The use of the kurtosis metric in the evaluation of occupational hearing loss in workers in China: Implications for hearing risk assessment

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Abstract

This study examined: (1) the value of using the statistical metric, kurtosis [β(t)], along with an energy metric to determine the hazard to hearing from high level industrial noise environments, and (2) the accuracy of the International Standard Organization (ISO-1999:1990) model for median noise-induced permanent threshold shift (NIPTS) estimates with actual recent epidemiological data obtained on 240 highly screened workers exposed to high-level industrial noise in China. A cross-sectional approach was used in this study. Shift-long temporal waveforms of the noise that workers were exposed to for evaluation of noise exposures and audiometric threshold measures were obtained on all selected subjects. The subjects were exposed to only one occupational noise exposure without the use of hearing protection devices. The results suggest that: (1) the kurtosis metric is an important variable in determining the hazards to hearing posed by a high-level industrial noise environment for hearing conservation purposes, i.e., the kurtosis differentiated between the hazardous effects produced by Gaussian and non-Gaussian noise environments, (2) the ISO-1999 predictive model does not accurately estimate the degree of median NIPTS incurred to high level kurtosis industrial noise, and (3) the inherent large variability in NIPTS among subjects emphasize the need to develop and analyze a larger database of workers with well-documented exposures to better understand the effect of kurtosis on NIPTS incurred from high level industrial noise exposures. A better understanding of the role of the kurtosis metric may lead to its incorporation into a new generation of more predictive hearing risk assessment for occupational noise exposure.

Keywords: *Gaussian noise, kurtosis, noise-induced permanent threshold shift, non-Gaussian noise*

Introduction

Experimental and epidemiological data from animal and human noise exposures indicates that the current noise exposure criterion (International Standard Organization; ISO-1999:1990)[1] underestimates the amount of noise induced hearing loss (NIHL) acquired by workers exposed to complex industrial noises. These results emphasize the inadequacy of current methods of measuring and evaluating noise exposures for the purpose of hearing conservation. The difficulty lies in our inability to identify the necessary and sufficient metrics that need to be measured in order to be able to estimate hearing loss from continued long-term exposure. Current hearing risk assessment as embodied in the ISO-1999:(1990)[1] document are based on data for NIHL and high level noise exposures acquired several decades ago using what by today's standards would be considered primitive technology. At the time the demographic data were incorporated into ISO-1999, energy metrics were the only metrics that could be measured from a noise environment with noise survey equipment. Thus, in the ISO-1999 document, all noise exposures are quantified by a single metric, that is a time-integrated pressure squared or energy metric incorporating a spectral weighting. However, the energy metric of time-integrated pressure squared, with or without spectral weighting (A-scale), is insufficient to characterize complex* non-Gaussian (non-G) industrial noise exposure.

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Thus, there is a need to better understand how to measure and evaluate noise exposures to better predict potential NIHL for hearing conservation purposes.

Temporal characteristics of noise exposure are known to affect hearing,[2-8] yet temporal variables are not incorporated into noise energy metrics, where infinite number of very different noise exposures can all have the same equivalent noise level (Leq). Noise metrics sensitive to temporal variation need to be incorporated into current measures. One metric sensitive to temporal noise characteristics is the kurtosis statistic. Kurtosis, β (t), is defined as the ratio of the fourth-order central moment to the squared second-order moment of the amplitude distribution. Kurtosis is a metric that can be used to quantify the departure from normality. For a normal distribution β (t) = 3 whereas distributions that are outlier prone have values of β (t) =>3. Examples of the former are Gaussian (G) noises or unvoiced fricatives (/s/, /f/, /sh/) while examples of the latter are running speech, impulse/impact noise, and complex noises. Complex noises are common in industrial environments where they consist of combinations of impact and continuous G noise, i.e., they are non-G. All the variables that characterize a non-G noise (e.g., transient peaks, inter-transient intervals, transient durations, crest factor etc.,) have an effect on the kurtosis value. This is necessary since there is no agreed upon method to predict the hazard to hearing in individuals resulting from complex occupational noise exposures,[9-14] That is, a valid and reliable method is needed in the construction of a noise dose resposne relation (DRR) for hearing conservation purposes. The main reason for this is the absence of published data on human NIHL that include sufficient numbers of workers with well-documented exposures. After many years of research, the question remains as to how current standards, developed from the results of steady-state exposures, and quantified by weighted energy alone, predict NIHL to complex noise environments. Studies which have shown both support for and against the equal energy hypothesis (EEH) that forms the basis of current hearing risk assessment for noise exposure, suggest that an energy approach is not sufficient to characterize a complex noise exposure.

If we can place the predictive value of both energy and kurtosis on a sound scientific footing we may be able to develop a very precise approach to the measurement and evaluation of noise environments. While there is currently no consensus on how to incorporate complex noise characteristics into predicting hearing hazard,[9-14] our own animal studies have shown that the kurtosis [β(t)] metric may be a reasonable candidate to adequately predict the risk of hearing loss from complex noise.[5-8] These studies demonstrate that: (1) kurtosis ordered the extent of hearing and sensory cell loss from a variety of complex noise exposures, i.e., for a fixed energy level, the noise-induced trauma increased as the kurtosis increased; (2) non-G exposures are more hazardous than G exposures of equivalent energy and the hazard is identified by the kurtosis, and (3) the EEH applies to exposures of the same class as defined by the kurtosis. This single result represents a significant advance in our understanding of the EEH and its application to noise standards.

The kurtosis statistic can be used to estimate the deviation of a distribution from the G, and can be computed on a filtered wave form to obtain a frequency specific kurtosis β(f) or on the unfiltered time-domain signal to obtain an overall kurtosis β(t). The kurtosis β(t) can then be associated with the Leq while β(f) can be associated with a frequency specific spectral energy Leq(f). It is possible that kurtosis may effectively discriminate the risk of hearing and sensory cell loss for noise exposures with equal energy but different temporal characteristics, a fundamental problem with current hearing conservation programs. If the human data that are collected reinforce our animal model data then a new approach to measuring industrial noise for hearing conservation purposes may be developed.[5-8]

Significant limitations of both the ISO-1999 and American National Standard Institute[1] models of noise-induced permanent threshold shift (NIPTS) have been associated with their inability to accurately estimate NIPTS to non-G noise. Both the National Institute of Occupational Safety and Health (NIOSH)[15] and the Committee on Hearing, Bioacoustics, and Biomechanics[11] working groups have acknowledged this and have recommended that such data be acquired. The NIOSH[15] document "Criteria for a Recommended Standard: Occupational Noise Exposure, Revised Criteria" for instance, emphasized the lack of data on the effects of temporal variables especially when the noise environment is non-G. The NIOSH[15] also acknowledged the need to improve the way in which current noise exposure standards (ISO-1999) predicts the probability of acquiring a NIPTS and the magnitude of NIPTS incurred from a given noise exposure. The ISO-1999 document, which incorporates field studies of NIPTS and studies of age-related hearing loss has also been criticized by the NIOSH[15] for data that is based on "mixed quality" which "constrain the ability to establish a widely accepted hearing risk assessment for impulsive and other sounds."

Compounding the issue above is the relative lack of research and conflicting results in the literature which attempt to evaluate the predictions of ISO-1999 for median NIPTS in workers exposed to diverse industrial noise environments. A few studies have evaluated the accuracy of how ISO-1999 quantifies risk of NIPTS based on a retrospective analysis of the 1968-1972 Occupational Noise and Hearing Survey (ONHS).[16-18] Some caution must be exercised however, when comparing the noise exposure and thresholds measures from the ONHS population to current populations since the ONHS database was developed using audiometric and noise measuring instrumentation available over 30 years ago.

Prince[17] cautioned against using the ISO-1999 model for populations exposed to "intermittent or highly variable exposure conditions" since the model assumes that workers were exposed to steady-state noise. Some studies have reported that the ISO-1999 model "underestimated" the risk or amount of NIPTS in workers exposed to high level industrial noise[16,17,19-22] whereas Dobie[18] using the ONHS data reported reasonable agreement between the high frequency median NIHL estimates with the predictions of ISO-1999.

Experimental animal data have challenged the validity of the ISO-1999 and ANSI S3.44 database and have indicated that non-G noise is more hazardous to hearing than G noise of the same spectrum and SPL.[5-8,10,12,13] Human demographic data[9,17-19] also challenge these measures. Considering that many industrial noise environments are non-G (e.g., impacts or noise bursts, intermittent and/or time-varying) and that energy metrics (e.g., Leq) are suitable for G noise, there is a need to evaluate alternative metrics or combination of metrics which have been found to be appropriate for assessing non-G noise environments.

