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RESEARCH ARTICLE

Associations between signs of sleep bruxism and insomnia: A polysomnographic study

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Summary

Sleep bruxism (SB) is a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible. Sleep bruxism has been linked with insomnia symptoms. Moreover, it has been suggested that there is a positive association between distress and the occurrence of sleep bruxism. However, the occurrence of sleep bruxism and its association with distress have not been studied in patients with insomnia. Therefore, we hypothesised that: (1) the occurrence of sleep bruxism is higher in patients with insomnia than in healthy controls; and (2) the occurrence of sleep bruxism in insomnia patients with moderate to high distress (IMHD) is higher than that in insomnia patients with slight distress (ISD). A total of 44 controls (34 females, 10 males, mean ± SD age = 46.8 ± 14.4 years) and 42 participants with insomnia (35 females, 7 males, mean ± SD age = 51.3 ± 12.1 years) were enrolled in this study. Among 42 participants with insomnia, 20 participants were subtyped as IMHD, 17 participants as ISD. Another five participants were not subtyped due to insufficient information. Group differences in

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rhythmic masticatory muscle activity (RMMA), a biomarker of sleep bruxism, were evaluated with Mann–Whitney U tests. The medians and interquartile ranges of the RMMA indices were 0.8|1.8|3.3 in controls, 1.1|1.6|2.3 in IMHD and 1.2|1.9|2.9 in ISD. There was no significant difference in the RMMA index, neither between participants with insomnia and controls ($P = 0.514$) nor between IMHD versus ISD ($P = 0.270$). The occurrence of RMMA indicators of possible sleep bruxism is not significantly different between individuals with insomnia and controls, nor between IMHD versus ISD.

KEYWORDS
distress, insomnia, RMMA, sleep arousal, sleep bruxism

1 | INTRODUCTION

Sleep bruxism (SB) is a repetitive jaw-muscle activity characterised by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible during sleep (Lobbezoo et al., 2013). In the case of a history or laboratory-based confirmation of these symptoms, a diagnosis of mild sleep bruxism may be present when at least 2 up to 4 episodes of rhythmic masticatory muscle activity (RMMA) per hour of sleep are present, while severe bruxism requires at least four episodes per hour (Carra et al., 2012, 2015). The aetiology of SB is multifactorial, including biological factors, psychosocial factors, and lifestyle factors. The potential negative consequences of sleep bruxism described in the literature are, amongst others, headache upon awakening, temporomandibular pain complaints, severe mechanical tooth wear, and tooth/dental restoration/implant fractures/failures (Manfredini et al., 2014; Manfredini & Lobbezoo, 2010). The prevalence of sleep bruxism is around 12.8% in a general population and decreases with age (Manfredini et al., 2013). There are many sleep-related disorders that have been suggested to be associated with SB, such as obstructive sleep apnea, periodic limb movement disorders, and insomnia (Kuang et al., 2022).

Insomnia is one of the most common sleep disorders, with a prevalence of approximately 10% (Morin et al., 2015). Insomnia is found to be associated with female gender, and older age (Aernout et al., 2021). There are several studies suggesting that insomnia by self-report is associated with sleep bruxism (Maluly et al., 2013; Saletu et al., 2010). In addition, a study by Khoury et al. (Khoury et al., 2016) suggested that it is more likely that patients with sleep bruxism report difficulty maintaining sleep than patients without sleep bruxism, which is one of the symptoms of insomnia. Furthermore, a recent large-scale polysomnographic study by Maluly et al. (Maluly et al., 2020) found that participants with sleep bruxism are more likely to report insomnia symptoms than those without sleep bruxism. Even though the specific aetiology of sleep bruxism is still unknown, many studies found it to be related to the occurrence of arousal (Kato et al., 2001; Lavigne et al., 2007). Likewise, all models of insomnia (i.e., physiologic, behavioural, cognitive, and neurocognitive) assume some degree of hyperarousal as a risk factor for the onset of insomnia (Bonnet & Arand, 2010; Lichstein et al., 2017).

