Application of the Kurtosis Statistic to the Evaluation of the Risk of Hearing Loss in Workers Exposed to High-Level Complex Noise

Yi-ming Zhao,[1] Wei Qiu,[2] Lin Zeng,[1] Shan-song Chen,[3] Xiao-ru Cheng,[1] Robert I. Davis,[2] and Roger P. Hamernik[2]

Objective: Develop dose-response relations for two groups of industrial workers exposed to Gaussian or non-Gaussian (complex) types of continuous noises and to investigate what role, if any, the kurtosis statistic can play in the evaluation of industrial noise-induced hearing loss (NIHL).

Design: Audiometric and noise exposure data were acquired on a population (N = 195) of screened workers from a textile manufacturing plant and a metal fabrication facility located in Henan province of China. Thirty-two of the subjects were exposed to non-Gaussian (non-G) noise and 163 were exposed to a Gaussian (G) continuous noise. Each subject was given a general physical and an otologic examination. Hearing threshold levels (0.5–8.0 kHz) were age adjusted (ISO-1999) and the prevalence of NIHL at 3, 4, or 6 kHz was determined. The kurtosis metric, which is sensitive to the peak and temporal characteristics of a noise, was introduced into the calculation of the cumulative noise exposure metric. Using the prevalence of hearing loss and the cumulative noise exposure metric, a dose-response relation for the G and non-G noise-exposed groups was constructed.

Results: An analysis of the noise environments in the two plants showed that the noise exposures in the textile plant were of a Gaussian type with an $Leq(A)_{8hr}$ that varied from 96 to 105 dB whereas the exposures in the metal fabrication facility with an $Leq(A)_{8hr} = 95$ dB were of a non-G type containing high levels (up to 125 dB peak SPL) of impact noise. The kurtosis statistic was used to quantify the deviation of the non-G noise environment from the Gaussian. The dose-response relation for the non-G noise-exposed subjects showed a higher prevalence of hearing loss for a comparable cumulative noise exposure than did the G noise-exposed subjects. By introducing the kurtosis variable into the temporal component of the cumulative noise exposure calculation, the two dose-response curves could be made to overlap, essentially yielding an equivalent noise-induced effect for the two study groups.

Conclusions: For the same exposure level, the prevalence of NIHL is greater in workers exposed to non-G noise environments than for workers exposed to G noise. The kurtosis metric may be a reasonable candidate for use in modifying exposure level calculations that are used to estimate the risk of NIHL from any type of noise exposure environment. However, studies involving a large number of workers with well-documented exposures are needed before a relation between a metric such as the kurtosis and the risk of hearing loss can be refined.

(Ear & Hearing 2010;31;527–532)

[1]Center for Clinical Epidemiological Research, Peking University Third Hospital, Beijing, People's Republic of China; [2]Auditory Research Laboratory, State University of New York at Plattsburgh, Plattsburgh, New York; and [3]Center for Disease Control of ZhengZhou, Henan, People's Republic of China.

INTRODUCTION

Equal energy hypothesis (EEH) postulates that the risk of noise-induced hearing loss (NIHL) is a function of the total exposure energy. The EEH thus implies that hearing loss is independent of the temporal properties of the noise. Industrial workers are often exposed to complex noise environments, i.e., a non-Gaussian (non-G) noise consisting of high-level transients embedded in a Gaussian (G) noise. Epidemiologic studies in industries, where workers are exposed to noise environments containing impact noise transients, have shown that there is an increased incidence of hearing loss (Taylor et al. 1984; Theiry & Meyer-Bisch 1988; Lataye & Campo 1996).

A number of animal-based laboratory experiments have shown that exposures that contain high-level transients (e.g., impacts or noise bursts) can produce increased levels of hearing loss when compared with an equivalent energy continuous G noise (Dunn et al. 1991; Lei et al. 1994; Lataye & Campo 1996). Such results confirm that the temporal distribution of energy is an important factor in NIHL. Although this research has reinforced the results obtained from industrial epidemiologic studies, none of these studies have provided information on the dose-response relation (DRR) between non-G industrial noise and hearing loss. Part of the difficulty in trying to establish a DRR is in how to measure or characterize the great diversity of non-G noises found in industry. For example, factors such as the histograms of both the peak levels and interpeak intervals of the embedded transients in addition to the overall SPL, spectra, and exposure durations may need to be taken into account. Neglecting any one of these factors will inevitably lead to an unacceptable DRR. Currently, noise measurements being used to access the risk of NIHL (American National Standard Institute [ANSI] S3.44 1996) are based on an 8-hr equivalent A-weighted SPL [$Leq(A)_{8hr}$]. However, there is considerable evidence that the effects of a non-G industrial noise exposure do not conform to the equal energy model (i.e., equivalent effects for a 3-dB increase or decrease in exposure intensity with a halving or doubling of the exposure duration, respectively).