Our current study was a clear extension of our animal experiments.[5-8] It allows us to test our findings in a human population to better understand: (1) the predictive accuracy of the ISO-1999 model for estimating median NIPTS in workers predictably exposed to a variety of industrial noise environments, and (2) how to measure and evaluate noise exposures for the purpose of hearing conservation, i.e., evaluate the kurtosis metric as an indicator of hazard resulting from the temporal features of industrial noise environments. To accomplish these objectives, we compared the ISO-1999 approach for determining median NIPTS estimates with actual recent epidemiological data obtained on 240 highly screened workers exposed to high-level noise in Chinese industry.

*In this study the term "complex noise" refers to non-Gaussian noise, which in the course of the work cycle is interrupted, intermittent and time varying in level.

Methods

Study design

A cross-sectional approach was used in this study. The four main study procedural elements were: (1) select workplaces based upon consistent noise and employment characteristics; (2) select and recruit subjects based upon strict quality criteria (i.e., health history and stable employment criteria); (3) obtain shift-long temporal wave forms of the noise that workers were exposed to for evaluation of noise exposures on selected subjects; and (4) obtain audiometric testing on all selected subjects. The details of each approach are addressed below.

Workplace selection for inclusion in the study

Workplace selection for this study was based upon criteria designed to assure necessary G and non-G noise exposure and a sufficient subject pool meeting subject criteria. Each workplace included in the study had: (1) a workforce that was stable over a number of years; (2) work processes and machinery that were stable for a number of years; (3) sufficiently high G and non-G noise exposure work areas, and (4) no use of hearing protective devices (HPD) by workers prior to the recent use of HPDs mandated in the last one-two years of employment. These selection criteria were designed to facilitate accurate cumulative noise exposure assessments over each worker's career. In these industries, workers are employed in high level noise environments which include a wide range of energy [L_{Aeq8h}= 85 to <94dBA]; kurtosis level [($\beta(t)$ = 3–196; mean = 30.5, s.d = 26.1], and exposure duration (1–30 year's). Industries were selected that were known to produce some of the highest noise levels with corresponding high levels of kurtosis. Most of the noise environments in these industries consist of what we have defined as complex noise i.e., refers to non-G noise that in the course of a work cycle, may be intermittent, interrupted and of variable level.

Subject selection and recruitment

A total of 240 workers were selected from two industries in Shiyan, Hubei province of China. Two hundred seven subjects (36.02.1 ± 4.93 years old, 125 males and 82 females) were exposed to complex noise (non-G; median [$\beta(t)$ ≥4] for 14.9 ± 5.9 years. The remaining 33 subjects (36.5 ± 5.08 years, 20 males and 13 females) were exposed to a continuous noise (G; median $\beta(t)$ <4) for 16.04 ± 6.2 years. All candidate subjects were required to complete a noise exposure and health questionnaire modified from Dobie[23] which was followed by a face-to-face interview for quality control (to clarify and affirm responses). Subject criteria were designed to assure that observed hearing loss is directly attributed to the measured industrial noise exposures and not related to other medical problems. The ideal subject is one whose high-level noise exposure was limited to that experienced on the job and who has been employed at that same job for his entire employment history.

The duration of the workers exposure to noise without the use of HPD's was determined from the noise exposure questionnaire and interview. The subjects in this study used HPDs only in the last one-two years of employment. All subjects met strict criteria. They must have consistently worked within the same job category and work (noise exposure) area for their entire current employment, and were excluded from the study for any of the following reasons: (1) prior employment (with a different employer) in a high noise environment; (2) military service or shooting activities; (3) reported hearing complaints; (4) present auditory symptoms; (5) family history of hearing loss; (6) history of ear disease; (7) use of ototoxic drugs; and

(8) history of diabetes. As self reports of prior noise exposure and hearing problems can be unreliable, personnel medical and employment history records were reviewed in conjunction with questionnaire responses and interviews in evaluating whether potential participants were acceptable. These criteria helped to ensure that the observed change in the subjects hearing threshold level during his/her employment in the target industries were primarily attributable to the subject's age, length of time in noise, and the noise measured during the study. A review of their work history records on file was performed to ensure that their current position is their first job with high-level noise exposure.

Compounding the problem of predicting NIHL over a working lifetime are the use of HPD, changes in production technology, and worker job instability. While the implementation of hearing conservation programs is an important and necessary practice, each worker's occupational exposure is attenuated in an unpredictable fashion, interfering with the effective estimation of noise exposure over time for experimental studies. Similarly, worker mobility and changes in machinery within a job also make historic noise estimation difficult. These issues have been addressed in this study.

Real-time sound recording: Collection and analysis of noise exposure waveforms

Personal noise dosimeters (Hangzhou Aihua Instruments Co. AWA5610B, class II) were used to collect noise exposure levels on individuals. The dosimeters were equipped with a microphone (Aihua, Hangzhou, AWA14421) fixed on the collar of each subject. The dynamic range of the dosimeters was 40 to 140 dB (A). The noise dosimeters could work continuously for fifteen hours. Leq(A) was recorded and calculated every two seconds. Prior to any measurements each dosimeter was calibrated using a 94 dB SPL, 1 kHz tone (Aihua Instruments, Model AWA6221B calibrator). Noise data collected in the dosimeter were transferred to an IBM-compatible computer. The eight-hour continuous equivalent, A-weighted sound pressure level [$Leq(A)_{8h}$] was calculated for each measurement. The workers were divided into 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 eight or ten hours. In the complex noise environment (non-G), subjects were measured over the course of one entire work shift. The $Leq(A)_{8h}$ for each subject in a group was averaged to produce a group mean daily noise exposure level [dB(A) SPL]. The statistical metric "kurtosis", which quantifies the 'peakedness' of an amplitude distribution was determined for the G and non-G noise exposure using a digital recorder (Kenwood, MGR-A7) connected to a dosimeter (Aihua, AWA5610B) to collect real-time samples of each subject's noise exposure in each industrial plant. Recordings were made at the level of the subject's ear. Full-shift (~8 hrs) noise for individual was recorded using MGR-A7 with 16 bit resolution at a 48 kHz sampling rate and fed to an IBM computer for subsequent analysis. The kurtosis of the noise was computed over consecutive 40-second time windows of each full-shift noise record using MATLAB software and the mean or median values were used to establish the kurtosis values for each series of noise records.

A detailed analysis of the temporal noise record that workers were exposed to and their audiograms was made to compare the actual NIHL in each worker with the predictions in ISO-1999. Noise environment characterization was achieved through: (1) full-shift noise signatures measured for each subject, (2) multiple subjects from each job category, and (3) historical review of each included job category to assure that no major changes in work organization or machinery used have taken place. Thus we were able to account for within and between worker variability for each job and construct a reasonably stable measure of noise exposure over the historical period covered by selected workers. Complete work shift noise waveforms were recorded for all subjects. Each subject had personal noise exposure data collected twice within the course of one month. The second record was used to make a rough estimate of exposure variability. Each shift-long personal noise exposure evaluation included two types of noise monitoring equipment. The first is a standard noise dosimeter (AWA 5610B) adapted to an output line of the microphone (AWA 14421). The second digital recorder (Kenwood MGR-A7), adapted to a precision dosimeter (AWA 5610B), obtained a full waveform record of the subject's noise exposure over a single work shift (~ eight hour) period. Both the dosimeter and recorder was calibrated before and after each sampling with a sound calibrator (AWA6221A) according to the manufacturer's instructions.

The microphone was placed on the collar of each subject at the beginning of an eight-hour work shift and collected at the end of the shift. Since the reliability of the noise data depends on the subject's cooperation in the proper use of the recorder, each subject was informed about the purpose of measurement and the importance of wearing it continuously for the full measurement period. The importance of the accuracy of noise data in assessing the need for noise control and the consequences of tampering with the microphone (shouting into it or knocking it on doors, etc.) was explained. With few exceptions, noise records were artifact free. Such artifacts will affect the value of kurtosis in the analysis window and skew the analytical results. Thus the recorded noise data needs to be preprocessed prior to analysis to eliminate these artifacts.

An artifact detecting and eliminating program was used to automatically track and delete artifact segments in the recorded noise data. Data were entered from the subject's history/medical questionnaire into a database using Epidata

3.1 software. The duplicated database was checked for errors and sent into an SPSS (version 13) software package for subsequent analysis.

