



UvA-DARE (Digital Academic Repository)

Sleep bruxism

Associations and comorbid conditions

Chattratjai, T.

Publication date

2024

[Link to publication](#)

Citation for published version (APA):

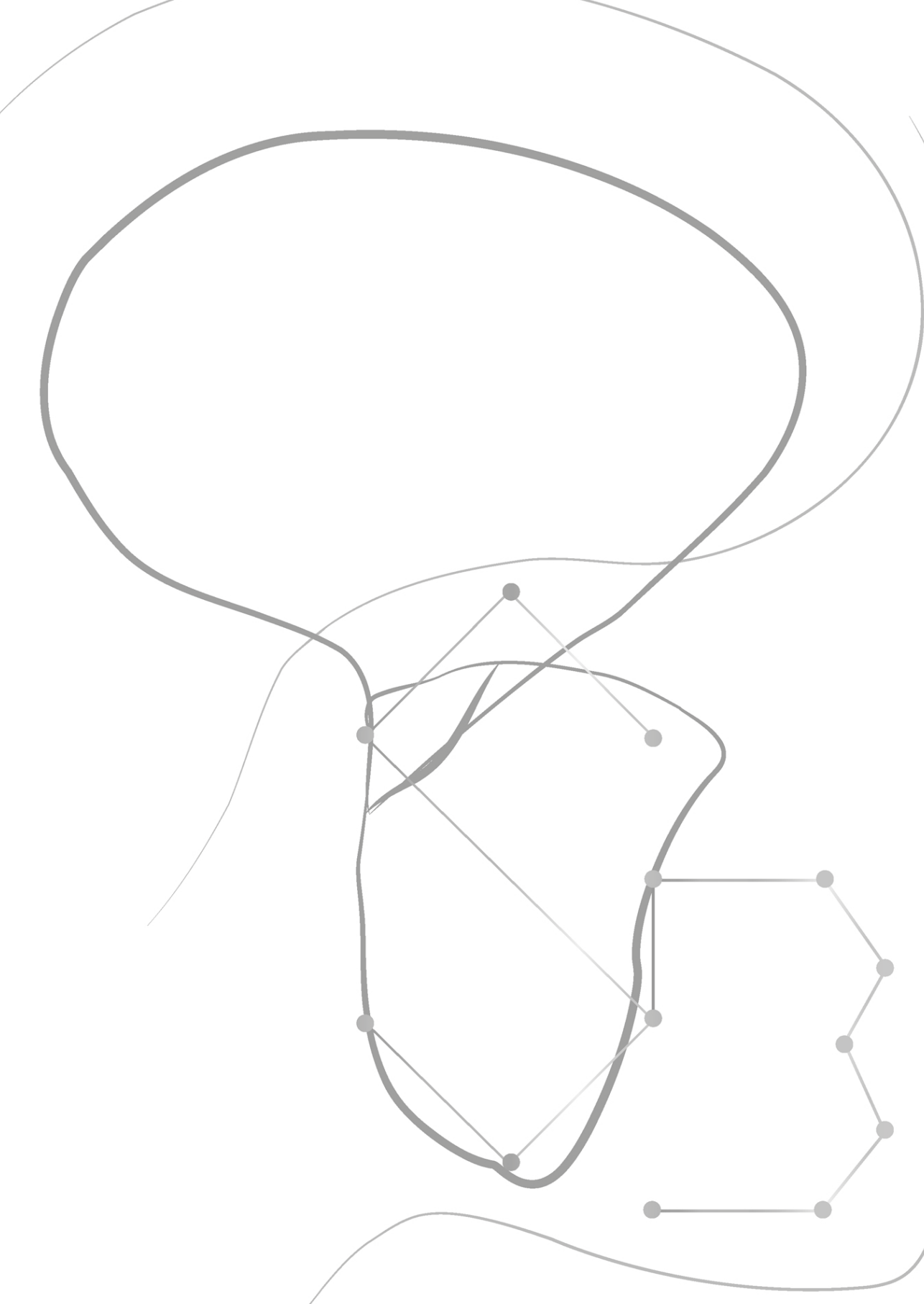
Chattratjai, T. (2024). *Sleep bruxism: Associations and comorbid conditions*. [Thesis, fully internal, Universiteit van Amsterdam].