Blanken et al. (Blanken et al., 2019) suggested that insomnia could be divided into five subtypes with high consistency over 4.8 years using latent class analysis, including highly distressed, moderately distressed but reward sensitive, moderately distressed and reward insensitive, slightly distressed with high reactivity, and slightly distressed with low reactivity. More specifically, they have demonstrated that insomnia patients with moderate to high distress showed higher pre-sleep arousal than controls, while insomnia participants with slight distress showed no difference. Regarding sleep bruxism, many studies (van Selms et al., 2020; Winocur et al., 2011) suggested that based on self-report, sleep bruxism is positively associated with emotional stress. Furthermore, the positive association between sleep bruxism and stress is supported by the findings that the levels of indicators of stress, namely, salivary cortisol, and catecholamines (Kaidonis et al., 2021; Vedhara et al., 2003), were elevated in patients with sleep bruxism (Flueraşu et al., 2021; Seraidarian et al., 2009).

Even though SB has been found to be positively associated with insomnia in the general population using questionnaires, SB has not been assessed objectively in a large population with insomnia. From a clinical point of view, a higher occurrence of SB in insomnia population than in controls suggests that an increase of awareness of physicians on this outcome is needed, and that they may refer their patients with insomnia to their dentist when negative consequences of sleep bruxism are present. We hypothesised for this study that (1) the occurrence of RMMA indicators of possible sleep bruxism is higher in individuals with insomnia than in controls; (2) the occurrence of RMMA indicators of possible sleep bruxism in insomnia individuals with moderate to high distress is higher than that in insomnia individuals with slight distress. Our aims were to (1) to determine the difference in occurrences of RMMA indicators of possible sleep bruxism between participants with insomnia and controls; (2) to determine the difference in occurrences of RMMA indicators of possible sleep bruxism between insomnia participants with moderate to high distress and insomnia participants with slight distress.

2 | MATERIALS AND METHODS

This study is a secondary analysis of the data collected in the prospective study by Christensen et al. (Christensen et al., 2019) and Wei et al. (Wei et al., 2017), which aimed to reveal group differences regarding sleep electroencephalography (EEG) between participants with insomnia and controls.

2.1 | Participants

Participants were enrolled by advertisement and via the Netherlands Sleep Registry website (NSR (Benjamins et al., 2017)). Participants were then screened by a telephone survey and invited to attend a face-to-face structured interview with a certified sleep specialist. Screening also included the Insomnia Severity Index (ISI) (Bastien et al., 2001). The ISI comprises seven items assessing the severity of sleep onset and sleep maintenance difficulties (both nocturnal and early morning awakenings), satisfaction with current sleep pattern, interference with daily functioning, noticeability of impairment attributed to the sleep problem, and the degree of distress or concern caused by the sleep problem. Each item is rated on a 0–4 scale and the total score ranges from 0 to 28. A higher score suggests more severe insomnia (Bastien et al., 2001). There were 44 controls (34 females, 10 males, mean ± SD age = 46.8 ± 14.4 years) and 42 participants with insomnia (35 females, 7 males, 51.3 ± 12.1 [mean ± SD] years) enrolled in this study. The diagnostic criteria for insomnia disorder, in accordance with the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) (American Psychiatric Association, 2013) and the International Classification of Sleep Disorders third edition (ICSD-3) (American Academy of Sleep Medicine, 2014), were used to select the insomnia group. Among the enrolled participants with insomnia, using the information gathered by questionnaires (see below, paragraph "Subtyping of the participants with insomnia"), 20 participants were subtyped as having moderate to high distress (18 females, 2 males, 47.3 ± 14.3 years) and 17 participants were subtyped as having slight distress (15 females, 2 males, 56.9 ± 7.8 years), and five participants had insufficient information to be subtyped.

