criteria for temporomandibular disorders (RDC/TMD) diagnoses. Cranio 2003b; 21: 279-85.

Manfredini D, Landi N, Romagnoli M, Bosco M. Psychic and occlusal factors in bruxers. Aust Dent J 2004a; 49: 84-9.

Manfredini D, Landi N, Tognini F, Montagnani G, Bosco M. Occlusal features are not a reliable predictor of bruxism. Minerva Stomatol 2004b; 53: 231-39.

Manfredini D, Landi N, Fantoni F, Segù M, Bosco M. Anxiety symptoms in clinically diagnosed bruxers. J Oral Rehabil 2005; 32: 584-8.

Marcusson A, List T, Paulin G, Dworkin S. Temporomandibular disorders in adults with repaired cleft lip and palate: a comparison with controls. Eur J Orthod 2001; 23: 193-204.

Marthol H, Reich S, Jacke J, Lechner KH, Wichmann M, Hilz MJ. Enhanced sympathetic cardiac modulation in bruxism patiens. Clin Auton Res 2006; 16: 276-80.

Martikainen K, Partinen M, Hasan J, Laippala P, Urponen H, Vuori I. The impact of somatic health problems on insomnia in middle age. Sleep Med 2003; 4: 201-6.

Maslach C. Burnout: A multidimensional perspective. In: Schaufeli WB, Maslach C, Marek T, editors. Professional burnout: recent developments in theory and research. Washington, DC: Taylor & Francis; 1993.

McParland P, Pearce JM. Restless legs syndrome in pregnancy. Case reports. Clin Exp Obstet Gynecol 1990; 17: 5-6.

Michelotti A, Farella M, Gallo LM, Veltri A, Palla S, Martina R. Effect of occlusal interference on habitual activity of human masseter. J Dent Res 2005; 84: 644-8.

Miyawaki S, Tanimoto Y, Araki Y, Katayama A, Fujii A, Takano-Yamamoto T. Association between noctural bruxism and gastroesophageal reflux. Sleep 2003a; 26: 888-92.

Miyawaki S, Lavigne GJ, Pierre M, Guitard F, Montplaisir JY, Kato T. Association between sleep bruxism, swallowing-related laryngeal movement, and sleep positions. Sleep 2003b; 26: 461-5.

Miyawaki S, Tanimoto Y, Araki Y, Katayama A, Imai M, Takano-Yamamoto T. Relationships among noctural jaw muscle activities, decreased esophageal pH, and sleep positions. Am J Orthod Dentofacial Orthop. 2004; 126: 615-19.

Molin C. From bite to mind: TMD - a personal and literature review. Int J Prosthodont 1999; 12: 279-88.

Molina OF, dos Santos Junior, Nelson SJ, Nowlin T. A clinical study of specific signs and symptoms of CMD in bruxers classified by the degree of severity. Cranio 1999; 17: 268-79.

Monaco A, Ciammella NM, Marci MC, Pirro R, Giannoni M. The anxiety in bruxer child. A case-control study. Minerva Stomatol 2002; 51: 247-50.

Ng DK, Kwok KL, Cheung JM, Leung SY, Chow PY, Wong WH et al. Prevalence of sleep problems in Hong Kong primary school children: a community-based telephone survey. Chest 2005; 128: 1315-23.

Niemi P, Le Bell Y, Koskinen-Moffett L. Self-reported symptoms of stress in Finnish patients with craniomandibular disorders. J Orofac Pain 1993; 7: 354-8.

Niemi PM, Le Bell Y, Kylmälä M, Jämsä T, Alanen P. Psychological factors and responses to artificial interferences in subjects with and without a history of temporomandibular disorders. Acta Odontol Scand 2006; 64: 300-5.

Ohayon MM, Caulet M, Priest RG, Guilleminault C. DSM-IV and ICSD-90 insomnia symptoms and sleep dissatisfaction. Br J Psychiatry 1997; 171: 382-8.

Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest 2001; 119: 53-61.

Ohayon MM, Zulley J. Correlates of global sleep dissatisfaction in the German population. Sleep 2001; 24: 780-7.

Ohayon MM, Smirne S. Prevalence and consequences of insomnia disorders in the general population of Italy. Sleep Med 2002; 3: 115-20.

Ohayon MM, Roth T. Prevalence of restless legs syndrome and periodic limb movement disorder in the general population. J Psychosom Res 2002; 53: 547-54.