Software was developed for analyzing the eight-hour long noise waveforms collected on each subject. The software is designed to extract: (1) the frequency and time domain kurtosis, a statistical metric shown to correlate with NIPTS,[5-8] and (2) the joint peak-interval histogram from complex noise environments. Conventional level and spectral metrics are also extracted in a variety of formats. These metrics taken together provide considerable temporal information that complements the energy and spectral metrics when the latter are insufficient to determine the hazard of a noise exposure.

Audiometric testing

Each subject had two audiometric examinations within one month. The second audiogram was used as a check on the initial measurements. Any discrepancy of more than 10 dB at any frequency necessitated additional testing and investigation and possible rejection of the subject. All audiometric testing took place immediately following a two-day respite from the industrial environment. These examinations included air-conduction pure-tone audiometry at. 5, 1, 2, 3, 4 and 6 kHz in each ear. In addition, an otologic examination and tympanometry testing were conducted to rule out conductive hearing impairment in each ear. Pure-tone audiometric thresholds were measured with an automatic microprocessor clinical audiometer (MADSEN ITERA) calibrated to appropriate ISO standards[24] and equipped with TDH-39 headphones mounted in MX-41/AR cushions. These tests were conducted in an audiometric sound suite with background sound pressure levels at or below the OSHA maximum allowable octave-band levels for audiometric test rooms. Prior to their day of testing each subject was instructed to avoid all sources of loud noise (e.g., loud music, gunfire, machinery, etc.) for the 48 hours prior to testing.

Statistical analysis

The median NIPTS for each noise exposed group was compared to the ISO-1999 predicted median NIPTS using a one-way analysis of variance (ANOVA) with repeated measures on one factor (frequency) and post-hoc paired comparisons (Fisher Protected Least Significant Difference Test). A statistical significance level of $P < 0.05$ was adopted for all analyses. Post hoc testing which showed the differences between the NIPTS for the noise exposure and ISO prediction to be significant ($P < 0.05$) is indicated by an asterisk (*) in Table 1. The median difference statistic determined median NIPTS. The predicted ISO-1999 threshold for each subject was determined using the equation described in the ISO-1999 document (ANSI S3.44-1996, Section 6.3)[1] as described below. Statistically significant effects of frequency were expected and found in all of the analyses because of the frequency-specific nature of the audibility curve and the noise exposure stimulus which caused greater NIPTS at some frequencies. For this reason, main effects of frequency are not addressed in the presentation of the results.

Evaluation of ISO-1999 median NIPTS estimates

A database composed of the worker's shift-long temporal noise waveform and associated audiometric results was developed and compared to the ISO-1999 predictions for median NIPTS. Hearing threshold levels at each test frequency (2, 3, 4 and 6 kHz) were adjusted by subtracting averaged hearing threshold levels in age and gender matched population adapted from ISO 1999:1990 Annex B (derived from an audiometric survey of the US population in 1960 to 1962).[25] Threshold results at 0.5 and 1 kHz were not reported since very little if any NIPTS was observed at these frequencies from the noise exposure conditions reported in the present study. Furthermore, NIOSH[15] recommended that 0.5 kHz no longer be tested in hearing conservation practice. Consistent with NIOSH reports, binaural average thresholds were determined for all subjects across the test frequencies. Median thresholds were then calculated for each noise exposed group.

The subjects were exposed to only one occupational high level complex noise and had a limited (past one-two years) or no history of hearing protection. Thus we were assured that the observed hearing loss was directly attributed to the measured industrial noise exposures. Subjects were partitioned into separate groups for three noise exposure level ranges (L_{Aeq} = 85- <88; 88- <91; 91- <94), and two exposure durations (≤10 years and >10 years). For each exposure condition defined by exposure level and duration, subjects were further partitioned into one of two groups based on the kurtosis level [$\beta(t)$ ≤10 or >10] of the noise. A similar analysis was performed on the entire subject population (N = 240) exposed to noise levels ranging from L_{Aeq} = 85 to <94 and exposure durations ranging between 1-30 years. For this analysis, the subjects were partitioned into one of two groups based on the kurtosis level [$\beta(t)$ <4 or ≥4] of the noise. By partitioning the subjects into groups represented by $\beta(t)$ <4 and ≥4, we attempted to compare the effect produced by G [$\beta(t)$ <4], and non-G [$\beta(t)$ ≥4] noise on median NIPTS for several exposure conditions. The predicted ISO-1999 thresholds for each subject was determined using the equation described in the ISO-1999 document (ANSI S3.44-1996, Section 6.3) for exposure times between 10 and 40 years [i.e., median potential NIPTS $N_{0.50} = [\mu + v\log(\Theta/\Theta_0)](L_{A8hn} - L_0)^2$] and less than 10 years [i.e., median potential NIPTS ($N_{0.50;\Theta,<10} = \log(\Theta + 1)/\log(11) N_{0.50;\Theta,=10}$). This calculation was also made to derive the ISO-1999 predictions for median NIPTS for each noise exposure condition classified by the L_{Aeq8h} and exposure duration. This approach allowed us to compare the ISO-1999 model prediction estimates for median NIPTS for each exposure condition to the median NIPTS incurred by the subjects for the same exposure condition. This was done to determine the accuracy of the ISO predictions for a variety of noise exposure conditions at each kurtosis level, e.g., [β(t) = ≤10 and >10], and G [β(t) = <4], and non-G (β(t) = ≥4]. This approach allowed us to better understand the: (1) relative contribution of the temporal features (i.e., kurtosis) of the noise exposure on NIPTS, and (2) degree of correspondence between the ISO-1999 prediction estimates for median NIPTS and that incurred for different group exposure conditions classified by L_{Aeq8h}, duration and kurtosis level. The median NIPTS group results for subjects in each noise condition (i.e., defined by L_{Aeq8h}, and exposure duration) were determined for both the mean and median noise exposure kurtosis level range of β(t) = ≤10 and >10, and β(t) = <4 and ≥4. The mean and median noise exposure kurtosis level

Table 1: The median (50th percentile) and extreme (10th and 90th percentile) NIPTS as a function of noise exposure level and duration, and median and mean kurtosis level in workers in chinese industry

Work duration	Kurtosis	L_{Aeq} range	*N*	Percentile	Frequency (kHz)			
					2	3	4	6
Duration≤10 years (G1)	Median kurtosis≤10	85≤L_{Aeq}<88	6	50th percentile	11.5*	14.75*	15.5*	10*
				10th percentile	7	1.75	0	0
				90th percentile	16.25	21	22.25	13
				Mean/s.d.	11.58/4.31	12.50/9.03	12.58/10.41	7.67/6.22
			7	ISO prediction	0.79	1.83	5.99	3.49
Duration>10 years (G2)	Median kurtosis≤10	85≤L_{Aeq}<88	31	50th percentile	12*	11.5*	16.5*	7.5
				10th percentile	8	1	2	0
				90th percentile	20.5	34.5	36.5	29
				Mean/s.d.	14.24/9.22	14.82/16.04	19.85/16.97	12.90/16.72
	Median kurtosis>10		10	50th percentile	12.25*	15*	22.25*	15.5*
				10th percentile	8.25	5.95	3.45	1.85
				90th percentile	21.3	25.55	34.5	40.25
				Mean/s.d.	14.10/6.71	15.65/10.79	21.45/14.00	18.05/17.14
			41	ISO prediction	1.53	5.25	7.52	4.51
Duration≤10 years (G3)	Median kurtosis≤10	88≤L_{Aeq}<91	10	50th percentile	10.25*	9.25*	15.5*	9.5*
				10th percentile	5.5	1	2.25	0.45
				90th percentile	18.75	15.5	33.65	32.05
				Mean/s.d.	12.25/7.38	9.80/9.62	16.30/13.39	15.90/17.58
	Median kurtosis>10		10	50th percentile	11.5*	14.25*	20.5*	16*
				10th percentile	7.95	6	13.75	9.65
				90th percentile	26.4	22.65	31.2	24.55
				Mean/s.d.	15.50/8.01	13.94/7.88	21.61/14.51	16.61/10.22
			20	ISO prediction	1.37	3.21	8.24	5.12
Duration>10 years (G4)	Median kurtosis≤10	88≤L_{Aeq}<91	43	50th percentile	13*	14.5*	19*	20*
				10th percentile	6.3	2.2	3.7	2.5
				90th percentile	20.5	48.3	44.6	34.1
				Mean/s.d.	15.38/11.32	18.55/16.62	22.43/16.00	18.05/13.51
	Median kurtosis>10		27	50th percentile	11*	11.5	20*	13.5*
				10th percentile	6	2.5	3	0
				90th percentile	19.9	37	49	36.6
				Mean/s.d.	11.89/5.53	17.33/14.86	23.46/18.92	19.11/18.69
			70	ISO prediction	3.14	8.67	11.37	7.31
Duration≤10 years (G5)	Median kurtosis>10	91≤L_{Aeq}<94	16	50th percentile	11.5*	9.25	8.75	18.25*
				10th percentile	6.75	1	2.5	0.5
				90th percentile	20.25	32.25	36.75	51.25
				Mean/s.d.	13.53/9.16	13.47/13.48	15.75/17.37	20.38/20.75
			17	ISO prediction	6.61	13.08	8.93	2.83
Duration>10 years (G6)	Median kurtosis≤10	91≤L_{Aeq}<94	31	50th percentile	10.5*	11	13.5	15
				10th percentile	6	1	2	3.5
				90th percentile	20.5	31	36.5	29
				Mean/s.d.	12.53/7.66	13.53/12.40	17.73/14.33	16.15/11.28
	Median kurtosis>10		38	50th percentile	10.75*	12.5	16.75	11.25
				10th percentile	7.4	3.85	5.05	2.05
				90th percentile	17.65	30.25	44.2	37.8
				Mean/s.d.	11.70/3.88	15.11/11.30	22.08/16.64	16.30/15.34
			69	ISO prediction	5.05	13.11	16.40	11.16