Using data obtained from industrial workers exposed to noise environments with and without embedded impact transients, Zhao et al. (2006) produced a DRR for each of the two populations using the $Leq(A)_{8hr}$ as the noise metric. They found that (1) hearing loss increased at a faster rate for the population exposed to the non-G noise environment than for the exposure that was free of the impulsive transients (i.e., a G noise) and (2) that for a given energy level, the

0196/0202/10/3104-0527/0 • Ear & Hearing • Copyright © 2010 by Lippincott Williams & Wilkins • Printed in the U.S.A.

non-G exposure produced more high-frequency hearing loss than did the G noise. The problem with these two DRRs and in particular the DRR for the non-G noise-exposed population was that they were, most likely, industry specific. These results, however, suggest that the single energy metric, Leq, although necessary, is not sufficient for the development of a DRR. An approach to incorporate the temporal features of a non-G noise should be developed for use in conjunction with the Leq to adequately evaluate the risk of hearing loss from any industrial noise environment.

Recent results from animal experiments (Hamernik et al. 2003; Qiu et al. 2006, 2007; Davis et al. 2009) have shown that the kurtosis (β) of the amplitude distribution, a statistical metric that is sensitive to the peak and temporal characteristics of a noise, could order the extent of hearing and sensory cell loss from a variety of complex noise exposures. They showed that for a fixed energy level, the noise-induced trauma increased as the kurtosis increased. Thus, there is the possibility that the kurtosis in combination with the Leq might be useful in the evaluation of any noise environment for hearing conservation purposes.

An issue of importance in formulating a damage risk criteria is whether the kurtosis metric can be incorporated into energy measurements from non-G industrial noise environments and whether this combined metric will reflect the hazard to hearing associated with such environments. Other attempts have been made to develop metrics (Zhu et al. 2009) that would be highly correlated with hearing loss from any industrial noise environment. However, to date, the ideal metric remains elusive.

In this report, an analysis of a limited database consisting of industrial noise recordings and hearing levels acquired from workers in industries that had high and low kurtosis noise environments will illustrate one approach to use the kurtosis statistic in combination with the Leq to unify a diverse set of hearing loss and noise level data.

MATERIALS AND METHODS

Subjects

Subjects (N = 220) were introduced to the purpose of and procedures to be followed in this study by an occupational physician and were asked to sign an informed consent form. The Institutional Review Boards for the protection of human subjects of the Peking University and State University of New York at Plattsburgh approved the protocol for this study. Industrial workers were recruited from two plants in Zhengzhou, Henan province of China. One of the plants was a metal fabrication facility where the subjects were primarily exposed to complex non-G noise. The other plant was a textile mill where subjects were exposed to a continuous G noise. For inclusion in the study, all subjects had to satisfy the following four criteria: (1) a minimum of at least 1-yr employment at the current task; (2) no history of genetic or drug-related hearing loss, head trauma, or ear diseases, (3) no military service, shooting activities, or high intensity nonindustrial noise exposure, and (4) no history of use of hearing protection. As a result, a total of 195 workers were acceptable from the original pool of 220 subjects in the two industries. Thirty-two of the subjects (35.1 ± 7.2 yrs, 12 men and 20 women) were exposed to non-G noise for an average of 12.3 ± 7.1 yrs in the metal fabrication industry. The remaining 163 subjects (31.5 ± 8.7 yrs, 82 men and 81 women) were exposed to a continuous G noise for an average 12.7 ± 8.4 yrs in a textile mill. Workers exposed to the complex noise were slightly older than workers exposed to the continuous noise ($t = 2.20$, $p < 0.03$). There was no statistical significance in the duration of the occupational exposure between the two populations.

