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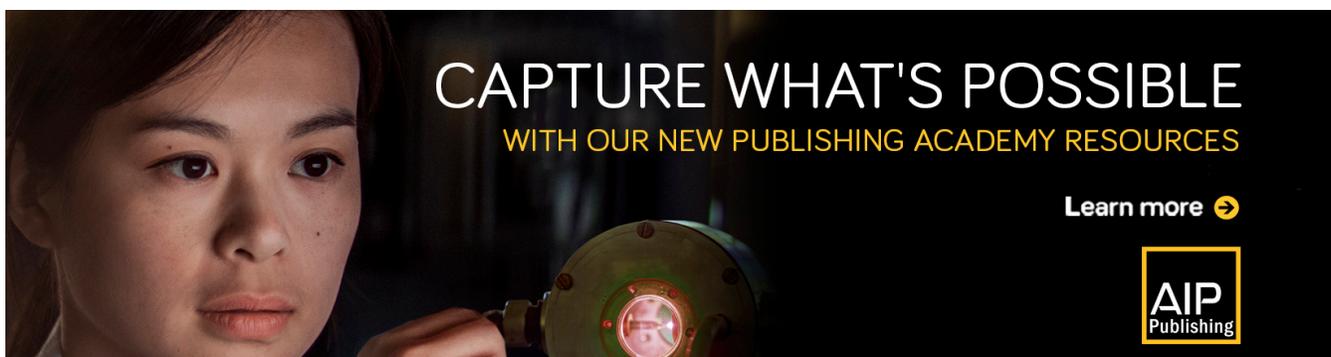
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# The effects of the amplitude distribution of equal energy exposures on noise-induced hearing loss: The kurtosis metric

Roger P. Hamernik,<sup>a)</sup> Wei Qiu, and Bob Davis

Auditory Research Laboratory, State University of New York, 107 Beaumont Hall, Plattsburgh, New York 12901

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Seventeen groups of chinchillas with 11 to 16 animals/group ( $\Sigma N=207$ ) were exposed for 5 days to either a Gaussian (G) noise or 1 of 16 different non-Gaussian (non-G) noises at 100 dB(A) SPL. All exposures had the same total energy and approximately the same flat spectrum but their statistical properties were varied to yield a series of exposure conditions that varied across a continuum from G through various non-G conditions to pure impact noise exposures. The non-G character of the noise was produced by inserting high level transients (impacts or noise bursts) into the otherwise G noise. The peak SPL of the transients, their bandwidth, and the intertransient intervals were varied, as was the rms level of the G noise. The statistical metric, kurtosis ( $\beta$ ), computed on the unfiltered noise  $\beta(t)$ , was varied  $3 \leq \beta(t) \leq 105$ . Brainstem auditory evoked responses were used to estimate hearing thresholds and surface preparation histology was used to determine sensory cell loss. Trauma, as measured by asymptotic and permanent threshold shifts (ATS, PTS) and by sensory cell loss, was greater for all of the non-G exposure conditions. Permanent effects of the exposures increased as  $\beta(t)$  increased and reached an asymptote at  $\beta(t) \sim 40$ . For  $\beta(t) > 40$  varying the interval or peak histograms did not alter the level of trauma, suggesting that, in the chinchilla model, for  $\beta(t) > 40$  an energy metric may be effective in evaluating the potential of non-G noise environments to produce hearing loss. Reducing the probability of a transient occurring could reduce the permanent effects of the non-G exposures. These results lend support to those standards documents that use an energy metric for gauging the hazard of exposure but only after applying a "correction factor" when high level transients are present. Computing  $\beta$  on the filtered noise signal [ $\beta(f)$ ] provides a frequency specific metric for the non-G noises that is correlated with the additional frequency specific outer hair cell loss produced by the non-G noise. The data from the abundant and varied exposure conditions show that the kurtosis of the amplitude distribution of a noise environment is an important variable in determining the hazards to hearing posed by non-Gaussian noise environments. © 2003 Acoustical Society of America. [DOI: 10.1121/1.1582446]

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## I. INTRODUCTION

The continuum of noise exposures that produce hearing loss runs from high level continuous Gaussian (G) noise through to military type impulse noise. Studies of noise-induced hearing loss (NIHL) using animal models have most often used either various bandwidths of continuous G noise or impact/impulse transients as the trauma-producing stimulus. Few experimental data are available on the effects of the infinite types of noise that lie between these two extremes, i.e., non-Gaussian (non-G) continuous noise. Industrial noise environments are often non-G, nonstationary and contain high-level noise bursts and/or impact sources.

Hamernik *et al.* (1974) combined high-level noise impulses of the Friedlander type with a G continuous noise to produce a continuous non-G signal. Chinchillas exposed to this noise showed a disproportionate increase in permanent threshold shift (PTS) and sensory cell loss than that produced by either the continuous or impulse noise alone. This potentiation of trauma was eliminated when the impulse was pre-

sented in a 2-s window of silence in the otherwise continuous G noise. More recently, Lei *et al.* (1994) showed that continuous non-G noise exposures would produce more trauma than energy and spectrally equivalent G exposures. The non-G character of the noise was produced by the insertion of high-level noise bursts or impact transients into the otherwise G noise. They showed that the increased trauma was related to the kurtosis,  $\beta(t)$ , of the non-G signal. The systematic relation between trauma metrics and  $\beta(t)$  was also shown to be reflected in the spectral distribution of energy in the transients that gave the noise its non-G character (Hamernik and Qiu, 2001). That is, for a fixed long-term spectrum and energy and a constant  $\beta(t)$ , trauma was directly related to the bandwidth of the transient stimulus. They also showed that over a limited range of  $\beta(t)$  the frequency specific kurtosis  $\beta(f)$ , computed on the filtered noise signal, was well correlated with the additional frequency specific OHC loss produced by the non-G noise relative to the G control condition. These and other animal model experiments (Dunn *et al.*, 1991; Lataye and Campo, 1996) as well as industrial epidemiological data (Thiery and Meyer-Bisch, 1988) suggest limitations on the use of energy-based metrics such as

<sup>a)</sup>Electronic mail: roger.hamernik@plattsburgh.edu

TABLE I. Summary of exposure conditions

Group no.	Noise type <sup>a</sup>	$N$	$\beta(t)$	Peak range (dB SPL)	Impulse probability	$L_n$ -background $L_{eq}$ (dBA)
43	(1)	16	3	-	-	-
44	(2)	11	25	[115, 128]	0.6	95.5
51	(2)	12	75	[118, 130]	0.6	91
52	(2)	11	53	[123, 127]	0.6	92.6
53	(2)	12	61	[117, 130]	0.2	94
54	(2)	11	25	[115, 128]	<sup>b</sup>	94
60	(2)	16	39	[115, 129]	0.6	93.5
63	(2)	11	95	[116, 128]	0.6	0
64	(2)	12	12	[116, 126]	0.6	97
49	(3)	12	33	[115, 129]	0.6	91.5
55	(3)	12	25	[115, 129]	0.6	94.5
65	(3)	12	105	[127, 132]	0.1	0
66	(3)	12	15	[113, 127]	0.6	96
68	(3)	11	65	[128, 133]	0.1	96
70	(4)	12	27	[104, 115]	0.6	91.3
71	(3)	12	55	[128, 133]	0.6	0
72	(3)	12	27	[125, 130]	0.1	96.5

<sup>a</sup>Noise type: (1) Broadband Gaussian. (2) Non-Gaussian noise, with or without a Gaussian background component defined by  $L_n$ . The transients which provide the non-Gaussian character of the noise were impacts created from three 400-Hz bands of energy centered at 1, 2, and 4 kHz. (3) Same as in (2) except the transients are broadband (710–5680 Hz for group 49 and 1–10 kHz for groups 55, 65, 66, 68, 71, and 72). (4) For this exposure the transient was a broadband (710–5680 Hz) noise burst (100 ms) having a rms SPL between 104 and 115 dB.

