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Hearing loss from interrupted, intermittent, and time varying non-Gaussian noise exposure: The applicability of the equal energy hypothesis

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Sixteen groups of chinchillas ($N=140$) were exposed to various equivalent energy noise paradigms at 100 dB(A) or 103 dB(A) SPL. Eleven groups received an interrupted, intermittent, and time varying (IITV) non-Gaussian exposure quantified by the kurtosis statistic. The IITV exposures, which lasted for 8 h/day, 5 days/week for 3 weeks, were designed to model some of the essential features of an industrial workweek. Five equivalent energy reference groups were exposed to either a Gaussian or non-Gaussian 5 days, 24 h/day continuous noise. Evoked potentials were used to estimate hearing thresholds and surface preparations of the organ of Corti quantified the sensory cell population. For IITV exposures at an equivalent energy and kurtosis, the temporal variations in level did not alter trauma and in some cases the IITV exposures produced results similar to those found for the 5 day continuous exposures. Any increase in kurtosis at a fixed energy was accompanied by an increase in noise-induced trauma. These results suggest that the equal energy hypothesis is an acceptable approach to evaluating noise exposures for hearing conservation purposes provided that the kurtosis of the amplitude distribution is taken into consideration. Temporal variations in noise levels seem to have little effect on trauma. © *2007 Acoustical Society of America.*
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I. INTRODUCTION

Industrial and military noise exposures are most often interrupted and intermittent with sound levels that vary over the course of a work shift (Taylor *et al.*, 1984). The presence of transients, either impacts or noise bursts, further complicates the evaluation of these noise environments for hearing conservation purposes. The hearing loss that results from such exposures usually accumulates over many years or decades of exposure. Much of our understanding of the effects of noise on hearing is derived from acute animal model experiments that typically have not taken into account the complexity of a daily industrial noise exposure. Further, in practice any concern for the temporal and level complexity of an industrial exposure is obviated by the acceptance of the equivalent energy principal as a guide to noise exposure criteria (e.g., ISO 1999). The equal energy hypothesis (EEH) initially proposed by Eldred *et al.* (1955), with early support from the guinea pig experiments of Eldredge and Covell (1958), suggests that equal amounts of acoustic energy (with some consideration given to frequency content) entering the ear will produce equal amounts of hearing loss under typical conditions of exposure. Over the ensuing years a number of studies have supported the concept of the EEH (Eldredge *et al.*, 1959; Dolan *et al.*, 1976; Clark, 1991; and others). This idea gave rise to the concept of the equivalent continuous noise level (L_{eq}), defined as the level of a continuous noise that, in the course of an 8 h workday, would cause the same sound energy to be received as that due to the actual noise over a typical workday. Energy as a basic metric for evaluating hazardous exposures was eventually extended to impact noise exposures (Atherley and Martin, 1971). While there is some recent support for an energy-based approach under limited exposure conditions (e.g., Qiu *et al.*, 2007), there is a consensus that such an approach will not cover all conditions of exposure (e.g., Lataye and Campo, 1996; Dunn *et al.*, 1991; Harding and Bohne, 2004; Hamernik *et al.*, 2003).

The impact that the temporal structure of an acoustic signal has on noise-induced hearing loss (NIHL) has been highlighted by the "toughening" and "conditioning" phenomena and an understanding of some of the cochlea's endogenous protective mechanisms underlying these phenomena. In the former case it has been shown (Miller *et al.*, 1963; Clark *et al.*, 1987; and others) that for subjects exposed to a daily interrupted noise, audiometric thresholds could improve despite the continuing exposure (i.e., the cochlea is gradually toughened). In the latter case, Canlon *et al.* (1988) and others have shown that exposure to a low level noise could reduce the effects of a subsequent traumatic exposure (i.e., the cochlea has been toughened by the low level conditioning noise). In a typical industrial noise exposure situation it could be argued that both of these phenomena would be operative. Temporal factors also become significant when the exposure is non-Gaussian (nonG), that is, when the noise contains transients that vary in their rate of appearance and in their distribution of peaks in the case of impacts, or their rms levels in the case of noise bursts. Such complex

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exposures were used by Hamernik *et al.* (2003) and Qiu *et al.* (2006) in showing the limitations of the energy metric and the advantage of using the statistical metric, kurtosis, in combination with energy to evaluate the hazard potential of a nonG exposure. They showed that for a constant energy there was an orderly relation between the kurtosis of an exposure and the resulting NIHL and sensory cell loss. As the kurtosis increased so did the hearing trauma. Experimental data on the effects of equivalent energy nonG exposures with time varying levels are not available.

In a recent study Qiu *et al.* (2007), using several different equivalent energy interrupted, intermittent, and time varying (IITV) Gaussian noise exposures, showed that despite some toughening effects the permanent threshold shifts (PTS) and sensory cell losses were reasonably similar across different IITV exposures and similar to an equivalent energy uniform and uninterrupted reference exposure. They concluded that the EEH is a reasonable approach to evaluating Gaussian noise exposures for hearing conservation purposes. The present paper, a continuation of the Qiu *et al.* (2007) work, presents data from a number of different nonG, IITV equivalent energy exposures. The exposures were designed to replicate some of the essential features of realistic industrial environments over a period of 3 weeks.

II. METHODS

A. Auditory evoked potential

One hundred and forty (140) chinchillas were used as subjects. Each animal was anesthetized [IM injection of Ketamine (35 mg/kg) and Xyalazine (1 mg/kg)] and made monaural by the surgical destruction of the left cochlea. During this procedure a bipolar electrode was implanted, under stereotaxic control, into the left inferior colliculus and the electrode plug cemented to the skull for the recording of auditory evoked potentials (Henderson *et al.*, 1973; Salvi *et al.*, 1982). The auditory evoked potential (AEP) was used to estimate pure tone thresholds. The animals were awake during testing and restrained in a yoke-like apparatus to maintain the animal's head in a constant position within the calibrated sound field. AEPs were collected to 20 ms tone bursts (5 ms rise/fall time) presented at a rate of 10/s. Each sampled wave form was analyzed for large-amplitude artifact, and, if present, the sample was rejected from the average and another sample taken. Averaged AEPs were obtained from 250 presentations of the 20 ms tone bursts. Thresholds were measured using an intensity series with 5 dB steps at octave intervals from 0.5 to 16 kHz. Threshold was defined to be one-half step size (2.5 dB) below the lowest intensity that showed a "response" consistent with the responses seen at higher intensities.

B. Experimental protocol

Following a 2 week postsurgical recovery, three AEP preexposure audiograms were obtained (on different days) on each animal at octave intervals between 0.5 and 16.0 kHz. If the mean of the three audiograms fell beyond one standard deviation (s.d.) of laboratory norms (Hamernik and Qiu, 2000), in the direction of poorer thresholds at more than one test frequency, the animal was rejected. The animals were randomly assigned to one of 16 experimental groups with 7–16 animals/group.

