Comparison between the rhythmic jaw contractions occurring during sleep and while chewing

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SUMMARY

The masticatory central pattern generator (CPG) may be implicated in the pathophysiology of sleep bruxism (SB). The aim of this study was to compare rhythmic masticatory muscle activity (RMMA) occurring during sleep related to SB with that of natural voluntary chewing in a sample of sleep bruxers. It was hypothesized that the pace of RMMA during sleep is correlated with the chewing pace. Electromyographic (EMG) surface activity was recorded unilaterally from the masseter muscle of 13 participants diagnosed with SB (mean age ± standard deviation = 26.1 ± 9.0 years) by means of portable recorders. For each participant, recordings were carried out in the natural environment setting, always including the dinner time and the entire sleeping period. The time–frequency features of RMMA episodes were extracted automatically offline using a previously validated algorithm. Comparisons between chewing and SB activity indicated that chewing RMMA episodes almost doubled sleep RMMA in duration and power. The mean frequency of SB episodes was 1.0 ± 0.3 Hz, whereas the mean frequency of chewing episodes was 1.5 ± 0.4 Hz. The pace of SB and that of chewing were not correlated significantly ($R = -0.13$; $P = 0.96$). We conclude that sleep RMMA is not related to that of chewing. Despite both activities being accompanied by rhythmic jaw contractions, the pace-generating mechanism of SB may be independent from that of chewing.

INTRODUCTION

Sleep bruxism (SB) is classified as a 'sleep-related movement disorder' that is characterized by repetitive (phasic) or sustained (tonic) contractions of the jaw-closing muscles (American Academy of Sleep Medicine, 2005; Lavigne *et al.*, 1999).

The consequences of SB include tooth destruction, breakage of dental restorations or rehabilitation, temporomandibular dysfunction (e.g. jaw pain or movement limitation), tension headache and tooth grinding sounds that may disrupt the sleep of family or life partners (Lavigne *et al.*, 2003, 2008). SB is reported by 5–8% of the adult population, with the prevalence decreasing with age from 10 to 20% in childhood to 3% in the elderly (Kato and Lavigne, 2010; Lavigne *et al.*, 2008).

The pathophysiology of SB is still poorly understood. Identified risk factors include higher levels of stress and anxiety, alcohol consumption, cigarette smoking, caffeine intake, drugs and a genetic predisposition (Bader and Lavigne, 2000; Kato and Lavigne, 2010; Lavigne *et al.*, 2001a; Lobbezoo and Naeije, 2001). Because sleep rhythmic masticatory muscle activity (RMMA) is usually preceded by brain and cardiac activation during sleep arousals, the autonomic and central nervous system may play a significant causal role for SB (Lavigne *et al.*, 2003, 2008; Lobbezoo and Naeije, 2001). The bruxism generator model proposes that interactions among motor, limbic and autonomic systems activate jaw muscle motoneurones either directly or through a facilitatory or release activity of the central pattern generator (CPG) for rhythmic jaw activity (Lavigne and Montplaisir, 1995), thus contributing to the increase in frequency, duration and intensity of RMMA as seen in patients with SB (Lobbezoo and Naeije, 2001). If this hypothesis is true, as the CPG is commonly active during mastication, then it can be

expected that the pace of SB activity would be correlated with that of chewing activity.

The gold standard for the assessment of RMMA activity during sleep is polysomnography, which allows bruxism episodes to be analysed in relation to several bioelectric, video and audio signals in a controlled experimental environment, thus limiting the confounding of bruxism scoring due to the presence of various types of orofacial activities during sleep (Lavigne *et al.*, 1996). However, polysomnography is expensive and requires a specialized clinic. Polysomnography studies also require the participant to sleep in an unusual environment, which may influence sleep behaviour, disturb sleep and render the collected data questionable in terms of generalization of findings to the general population (Gallo *et al.*, 1999).

Portable electromyographic (EMG) recorders have been developed and used successfully to gather data on RMMA (Farella *et al.*, 2005; Gallo *et al.*, 1999; Michelotti *et al.*, 2005; Po *et al.*, 2011). The activity recorded by this equipment may be more representative of muscle contraction patterns during both wakefulness and sleep than laboratory equipment. An algorithm for an automated detection of RMMA from EMG signals has also been developed and validated under laboratory conditions (Farella *et al.*, 2009). This algorithm allows for time–frequency analysis of rhythmic episodes and is especially suitable for investigating the features of RMMA, as it occurs in the natural environment.

