Legislation Hinders Research Into Low Frequency Noise

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Abstract— **Routine noise evaluations, conducted primarily to avoid hearing loss, characterize the overall amplitude of the acoustic environment, while frequency distribution analyses are rarely performed. Alone, a dB-level measurement does not adequately characterize an acoustic environment; two locations may be comparable in terms of dB-levels, but quite distinct when their frequency distributions are taken into account. Vibroacoustic disease is a consequence of exposure to acoustic phenomena within the low frequency ranges (<500 Hz, including infrasound). However, because legislation does not require frequency distribution analyses, the real acoustic content of the vast majority of occupational and environmental noise exposure remains unknown.**

***Index Terms*—noise exposure, noise pollution, non-auditory pathology, vibroacoustic disease.**

I. Introduction

Excessive exposure to noise has always been equated with hearing loss: in Ancient Greece, metalwork involving hammers was banned within city limits in 600 BCE [1], and Pliny the Elder, in 50 CE, noted that people living near the cataracts of the Nile became hard of hearing [2]. 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 [3].

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 can capture 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. 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" [4]. 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 are usually based on dBA level measurements, and protection against noise is *exclusively* equated with hearing protection devices.

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 [5]. Noise-induced, non-auditory pathology has led to a vigorous

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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 [6]. Numerous authors have referred to non-auditory pathology [7]-[18], however, as states the 13th edition of Public Health and Preventive Medicine, "the effects of noise on bodily functions other than hearing are poorly understood" [19]. Regarding non-auditory effects of noise, the 4th edition of Industrial Hygiene, published in 1996 by the United States National Safety Council [20] states: "Research on [nonauditory] effects of noise has addressed interference with communication, altered performance, annoyance, and physiologic responses such as elevated blood pressure and sleep disturbances. Definitive studies have yet to be done on most of these issues." According to OSHA Technical Manual, "in addition to effects on hearing, noise: Interferes with speech; Causes a stress reaction; Interferes with sleep; Lowers morale; Reduces efficiency; Causes annoyance; Interferes with concentration; Causes fatigue" [21]. The Environmental Engineering Handbook states: "Noise is recognized as a form of pollution because it is a public health hazard causing hearing impariment, and a nuisance causing psychological stress" [22]. Controversial, contradictory, and hence, inconclusive is still the mainstream belief regarding noise-induced, non-auditory pathology [5], [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], [25]. LFN has been identified as an agent of disease [26]-[28] with a genotoxic component [29]-[32]. Long-term exposure (years) to LFN has been shown to cause vibroacoustic disease (VAD) [26]. Initially identified among aeronautical technicians [26]-[28], VAD has also been observed in military [33] and commercial pilots [34] and aircrew, and in a civilian population exposed to environmental LFN [35]. Other individuals who were unsuspectingly exposed to LFN have also been identified with VAD [27]. In an attempt to compare these studies with research conducted by other authors, one reaches a dead-end: no frequency spectrum is provided by the vast majority of noise-related studies. In fact, most biomedical studies describe their corresponding acoustical environments, or stimuli, only in terms of a dB-level measurement, most often than not, a dBA-level measurement.

Question: Is it valid to compare two acoustic environments based solely on their dBA (or dB) level?

II. Methods

Within the context of ongoing studies on LFN-induced pathology and VAD, LFN was evaluated in a variety of locations, in and around Lisbon. Those that had similar dBA levels were selected, and their frequency distributions were compared. Previous studies have shown that the acoustic environment of commercial airliner cockpits, rich in LFN, is conducive to the development of VAD [34]. Thus, the 1/3 octave band frequency spectra of the various locations were analyzed comparatively to the cockpit of the Airbus-340.

Noise was evaluated in the following locations: a) cockpit of commercial aircraft [36]; b) in the kitchen of an expensive restaurant during lunch hour; c) in a modern electric commuter train stopped at a station; d) and in a common European-made automobile, travelling alone on a highway (at 3 am), at a steady 120 km/h, with windows closed and radio off. For comparative purposes, noise was also evaluated within e) a modern trolley car; f) a dance club; g) a textile factory in the northern Portugal; and h) a boom car while stopped. (Boom cars are the names given to automobiles that possess sophisticated bass amplification devices, such as woofers and sub-woofers.)

Sound pressure levels (in dBA and dBLin) were measured with a modular precision sound level meter (Bruel & Kjaer, 2231, Denmark). Frequency spectra were obtained using a real-time frequency analyzer (Hewlett Packard, 3569 A, USA) in 1/3 octave frequency bands (from 6.3 Hz to 20000 Hz). Microphone calibration was achieved with a 250 Hz pistonphone (Bruel & Kjaer, 4228, Denmark) to a sound pressure level of 124 dB re: 20 μPa. To expand the lower limiting frequency, the 1/2 inch microphone (Bruel & Kjaer, 4165, Denmark) was attached with a coupler (Bruel & Kjaer, UC5265, Denmark), thus permitting the measurements to begin at 1.6 Hz

III. Results

A. Sound Pressure Levels

Table I describes the sound pressure levels, in dBA and dBLin, obtained at each location. Table II describes the difference in dBA and dBLin, when compared to the A340 cockpit.

B. Frequency Distribution

Figs. 1-7 compare the frequency distribution of the A340 cockpit with all other locations, within the 6.3-20 000Hz range. Fig. 8 shows the frequency distributions within the 1.6-500Hz range.

IV. Discussion

These results clearly indicate that "noisy" environments described merely by a dB-level measurement are acoustically insufficiently characterized. Considering that exposure to different frequencies induces different effects [37]-[41], comparing acoustical environments merely on the basis of a dB-level measurement is invalid. 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 frequency bands.

It should not be supposed, nor assumed, that individuals who attend dance clubs or who ride in trolley cars will develop VAD because, despite the higher dB-levels higher than in the cockpit, the time exposure pattern is very different. However, the effects of LFN are cumulative, and thus all sources of LFN must be taken into account when evaluating an individual's exposure.

A. No Frequency Spectra

The current working hypothesis for VAD researchers is that infrasound accelerates pericardial thickening [34], which, in the absence of any inflammatory process, is a specific sign of LFN exposure [42]. Thickening of cardiovascular structures is a whole-body response to LFN exposure, seen in autopsy [43], through echo-imaging [34], [35], [44] and in LFN-exposed rodents [45]. Thus, measuring noise while ignoring infrasound is an useless procedure if the goal is scientific research. However, since legislation does not require that a spectrum analysis be performed, the vast majority of biomedical studies only report their acoustic environment in terms of a dB-level measurement. Hence, parallel, but non-comparable studies are produced, as is seen below.

In several studies out of the University of Pisa, Italy, investigations regarding noise stress have been conducted over the past years [45]-[50]. Noise is described merely in terms of a dBA-level measurement, but the morphological responses that were observed through ultrastructural studies, such as cellular re-organizations and mitochondrial alterations, are similar to those observed within the context of VAD and LFN-induced pathology [33], [51]-[53]. However, no truly valid comparison can be made because no frequency spectra are provided. Many other such situations can be referenced [54]-[60] and have been discussed in more detail in [5].