Ohayon MM, Partinen M. Insomnia and global sleep dissatisfaction in Finland. J Sleep Res 2002; 11: 339-46.

Ohrbach R, Dworkin SF. Five-year outcomes in TMD: relationship of changes in pain to changes in physical and psychological variables. Pain 1998; 74: 315-26.

O'Keeffe ST, Gavin K, Lavan JN. Iron status and restless legs syndrome in the elderly. Age Ageing 1994; 23: 200-3.

Okeson JP, American Academy of Orofacial Pain. ed. Orofacial pain. Guidelines for assessment, diagnosis, and management. Chicago: Quintessence Publishing Co; 1996.

Olkinuora M. Bruxism as a psychosomatic phenomenon. [dissertation]. Helsinki: University of Helsinki, 1972.

Ommerborn MA, Giraki M, Schneider C, Schaefer R, Gotter A, Franz M, Raab WH. A new analyzing method for quantification of abrasion on the Bruxcore device for sleep bruxism diagnosis. J Orofac Pain 2005; 19: 232-8.

Ommerborn MA, Schneider C, Giraki M, Schäfer R, Handschel J, Franz M, Raab W. Effects of an occlusal splint compared with cognitive-behavioral treatment on sleep bruxism activity. Eur J Oral Sci 2007; 115: 7-14.

Onodera K, Kawagoe T, Sasaguri K, Protacio-Quismundo C, Sato S. The use of a bruxchecher in the evaluation of different grinding patterns during sleep bruxism. Cranio 2006; 24: 292-9.

Österberg T, Carlsson GE, Wedel A, Johansson U. A cross-sectional and longitudinal study of craniomandibular dysfunction in an elderly population. J Craniomandib Disord 1992; 6: 237-45.

Pallesen S, Nordhus IH, Nielsen GH, Havik OE, Kvale G, Johnsen BH, Skjøtskift S. Prevalence of insomnia in the adult Norwegian population. Sleep 2001; 24: 771-9.

Pergamalian A, Rudy TE, Zaki HS, Greco CM. The association between wear facets, bruxism, and severity of facial pain in patients with temporomandibular disorders. J Prosthet Dent 2003; 90: 194-200.

Phillips B, Young T, Finn L, Asher K, Hening WA, Purvis C. Epidemiology of restless legs symptoms in adults. Arch Intern Med 2000; 160: 2137-41.

Pierce CJ, Chrisman K, Bennett ME, Close JM. Stress, anticipatory stress, and psychologic measures related to sleep bruxism. J Orofac Pain 1995; 9: 51-6.

Pingitore G, Chrobak V, Petrie J. The social and psychologic factors of bruxism. J Prosthet Dent 1991; 65: 443-6.

Pintado MR, Anderson GC, DeLong R, Douglas WH. Variation in tooth wear in young adults over a two-year period. J Prosthet Dent 1997; 77: 313-20.

Ramfjord SP. Bruxism, a clinical and electromyographic study. J Am Dent Assoc 1961; 62: 21-44.

Rantala MA, Ahlberg J, Suvinen TI, Savolainen A, Könönen M. Symptoms, signs, and clinical diagnoses according to the research diagnostic criteria for temporomandibular disorders among Finnish multiprofessional media personnel. J Orofac Pain 2003; 17: 311-16.

Rantala MA, Ahlberg J, Suvinen TI, Savolainen A, Könönen M. Chronic myofascial pain, disk displacement with reduction and psychosocial factors in Finnish non-patients. Acta Odontol Scand 2004; 62: 293-7.

Rauhala K, Oikarinen KS, Järvelin MR, Raustia AM. Facial pain and temporomandibular disorders: an epidemiological study of the Northern Finland 1966 Birth Cohort. Cranio 2000; 18: 40-6.

Reding GR, Zepelin H, Robinson JE Jr, Zimmerman SO, Smith VH. Nocturnal teeth-grinding: all-night psychophysiologic studies. J Dent Res 1968; 47: 786-97.

Ribet C, Derriennic F. Age, working conditions, and sleep disorders: a longitudinal analysis in the French cohort E.S.T.E.V. Sleep 1999; 22: 491-504.

Riley JL 3rd, Gilbert GH, Heft MW. Orofacial pain symptom prevalence: selective sex differences in the elderly? Pain 1998; 76: 97-104.