The control group consisted of age- and gender-matched volunteers reporting no sleep complaints during telephone survey and the face-to-face interview and were further confirmed by an ISI score <8. The exclusion criteria were: (1) diagnosed sleep apnea, restless legs syndrome, narcolepsy, or other somatic, neurological, or psychiatric disorders; (2) use of sleep medications within the past 2 months up to and including the recording days; (3) overt shifted or irregular sleep–wake rhythms, assessed using 1 week of actigraphy (Actiwatch AW4, Cambridge Neurotechnology Ltd, Cambridge, UK or GENEActiv Sleep, Activinsights Ltd, Kimbolton, UK) supplemented by sleep diaries. The Medical Ethical Committee of the Academic Medical Center of Amsterdam and the Central Committee on Research Involving Human Subjects approved this study (#09.17.1396). All participants signed the written informed consent.

2.2 | Subtyping of participants with insomnia

The participants with insomnia were subtyped following the recently identified subtypes (Blanken et al., 2019). These subtypes were identified using latent class analysis including 19 different questionnaires spanning different non-sleep characteristics, such as life history, personality, mood, happiness, that have all been shown to be of relevance for insomnia (Benjamins et al., 2017).

The score of each questionnaire was used to represent one characteristic. Subsequently, the patterns of the characteristics were used to determine the subtype of the participants with insomnia. There are five subtypes of insomnia including highly distressed, moderately distressed but reward sensitive, moderately distressed and reward insensitive, slightly distressed with high reactivity, and slightly distressed with low reactivity. All the participants who were diagnosed with insomnia were assigned one subtype. For the complete subtyping procedures and validation, please refer to Blanken et al. (Blanken et al., 2019)

2.3 | Procedure and polysomnographic recording

Participants underwent two nights PSG in Netherlands Institute of Neuroscience (NIN). The first night was only intended for habituation. The second night was used for further analysis. For both nights, the PSG included signals of electrooculogram (EOG), electroencephalogram (EEG), electrocardiogram (ECG), electromyogram (EMG) channels on the bilateral masseter, bilateral temporalis, and mentalis using a 256-electrode Hydrocel Geodesic Sensor net connected to a Net Amps 300 amplifier (Electrical Geodesics Inc., Eugene, OR, USA). The signals were further processed using MEEGPIPE toolbox (GitHub-Meegpipe, 2014) and EEGLAB (Delorme & Makeig, 2004) in Matlab R2014a (The MathWorks Inc., Natick, MA, USA). Regarding the detailed data processing procedures please refer to the previously published article (Christensen et al., 2019).

2.4 | Analysis of polysomnographic recordings

The analysis of all the PSG recordings consisted of two parts, namely a sleep analysis and a bruxism analysis. The scoring of the sleep stages has been performed by trained scorers (JR, RW) according to the American Academy of Sleep Medicine (AASM) Manual for the Scoring of Sleep and Associated Events (Berry et al., 2020).

The analysis of bruxism and arousal related events, was performed by a trained scorer (BK) based on well-accepted criteria published by Carra et al. (Carra et al., 2015) and AASM manual for the Scoring of Sleep and Associated Events (Berry et al., 2020) at the

TABLE 1 Demographics, sleep, and EMG variables of participants with insomnia and controls

Outcome variables	Insomnia participants ($n = 42$)	Controls ($n = 44$)	Test statistic[a]	p
Demographics				
Age	45.0|54.4|60.3	35.5|46.2|60.7	$Z = -1.443$	0.149
Gender	35F, 7M	34F, 10 M	$\chi^2 = -0.498$	0.481
Sleep profiles				
TST (h)	6.2|6.8|7.6	7.0|7.4|7.8	$Z = -2.536$	0.011*
N1 (%)	4.0|8.3|12.2	3.0|4.9|6.5	$Z = -3.266$	0.001*
N2 (%)	44.4 ± 13.2	43.8 ± 10.6	$T = 0.240$	0.811
N3 (%)	22.1 ± 12.0	25.7 ± 9.8	$T = -1.519$	0.133
REM (%)	24.2 ± 12.8	25.4 ± 6.8	$T = -0.573$	0.569
SE (%)	81.8|90.0|95.0	89.0|93.0|95.8	$Z = -1.983$	0.047*
SL (min)	6.0|15.3|23.6	8.1|15.5|31.5	$Z = -0.860$	0.390
WASO (min)	15.0|26.5|67.4	7.3|16.5|29.1	$Z = -2.951$	0.003*
Interruption index[b]	4.9|5.9|7.4	3.8|5.1|6.1	$Z = -2.566$	0.010*
AI (events/h)	3.7|4.5|5.9	3.1|4.0|5.3	$Z = -1.582$	0.114
EMG parameters				
RMMA index	1.1|1.5|2.4	0.8|1.8|3.3	$Z = -0.653$	0.514
OA index	2.6|3.6|5.1	1.7|2.8|4.3	$Z = -1.586$	0.113
OMA index	1.1|1.5|2.4	1.2|2.0|2.8	$Z = -0.597$	0.551