When studies involve aircraft/airport noise, and the predominance of low frequency components can be safely assumed, symptoms seen in VAD are frequently identified. The impact of military aircraft noise exposure on the health of individuals, living around the Kadena and Futenma military US airfields in Japan, was investigated by Miyakita et al. [61]. People were given the Todai Health Index questionnaire, and were stratified into 5 noise-level groups in accordance with Ldn (averaged day-night dB level): 55, 55-60, 60-65, 65-70 dB (no frequency spectra). Significant dose-response relationships were found for vague complaints (dullness or heaviness in the legs, desire to lie down, head feels heavy or dull, headaches, stiffness or pain in the shoulders, pains in the various parts of the body, feeling flushed or feverish), respiratory (cough up phlegm, sneeze, have a runny nose, cough, have mucous in the throat, irritation or pain in the throat), digestive (stomach problems, stomach pain, discomfort in the stomach, diarrhea, indigestion), mental instability (worry about small things, feel uneasy when work is observed by others, nervous and shaky, tremble or feel weak, worry about the past, cold sweats, become mentally tired, mania), depression and nervousness. The authors concluded that residents around Kadena airfield suffer "both physical and mental effects due to the exposure of military aircraft noise and the extent of such responses increases with the level of noise exposure." Many of the symptomatic complaints referred to in this, and other [7], [8], [11], [12], studies are part of the VAD clinical picture [26], and given the type of noise exposure (despite the lack of frequency distribution analysis), they seem to fit into the framework of LFN-induced pathology.

Clearly, the lack of frequency distribution analysis in the overwhelming majority of biomedical studies has hindered LFN-induced pathology researchers in their search for replication or confirmation of their work.

B. Annoyance

Annoyance is a subjective parameter that is *felt* by persons exposed to noise. In order to better assess the effects of noise on people, the evaluation of the level of annoyance has been common to many studies.

To investigate community response to noise, annoyance levels were compared among individuals living in Gothenburg, Sweden, and Sapporo and Kumamoto in Japan. Noise exposure was defined as 46.2-73.6 dB in Gothenburg, 49.3-73.7 dB in Kumamoto, and 53.3-73.6 dB in Sapporo (L_{Aeq}(24) were also provided, frequency spectra were not), and annoyance levels were classified in 5 levels, from "not noticed" to "very annoyed" [62]. The type of housing was also taken into account (detached housing or apartment), and in Gothenburg, the degree of sound insulation was higher than in the Japanese houses. However, sound insulation of houses did not have a significant effect of annoyance responses. This would be a strong indication that annoyance is associated with LFN. Sound insulation in homes is designed to block out sounds that interfere with speech and sleep, i.e., audible sounds. LFN is not blocked out by the standard acoustic insulation devices. Walker et al. [63] showed that noise structurally radiated by railways was predominantly in the 20-200 Hz range, and that annoyance was specifically related to LFN. Persson-Waye et al. [64] showed that annoyance levels do, indeed, seem to be closely related to the predominance of LFN, and dBA levels did not predict annoyance.

In VAD, one of the most telling complaints is "I hear too much" and "I can't stand any type of noise, even television or music" [26]. Unlike the noise-exposed individuals who suffer hearing loss, VAD patients usually have minor audiometric losses within the 1000-5000 Hz range, and large losses at the 250-500 Hz notches. In rats exposed to LFN, the cochlear ciliated cells are seen to fuse with the upper tectonic membrane [51]. This is in stark contrast with the control rats, who lost cochlear cilia with the normal aging process. Cilia are supposed to move freely as the basilar membrane vibrates with the transduction of an acoustic pressure wave. If these ciliated cells are, instead, fused with the upper membrane, their movement will most probably cause discomfort, and even pain. The working hypothesis has been that individuals who complain about being annoyed by noise have probably already been excessively exposed to LFN.

Underground railway noise in dwellings, and consequent resident complaints, was the object of study by Vadillo et al. [65]. Dwellings were divided into 3 groups: I-exposed to high levels – ground floors in one-family houses; II-intermediate levels – first floors in multi-storied buildings; III-moderate levels – 2nd or 3rd floors in multi-storied buildings. LFN spectra were obtained, and revealed an absolute peak at 40 Hz which in Group I reached 66-70 dB(Lin) (sound pressure level is measured linearly, with no filter); in Group II, 55-66 dB(Lin); and in Group III, 52-61 dB(Lin). In each case there was virtually no acoustic phenomena present at frequencies above 300 Hz. The authors found that residents exposed to levels below 32 dBA were not bothered and did not complain, although they sometimes felt the passage of the train. When maximum levels were between 32-42 dBA, resident answers were not consistent. Above 42 dBA, all residents complained strongly about noise and vibration. Here, the dBA-level measurement seemed to be a good predictor for annoyance.

If people are not annoyed, this does not necessarily mean that they are not being exposed to an agent of disease. The simplest argument is that agents of disease need not be perceived to be noxious, take radiation, for example. Clearly a new attitude toward noise is urgently needed [66].

C. *LFN Ignored*

The erroneous assumption that acoustic phenomena impinges only on, or via, the auditory system is still deeply embedded in researchers minds. The concept that acoustic phenomena can directly impinge on, for example, the respiratory tract [51], [67] is still not integrated into many research designs. One single histological type of tumor has been found in VAD patients with malignancies of the respiratory tract: squamous cell carcinoma [68]. Knowing this, what is the validity of studying lung cancer without performing the breakdown of tumor types, especially among LFN-exposed workers [69]?

Space exploration is another situation where LFN is being ignored [70]. In Space, noise exposure is continuous, even during resting periods. All life-support equipment generates LFN to some extent, but there is no acoustic propagation into outer space. Thus, acoustic phenomena is contained within the living spaces, and is absorbed by surrounding structures, including human tissue. By insisting that acoustic phenomena only impinges upon humans through the auditory system, is apparently sufficient justification not to perform a frequency spectrum analysis [70]. Shipworkers are in a similar situation in that their exposure is continuous. However, many shipworkers are not protected by any type of noise legislation. British Seafarers, for example, including Engine Room Staff, are not protected by any noise-related legislation, do not have mandatory audiometric evaluations, and no limit for noise levels has been established for these workers [71]. Most young job applicants for noisy jobs have already had substantial and extensive exposure to LFN. This creates a concern for their mid-term job performance, and long-term health effects. Screening programs are not yet in place to protect the young LFN-workers who will, most probably, develop LFN-induced pathology at a much faster rate than older generation workers.

LFN as a confounding factor has also been neglected. Knowing that increased irritability and aggressiveness, accompanied by a cognitive deterioration, are initial signs of LFN-induced pathology, confinement studies, for example, must control for possible LFN that may be present [72]. Similarly, in many studies involving human populations, screening for previous LFN exposure histories could rule out LFN as a confounding factor. With animal studies, laboratories are often located in basements where ventilation and refrigeration systems are significant sources of LFN. This is a parameter that must be controlled when physiological studies are being conducted on such animals.

V. Conclusion

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 low 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.
4 – LFN should be considered as a possible confounding factor in biomedical studies, especially those involving human populations and laboratory animals.

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References

[1] W. D. Ward, “Noise-induced hearing damage,” in *Otolaryngology*, M. M. Paparella, D. A. Shumrick, Eds., Philadelphia: W. B. Saunders Co., 1973.

[2] R. W. Cantrell, “Effects of noise exposure”, in: *AGARD Conference Proceedings No. 171 Effects of long duration noise exposure on hearing and health*. AGARD C-10-1: France, 1975.

[3] Occupational Safety and Health Administration 3074. *Hearing Conservation*, Washington DC: US Dept. Labor, 1995.

[4] E. H. Berger, W. D. Ward, J. C. Morrill and L.H. Royster, Eds. *Noise and hearing conservation Manual*. 4th ed. Fairfax, Va, USA: American Industrial Hygiene Association, 1995.

[5] M. Alves-Pereira, “Extra-aural noise-induced pathology. A review and commentary,” *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A7-A21, Mar. 1999.

[6] D. A. Laird, “Experiments on the physiological cost of noise,” *J. Natl. Inst. Indust. Psychology*, vol. 4, pp. 251-258, 1928.

[7] A. Cohen, “The influence of a company hearing conservation program on extra-auditory problems in workers,” *J. Safety Res.*, vol. 8, pp. 146-162, 1976.