The aim of this study was to compare RMMA occurring during sleep with that of natural chewing in a sample of sleep bruxers. It was also hypothesized that the pace of RMMA during sleep is correlated with the chewing pace.

METHODS

Participants

A convenience sample of 13 participants [two males, 11 females; mean age $\pm$ standard deviation (SD) = 26.1 $\pm$ 9.0 years] were recruited as paid volunteers by means of advertisements. The participants underwent a clinical examination of the dentition, masticatory muscles and temporomandibular joints (TMJs). To be included into the study, they had to be above 18 years and fulfil the minimal diagnostic criteria for sleep bruxism as stated in the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005). In addition, all participants had a history of sounds associated with bruxism reported by their siblings, flatmates or bed partners, and their bruxism could not be explained by other sleep disorders, medications and/or substance use. Exclusion criteria included neurological disorders, eating disorders, use of medications that may affect tooth grinding, missing teeth (with the exception of third molars or orthodontic extractions) and having a beard during recording sessions. None of the participants were affected by any major skeletal and/or dental malocclusion. Six of the 13 participants reported orofacial pain or discomfort upon awakening. No TMJ clicking/sound was recorded or reported at the time of examination in any participant. Participants were informed carefully about the experiment and assured that they could leave the study at any time. The study protocol was approved by the Human Ethics Committee of the University of Otago (09/173) and written informed consents were collected.

EMG equipment

The EMG activity was recorded by means of portable recorders (BSR release 2, Zürich, Switzerland). The recorder has two-channel inputs, which are band-pass-filtered (70–500 Hz). The collected signals are digitized with 10-bit resolution at a sampling rate of 2 kHz, amplified ($\times$8692), and stored in a memory card (MMC: 512 MB) in waveform audio file format (WAV). The portable recorder is supplied with a rechargeable battery and can be programmed to acquire data for up to 24 h per day.

Procedure

The entire experiment for each participant included one clinical session and two EMG recording sessions. During the clinical session, participants were assessed for eligibility and were informed carefully about the study procedure. At the start of each EMG recording session, the skin overlying the masseter muscle was scrubbed with abrasive paste (Natural facial scrub; Natural Instinct, Scoresby, Vic., Australia) and an alcohol wipe (product WP1010; Briemar Nominees Pty Ltd., Koo Wee Rup, Vic., Australia) for skin impedance reduction. Two surface EMG electrodes (model 9013S0212; Alpine Biomed ApS, Skovlunde, Denmark; 20 $\times$ 15 mm) were positioned unilaterally on the masseter muscle of the self-reported preferred chewing side at a centre-to-centre distance of 20 mm, always by the same examiner (J.P.). If the participant did not report a preferred side, the electrodes were applied on the right side. Electrodes were placed at the most prominent point of the masseter muscle during maximum contraction, at its centre, and at a position above the latter, parallel to the main direction of the muscle fibres. A reference electrode was attached on the skin overlying the mastoid process.

In the first EMG recording session, the participants were acquainted with the experimental procedures and performed a series of standardized chewing tasks while sitting in an upright position. The tasks were: chewing toasted bread (1 g), carrot (2 g) and confectionery (2 g) at their habitual rhythm, without any feedback. These tasks were separated by 30-s rest pauses. As a final task, the participants were asked to clench their teeth as hard as possible, three times for 2–3 s each, with 15-s pauses in between clenching efforts. These tasks were completed within a 6-min time-frame. After completing these tasks, the EMG recordings continued for approximately 12 h duration in the participants' natural environment, and were scheduled to always include dinner and the entire sleep period. Gum chewing was

not allowed during the recording period. Upon awakening, the recorder was switched off and the electrodes removed. Detailed self-report diaries of foods eaten were obtained.

The EMG recordings were obtained over two different nights, not necessarily contiguous. The EMG recording for the first night was used for habituation and was consistently discarded (i.e. not included in the results presented here).