General rights

It is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), other than for strictly personal, individual use, unless the work is under an open content license (like Creative Commons).

Disclaimer/Complaints regulations

If you believe that digital publication of certain material infringes any of your rights or (privacy) interests, please let the Library know, stating your reasons. In case of a legitimate complaint, the Library will make the material inaccessible and/or remove it from the website. Please Ask the Library: <https://uba.uva.nl/en/contact>, or a letter to: Library of the University of Amsterdam, Secretariat, P.O. Box 19185, 1000 GD Amsterdam, The Netherlands. You will be contacted as soon as possible.





CHAPTER 7

GENERAL DISCUSSION

The general aim of this thesis was to assess the associations of sleep bruxism (SB) on the one hand, and awake bruxism (AB), psychological factors, temporomandibular disorders (TMD), and insomnia on the other hand. We used self-report and instrumental techniques to establish the presence of bruxism. An interesting point of this thesis is that we studied SB in different populations, i.e., the American, Thai, and Dutch TMD-patient populations, and that the results obtained from these populations show the same trends. Amongst others, we found that objectively assessed SB and AB are not associated across individuals (**chapter 2**). Further, SB is conditionally independent from AB and TMD (**chapter 3**) and the improvement of SB is not associated with TMD treatment (**chapter 4**). In contrast, we did find a direct association between AB and TMD (**chapters 3 and 4**). Besides, we investigated the association between SB and insomnia in a specific population sample, i.e., participants who are interested in sleep problems, as well as in a general population sample. We investigated the association of SB, assessed with self-report and polysomnography (PSG), with insomnia and psychosocial factors, and we determined whether the associations of self-reported SB and PSG-confirmed SB are similar. We found that only self-reported SB, i.e., possible SB, was associated with insomnia and psychosocial factors (**chapters 5 and 6**). In contrast, PSG-confirmed SB, i.e., definite SB, was not associated with either insomnia or psychosocial factors. In the present chapter, we discuss our findings, clinical implications, and future research from three perspectives: the associations between SB and AB; the associations between SB, AB, and TMD; and the association between SB and sleep disorders. Finally, we provide a general conclusion.

SB AND AB

The results of this thesis support the idea that SB and AB are different entities.¹ **Chapter 2** shows that the main characteristic of SB is grinding activity, while that of AB is mainly clenching activity. SB and AB, assessed with self-report in **chapter 3** and with electromyography (EMG) and/or PSG in **chapter 2**, are not associated in the same individual. In **chapter 2**, we showed that masticatory muscle activity during SB events and the event rates (i.e., the repetitive masticatory muscle activity (RMMA) episodes that were standardized as events per hour) of SB are not associated with those of AB, neither at rest nor during stress-related activity. Only masticatory muscle activity during AB events and the event rates of AB at rest are associated with those of AB during stress-related activity. Using a network analysis, **chapter 3** shows no direct association between possible SB and AB. Importantly, SB and AB are not only different in terms of their characteristics but also have different associations with psychosocial factors. This result is consistent among different populations (**chapters 2 and 3**). AB

tends to increase during a stress-induced condition in **chapter 2**, and it is associated with stress in **chapter 3**. In contrast, in **chapter 3**, SB was not directly associated to psychosocial factors. Additionally, we cannot find any significant association between SB and psychosocial factors in **chapter 4**. The combined evidence summarized above corroborates the notion that SB and AB are different entities.

In this thesis, we assessed SB with two different approaches: self-report and PSG. We used the cut-off criteria according to Rompré et al.² to assess definite SB in **chapters 2 and 6**. PSG is a valuable assessment for SB physiology, and these cut-off criteria for scoring PSG data were constructed for the study of the physiologic characteristics of SB.³ However, definite SB seems to lack a link with its clinical consequences if the cut-off point is used (**chapter 6**). Using the cut-off point to determine the presence of SB, no conditional dependence association between definite SB and other factors like anxiety and depression was found in **chapter 6**, while direct associations of self-reported SB with anxiety and insomnia were found in **chapters 5 and 6**. In addition, **chapter 2** shows that some patients who did not have SB events above the cut-off point still have SB events. Fortunately, there are some promising developments in how to analyze the instrumentally obtained data of SB in a meaningful way.³ For example, Manfredini et al. (2019) and Lavigne et al. (2021) suggested the assessment of indicators relevant to support SB diagnosis and treatment decisions, such as age, sex, heart rates, sleep arousals, RMMA index, the duration of RMMA, background EMG, and comorbid conditions like TMD, tooth wear, and sleep conditions.^{3,4} Clearly, PSG scoring requires validation that provides consistency with clinical relevance.^{3,4} Furthermore, SB has a considerable night-to-night variability.⁵ Keeping that in mind, self-report has the advantage of long-term observation compared with the result from a single-night PSG. We therefore suggest that self-report remains a suitable technique for the monitoring of SB and its clinical relevance.

