Development of a New Night-Time Noise Index: Integration of Neurophysiological Theory and Epidemiological Findings

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Abstract

Background: The effects of environmental noise on sleep are of great interest to public health. Numerous studies have been conducted to investigate these effects; however, these previous studies applied existing sound-level statistics that were not based on neurophysiology.

Aims: This study aimed to develop a new night-time noise index based on neurophysiology and epidemiology.

Methods: First, we derived a formula for predicting the noise effects on sleep based on a neurophysiological model of brainstem sleep regulation, where awakening was associated with greater electrical potentials in the brainstem. Second, we investigated the noise effects on sleep using the results of an epidemiological study conducted in the vicinity of the Kadena military airfield in Okinawa, Japan. Thirty volunteers participated in the study. Vibrations of whole-body movements were recorded using sheet-shaped sleep monitors for 26 consecutive nights. The onset of motility, which was defined by monitor vibrations, was used to index awakening reactions.

Results: Our statistical model could properly predict the fluctuating risk of motility onset. The new index, which is the mean of the sound level above 60 dB, can be successfully used, irrespective of the duration of noise exposure. Additionally, it out-performed existing event-related noise indices.

Conclusions: We derived a new night-time noise index for evaluating the noise effects on sleep. To our knowledge, this is the first study to explain the noise effects on sleep with the consideration of neurophysiology and epidemiology.

Keywords: Noise-induced sleep disturbance, sleep science, neurophysiology, epidemiology, motility

Introduction

Night-time noise greatly affects sleep and public health. Short-term noise exposure can induce a single awakening reaction while long-term repeated noise exposures may affect health and cause sleep disorders, hypertension, ischaemic heart disease, stroke, and diabetes [1,2]. In European countries, approximately 6.5 million people suffer from chronic sleep disturbances due to long-term exposure to environmental noise, resulting in 48,000 cases of ischaemic heart disease and 12,000 premature deaths per year [3]. Traffic noise is a leading environmental risk factor and has a burden of disease [4,5].

There have been numerous studies using experimental and epidemiological designs to investigate the effects of night-time noise on sleep [6,7]. However, these studies applied existing sound-level statistics not based on neurophysiology, such as the average of acoustic intensity (night equivalent sound level). Previous studies have examined the association between noise indices and noise effects on sleep [7]. These indices include body movements (motility), arousal, awakening, and self-reported sleep disturbance; however, they do not define physiological mechanisms.

Neurophysiological findings have shown that the states of sleep and wakefulness in the forebrain are regulated by interactions between brainstem nuclei [8]. During sleep, the ventrolateral preoptic (VLPO) nuclei are activated, which inhibit the activation of monoaminergic (MA) nuclei. Conversely, MA nuclei activation inhibit VLPO nuclei activation during wakefulness. External stimuli, including vibrations, can disrupt these interactions and lead to awakening reactions.

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noise exposure, may induce awakening reactions that activate the MA nuclei. Using a mathematical model of the interaction between the brainstem nuclei \cite{10,11}, we previously developed an equation that could account for external stimuli (noise exposure) and electrical potentials in the brainstem associated with awakening reactions \cite{10,11}. The electrical potentials were represented as a first-order lag system, including some undetermined constants, that could be mathematically connected to a new night-time noise index.

The present study aimed to develop a new night-time noise index based on neurophysiology and the findings of an epidemiological study on the noise effects on sleep. We hypothesized that noise-induced awakening is caused by electrical potentials in the brainstem and can be explained by a neurophysiological model of how the brainstem regulates sleep. We derived a formula from our neurophysiological equation for explaining awakening reactions due to noise exposure. Subsequently, we estimated the undetermined constants by analyzing epidemiological findings regarding sleep to obtain the new night-time noise index. To our knowledge, there has been no study considering how noise influences the brain when investigating the noise effects on sleep.