Data processing and analyses

Raw EMG data were first evaluated for noise signal artefacts using software (Adobe Audition Version 3; Adobe Systems Inc., San Jose, CA, USA). Collected data were then analysed using a time–frequency analysis of the EMG signal linear envelope. Differing from the classical EMG analysis performed in the time domain, which is based on the amplitude-versus-time representation, the EMG envelope in the time–frequency domain is described in terms of frequency (i.e. number of bursts per unit of time), spectral power and time. An algorithm for detecting RMMA, which is based on time–frequency analysis, has been described extensively in previous publications (Farella *et al.*, 2009; Po *et al.*, 2011). Computational details of this algorithm are summarized as follows: the stored EMG signals were baseband demodulated using root mean square amplitude values calculated over 125 ms contiguous rectangular windows by MatLab version 8.0 (MathWorks, Natick, MA, USA). This allowed for extraction of the EMG signal linear envelope (Fig. 1). Demodulated EMG signals were analysed using the windowed short-time fast-Fourier transform to 64 points with a one-point sliding Hamming window. The resulting spectrum (i.e. spectrogram) had a frequency band ranging from 0 to 4 Hz, a frequency resolution of 0.125 Hz and a time resolution of 125 ms (Fig. 2). For each spectrum, peak frequency (Hz) and peak power (dB) were calculated. The standardized chewing tasks were used to determine the spectral power peak values, which were used to calculate the relative power (%) of EMG spectrograms.

The spectrogram of the demodulated EMG activity envelope was then used to score RMMA episodes. An RMMA episode was defined as a portion of the spectrogram above two predefined frequency and power thresholds. An episode had to last more than 1.5 s and had to contain at least three bursts. The algorithm allowed automatic detection of onset and cessation of rhythmic contraction episodes based on thresholds for peak frequency and relative power, set at 0.625 Hz and 2%, respectively. These settings were calculated using receiving operating characteristics curves to discard possible movement artefacts due to a variety of oral behaviours, including swallowing (Farella *et al.*, 2009). When two RMMA episodes were separated by <2 s, they were merged into one episode.

To indicate whether the frequency increased or decreased across a rhythmic episode, robust linear regression analysis was used to evaluate linear trends of frequency within each rhythmic episode. The slope was expressed in mHz s^{-1} and could be preceded by a positive or negative sign indicating either an increase (up-chirp) or a decrease (down-chirp) in frequency across each episode. Non-significant slopes indicated either a steady frequency of the rhythmic episode or a non-linear trend of frequency over time. To obtain an estimate of the frequency and power of a single rhythmic chewing or SB episode, peak frequency and percentage of power were averaged across each episode's duration, and henceforth in this report they are simply referred to as frequency and power, respectively.

The episode frequency averaged across either daytime or sleep time was regarded as an indication of an individual's chewing pace and SB pace, respectively.

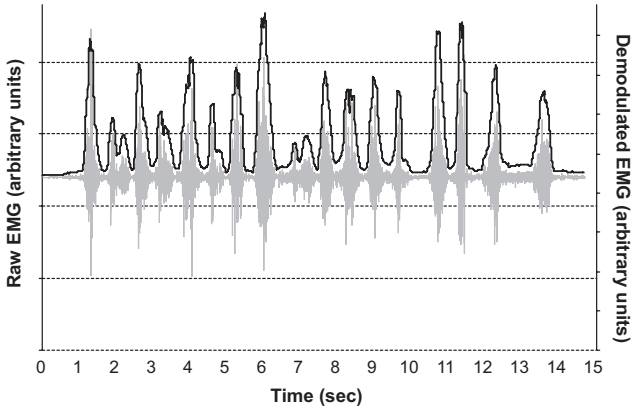

Figure 1. Example of raw electromyographic (EMG) activity (light grey tracing) showing rhythmic masseter muscle contractions recorded during sleep in one of the study participants. The dark grey linear envelope indicates the EMG signal demodulated using root mean square amplitude values calculated over 125 ms contiguous rectangular windows.