Questionnaire Survey

At the Zhengzhou Center for Disease Control and Prevention, an occupational hygienist administered a questionnaire to each subject to collect the following information: general personal information (age, sex, etc.), occupational history (factory, workshop, military, job description, length of employment, duration of daily noise exposure, and history of hearing protector use), personal life habits (e.g., shooting, smoking, and alcohol use), overall health, history of ear disease, and use of ototoxic drugs. An occupational physician entered all information into a database.

Physical and Audiometric Evaluation

Each subject that passed the screening protocol was given a general physical and an otologic examination. Pure tone, air conduction hearing threshold levels at 0.5, 1.0, 2.0, 3.0, 4.0, 6.0, and 8.0 kHz were measured in each ear by an experienced physician. Testing was conducted in an audiometric booth (baseline noise <30 dB SPL) using an audiometer (Madsen, OB40, Copenhagen, Denmark) calibrated according to the Chinese national standard (GB4854-84). Audiograms were measured at least 16 hrs after the subjects' last occupational noise exposure.

Determination of an Adjusted High-Frequency NIHL

Hearing threshold levels (HTLs) at each frequency were adjusted for age and gender using the 50th percentile values found in the International Standard Organization (ISO-1999 1990) Annex B. The prevalence of an adjusted high-frequency NIHL (AHFNIHL) in workers exposed to non-G or G noise was defined as one or more hearing levels, in either ear, at 3, 4, or 6 kHz equal to or higher than 30 dB.

SPL Noise Measurements

Personal noise dosimeters (Institute of Acoustics, Chinese Academy of Science and Hengyang Instruments, SH-126, class II) were used to collect noise-exposure sound levels on individuals. The dosimeters were equipped with a microphone (HY205; Hengyang Instruments, Hunan, China) fixed on the collar of each subject. The dynamic range of the dosimeters was 40 to 140 dBA. The noise dosimeters could work continuously for 8 hrs at a sampling rate of 5 Hz. Before any measurements, each dosimeter was calibrated using a 94 dB SPL, 1 kHz tone calibrator (AWA6221B; AIHUA Instruments, Hangzhou, China). Noise data collected in the dosimeter were transferred to an IBM-compatible computer. The $Leq(A)_{8hr}$ was calculated for each 8-hr set of measurements using a software package designed for the dosimeter. The workers were divided into six groups based on the type of noise exposure, factory, workshop, type of work and type of machine they were using. In the continuous G noise environment, four or five workers in each group were selected for shift-long personal noise exposure level measurements using the dosimeter over a workday lasting 8 or 10 hrs. In the non-G noise environment, 25 of the 32 subjects were measured over the course of one entire work shift. All the Leq(A)$_{8hr}$ measurements for each group were averaged to produce a group mean daily noise exposure level (dBA SPL).

Real-Time Sound Recording and Kurtosis Measurement

A sound level meter (Hong Sheng, Model HS5670, Jiangxi, China) was used to collect real-time samples of each subject's noise exposure. Recordings were made at the level of the subject's ear. Nineteen 5-min samples of the real-time signal from the complex non-G noise environments and 20 from the G noise environments were recorded with 16-bit resolution at an 11 kHz sampling rate and fed to a computer for subsequent analysis. The kurtosis of the noise signal was computed over consecutive 40-sec time windows of each 5-min noise record using commercial software (MATLAB), and the mean value was used to establish the kurtosis value for each series of noise records. Because kurtosis is dependent on the length of the window over which the calculation is made, and its calculation is limited by the computer's processing capabilities, a compromise was made to use a 40-sec time window which, based on animal data (Hamernik et al. 2003) was found to be sufficient for establishing an acceptable measure of the kurtosis. The Nyquist limit (5.5 kHz) would have had only a small effect on the kurtosis value of the noise waveform recordings containing frequencies above this limit.

Cumulative Sound Energy Exposure Assessment

The cumulative noise exposure (CNE), a composite noise exposure index (Earshen 1986), was used to quantify the noise exposure for each subject. The CNE is defined as:

$$CNE = 10\log\left[\frac{1}{T_{ref}}\sum_{i=1}^{n}(T_i \times 10^{L_{Aeq,8hr}/10})\right] \quad (1)$$

where $L_{Aeq,8hr}$ is the equivalent continuous A-weighted noise exposure level normalized to an 8-hr working day, in decibels, occurring over the time interval T_i in years; n is the total number of different noise levels (i.e., different working tasks/environments) the subject was exposed to during their employment history; and $T_{ref} = 1$ yr. Because all subjects in this study never changed their working environment, n was set equal to 1 and T_1 was simplified as T. Thus, for the 195 subjects in this study $n = i = 1$ and Eq. (1) can be written as:

$$CNE = L_{Aeq,8h} + 10\log T \quad (2)$$

This relation is typically applied to the evaluation of noise environments that require an estimate of the total exposure energy.