**Materials and Methods**

**Neurophysiological formula on awakening reactions due to noise exposure**

In the present study, we derived a neurophysiology-based formula for explaining awakening reactions due to noise exposure. This includes an equation modelling electrical potentials in the brainstem that regulate sleep \cite{10,11}. It is represented as the output of a first-order lag system of external stimuli, as follows:

\[
\phi(t) = \int_{-\infty}^{t} \left\{ \exp \left( -\frac{t-\tau}{t_0} \right) f(L(\tau)) \right\} d\tau
\]  

(1).

where, \( \phi \) (mV) is the electrical potential, \( t \) (s) is time, \( t_0 \) (s) is a time constant, \( L(t) \) (dB) is sound level, and \( f(L(t)) \) (mVs\(^{-1}\)) is a function that converts sound level into electrical potentials in the brainstem. The \( t_0 \) was estimated as 10–100 seconds \cite{11}.

Since the electrical potentials in the brainstem relate to awakening reactions, the probability of awakening \( h(t) \) is a value of a function \( g \) of \( \phi(t) \),

\[
h(t) = g(\phi(t))
\]  

(2).

The cumulative probability of awakening over a specific time interval \( P(t_a \leq t \leq t_b) \) is represented by the time integral of the instantaneous probability \( h(t) \), as follows:

\[
P(t_a \leq t \leq t_b) = \int_{t_a}^{t_b} h(t) \, dr.
\]  

(3)

Thus, the probability of awakening is associated with fluctuations in sound level.

To our knowledge, there has been no study conducting longitudinal statistical analyses of the noise effects on sleep given the unclear relationship between fluctuations in sound level and the awakening risk. However, neurophysiological equations with a first-order lag system can be used to help explain the relationship, and therefore enable assessment of the fluctuations in sound level and noise effects on sleep. In our previous study, we did not conduct longitudinal analyses. However, we estimated the cumulative probability of awakening using existing exposure-response curves based on the assumption that noise exposure is much shorter than the time constant \cite{10}. In the present study, we constructed a statistical model by integrating the longitudinal epidemiological findings.

Since the cumulative probability is calculated from the time integral of \( h(t) \), these equations are equivalent to the survival analysis using a hazard function of \( h(t) \). If \( g \) is an exponential function and \( h(t) \) depends on several covariates related to external stimuli, the hazard function (Eq. (2)) is expressed as follows:

\[
h(t) = \exp \left( \beta_0 + \beta_1 x_1 + \cdots + \beta_p x_p + \beta_0 \int_{t_0}^{t} \exp \left( -\frac{t-\tau}{t_0} \right) f(L(\tau)) \right) d\tau
\]  

(4),

where, \( x_1, \ldots, x_p \) are covariates and \( \beta_0, \beta_1, \ldots, \beta_p, \beta_0 \) are coefficients of the variables.

The coefficients of the hazard function, including a time-varying hazard, can be estimated based on the survival analysis using a newly developed statistical model known as the distributed lag non-linear model (DLNM) \cite{12}. Further, we assumed that \( f(L(t)) \) is a ramp function to simplify the relationship, as follows:

\[
f(L(t)) = \begin{cases} 
L(t) - 60 & \text{for } L(t) \geq 60 \text{dB} \\
0 & \text{for } L(t) < 60 \text{dB}
\end{cases}
\]  

(5).

The threshold for awakening reactions was set to 60 dB, as defined by the World Health Organization \cite{13}.

Therefore, the neurophysiological formula integrated the epidemiological results. Fixed values were substituted and the likelihood of statistical results was examined to determine the undetermined \( t_0 \) parameter.

**New night-time noise index**

We derived a new night-time noise index using the neurophysiological formula. Let \( h_0 = \exp(\beta_0 + \beta_1 x_1 + \cdots + \beta_p x_p) \), which represents a hazard without noise exposure. The hazard function (Eq. (4)) can be transformed as follows:

\[
h(t) \approx h_0 \cdot \left( 1 + \beta_0 \int_{-\infty}^{t} \exp \left( -\frac{t-\tau}{t_0} \right) f(L(\tau)) \right) d\tau
\]  

(6).

