ARTIGO ORIGINAL/ORIGINAL ARTICLE

O ruído e o aparelho respiratório

Noise and the respiratory system

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RESUMO

Apesar do importante acervo de evidência científica já existente, a patologia respiratória provocada pela exposição crónica a ruído de baixa frequência (RBF) continua por reconhecer. O objectivo deste trabalho de revisão consiste em:

ABSTRACT

Noise-induced pulmonary pathology is still an issue that is regarded with much suspicion despite the significant body of evidence demonstrating that acoustic phenomena target the respiratory tract. The goal of this review paper is threefold:

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a) descrever o fenómeno acústico como agente de doença, demonstrando a inépcia da legislação no que diz respeito à patologia extra-auditiva; b) esclarecer os motivos que conduziram ao interesse na patologia respiratória nos indivíduos expostos a RBF; e c) enaltecer outros estudos que denunciam o efeito deletério do ruído sobre o aparelho respiratório. Por último, apresentar e discutir as perspectivas futuras sobre os estudos dirigidos à caracterização dos efeitos do RBF e da necessidade de reproduzir estes efeitos em modelos animais.

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Palavras-chave: ruído de baixa frequência, derrame pleural, bronquite, fibrose pulmonar, audição, decibel, ocupacional, ambiental

a) to describe acoustic phenomena as an agent of disease, and the inadequacies of current legislation regarding noise-induced, non-auditory pathology; b) to trace how the interest in noise-induced pulmonary pathology emerged within the scope of studies on vibroacoustic disease; and c) to bring to light other studies denouncing noise as an agent of disease that impinges on the respiratory tract. As concluding remarks, future perspectives in LFN-related research will be discussed. The need for animal models will be emphasized.

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Key-words: low frequency noise, pleural effusion, bronchitis, pulmonary fibrosis, hearing, decibel, occupational, environmental

INTRODUCTION

Noise exposure and lung pathology is not an immediate association in the minds of most scientists and physicians. In fact, noise-induced pulmonary lesions may seem to be more a speculative idea rather than an actual occurrence. Nevertheless, the link between respiratory pathology and noise exposure has been hinted at since the 1960's, and today a consistent body of scientific knowledge has successfully been gathered, definitively linking noise-exposure with respiratory pathology.

The interdisciplinary field concerning the biological effects of acoustic phenomena is wide-ranging. Most acousticians do not have the biological background to grasp the fundamental biological features of respiratory pathology, and many physicians lack the physical and biomechanical backgrounds related to acoustic propagation at the interface with biological tissue.

Thus, the goal of this report is threefold: a) to describe acoustic phenomena as an agent of disease, and the inadequacies of current legislation regarding noise-induced, non-auditory pathology; b) to trace how the interest in noise-induced pulmonary pathology emerged within the scope of studies on vibroacoustic disease; and c) to bring to light other studies that denounce noise as an agent of disease which impinges on the respiratory tract.

ACOUSTIC PHENOMENA

Decibels, frequency and hearing

Sound is an acoustic phenomenon that is perceived by the human auditory system. When this sound is undesirable or unwanted, it is called noise. However, human hearing is not able to capture the entire spectrum of acoustic phenomena that exists in Nature, just as human vision is

unable to capture the entire electromagnetic spectrum (light, the only visible electromagnetic radiation, is within the 1-100 mm wavelength range). The auditory system only captures acoustic phenomena that occurs within the 20-20000 Hz frequency range. Below 20 Hz, acoustic events are inaudible, and are designated infrasound. Ultrasounds, in the megahertz range of the spectrum, and are also inaudible and are used, for example, in medical diagnostic procedures.

Physically, acoustic phenomena are airborne travelling pressure waves[1]. As wave events, they are only clearly identified if *both* amplitude and frequency are known. The decibel (dB) is the unit in which acoustic amplitude, or sound pressure level (SPL), is measured. The human auditory system does not have equal sensitivity throughout entire audible spectrum. It is more tuned to acoustic events that occur within the 500-10000 Hz range. For example, at an amplitude of 40dB, a tone at 1000 Hz will be clearly perceived, at 100 Hz it will be barely audible, and at 50 Hz it will go undetected. Legal deafness is defined as a >30dB loss at the 4000 Hz notch.

Noise has been associated with hearing impairment and annoyance since ancient times. In Ancient Greece, metalwork that involved hammers was banned within city limits in 600 BCE[2], while in 50 CE, Pliny the Elder noted that people who lived near the cataracts of the Nile River were hard of hearing[3]. Today, noise is related to hearing impairment and deafness, and acoustic phenomena are generally thought to only impinge on, or via, the auditory system. Appropriately, the vast majority of noise protection devices belong to the class of *hearing* protection devices.