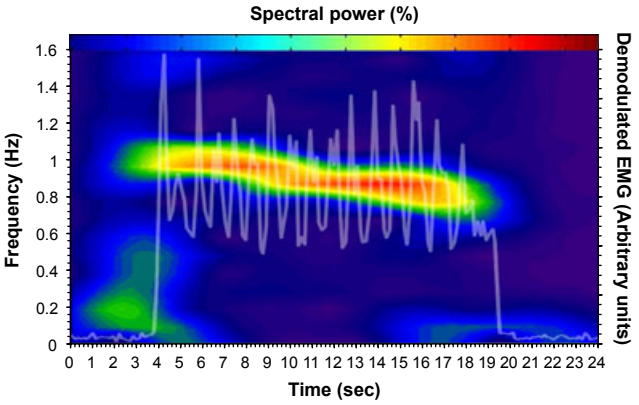

Figure 2. Rhythmic masticatory contraction episode represented in the time–frequency domain, with the corresponding demodulated electromyographic (EMG) activity shown in overlay. The left vertical axis indicates chewing frequency (Hz); the right vertical axis indicates demodulated EMG activity in the time–amplitude domain; the lower horizontal axis indicates time (s). A colour map is used to represent the relative magnitude of spectral power (% of maximum). The mean frequency of this episode is 0.92 Hz. The episode started at a frequency of 1.03 Hz and decreased gradually to 0.82 Hz, showing a down-chirp frequency profile.

Table 1 Demographic data and recording times of participants investigated

Participant ID no.	*Gender*	*Age (years)*	*Start time (hours:min)*	*Sleep time (hours:min)*	*Wake-up/stop time (hours:min)*	*Length of sleep (hours:min)*	*Length of recording (hours:min)*
# 1	F	49.3	19:40	22:00	07:15	09:15	11:35
# 2	F	24.3	18:55	21:50	06:10	08:20	11:15
# 3	F	20.3	17:25	23:30	06:00	06:30	12:35
# 4	F	19.3	18:10	00:00	07:30	07:30	13:20
# 5	F	20.5	17:23	23:30	08:00	08:30	14:37
# 6	M	21.9	18:30	23:45	06:00	06:15	11:30
# 7	F	24.4	20:22	22:05	07:45	09:40	11:08
# 8	F	24.4	18:10	00:00	08:00	08:00	13:40
# 9	F	21.4	17:35	23:30	07:00	07:30	13:25
# 10	F	29.1	17:26	22:50	07:00	08:10	13:34
# 11	M	40.9	18:32	00:00	06:00	06:00	11:28
# 12	F	22.7	19:41	01:50	07:00	05:10	11:19
# 13	F	20.9	18:00	23:45	06:00	06:15	12:00

F, female; M, male.

The accuracy of this algorithm for detecting RMMA has been tested previously against sustained activities (e.g. tooth clenching, biting, yawning etc.) and a variety of possible rhythmic confounding activities and movement artefacts, including swallowing, grimacing, smiling, coughing, reading, jumping, whistling, deep breathing, head movements and repetitive touches and tractions of EMG electrodes/cables. The algorithm could identify chewing and grinding activities successfully, with a sensitivity of 90.0% and a specificity of 97.4% (Farella *et al.*, 2009).

Preliminary analyses consisted of descriptive statistics and normality tests. Comparisons between the time–frequency features of chewing and sleep bruxism episodes were performed by means of linear mixed-model analysis. Correlations were tested by Pearson's correlation analysis. Chi-square tests were used to compare the proportion of down-chirp, up-chirp and steady episodes between daytime and sleep time. All contraction episodes detected after the reported sleeping time were scored as SB RMMA. Statistical analyses were performed using the SAS package (version 8.01; SAS Institute, Cary, NC, USA) and SPSS (version 15.0; Chicago, IL, USA). Type I risk error for all statistical tests was set at 0.05.

RESULTS

All the study participants completed the recording sessions. Mean recording length (±SD) was 12 h 25 min (±1 h 10 min). Self-reported mean duration of sleep (±SD) was 7 h 28 min (±1 h 21 min). Details of the recordings for all participants can be seen in Table 1. All participants reported a good quality of sleep with no arousal.

Self-report diaries of eaten foods showed that the participants ate a wide variety of food types, amounts and consistencies across the different recording days. The mean number of food types eaten (±SD) for all participants was eight (±3), with all participants reporting a variety in amounts and consistencies for each food type on a scale of small–medium–large and soft–medium–tough, respectively. Two examples of these food diaries have been given as Supporting information.