(Contd...)

Table 1: *Continued*

Work duration	Kurtosis	L_{Aeq} range	*N*	Percentile	Frequency (kHz)			
					2	3	4	6
Duration>10 years (G7)	Mean kurtosis≤10 `	85≤L_{Aeq}<88	9	50th percentile	12*	6	14*	2.5
				10th percentile	7.1	1	1.2	0
				90th percentile	20.1	26	24.1	15.8
				Mean/s.d.	12.28/5.56	10.61/9.92	13.11/9.53	6.72/9.52
	Mean kurtosis>10		32	50th percentile	12*	12.75*	22.25*	9*
				10th percentile	8.05	1.1	3.05	2
				90th percentile	22.75	38.55	37.85	53.35
				Mean/s.d.	14.75/9.28	16.27/15.82	22.25/17.15	16.25/17.84
			41	ISO prediction	1.53	5.25	7.52	4.51
Duration≤10 years (G8)	Mean kurtosis>10	88≤L_{Aeq}<91	19	50th percentile	10.5*	11*	20*	13*
				10th percentile	7.1	1	2.5	1.7
				90th percentile	26.8	23.3	33.8	31.3
				Mean/s.d.	13.79/765	12.16/8.94	19.61/13.85	15.89/14.66
			20	ISO prediction	1.37	3.21	8.24	5.12
Duration>10 years (G9)	Mean kurtosis≤10	88≤L_{Aeq}<91	10	50th percentile	11.75*	7.5	12.5	7.75
				10th percentile	5.7	0.9	1.35	0
				90th percentile	18.25	26.15	27.45	21.45
				Mean/s.d.	11.60/5.74	11.35/12.71	15.05/13.94	9.80/10.86
	Mean kurtosis>10		60	50th percentile	13*	15.25*	20.25*	20.25*
				10th percentile	6	3.35	3.9	3.4
				90th percentile	20.75	49.75	50.5	36.6
				Mean/s.d.	14.44/10.10	19.20/16.15	24.13/17.28	19.90/15.86
			70	ISO prediction	3.14	8.67	11.37	7.31
Duration≤10 years (G10)	Mean kurtosis>10	91≤L_{Aeq}<94	17	50th percentile	12.5*	10	10	19.5*
				10th percentile	7	1	2.5	0.6
				90th percentile	20.2	31.5	36.6	50.6
				Mean/s.d.	13.47/8.87	13.71/13.09	15.85/16.83	20.35/20.09
			17	ISO prediction	2.83	6.61	13.08	8.93
Duration>10 years (G11)	Mean kurtosis>10	91≤L_{Aeq}<94	63	50th percentile	11	11.5	15.5	12.5
				10th percentile	6	1.2	3.2	2.6
				90th percentile	18.4	30.7	41.3	33.3
				Mean/s.d.	12.18/5.25	14.39/11.54	20.22/15.57	15.47/13.02
			69	ISO prediction	4.97	13.07	16.38	11.14
Duration 1~30 years (G12)	Median kurtosis≤4	85≤L_{Aeq}<94	33	50th percentile	10.25	10	13.75	10.5
				10th percentile	6	0.1	1.6	2.5
				90th percentile	19.6	26.45	35.5	36.6
				Mean/s.d.	11.67/6.11	12.39/11.98	17.48/14.92	14.31/13.45
	Median kurtosis>4		207	50th percentile	12*	11.5	17.5*	13*
				10th percentile	6	1	2.5	0.5
				90th percentile	20.5	35.1	43	37.5
				Mean/s.d.	13.41/8.07	15.57/13.81	20.67/16.31	16.87/15.42
			240	ISO prediction	3.94	9.93	13.21	8.84

range metrics were both evaluated to better understand their relative degree of correspondence for median NIPTS for: (1) two different kurtosis level ranges [e.g., $\beta(t) = \leq 10$ and >10, and $\beta(t) = <4$ (G) and ≥4 (non-G)] and, (2) the ISO-1999 prediction estimate with epidemiological data obtained on 240 subjects divided among several noise exposure conditions. It should be noted that the different nature of each statistic (i.e., mean vs. median) is reflected in the differences reported for median NIPTS in (1) and (2). This analysis was performed using both the mean and median kurtosis statistic of the noise level to evaluate each statistic on (1) and (2) above.

Results

Evaluation of ISO-1999 median NIPTS estimate predictions

Figure 1 (Panels G1-G11) compares the ISO-1999 median NIPTS predictions with the median NIPTS incurred by subjects for the variety of noise exposures indicated by L_{Aeq} range (L_{Aeq} = 85- <88, L_{Aeq} = 88- <91, and L_{Aeq} = 91- <94) and duration (≤10 or >10 years). For each exposure condition, subjects were divided into two groups based on the mean and median kurtosis levels [$\beta(t) = \leq 10$ and/or >10] of the noise.

In a few exposure conditions only one kurtosis level was reported due to a lack of subjects. A similar analysis shown in Figure 2 (Panel G12) compares the ISO-1999 median NIPTS predictions with the mean and median NIPTS incurred by the total population of 240 subjects for L_{Aeq} = 85 - <94 and 1-30 years exposure duration. This population was divided into two groups based on the median kurtosis level of the noise [β(t) = <4 (G) or ≥4 (non-G)]. Summary data for the groups defined in Figures 1 and 2 are represented in Table 1. For all noise exposures where comparisons between the ISO-1999 median NIPTS predictions and high kurtosis noise [β(t) = >10] exposed groups could be made, the ISO predictions significantly ($P < 0.05$) under estimated the NIPTS for both the median (5 out of 5 group comparisons; panels G2-G6) and mean (4 out of 5 group comparisons; panels G7-G10) kurtosis groups. Similarly, for all noise exposures where comparisons between the ISO-1999 median NIPTS predictions and low kurtosis noise [β(t) = ≤10] exposed groups could be made, the ISO predictions significantly ($P < 0.05$) underestimated the NIPTS for both the median (4 out of 5 groups; panels G1-G4) and mean (1 out of 2 groups; panel G7) kurtosis groups. Post hoc testing showed the differences to be significant ($P < 0.05$) across most of the test frequencies as indicated by an asterisk (*) in Table 1.