Note: Mean value and standard deviations are provided for normally distributed variables, percentiles (25%|median|75%) are provided for not normally distributed variables.

[a]T = Test statistic of independent sample t-test, Z = test statistic of Mann–Whitney U test, χ^2 = chi − square test.

[b]Interruption index was calculated as $\frac{\text{number of sleep arousals} + \text{number of bouts of wakefulness}}{\text{total sleep time in hours}}$.

*A $p < 0.05$ is considered statistically significant.Abbreviation: AI, arousal index; OA, orofacial activity; OMA, other muscle activity; RMMA, rhythmic masticatory muscle activity; SE, sleep efficiency; SL, sleep latency; TST, total sleep time; WASO: wake after sleep onset.

Academic Centre for Dentistry Amsterdam (ACTA), using the software RemLogic (Natus®, Middleton, WI, USA).

2.5 | Outcome variables

The primary outcome variable for both aims was the RMMA index calculated as RMMA episodes per hour of sleep. An RMMA episode can consist of different types of RMMA bursts, i.e., phasic, tonic, and mixed RMMA. To qualify as an RMMA burst, the mean EMG amplitude must increase to two times the baseline EMG amplitude on at least three out of four EMG channels, monitoring bilateral masseter and bilateral temporalis. A phasic RMMA burst lasts 0.25–2 s. A tonic RMMA burst has a duration of at least 2 s. A phasic RMMA episode has at least three phasic bursts. A tonic RMMA episode has at least one tonic burst. A mixed RMMA episode has at least one phasic and one tonic burst. A time interval of at least 3 s of baseline EMG must occur between different RMMA episodes.

The secondary outcome variables included standard sleep descriptives, including total sleep time (TST) in hours, percentages of stage N1, N2, N3, and REM, sleep efficiency (SE), wake after sleep onset (WASO) in minutes, and interruption index. The interruption index was adapted from Wassing et al. (Wassing et al., 2019), which was calculated by the sum of the number of sleep arousals and the number of bouts of wakefulness and divided by the total sleep time in hours. If a masticatory muscle activity episode does not meet the criteria for RMMA, it was named as orofacial activity (OA), which typically represents coughing, yawning, chewing, swallowing and the movement of the lips and tongue during sleep. Other muscular activity (OMA) was defined as major body movement, involving the masticatory, facial, and neck muscles (Carra et al., 2015).The secondary outcome variables also include masticatory muscle activity related variables, namely OA index, and OMA index (number of episodes per hour of sleep).

2.6 | Statistical analysis

The normality of the data was tested using Shapiro–Wilk tests and histograms were also drawn to offer visual distribution of the data.

Group differences in the proportion of females and males were tested by X^2 tests, and age differences by Mann–Whitney U tests. Group differences in sleep and masticatory muscle activity related variables were evaluated using independent sample t-tests for normally distributed data, or Mann–Whitney U tests for non-normally distributed data. All the above-mentioned tests were performed using SPSS 26 (IBM, Chicago, IL, USA) and differences were considered significant when the p value was <0.05.