[8] E. E. Dart, “Effects of high speed vibrating tools on operators engaged in airplane industry,” *Occ. Med.*, vol. 1, pp. 515-550, 1946.

[9] A. R. Moller, “Occupational noise as a health hazard - physiological viewpoints,” *Scand. J. Work Environ.*, vol. 3, pp. 73-99, 1977.

[10] K. Millar and M. J. Steels, “Sustained peripheral vasoconstriction while working in continuous intense noise,” *Aviat. Space Environ. Med.*, vol. 61, pp. 695-698, 1990.

[11] G. I. Rumancev, "[Investigations concerning the hygienic evaluation of vibration in factories producing reinforced concrete]," *Gig. Tr. Prof. Zabol.*, vol. 5, pp. 6-12, 1961.

[12] V. E. Ostapkovich, "Noise pathology in otorhinolaryngology," *Vestn. Otorinolaringol.*, vol. 3, pp. 6-10, 1991.

[13] F. Tomei, B. Papaleo, T. P. Baccolo, E. Tomao, P. Alfi and S. Fantini, "[Chronic noise exposure and the cardiovascular system in aircraft pilots.] Esposizione cronica a rumore e apparato cardiovascolare in piloti di aeromobili," *Med. Lav.*, vol. 87, pp. 394-410, 1996.

[14] H. Ising, "Extraaural effects of chronic noise exposure in animals - A review," *Schrift Ver. Wasser Boden Luft Hyg.*, vol. 88, pp. 48-64, 1993.

[15] N. F. Izmerov, G. A. Suvorov, N. A. Kuralesin and V. G. Ovakimov, "[Infrasound: body's effects and hygienic regulation]," *Vestn. Ross. Akad. Med. Nauk.*, vol. 7, pp. 39-46, 1997.

[16] P. Knipschild, "Medical effects of aircraft noise: review and literature," *Int. Arch. Occup. Environ. H*ealth, vol. 40, pp. 201-204, 1977.

[17] N. V. Grechkovskaia and I. A. Parpalei, "[The impact of the working conditions on morbidity in workers in jobs hazardous for vibration and noise in aviation enterprises]," *Lik. Sprava.*, vol. 5, pp. 20-23, 1997.

[18] C. Giordano, S. Conticello, F. Beatrice, A. Montemagno and R. Boggero, "Non-auditory effects of environmental noise: a study of metallurgical and mechanical workers," *Acta Otorhinolaryngol. Ital.*, vol. 21(5), pp. 281-6, 2001.

[19] J. M. Last and R.D. Wallace, Eds. *Public health and preventive medicine*, 13th ed. Norwalk CT: Appleton & Langen, 1992, pp. 529-31.

[20] B. A. Plog, J. Niland and P. J. Quinlan, Eds. *Fundamentals of industrial hygiene*. Itasca, IL: National Safety Council, 1996.

[21] Occupational Safety and Health Administration Technical Manual, "Section II - Chapter 5, Noise Measurement. Directive Number: TED 1.15 CH-1," Washington DC : US Dept Labor, 1996.

[22] R. A. Corbitt. *Standard handbook of environmental engineering*, 2nd edition, New York : McGraw-Hill Handbooks, 1998, pp.4.96.

[23] H. von Gierke and S. Mohler, "Letter to the editor: 'vibroacoustic disease'," *Aviat. Space Environ. Med.*, vol. 73 (8), pp. 828-830, Aug. 2002.

[24] GIMOGMA, "[Epilepsy of vascular etiology, a clinical picture of vibration disease?] Epilepsia sintomática de etiologia vascular, manifestação da síndrome das vibrações?," *Rev Port Med Mil*, vol. 32, pp. 5-9, 1984, in Portuguese.

[25] GIMOGMA, "[Evoked potential study in a population exposed to occupational vibration.] Estudo dos potenciais evocados em populações sujeitas a vibrações ocupacionais," *Rev Port Med Mil*, vol. 32, pp. 10-16, 1984, in Portuguese.

[26] N. A. A. Castelo Branco, "The clinical stages of vibroacoustic disease," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A32-A39, Mar. 1999.

[27] N. A. A. Castelo Branco, E. Rodriguez Lopez, M. Alves-Pereira and D. R. Jones, "Vibroacoustic disease: some forensic aspects," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A145-A151, Mar. 1999.

[28] N. A. A. Castelo Branco and E. Rodriguez Lopez, "The vibroacoustic disease – An emerging pathology," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A1-A6, Mar. 1999.

[29] M. J. Silva, A. Carothers, N. A. A. Castelo Branco, A. Dias and M. G. Boavida, "Increased levels of sister chromatid exchanges in military aircraft pilots", *Mutation Res*; vol. 44(1), pp. 129-134, 1999.

[30] M. J. Silva, A. Carothers A, N. A. A. Castelo Branco, A. Dias and M. G. Boavida, "Sister chromatid exchanges workers exposed to noise and vibration," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A40-A45, Mar. 1999.

[31] M. J. Silva, A. Dias, A. Barreta, P. J. Nogueira, N. A. A. Castelo Branco and M. G. Boavida, "Low frequency noise and whole-body vibration cause increased levels of sister chromatid exchange in splenocytes of exposed mice," *Teratog Carcinog Mutagen*, vol. 22(3), pp. 195-203, 2002.

[32] M. G. Boavida, A. Dias, N. A. A. Castelo Branco and M. J. Silva, "Analysis of sister chromatid exchanges in splenocytes of mice exposed to noise and vibration," *Aviat. Space Environ. Med.*, vol. 70(4), pp. 376, Apr. 1999.

[33] N. A. A. Castelo Branco, E. Monteiro, M. Alves-Pereira, A. P. Águas, A. Sousa Pereira and N. R. Grande, "Morphological changes in the pericardia of military helicopter pilots," *Proc. Microscopy Barcelona 2001*, 2001, pp. 318-319.

[34] A. Araujo, F. Pais, J. M. C. Lopo Tuna, M. Alves-Pereira and N. A. A. Castelo Branco, "Echocardiography in noise-exposed flight crew," *Proc. Internoise 2001*, 2001, pp. 1007-1010.

[35] R. Torres, G. Tirado, A. Roman, R. Ramirez, H. Colon, A. Araujo, F. Pais, W. Marciniak, J. Nóbrega, A. Bordalo e Sá, J. M. C. Lopo Tuna, M. S. N. Castelo Branco, M. Alves-Pereira and N. A. A. Castelo Branco, "Vibroacoustic disease induced by long-term exposure to sonic booms," *Proc. Internoise 2001*, 2001, pp. 1095-1098.

[36] M. Alves-Pereira, M. M. N. Castelo Branco, J. Motylewski, A. Pedrosa and N. A. A. Castelo Branco, "Airflow-induced infrasound in commercial aircraft," *Proc. Internoise 2001*, 2001, pp. 1011-14.

[37] A. S. Nekhoroshev and V. V. Glinchikov, "Morphological research on the liver structures of experimental animals under the action of infrasound," *Aviakosm. Ekolog. Med.*, vol. 26, pp. 56-59, 1992.

[38] A. S. Nekhoroshev and V. V. Glinchikov, "Effect of infrasound on morphofunctional changes in myocardium exposed to infrasound," *Gig. Sanit.*, vol. 12, pp. 56-58, 1991.

[39] V. I. Svigovyi and V. V. Glinchikov, "The effect of infrasound on lung structure," *Gig Truda Prof* Zabol, vol. 1, pp. 34-7, 1987.

[40] J. N. Cole, G. C. Mohr, E. G. Guild and H. E. von Gierke, "The effects of low frequency noise on man as related to the Apollo Space Program," AMRL Memorandum B-66, 1966.

[41] G. C. Mohr, J. N. Cole, E. Guild, H. E. von Gierke "Effects of low-frequency and infrasonic noise on man," *Aerosp. Med.*, vol. 36, pp. 817-824, 1965.