<sup>b</sup>Impacts occur regularly 1/1.5 s.

the  $L_{eq}$  which are the foundation of current international standards.

The data presented here extend the results of Lei *et al.* (1994) and Hamernik and Qiu (2001) by (1) extending the range of  $\beta(t)$  for each of two series of exposures whose transients have different spectra, and (2) varying the probability of the occurrence of a transient.  $\beta(t)$  was varied by changing both the interval and peak histograms of the noise signal.

## II. METHODS

Two hundred and seven (207) chinchillas (between 1 and 2 years old), randomly distributed among 17 exposure groups, were used as subjects. Each animal was made monaural by the surgical destruction, under anesthesia, 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 (AEP). The AEP was used to estimate pure tone thresholds and surface preparations of the organ of Corti were used to estimate the inner and outer hair cell (IHC, OHC) populations. Additional details of the experimental methods, beyond those presented below, may be found in Ahroon *et al.* (1993).

### A. Threshold testing

AEP audiograms were measured at octave intervals from 0.5 to 16.0 kHz. The mean (in dB SPL) of three threshold determinations measured on different days defined each animal's pre- and 30-d post-exposure audiogram. A complete audiogram was measured once daily during each of the 5 days of the exposures and the average (in dB SPL) taken over the 5 days established the mean asymptotic threshold levels and shifts.

### B. 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 to 24 h of fixation the cochlea was postfixed in 1% OsO<sub>4</sub> in veronal acetate buffer. Surface preparation mounts of the entire organ of Corti were prepared and IHC, 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 their characteristic phalangeal scars. For purposes of this presentation, sensory cell population data is presented as group averages (in percent or numbers missing) taken over octave-band lengths of the cochlea centered on the primary AEP test frequencies.

### C. Noise exposures

The noise field was monitored with a Larson Davis 814 sound level meter equipped with a  $\frac{1}{2}$ -in. microphone. The acoustic signal produced by the Electro-Voice Xi-1152/94 speaker system was transduced by a Brüel and Kjær  $\frac{1}{2}$ -in. microphone (Model 4134), amplified by a Brüel and Kjær (Model 2610) measuring amplifier and fed to a Windows PC-based analysis system. The design and digital generation of the acoustic signal is detailed in Hsueh and Hamernik (1990, 1991).

During exposure, individual chinchillas were confined to cages (10×11×16 in.<sup>3</sup>) with free access to food and water. The 17 groups of animals were exposed to one of the exposure protocols outlined in Table I. The exposure field was uniform to within 2 dB. The exposures lasted 24-h/day for 5

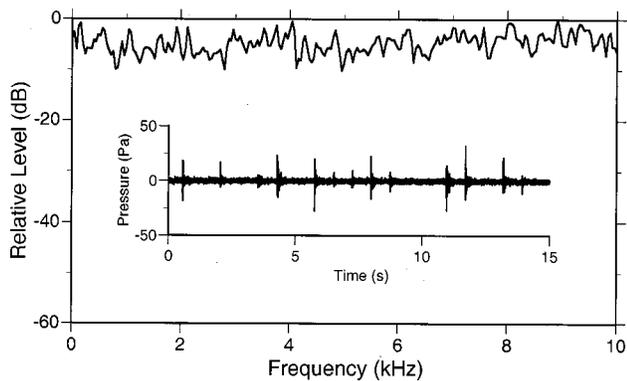


FIG. 1. The average spectrum of a 40-s sample of one of the noise signals. This long-term spectrum was common to all 17 noise exposures. Inset: A 15-s sample of one of the non-Gaussian waveforms. The peak SPL was randomly varied and the probability of an impact occurring in a 750-ms window was 0.6.

days and were interrupted once daily for approximately 20–30 min. for AEP testing. The 5-day continuous exposures produced an asymptotic threshold shift (ATS).

Each exposure had in common approximately the same flat spectrum between 0.125 and 10.0 kHz shown in Fig. 1 and was presented at an  $L_{eq} = 100$  dB(A). This level was chosen so that the G exposure condition would produce consistent but relatively small permanent changes in hearing and sensory cell populations. The 17 exposures differed only in their temporal structures, which were designed to produce 1 G and 16 non-G exposure conditions. The non-G conditions were designed in the frequency domain as described by Hsueh and Hamernik (1990, 1991) and were the result of inserting impacts or noise bursts (group 70), whose spectra were complementary to the background G noise, into the otherwise G signal. Two classes of non-G signals that incorporated impacts were generated. One was produced by inserting impact transients that were generated from three

400-Hz bands of energy centered at 1, 2, and 4 kHz. The other class was produced using broadband impacts that derived their energy from the 710–5680-Hz or 1–10-kHz region of the spectrum. The former are referred to as the “three band” and the latter as the “broadband” non-G exposures. The transient peak levels were randomly varied between the limits indicated in Table I and the probability ( $P$ ) of an impact or noise burst occurring in a 750-ms window was set at either 0.6 or 0.1 except for groups 53 and 54. Peak and interval histograms, which varied for each condition, were constructed for each of the exposure conditions and  $\beta(t)$  and  $\beta(f)$  were computed.  $\beta(f)$  was computed on octave bandwidths of the filtered signal centered at the audiometric test frequencies to provide, in effect, a “kurtosis spectrum.”

### III. RESULTS AND DISCUSSION

#### A. Noise analysis

Figure 1 shows the average spectrum (relatively flat, broadband) obtained from a 40-s sample of the digitized waveform. This long-term spectrum was common to each of the 17 different exposure conditions. The inset shows a 15-s sample of one of the non-G, nonstationary waveforms. Figures 2(a) and (c) show the spectra of the “three band” and “broadband” impact transients that produced the non-G signals along with a sample waveform. A second type of broadband (0 to 10 kHz) transient stimulus, whose spectrum is not shown, was used for the exposure of groups 55, 65, 66, 68, 71, and 72. Figures 2(b) and (d) show typical complementary spectra of the G components of the two broad classes of non-G signals. Table II presents the octave band and overall levels of each noise exposure. Values shown are the mean values obtained from eight 40-s samples of the digitized waveform.

