# Association Between the Amount of Alcohol Intake and Masseter Muscle Activity Levels Recorded During Sleep in Healthy Young Women

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***Purpose:*** The aim of this study was to determine if the amount of alcohol intake is associated with masseter muscle activity recorded during sleep. ***Materials and Methods:*** Sixty healthy young female subjects (average age, 23.0 ± 1.9 years; range, 21 to 32 years) participated in the study. Subjects were asked to perform 6 consecutive nightly masseter electromyography (EMG) recordings by using a portable EMG recording system in their homes. Using a minimum threshold criterion, which was set at 20% of the maximum voluntary contraction level, the total duration of muscle activity per hour of sleep was calculated. EMG data obtained on the first night were excluded from the analysis to avoid the first-night effect, and the data of the remaining 5 nights were averaged. Further, the subjects were asked to fill out questionnaires regarding their daily alcohol intake during the recording period. The total ethanol content of the consumed alcohol was calculated using a standard conversion table for alcoholic beverages. The ethanol concentration of each type of alcohol was multiplied by the reported amount consumed on each day, and the average value for 5 days was calculated. The EMG data were considered as a dependent variable, while the alcohol data were considered as an independent variable. Linear regression analysis was used to assess a possible association between these variables. ***Results:*** The subjects who did not consume alcohol during the recording period (n = 28) or who provided incomplete data sets as a result of missing data (n = 9) were excluded. The data of the remaining 23 subjects (n = 23) were exclusively analyzed. The result of this analysis revealed that the total ethanol content of the consumed alcohol was significantly and positively related to the EMG duration variable (coefficient = 0.51, 95% confidence interval: 0.20 to 0.82, adjusted $R^2$ = 0.33, $P < .01$). ***Conclusion:*** The results suggest that the amount of alcohol intake is substantially associated with masseter muscle activity levels during sleep in young women. *Int J Prosthodont 2007;20:251–255.*

Sleep bruxism is defined as "spasmodic grinding or clenching of teeth during sleep"[1,2] and is classified as a sleep-related movement disorder.[3] When sleep bruxism is frequent or performed with great force, it has the potential to cause a variety of pains and dysfunctional conditions in the orofacial region. Psychosocial factors,[4–8] pathophysiologic factors,[9–15] morphologic factors,[9,16–18] and the use of psychoactive drugs[19–21] have been suggested as possible risk factors for sleep bruxism. Among these factors, it is generally accepted that alcohol intake increases sleep bruxism; however, supporting evidence-based data are rather scarce.[19,20]

Based on the observation of 4 patients over a 4- to 12-month period, Hartman[22] initially suggested that alcohol aggravated sleep bruxism. In a later placebo-controlled crossover sleep laboratory–based polysomnographic (PSG) study, Hartman et al[23]

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administered 3 different alcohol doses plus a placebo on 4 separate nights to 16 subjects; however, a statistically significant dose-response relationship was not observed. Considering the significant daily variation in sleep bruxism, the result drawn from a single night of measurements should be interpreted with caution. In an epidemiologic study, Ohayon et al[4] found that bruxers reported drinking alcohol at bedtime more often than non–bruxers. However, there are certain limitations regarding the ability of subjects to be aware of their own behavior during sleep.[24] Further, the accurate ethanol content of the consumed alcohol was not available. In the interim, the alcohol-bruxism relationship could neither be proven nor refuted, resulting in much controversy.

To overcome these problems, recordings over multiple nights have been performed in the home environment by subjects using portable electromyography (EMG) recording devices.[25–31] Recently, a computer-assisted masseter muscle activity detection system has been developed, which allows high-resolution digital data collection and systematic discrimination of artifact signals.[32] In a preliminary study of the potential risk factors of sleep bruxism using the system described above, Rosa et al[33] found that the amount of alcohol intake was positively associated with masseter muscle activity levels during sleep. However, this association was weak and only valid in female subjects.