Note that the exponentiation was approximated to a linear expression using the Taylor expansion around zero.
Suppose that \( h_0 \) is constant over a specific time interval \([t_a, t_b]\) and \( t_b - t_a \gg t_0 \). The latter indicates that delayed effects of noise exposure outside the time interval are omitted. The relative risk of an awakening reaction occurring during the time interval, defined as \( \text{RR}_{\text{awake}} \), is expressed as follows:

\[
\text{RR} = \left[ 1 + \beta_0 \int_{t_a}^{t_b} \left( 1 + \int_{0}^{t} \exp\left( -\frac{t - \tau}{t_0}\right) f(L(\tau)) \right) d\tau \right] \left/ \left[ \int_{t_a}^{t_b} h_0 dt \right] \right.
\]

\[
= 1 + \frac{\beta_0}{t_0} \int_{t_a}^{t_b} \int_{0}^{t} \exp\left( -\frac{t - \tau}{t_0}\right) f(L(\tau)) d\tau d\tau
\]

\[
= 1 + \frac{\beta_0 d_0}{t_0} \int_{t_a}^{t_b} f(L(t)) dt
\]  \hspace{1cm} (7)

Consequently, a time average of \( f(L(t)) \) plays a crucial role in determining the risk of the noise effects on sleep. Therefore, if we can accept the linearity assumption of the \( f(L(t)) \), an arithmetic mean of the sound level above 60 dB, \( L_{60\text{dB}} \), is proposed as the new night-time noise index, as follows.

\[
\frac{1}{L_{60\text{dB}}} = \frac{1}{T} \int_{0}^{T} f(L(t)) dt, \quad f(L(t)) = \begin{cases} L(t) - 60 & \text{for } L(t) \geq 60\text{dB} \\ 0 & \text{for } L(t) < 60\text{dB} \end{cases}
\]  \hspace{1cm} (8)

where, \( T \) is the time where the noise effects are evaluated.

Thus, the new noise index can be used to model noise exposure with varying durations. The linear relationship between the noise index and the awakening risk was mathematically derived.

**Epidemiological study investigating the effects of noise exposure on sleep**

**Volunteers**

We investigated the results of an epidemiological sleep study conducted by the Japanese Society of Sonic Environment to estimate the underestimated constants in the aforementioned neurophysiological formula, which facilitated the obtaining of a new night-time noise index. Volunteers were recruited from the Mihara district, Uruma, Okinawa in Japan, where there are high levels of noise caused by military aircraft from the Kadena airfield. The area, which is approximately 7 km from the airfield, is under the flight paths of the aircraft. The local Okinawa government has set up a noise monitoring station. We enrolled 30 volunteers who lived within 200 m from this noise monitoring station, including 15 males and 15 females (age range between teens and sixties). This study was approved by the Research Ethics Committee of the Faculty of Engineering, Hokkaido University. All the volunteers signed an informed consent before participating in the study.

**Sleep measurement**

To evaluate sleep states, bodily vibrations during sleep were measured using a sheet-shaped sleep monitor (SSSM; SleepScan SL504 by Tanita, Tokyo, Japan) \[14\]. This non-invasive method measures whole-body movements, which may have advantages over other methodologies, including actigraphy and polysomnography. The volunteers were instructed to place the SSSM under their mattress at home, turn it on when they went to bed, and turn it off when they got up. The measurements were repeated for 26 consecutive nights between October 20th, 2014 and November 15th, 2014. The time resolution was 0.0625 seconds (sampling rate: 16 Hz).

We estimated rapid eye movement (REM) sleep cycles according to previous reports regarding the association of respiration rates with REM sleep cycles \[15\]. We estimated respiration cycles using the autocorrelation function of the vibrations.