For future research, to measure SB and AB events, it is considered better to approach these events as continuous values rather than as the presence or absence of SB based on the cut-off point.⁴ In addition, the Standardized Tool for the Assessment of Bruxism (STAB)^{6,7}— a bruxism evaluation system that consists of the assessment domain, i.e., axis A, and factors and conditions related to bruxism, i.e., axis B— and BruxScreen⁸— a questionnaire and a clinical assessment form for SB and AB— should be implemented in future research to obtain standardized measurements. Besides, using these standardized tools can help us compare possible and probable SB and AB research across populations. As a result, we can incorporate data related with SB, AB, and their clinical relevance and comorbidity from different populations, and identify phenotyping of SB and AB regarding to these data. Consequently, we may establish

treatment options regarding to phenotyping of SB and AB.³ Additionally, more research in endotyping, i.e., the underlying biological mechanisms of SB and AB, is recommended to explain their phenotyping and help clinicians to deliver the most suitable treatments to their patients.

SB, AB, AND TEMPOROMANDIBULAR DISORDERS (TMD)

Even though **chapter 2** shows similar levels of SB and AB between TMD-pain cases and pain-free controls, network analysis in **chapter 3** shows that TMD pain is directly associated with possible AB but not with possible SB. In **chapter 4**, we studied the association of usual TMD management with the changes of possible SB and AB, including different activities of AB, i.e., awake grinding, awake clenching, and jaw bracing. To the best of our knowledge, no study has investigated the changes in different types of AB activity six weeks after receiving counselling without or with any other TMD treatment, in a TMD population. The management of TMD through counseling with other treatments, for a brief period is useful for possible AB (**chapter 4**). Patients who believe that oral behavior causes jaw pain and have high psychological distress tend to report higher AB frequency than those who do not believe that or have low psychological distress.⁹ So, AB may reflect psychosocial status, or the other way around. If this is actually the case, counselling including stress management may affect the changes in AB.

Surprisingly, possible SB is not associated with TMD or psychosocial factors, and usual TMD management for a brief period does not improve possible SB (**chapters 3 and 4**). **Chapters 3 and 4** used item 1 from the Oral Behavioral Checklist that asks how frequently one bruxes while asleep.¹⁰ SB may not be easily recognized compared with AB by the patient. Thus, some sleep bruxers may miss how frequently they brux during sleep. The report of grinding sounds from their sleep partners can increase the validity of sleep bruxing.¹¹ On the other hand, self-report can be used to screen non-bruxers as it has high specificity, but self-report is not a good indicator for identifying SB because of the low sensitivity.¹² Another assumption is that the main characteristic of AB, i.e., mainly clenching activity, may yield delayed-muscle onset soreness (DOMS), and this “repetitive, low-level, tonic activity” may thus play a role in the onset and progression of TMD.^{13,14} In contrast, SB is mainly grinding activity that lasts 0.2-2 seconds, which may not be long enough to induce DOMS and subsequently TMD.

This thesis confirms the biopsychosocial nature of TMD, which involves psychosocial factors like stress and oral behaviors like AB. It is suggested that AB is closely related to

TMD (see above). The management of TMD thus requires multidisciplinary approaches, whereby our findings suggest that we should focus on the management of AB. Even though the study in **chapter 4** measured the changes in SB and AB in a brief period after implementing usual TMD treatment, the results support the idea that counseling and physical therapy may influence the TMD-treatment effect and the change in AB. Therefore, increasing awareness of AB is recommended in the management of TMD. Although **chapter 4** did not find an association between AB and other psychosocial factors, **chapters 2** and **3** clearly show that AB is associated with stress. AB may play a role in stress coping, although stress coping strategies may vary between individuals.¹⁵ On the other hand, SB may co-occur with multiple factors, such as psychosocial factors and sleep-related conditions.^{6,7} SB is managed when there are severe negative consequences, e.g., extensive mechanical tooth wear and restoration failure, due to the presence of SB.⁷