[42] B. D. Holt, "The pericardium," in *Hurst's The Heart*. 10th ed., V. Furster, R. Wayne Alexander and F. Alexander, Eds. New York: McGraw-Hill Professional Publishing, 2000, pp. 2061-82.

[43] N. A. A. Castelo Branco, "A unique case of vibroacoustic disease. A tribute to an extraordinary patient," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A27-A31, Mar. 1999.

[44] W. Marciniak, E. Rodriguez, K. Olsowska, I. Botvin, A. Araujo, F. Pais, C. Soares Ribeiro, A. Bordalo, J. Loureiro, E. Prazeres de Sá, D. Ferreira, M. S. N. Castelo Branco and N. A. A. Castelo Branco, "Echocardiography in 485 aeronautical workers exposed to different noise environments," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A46-A53, Mar. 1999.

[45] J. Martins dos Santos, N. R. Grande, N. A. A. Castelo Branco, C. Zagalo and P. Oliveira, "Vascular lesions and vibroacosutic disease," *Eur. J. Anat.*, vol. 6(1), pp. 17-21, 2002.

[46] M. Gesi, P. Lenzi, M. G. Alessandri, M. Ferrucci, F. Fornaia and A. Paparelli, "Brief and repeated noise exposure produces different morphological and biochemical effects in noradrenaline and adrenaline cells of adrenal medulla," *J. Anat.*, vol. 200(2), pp. 159-168, 2002.

[47] M. Gesi, F. Fornai, P. Lenzi, G. Natale, P. Soldani and A. Paparelli, "Time-dependent changes in adrenal cortex ultrastructure and corticosterone levels after noise exposure in male rats," *Eur. J. Morphology*, vol. 39(3), pp. 129-135, 2001.

[48] M. Gesi, A. Riva, P. Soldani, F. Fornai, G. Natale, P. Lenzi, A. Pellegrini and A. Paparelli, "Central and peripheral benzodiazepine ligans prevent mitochondrial damage induced by noise exposure in the rat myocardium: an ultrastructural study," *Anat. Rec.*, vol. 255(3), pp. 334-341, 1999.

[49] F. Salvetti, B. Chelli, M. Gesi, A. Pellegrini, G. Giannaccini, A. Lucacchini and C. Martini, "The effect of noise exposure on rat cardiac peripheral benzodiazepine receptors," *Life Sciences*, vol. 66(13), pp. 1165-1175, 2000.

[50] P. Soldani, A. Pellegrini, M. Gesi, P. Lenzi, R. Cristofani and A. Paparelli, "SEM/TEM investigations of rat cardiac subcellular alterations induced by changing duration of noise stress," *Anat. Rec.*, vol. 248(4), pp. 521-532, 1997.

[51] N. A. A. Castelo Branco, M. Alves-Pereira, J. Martins dos Santos and E. Monteiro, "SEM and TEM study of rat respiratory epithelia exposed to low frequency noise," in *Science and Technology Education in Microscopy: An Overview*, A. Mendez-Vilas, Ed. Badajoz, Spain, Formatex, 2002, to be published.

[52] N. A. A. Castelo Branco, A. P. Águas, A. Sousa Pereira, E. Monteiro, J. I. G. Fragata and N. R. Grande, "The pericardium in noise-exposed individuals," *Proc. Internoise 2001*, 2001, pp. 1003-1006.

[53] N. A. A. Castelo Branco, A. P. Águas, A. Sousa Pereira, E. Monteiro, J. I. G. Fragata, F. Tavares and N. R. Grande, "The human pericardium in

vibroacoustic disease," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A54-A62, Mar. 1999.

[54] J. Zenk, D. Scheffler, P. Scheffler, W. Delb and H. Iro, "The influence of noise on blood flow in the basilar artery – measurements with transcranial color-coded duplex sonography," *Hearing Res.*, vol. 140(1-2), pp. 157-64, 2000.

[55] M. I. Weintraub, "Vestibulopathy induced by high-impact aerobics – a nre syndrome – discussion of 30 cases," *J. Sports Med. Physical Fitness*, vol. 34(1), pp. 56-63, 1994.

[56] W. Babisch, H. Fromme, A. Beyer and H. Ising, "Increased catecholamine levels in urine in subjects exposed to road traffic noise – the role of stress hormones in noise research," *Envrion. Intern.*, vol. 26(7-8), pp. 475-481, 2001.

[57] W. S. Quirk, J. K. M. Coleman, J. M. Hanesworth, J. W. Harding and J. W. Wright, "Noise-induced elevations of plama membrane endothelin (ET-3)," *Hearing Res.*, vol. 80(1), pp. 119-122, 1994.

[58] N. A. Kumar, D. C. Mathangi and A. Namasivayam, "Noise-induced changes in fress radical scavenging enzymes in the blood and brain of albino rats," *Medical Sci. Res.*, vol. 26(12), pp. 811-812, 1998.

[59] F. Mocci, P. Cannalis, P. A. Tomasi, F. Casu, S. Pettinato, "The effect of noise on serum and urinary magnesium and catecholamines in humans," *Occ. Med. Oxford*, vol. 51(1), pp. 56-61, 2001.

[60] M. T. M. van Raaij, C. J. G. Dobbe, B. Elvers, A. Timmerman, E. Schenk, M. Oortgiesen and V. M. Wiegant, "Hormonal status and the neuroendocrine response to a novel heterotypic stressor involving subchronic noise exposure," *Neuroendocrinology*, vol. 65(3), pp. 200-209, 1997.

[61] T. Miyakita, T. Matsui, A. Ito, T. Tokuyama, K. Hiramatsu, Y. Osada and Y. Yamamoto, "Population-based questionnaire survey on health effects of aircraft noise on residents living around U.S. airfields in the Ryikyus – Part I: and analysis of 12 scale scores," *J. Sound Vib.*, vol. 250(1), pp. 129-137, 2002.

[62] T. Sato, T. Yano, M. Bjorkman and R. Rylander, "Comparison of community response to road traffic noise in Japan and Sweden – Part I: Outline of surveys and dore-response relationships," *J. Sound Vib.*, vol. 350(1), pp. 161-7, 2002.

[63] J. G. Walker and M. F. K. Chan. "Human response to structurally radiated noise dus to underground railway operations," *J. Sound Vib.*, vol. 193(1), pp. 49-63, 1996.

[64] K. Persson-Waye and R. Rylander, "The prevalence of annoyance and effects after long-term exposure to low frequency noise," *J Sound* Vib, vol. 240(3), pp. 483-97, 2001.

[65] E. G. Vadillo, J. Herreros and J. G. Walker, "Subjective reaction to structurally radiated sound from underground railways: field results," *J. Sound Vib.* 1996; 193(1): 65-74.

[66] M. Alves-Pereira and N. A. A. Castelo Branco, "Vibroacoustic Disease: The need for a new attitude towards noise, " *Proc Inter Conf Public Participation & Information Technologies*, Lisbon, Portugal, 1999, pp. 340-7. Available: http://www.citidep.pt/papers/articles/alvesper.htm.

[67] J. M. Reis Ferreira, A. R. Couto, N. Jalles-Tavares, M. S. N. Castelo Branco and N. A. A. Castelo Branco, "Airflow limitations in patients with vibroacoustic disease," *Aviat. Space Environ. Med.*, vol. 70 (3, Suppl), pp. A63-A69, Mar. 1999.

[68] N. A. A. Castelo Branco. The respiratory system as a target of low frequency noise. Reports on human and animal models. *Proc. 8th Intern Cong Sound & Vibration*, 2001, pp. 1501-1508.