Riley JL 3rd, Gilbert GH. Orofacial pain symptoms: an interaction between age and sex. Pain 2001; 90: 245-56.

Rompré PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 2007; 86: 837-42.

Rothdach A, Trenkwalder C, Haberstock J, Keil U, Berger K. Prevalence and risk factors of RLS in an elderly population: MEMO study. Memory and Morbidity in Augsburg Elderly. Neurology 2000; 54: 1064-8.

Rutkiewicz T, Könönen M, Suominen-Taipale L, Nordblad A, Alanen P. Occurence of clinical signs of temporomandibular disorders in adult Finns. J Orofac Pain 2006; 20: 208-17.

Sallinen M, Härmä M, Mutanen P, Ranta R, Virkkala J, Müller K. Sleep-wake rhythm in an irregular shift system. J Sleep Res 2003; 12: 103-12.

Schapira AH. Restless legs syndrome: an update on treatment options. Drugs. 2004; 64: 149-58.

See SJ, Tan EK. Severe amphethamine-induced bruxism: treatment with botulinum toxin. Acta Neurol Scand 2003; 107: 161-63.

Shochat T, Gavish A, Arons E, Hadas N, Molotsky A, Lavie P, Oksenberg A. Validation of the BiteStrip screener for sleep bruxism. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2007; 104: e32-e39. Epub 2007 Jul 6.

Sipilä K, Veijola J, Jokelainen J, Järvelin MR, Oikarinen KS, Raustia AM, Joukamaa M. Association between symptoms of temporomandibular disorders and depression: an epidemiological study of the Nothern Finland 1966 Birth Cohort. Cranio 2001; 19: 183-7.

Sipilä K. Facial pain and temporomandibular disorders [dissertation]. Oulu: University of Oulu; 2002.

Sipilä K, Zitting P, Siira P, Niinimaa A, Raustia AM. Generalized pain and pain sensitivity in community subjects with facial pain: a case-control study. J Orofac Pain 2005; 19: 127-32.

Sipilä K, Ylöstalo PV, Joukamaa M, Knuuttila ML. Comorbidity between facial pain, widespread pain, and depressive symptoms in young adults. J Orofac Pain 2006; 20: 24-30.

Sjöholm T. Sleep Bruxism: pathophysiology, diagnosis, and treatment [dissertation]. Turku: University of Turku; 1995.

Sjöholm TT, Piha SJ, Lehtinen I. Cardiovascular autonomic control is disturbed in nocturnal teethgrinders. Clin Physiol 1995; 15: 349-54.

Sjöholm TT, Lowe AA, Miyamoto K, Fleetham JA, Ryan CF. Sleep bruxism in patients with sleep-disordered breathing. Arch Oral Biol 2000; 45: 889-96.

Smith A, Johal S, Wadsworth E, Davey Smith G, Peters T. The scale of occupational stress: the Bristol Stress and Health at work study. Contact research report 265/2000. Health & Safety Executive. Sudbury, UK: HSE Books, 2000. pp. 18-29.

Stefansson H, Rye D, Hicks A, Petursson H, Ingason A, Thorgeirsson T, et al . A genetic risk factor for periodic limb movements in sleep. N Engl J Med 2007; 357: 639-47.

Sun ER, Chen CA, Ho G, Earley CJ, Allen RP. Iron and the restless legs syndrome. Sleep 1998; 21: 371-7.

Sutton DA, Moldofsky H, Badley EM. Insomnia and health problems in Canadians. Sleep 2001; 24: 665-70.

Suvinen TI, Reade PC. Temporomandibular disorders: a critical review of the nature of pain and its assessment. J Orofac Pain 1995; 9: 317-39.

Suvinen TI, Reade PC, Kemppainen P, Könönen M, Dworkin SF. Review of aetiological concepts of temporomandibular pain disorders: towards biopsychosocial model for integration of physical disorder factors with psychological and psychosocial illness impact factors. Eur J Pain 2005; 9: 613-33.

Svensson P, Jadidi F, Arima T, Baad-Hansen L, Sessle BJ. Relationships between craniofacial pain and bruxism. J Oral Rehabil 2008; 35: 524-47.

Tamakoshi A, Ohno Y, JACC Study Group. Self-reported sleep duration as a predictor of all-cause mortality: results from the JACC Study, Japan. Sleep; 2004; 27: 51-4.

Tepas DI, Carvalhais AB. Sleep patterns of shiftworkers. Occup Med 1990; 5: 199-208.