The animals were exposed four (or less) at a time to one of the noise conditions detailed in the following. During exposure, animals were given free access to food and water and were rotated through a bank of six cages daily. The SPLs, across cages, in the middle of each cage, varied within less than ±1 dB. For the 5 day uniform, Gaussian or nonG continuous (uninterrupted) reference exposures, animals were removed daily for less than 0.5 h for AEP testing. A complete AEP audiogram was obtained on each animal each day of the exposure and the mean of the five audiograms defined asymptotic threshold (AT). For all the IITV groups the animals were tested at the end of the daily exposure on days 1, 17, 18, and 19. The difference between the threshold measured following the first day of exposure (T_1) and the mean of the thresholds measured following the last three days (T_{17-19}) of the exposure was accepted as an estimate of any threshold recovery or toughening (T_r) effect [i.e., $T_r=(T_1)-(T_{17-19})$]. Thirty days following the last exposure day for all exposure paradigms, three more audiograms were collected on different days and the mean used to define permanently shifted thresholds or permanent threshold shift.

C. Histology

Following the last AEP test protocol, each animal was euthanized under anesthesia and the right auditory bulla removed and opened to gain access to the cochlea for perfusion. Fixation solution consisting of 2.5% glutaraldehyde in veronal acetate buffer (final pH=7.3) was perfused through the cochlea. After 12–24 h of fixation the cochlea was postfixed in 1% OsO_4 in veronal acetate buffer. Surface preparation mounts of the entire organ of Corti were prepared (Engstrom *et al.*, 1966) and inner hair cell (IHC) and outer hair cell (OHC) populations were plotted as a function of frequency and location using the frequency-place map of Eldredge *et al.* (1981). Missing cells were identified by the presence of a characteristic phalangeal scar. For purposes of this presentation, sensory cell population data are presented as group averages (in percent missing) taken over octave band lengths of the cochlea centered on the primary AEP test frequencies.

D. Noise measurement and analyses

During the exposures the noise field was monitored with a Larson Davis 814 sound level meter equipped with a 1/2 in. microphone. The computer generated acoustic signal, produced by the Electro-Voice Xi-1152/94 speaker system, was transduced by a Brüel and Kjær 1/2 in. microphone (Model 4134), amplified by a Brüel and Kjær (Model 2610) measuring amplifier and fed to a Windows PC-based analysis system. The signal was sampled at 48 kHz with a recording duration of 5.5 min. The design and digital generation of the acoustic signal is detailed in Hsueh and Hamernik (1990, 1991). Calibration of the exposure was accomplished "off line" by analyzing two to eight, 5.5 min segments of the digitized acoustic wave form. In order to maintain equal en-

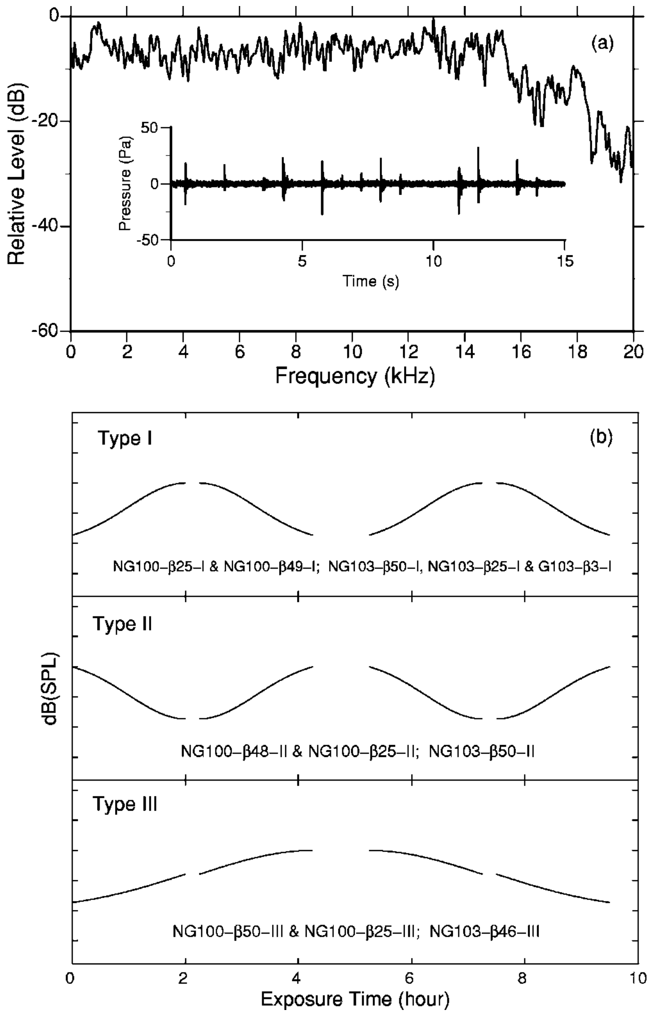

FIG. 1. (a) The relative spectral level of the unweighted 100 dB(A) SPL noise. The 103 dB(A) SPL noise had an unweighted spectrum that was qualitatively similar to the 100 dB(A) SPL noise. The inset shows a 15 s segment of a non-Gaussian noise. The impact peaks and interimpact intervals were randomly varied. (b) A sketch of the daily SPL variations that were used for the intermittent, interrupted, and time varying (IITV) noise exposure paradigm for the 100 and 103 dB(A) SPL groups. Groups exposed to each type of IITV exposure are identified. Each exposure sequence lasted for 19 days. Each daily exposure consisted of two 4.25 h periods with an hour break in between. Each 4.25 h exposure was interrupted for 15 min and each 5 day exposure sequence was separated by a 2 day break.

ergy exposure conditions across nonG noise exposed groups at the same overall SPL the peak and interval histograms of the impact transients and the level of the background noise (L_b), that is, the rms level of the noise between the transients, had to be adjusted. For the nonG, IITV exposures a mean value of the kurtosis, $\beta(t)$, was calculated by averaging the $\beta(t)$ from eight, 5.5 min samples of the exposure wave form.

E. Noise exposures

Hearing and cell loss data are presented from 16 different exposures. Each exposure had in common the same flat spectrum between 0.125 and approximately 15.0 kHz shown in Fig. 1(a). The nonG exposures were created by inserting impacts, having randomly varied peak levels and interimpact intervals, into a Gaussian background noise as described by Hsueh and Hamernik (1990, 1991). A 15 s sample of a nonG wave form is shown in the inset of Fig. 1(a). The identification of each experimental group and the exposure parameters for the groups are detailed in Fig. 1(b) and Table I. Nine (9) exposures were presented at an overall SPL of 100 dB(A) and seven exposures were presented at 103 dB(A). All the 100 dB(A) exposures had the same total energy. Group G100-β3-U was exposed to a Gaussian noise and the remaining eight groups were exposed to a nonG noise that differed in either the value or range of values of the kurtosis or in the configuration of the SPL temporal variation [Fig. 1(b)]. Two of the nonG exposures (groups NG100-β25-U and NG100-β55-U) that were used as equal energy reference groups lasted for 24 h/day for 5 days and had a uniform, uninterrupted 100 dB(A) SPL. Group G100-β3-U, another reference group, was exposed to a uniform Gaussian noise for 5 days, 24 h/day. Six of the 100 dB(A) exposures were structured to model an idealized 3 week work shift with a varying noise level [IITV exposures, Fig. 1(b)]. Each daily IITV exposure consisted of two 4.25 h periods with an hour break in between. Each 4.25 h exposure was interrupted for 15 min and each 5 day sequence was separated by a 2 day break. Three different temporal variations in the sound pressure level (identified as type I, II, or III) were used for the exposures and are shown schematically in Fig. 1(b) along with the exposure group identification. It should be noted that there was no *a priori* reason for the choice of the Gaussian shaped patterns of sound level variation shown in Fig. 1(b).