Awakening reactions were indexed using motility data, which was detected from the SSSM vibrations. Motility was defined as a peak 5-times higher than other peaks likely to represent respiration. The onset of motility was defined as isolated movement not preceded by other movements in the 60 seconds before its occurrence.

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**Noise exposure**

Since the volunteers’ homes were < 200 m from the noise monitoring station, we employed sound levels measured at the monitoring station. The data had a time resolution of 1 second. We did not measure the façade or individual indoor sound levels. Further, we collated data provided by the Okinawa government regarding noise events that exceeded 60 dB. To exclude possible artefacts, we only employed noise events if there were no other noise events 2 minutes before their occurrence.

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**Data analysis**

We conducted survival analysis with the DLNM \[12\] using the multivariate Poisson regression analysis. The onset of motility was used to index awakening. The sound level was included in the statistical model as a time-dependent variable. Individual differences, sleeping duration, and sleeping status (REM sleep vs. non-REM sleep) upon the occurrence of noise events were included as time-independent covariates. The time resolution of the data was aligned to one second. The linearity assumption of the sound level was evaluated using a model with the inclusion of categorised variables.

Moreover, multivariate logistic regression analysis was performed to compare noise indices; namely, maximum sound levels (\( L_{\text{max}} \)), sound exposure levels (SEL), and mean of sound levels above 60 dB (\( L_{60\text{dB}} \)). We investigated the onset of motility 90 seconds after the onset of the noise event as previously reported \[7\]. All statistical analyses were conducted using R (Version 3.6.0) \[16\].

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**Results**

The planned sample size of the present study was 780 participant-nights (26 consecutive nights with 30
However, due to technical issues regarding the use of the SSSM and participants forgetting to record their data, only 637 participant-nights were recorded for 29 volunteers. In addition, we excluded 20 datasets due to the volunteers forgetting to turn off the recording after awakening. Thus, the final dataset included 617 participant-nights for 29 volunteers.

Figure 1: An example of the vibration measured by the SSSM. The lower figure shows the first 10 seconds of the upper figure. There were periodic bodily vibrations due to respiration (around 4-second cycle), tiny vibrations due to heartbeat (around 0.8-second cycle) and strong vibrations due to motility (around at 21 seconds in the upper figure).

Figure 2: An example of the estimated respiratory rate (solid line) and the REM sleep (thick lines) vs. the elapsed sleep time.

Figure 3: A scatter plot of the maximum sound level and duration above 60 dB of the observed noise events.

Figure 4: The probability of the onset of motility for every 10 seconds after the onset of noise events. There were 879 recordings of time-to-event data. The probability of the onset of motility was highest at approximately 20–30 seconds after the onset of noise events.
Table 1 displays a descriptive summary of the individual time-to-event data. There was a wide variation range in the rate of motility onset with elapsed sleep time, sleep status, and volunteers.

To examine the noise effects on sleep that lasted over several tens of seconds, we obtained the 1-second data, which ranged from the time the noise event began until 60 seconds after the event ended. Additionally, the data were censored at the time of motility onset. The sample size of the data for the survival analysis was 72,198.

Figure 5 shows the relationship between the time constant of the first-order lag system and the log likelihood in the statistical regression model. The maximum likelihood estimate of the time constant was approximately 15 seconds. By fitting a quadratic curve, we estimated a 95% confidence interval, which ranged from 5.1 to 44.2 seconds. Since the confidence interval was relatively large, we rounded the maximum likelihood estimate of time constant to 15.0 seconds in the subsequent analyses. Minor differences in the time constant did not influence the results.

Table 2 lists the results of the survival analysis. The hazard ratio was significantly correlated with sound level but not with the elapsed sleeping time or sleeping status. The effects of individual differences were relatively large compared with other time-independent covariates, including differences in housing insulation.

As shown in Figure 7, there was a good correlation of the results derived from statistical analyses and observations, which indicates good agreement between both 1-second and cumulative probabilities.
the 1-second probability gradually changed over time, according to the first-order lag system. Both the 1-second and cumulative probabilities were divided into noise-induced and spontaneous probabilities.