The results in Figure 1 and Table 1, which show that the ISO-1999 predictions underestimated the median NIPTS by up to ~ 15 dB in workers exposed to the industrial noise exposure conditions in this study, was reasonably consistent in the two lower noise level exposure groups (L_{Aeq} = 85- <88;

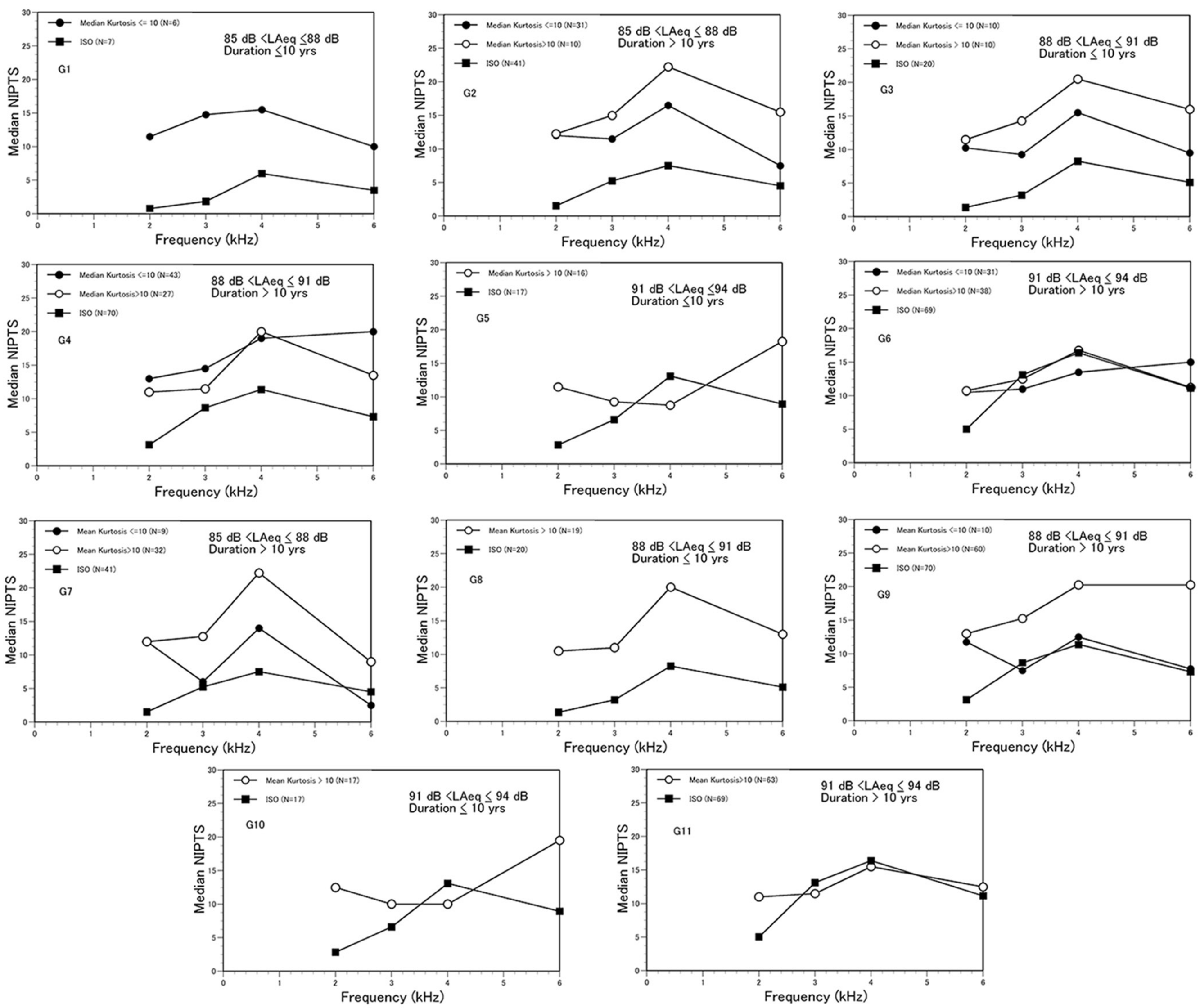

Figure 1: Panels G1-G11 show a comparison of the ISO-1999 median NIPTS predictions with the median NIPTS incurred by the subjects for several noise exposure conditions at the indicated L_{Aeq} range (L_{Aeq} = 85- < 88, L_{Aeq} = 88- < 91, or L_{Aeq} = 91- < 94) and exposure duration (≤ 10 or > 10 years). For each exposure condition, subjects were divided into two groups based on the mean and median kurtosis levels [β(t) = ≤10 and/or > 10] of the noise

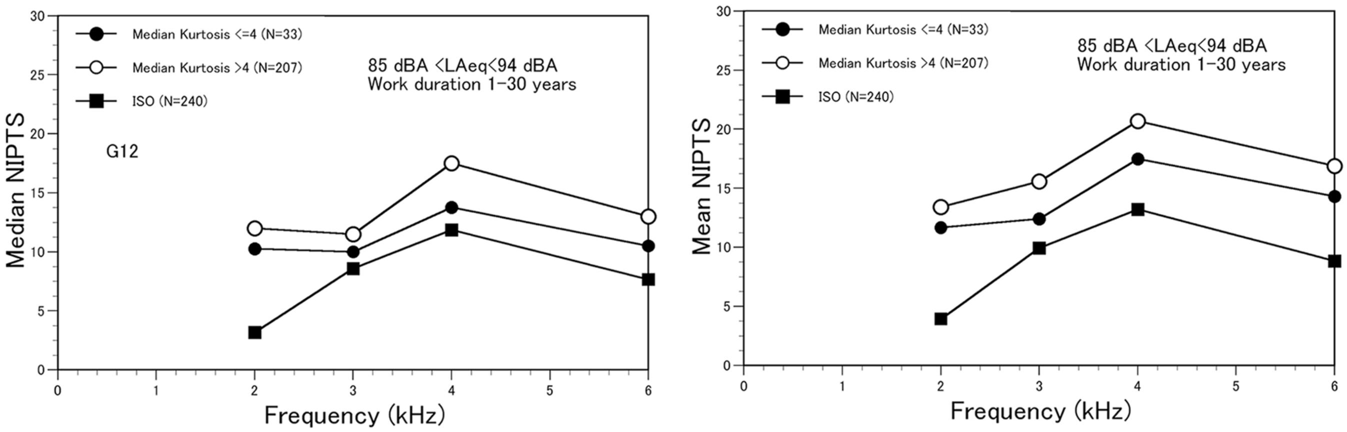

Figure 2: Panel G12 shows a comparison of the ISO-1999 median NIPTS predictions with the median and mean NIPTS incurred by the total population at the indicated L_{Aeq} range (L_{Aeq} = 85- < 94) and exposure duration (1–30 years). For each exposure condition, subjects were divided into two groups based on two median kurtosis levels [β(t) = <4 and ≥ 4] of the noise

and L_{Aeq} = 88- <91) across the test frequencies. In the higher noise level group (L_{Aeq} = 91- <94), the ISO-1999 predictions underestimated the median NIPTS by up to ~10-15dB at 2 and/or 6 kHz with similar results occurring at 3 and 4 kHz. The extent by which the ISO model underestimated the group median NIPTS was slightly greater (Mean = 2.1 dB) for the higher median kurtosis noise level [β(t) = ≥10; Mean = 8.8 dB] than for the lower median kurtosis noise level [β(t) = ≤10; Mean = 6.7 dB] across the test frequencies. A similar result was observed for the mean kurtosis statistic, i.e., the extent by which the ISO model underestimated the group median NIPTS was greater (Mean = 6.8 dB) for the higher mean kurtosis noise level [β(t)≥10; mean = 9.8 dB] than the lower mean kurtosis noise level [β(t) ≤10; mean = 3.0 dB]. The ISO median NIPTS predictions were more closely aligned (average of ~5 dB across the test frequencies) with the median NIPTS for the mean kurtosis than the median kurtosis statistic across the exposure conditions in this study. Figure 2 (Panel G12) and Table 1 show the median NIPTS results for the entire population (*N* = 240) divided by kurtosis level for G[β(t) = <4] and non-G [β(t) = ≥4] noise exposures ranging by level (L_{Aeq} = 85 - <94) and duration (1-30 years). The ISO-1999 predictions significantly [$F(2,643)$ = 10.60, $P < 0.05$] underestimated the median NIPTS at 2, 4, and 6 kHz for the high kurtosis level [β(t) = >4] group but not for the low kurtosis level group [β(t) = <4]. The ISO-1999 model: (1) consistently underestimated the mean and median NIPTS for each kurtosis level group across the test frequencies, and (2) was more closely aligned with the mean and median NIPTS incurred by the lower [β(t) = <4; mean = 2 dB] than higher [β(t) = ≥4; mean = 4.5 dB] kurtosis level group across the test frequencies, i.e., the ISO-1999 model underestimated the mean and median NIPTS by up to 10 dB for the higher kurtosis level group [β(t) = ≥4] across the test frequencies.