There were seven groups exposed at an overall SPL of 103 dB(A). One of these groups (G103-β3-U) was exposed to a uniform Gaussian noise, 24 h/day for 5 days and one group (G103-β3-I) was exposed to a Gaussian, IITV noise with a type I SPL variation. One group (NG103-β50-U) was exposed to a uniform nonG noise, 24 h/day for 5 days. These three groups served as reference exposures for the five, nonG, IITV exposed groups. Summary AEP and sensory cell loss data across different exposure groups are compared in the figures identified in Table I.

F. Statistical analysis

Threshold shifts and the percent sensory cell loss in octave-band lengths of the cochlea were compared among the groups of animals for each noise exposure level [100 and 103 dB(A) SPL] using a mixed model analysis of variance (ANOVA) with repeated measures on one factor (frequency). The probability of a type I error was set at 0.05. Statistically significant main effects of frequency were expected and found in all of the following analyses because of the frequency-specific nature of the audibility curve of the chinchilla and the noise exposure stimulus. For this reason the main effects of frequency are not addressed in the following presentation of the results.

III. RESULTS AND DISCUSSION

A. Preexposure thresholds

The group mean preexposure AEP thresholds are shown as symbols in Fig. 2. The solid and broken lines show the mean thresholds for all the animals exposed at 100 and

TABLE I. Outline of the noise exposure conditions for all experimental groups.

Fig.	Group I.D.	Group size	Noise type[a]	Exposure type	L_{eq} dB(A)	L_{eq} range dB(A)	Mean kurtosis	Kurtosis range	Peak SPL (dB)	L_b dB(A)[b]
3	G103-β3-U	$N=8$	G	Uniform 5 day	103	⋯	3	⋯	⋯	⋯
4 and 5	NG103-β50-U	$N=7$	nonG	Uniform 5 day	103	⋯	50	[43, 57]	[122, 131]	93
	NG103-β46-III	$N=8$	nonG	IITV 19 day, III	103	[89, 106]	46	[35, 55]	[104, 134]	[80, 97]
	NG103-β50-I	$N=8$	nonG	IITV 19 day, I	103	[89, 106]	50	[33, 56]	[105, 134]	[81, 98]
	NG103-β50-II	$N=7$	nonG	IITV 19 day, II	103	[89, 106]	50	[38, 62]	[102, 134]	[80, 97]
6 and 7	G103-β3-I	$N=8$	G	IITV 19 day, I	103	[89, 105]	3	⋯	⋯	⋯
	NG103-β25-I	$N=7$	nonG	IITV 19 day, I	103	[89, 105]	25	[19, 32]	[101, 132]	[81, 98]
3	G100-β3-U	$N=16$	G	Uniform 5 day	100	⋯	3	⋯	⋯	⋯
8	NG100-β25-U	$N=12$	nonG	Uniform 5 day	100	⋯	25	[23, 28]	[115, 129]	95
	NG100-β25-III	$N=8$	nonG	IITV 19 day, III	100	[67, 105]	25	[17, 31]	[80, 130]	[60, 98]
	NG100-β25-II	$N=7$	nonG	IITV 19 day, II	100	[69, 105]	25	[18, 35]	[90, 130]	[63, 97]
	NG100-β25-I	$N=8$	nonG	IITV 19 day, I	100	[70, 105]	25	[16, 35]	[92, 130]	[62, 98]
9	NG100-β55-U	$N=12$	nonG	Uniform 5 day	100	⋯	55	[46, 62]	[121, 132]	0
	NG100-β50-III	$N=8$	nonG	IITV 19 day, III	100	[68, 106]	50	[33, 68]	[84, 132]	[60, 97]
	NG100-β48-II	$N=8$	nonG	IITV 19 day, II	100	[70, 105]	48	[35, 57]	[91, 132]	[63, 97]
	NG100-β49-I	$N=8$	nonG	IITV 19 day, I	100	[70, 105]	49	[31, 57]	[92, 133]	[63, 97]

[a]G=Gaussian; nonG=non-Gaussian.
[b]L_b, the level of the Gaussian background noise.

103 dB(A) respectively. Standard errors were small and all animals and groups fell within ±1 s.d. of laboratory norms based on 1572 chinchillas (shaded area). These data are consistent with published thresholds for the chinchilla (Fay 1988). There were no systematic differences in preexposure thresholds among the experimental groups.

B. The 100 and 103 dB(A) Gaussian reference exposures

Figure 3 presents the results of exposure to a uniform Gaussian noise at 100 and 103 dB(A) for 5 days, 24 h/day (groups G100-β3-U and G103-β3-U, respectively). The exposure was interrupted for only approximately 20 min each day for AEP testing in order to obtain estimates of asymptotic threshold shift (ATS). The upper panels show the AEP data and the lower panels the inner and outer hair cell loss data. The shaded area in the upper panels represents the PTS. The difference between AT and the preexposure thresholds is an estimate of the ATS. The bars on the data symbols in this and subsequent figures represent the standard error (s.e.) of the mean. If a bar is not present the s.e. is less than the size of the symbol.

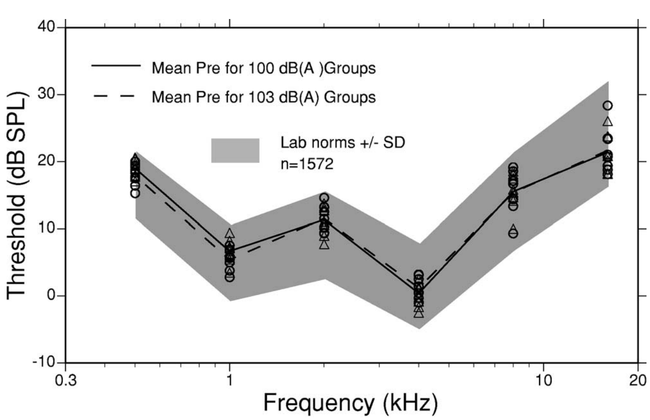

FIG. 2. Mean preexposure thresholds (symbols) for the 16 experimental groups defined in Table I. The shaded area represents the mean preexposure thresholds ±1 s.d. of the laboratory norm based on 1572 chinchillas. The solid line represents the mean preexposure thresholds of the 87 chinchillas included in the groups exposed to the 100 dB(A) SPL noises and the broken line the preexposure thresholds for the 53 chinchillas exposed at 103 dB(A).