TABLE 2 Demographics, sleep, and EMG variables of insomnia participants with slight distress and insomnia participants with moderate to high distress

Outcome variables	Insomnia participants with slight distress ($n = 17$)	Insomnia participants with moderate to high distress ($n = 20$)	Test statistic[a]	p
Demographics				
Age	50.7|56.7|62.1	31.6|50.8|59.4	$Z = -1.859$	0.063
Gender	15F, 2M	18F, 2M	$\chi^2 = 0.030$	0.863
Sleep profiles				
TST (h)	6.9 ± 0.8	6.6 ± 1.0	$T = 0.925$	0.361
N1 (%)	7.2 ± 4.2	10.5 ± 6.8	$T = -1.730$	0.092
N2 (%)	35.4|46.4|61.1	35.2|39.5|53.2	$Z = 1.128$	0.270
N3 (%)	18.9 ± 13.5	25.6 ± 10.3	$T = -1.699$	0.098
REM (%)	25.6 ± 13.9	21.2 ± 11.3	$T = -1.069$	0.292
SE (%)	79.5|85.0|94.0	81.8|90.0|94.8	$Z = -0.580$	0.562
SL (min)	6.8|15.0|17.8	5.3|16.8|31.6	$Z = -0.884$	0.377
WASO (min)	17.0|49.5|87.5	14.6|26.5|63.3	$Z = -0.914$	0.373
Interruption index[b]	5.2|5.9|6.8	4.4|6.2|7.8	$Z = -0.061$	0.951
AI (events/h)	4.2|4.4|5.9	3.1|4.5|5.8	$Z = -0.565$	0.572
EMG parameters				
RMMA index	1.3|1.9|2.9	1.1|1.6|2.3	$Z = -1.114$	0.265
OA index	2.7|3.6|6.0	2.1|3.3|5.0	$Z = 0.368$	0.373
OMA index	1.0|1.4|2.1	1.1|1.8|2.8	$Z = 0.321$	0.326

Note: Mean value and standard deviations are provided for normally distributed variables; percentiles (25%|median|75%) are provided for not normally distributed variables.

[a]T = test statistic of independent sample t-test, Z = test statistic of Mann–Whitney U test, χ^2 = chi − square test.

[b]Interruption index was calculated as $\frac{\text{number of sleep arousals} + \text{number of bouts of wakefulness}}{\text{total sleep time in hours.}}$.

Abbreviation: AI, arousal index; OA, orofacial activity; OMA, other muscle activity; RMMA, rhythmic masticatory muscle activity; SE, sleep efficiency; TST, total sleep time; WASO, wake after sleep onset.

3 | RESULTS

3.1 | Demographics

There was no significant difference in age and gender, neither between insomnia participants and controls ($p = 0.149$ and $p = 0.481$, respectively), nor between insomnia participants with moderate to high distress and insomnia participants with slight distress ($p = 0.063$ and $p = 0.863$, respectively), see Tables 1 and 2.

3.2 | Primary outcome variable

Tables 1 and 2 show the medians, 25% and 75% percentiles for RMMA indices for controls, for all participants with insomnia, for participants with insomnia of a moderate to high distress subtype, and for participants with insomnia of slight distress subtype. There was no significant difference in RMMA index, neither between insomnia participants and controls ($p = 0.514$), nor between insomnia subtypes ($p = 0.265$). The range of RMMA observed suggested mostly an absence or at worst only mild sleep bruxism in the population studied. When using a RMMA index higher than 2 events per hour as the cutoff (Carra et al., 2012), 20 out of 44 (45.5%) participants in the control group had signs that could indicate a diagnosis of mild SB, while 14 of 42 (33.3%) participants with insomnia had these signs. Further, 7 of 17 (45.5%) insomnia participants with slight distress had these signs, compared with 7 of 20 (35%) insomnia participants with moderate to high distress.

3.3 | Secondary outcome variables

The sleep profiles comparing insomnia and controls are shown in Table 1. The TST and SE were significantly lower in individuals with insomnia than in controls ($p = 0.011$ and $p = 0.047$, respectively), while the percentage of stage N1, WASO, and interruption index were significantly higher in individuals with insomnia than in controls ($p = 0.001$, $p = 0.003$ and $p = 0.010$, respectively). There was no group difference in the arousal index between both groups ($p = 0.114$, Table 1). Table 1 shows no group differences in the OA index, nor in the OMA index ($p = 0.113$ and 0.551, respectively).