These results support the use of the new night-time noise index. The relative risk would be estimated using the index as follows:

$$RR_{\text{awake}} = 1 + 0.053 \cdot L_{60\text{dB}}$$ (9)

This indicates that a 1-dB increase in the new index would lead to a 5.3% increase in the awakening risk.

Table 3 lists the results of the multivariate logistic regression where $L_{\text{max}}$, SEL, or $L_{60\text{dB}}$ was used as a noise index. The $L_{60\text{dB}}$ showed the highest significance and $R^2$ value among the indices.

### Discussion

Noise-induced sleep disturbances are a leading cause of adverse health effects due to noise exposure [2]. However, previous studies have only used sound-level statistics to evaluate night-time noise exposure without considering the importance of neurophysiology [1,6]. We developed a new night-time noise index for investigating the noise effects on sleep based on neurophysiology and epidemiological findings.

To our knowledge, this is the first study to assess the effects of noise exposure on sleep with consideration of neurophysiology and epidemiological findings. Previous studies using sound-level statistics, such as the night

![Figure 5: Relationship between the time constant of the first-order lag system in the mathematical model (log scale) and the log likelihood of the survival analysis](image)

Table 2: Results of the survival analysis regarding the onset of motility

| Variable         | Category/unit | Hazard ratio | 95% CI         | $P$ value |
|------------------|---------------|--------------|----------------|-----------|
| Constant         | —             | 2.014x10^{-3} | 0.741x10^{-3}–5.47x10^{-3} | < 0.0001 |
| Sound level above 60 dB | 10 dB | 1.704 | 1.353–2.145 | < 0.0001 |
| Elapsed sleep time | < 2 h | 1.000 | — | — |
|                  | 2–4 h | 1.018 | 0.628–1.648 | 0.942 |
|                  | 4–6 h | 1.231 | 0.790–1.918 | 0.360 |
|                  | 6 h < | 1.190 | 0.771–1.837 | 0.433 |
| Sleep status     | Non-REM      | 1.000 | — | — |
|                  | REM          | 1.264 | 0.888–1.800 | 0.193 |
| Volunteer*       | 10th (4/29)  | 0.249 | 0.051–1.134 | 0.072 |
|                  | 25th (8/29)  | 0.674 | 0.167–2.726 | 0.580 |
|                  | 50th (15/29) | 1.000 | — | — |
|                  | 75th (22/29) | 1.256 | 0.360–4.383 | 0.720 |
|                  | 90th (26/29) | 1.668 | 0.521–5.341 | 0.389 |

*Volunteers were sorted by the rate of onset of motility.
equivalent sound level, could not account for how sleep may be disturbed by fluctuations in noise exposure. Although several studies have attempted to evaluate the temporal aspects [18-21], neurophysiological validity remains unclear.

Table 3: Summary of the results of the multivariate logistic regression analysis regarding the onset of motility in a time window of 90 seconds after the beginning of the noise event, using different noise indices (n=879)

| Noise index | Unit | Odds ratio (95% CI)* | P value | McFadden’s $R^2$ |
|-------------|------|----------------------|---------|------------------|
| $L_{\max}$ | 10 dB | 1.399 (1.049–1.866)  | 0.0225  | 0.0808           |
| SEL         | 10 dB | 1.434 (1.084–1.897)  | 0.0115  | 0.0820           |
| $L_{Aeq}$   | 10 dB | 1.971 (1.270–3.060)  | 0.0025  | 0.0848           |

Figure 7: Correlation between the predicted and observed probabilities of the onset of motility. The 1-second probability is shown in the left figure, in which the observed probability and its CI were obtained by categorising the predicted probability (<0.2%, 0.2–0.4%, 0.4–0.6%, 0.6–0.8%, >0.8%). The cumulative probability is shown in the right figure, in which the observed probability and its CI were obtained by categorising the predicted probability (<10%, 10–20%, 20–30%, 30–40%, >40%). Equal-probability lines are also shown.