These two exposures, at each level, serve as reference points for all the Gaussian and nonG, IITV or uninterrupted exposure groups discussed in the following. For the 100 dB(A) exposure (group G100-β3-U) ATS increased with increasing frequency from about 37 dB at 0.5 kHz to 73 dB at 16 kHz, while PTS varied from near 0 dB at 0.5 kHz to 24 dB at 16 kHz. The IHC loss did not exceed 10% at the higher frequencies and OHC losses varied from less than 10% to almost 20% in the highest frequency band. Increasing the level to 103 dB(A) (group G103-β3-U) produced from about 65 dB ATS at 16 kHz to a maximum of 84 dB at 4 kHz. During the 5 day exposure there were large shifts in threshold across the entire range of AEP test frequencies. PTS increased with increasing frequency from 10 to 30 dB. IHC loss was small with about a 12% loss at 8 kHz and no loss at and below 2 kHz. OHC loss showed a bimodal distribution with 26% losses in the 0.250 and 0.5 kHz bands and 36% loss at 8 kHz with little loss in the 1.0 and 2.0 kHz region. It should be noted that any thresholds at 90 dB seen in Fig. 3 or in any of the subsequent figures represent the upper limit of the AEP test system and a threshold shift computed at such frequencies represents an estimate of the lower bound only. The most noticeable effect of the 3 dB increase in level is the increased threshold shifts

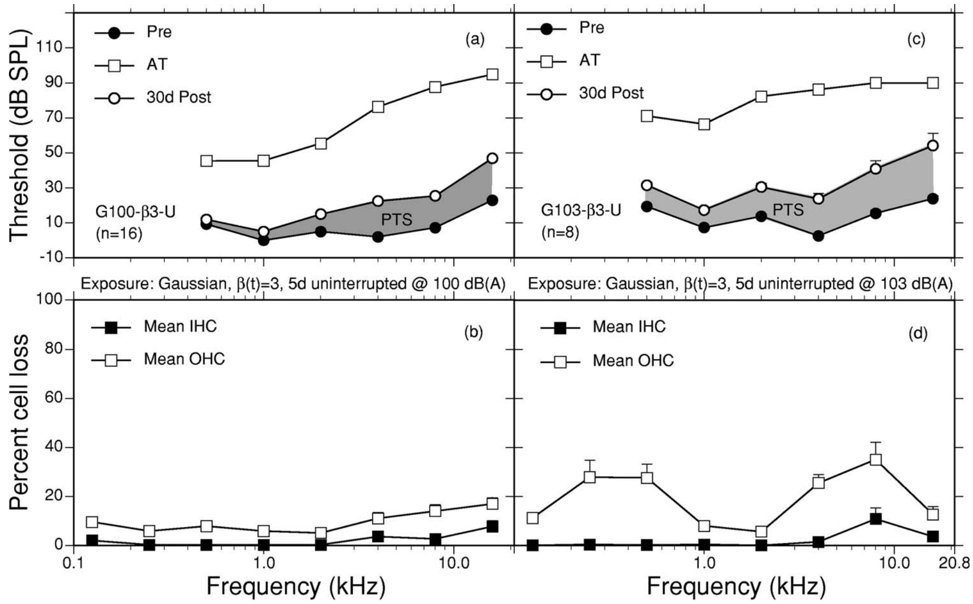

FIG. 3. Summary data for reference group G100-β3-U exposed to the 100 dB(A) SPL Gaussian, uninterrupted 5 day exposures and group G103-β3-U exposed to a similar noise but at 103 dB(A) SPL. Upper panels show the group means preexposure thresholds, asymptotic threshold (AT), and 30 day postexposure thresholds for each group. The lower panels show the group mean percent outer and inner sensory cell (OHC and IHC) losses for each group. Error bars indicate the standard error (s.e.) of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

and OHC loss especially at the low frequencies. A two-way ANOVA confirmed these differences. There was a statistically significant main effect of group for the ATS, PTS, and OHC loss and no significant main effect of group for IHC loss. There was no significant interaction of group and frequency for PTS but there was a significant interaction for ATS, IHC loss, and OHC loss.

C. The 103 dB(A) exposures

All the exposures discussed in the following had the same total energy and the same spectrum. They differ only in the nature of their level variations, their kurtosis, and whether or not the exposure was interrupted and intermittent.

1. The effects of exposures having an equal energy and kurtosis but a variable level

Figure 4 presents complete group mean data sets for four of the 103 dB(A), nonG exposure conditions. Figure 4 illustrates the type of data set that was obtained for each experimental group and from which any subsequent summary figures are distilled. The effects of three different nonG, IITV exposures (groups NG103-β46-III, NG103-β50-I, and NG103-β50-II) and an uninterrupted nonG, 5 day reference exposure (NG103-β50-U) are shown. The four exposures had approximately the same mean value of the kurtosis $[\beta(t) \sim 50]$ and the same total energy and spectra. For group NG103-β50-U exposed for 5 days to the uninterrupted nonG noise, group mean asymptotic thresholds were 90 dB across the range of test frequencies. These threshold values do not represent actual AEP thresholds but rather the upper limit of the AEP measurement system. After a 30 day recovery period, thresholds recovered to between 48 and 80 dB SPL with the higher frequencies showing the highest thresholds. Group mean thresholds for the three IITV exposures measured following the first day's exposure were similarly at or very near the limit of the AEP test system except for the 0.5 and 1.0 kHz test frequencies in group NG103-β46-III where thresholds of about 76 dB SPL were measured. All three IITV exposures produced a T_r that was greatest at the lower frequencies and disappeared at the highest test frequencies. While a T_r was measured, the true magnitude could not be determined at a number of frequencies as a result of the ceiling effect in the AEP test system. All the IITV exposures produced a T_r and when thresholds were not limited by the ceiling effect, T_r was as much as 22 dB at 0.5 kHz for the 103 dB(A) exposures. During the 30 day recovery period, thresholds only recovered 10–25 dB leaving each group with as much as 65 dB permanent losses. Inner and outer hair cell losses generally reflect the severity of the shifted thresholds. Outer hair cell loss is severe and in some frequency regions complete, with greatest loss occurring at the higher frequencies. Inner hair cell losses are also severe with maximum loss in the 4 kHz region of the basilar membrane. Threshold and cell loss data from these four groups are summarized and compared in Fig. 5. Apparent from Fig. 5 is the similarity of the PTS, IHC loss, and OHC loss across the four groups. A two-way ANOVA confirmed that there was no significant main effect of group and no interaction between group and frequency for PTS, IHC loss, and OHC loss.