[69] E. Pukkala, R. Aspholm, A. Auvinen, H. Eliasch, M. Gundestrup, T. Haldorsen, N. Hammar, J. Hrafnkelsson, P. Kyyronen, A. Linnersjo, V. Rafnsson, H. Storm and U. Tveten, "Cancer incidence among nordic airline pilots," *Aviat. Space Environ. Med.*, vol. 72 (3), pp. 292, 2001

[70] J. C. Buckey, F. E. Musiek, R. Kline-Schoder, J. C. Clark, S. Hart and J. Havelka, "Hearing loss in space," *Aviat. Space Environ. Med.*, vol. 72(12), pp. 1121-1124, 2001.

[71] J. W. Arnot, Personal communication. (email: j.w.arnot@ukgateway.net).

[72] N. O. Kraft, H. Oshima, N. Inoue, K. Mizuno, T. Murai and C. Sekiguchi, "Physiological parameters and accentuation of personal traits during confinement in an isolated environment," *Aviat. Space Environ. Med.*, vol. 70 (4), pp. 412, 2001.

Table I. Description of the overall acoustic amplitude at each of the locations, in both dBA and dBLin.

Location	dB-Level (dBA)	dB-Level (dBLin)
Cockpit	72.1	83.2
Kitchen Restaurant	71.6	80.1
Train (in station)	71.4	92.0
Car @ 120Km/h	71.2	100.8
Trolley Car	72.7	97.1
Textile Factory	97.2	98.2
Dance Club	95.1	114.7
Boom Car (Stopped)	96.6	124.4

Table II. Comparison of the dB-levels in the A340 Cockpit and all other locations. Peak frequencies refers to the 1/3 octave bands where the acoustical energy was highly concentrated.

Locations (Cockpit vs. ...)	Difference in dBA	Difference in dBLin	Peak Frequency (Hz)
Restaurant	0.5	3.1	12.5, 125 - 200
Train	0.7	8.8	25 – 125
Car	0.9	17.6	6.3 - 31.5
Trolley	0.6	13.9	6.3 – 100
Textile	25.1	15	80, 400
Dance Club	23	31.5	40 – 80
Boom Car	24.5	41.2	40 – 100

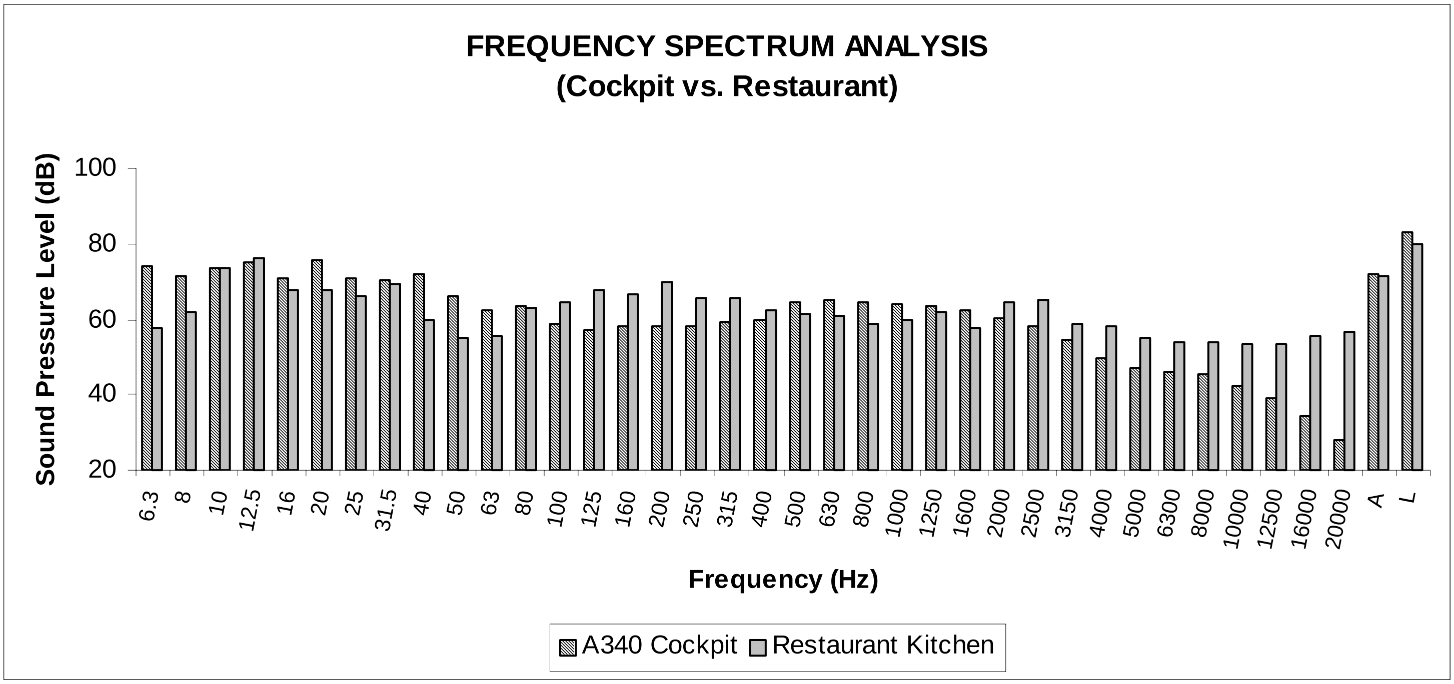

Fig. 1. Comparison of the frequency distributions in the Airbus 340 cockpit and in the restaurant kitchen. Even though the dBA levels (A) are comparable (72.1 vs. 71.6), dBLin levels (L) differ (83.2 vs. 80.1) due to the substantial differences in dB level at the 6.3 Hz, 8 Hz, 40 Hz, and 125-315 Hz.