TABLE 1. Group mean noise levels [Leq(A)$_{8h}$ dB ± 1SD] for the six groups of subjects

Noise Type	Noise Source	N_1	N_2	Leq(A)$_{8hr}$ dB
Gaussian	Loom ZA205i	24	4	98.1 ± 2.1
	Loom 1511	75	5	105.4 ± 2.2
	Spinner FA507A	23	5	99.5 ± 2.2
	Spinner 1301	41	5	96.1 ± 2.7
Complex	Punch press	17	11	95.3 ± 2.5
	Plate clipper	15	14	95.2 ± 3.5

N_1, total number of subjects at the indicated workstation; N_2, number of subjects with full shift noise recordings.

Table 2. Cumulative noise exposure (CNE) and adjusted high-frequency noise-induced hearing loss (AHFNIHL) in complex (non-Gaussian) noise-exposed workers and Gaussian noise exposed workers using the 3 dB equal energy rule

Group	N_1	CNE [dB(A)·yr] ± 1SD	AHFNIHL N_2	AHFNIHL Prevalence (%)
Complex noise	32	103.2 ± 4.2	21	65.6
Gaussian noise	163	110.6 ± 6.0	105	64.4

N_1, number of subjects in each group; N_2, number of cases of AHFNIHL.

Data Processing and Statistical Analysis

Two staff members each entered the data from the subject's questionnaire into a database using Epi Info 6.04D software (Centers for Disease Control and Prevention, Atlanta, GA). The duplicated database was then checked for errors and sent into an SPSS (version 13) software package for subsequent analysis.

RESULTS

The number of subjects in each noise-exposed group, the noise source to which the group was exposed, the number of subjects from whom full-shift noise levels were recorded, and the mean Leq(A)$_{8hr}$ for that group is shown in Table 1. The field investigation as well as the personal questionnaire indicated that none of the subjects used hearing protectors during their employment in the metal fabrication or textile industry. The Leq(A)$_{8hr}$ that these subjects were exposed to varied from a low value of 95 dBA for the non-G noise exposures to as much as 105 dBA for the continuous G noise. Peak levels of the individual impacts in the non-G noise reached as much as 125 dB peak SPL. Noise exposure

Table 3. Prevalence (%) of adjusted high-frequency noise-induced hearing loss (AHFNIHL) in workers exposed to complex (non-Gaussian) and Gaussian noise

	Complex Noise			Gaussian Noise		
CNE (dBA·yr)	N_1	N_2	AHFNIHL (%)	N_1	N_2	AHFNIHL (%)
90	2	0	0	—	—	—
95	5	1	20.0	9	1	11.1
100	13	9	69.2	23	7	30.4
105	11	10	90.9	40	20	50.0
110	1	1	100.0	39	30	76.9
115	—	—	—	52	47	90.4

The subjects were separated into 5 dB cumulative noise exposure (CNE) bins. The CNE for each subject was computed using the 3 dB time-intensity trading rule [i.e. Eq. (2)].
N_1, total number of subjects in the indicated CNE bin; N_2, number of subjects with AHFNIHL.