Amplitude histograms for four of the non-G exposures are shown in Figs. 3(a)–(d). These were constructed

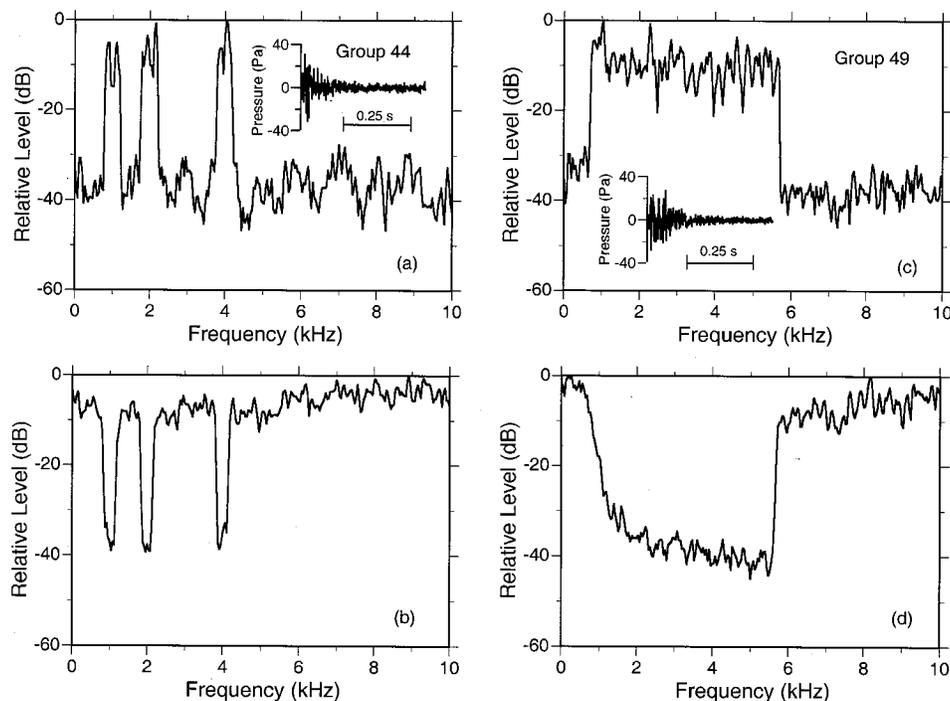


FIG. 2. The upper panels show the spectrum of (a) one of the “three band” impacts that was created from three 400-Hz bands of energy centered on 1.0, 2.0, and 4.0 kHz and (c) one of the “broadband” impacts that derived its energy from the 710–5680-Hz region of the spectrum. The insets show sample impact waveforms. Panels (b) and (d) show the respective complementary spectra of the Gaussian noise which was mixed with the impacts to create the non-Gaussian noise exposures. Group numbers 44 and 49 identify the specific exposures outlined in Table I.

TABLE II. Total and octave band sound pressure levels (dB SPL) over the 5-day exposure period for all experimental groups.

Group no.	Octave band cf (kHz)						Mean Leq	Mean Leq(A)	s.d.
	0.5	1	2	4	8	16			
43	89	89	88	91	99	98	103	100	0.04
44	82	93	95	95	95	95	101	100	0.26
49	86	95	94	96	93	93	101.5	101	0.65
51	84	97	93	93	91	89	101	100	0.89
52	88	98	94	94	93	94	102	101	0.18
53	87	97	93	93	94	94	101	100	0.81
54	86	97	93	93	94	96	102	100	0.40
55	93	93	89	91	97	93	102	100	0.77
60	86	96	92	93	92	93	101	100	0.67
63	85	98	92	92	83	77	100	100	0.44
64	88	96	93	95	96	96	103	101	0.39
65	96	95	89	90	94	84	103	100	0.35
66	91	91	90	94	99	95	103	100	0.17
68	95	94	89	92	97	94	103	101	0.52
70	86	93	93	96	93	94	101	100	0.34
71	95	93	90	94	97	86	103	100	0.45
72	92	91	89	94	98	95	103	100	0.52

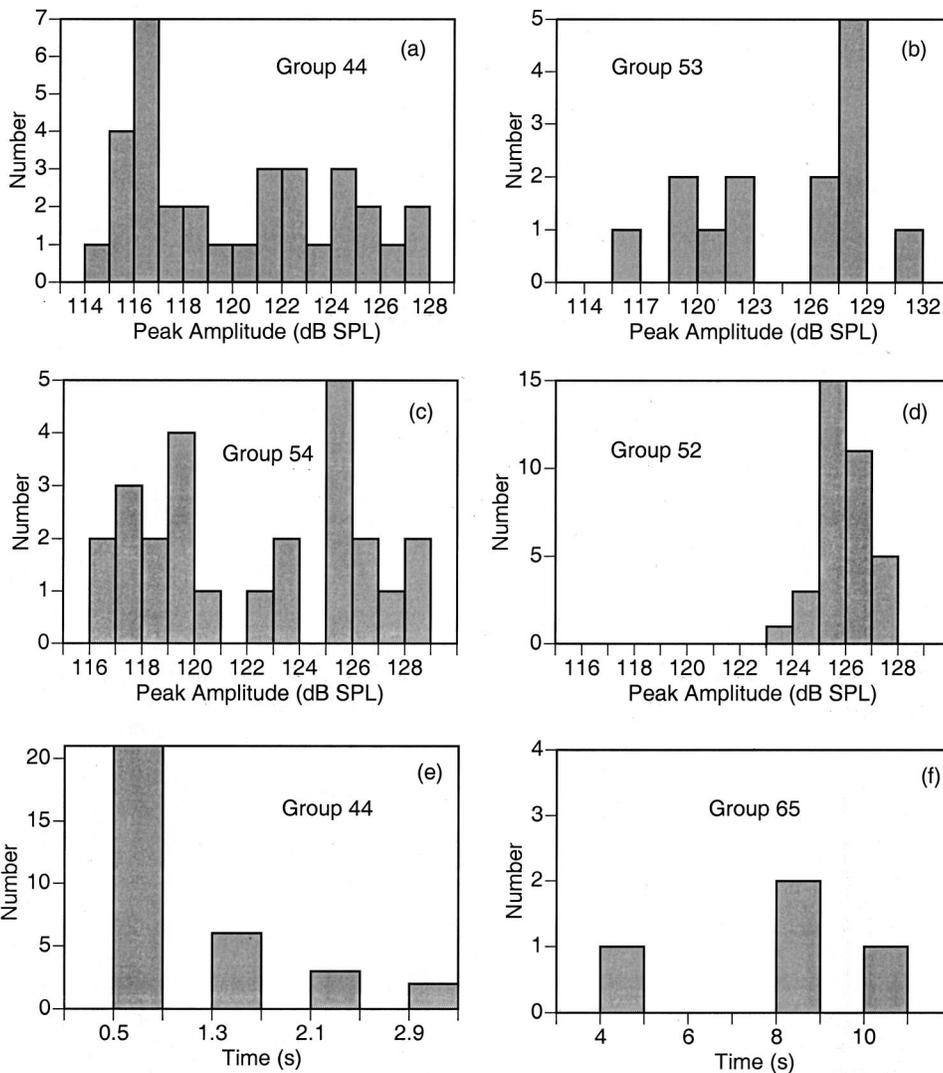


FIG. 3. Panels (a)–(d) show impact peak histograms from a 40-s sample of the exposures identified in each panel. Panels (e) and (f) show impact interval histograms for impact probabilities of 0.6 and 0.1, respectively.