Based on the above presented results, nonG exposures of equivalent energy, spectra, and kurtosis can produce equivalent trauma whether or not the exposure is interrupted, intermittent, or follows a different temporal pattern of level variation. This result is similar to the conclusions drawn in a recent paper (Qiu *et al.*, 2007) where Gaussian IITV exposures were used. The toughening, T_r, seen in the IITV exposures did not seem to have any effect on the final PTS. While an accurate T_r cannot be measured because of the AEP ceiling effect, all the IITV exposures at 103 dB(A) did produce toughening but no subsequent protective effect. Similar levels of PTS were found in both the uninterrupted exposures where there was no toughening and the IITV exposures that showed some toughening. Also note the considerable increase in trauma associated with these nonG exposures when compared to the equivalent energy Gaussian $[\beta(t)=3]$ reference exposure shown in Figs. 3(c) and 3(d). The increase in trauma from nonG exposures has been documented from a

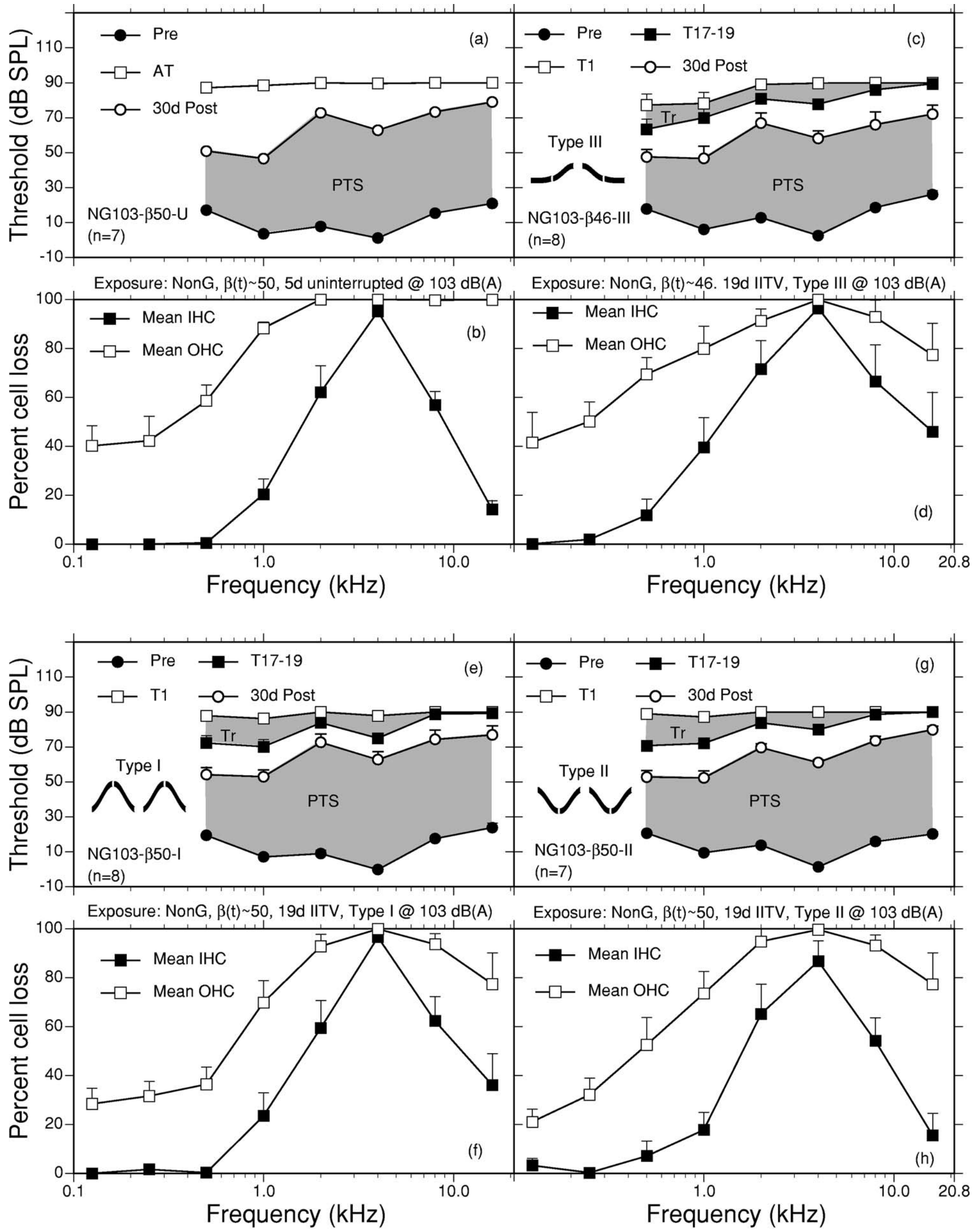

FIG. 4. Summary data from four of the groups exposed to 103 dB(A) SPL, non-G, $\beta(t) \sim 50$, 5 or 19 day noise paradigms. (a) The group (NG103-β50-U) mean preexposure thresholds, AT, and 30 day postexposure thresholds for the 5 day uninterrupted, uniform reference exposure. (b) The corresponding group mean outer and inner sensory cell (OHC, IHC) losses. (c), (d); (e), (f); and (g), (h) A similar presentation of data for the three IITV equivalent energy exposures, NG103-β46-III, NG103-β50-I, and NG103-β50-II, respectively. Instead of AT, the group mean thresholds measured immediately following exposure on day 1 (T_1) and the group mean thresholds measured immediately following exposure on days 17, 18, and 19 (T_{17-19}) are shown. Toughening (T_r) and permanent threshold shifts (PTS) are shown shaded. Error bars indicate the s.e. of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

variety of exposures that incorporated impacts or noise bursts to produce nonG exposure conditions (Hamernik *et al.*, 2003) as well as from comparisons of equal energy continuous noises and pure impact noise exposures (Dunn *et al.*, 1991).

2. The effects of IITV exposures having an equivalent energy and temporal structure but varying in their kurtosis

Figure 6 presents the group mean data from groups G103-β3-I and NG103-β25-I. Animals in group G103-β3-I were exposed to a 19 day, IITV Gaussian noise, $\beta(t)=3$ and group NG103-β25-I to a 19 day, IITV nonG noise, $\beta(t) \sim 25$. Group NG103-β50-I discussed earlier [Figs. 4(e) and 4(f)] was exposed to the same noise except with a $\beta(t) \sim 50$. All three exposures had a type I level variation. All three exposures produced large increases in threshold (T_1) following the first day of exposure that varied from about 68 to the 90 dB SPL limit of the AEP test system. The nonG groups showed the greatest increases in threshold. All three exposures produced similar amounts of toughening T_r with a maximum T_r of about 22 dB at the lower frequencies and very little or no toughening at the highest frequencies. This was similar in magnitude and frequency specificity to the exposures discussed above in Sec. III C 1. Following a 30 day recovery period, thresholds had recovered 22–46 dB in the group exposed to the Gaussian IITV noise (G103-β3-I), while thresholds in the nonG noise exposed group NG103-β25-I only recovered 16–26 dB. Inner and outer hair cell loss was also considerably less in the Gaussian noise exposed group G103-β3-I. A summary and comparison of the data from the three 103 dB(A) exposures with the type I level variation but having different $\beta(t)$ is shown in Fig. 7. Figures 7(a)–7(c) show the PTS, %IHC, and %OHC loss for the three groups, respectively. There is a clear ordering of PTS, IHC loss, and OHC loss. The Gaussian noise exposed group G103-β3-I showed a 10 dB PTS at the lowest frequencies that increased to 45 dB at the highest test fre-