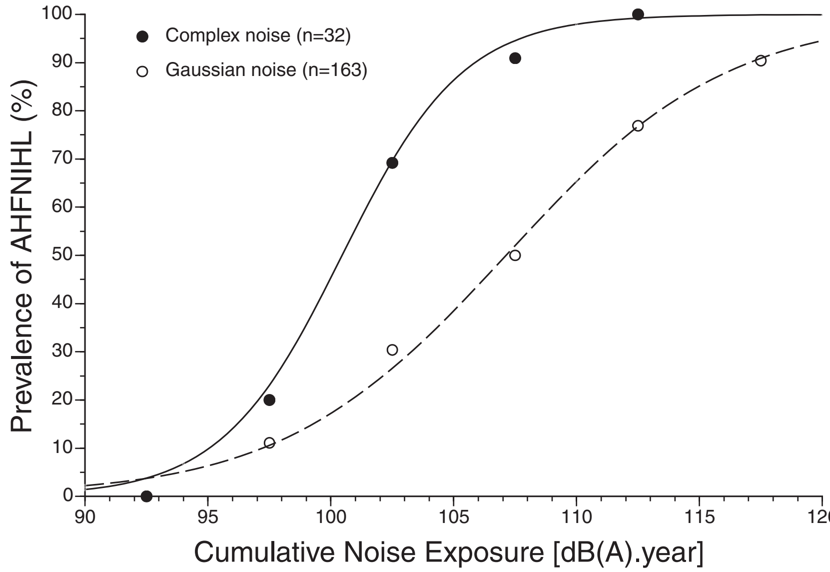

Fig. 1. The dose-response relation for long-term complex (non-Gaussian) and Gaussian noise exposure. The cumulative noise exposure was calculated using a 3-dB time-intensity trading relation [i.e., Eq. (2)]. These data, taken from Table 3, were plotted at the midpoint of each bin.

levels in the four G noise environments were generally higher than those in the two non-G noise environments.

Prevalence of AHFNIHL From Gaussian and non-Gaussian Noise Exposures

The CNE level and the prevalence of AHFNIHL for the two study groups (i.e., G and non-G noise-exposed groups) are shown in Table 2. The CNE level for workers in the G noise environment (110.6 dBA·yr) was higher than for those in the non-G noise environment (103.2 dBA·yr). This difference in level was not statistically significant (analysis of variance, $p < 0.05$). The prevalence of AHFNIHL in the two groups was similar. For the group exposed to the G noise, the prevalence was 64.4%, and for the group exposed to the non-G noise, it was 65.6%. There was no statistically significant difference between the two groups in the prevalence of AHFNIHL. However, separating the exposure levels of the subjects into 5-dB CNE bins showed a clear difference between the prevalence of AHFNIHL in the non-G and G noise-exposed groups for the 100 and 105 dBA in Table 3. The prevalence in the non-G noise-exposed workers was significantly higher than that of the workers exposed to G noise with differences of 69% versus 30% in the 100 dBA bin and 91% versus 50% in the 105 dBA bin. These differences were statistically significant (analysis of variance, $p < 0.05$).

Cumulative Noise Exposure and the Kurtosis Metric

A logistic regression model was used to fit the dose-response data shown in Table 3 for both the non-G and the G noise groups. The results shown in Figure 1 yielded a typical DRR for both groups with the complex noise-exposed group shifted to the left and with a steeper slope relative to G noise-exposed subjects. Although the number of subjects in this study is limited, the results suggest that, as with the animal model data, complex non-G noise exposure is more hazardous to hearing than is an energy equivalent continuous G noise (Hamernik et al. 2003; Qiu et al. 2006, 2007; Davis et al. 2009).

A mean value of the kurtosis, representative of both noise environments, was calculated from 19 5-min real-time segments of the non-G noise recorded from the manufacturing workshops and 20 5-min segments of G noise from the textile workshops. These two sets of recordings were sampled over the course of the measured subject's work shift. Figure 2a shows a 40-sec record of the G noise and Figure 2b the non-G noise. For the illustrated waveform, the kurtosis computed over the 40-sec sample of the G noise was approximately 3.1, and

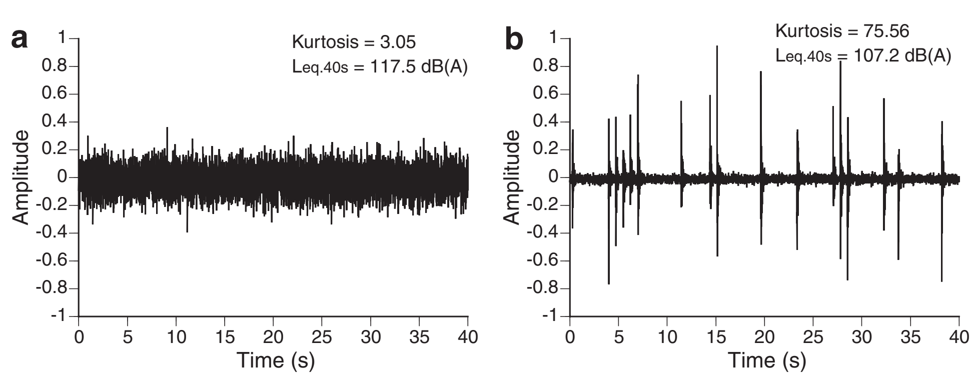

Fig. 2. Forty-second samples of the temporal waveform of (a) the continuous Gaussian noise and (b) the complex (non-Gaussian) noise.