The effect of kurtosis level on NIPTS

The effect of kurtosis level on NIPTS for β(t) = ≤10 and >10, and for β(t) = <4 (G) and ≥4 (non-G) is shown in Figures 1 (Panels G1-G11) and 2 (Panel G12), respectively. Summary data for the groups defined in Figures 1 and 2 are represented in Tables 1 and 2. The results show that the median NIPTS increased by an average of 3 dB (range 0-11 dB) as the median kurtosis level increased from β(t) ≤10 to >10 and by an average of 8.1 dB (range 3-13 dB) as the mean kurtosis level increased from β(t) ≤10 to >10 across the test frequencies among the noise exposed groups. This kurtosis level effect was reasonably consistent and most pronounced for the lower L_{Aeq} exposure groups (L_{Aeq} = 85- <88; and L_{Aeq} = 88- <91) than for the higher noise level group (L_{Aeq} = 91- <94). This effect was not statistically significant (P = 0.05) for the median β(t) = ≤10 and >10 group comparisons (4 out of 4; Panels G2, G3, G4, G6). However, the amount of median NIPTS was significantly greater for the higher mean kurtosis level [β(t) = >10] than lower mean kurtosis level [β(t) = ≤10] in the exposed groups where comparisons could be made (2 out of 2) across the test frequencies (2-6 kHz) and at the test frequencies of 3, 4 and 6 kHz [e.g., Panel G7- L_{Aeq} = 85- <88 and exposure duration >10 years; $F(2,79)$ = 8.02, $P < 0.05$; and Panel G9- L_{Aeq} = 88- <91 and exposure duration >10 years $F(2,137)$ = 18.04, $P < 0.05$]. The range in median NIPTS for the β(t) = ≤10 and >10 kurtosis level groups between the 10% and 90% percentile was ~ 10-50 dB across the test frequencies and was most pronounced for those frequencies having the greatest amount of NIPTS (e.g., 3, 4, and 6 kHz).

A comparison of the median NIPTS for the groups exposed to the two kurtosis levels β(t) = <4 and ≥4 and ISO-1999 predictions is shown in Figure 2 and Tables 1 and 2. The median NIPTS increased by ~ 2-4 dB (mean = 2.4 dB) as the kurtosis level of the noise exposure increased from β(t) <4 to ≥4 across the test frequencies. This effect was not significant (P = 0.05) for the kurtosis level [β(t) = <4 to ≥4] group comparison. The high frequency median NIPTS (average of 3, 4 and 6 kHz) increased by

Table 2: Median (50th percentile) high frequency NIPTS (average of 3, 4 and 6 kHz) as a function of noise exposure level (L_{Aeq}) and kurtosis level

Kurtosis level	L_{Aeq} range	*N*	NIPTS@3-4-6 kHz (Mean/std)
3≤ β(t)<10	$85 \le L_{Aeq} < 88$	12	10.53/8.87
	$88 \le L_{Aeq} < 91$	11	12.26/11.27
	$91 \le L_{Aeq} < 94$	6	19.28/15.84
10≤β(t)<30	$85 \le L_{Aeq} < 88$	24	17.35/16.96
	$88 \le L_{Aeq} < 91$	53	17.99/14.86
	$91 \le L_{Aeq} < 94$	37	14.85/12.31
β(t)β≥30	$85 \le L_{Aeq} < 88$	12	16.94/11.59
	$88 \le L_{Aeq} < 91$	27	22.90/13.36
	$91 \le L_{Aeq} < 94$	43	18.26/12.73
β(t)β<4	$85 \le L_{Aeq} < 94$	33	14.73/12.52
β(t)≥4	$85 \le L_{Aeq} < 94$	207	17.57/13.43

The Use of the kurtosis metric in the evaluation of occupational hearing loss

2.8 dB as the kurtosis level increased from β(t) = <4 (G) to ≥4 (non-G) for the noise level range represented by L_{Aeq} = 85- <94 [Table 2]. The range in median NIPTS for the β(t) = <4 and ≥4 kurtosis level groups between the 10% and 90% percentile was ~ 15-40 dB across the test frequencies and was most pronounced for those frequencies with the greatest amount of NIPTS (e.g., 3, 4, and 6 kHz).

The effect of noise exposure level and duration on NIPTS

The mean high frequency NIPTS (3, 4, and 6 kHz) incurred among different subjects as a function of exposure duration range (1 ≤ D < 10, 10 ≤ D < 20, 20 ≤ D < 30) for the three exposure levels is illustrated in Table 3. Summary data of the results in Figures 1 and 2 shown in Table 1 also represent the effect of exposure duration (≤10 and >10 years) on median NIPTS across the test frequencies. The results show the characteristic pattern for development of NIHL over time, i.e., NIHL develops most rapidly in the first 10 years, and then decelerates with additional exposure to noise.[15,23] For the two lower level noise ranges, the median high frequency NIPTS increased by an average of 6.9 dB for L_{Aeq} = 85- <88, and by 3.9 dB for L_{Aeq} = 88- <91 as exposure duration increased from 1 ≤ D < 10 to 20 ≤ D < 30. In contrast, there was a negligible increase in NIPTS as exposure duration increased over this range for the higher noise level group (L_{Aeq} = 91- <94), i.e, may be attributed to the large variance in NIPTS in high frequency median NIPTS (up to ~50 dB) at similar durations for each exposure level. The distribution of median NIPTS among exposure levels and durations was also large e.g., the average difference between the 10% and 90% percentile was 26 dB (range from 14-33 dB). The range in median NIPTS between the 10% and 90% percentile for the same noise exposure duration was reasonably consistent (3 dB) among the three noise exposure level groups. In contrast, the range in median NIPTS between the 10% and 90% percentile for the same noise exposure level was somewhat larger (11 dB) among the three exposure durations.

Table 3: Median (50th percentile) high frequency NIPTS (average of 3, 4 and 6 kHz) as a function of noise exposure level (L_{Aeq}) and duration

Exposure duration(years)	L_{Aeq} range	*N*	NIPTS@3-4-6 kHz (Mean/std)
1≤D<10	$85 \le L_{Aeq} < 88$	7	10.10/7.82
	$88 \le L_{Aeq} < 91$	20	15.15/10.88
	$91 \le L_{Aeq} < 94$	19	16.93/13.99
10≤D<20	$85 \le L_{Aeq} < 88$	33	16.27/13.33
	$88 \le L_{Aeq} < 91$	60	20.14/15.48
	$91 \le L_{Aeq} < 94$	56	16.71/12.67
20≤D<30	$85 \le L_{Aeq} < 88$	10	17.20/20.12
	$88 \le L_{Aeq} < 91$	12	17.88/13.44
	$91 \le L_{Aeq} < 94$	14	17.41/12.18

Discussion

The role of the kurtosis metric for hearing risk assessment

The results from the present study indicated that the kurtosis metric is an important variable in determining the hazards to hearing posed by a high-level industrial noise environment for hearing conservation purposes, i.e., the kurtosis differentiated between the hazardous effects produced by G and non-G noise environments. This finding suggests that an energy metric (Leq) alone should not be used to predict NIPTS (as applied in the ISO model) since it appears to be insensitive to the effects of the temporal characteristics of a noise exposure known to be important in affecting hearing. This conclusion is based on the results which showed that the: (1) median NIPTS increased (mean = 8.1 dB; range 3-13 dB) as the mean kurtosis level increased [β(t) ≤10 to >10], and (2) amount of median NIPTS was significantly greater ($P < 0.05$) for the higher mean kurtosis level [β(t) = >10] than lower mean kurtosis level [β(t) = ≤10] at the test frequencies of 3, 4 and 6 kHz in the exposed groups where comparisons could be made (2 out of 2). Collectively, the findings from this study in humans, concomitant with the results from our experiments with animals,[5-8, 12] suggest that the use of an energy metric (Leq) in combination with the kurtosis [β(t)] of the amplitude distribution of a noise environment can be used to more accurately estimate the hazards to hearing from the diversity of complex noise environments found in industry. These results are supported by recent studies[26,27] in humans which have demonstrated the value of the kurtosis in hearing risk assessment to noise. In a study by Zhao *et al.* (2010),[26] the kurtosis metric was shown to more accurately assess the risk of developing high frequency NIHL in workers in Chinese industry exposed to high level G and non-G noise, e.g., the noise exposure SPL combined with a kurtosis correction term to match a dose-response relationship in those exposed to a G and non-G noise environment served as a “good metric for assessment of risk for NIHL”. By introducing the kurtosis variable into the temporal component of the cumulative noise exposure calculation, the two dose response curves (for G and non-G exposed groups) were made to overlap, essentially yielding an equivalent noise-induced effect for the two study groups, i.e., the kurtosis statistic quantified the deviation of the complex noise from the G. Goley *et al.*,[27] who applied the approach by Zhao *et al*.,[26] using human data to data collected in our auditory research laboratories in the chinchilla, showed that the kurtosis correction term improved the predictive accuracy (e.g., from $r^2 = 0.46$ for L_{Aeq8h} to $r^2 = 0.67$ for the kurtosis corrected constant) of the kurtosis metric on NIHL.

