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LOW FREQUENCY NOISE LEGISLATION

Mariana Alves-Pereira[1], Jersy Motylewski[2], Elzbieta Kotlicka[3], Nuno A. A. Castelo Branco[4]

[1]Department of Environmental Sciences & Engineering (DCEA-FCT),
New University of Lisbon, 2829-516 Caparica, Portugal
[2]Institute of Fundamental Technological Research, Polish Academy of Sciences,
Warsaw, Poland
[3]Department of Physics, Warsaw University of Technology, Warsaw, Poland
[4]Center for Human Performance
Estrada Nacional No. 10, Edifício Cinema, Sala 109, 2615 Alverca, Portugal
vibroacoustic.disease@gmail.com

Abstract

Introduction. Legislation regarding low frequency noise (LFN, <500 Hz including infrasound), when existent, is highly deficient. Not only is it expressed in dBA, actually defeating the purpose of evaluating LFN, but no concrete measures are prescribed if excessive LFN is identified. The *status quo* notion that acoustical phenomena are only harmful when perceived by humans cannot be sustained given current scientific facts. The purpose of this report is to demonstrate just how inadequate legislation is regarding LFN control, and how ubiquitous LFN is in locations common to the general public. Methods. Noise assessments were conducted in homes, clubs, public transportation and common automobiles, in 1/3 octave bands and with a lower limiting frequency of 6.3 Hz, measured in dBLin. Overall average noise levels are reported in both dBA and dBLin. Results. Comparative frequency analysis among acoustic environments that possess the same dBA levels show that it is not scientifically valid to presume the existence of comparable acoustic environments merely based on a dBA level, i.e., equal dBA levels does not mean equal acoustic environments. Neither the dBA nor the dBLin parameter adequately reflect the presence of LFN components. Discussion. LFN is ubiquitous in modern society, and yet it is not adequately legislated. Noise-related studies do not take LFN in account and thus yield results that are deemed controversial, contradictory, and inconclusive. No effort is made to control LFN in the homes, nor in other locations of common use to the general public. The implications of ignoring LFN as an agent of disease for the public health is detrimental to us all as a human society, and a nightmare for future generations.

INTRODUCTION

Noise has always been an important environmental problem. According to the Epic of Gilgamesh, a Babylonian king who lived in 2700 BC, the Great Flood was brought to the planet Earth because the demigods were unable to sleep due to the noise produced by humans (1). In Ancient Greece, metalwork involving hammers was banned within city limits in 600 BC (2), and Pliney the Elder, in 50 CE, noted that people living near the cataracts of the Nile became hard of hearing (3). In Ancient Rome, legislation existed pertaining to the noise associated with iron wheels of wagons that disrupted sleep, while in certain cities of Medieval Europe, horse carriages were not allowed during night time (4).

Today, hearing conservation programs are ubiquitous among most noise-exposed workers, and seem to be effective in the prevention of hearing loss. Legislation is quite specific and limits noise exposure, by dB-level, to well-determined periods of time (in hourly increments), after which the worker must remove him or herself from the noisy environment (5,6).

Acoustical phenomena, however, can be much more than just “noise” that causes hearing impairment. Ultrasound (MHz range), is used in a variety of medical diagnostic procedures (for example) and is inaudible to humans. Infrasound, on the other end of the spectrum (<20 Hz), is also inaudible to humans. “Noise” at these frequencies is not heard, and thus cannot produce hearing loss. Consequently, neither ultrasound nor infrasound, are required to be assessed during routine noise evaluation procedures.

The human auditory system captures acoustical phenomena in the range of 20 to 20000 Hz. But the sensitivity at each frequency band is not the same, i.e., different dB levels are required at different frequency bands in order to perceive a sound with the same loudness. The human ear is most tuned to frequencies within the 1000-5000 Hz range; the resonance frequency of the ear is 3500 Hz, and it is within this range that most speech and language occur. Thus, to prevent hearing loss in noise-exposed individuals, measurements mandated by legislation focus primarily on the ranges where the smallest dB-level (sound pressure amplitude) produces audible sound: 1000-5000Hz.

Through a weighting network, or filter, routine noise measurements capture the overall amplitude of the acoustical environment *as if* it were being perceived by the human auditory system, i.e., linearly evaluating the sounds in the 1000-5000 Hz range while de-emphasizing acoustic phenomena below 500 Hz (See Fig. 1). The A-weighting network, which measures the overall amplitude in dBA, is “an approximation of equal loudness perception characteristics of human hearing for pure tones relative to a reference of 40 dB SPL (sound pressure level) at 1 kHz" (7). The A filter simulates human auditory thresholds and is appropriately employed when the goal is to avoid hearing loss. As a result, legislation regarding permissible exposure levels is usually based on dBA level measurements, and protection against noise is *exclusively* equated with hearing protection devices.