Torsvall L, Åkerstedt T, Gillander K, Knutsson A. Sleep on the night shift: 24-hour EEG monitoring of spontaneous sleep/wake behavior. Psychophysiology 1989; 26: 352-8.

Turek F, Dugovic C, Laposky A. Master Circadian Clock, Master Circadian Rhythm.In: Kryger MH, Roth T, Dement WC, editors. Principles and Practice Sleep Medicine. Philadelphia: Elsevier Saunders; 2005. pp. 318-20.

Turner JA, Dworkin SF, Mancl L, Huggins KH, Truelove EL. The roles and beliefs, catastrophizing, and coping in the functioning of patients with temporomandibular disorders. Pain 2001; 92: 41-51.

Türp JC, Kowalski CJ, O´Leary N, Stohler CS. Pain maps from facial pain patients indicate a broad pain geography. J Dent Res 1998; 77: 1465-72.

Ulfberg J, Nyström B, Carter N, Edling C. Prevalence of restless legs syndrome among men aged 18 to 64 years: an association with somatic disease and neuropsychiatric symptoms. Mov Disord 2001a; 16: 1159-63.

Ulfberg J, Nyström B, Carter N, Edling C. Restless legs syndrome among working-aged women. Eur Neurol 2001b; 46: 17-9.

van Selms MK, Lobbezoo F, Wicks DJ, Hamburger HL, Naeije M. Craniomandibular pain, oral parafunctions, and psychological stress in a longitudinal case study. J Oral Rehabil 2004; 31: 738-45.

van der Zaag J, Lobbezoo F, Wicks DJ, Visscher CM, Hamburger HL, Naeije M. Controlled assessment of the efficacy of occlusal stabilization splints on sleep bruxism. J Orofac Pain 2005; 19: 151-8.

Vassend O, Krogstad BS, Dahl BL. Negative affectivity, somatic complaints, and symptoms of temporomandibular disorders. J Psychosom Res 1995; 39: 889-99.

Vimpari SS, Knuuttila ML, Sakki TK, Kivelä S-L. Depressive symptoms associated with symptoms of the temporomandibular joint pain and dysfunction syndrome. Psychosom Med 1995; 57: 439-44.

Von Korff M, Dworkin SF, Le Resche L, Kruger A. An epidemiologic comparison of pain complaints. Pain 1988; 32; 173-83.

Wahlund K, List T, Dworkin SF. Temporomandibular disorders in children and adolescents: reliability of a questionnaire, clinical examination, and diagnosis. J Orofac Pain 1998; 12: 42-51.

Walters AS, Hickey K, Maltzman J, Verrico T, Joseph D, Hening W, et al. A questionnaire study of 138 patiens with restless legs syndrome: the 'Night-Walkers' survey. Neurology 1996; 46: 92-5.

Walters AS, Lavigne G, Hening W, Picchietti DL, Allen RP, Chokrovertry S, et al. The scoring of movements in sleep. J Clin Sleep Med 2007; 3 : 155-67.

Watanabe T, Ichikawa K, Clark GT. Bruxism levels and daily behaviors: 3 weeks of measurement and correlation. J Orofac Pain 2003; 17: 65-73.

Wedel A, Carlsson GE. Sick-leave in patients with functional disturbances of the masticatory system. Swed Dent J 1987; 11: 53-59.

Wenning GK, Kiechl S, Seppi K, Müller J, Högl B, Saletu M, et al. Prevalence of movement disorders in men and women aged 50-89 years (Bruneck Study cohort): a population based study. Lancet Neurol 2005; 4: 815-20.

Winkelman JW, Chertow GM, Lazarus JM. Restless legs syndrome in end-stage renal disease. Am J Kidney Dis 1996; 28: 372-8.

Winkelmann J, Wetter TC, Collado-Seidel V, Gasser T, Dichgans M, Yassouridis A, Trenkwalder C. Clinical characteristics and frequency of the hereditary of restless legs syndrome in a population of 300 patients. Sleep 2000; 23: 597-602.

Yatani H, Studts J, Cordova M, Carlson CR, Okeson JP. Comparasion of sleep quality and clinical and psychologic characteristics in patiens with tempomandibular disorders. J Orofac Pain 2002; 16: 221-8.

Youngstedt SD, Kripke DF. Long sleep and mortality: rationale for sleep restriction. Sleep Med Rev 2004; 8: 159-74.