Future research should use a continuum spectrum of SB activities (i.e., continuous values of SB parameters) to investigate the associations of the intensity, frequency, and duration of SB events with other factors, including AB, TMD, psychosocial factors, and other conditions. Only then, we may conclude that the intensity of SB is associated with TMD or that background EMG during sleep is associated with TMD pain.¹⁶ Moreover, longitudinal studies are needed to investigate the positive and negative consequences of SB, preferably together with a novel statistical method like network analysis that allows to distinguish direct from indirect effects. Due to such approach, we may discover possible direct associations between SB and other factors that have not been identified before, or we may find meaningful indirect associations between SB and other factors. Interestingly, a network model can be a patient education tool that provides insight into the importance of multidisciplinary approaches, i.e., dentistry, sleep medicine, physical therapy, and psychological treatment. Moreover, the network model can investigate the role of bridge symptoms, i.e., a group of symptoms that overlap between two disorders, such as the common symptoms of anxiety and depression.¹⁷ In this thesis, we defined potential bridge factors as factors that bridge indirect associations between factors. For instance, TMD pain, as described in **chapter 3**, is a bridge factor between AB, TMD dysfunction, and stress. Such bridge factors could potentially highlight treatment targets, as it suggests that treating the bridge factor may alleviate or affect the connecting factors. While this should be tested in practice, the network models may inform tailor-made treatment.

SB AND SLEEP DISORDERS

The association between possible SB and insomnia was found in both sleep-interested and general populations. A novel finding is that insomnia is associated with possible SB but not with definite SB (**chapter 5** and **6**). It supports the aforementioned recommendation that the cut-off criteria of definite SB should be considered in a continuum spectrum. Network analysis shows that psychosocial factors play a role in the connection between SB and insomnia. The tripartite association between anxiety, depression, and insomnia remains in both sleep-interested and general populations (**chapters 5** and **6**).

While the first part of this thesis could not demonstrate any association between SB and other factors like AB, TMD, and psychosocial factors, the second part of this thesis found an association between SB and insomnia as well as anxiety. It should be noted that, in the first part of this thesis, we used different questionnaires to measure stress as a psychosocial factor related to SB, AB, and TMD, while in the second part of this thesis, we assessed anxiety and depression as psychosocial factors for SB and insomnia. Consequently, it is not possible to compare psychosocial factors across studies. Network analysis in **chapters 5** and **6** shows direct and indirect associations between SB, insomnia, and psychosocial factors. Thus, psychosocial factors may be comorbid conditions of SB. Conversely, the regulation of serotonin and dopamine is associated with SB, insomnia, and psychosocial factors like anxiety and depression.^{18,19} Excessive serotonin, e.g., due to medication with selective serotonin reuptake inhibitors, can increase SB and AB.²⁰ More research on SB and neurotransmitters is needed to draw a definite conclusion about the association between SB and psychosocial factors. In addition, PSG studies are needed to investigate sleep arousal in patients with SB and insomnia, as to enable drawing a conclusion about the biological association between SB and insomnia.

A clinical implication of the above is that insomnia patients tend to be sensitive to SB events. When patients notice their SB, it may be a cue to further investigate insomnia and other sleep conditions. It emphasizes that various fields of expertise, i.e., sleep medicine, dentistry, and psychiatry, are needed to manage SB (in case of negative consequences) and other sleep disorders.

Since the definite diagnosis of insomnia requires a clinical assessment,²¹ future research is needed to investigate the association between SB on the one hand and definite insomnia and psychological status on the other hand. Finally, to identify whether psychosocial factors are associated with, or are only a comorbid condition of SB, it is suggested to study SB in patients with anxiety and/or depression.

GENERAL CONCLUSION

Different assessments ranging from self-report to instrumental techniques suggest different clinical consequences and associated conditions of SB. SB is not directly associated with psychosocial factors and TMD, while AB is associated with those factors. In addition, evidence is accumulating that SB and AB are different entities. Furthermore, possible SB connects with insomnia through psychosocial factors, but the associations between definite SB and insomnia as well as psychosocial factors could not be confirmed.