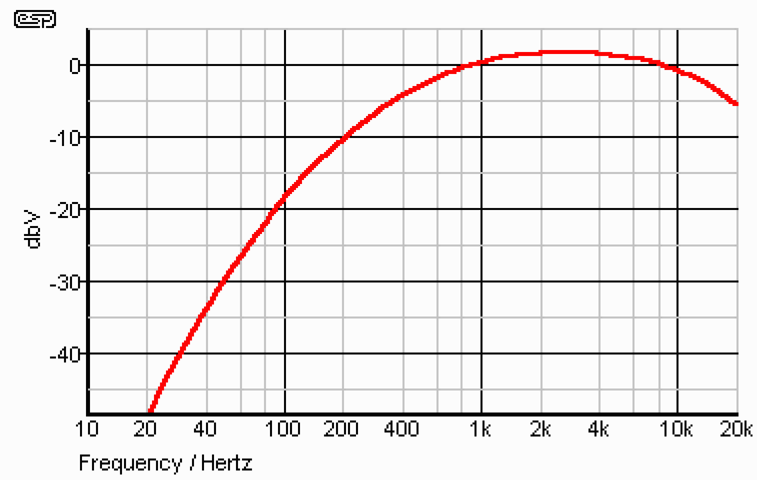

Figure 1 - A-weighting response curve. At 1000 Hz, there is a 0 dB difference between what is measured and what exists in the environment. At 100 Hz, there is a 20 dB difference between what is measured with this weighting system and what actually exists in the environment. Acoustic phenomena below 20 Hz and above 20 kHz are not assessed.

Throughout the decades, it has been assumed that two environments with similar dBA levels are comparable. Throughout the decades, biomedical studies regarding non-auditory pathology caused by noise exposure have been controversial, contradictory, and hence, inconclusive (8)[1]. Noise-induced, non-auditory pathology has led to a vigorous proliferation of scientific articles since Laird, in 1928, studied the effects of noise on typists and concluded that working in this type of noise environment had a physiological cost to humans (9). Numerous authors have referred to non-auditory pathology (10-21), however, controversial, contradictory, and hence, inconclusive is still the mainstream belief regarding noise-induced, non-auditory pathology (8,22,23).

Given that the vast majority of studies are only measuring the acoustic phenomena *as if* perceived by the human auditory system, it is possible that other acoustical phenomena, not perceived by the auditory system, or not conducive to hearing impairment, be present in the environment. Low frequency noise (LFN) (≤500 Hz, including infrasound) has been the object of study by Portuguese researchers since 1980 (24-26)[2]. LFN has been identified as a genotoxic agent of disease (27-30)[3], capable of inducing blood vessel wall thickening as seen in autopsy (8), as well as through light and electron microscopy studies (31-37)[4]. This can lead to well known consequences such as tumour development (38)[3], cardiac infarcts and/or the need for cardiac bypass surgery (24,31-33,39,40). The pathology caused by excessive exposure to LFN is termed vibroacoustic disease (VAD), and has been

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[1] See "Low frequency noise exposure as a confounding factor in biomedical science," included in these Proceedings.
[2] See "Vibroacoustic disease – a 25-year-old saga", included as a keynote speech in these Proceedings.
[3] See "Mutagenesis and malignancy in vibroacoustic disease," included in these Proceedings.
[4] See "Respiratory pathology in vibroacoustic disease II – Specific morphological changes", included in these Proceedings.

diagnosed among several occupational and environmentally exposed populations (24). In an attempt to compare VAD-related studies with research conducted by other authors, one reaches a dead-end: since no frequency spectra are usually provided by the vast majority of noise-related studies, the LFN content of the acoustic environment under study remains unknown.

The goal of this report is to show why acoustic environments must cease to be reported merely in terms of a dB- or dBA-level measurement, and should always include a frequency distribution analysis in order to correctly evaluate noise-induced changes.

METHODS

Within the context of ongoing studies on LFN-induced pathology and VAD, LFN is being evaluated in a variety of different locations in Portugal. All data were obtained with a sound level meter & real-time frequency analyser (Bruel & Kjaer, 2260 Observer, Denmark). Sound pressure levels were documented in dBA and dBLin. Frequency distributions (in dBLin) were obtained in 1/3 octave bands, with a lower limiting frequency of 6.3 Hz. Microphone (Bruel & Kjaer, 4189, Denmark) calibration was achieved with a 1000 Hz pistonphone (Bruel & Kjaer, 4231, Denmark).