To verify and extend the results of that preliminary study, this study examined the possible relationship between alcohol intake and masseter muscle activity in female subjects. The null hypothesis of this study was that the amount of alcohol intake is not related to masseter activity levels during sleep in healthy young female adults.

## Materials and Methods

### *Subjects*

Subjects were recruited from the female students enrolled at Tokyo Medical and Dental University, Tokyo, Japan. A total of 63 consecutively listed female students (average age, 23.0 ± 1.9 years; range, 21 to 32 years) participated in the study. Each subject was provided with a full verbal description of the study, and those who elected to enroll signed a university-approved human subject consent form. Three subjects declined to participate because of time constraints. All subjects were in good physical health. None of the subjects used any prescription medication. Subjects who had acute dental disease or were receiving ongoing dental treatment were excluded. All subjects filled out a questionnaire, which asked if they were aware of bruxism behaviors. Some subjects were aware of clenching and sleep bruxism habits, but none had ever sought treatment or felt the need to seek treatment for this behavior. To encourage compliance, each subject was paid an amount equivalent to $40 after completion of all experimental procedures.

### *Alcohol Intake Questionnaire*

A questionnaire was administered to quantify the amount of alcohol consumed during the study period. The subjects reported details regarding the amount (mL) and type of alcohol (sake, wine, whiskey, beer, etc) consumed on each day before going to bed during the study period. The total ethanol content of the consumed alcohol was calculated using a standard conversion table for alcoholic beverages.[34] The ethanol concentration of each type of alcohol was multiplied by the reported amount of the alcohol consumed on that day, and the average value for 5 days, ie, days 2 to 6, was calculated (g).

### *Masseter Muscle EMG Recording*

Using a portable EMG recording system,[35] EMG signals from the right superficial masseter muscle were amplified and digitized at a sampling frequency of 200 Hz and stored on a personal computer for offline analysis. The subjects were instructed on how to use the device as well as the placement of the electrodes. They then took the recording system to their homes to perform nightly EMG recordings.

At the beginning of each recording night, the subjects performed 3 brief (2 seconds) maximum voluntary contractions (MVCs) in maximum intercuspation. Upon waking in the morning, the subjects were asked to record the following additional information in a sleep diary: the time between when they turned on the recorder and when they actually went to sleep; the time between when they woke up and when they turned off the recorder; and the number, duration, and reason for any awakenings during the recording period. After the recording session, the subjects returned the system, and the data were downloaded onto a laboratory computer.

### *Data Reduction*

The first-night EMG data were excluded from the analysis to avoid the first-night effect.[36] The EMG data recorded over the remaining 5 nights during the sleeping time reported in the sleep diary were analyzed using a semiautomated custom software program in a blind-to-subject status manner.[35] First, the EMG signals were rectified and smoothed. All instances in which the EMG activity was above a minimum threshold level,

which was individually set at 20% of the established MVC level of each subject,[36] were considered potential bruxism events. In addition, interval and duration criteria were used to further condition these potential bruxism events as follows: *(1)* events that were separated by an interval of less than 2 seconds were linked together in pairs, and *(2)* events that were shorter than 2 seconds in duration were excluded. Next, data cleaning was performed to remove any EMG signal artifacts. In this step, the raw EMG activity of the remaining data was displayed on a computer screen and examined by 2 scorers who were chosen for their ability to precisely and accurately discriminate artifact signals from potential bruxism signals.

Every signal that was judged to be an artifact was excluded from further analysis in a blind-to-subject status manner. The intraclass correlation (ICC) for intrascorer reproducibility ranged from 0.70 to 0.97, and the ICC for interscorer reliability ranged from 0.84 to 0.99, as evaluated using 5 nights of data from 10 subjects. Details of the validity and reliability of the system have been described previously.[32] Finally, the cleaned data were conditioned by the interval and duration criteria once again. The EMG duration variable, which was the total duration of muscle activity per hour, was established for each night and then averaged across the 5-night study period for each subject.