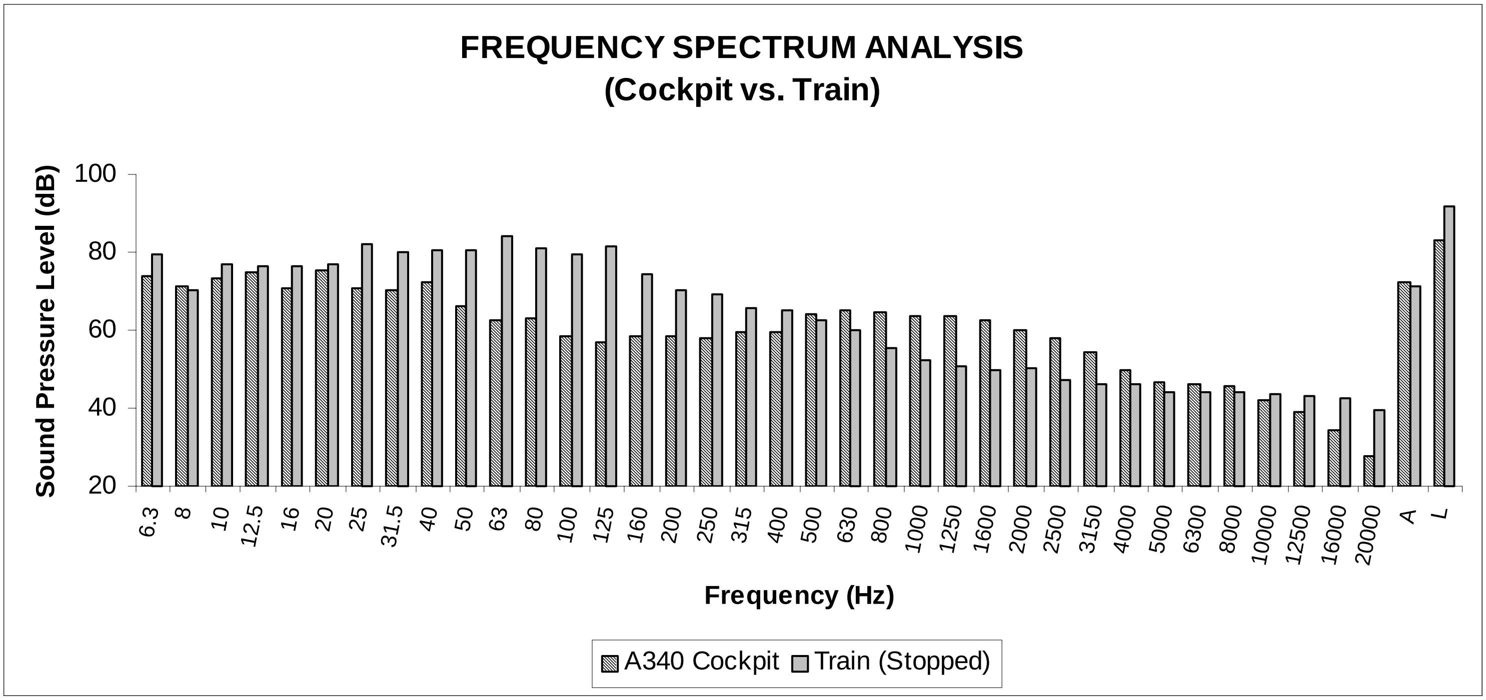

Fig. 2. Comparison of the frequency distributions in the Airbus 340 cockpit and in a stopped, electric, commuter train. Even though the dBA levels (A) are comparable (72.1 vs. 71.4), dBLin levels (L) differ (83.2 vs. 92.0) due to the substantial differences in dB level at the 25-250 Hz bands.

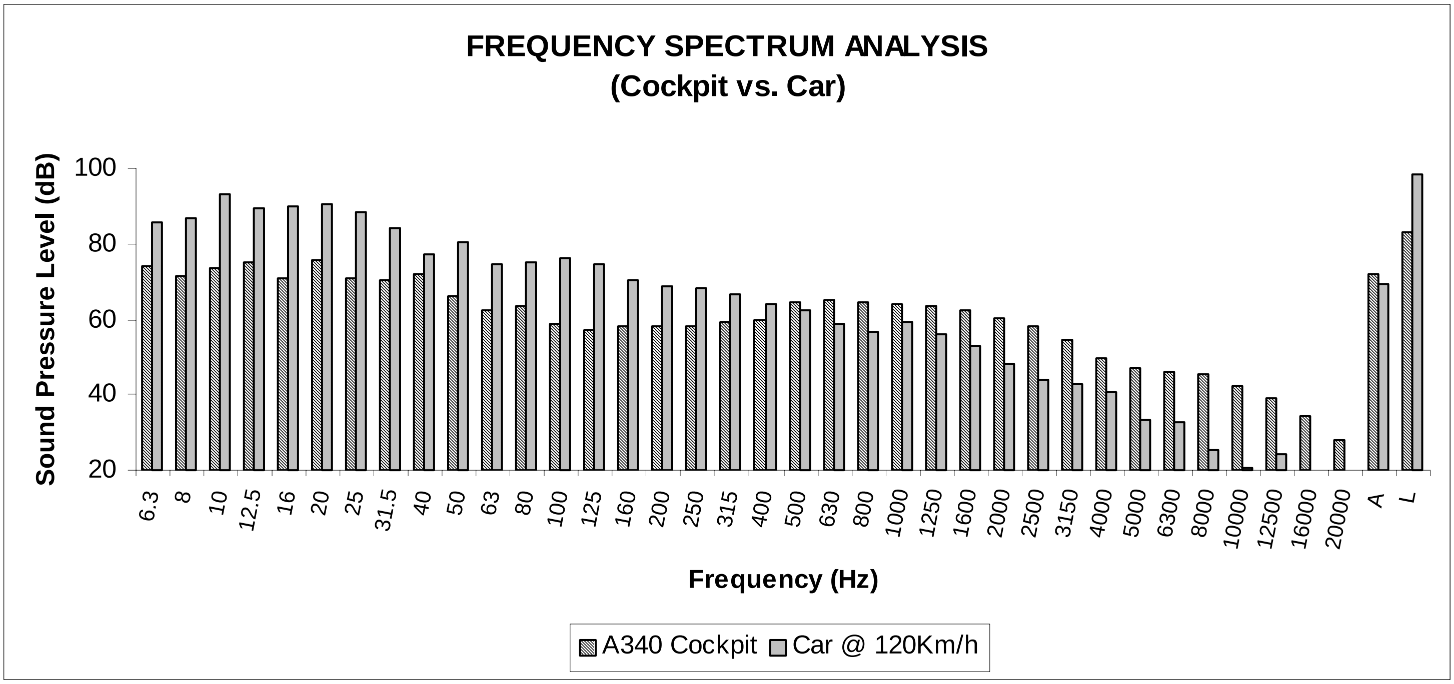

Fig. 3. Comparison of the frequency distributions in the Airbus 340 cockpit and in a car, alone on a highway (at 3 a.m.), travelling at 120 Km/h, with radio off and windows closed. Even though the dBA levels (A) are comparable (72.1 vs. 71.2), dBLin levels (L) differ (83.2 vs. 100.8) due to the substantial differences in dB level at the 6.3-315 Hz bands.

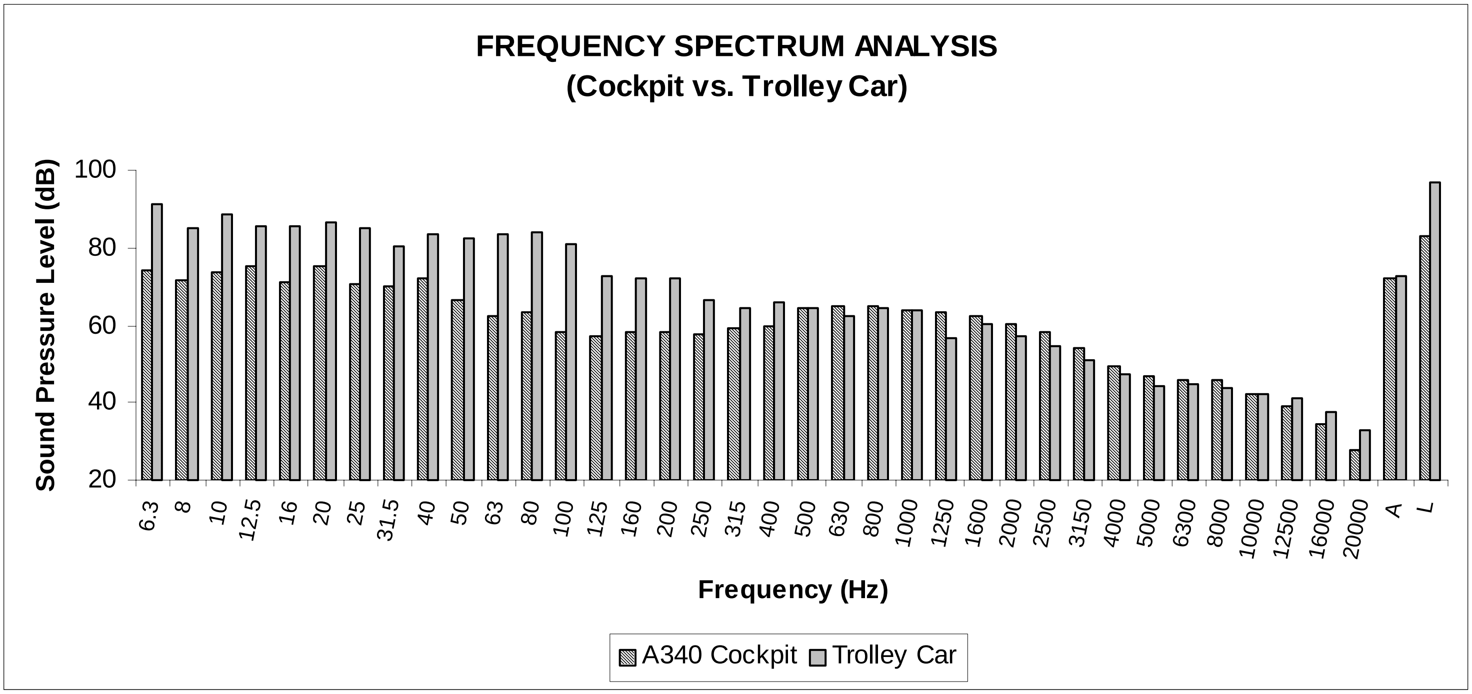

Fig. 4. Comparison of the frequency distributions in the Airbus 340 cockpit and in a modern trolley car, travelling in Lisbon, around midday. Even though the dBA levels (A) are comparable (72.1 vs. 72.7), dBLin levels (L) differ (83.2 vs. 97.1) due to the substantial differences in dB level at the 6.3-200 Hz bands.