Noise was evaluated in the following locations: a) outdoor café in a Portugeuse northern interior city of Viseu, with a 90 mm windscreen; b) inside the passenger cabin of a Lisbon subway, en route between the stations of Bela Vista and Chelas; and c) in the waiting room of a doctor’s office, located near a busy street in São Domings de Rana, a suburb of Lisbon.

RESULTS

Even though many locations had comparable dB(A) levels, the corresponding dB(Lin) values differed greatly (See Table 1).

Table 1 - Summary of dB-level data.

Location	**dB-Level (dBA)**	**dB-Level (dBLin)**
Outdoor Café	64.6	73.7
Subway	64.3	87.3
Doctor’s Office	64.0	75.4

The distribution of acoustic energy along the different 1/3 octave bands also differed among the 3 different locations (See Fig. 2).

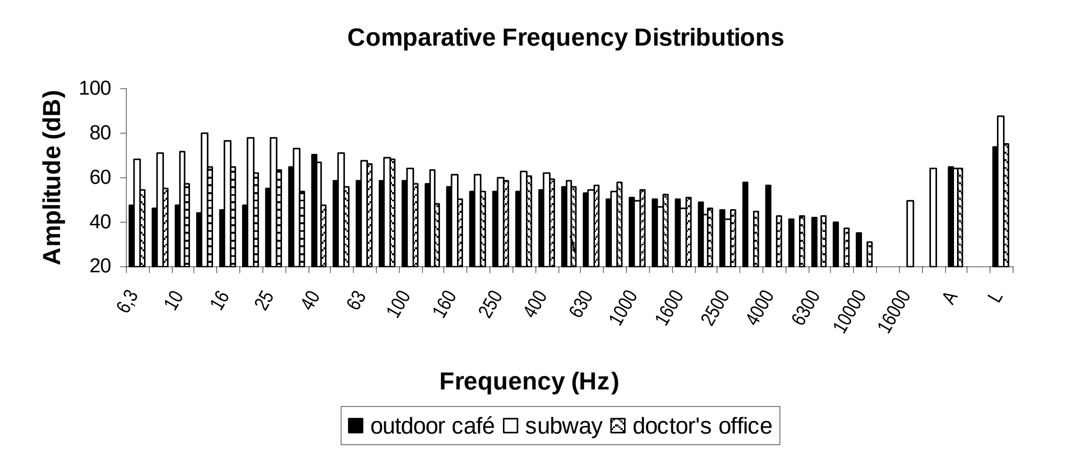

Figure 2 - Comparison of frequency distributions of measurements taken in 3 different locations: a) an outdoor café, b) passenger cabin of a Lisbon subway, and c) in a doctor's office waiting room. Although location dBA-level measurements are comparable (Bars labelled A) (64.6 vs. 64.3 vs. 64.0 respectively), their dBLin levels (Bars labelled L) show a much larger difference (73.7 vs. 87.3 vs. 75.4, respectively). The distribution of acoustic energy throughout the 1/3 octave frequency bands differs among all three locations, particularly in the lower frequency bands.

DISCUSSION

These results clearly indicate that "noisy" environments merely described by a dB-level measurement are acoustically insufficiently characterized. Considering that exposure to different frequencies induces different effects (41-44), comparing acoustical environments merely on the basis of a dB-level measurement is not a scientifically valid methodology. Each organ has its own resonance frequency so it cannot be assumed that they will respond equally when presented with "noisy" environments that have a dissimilar predominance of acoustic energy among the different 1/3 octave frequency bands.

Law- and policy-makers who insist on maintaining the dBA standard for all noise assessments, are providing a great disservice to human societies. The lack of frequency spectral analysis impedes any possible comparison amongst noise-related studies, consequently promoting the development of controversial, contradictory, and inconclusive studies. By not acknowledging LFN as an agent of disease, many noise studies conducted today and that do not adequately describe their acoustic environments (with information of amplitude and frequency) will certainly be deemed non-useful. A possible solution to the quagmire is the topic of an independent report[5].

[5] See "...And again low frequency noise – A possible solution," included in these Proceedings.

SUMMARY

1 – It is invalid to compare acoustical environments merely based on dB-level measurements because, despite comparable dB-level measurements, the distribution of the acoustic energy over the frequency spectra can be substantially distinct.

2 – Despite the fact that legislation does not require a frequency spectrum analysis, the amount and type of low frequency noise present in a “noisy” environment should be assessed.

3 – Since each organ has its own resonance frequency, it cannot be expected that equal responses will be obtained with acoustical environments that have dissimilar frequency distributions. Hence, biomedical studies, in particular, should cease to report their acoustical stimuli merely as a dB-level measurement, and should always include a frequency spectrum analysis.

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