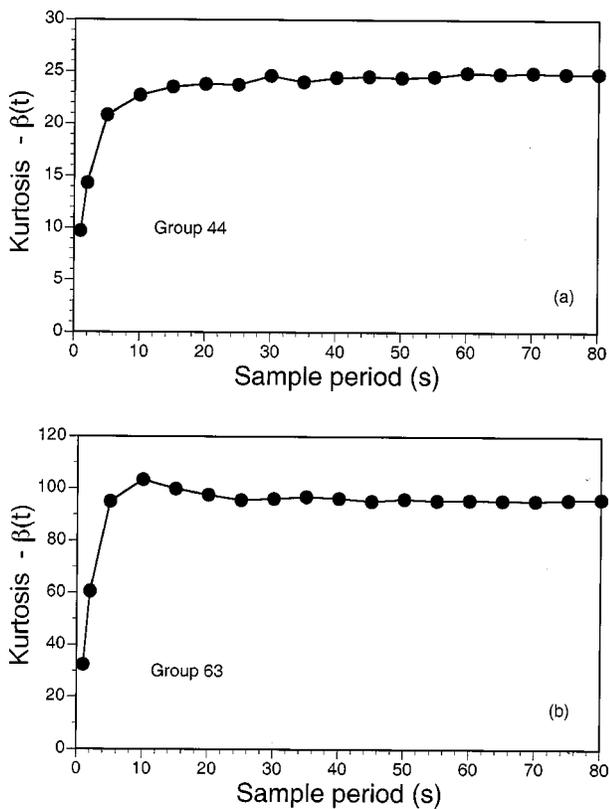


FIG. 4. The kurtosis  $\beta(t)$  computed on sample windows varying from 1 to 80 s for two of the non-Gaussian exposure groups 44 and 63.  $\beta(t)$  reaches a relatively stable value for windows greater than about 30 s.

from a 40-s sample of the noise. Since the peak levels were randomly generated, each sample of the signal will yield a different histogram. These histograms, however, illustrate that the net effect of randomizing the peaks is to produce very different distributions for each of the non-Gaussian exposures. Note that for group 52 the peak SPLs are, by design, nearly constant. The peak interval histograms for two exposure conditions illustrated in Figs. 3(e) and (f) reflect the 0.6 and 0.1 probability of an impact occurring in a 750-ms window respectively. Note that for group 54 the impacts were presented at a constant rate of 1/1.5 s and that the probability

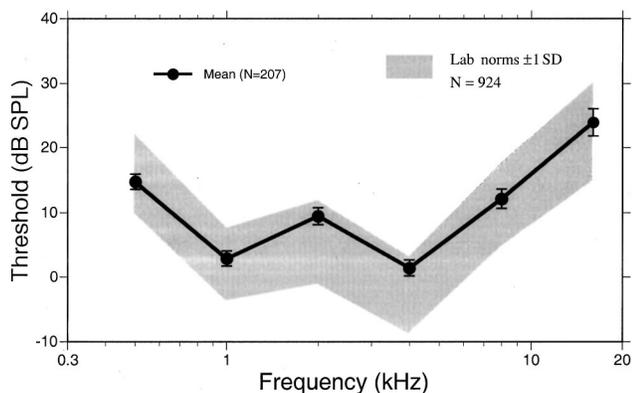


FIG. 5. The mean ( $N=207$ ) AEP audiogram for the animals used in the various exposure conditions outlined in Table I compared to the laboratory average based on  $N=924$  animals. The shaded region represents  $\pm$  one standard deviation of the laboratory standard. Bars on the data points represent one standard error of the mean.

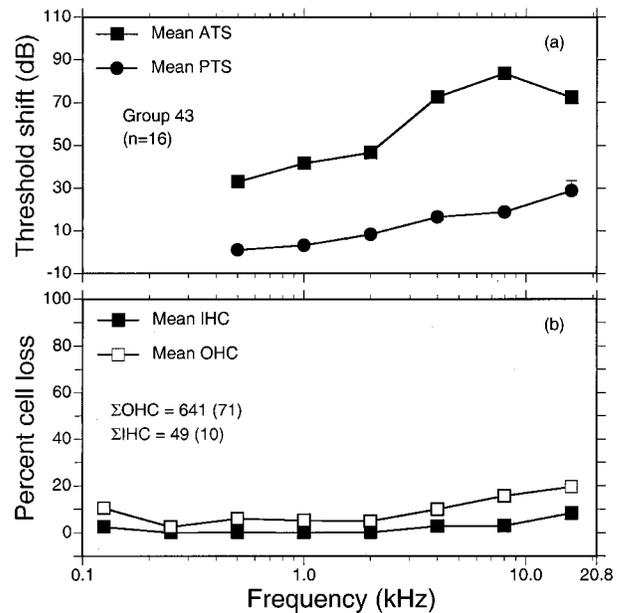


FIG. 6. (a) The group mean asymptotic and permanent threshold shifts (ATS, PTS) for group 43 exposed to the Gaussian noise. ATS is the average of the daily AEP threshold estimates over the 5-day exposure. PTS is the average of three threshold estimates taken 30 days postexposure. (b) The group mean percent outer and inner hair cell (OHC, IHC) loss averaged over adjacent octave band lengths of the basilar membrane. Bar=standard error. Total average numbers of IHCs and OHCs missing and standard errors are also given.

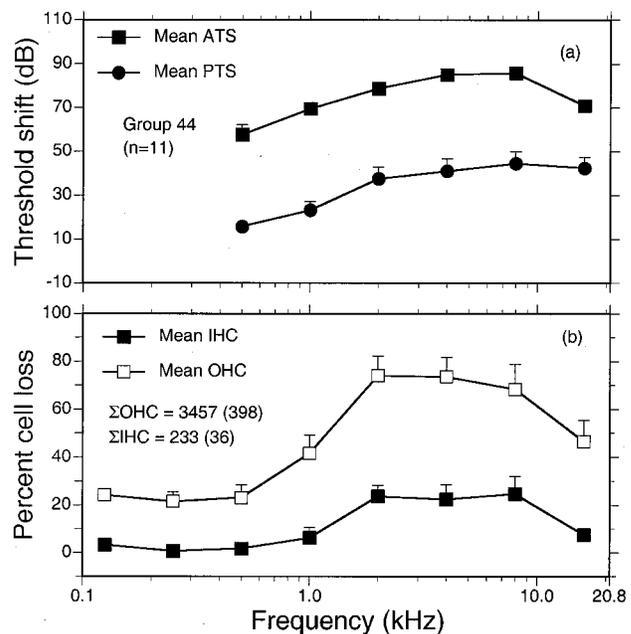


FIG. 7. (a) The group mean asymptotic and permanent threshold shifts (ATS, PTS) for group 44 exposed to a non-Gaussian noise. ATS is the average of the daily AEP threshold estimates over the 5-day exposure. PTS is the average of three threshold estimates taken 30 days postexposure. (b) The group mean percent outer and inner hair cell (OHC, IHC) loss averaged over adjacent octave band lengths of the basilar membrane. Bars = standard errors. Total average numbers of IHCs and OHCs missing and standard errors are also given.