The sleep profiles comparing insomnia subtypes are shown in Table 2. None of the sleep-related variables differed significantly

($0.062 < p < 0.951$). Table 2 shows no subtype differences in the OA index, nor in the OMA index ($p = 0.373$ and 0.326, respectively).

4 | DISCUSSION

Our study is the first to compare the occurrences of RMMA indicators of possible sleep bruxism objectively between participants with insomnia and controls. Against our hypotheses, occurrences of RMMA indicators of possible SB did not differ significantly between participants with insomnia and controls, nor between insomnia subtypes characterised by either moderate to high distress or slight distress.

4.1 | Primary outcome variable

The results of this study are in line with the findings of Blanken et al. (Blanken et al., 2019) who reported no significant difference regarding sleep bruxism prevalence between controls and patients with insomnia in a large sample study carried out using questionnaires and interviews. Maluly et al. (Maluly et al., 2013) previously suggested that SB was significantly associated with self-reported insomnia. One possible reason why our result was different from the results of previous questionnaire studies (Ahlberg et al., 2008; Maluly et al., 2013) could be related to the definition of insomnia. In previous studies (Ahlberg et al., 2008; Maluly et al., 2013), insomnia was assessed with questionnaires. In our study, the diagnosis of insomnia included interviews by a certified sleep specialist following the criteria set in DSM-5 and ICSD-3. In both guidelines, the insomnia diagnosis must fulfil the requirement that "the sleep/wake difficulty is not better explained by another sleep disorder". Consequently, it is possible that for previous questionnaire studies (Ahlberg et al., 2008; Maluly et al., 2013), the sleep/wake difficulties that participants self-reported were related to other SB-related disorders, namely obstructive sleep apnea (OSA) or restless legs syndrome (RLS), which have both been found to be related to insomnia (Hosoya et al., 2014; Saletu et al., 2010).

Our study showed that there is no significant difference in the occurrence of RMMA indicators of possible sleep bruxism between subtypes of insomnia with moderate to high distress and subtypes with slight distress. Our result is in accordance with Blanken et al. (Blanken et al., 2019) suggesting that there is no significant difference of sleep bruxism prevalence among insomnia subtypes with different distress levels. Given that many studies showed that sleep bruxism is associated with emotional stress (van Selms et al., 2020; Winocur et al., 2011), there could be several possible reasons for the different results between these studies and ours. According to the American Psychology Association Dictionary of Psychology (VandenBos, 2015), stress is defined as the physiological or psychological response to internal or external stressors, while distress is defined as the negative stress response, often involving negative affect and physiological reactivity. Thus, the concept of stress is broader than that of distress. While sleep bruxism is shown to be associated with stress (van Selms et al., 2020; Winocur et al., 2011), the association between sleep bruxism and distress has been reported to be relatively weak (Bayar et al., 2012; Huhtela et al., 2021). Although distress and stress are closely related, there is no clear distinction between distress and stress in any of the above studies. Therefore, future studies should further investigate the sleep bruxism occurrence using polysomnography among participants with different distress levels in the insomnia population.

4.2 | Secondary outcome variables

In line with the finding that interrupting wakefulness during sleep is an important characteristic of insomnia (Wei et al., 2017), it was found that participants with insomnia have significantly higher interruption index than controls. However, the arousal index was not significantly different between participants with insomnia and controls in our study. Wassing et al. (Wassing et al., 2019) also found that participants with insomnia have only significantly higher arousal index during REM than normal sleepers. RMMA episodes occur more frequently during NREM sleep stages N1 and N2 (Carra et al., 2012), which are often associated with arousals (Hosoya et al., 2014; Sjöholm et al., 2000), while muscle atonia is a typical feature of REM sleep (Svensson et al., 2017). Thus, it is unlikely that the arousal difference during REM could result in a difference of RMMA index between individuals with insomnia and controls. Possibly, separating arousals and transitions to wakefulness is sub-optimal in characterising the sleep instability in insomnia. Sleep bruxism can only be scored during sleep, not during the interrupting epochs of wakefulness, which could be another reason why no significant difference in RMMA index was found between participants with insomnia and controls.