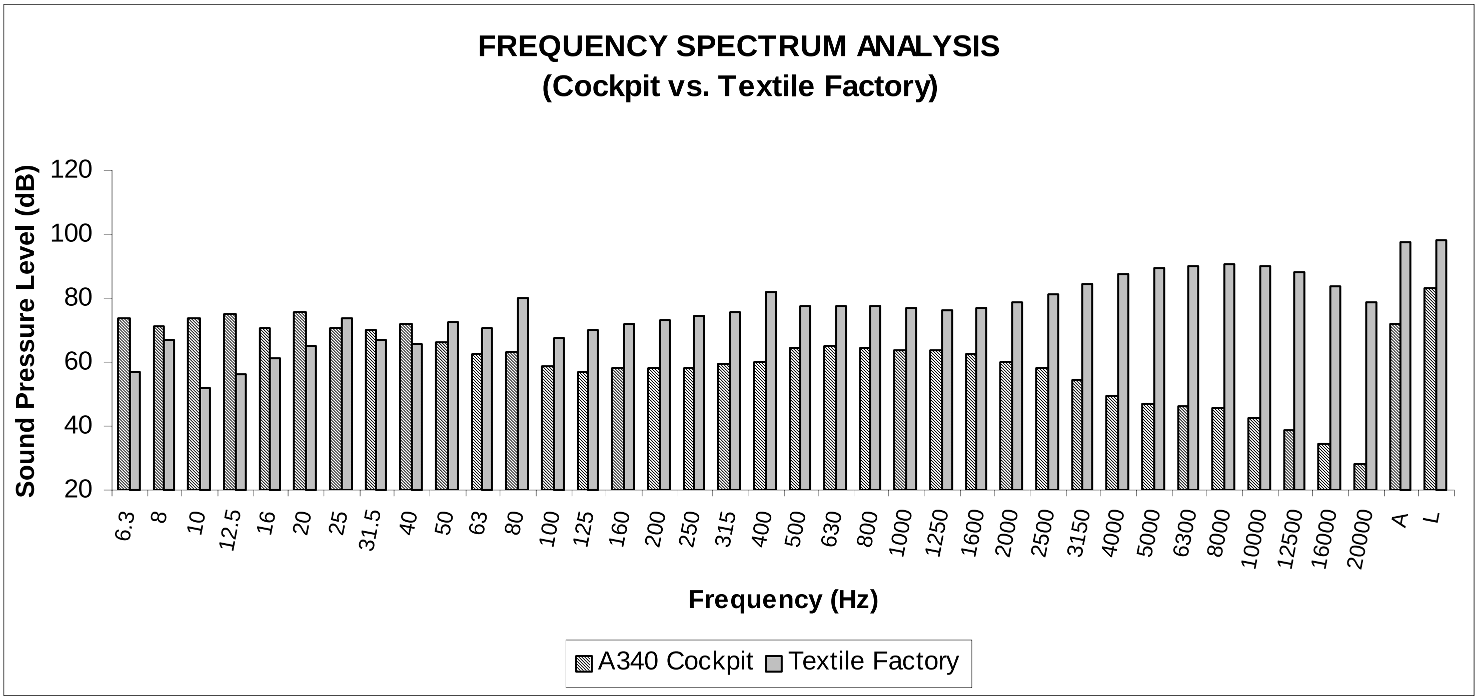

Fig. 5. Comparison of the frequency distributions in the Airbus 340 cockpit and in a working hall of a textile factory. dBA levels (A) in the textile factory are higher than in the cockpit (72.1 vs. 95.1), and the dBLin levels (L) differ greatly (83.2 vs. 98.2) due to the substantial differences in dB level in the bands >80 Hz.

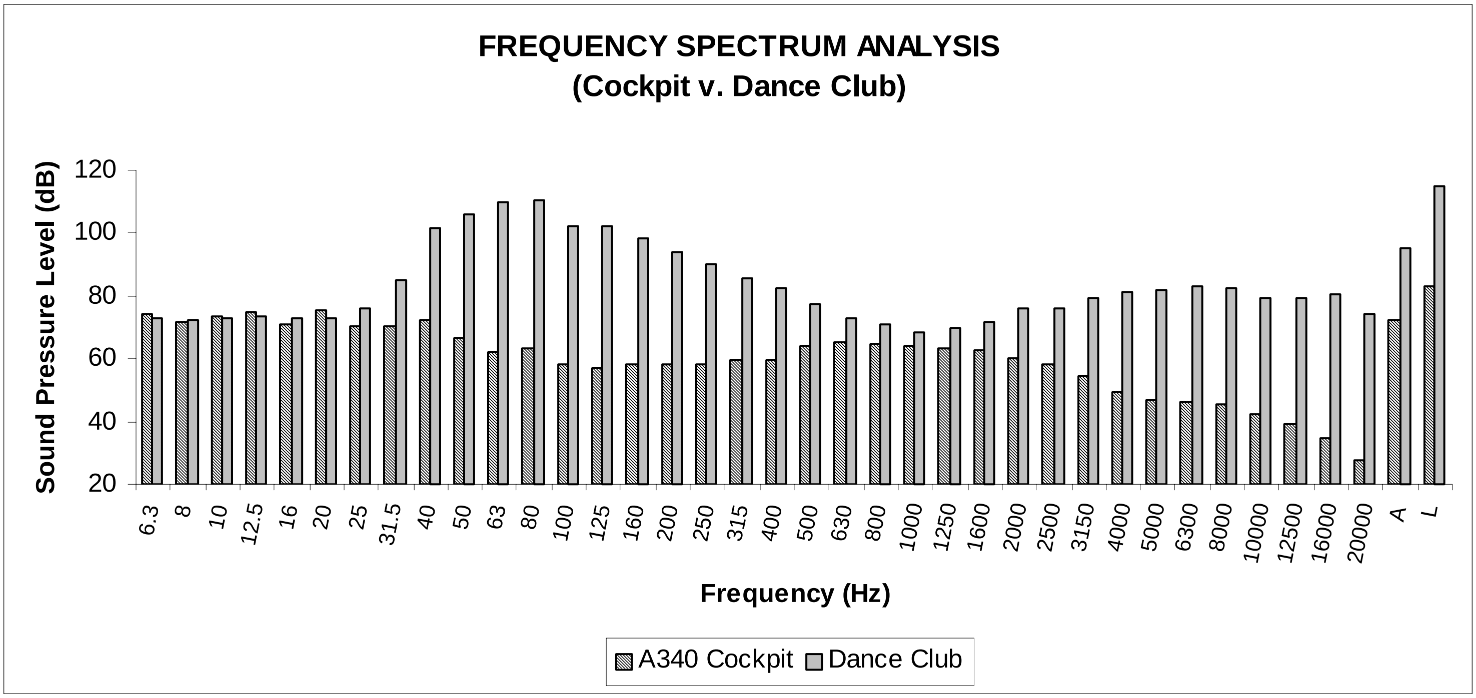

Fig. 6. Comparison of the frequency distributions in the Airbus 340 cockpit and in a dance club (techno music). dBA levels (A) in the dance club are much higher than in the cockpit (72.1 vs. 95.1), and the dBLin levels (L) differ greatly (83.2 vs. 114.7) due to the substantial differences in dB level at the 31.5-500 Hz bands.