Overall, 1421 RMMA episodes could be detected from all the participants investigated over all recording days. Of these RMMA episodes, 1048 occurred during daytime (i.e. chewing) and 373 during sleep time (i.e. SB). Examples of RMMA episodes occurring during sleep are given in Fig. 3. The mean number of RMMA episodes per night was 30.9 and ranged from five to 74, indicating that the severity of bruxism during the recording night varied markedly across the participants.

The mean duration (±SD) of the pooled daytime RMMA episodes was 10.2 s (±8.2 s) and the 5, 50 and 95th percentiles were 2.5, 7.4 and 28.1 s, respectively; whereas the mean duration (±SD) of the pooled sleep time RMMA episodes was 5.6 s (±3.1 s) with the 5, 50 and 95th percentiles was 2.5, 4.6 and 11.6 s, respectively.

The distribution of the daytime and sleep time RMMA episodes of all recordings for all participants according to their frequency and power is given in Fig. 4.

The mean (±SD) and median frequencies of all the pooled daytime RMMA episodes were 1.53 (±0.41) and 1.58 Hz, respectively. The large majority (90%) of these episodes ranged in frequency from 0.75 Hz (5th percentile) to 2.19 Hz (95th percentile). The mean (±SD) and median powers were 24.0 (±16.4%) and 22.3% respectively, with the 5 and 95th percentile being 3.2 and 52.8%, respectively.

For the pooled sleep time RMMA episodes, the mean (±SD) and median frequencies were 1.05 (±0.27 Hz), and 1.00 Hz, respectively. The large majority (90%) of these episodes ranged in frequency from 0.69 (5th percentile) to 1.50 Hz (95th percentile). The mean (±SD) and median powers were 10.0 (±8.2%), and 6.3%, respectively, with the 5 and 95th percentiles being 2.6 and 25.7%, respectively.

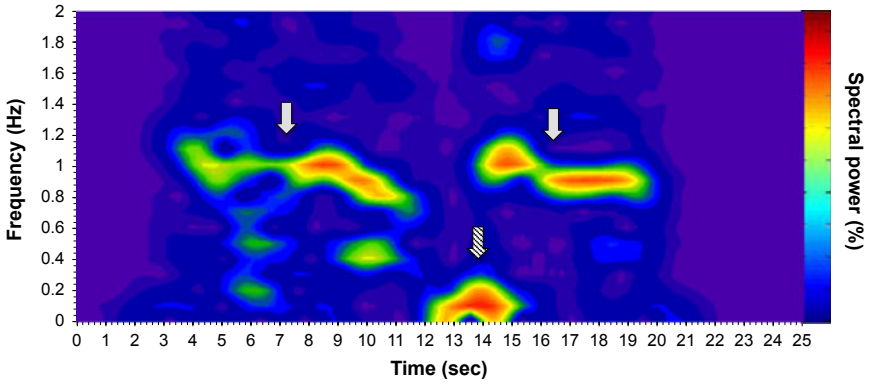

Figure 3. Examples of rhythmic masticatory muscle activity occurring during sleep in one of the study participants. The time–frequency representation shows two contiguous rhythmic masticatory muscle activity (RMMA) episodes (plain arrows) separated by a clenching episode (patterned arrow).

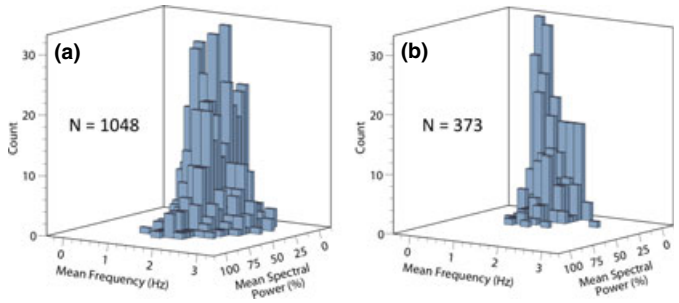

Figure 4. Contraction episodes obtained from sleep bruxers (n = 13) during chewing (a) and during sleep (a), plotted against their mean frequency and their mean power. A comparison of the three-dimensional histograms shows marked differences in counts, mean frequency and mean power, with sleep rhythmic masticatory muscle activity (RMMA) episodes being skewed towards lower frequencies and lower power than chewing episodes.