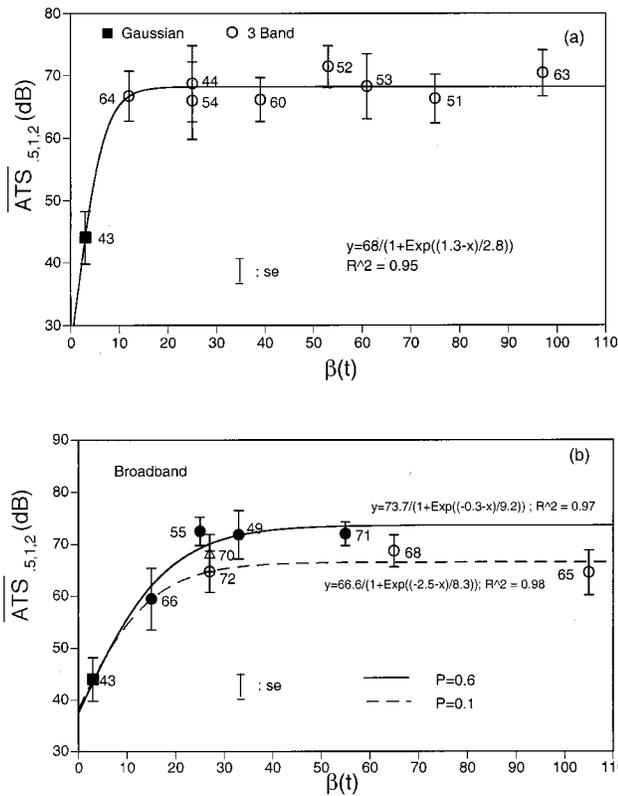


FIG. 8. The group mean asymptotic threshold shift averaged over the 0.5, 1.0, and 2.0 kHz AEP test frequencies ( $\overline{ATS}_{.5,1,2}$ ) as  $\beta(t)$  is increased for (a) the “three band” class of non-Gaussian noise exposures (○) and (b) the “broadband” class of non-Gaussian noise exposures (○,  $P=0.1$ ; ●,  $P=0.6$ ). ■ = the Gaussian reference exposure. Bars = standard errors. The nonlinear regression equation and the coefficient of determination ( $r^2$ ) are given.

of an impact occurring in group 53 was set at 0.2.

The kurtosis  $\beta$  of the sample distribution is defined as the ratio of the fourth-order central moment to the squared second-order central moment of the distribution.  $\beta$  is sensitive to the various parameters that define a complex noise such as the levels and durations of the transients, temporal structure of the noise, the crest factor, etc., as well as to the duration of the noise sample over which  $\beta$  is computed. The values of  $\beta$  presented in Table I are an average computed over eight 40-s sample of the noise. Figure 4 shows the typical asymptotic behavior of  $\beta$  for two of the exposure conditions as the sample duration is increased. For all the noise conditions used,  $\beta$  reached an approximately constant value for a sample window duration of approximately 40 s or more.

## B. Audiometric and histological effects of exposure

Mean AEP preexposure thresholds for all animals ( $N = 207$ ) are compared to the laboratory standard ( $N = 924$ ) in Fig. 5. Standard errors were small and all animals and groups fell within  $\pm$  one standard deviation of laboratory norms and reported thresholds for the chinchilla (Fay, 1988). ANOVA analysis indicated no statistically significant effect of group. In this and all other figures bars denote the standard error. Where a bar is not shown, the standard error was less than the size of the datum symbol.

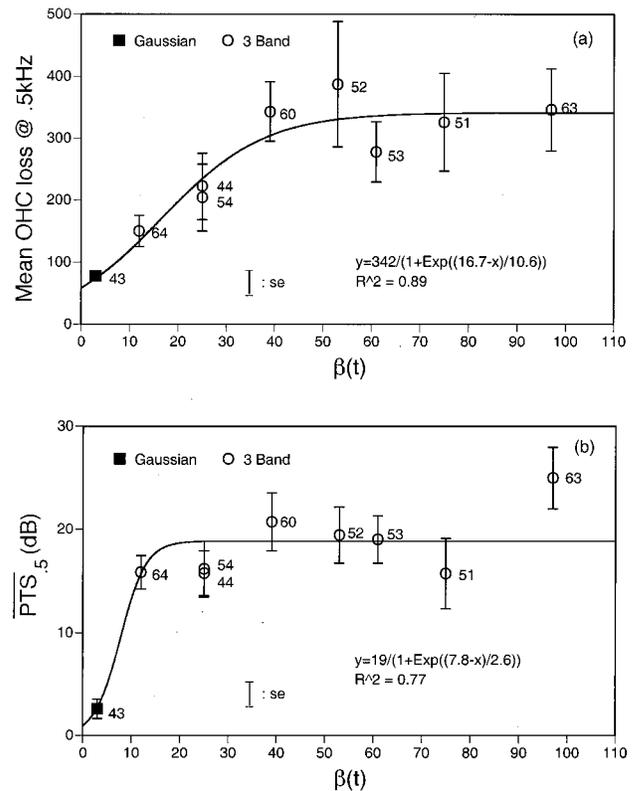


FIG. 9. (a) The group mean number of outer hair cells (OHC) lost in the 0.5-kHz octave band region of the basilar membrane and (b) the group mean permanent threshold shift measured at the 0.5-kHz test frequency ( $\overline{PTS}_5$ ) as  $\beta(t)$  is increased. ○ = the “three band” class of non-Gaussian noise exposures. ■ = the Gaussian reference condition. Bar = standard error. The nonlinear regression equation and the coefficient of determination ( $r^2$ ) are given.

Figures 6 and 7 show examples of complete data sets for two of the exposure conditions; group 43, the G,  $\beta(t) = 3$  reference condition, and group 44, a non-G,  $\beta(t) = 25$  condition. The upper panels present the group mean ATS and PTS while the lower panels show the corresponding cochleograms. Total OHC and IHC losses are also presented along with the standard errors. These two figures illustrate one of the consistent findings to emerge from all of these exposures. Specifically, for an equivalent spectral distribution of energy and total energy, the non-G exposures produce more ATS, PTS and sensory cell loss than does the energy equivalent G exposure. Similar data summaries were prepared for the other 15 exposure conditions. It is from data files such as these that all the remaining figures are derived.

Figure 8 shows the mean ATS averaged over the 0.5-, 1.0-, and 2.0-kHz test frequencies ( $\overline{ATS}_{.5,1,2}$ ) for the “three band” (upper panel) and the “broadband” (lower panel) non-G exposures. (Note: The “three band” and “broadband” designations in this and remaining figures refer to the spectral content of the transients that produced the non-G noise exposures. The numbers next to the datum points in this and subsequent figures refer to exposure conditions shown in Table I.) The lower test frequencies were chosen for this average because the ATS levels at the higher test frequencies, for the non-G conditions, reached or exceeded the upper limits of our AEP testing system. For all exposure conditions