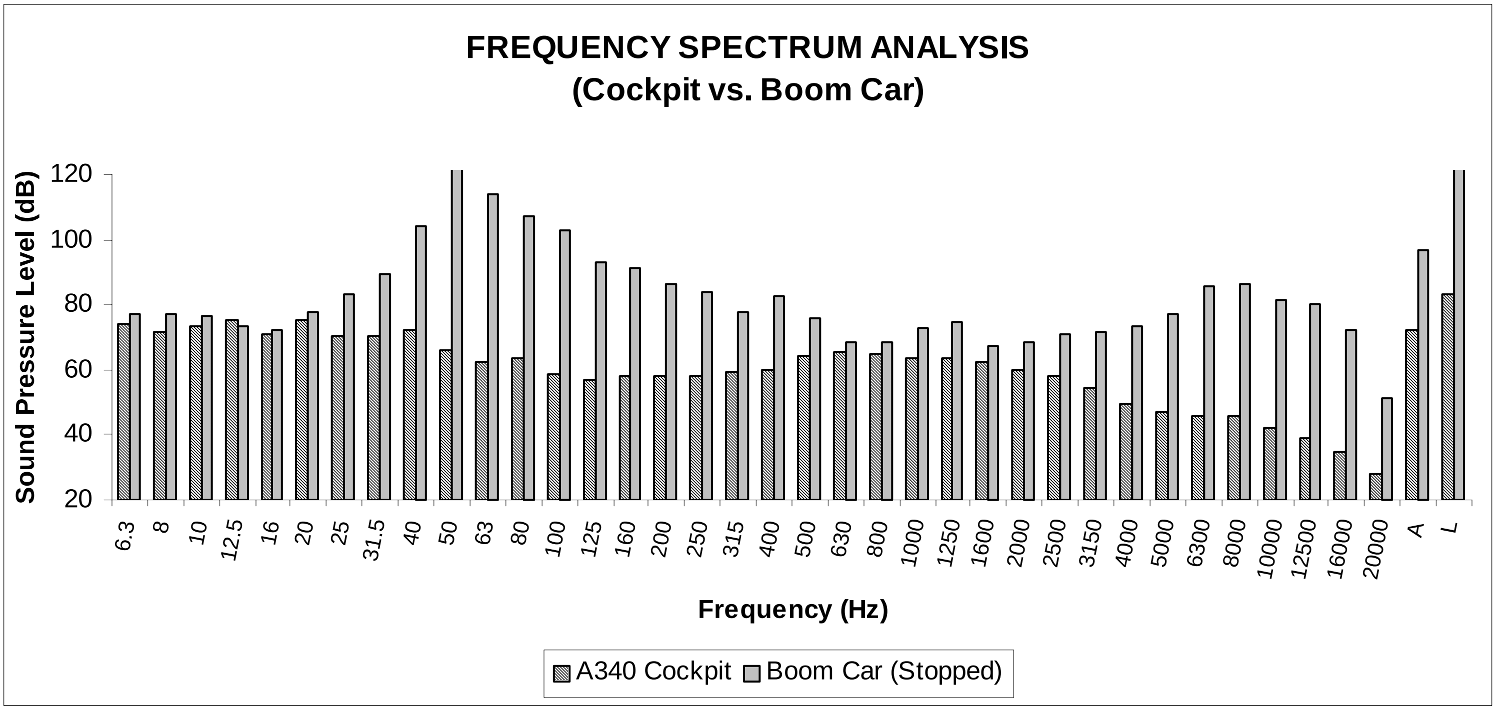

Fig. 7. Comparison of the frequency distributions in the Airbus 340 cockpit and in stopped boom car playing techno music. dBA levels (A) are much higher than in the cockpit (72.1 vs. 96.6), and the dBLin levels (L) differ greatly (83.2 vs. 124.4) due to the substantial differences in dB level at the 25-500 Hz bands.

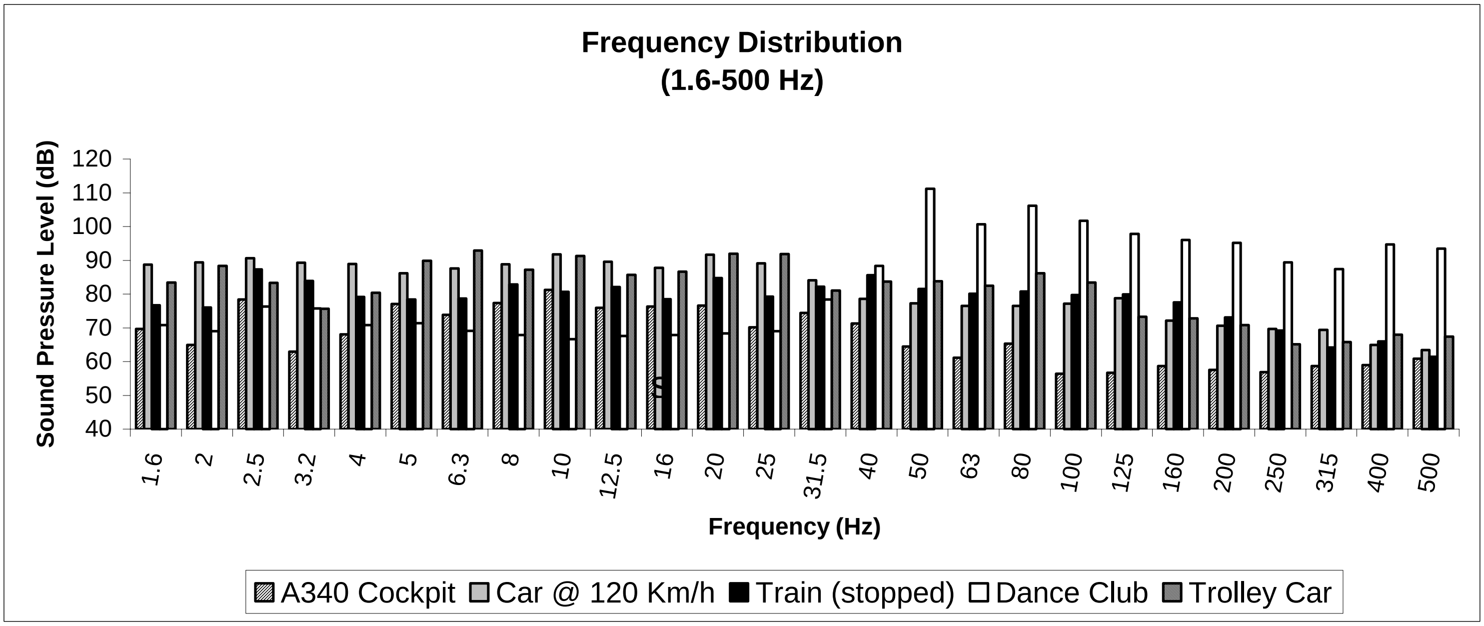

Fig. 8. Comparison of the frequency distribution within the 1.6-500 Hz (LFN) range among the cockpit, the car travelling on highway, the train stopped at the station, the dance club playing techno music, and the trolley car travelling in Lisbon at midday. The cockpit seems to have an overall less amount of LFN than all other locations, while the dance club is the location with the highest peaks of LFN.