The mean duration, the mean frequency and the mean power of daytime rhythmic episodes were significantly different from those of sleep RMMA episodes ($F \geq 38.0$; $P \leq 0.001$).

The proportions of down-chirp (35.5%) up-chirp (29.7%) and steady (34.8%) RMMA episodes were similar during the day. Conversely, only 14.4% of RMMA episodes exhibited an up-chirp frequency profile during sleep, 38.6% were down-chirp and 46.9% were steady, the differences between daytime and sleep time being statistically significant ($P < 0.001$).

Mean frequency of daytime RMMA was not correlated significantly ($R = -0.13$; $P = 0.96$) with that of sleep time RMMA (Fig. 5).

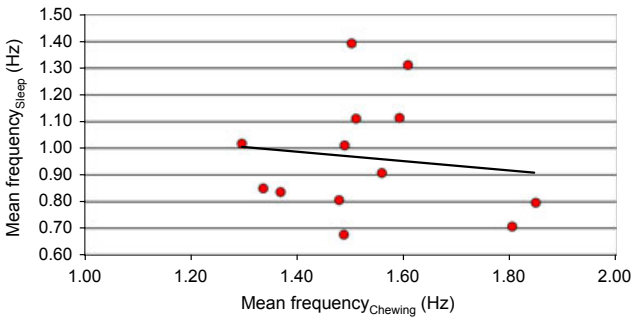

Figure 5. Scatterplot (n = 13) of mean frequency of sleep rhythmic masticatory muscle activity versus chewing activity. The pace of chewing and of sleep bruxism were highly scattered and not correlated significantly ($R = -0.13$; $P = 0.96$).

DISCUSSION

To the best of our knowledge, this is the first study to compare the time–frequency features of sleep bruxism episodes with chewing episodes occurring under unrestrained conditions in the natural environment.

The mean frequency for chewing episodes was consistent with previous laboratory findings obtained from participants with natural dentitions (Bates *et al.*, 1975) and with our previous findings on the natural human chewing pace (Po *et al.*, 2011). The mean frequency of sleep RMMA episodes was also similar to that of a previous report (0.92 Hz) (Lavigne *et al.*, 2001b).

The mean duration of sleep time RMMA episodes is slightly lower than other reports of 7–10 s (Clarke *et al.*, 1984; Lavigne *et al.*, 2001b; Okeson *et al.*, 1994), but higher than the reported mean duration of RMMA episodes in

non-bruxing participants (4.6 s) (Gallo *et al*., 1999). The shorter duration of SB episodes found in our study can be ascribed to the fact that our algorithm identifies only the rhythmic activity and discards sustained activity such as clenching, which may also be part of SB episodes (Camparis *et al*., 2006).

Comparison between chewing and SB activity indicated that chewing episodes were almost twofold longer and twofold more powerful than sleep RMMA episodes. Furthermore, chewing frequency was approximately 50% faster than sleep RMMA frequency. It is also interesting to note that the frequency profiles of chewing episodes differed from that of SB RMMA episodes. Indeed, the proportion of up-chirp RMMA episodes occurring during chewing was approximately twice as high as that of SB activity. Differences in the time–frequency features of RMMA episodes between chewing and sleep are explained partly by the presence of food during chewing, which provides a positive feedback from the periodontal ligament to the CPG (Lavigne *et al*., 1987). During food processing, this may cause a progressive increase of chewing frequency across the contraction episode. The lack of this positive feedback in SB activity may also help to explain why the proportion of RMMA episodes with down-chirp and steady frequency profiles occurring during sleep was higher than during chewing.

Our previous findings indicated that the chewing frequency averaged across an entire day is surprisingly stable over different recording sessions, as it shows very little variation within an individual and large variation between different individuals (Po *et al*., 2011). This indicates that there is an innate chewing pace that is unique for each individual and stable over time, regardless of the variety of food consumed.

The stability of this chewing pace supports the existence of the CPG and its operation during normal chewing conditions in humans. The CPG works as a 'chewing center', generating the basic masticatory motor rhythm of jaw opening and closing movements (Dellow and Lund, 1971). It needs to be emphasized that the CPG is not a simple on–off-switch; rather, it represents a complex network of many brain stem cells with influences from the autonomic nervous system (Westberg and Kolta, 2011).