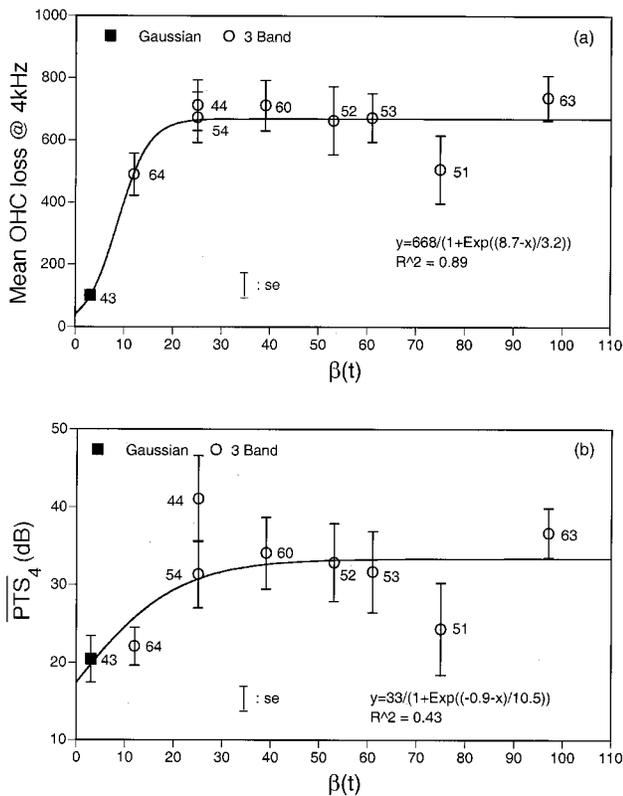


FIG. 10. (a) The group mean number of outer hair cells (OHC) lost in the 4.0-kHz octave band region of the basilar membrane and (b) the group mean permanent threshold shift measured at the 4.0-kHz test frequency ( $\overline{PTS}_4$ ) as  $\beta(t)$  is increased.  $\circ$  = the “three band” class of non-Gaussian noise exposures.  $\blacksquare$  = the Gaussian reference condition. Bar = standard error. The nonlinear regression equation and the coefficient of determination ( $r^2$ ) are given.

shown in Fig. 8(a),  $\overline{ATS}_{.5,1,2}$  exceeded that produced by the G noise (group 43) by 20 dB or more and was relatively constant across the eight different non-Gaussian exposure conditions regardless of the statistical properties of the non-G noise. The exposure of group 63 (the end point of this sequence of exposures) consisted of a sequence of random transients of the type shown in Figs. 2(a) and (b), i.e., there was no background G component ( $L_b = 0$ ). Thus, although group 63 has a different spectrum, it represents the end of a continuum of exposures that start with the G exposure of group 43. Note that in order to increase  $\beta(t)$  the level of the G component of the non-G exposures was gradually reduced from a high of 97 dB for group 64, to 91 dB for group 51, and finally to zero for group 63, i.e., the crest factor was increased. A similar presentation of data is shown in Fig. 8(b) for the “broadband” conditions and for two different probabilities ( $P = 0.6$  and  $P = 0.1$ ) for generating the transients in the non-G noise. For the “broadband” non-G exposures,  $\overline{ATS}_{.5,1,2}$  gradually increased as  $\beta(t)$  increased and appeared to reach an upper limit at a  $\beta(t)$  of approximately 40, of about 75 dB for the  $P = 0.6$  condition and 65 dB for the  $P = 0.1$  condition. This is about 30 and 20 dB respectively greater than that produced by the G condition. Note also in this series of exposures that groups 71 and 65 were exposed to pure impact noise.

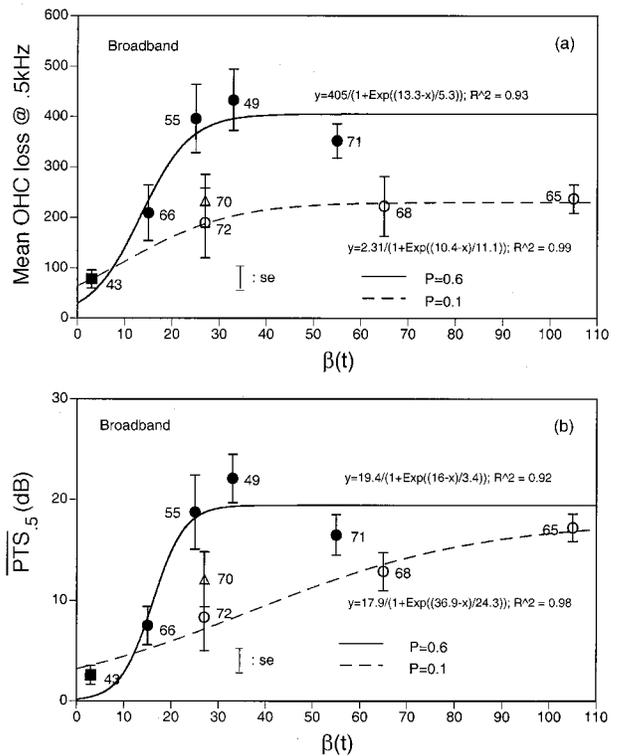


FIG. 11. (a) The group mean number of outer hair cells (OHC) lost in the 0.5-kHz octave band region of the basilar membrane and (b) the group mean permanent threshold shift measured at the 0.5-kHz test frequency ( $\overline{PTS}_5$ ) as  $\beta(t)$  is increased for the “broadband” class of non-Gaussian noise exposures.  $\bullet$ ,  $P = 0.6$ ;  $\circ$ ,  $P = 0.1$ ;  $\blacksquare$  = the Gaussian reference condition. Bar = standard error. The nonlinear regression equations and the coefficients of determination ( $r^2$ ) are given.

The permanent effects of these exposures, quantified by PTS and OHC loss, are presented in Figs. 9–14. Figures 9–12 [(a) panels] show OHC losses in the octave band length of the basilar membrane centered at 0.5 and 4.0 kHz, while the (b) panels show the corresponding PTS for the “three band” (Figs. 9 and 10) and “broadband” (Figs. 11 and 12) conditions. As seen with the ATS data, there is a consistent increase in the PTS of up to 35 dB and very large increases in OHC loss for the non-G conditions. Generally the greatest differences between the G and non-G conditions were found in the “broadband” groups for  $\beta(t) > 30$ . This result is in agreement with the data of Hamernik and Qiu (2001). The asymptotic behavior of both PTS and OHC loss for  $\beta(t) > \sim 40$  is also reflected in the frequency specific permanent losses. In the “broadband” conditions the exposures with  $P = 0.1$  consistently showed less permanent effects while in the “three band” conditions groups 53 with  $P = 0.2$  and 54 with impacts occurring at a constant rate of 1/1.5 s produced permanent effects that were similar to the  $P = 0.6$  exposure conditions.

Figures 13 and 14 show the group mean total number of OHCs lost and the mean PTS computed at 2.0, 4.0, and 8.0 kHz ( $\overline{PTS}_{2,4,8}$ ) for all exposure conditions. These frequencies were chosen for an average index of trauma since they generally showed the most PTS. There is a consistent relation between  $\beta(t)$  and the number of missing OHCs. In the “three band” non-G exposures, OHC loss exceeded that found fol-