Figure 8: An example of the fluctuations of the sound level (upper figure), the 1-second probability of the onset of motility (middle figure), and the cumulative probability (lower figure). Each of the probabilities can be divided into the spontaneous and noise-induced (shading) probabilities. The observation of the onset of motility is also shown (step line).
Evaluating the effects of night-time noise based on neurophysiology could contribute to the estimation of the cumulative noise effects on health. An SSSM was used to measure sleeping states, which is a method used in epidemiological studies. An SSSM is objective, non-invasive, inexpensive, and has a high time resolution, which is applicable to both short- and long-term measurements. Further, this method enables the investigation of awakening reactions in a volunteer’s bedroom. Moreover, this method for measuring whole-body movements may be more favourable than approaches that only measure wrist movement, such as actigraphy. The onset of motility assessed in this study is indicative of short-term arousal and can be easily detected. Future studies should investigate the association of physiological reactions during sleep with adverse health effects (e.g. changes in heart rate).

Longitudinal–survival analysis using a newly developed statistical model played a critical role in identifying the instantaneous noise effects on sleep using this neurophysiological formula. This may explain the temporal causality and delayed noise effects not reported by previous cross-sectional studies. Future studies using the neurophysiological formula should investigate how sleep is affected by external stimuli, including noise exposure and others not recorded in this study, e.g., external vibrations, light, and temperature.

Survival analysis of delayed effects provided important insight into noise-induced sleep effects. First, the time constant of the time-integral system in the brainstem was estimated (95% CI: 5.1–44.2 seconds), which was consistent with our rough estimation of 10–100 seconds. This indicated that noise caused delayed effects that lasted over several tens of seconds; moreover, the brainstem is relatively insensitive to short-term stimuli. The estimated constant has scientific value since it may determine the dynamic characteristics of circadian transitions between sleep and wakefulness in the brainstem. Second, statistical hazard estimation revealed that a 10 dB increase in the sound level was equivalent to a 1.7-fold increase in noise duration. It quite differs from energy-based evaluations used in most studies, where a 10 dB increase is equivalent to a 10-fold increase in the noise duration. Therefore, the energy-based evaluation may underestimate the effects of noise duration.

The new night-time noise index (L60dB) is the first noise index based on neurophysiology and epidemiological findings. Moreover, it can be used to investigate the short- and long-term effects of noise exposure. Existing noise indices are divided into those used for single noise events (e.g. Lmax, SEL) and long-term noise exposure (e.g. night equivalent sound level). In addition, the new index may help evaluate chronic health risks resulting from long-term noise exposure. However, it should be noted that the threshold of the noise effects on sleep may be lower than 60 dB because we did not investigate the effects of low levels of noise. Future epidemiological studies are needed to investigate the reliability of the new index when evaluating the noise effects on sleep. Further, there is a need for future studies regarding the threshold for protecting health.

This study has several limitations. First, we did not measure the sound levels at each volunteer’s home. Estimated individual differences should also include differences in the insulation of each volunteer’s home. Second, we did not measure within-person covariates that may influence sleep, including the daily use of alcohol and illicit substances. Future studies should investigate these variables since they may lead to substantial errors in quantitative estimations.

Conclusions
To develop a new night-time noise index for evaluating the noise effects on sleep based on neurophysiology, we derived a formula that could explain how sleep may be disturbed by fluctuations in noise exposure. We integrated it with the results of an epidemiological study using longitudinal statistical analysis. The new index, L60dB, out-performed existing event-related indices. Moreover, our formula and the new index can be used to investigate the short- and long-term effects of noise exposure on sleep. It may also help evaluate the noise effects on chronic adverse health problems, including cardiovascular disease. Future studies should assess the reliability of the new index, which could help evaluate the noise effects on sleep and health.

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Conflicts of interest
There are no conflicts of interest.

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