Polysomnographic studies have shown a relationship between sleep micro-arousal and SB episodes, suggesting that autonomic/central nervous system activation are primary factors responsible for initiating SB (Kato *et al*., 2003). However, it has also been postulated that CPG neuronal networks responsible for chewing and swallowing during wakefulness remain active during sleep. The triggering of cortical masticatory areas, which results in RMMA, has been shown to not be exclusive to the genesis of rhythmic chewing and has also been observed to trigger swallowing, as noted in nearly 60% of SB episodes (Lavigne *et al*., 2003; Miyawaki *et al*., 2003). The observation that chewing RMMA is 50% faster than SB RMMA may be ascribed to the overall reduction of the drive to motor system occurring during sleep.

The current study has shown the lack of correlation between the paces of chewing RMMA and sleep RMMA. This observation does not support the hierarchical structure of the bruxism generator model and the notion that SB can be ascribed simply to a disruption of the chewing CPG (Lavigne and Montplaisir, 1995). It may be possible that there is a separate pace-generating mechanism for SB, which is independent from that of chewing. This suggestion, however, remains highly speculative, as the lack of correlation between natural chewing and spontaneous RMMA during sleep does not allow definitive conclusions on the relationship between the pace-generating mechanisms of chewing and SB. Furthermore, due to the small sample size investigated in the present study, the lack of correlation between the chewing pace and SB pace also needs to be interpreted with caution. It can be noted, however, that the correlation coefficient was very small and the plotted data were highly scattered. It is therefore unlikely that a future study performed in a larger sample size will yield a different result.

It has been reported that there is no gender predilection for SB (Bader and Lavigne, 2000; Glaros, 1981; Kato and Lavigne, 2010; Lavigne *et al*., 1999, 2008; Reding *et al*., 1966). The time–frequency features of chewing episodes in the natural environment have also been shown to have no gender differences (Nagasawa *et al*., 1997; Po *et al*., 2011). Thus, investigation of possible gender differences in the time–frequency features of SB was not a focus of interest in the present study.

Sleep bruxers recruited in our study were diagnosed according to the clinical criteria of American Association of Sleep Medicine, and EMG data were collected during a single night's recording. This may be a limitation of the present investigation, as the intensity of SB can fluctuate markedly over time (Lobbezoo *et al*., 2008), with significant variability in some individuals (Lavigne *et al*., 2001a).

Although we followed the recommendation that SB should be diagnosed along multiple axes, namely questionnaires, an oral history-taking (including a bed partner's report of grinding sounds) and an extra- and intra-oral inspection for clinical signs of bruxism, we may have over- or underestimated the severity of SB for any given participant. We wish to emphasize, however, that the main focus of our study was to compare the time–frequency features of chewing and sleep RMMA using a within-subject study design, regardless of the SB severity.

The portable EMG recorders utilized in this study interfere minimally with chewing functions performed in the natural environment. However, our participants may still have been aware of the surface EMG electrodes and portable EMG recorder. This might have altered the participants' environments, affecting their habitual activities. It must also be pointed out that artefacts may have been recorded during data collection. Although the accuracy of the algorithm used was rather high, the occurrence of false-positives due to quasi-rhythmic activities such as coughing may still have influenced the results (Farella *et al*., 2009).

Finally, our participants were volunteers, not sampled randomly, and the age distribution of our study population was concentrated at adults in their 20 s. Given that the prevalence of SB has been reported to decrease with age

(Bader and Lavigne, 2000; Lavigne *et al*., 1999; Reding *et al*., 1966), the results of our sample may not be representative of the general population.

In conclusion, the findings of this study indicate that the contraction episodes of chewing and SB have different time–frequency features. Chewing episodes are more powerful and longer-lasting than SB episodes. The pace of chewing activity is unrelated to that of SB. Despite both activities being accompanied by rhythmic jaw contractions, the pace-generating mechanism of sleep bruxism may be independent from that of chewing.

CONFLICTS OF INTEREST

No conflicts of interest declared.

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SUPPORTING INFORMATION

Additional Supporting Information may be found in the online version of this article:

Data S1. Comparison between the rhythmic jaw contractions occuring during sleep and while chewing.