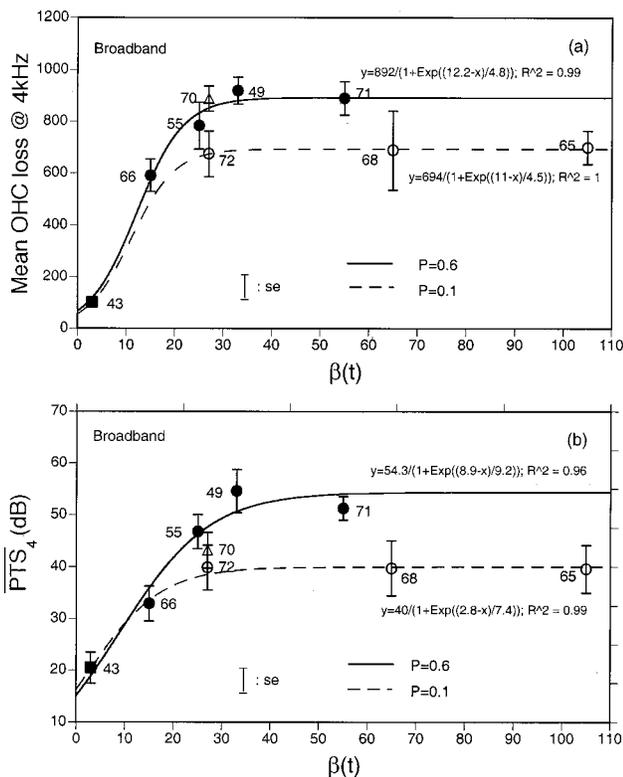


FIG. 12. (a) The group mean number of outer hair cells (OHC) lost in the 4.0-kHz octave band region of the basilar membrane and (b) the group mean permanent threshold shift measured at the 4.0-kHz test frequency ( $\overline{PTS}_4$ ) as  $\beta(t)$  is increased for the “broadband” class of non-Gaussian noise exposures. ●,  $P=0.6$ ; ○,  $P=0.1$ ; ■=the Gaussian reference condition. Bar = standard error. The nonlinear regression equations and the coefficients of determination ( $r^2$ ) are given.

lowing the G exposure by a factor of 5. The increased OHC loss was even greater for the “broadband” conditions.  $\overline{PTS}_{2,4,8}$  in the non-G conditions exceeded that produced by the G condition by 20 to 35 dB depending on the spectral features of the noise. There were no differences in the permanent effects among exposure groups 60, 52, and 53. For group 53 the peaks were random and the probability of occurrence was 0.2, while for exposure of group 52 with  $P=0.6$ , the peaks were kept relatively constant [Fig. 3(d)]. Note also that in Fig. 14 the level of trauma sustained by group 70 from a non-G exposure that incorporated noise bursts having  $P=0.6$  instead of impacts was considerably reduced compared to group 55 that had similar statistical properties but incorporated impacts. The trauma in group 70 fell roughly near the “broadband” impact conditions with  $P=0.1$ . The asymptotic behavior of the regression lines in Figs. 13 and 14 would indicate that, for the chinchilla model, under our limited exposure conditions, the use of an energy principal as proposed a number of years ago by Martin (1976) and others may be useful for the evaluation of non-G noise but only for  $\beta(t) > 40$ .

Figure 15 illustrates, for three of the exposure conditions, the relation between the  $\beta(f)$  spectrum and the frequency specific excess OHC loss produced by the non-Gaussian exposures (i.e., the OHC loss produced in each octave band by the non-Gaussian minus that produced by the

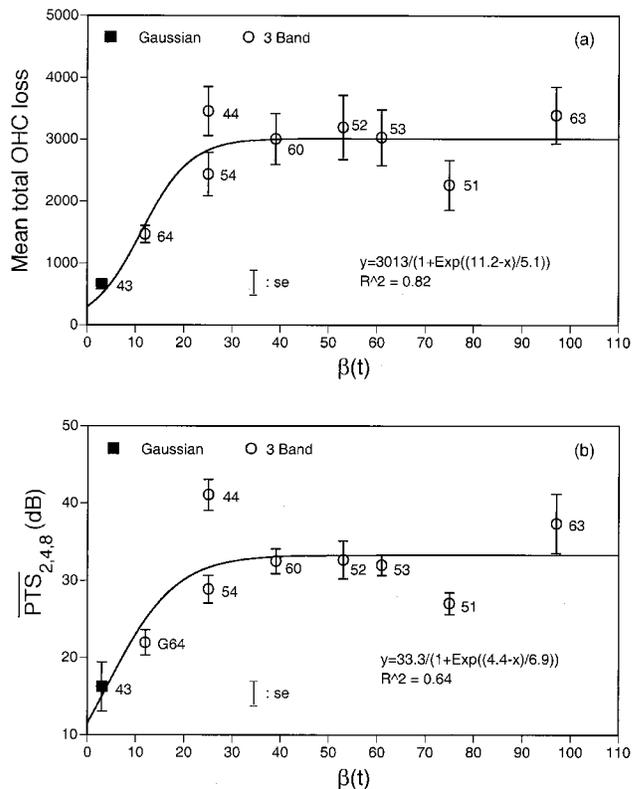


FIG. 13. (a) The group mean total number of outer hair cells (OHC) lost and (b) the group mean permanent threshold shift averaged at the 2.0-, 4.0-, and 8.0-kHz test frequencies ( $\overline{PTS}_{2,4,8}$ ) as  $\beta(t)$  is increased. ○=the “three band” class of non-Gaussian noise exposures. ■=the Gaussian reference condition. Bar=standard error. The nonlinear regression equation and the coefficient of determination ( $r^2$ ) are given.

Gaussian noise). In agreement with the results of Lei *et al.* (1994) and Hamernik and Qiu (2001), these examples illustrate the relatively close relation between  $\beta(f)$  and the additional OHC loss produced by non-Gaussian noise exposures.

#### IV. SUMMARY AND CONCLUSIONS

Under a variety of exposure conditions the results presented here agree with our earlier results (Hamernik and Qiu, 2001; Lei *et al.*, 1994) in showing the following: (1) Non-G noise exposures produce more hearing trauma than do energy and spectrally equivalent G noise exposures. This is true whether the transients that produce the non-G character of the noise are impacts or noise bursts (e.g., group 70). (2) The statistical metric, kurtosis ( $\beta$ ), in conjunction with an energy metric can identify hazardous exposure conditions not identified by conventional energy based metrics alone. In the chinchilla model, PTS and OHC loss are monotonically related to  $\beta(t)$  over the range  $3 < \beta(t) < 40$ . (3) The extent of trauma is related to the bandwidth of the transients that give the noise its non-G character. (4) A frequency specific kurtosis  $\beta(f)$  can be computed on the filtered acoustic signal.  $\beta(f)$  is well correlated with the frequency-specific OHC loss produced by non-G noise exposures relative to the energy and spectrally equivalent G exposures.

The results in this paper also extend our earlier results by showing the following:

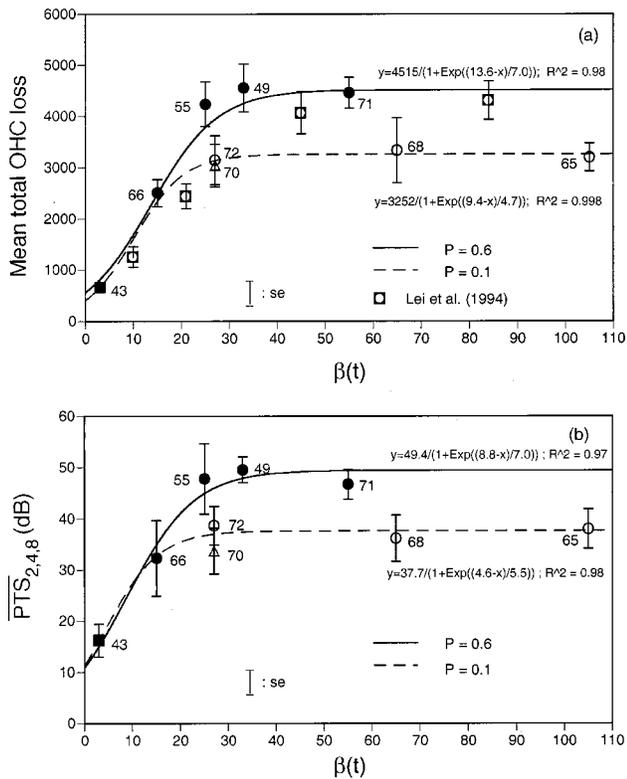


FIG. 14. (a) The group mean total number of outer hair cells (OHC) lost and (b) the group mean permanent threshold shift averaged at the 2.0-, 4.0-, and 8.0-kHz test frequencies ( $\overline{PTS}_{2,4,8}$ ) as  $\beta(t)$  is increased for the “broadband” class of non-Gaussian noise exposures. ●,  $P=0.6$ ; ○,  $P=0.1$ ; ■ = the Gaussian reference condition. Bar = standard error. The nonlinear regression equations and the coefficients of determination ( $r^2$ ) are given.