The results above indicate the need to develop and analyze a larger database of workers with well-documented exposures to better understand the effect of kurtosis on NIHL incurred from high level industrial noise exposures for hearing risk assessment. If we can place the predictive value of both energy and kurtosis on a sound scientific footing we may be able to develop a very precise approach to the measurement and evaluation of noise environments.

Considering the results from this study, a data acquisition and analysis strategy which incorporates the kurtosis level of the noise should be designed to take into account the diversity of industrial exposure conditions which contribute to NIHL. This is necessary since current occupational noise measurement practice relies primarily on measures such as an A-weighted Leq measure that completely ignores the temporal characteristics of complex industrial noise environments. Unfortunately there is at present no other generally accepted metric or combination of metrics to be used as an alternative to weighted energy. The NIOSH has supported research designed to investigate the effects of complex noise on hearing in an animal model. However, while the parametric approach used in these studies has given us insights into the relative importance of the noise variables in causing hearing loss, it has not had an impact on exposure criteria because we do not know how to apply this information to the industrial environment or even to what extent it applies. Other attempts have been made to develop metrics[28] that would be highly correlated with hearing loss from any industrial noise environment. However, to date the ideal metric remains elusive.

Evaluation of ISO-1999 median NIPTS estimates

The results from the present study indicated that the: (1) ISO-1999 predictive model significantly ($P < 0.05$) underestimated (up to ~ 15 dB across most test frequencies) the amount of median NIPTS in almost all noise exposed groups (9 out of 10) with both high median and mean kurtosis levels [$\beta(t) = >10$ and $\beta(t) = \geq 4$]; (2) ISO-1999 predictive model significantly ($P < 0.05$) underestimated (up to ~ 10-15 dB at 2 and/or 6 kHz) the amount of median NIPTS in the majority of noise exposed groups (5 out of 7) with both low median and mean kurtosis levels [$\beta(t) = \leq 10$], and (3) the ISO-1999 median NIPTS predictions were more closely aligned (average of ~5 dB across the test frequencies) with the median NIPTS for the mean kurtosis than the median kurtosis statistic across the exposure conditions in this study. The different nature of each statistic (i.e., mean and median) is reflected in the differences for median NIPTS reported between the predicted ISO estimates and that incurred by the noise exposed groups.

The results in the present study noted above are consistent with several studies which reported the ISO model to underestimate the amount of NIPTS. For example, hearing thresholds of railway workers were reported to exceed ISO-1999 predictions by 9 dB over 2 and 4 kHz.[20] Carlsson *et al*.,[21] who conducted an audiometric analysis in 1200 noise-exposed workers in Sweden, reported that in seven out of nine subgroups, the audiometric curves fell above 0.1 fractile of the ISO model and in only two groups they were at or below 0.1 fractile predicted with the ISO model. The NIOSH,[29] who compared the excess of risk of hearing impairment (binaural average >25 dB HL) expected to result from 40 years of occupational noise exposures of 80, 85, and 90 dBA (8 hour TWA) among 60-year old workers, also found that the ISO-1999 underestimated the amount of risk compared to other methods. Furthermore, comparisons between the jute weaving NIPTS data from Carlsson *et al*.,[19] with the NIPTS predictions by ISO-1999 for this data showed a close correspondence during the first 10 years of exposure with significant differences (i.e., the ISO predictions underestimated the Carlsson *et al*., data[21] appearing after 15-20 years of exposure for the 50th and 75th percentile. In a recent study by Leensen *et al*.,[22] an analysis of hearing thresholds in over 29,000 construction workers in Dutch industry indicated a similar relationship to ISO-1999 predictions for exposure duration greater than 10 years. However, the ISO model was not consistent with the population data during the first 10 years of exposure, i.e., pure-tone average at 3, 4, and 6 kHz was ~ 10 dB poorer than that predicted by ISO-1999. Leensen *et al*.,[22] also found that construction workers employed for less than ten years had greater hearing losses than expected based on the interpolation of ISO-1999. The median values of worker NIPTS however, was similar to the ISO predictions for exposure times between ten and 40 years. Recent studies,[30,31] which have attempted to evaluate the ISO-1999 database, commonly used to compensate for age-related hearing loss (i.e., to determine NIPTS), revealed the following results: (1) there was a greater amount of NIPTS in men (65-74 years) than that observed in the 1999-2006 National Health and Nutrition Examination Survey (NHNES) i.e., prevalence of hearing impairment was lower in the 1999-2006 NHNES in comparison to the 1959-1962 database,[25] and (2) using the 1999-2002 NHNES ($N = 3{,}527$), occupational noise exposure was significantly associated with several non-occupational noise factors (e.g., smoking, educational level, leisure time and firearm noise),[31] The implication of the findings above suggest that: (1) the use of the use of the 1999-2006 NHNES database (instead of ISO-1999) would result in greater NIHL in unscreened older male adults, and (2) an overestimation of NIHL may occur using the ISO-1999 age-correction database if non-occupational noise factors (e.g., smoking, educational level, leisure time and firearm noise) are not considered.

Susceptibility to noise-induced hearing loss

The results reported in this study illustrate characteristic differences in individual susceptibility to noise exposure reflected in very little to large median NIPTS of up to 50 dB (pure tone average for 3, 4, and 6 kHz) for comparable noise exposure conditions. This finding is consistent with that reported by Taylor *et al*.,[19] who examined hearing loss in a group of forge operators who had worked in the same factory for up to 40 years i.e., workers exposed to the same constant noise (99 or 102 dB) for long periods of time (1-54 years) showed as much as 70 dB difference in auditory threshold between the least and most affected workers with NIHL. In the ISO-1999 model the distribution of median NIPTS is also large e.g., for men working at 100 dB (A) noise for 30 years the difference between 10% and 90% percentile of hearing loss is 60 dB HL. In our present study, differences between 10% and 90% percentile of median NIPTS were also large and greatest (up to ~50 dB) at the higher test frequencies where the amount of NIPTS was most pronounced. Even in controlled laboratory experiments the range of hearing loss in chinchillas exposed to the same high level noise show a range of variability that is similar to variability found in demographic studies.[5-7] The large variability in NIHL has been attributed to the variation in acoustic transfer characteristics of the external auditory meatus,[32] contribution of the stapedius reflex to protection from noise exposures,[33,34] and/or genetic factors.[35,36] Recently, gene expression differences induced by a noise exposure were demonstrated in mice.[35] Other intrinsic factors such as gender, race, hypertension, diabetes, and external factors[36-39] which include ototoxicity, leisure noise exposure, smoking, and use of HPD, have been reported. These potential factors, combined with the parameters of the noise exposure itself (e.g., Leq, duration, interrupted, intermittent, and time varying) contribute, in varying ways, to the probability of developing NIHL and the magnitude of NIHL among individuals exposed to equal acoustic energy exposures. In light of the results from this present study, another potential factor to consider is the kurtosis level of the noise exposure, i.e., the median NIPTS increased as the kurtosis level of the noise exposure increased.

Conclusion

The results from the varied exposure conditions in the present study suggest that: (1) the kurtosis metric is an important variable in determining the hazards to hearing posed by a high-level industrial noise environment for hearing conservation purposes, i.e., the kurtosis differentiated between the hazardous effects produced by G [$\beta(t) = <4$] and non-G [$\beta(t) = \geq 4$] noise environments, and noise environments with high and low mean and median kurtosis levels [$\beta(t) = \leq 10$ and >10], and (2) the ISO-1999 predictive model is not as accurate estimating the amount of median NIPTS incurred from high level kurtosis industrial noise as it is to low level kurtosis industrial noise. This conclusion is supported by the findings which showed: a) that the ISO-1999 model was more closely aligned with the group exposed to the G [$\beta(t) = <4$] than to the non-G [$\beta(t) = \geq 4$] industrial noise, e.g., the ISO model significantly ($P < 0.05$) underestimated the median NIPTS for the high level kurtosis group at 2, 4 and 6 kHz, b) the extent by which the ISO model underestimated the group median NIPTS which was greater (Mean = 6.8 dB) for the higher mean kurtosis noise level [$\beta(t) = \geq 10$; mean = 9.8 dB] than the lower mean kurtosis noise level [$\beta(t) = \leq 10$; mean = 3.0 dB] among the noise conditions across the test frequencies, and c) the ISO model significantly ($P < 0.05$) underestimated the amount of median NIPTS in the majority (14 of 17) of comparisons with noise exposed groups having both high [$\beta(t) = >10$] and low [$\beta(t) = \leq 10$] mean and median kurtosis levels.