Table 4. Mean kurtosis (β) and the factor *K* for the two classes of noise

Group	N	$\beta \pm$ 1SD	$K = \ln(\beta) + 1.9$
Complex noise	19	40 ± 44	5.6
Gaussian noise	20	3.3 ± 0.3	3.1

N is the number of 5-min real-time segments of noise recordings. Kurtosis was calculated over consecutive 40-sec segments of each noise sample and an average calculated.

for the non-G noise sample, it was 75.6. The mean value of the kurtosis, computed over successive 40-sec windows of the sampled segments for both classes of noise, is shown in Table 4. For the G noise, it was approximately 3, confirming the G character of the noise. For the non-G noise, it was approximately 40 with a large SD. This difference was statistically significant (analysis of variance, $p < 0.05$).

To incorporate the kurtosis metric into the evaluation of non-G noise environments and to unify the epidemiologic data from the two classes (i.e., G and non-G) of noise, Eq. (2) was rewritten equivalently as:

$$\mathrm{CNE} = \mathrm{L}_{\mathrm{Aeq,8h}} + \mathrm{K}\,[\log \mathrm{T}/\log 2] \quad (3)$$

For a G noise environment, where hearing loss accumulates roughly in accordance with equal energy principle, the variable K can be viewed as the time-intensity trading value, $K = 3$.

However, because the effects of non-G noise on hearing do not conform to an equal energy model, the CNE for the subjects exposed to the non-G noise was adjusted by introducing the kurtosis (β) into the variable K in the form:

$$\mathrm{K} = \ln(\beta) + 1.9 \quad (4)$$

The form of Eq. (4) was chosen so that for a G noise with $\beta = 3$, the term $K/\log 2$ in Eq. (3) would be approximately 10 and thus Eq. (3) would reduce to Eq. (2). Using the mean values $\beta = 3.3$ (SD = 0.3) and $\beta = 40$ (SD = 44) that were computed for the G and non-G noise environments, respectively, yielded a value of $K = 3.1$ for the G noise data in accordance with the EEH and a value of $K = 5.6$ for the non-G noise data. With this empirically established relation between K and β, the kurtosis adjusted CNE levels of the non-G noise groups were very similar to those of the G noise groups (110.6 ± 6.7 versus 110.9 ± 6.0).

All the data in Table 3 and Figure 1 were recalculated using Eqs. (3) and (4). The new "kurtosis-adjusted" CNE values for the subjects are presented in Table 5 and are shown plotted in Figure 3. The two data sets now have a similar DRR. That is, the prevalence of AHFNIHL for the G-exposed subjects and the CNE computed with an approximate equal energy rule ($K = 3.1$) were essentially unchanged, whereas the AHFNIHL prevalence data and the kurtosis-adjusted CNE with $K = 5.6$ are substantially changed and nearly superimposed on the $K = 3.1$ data. The $K = 5.6$ curve in Figure 3 essentially represents an equal noise-induced effect from the non-G noise exposure relative to the $K = 3.1$ equal energy analysis of the G noise exposure.

DISCUSSION

Several early epidemiological studies (Passchier-Vermeer 1974; Kuzniarz et al. 1976; Taylor et al. 1984) have suggested that noise environments that contain high-level transients, i.e., complex or non-G noise, are more hazardous to hearing than energy equivalent uniform (G) noise environments. For example, when the function relating hearing loss and noise exposure level found in the Passchier-Vermeer (1974) data is compared with that of Burns and Robinson (1970), the former has a steeper slope and, at any given noise level, a greater hearing loss. The former data were acquired from a mixed population exposed to complex as well as G noise environments, whereas the latter data were acquired from a select population that had no impact noise exposures. The Kuzniarz et al. (1976) and Taylor et al. (1984) data included subjects exposed to complex noise, and these data were in accord with the Passcher-Vermeer results (see also Henderson and Hamernik 1986).