REFERENCES

1. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. *J Oral Rehabil.* 2018;45(11):837-844.
2. Rompre 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(9):837-842.
3. Lavigne G, Kato T, Herrero Babiloni A, et al. Research routes on improved sleep bruxism metrics: Toward a standardised approach. *J Sleep Res.* 2021:e13320.
4. Manfredini D, Ahlberg J, Wetselaar P, Svensson P, Lobbezoo F. The bruxism construct: From cut-off points to a continuum spectrum. *J Oral Rehabil.* 2019;46(11):991-997.
5. Ohlmann B, Bomicke W, Behnisch R, Rammelsberg P, Schmitter M. Variability of sleep bruxism-findings from consecutive nights of monitoring. *Clin Oral Investig.* 2022;26(4):3459-3466.
6. Manfredini D, Ahlberg J, Aarab G, et al. Towards a Standardized Tool for the Assessment of Bruxism (STAB)-Overview and general remarks of a multidimensional bruxism evaluation system. *J Oral Rehabil.* 2020;47(5):549-556.
7. Manfredini D, Ahlberg J, Aarab G, et al. Standardised Tool for the Assessment of Bruxism. *J Oral Rehabil.* 2023.
8. Lobbezoo F, Ahlberg J, Verhoeff MC, et al. The bruxism screener (BruxScreen): Development, pilot testing and face validity. *J Oral Rehabil.* 2023.
9. van Selms MKA, Thymi M, Lobbezoo F. Psychological distress and the belief that oral behaviours put a strain on the masticatory system in relation to the self-report of awake bruxism: Four scenarios. *J Oral Rehabil.* 2023.
10. van der Meulen MJ, Lobbezoo F, Aartman IH, Naeije M. Validity of the Oral Behaviours Checklist: correlations between OBC scores and intensity of facial pain. *J Oral Rehabil.* 2014;41(2):115-121.
11. Raphael KG, Janal MN, Sirois DA, et al. Validity of self-reported sleep bruxism among myofascial temporomandibular disorder patients and controls. *J Oral Rehabil.* 2015;42(10):751-758.
12. Casett E, Reus JC, Stuginski-Barbosa J, et al. Validity of different tools to assess sleep bruxism: a meta-analysis. *J Oral Rehabil.* 2017;44(9):722-734.
13. Koutris M, Lobbezoo F, Sumer NC, Atis ES, Turker KS, Naeije M. Is myofascial pain in temporomandibular disorder patients a manifestation of delayed-onset muscle soreness? *Clin J Pain.* 2013;29(8):712-716.
14. Bracci A, Lobbezoo F, Colonna A, et al. Research routes on awake bruxism metrics: implications of the updated bruxism definition and evaluation strategies. *J Oral Rehabil.* 2023.
15. Soto-Goñi XA, Alen F, Buiza-González L, et al. Adaptive Stress Coping in Awake Bruxism. *Front Neurol.* 2020;11:564431.
16. Raphael KG, Janal MN, Sirois DA, et al. Masticatory muscle sleep background electromyographic activity is elevated in myofascial temporomandibular disorder patients. *J Oral Rehabil.* 2013;40(12):883-891.
17. Cramer AO, Waldorp LJ, van der Maas HL, Borsboom D. Comorbidity: a network perspective. *Behav Brain Sci.* 2010;33(2-3):137-150; discussion 150-193.
18. Blake MJ, Trinder JA, Allen NB. Mechanisms underlying the association between insomnia, anxiety, and depression in adolescence: Implications for behavioral sleep interventions. *Clin Psychol Rev.* 2018;63:25-40.

19. Smardz J, Martynowicz H, Wojakowska A, et al. Lower serotonin levels in severe sleep bruxism and its association with sleep, heart rate, and body mass index. *J Oral Rehabil.* 2022;49(4):422-429.
20. de Baat C, Verhoeff M, Ahlberg J, et al. Medications and addictive substances potentially inducing or attenuating sleep bruxism and/or awake bruxism. *J Oral Rehabil.* 2021;48(3):343-354.
21. Schutte-Rodin S, Broch L, Buysse D, Dorsey C, Sateia M. Clinical Guideline for the Evaluation and Management of Chronic Insomnia in Adults. *Journal of Clinical Sleep Medicine.* 2008;04(05):487-504.