### *Statistical Analysis*

The average masseter EMG duration was considered as a dependent variable, and the average ethanol content was considered as an independent variable. A linear regression analysis was performed between the independent and dependent variables using SPSS 11.0J software (SPSS Japan).

## Results

Two of the 63 subjects carried out additional EMG measurements because of unsuccessful recordings caused by an unacceptable noise level of recorded EMG signals. These subjects were given additional instructions on electrode settings and then completed their recording sessions successfully. No subjects reported detachment of the electrodes upon waking or substantial difficulty with their sleep as result of the device after the second night of recording. Finally, data from 9 subjects were excluded from the analysis as a result of incomplete recordings of the required nights (7 subjects) or major data contamination despite rerecordings (2 subjects).

The data for the independent and dependent variables acquired from 51 subjects are summarized in Table 1. Since 28 of 51 subjects did not consume any alcohol during the recording period, the data from these subjects were excluded from the regression analysis. Instead, group differences in muscle activity between the excluded and included groups were tested using the unpaired *t* test, which found no significant difference between the 2 groups ($P = .35$). The average muscle activity duration was $35.2 \pm 14.6$ seconds in subjects who consumed alcohol and $30.3 \pm 22.9$ seconds in those who did not. The average amount of consumed alcohol was $16.9 \pm 17.2$ g (range, 1.3 to 57.4 g), and the average number of nights subjects consumed any alcohol was $2.3 \pm 1.1$ nights in the included group.

**Table 1** Data for the Independent and Dependent Variables for 51 Subjects

| Self-awareness of sleep bruxism | n | EMG duration (s) | Alcohol intake (g) |
|---|---|---|---|
| All subjects | | | |
| Total | 51 | $32.5 \pm 19.6$ | |
| Positive | 16 | $42.7 \pm 28.1$ | |
| Negative | 35 | $27.9 \pm 12.1$ | |
| Included group | | | |
| Total | 23 | $35.2 \pm 14.6$ | $16.9 \pm 17.2$ |
| Positive | 9 | $42.9 \pm 15.7$ | $21.2 \pm 20.8$ |
| Negative | 14 | $30.4 \pm 12.0$ | $14.1 \pm 14.6$ |
| Excluded group | | | |
| Total | 28 | $30.3 \pm 22.9$ | 0 |
| Positive | 7 | $42.5 \pm 40.5$ | 0 |
| Negative | 21 | $26.2 \pm 12.1$ | 0 |

Linear regression analysis revealed a positive correlation between the ethanol content and masseter muscle activity duration ($Y = 0.51X + 26.6$: coefficient $= 0.51$, 95% confidence interval: 0.20 to 0.82, $R = 0.60$, adjusted $R^2 = 0.33$, $P = .003$) (Table 2 and Fig 1).

## Discussion

The data showed a significant association between the independent and dependent variables. Thus, the null hypothesis was rejected. More specifically, a 10-g increase in ethanol consumption was associated with a 5.1-second increase in EMG duration (95% confidence interval: 2.0 to 8.2 seconds).

The results of this study are not in agreement with those of the PSG study by Hartman et al,[23] who found no significant relationship between bruxism and alcohol consumption. However, it should be noted that the failure to find a significant association in that study might be the result of a daily fluctuation of this behavior, which the PSG could not capture. The results of the present study support the results of an epidemiologic study that reported that sleep bruxers drink alcohol at bedtime significantly more often than nonbruxers.[4] Finally, this study reproduced the results of a previous preliminary study of the risk factors for sleep bruxism.[20]

**Table 2** Linear Regression Models

| Sum of squares | *df* | Mean square error | Coefficient | $R^2$ | Adjusted $R^2$ | F | *P* |
|---|---|---|---|---|---|---|---|
| 4705.43 | 22 | 11.99 | 0.51 | 0.36 | 0.33 | 11.75 | < .01 |