Table 5. Prevalence (%) of the adjusted high-frequency noise-induced hearing loss (AHFNIHL) in workers exposed to complex (non-Gaussian) or Gaussian noise

	Complex Noise $K = 5.6$			Gaussian Noise $K = 3.1$		
CNE (dBA·yr)	N_1	N_2	AHFNIHL (%)	N_1	N_2	AHFNIHL (%)
90∼	1	0	0	—	—	—
95∼	1	0	0	9	1	11.1
100∼	3	1	33.3	22	7	31.8
105∼	10	6	60.0	41	20	48.8
110∼	8	5	62.5	39	30	76.9
115∼	9	9	100.0	52	47	90.4

The subjects' cumulative noise-exposure (CNE) values were adjusted using the value of K determined from the kurtosis (β) by $K = \ln(\beta) + 1.9$. The subjects were separated into 5 dB CNE bins.

N_1, total number of subjects in the indicated CNE bin; N_2, number of subjects with AHFNIHL.

The data shown in Figure 1 and Table 3, although based on an admittedly small sample size, does conform to the results mentioned above in showing the increased hazard associated with complex noise exposures. Current noise regulations (ANSI, ISO 1999) are based on the EEH that embodies a 3-dB time-intensity trading relation for noise exposures. It is clear from Figure 1 that the 3-dB exchange relation yields two different functional relations between the prevalence of AHFNIHL and the CNE exposure metric. However, the two nearly congruent sets of data in Figure 1 suggest that a kurtosis-based modification of the variable K might yield a consistent data set. Although an arbitrary adjustment in the logistic function could achieve the desired result, a more efficacious approach might be to relate K to some variable that can be extracted from the noise measurements. Considering that the animal data discussed in the introduction have shown a consistent relation between the kurtosis of the noise and the subsequent hearing loss and cochlear pathology, the kurtosis would seem to be a reasonable candidate for use in modifying energy calculations for hearing risk assessment.

The diversity of industry-specific complex noise environments makes it difficult to establish a single DRR (i.e., prevalence of NIHL as a function of CNE level) suitable for all types of complex noises. However, the relative stability of the DRR for continuous G noise exposure may serve as a

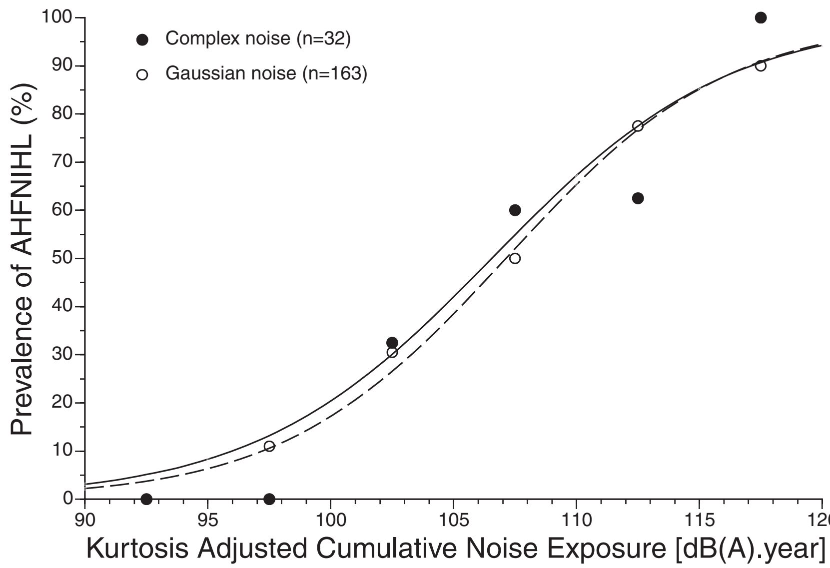

Fig. 3. The dose-response relation for long-term complex and Gaussian noise exposure. The cumulative noise exposure was calculated using the kurtosis-based adjustment factor $K = \ln(\beta) + 1.9$, where β = kurtosis. These data, taken from Table 5, were plotted at the midpoint of each bin.

reference to which the CNE from a complex noise exposure can be compared to estimate the prevalence of NIHL that can be expected from prolonged exposure to any noise. Another way of viewing the exercise presented in this article is simply that the kurtosis can be used to "adjust" the CNE to yield a consistent estimate of the prevalence of hearing loss for exposure to any noise environment using a single number metric. Whether there is any practical use for this kind of an adjustment process can only be determined by repeating this exercise with data acquired from a large number of workers, with well-documented exposures, to a diverse set of complex noises.