4.3 | Strengths and limitations

Our study is a secondary analysis of the data collected by previous studies. Therefore, the selection of participants may be biased. Even though insomnia has been shown to be more prevalent in females (Aernout et al., 2021), around 80% of our participants in both insomnia group and control group were females, while a recent large scale multi-center study showed that the prevalence rate of insomnia is 9.9% among males and 12.5% among females in the general population (Aernout et al., 2021). Moreover, Manfredini et al. (Manfredini et al., 2013) suggested that the prevalence of sleep bruxism decreases with aging. However, there were no significant differences in gender and age, neither between insomnia participants and controls, nor between insomnia participants with moderate to high distress and insomnia participants with slight distress. Thus, the influence of gender and age on the comparisons between different groups may be negligible.

The medians for the RMMA indices of the two groups (insomnia participants and controls) and the two subgroups within insomnia participants (insomnia participants with moderate to high distress and insomnia participants with slight distress), were around 1.5 events per

hour. When taking into consideration that a RMMA index at least two events per hour may indicate bruxism (Carra et al., 2015), the RMMA indices of both groups and subgroups were relatively low. Moreover, the RMMA index reported here is likely to be overestimated because of the lack of concurrent audio-video data (Carra et al., 2012, 2015). The generally low frequency of RMMA index of this particular population could potentially make the influence of insomnia and distress on RMMA index difficult to manifest.

Despite the relatively low RMMA indices for both the insomnia group and controls, when using RMMA index at least two events per hour as the cut-off in research settings, more than a third of the participants of both groups could be diagnosed with sleep bruxism. Even though the RMMA index cut-off of at least two events per hour for diagnosing sleep bruxism has been well-established (Carra et al., 2012), the RMMA index cut-off has no links to the clinical phenomenon of sleep bruxism and its consequences (Manfredini et al., 2019), including masseter hypertrophy, indentations on the tongue or lips, or mechanical dental wear, etc. (Lobbezoo et al., 2018). Future studies should work with continuous measurements of sleep bruxism rather than with cut-off values (Manfredini et al., 2019).

Raphael et al. (Raphael et al., 2015) reported that the concordance rate between sleep bruxism diagnosed by self-report and sleep bruxism diagnosed by polysomnography is generally low, with large variability in different circumstances. Lobbezoo et al. (Lobbezoo et al., 2018) speculated that a self-reported sleep bruxism diagnosis might reflect distress rather than actual masticatory muscle activity. To our best knowledge, our study is the first to report on the occurrence of RMMA indicators of possible sleep bruxism in individuals with insomnia using polysomnography and to compare the occurrence with that in controls. The utilisation of polysomnography, an objective assessment tool, could be another reason why our results are different from the results of previous study (Ahlberg et al., 2008) using questionnaires to assess sleep bruxism.

Within the limitations of this study, we concluded that the occurrence of RMMA indicators of possible sleep bruxism is not significantly different between individuals with insomnia and controls. In addition, the occurrence of RMMA indicators of possible sleep bruxism is not significantly different between insomnia individuals with moderate to high distress and insomnia individuals with slight distress.

AUTHOR CONTRIBUTIONS

Initial study design: Frank Lobbezoo, Eus J.W. Van Someren, Ghizlane Aarab; study protocol: Frank Lobbezoo, Eus J.W. Van Someren, Ghizlane Aarab; data collection and analysis: Rick Wassing, Jennifer R. Ramautar; data analysis and first draft of the manuscript: Boyuan Kuang; critical review of analysis results and editing of the manuscript: all authors; final approval of the manuscript: all authors.

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CONFLICT OF INTEREST

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DATA AVAILABILITY STATEMENT

The collection, storage and analysis of participants' information were approved by the Medical Ethical Committee of the Academic Medical Center of Amsterdam and the Central Committee on Research Involving Human Subjects (#09.17.1396). Detailed analysis results pertaining to this study are available from the corresponding author upon request.

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