- (1) For  $\beta(t) > \sim 40$  and a fixed probability ( $P$ ) of a transient occurring, the degree of trauma as measured by ATS, PTS, or OHC loss remains constant despite changes in the statistical characteristics of the noise exposure. That is, varying the amplitude or the interval histogram of the transients had no effect on the indices of trauma. These variables are incorporated in the kurtosis metric. The function relating trauma to  $\beta(t)$  reaches an asymptote for  $\beta(t) > \sim 40$ . The asymptotic behavior of the function relating the indices of trauma to  $\beta(t)$  for  $P=0.6$  and  $P=0.1$  suggests that, over a range of values of the kurtosis, an energy-based approach can be used to assess the potential of an exposure to produce trauma. This result supports the approach in some standards documents (e.g., ISO-1999, 1990) that assign a “correction” factor for noise environments that contain impacts. There does not appear, however, to be experimental justification for what the value of the factor should be.
- (2) Decreasing the probability of a transient occurring from  $P=0.6$  to  $P=0.1$  reduced the degree of trauma for the exposures that incorporated broadband impacts. This would be expected since in the limit as  $P$  goes to zero, the exposure approaches the G condition. However, for the “3 band” series of exposures neither the  $P=0.2$  (group 53) nor the constant impact presentation rate (group 54) showed a level of trauma that differentiated these groups from those with exposures having  $P=0.6$ .

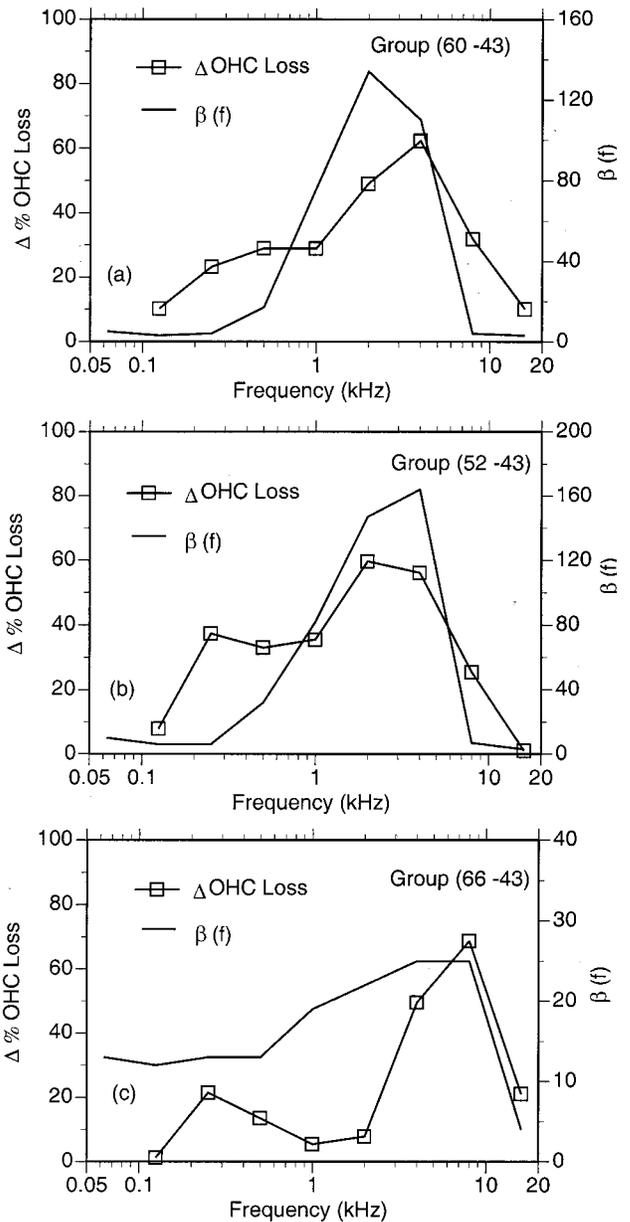


FIG. 15. Three examples of the relation between the increased octave band percent outer hair cell loss produced by the non-Gaussian exposure relative to the energy equivalent Gaussian exposure and the “kurtosis spectrum”  $\beta(f)$ .  $\Delta\%OHC$  loss = the percent OHC loss produced in a given octave band region by the non-Gaussian noise minus that produced by the Gaussian noise.

- (3) One of the exposures (group 70) used random noise bursts instead of impacts to produce the non-G exposure. The trauma produced by this exposure, while greater than that produced by the G noise, was significantly less than that which was produced by the exposure conditions for group 55. The exposures for groups 55 and 70 had approximately the same  $\beta(t)$  and differed only in the envelope of the transient and  $L_b$ . Although the envelope of the transient and its rise time are incorporated in  $\beta(t)$ ,  $\beta(t)$  alone could not be used to differentiate between these exposures. However, when data from two groups that used noise burst transients to produce non-G noise in the Lei *et al.* (1994) paper are compared with the results of group 70 [Fig. 14(a)], a trauma versus  $\beta(t)$  func-

tion emerges that parallels the function derived from the impact data. Specifically, in the Lei *et al.* paper, groups CN VII with  $\beta(t)=21$  and CN IX with  $\beta(t)=10$  showed an increased trauma relative to the G condition as seen with our group 70 with  $\beta(t)=27$ . The data from these three conditions are well ordered with respect to  $\beta(t)$ . This suggests that “correction” factors in standards need to be used not only for impact containing noise environments but also for any non-G environment whether or not the transients are impacts. Note further that for the three exposures identified above there is a linear relation between trauma and  $\beta(t)$  with, as in the case of impacts, trauma increasing with increasing  $\beta(t)$ . For these exposures having  $\beta(t)<40$ , any asymptotic behavior of the trauma versus  $\beta(t)$  function could not be determined. Note also that the data from the two exposure conditions in the Lei *et al.* paper that employed impacts (i.e., Groups CN VI and IX) and whose statistical properties were different from those used in the present paper, fall along the asymptotic line determined by the  $P=0.6$  exposure conditions (Fig. 14).

In conclusion, the data from the abundant and varied exposure conditions show that the kurtosis of the amplitude distribution of a noise environment is an important variable in determining the hazards to hearing posed by non-Gaussian noise environments.

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ratory Animals,” prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council [DHHS Publication No. (NIH) 86-23, revised 1985].

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