ACKNOWLEDGMENTS

This work was partially supported by grant no. 1-R01-OH008967 from the National Institute for Occupational Safety and Health.

Address for correspondence: Yi-ming Zhao, Ph.D., Research Center of Occupational Medicine, Research Center for Clinical Epidemiological Research, Peking University Third Hospital, Beijing 100191, People's Republic of China. E-mail: yimingzhao115@163.com.

Received June 25, 2009; accepted February 6, 2010.

REFERENCES

ANSI (American National Standard Institute) (1996). *ANSI S3.44. Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment*. New York: Acoustical Society of America.

Burns, W., & Robinson, D. W. (1970). An investigation of the effects of occupational noise on hearing. In *Hearing and Noise in Industry* (pp, 177–192). London: HMSO.

Davis, R. I., Qiu, W., Hamernik, R. P. (2009). The role of the kurtosis statistic in evaluating complex noise exposures for the protection of hearing. *Ear Hear*, *30*, 628–634.

Dunn, D. E., Davis, R. R., Merry, C. J., et al. (1991). Hearing loss in the chinchilla from impact and continuous noise exposure. *J Acoust Soc Am*, *90*, 1979–1985.

Earshan, J. J. (1986). Sound measurement: Instrumentation and noise descriptors. In E. H. Berger, W. D. Ward, J. C. Morrill, et al. (Eds). *Noise and Hearing Conservation Manual*. Akron, Ohio: American Industrial Hygiene Association.

Hamernik, R. P., Qiu, W., Davis, R. (2003). The effects of the amplitude distribution of equal energy exposures on noise-induced hearing loss: The kurtosis metric. *J Acoust Soc Am*, *114*, 386–395.

Henderson, D. & Hamernik, R. P. (1986). Impulse noise: Critical review. *J Acoust Soc Am*, *80*, 569–584.

ISO (International Standard) 1999 (1990). ISO 1999: Acoustics-Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment. ISO/TC43-Acoustics. Geneva, Switzerland: International Organization for Standardization.

Kuzniarz, J.J., Swierczynski, Z. Lipowczan, A. (1976). Impulse noise induced hearing loss in industry and the energy concept: A field study. In *Proceedings of the 2nd Conference on Disorders of Auditory Function*. Southampton, London: Academic Press.

Lataye, R., & Campo, P. (1996). Applicability of the Leq as a damage-risk criterion: An animal experiment. *J Acoust Soc Am*, *99*, 1621–1632.

Lei, S.-F., Ahroon, W. A., Hamernik, R. P. (1994). The application of frequency and time domain kurtosis to the assessment of hazardous noise exposures. *J Acoust Soc Am*, *96*, 1435–1444.

Passcher-Vermeer, W. (1974). Hearing loss due to continuous exposure to steady-state broad-band noise. *J Acoust Soc Am*, *56*, 1585–1593.

Qiu, W., Hamernik, R. P., Davis, R. (2006). The kurtosis metric as an adjunct to energy in the prediction of trauma from continuous, non-Gaussian noise exposures. *J Acoust Soc Am*, *120*, 3901–3906.

Qiu, W., Davis, R., Hamernik, R. P. (2007). Hearing loss from interrupted, intermittent, and time-varying Gaussian noise exposures: The applicability of the equal energy hypothesis. *J Acoust Soc Am*, *121*, 1613–1620.

Taylor, W., Lempert, B., Pelmear, P., et al. (1984). Noise levels and hearing thresholds in the drop forging industry. *J Acoust Soc Am*, *76*, 807–819.

Theiry, L., & Meyer-Bisch, C. (1988). Hearing loss due to partly impulsive industrial noise exposure at levels between 87 and 90 dB. *J Acoust Soc Am*, *84*, 651–665.

Zhao, Y. M., Chen, S. S., Cheng, X. R., et al. (2006). Adjustment of dose-response relationship of industrial impulse noise induced high frequency hearing loss with different exchange rate. *Nat Med J China*, *86*, 48–51.

Zhu, X., Kim, J. H., Song, W. J., et al. (2009). Development of a noise metric for assessment of exposure risk of complex noises. *J Acoust Soc Am*, *126*, 703–712.