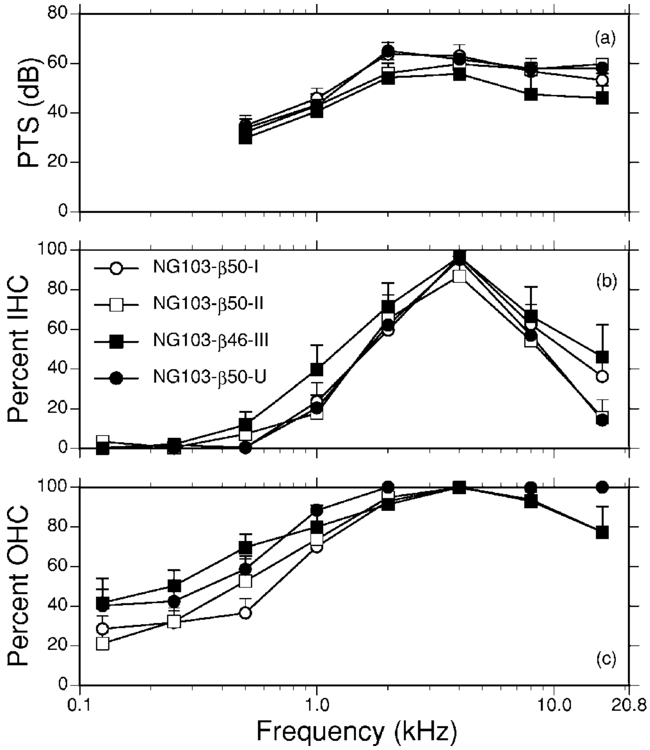

FIG. 5. A comparison of the PTS and cell loss data for the four groups shown in Fig. 4. (a) The PTS. (b) The percent IHC loss. (c) The percent OHC loss. All four groups were exposed to a nonG noise having the same energy and kurtosis. For group NG103-β50-U the uniform exposure lasted for 5 uninterrupted days. Groups NG103-β50-II, NG103-β46-III, and NG103-β50-I received the 19 day, IITV exposures. Error bars indicate the s.e. of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

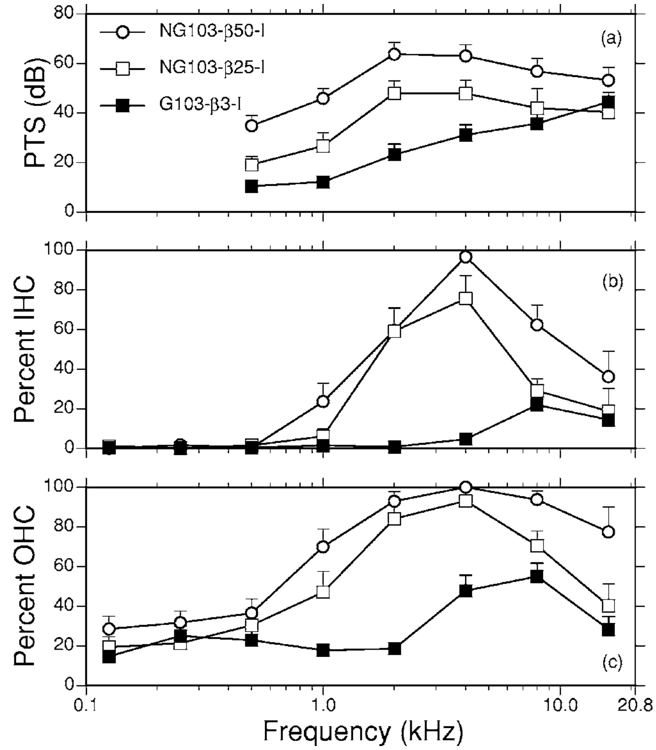

FIG. 7. A comparison of the PTS and cell loss data for the two groups shown in Fig. 6 along with the data from group NG103-β50-I (Fig. 4). (a) The PTS. (b) The percent IHC loss. (c) The percent OHC loss. All three groups were exposed to an IITV noise having the same energy and a type I level variation but different mean $\beta(t)$. For group G103-β3-I, $\beta(t)=3$ (i.e., a Gaussian noise); for group NG103-β25-I, $\beta(t)\sim 25$; and for group NG103-β50-I, $\beta(t)\sim 50$. Error bars indicate the s.e. of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

quency. Group NG103-β25-I, the nonG, $\beta(t)\sim 25$ exposure was intermediate in loss with 19–47 dB PTS that peaked at 2 kHz. The greatest loss was seen in the high kurtosis, $\beta(t)\sim 50$, nonG noise exposed group NG103-β50-I. PTS in this group varied from 35 dB at 0.5 kHz to a maximum of 64 dB at 2.0 kHz. IHC and OHC losses generally followed the trends seen in the PTS with maximum IHC losses of 75% and 95% in the 4 kHz region of the basilar membrane for the $\beta(t)\sim 25$ and $\beta(t)\sim 50$ exposures, respectively. The pattern of loss for the nonG exposures was broadly distributed across frequency. The Gaussian exposure produced maximum IHC loss of 22% in the 8 kHz region and little or no loss at the lower frequencies. OHC losses were considerably larger; more broadly distributed and generally paralleled the

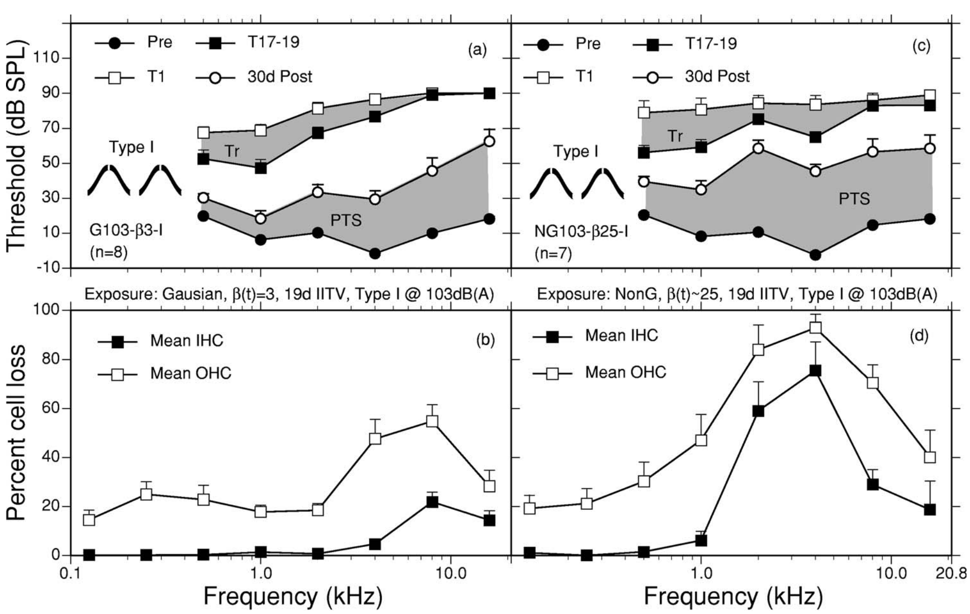

FIG. 6. Summary data from two of the groups exposed at 103 dB(A) SPL, to a 19 day, type I, IITV noise paradigm. Group G103-β3-I was exposed to a Gaussian $\beta(t)=3$ noise while group NG103-β25-I was exposed to a non-G, $\beta(t)\sim 25$ noise. (a) The group (G103-β3-I) mean thresholds measured immediately following exposure on day 1 (T_1) and the group mean thresholds measured immediately following exposure on days 17, 18, and 19 (T_{17-19}). T_r and PTS are shown shaded. The group mean preexposure thresholds, and 30 day postexposure thresholds are also shown. (b) The group mean outer and inner sensory cell (OHC, IHC) losses. (c), (d) A similar presentation of data for group NG103-β25-I. Error bars indicate the s.e. of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