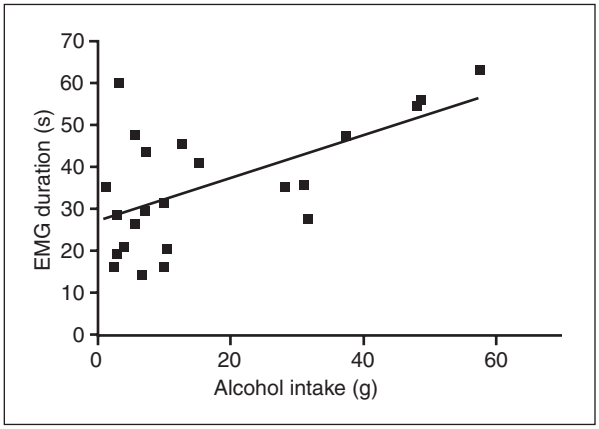


**Fig 1** Relationship between alcohol intake and EMG duration.

It is widely accepted that sleep bruxism occurs as a consequence of transient arousal from sleep.[1] Since alcohol intake disrupts sleep consolidation and affects sleep stage distribution,[37] these effects may indirectly influence sleep bruxism. Additionally, the use of selective serotonin reuptake inhibitors has been reported to cause sleep bruxism, which suggests serotonergic involvement in sleep bruxism occurrence.[38–41] As alcohol causes an acute increase in the local concentration of serotonin, opioids, and dopamine in the brain,[42] alcohol-induced changes in these neurotransmitters may also influence sleep bruxism. However, it is obvious that additional studies are necessary to clarify how alcohol affects sleep bruxism.

The strengths of this study were the number of subjects and the number of nights over which actual masseter EMG data, excluding the first-night data, were collected. Moreover, the masseter muscle activity levels were quantified using high-resolution, artifact-cleaned, noncumulative EMG recordings, which were performed during sleep in the home of the subject. Another strength was that alcohol consumption was quantified based on the total ethanol content calculated using a standard conversion table. These aspects of the study are important because of the limited availability of multiple-night EMG-based bruxism data in relation to accurate ethanol consumption data, as discussed earlier.

With regard to the limitations of this study, all possible efforts were made to prevent data contamination,[32] but the validity and reliability of a single-channel, home-based recording method have certain limitations compared to a sleep laboratory–based recording method using electroencephalography and an audio-video mode, which allows the detection of muscle activity during an actual sleep bruxism event.[42]

The exclusion of the data of non–alcohol users from the regression analysis needs to be addressed. These data were excluded because the main focus of this study was to clarify the relationship between the amount of alcohol consumed and EMG level. Since half the subjects were not alcohol drinkers, the distribution of the independent variable was distorted. The EMG data of the excluded group were compared with those of the included group. This analysis revealed that non–alcohol users tended to exhibit shorter EMG duration; however, it should be noted that this difference was not statistically significant. The excluded group comprised subjects who were sensitive to alcohol because of an inherent lack of aldehyde dehydrogenase and subjects who happened not to drink alcohol during the study period for other unknown reasons. The included group did not have any alcohol-sensitive subjects. Furthermore, the amount of the alcohol consumed varied significantly between individuals within the included group, and the amount of consumed alcohol was generally low (Fig 1). The authors speculate that the actual difference might not have been detected because of the heterogeneous characteristics of the non-alcohol group and the distribution of the amount of alcohol consumed in the included group. The former issue could have been overcome by excluding alcohol-sensitive subjects; unfortunately, however, the authors have no information on the reasons why these subjects did not consume alcohol, and this could be considered as another limitation of this study. These issues should be addressed in future studies.

## Conclusion

Within the forementioned limitations of this study, the results suggest that sleep bruxism is substantially associated with the amount of alcohol consumption in young female subjects. The authors believe that these data are important for a better understanding of sleep bruxism.

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