The inherent large variability in NIPTS among subjects, in combination with the relatively small sample sizes in a few noise exposure groups, emphasize the need to develop and analyze a larger database of workers with well-documented exposures to better understand the effect of kurtosis on NIPTS incurred from high level industrial noise exposures. This will enable us to determine if the kurtosis metric provides more value in identifying high level complex industrial noise exposures that have the potential to increase risk of NIHL, than the traditional energy based approach for hearing conservation. A better understanding of the role of the kurtosis metric in NIHL may lead to its incorporation into a new generation of more predictive hearing risk assessment for noise exposure provided that human exposure and hearing loss data can continue to be acquired from suitably designed epidemiological studies.

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References

1. International Standard ISO 1999:1990. Acoustics–Determination of

occupational noise exposure and estimation of noise-induced hearing impairment. Technical Comm. ISO/TC 43-Acoustics. Geneva, Switzerland: International Organization for Standardization; 1990.
2. Canlon B, Borg E, Flock A. Protection against noise trauma by pre-exposure to a low-level acoustic stimulus. Hear Res 1988;34:197-200.
3. Clark WW, Bohne BA. Effects of periodic rest on cochlear damage and hearing loss. In: Dancer D, Henderson D, Salvi RJ, Hamernik RP, editors. Noise-Induced Hearing Loss. St. Louis MO: Mosby-Year Book; 1991. p. 445-55.
4. Ward WD. The role of intermittence in PTS. J Acoust Soc. Am 1991;90:164-9.
5. Hamernik RP, Qiu W, Davis RI. The effects of the amplitude distribution of equal energy exposures on noise-induced hearing loss: The kurtosis metric. J Acoust Soc Am 2003;114:386-95.
6. Qiu W, Hamernik RP, Davis B. The kurtosis metric as an adjunct to energy in the prediction of trauma from continuous, non Gaussian noise exposures. J Acoust Soc Am 2006;120:3901-6.
7. Qiu W, Davis B, Hamernik RP. Hearing loss from interrupted, intermittent, and time-varying Gaussian noise exposures: The applicability of the equal energy hypothesis. J Acoust Soc Am 2007;121:1613-20.
8. Davis RI, Qiu W, Hamernik RP. The role of the kurtosis statistic in evaluating complex noise exposures for the protection of hearing. Ear Hear 2009;30:628-34.
9. Theiry L, Meyer-Bisch, C. Hearing loss due to partly impulsive industrial noise exposure at levels between 87 and 90 dB. J Acoust Soc Am 1988;84:651-9.
10. Dunn DE, Davis RR, Merry CJ, Franks JR. Hearing loss in the chinchilla from impact and continuous noise exposure. J Acoust Soc Am 1991;90:1979-85.
11. Committee on Hearing, Bioacoustics, and Biomechanics (CHABA). Hazardous exposure to impulse noise. Washington DC: National Research Council; 1992.
12. Ahroon WA, Hamernik RP, Davis RI. Complex noise exposures: An energy analysis. J Acoust Soc Am 1993;90:997-1006.
13. Lataye R, Campo P. Applicability of the Leq as a damage-risk criterion: An animal experiment. J Acoust Soc Am 1996;99:1621-32.
14. Kardous CA, Franks JR, Davis RR. NIOSH/NHCA Best-Practices Workshop on impulsive noise. J Noise Control Eng 2005;53:34-45.
15. National Institute of Occupational Safety and Health NIOSH Criteria for a Recommended Standard: Occupational Noise Exposure-Revised Criteria U.S. Department of Health, Education, and Welfare. Cincinnati, OH: National Institute for Occupational Safety and Health; 1998. p. 98-126.
16. Prince MM, Stayner LT, Smith RJ, Gilbert SJ. A re-examination of risk estimates from the NIOSH Occupational Noise and Hearing Survey (ONHS). J Acoust Soc Am 1997;101:950-63.
17. Prince MM, Gilbert SJ, Smith RJ, Stayner LT. Evaluation of the risk of noise-induced hearing loss among unscreened male industrial workers. J Acoust Soc Am 2003;113:871-80.
18. Dobie RA. Noise-induced permanent threshold shifts in the occupational noise and hearing survey: An explanation for elevated risk estimates. Ear Hear 2007;28:580-91.
19. Taylor W, Pearson J, Mair A, Burns W. Study of noise and hearing in jute weaving. J Acoust Soc Am 1965;38:113-20.
20. Henderson D, Saunders SS. Acquisition of noise-induced hearing loss by railway workers. Ear Hear 1998;19:120-30.
21. Carlsson P, Borg E, Grip L. Variability in noise susceptibility in Swedish population: The role of 35 delG mutation in the connex in 26 (GJB2) gene. Audiol Med 2004;2:123-30.
22. Leensen MC, van Duivenbooden JC, Dreschler WA. A retrospective analysis of noise-induced hearing loss in the Dutch construction industry. Int Arch Occup Environ Health 2011;84:577-90.
23. Dobie RA. Medical-Legal Evaluation of Hearing Loss. 2nd ed. San Diego CA: Singular; 2001. p. 270-7.
24. International Standard Organization (ISO 389-1). Acoustics - Reference zero for the calibration of audiometric equipment. Part 1 - Reference equivalent threshold sound pressure levels for pure tones and supra-aural earphones. 1998.
25. Glorig A, Roberts J. Hearing Levels of Adults by Age and Sex: United States. 1960-1962. Vital Health Stat Series 1965;11:11.
26. Goley SG, Song WJ, Kim JH. Kurtosis corrected sound pressure level as a noise metric for risk assessment of occupational noises. J Acoust Soc Am 2011;129:1475-81.
27. de Moraes Marchiori LL, de Almeida Rego Filho E, Matsuo T. Hypertension as a factor associated with hearing loss. Bras J Otorhinolaringol 2006;72:533-40.
28. Zakrisson JE, Borg E, Liden G, Nilsson R. Stapedius reflex in industrial impact noise: Fatigability and role for temporary threshold shifts (TTS). Scand Audiol Suppl 1980;(Suppl12):326-34.
29. National Institute of Occupational Safety and Health Best Practices Workshop on Impulsive Noise and its Effect on Hearing. National Institute for Occupational Safety and Health. Noise Control Eng J 2005;53:53-60.
30. Agrawal Y, Niparko JK, Dobie RA. Estimating the effect of occupational noise exposure on hearing thresholds: The importance of adjusting for confounding variables. Ear Hear 2010;31:234-7.
31. Hoffman HJ, Dobie RA, Ko CW, Themann CL, Murphy WJ. Hearing threshold levels at age 70 years (65-74 years) in the unscreened older adult population of the United States, 1959-1962 and 1999-2006. Ear Hear 2012;33:437-40.
32. Hellstrom PA. Individual differences in peripheral sound transfer functions: Relation to NIHL. In: Axelsson A, Hamernik RP, Henderson D, editors. Noise Induced Hearing Loss. New York: Thieme Publishers; 1995. p. 129-37.
33. Colletti V, Sittoni W. Noise history, audiometric profile and acoustic reflex responsivity. In:Salvi R, Henderson D, Hamernik R, Colletti V, editors. Basic and Applied Aspects of Noise Induced Hearing Loss. New York: Plenum Press; 1985.p. 269-347.
34. Erway LC, Shiau YW, Davis RR, Krieg EF. Genetics of age-related hearing loss in mice. III. Susceptibility of inbred and F_1 hybrid strains to noise-induced hearing loss. Hear Res1996;93:181-7.
35. Gratton M, Eleftheriadou A, Garcia J, Verduzco E, Martin GK, Lonsbury-Martin BL, *et al*. Noise-induced changes in gene expression in the cochleae of mice differing in their susceptibility to noise damage. Hear Res 2011;277:211-26.
36. Zhao YM, Qiu W, Zeng L, Chen SS, Cheng XR, Davis RI, *et al*. Application of the kurtosis statistic to the evaluation of the risk of hearing loss in workers exposed to high-level complex noise. Ear Hear 2010;31:527-32.
37. Mizoue T, Miyamoto T, Shimizu T. Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. Occup Environ Med2003:60;56-9.
38. Wild DC, Brewster MJ, Banerjee AR. Noise-induced hearing loss is exacerbated by long-term smoking. Clin Otolaryngol 2005;30:517-20.
39. Zhu X, Kim JH, Song WJ, Murphy WJ, Song S. Development of a noise metric for assessment of exposure risk of complex noises. J Acoust Soc Am 2009;126:703-12.

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