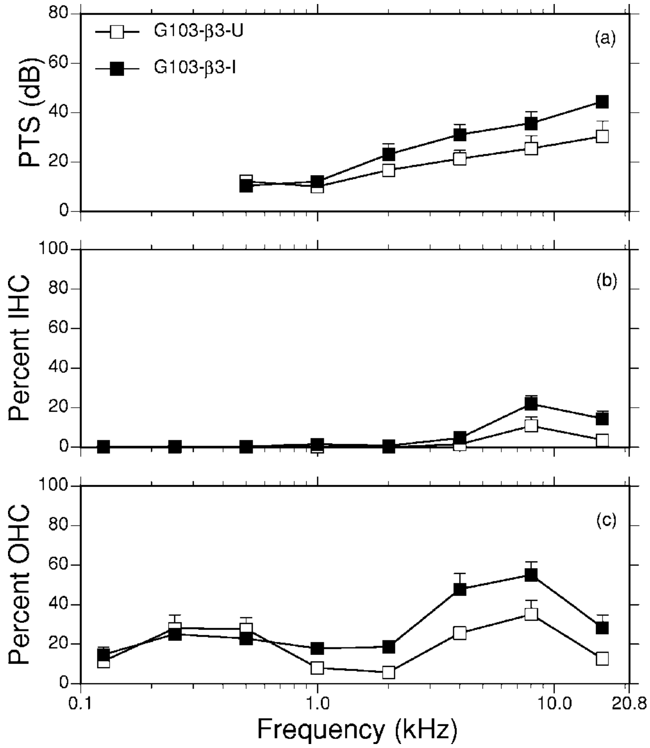

FIG. 8. A comparison of the (a) PTS, (b) IHC loss, and (c) OHC loss measured in group G103-β3-U exposed to a 5 day, uniform uninterrupted Gaussian noise and group G103-β3-I exposed to a 19 day Gaussian type I, IITV noise (Fig. 6) of equivalent energy. Error bars indicate the s.e. of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

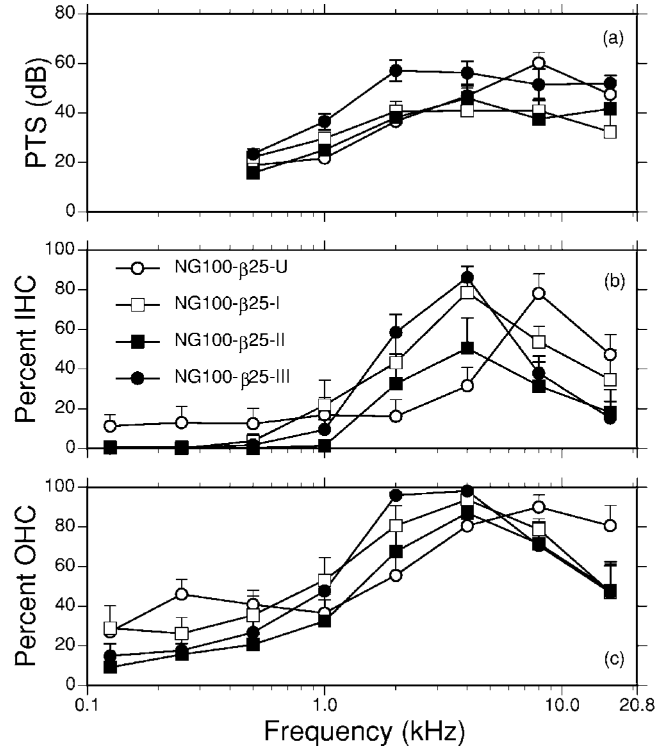

FIG. 9. A comparison of the (a) PTS, (b) IHC loss, and (c) OHC loss measured in four groups exposed at 100 dB(A) SPL noise at $\beta(t)\sim 25$. The three IITV groups NG100-β25-III, NG100-β25-I, and NG100-β25-II were exposed to a 19 day noise with type I, II and III level variations respectively. Group NG100-β25-U received an uninterrupted, uniform 5 day exposure. Error bars indicate the standard error (s.e.) of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

IHC loss. A two-way ANOVA confirmed these differences. For PTS, IHC loss, and OHC loss there was a statistically significant main effect of group and a significant interaction of group and frequency.

In this set of IITV exposures, differing only in the value of the kurtosis, there is a clear difference in trauma produced as a result of increasing the kurtosis; trauma increases with an increase in $\beta(t)$. These results are in agreement with the results of Hamernik *et al.* (2003) where a very different exposure paradigm was followed. Results from a variety of exposure conditions are now available showing that the kurtosis of the amplitude distribution of a noise is a variable that should be considered when trying to establish the hazards to hearing posed by long-term exposure to acoustically diverse noise exposures.

3. A comparison between the 5 day *uninterrupted and* 19 day *IITV Gaussian exposures*

Based on the results of Qiu *et al.* (2007) where Gaussian uninterrupted and IITV exposures at 100 and 106 dB(A) SPL were used one would anticipate that groups G103-β3-U [Figs. 3(c) and 3(d)] and G103-β3-I [Figs. 6(a) and 6(b)] would produce similar levels of trauma. These two groups are compared in Fig. 8. An ANOVA analysis of these data indicates that there is a significant main effect of group for the PTS and no interaction between group and frequency. For the IHC and OHC loss there is a significant main effect of group and an interaction of group and frequency. These differences are relatively small, amounting to a 12 dB maximum difference at 16 kHz in the PTS, 12% difference in the IHC loss at only 8 and 16 kHz, and less than 22% for OHC loss at the higher frequencies. The more traumatic of the two exposures was the 19 day IITV exposure. This is contrary to what would be expected since the IITV exposure produced a modest toughening effect and the interrupted paradigm should have given ample opportunity for thresholds to recover between the daily and weekly exposures.

D. The 100 dB(A) exposures

As discussed earlier (Fig. 3), the 100 dB(A) Gaussian reference exposure produced statistically significant less ATS, PTS, and OHC loss than did the 103 dB(A) Gaussian exposure. The results from the 103 dB(A) nonG exposures (Figs. 4 and 5) suggested that the equal energy principle applied to IITV exposures provided that the exposures had the same $\beta(t)$. In an effort to confirm this result, several groups of animals were exposed at 100 dB(A) SPL to various nonG, IITV exposures at two values of $\beta(t)$.

In addition to the IITV groups, one group at each $\beta(t)$ received an uninterrupted nonG exposure for 5 day, 24 h/day. Data summaries for the four groups (NG100-β25-U, NG100-β25-I, NG100-β25-II, NG100-β25-III) exposed to a $\beta(t)\sim 25$ noise are shown in Fig. 9 and for the $\beta(t)\sim 50$ groups (NG100-β55-U, NG100-β49-I, NG100-

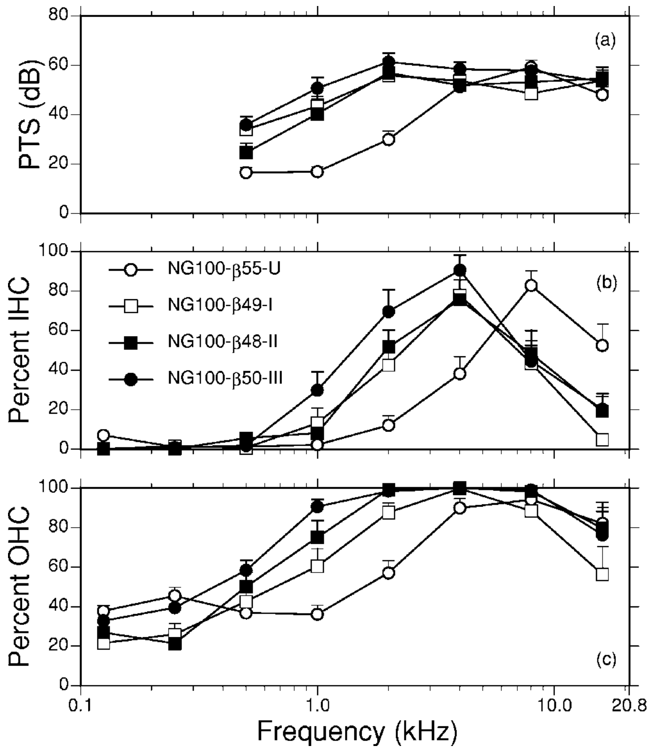

FIG. 10. A comparison of the (a) permanent threshold shift (PTS), (b) inner hair cell loss (IHC) and (c) outer hair cell loss (OHC) measured in four groups exposed at 100 dB(A) SPL noise at β(t) ~ 50. The three IITV groups NG100-β50-III, NG100-β49-I and NG100-β48-II were exposed to a 19 day noise with type I, II and III level variations respectively. Group NG100-β55-U received an uninterrupted, uniform 5 day exposure. Error bars indicate the standard error (s.e.) of the mean. If no error bar is present the s.e. is smaller than the size of the symbol.

β48-II, NG100-β50-III) in Fig. 10. Groups NG100-β25-U with $\beta(t) \sim 25$ and NG100-β55-U with $\beta(t) \sim 50$ received the 5 day, uninterrupted nonG exposure.

The two-way ANOVA on the data in Fig. 9 indicated that there was no significant main effect of group for the PTS, IHC loss, and OHC loss but there was a significant interaction of group and frequency. This interaction, indicating frequency specific effects, is the result of a shift in the pattern of PTS and sensory cell loss toward the higher frequencies for group NG100-β25-U, the group exposed for 5 continuous days, relative to the three IITV groups (NG100-β25-I, NG100-β25-II, NG100-β25-III). When an ANOVA is performed on only the three IITV groups, there is no interaction effect and no main effect of group for the OHC or IHC loss. There is, however, a main effect of group for the PTS. Group NG100-β25-III with the type III level variation tended to have the greater PTS, although the differences overall were small except at 2 kHz where the PTS exceeded that of the other two IITV groups by about 18 dB.

Increasing the kurtosis of four similar exposures from $\beta(t) \sim 25$ to $\beta(t) \sim 50$ produced an increased level of trauma (Fig. 10) as anticipated but an almost parallel set of frequency specific results to those shown in Fig. 9 for the $\beta(t) \sim 25$ exposures. A two-way ANOVA on the data in Fig. 10 indicated a significant interaction of group and frequency for all dependent variables as well as a main effect of group for the PTS and OHC loss but no main effect of group for the IHC loss. An ANOVA on the data from only the three groups receiving the IITV exposures (i.e., eliminating group NG100-β55-U that received the 5 day exposure) indicated no main effect of group and no interaction of group and frequency for all dependent variables. Thus all three IITV exposures at the higher level of kurtosis produced the same degree of trauma regardless of the type of level variation.

The data from the four exposures shown in Fig. 5 were consistent in showing the same effects on hearing regardless of whether the exposures were 19 day, IITV, or 5 day continuous, thus supporting the use of an energy metric for evaluating equivalent $\beta(t)$ exposures. Also surprising was that the 5 day uninterrupted exposure at 103 dB(A) produced the same effects as the three IITV exposures. Lowering the overall level of the noise by 3 dB did show that the IITV exposures were statistically similar regardless of the type of level variation as long as $\beta(t)$ was the same. However, unlike the results at 103 dB(A) shown in Fig. 5, there were small but significant differences between the 5 day uninterrupted and 19 day IITV exposures.

The toughening effects (T_r) measured from exposure to the 100 dB(A), IITV exposures were similar in magnitude and frequency distribution to that reported earlier for the 103 dB(A) exposures with over 20 dB of toughening being measured at 0.5 and 1.0 kHz. Since asymptotic thresholds at many frequencies were not limited by the AEP ceiling effect more accurate estimates of T_r could be made. As with the 103 dB, IITV groups this amount of toughening did not reduce the trauma. In general, the 100 dB(A), IITV exposures produced more PTS and cell loss at most frequencies than did the uninterrupted 5 day exposures (Figs. 9 and 10). A number of papers have shown that, as one might expect, interrupted exposures will typically produce less trauma than equivalent energy continuous exposures (Patuzzi, 1998; Campo and Lataye, 1992; and others). In the above-noted exposure paradigms the IITV exposures that produced modest levels of toughening also produced the same or more trauma than the uninterrupted reference exposures at the same $\beta(t)$.

This surprising result was also seen in the Gaussian IITV exposures reported by Qiu *et al.* (2007) and in the long-term impact noise exposures reported by Hamernik and Ahroon (1998).

IV. CONCLUSION

The above-presented data along with the results in Qiu *et al.* (2007) show that for a given total energy, temporal variations in the rms level of a noise exposure do not have a significant effect on the degree of trauma associated with Gaussian and nonG complex noise exposures. The data are also consistent in showing that under a variety of exposure conditions the kurtosis of the amplitude distribution is an exposure variable that should be considered when evaluating complex noise exposures for the purpose of hearing conservation practice. Increasing the kurtosis of an exposure at the same energy increases the PTS and sensory cell loss while exposures at the same kurtosis and energy regardless of their temporal complexity produce similar levels of trauma.

These data as well as the data from a number of referenced papers showing both support for and against the EEH suggest that while energy is a necessary metric for the evaluation of noise environments for hearing conservation purposes it is not sufficient. However, the results presented here along with those of Qiu *et al.* (2006, 2007) and Hamernik *et al.* (2003) suggest that energy and kurtosis may represent a necessary and sufficient set of metrics for such an evaluation. A better understanding of the role of the kurtosis metric in NIHL should lead to its incorporation into a new generation of more predictive damage risk criteria for noise exposure provided that human exposure and hearing loss data can be acquired from suitably designed epidemiological studies.

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