Since the goal of noise protection is the avoidance of hearing impairment, it becomes necessary to be able to evaluate a noisy environment *as if it were being heard*. Only then is it possible to ascertain if protection against the risk of deafness is required. This is achieved through the use

of the A-weighting system, which measures the acoustic amplitude (in dBA) as if it were being perceived by the human auditory system, i.e., it de-emphasizes the acoustic energy that is concentrated in the lower frequency ranges (<500 Hz). "The A-weighting is an approximation of equal loudness perception characteristics of human hearing for pure tones relative to a reference of 40 dB SPL at 1 kHz"[4], i.e. it best simulates the characteristics of the human auditory system.

Thus, legislation has adopted the dBA value in establishing the permissible noise exposure levels, i.e., a measure of the overall amplitude of the acoustic energy present in an environment, where frequency distribution is not assessed, and frequencies ≤500 Hz are de-emphasized.

A real life example

Acoustic phenomena were evaluated at four different locations: 1) cockpit of an Airbus-340, at cruise flight[5]; 2) in a restaurant kitchen, at lunch time; 3) in an electric commuter train while stopped at a station; and 4) within a common passenger vehicle, alone on a highway (at 3 a.m.), travelling at a steady 120 km/h, with windows closed and radio off.

Amplitude levels, or sound pressure levels, were measured in both dBA (with the A-weighting network) and in dBLin (with no network, linearly).

Table 1 summarizes the amplitude levels obtained. All dBA-level values are within 0.9 dB of one another. However, the dBLin values reflect a different reality. Indeed, these four environments are comparable if the issue is what is being *heard*; however, in terms of the actual acoustical energy present, these environments vary significantly – there is a 20 dB difference between the dBLin values of the kitchen and the car.

TABLE I

Description of the overall acoustic amplitude at each location. dBA levels are comparable, with a maximum of 0.9 dB difference between them. dBLin levels are much larger and present a maximum difference of 20 dB.

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

Simultaneously, real-time frequency analyses were performed, providing information on the frequency distribution of the acoustic energies present in these environments. Figs. 1-3 show the frequency distributions at each location.

As can be clearly observed, in these four locations a substantial amount of acoustic energy is concentrated within the lower frequency bands. This is reflected by the dBLin value but not by the dBA value. Moreover, the predominance of different frequency bands are different for all four

locations. Alone, a dB level value (whether in dBA or dBLin) does not provide information on the frequency distribution of the acoustic energy.

Consequences of erroneous assumptions

Tissues and organ systems have distinct acoustic properties. The acoustic impedance[6] of the liver is different from that of the lungs. Similarly, the resonance frequency[7] of the skull is different than that of the abdominal cavity. This means that different acoustical frequencies will elicit different responses from all the different types of tissues and organ systems.

By assuming that acoustical environments are comparable just because their dB levels are comparable, an error is introduced into the studies. As seen above, environments with comparable dB-levels may have entirely distinct frequency distributions.

Thus, acoustic environments with different distributions of frequency bands cannot be expected to induce the same effects. However, since
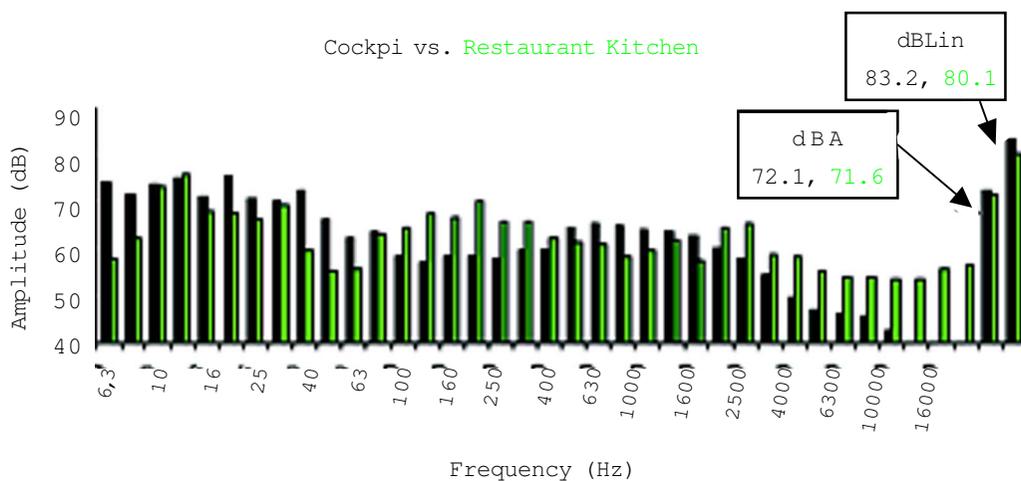

Fig. 1 — Comparison between the frequency distributions obtained within the Airbus-340 cockpit (black) and within a restaurant kitchen at lunch hour (green). dBA levels are comparable (72.1 vs. 71.6). dBLin levels are higher than dBA levels and the difference between both environments is more substantial (83.2 vs. 80.1). In both locations, the lower frequency components (<500Hz) concentrate a substantial part of the acoustic energy. At the more audible frequencies (>1000 Hz), the amplitude is lower.
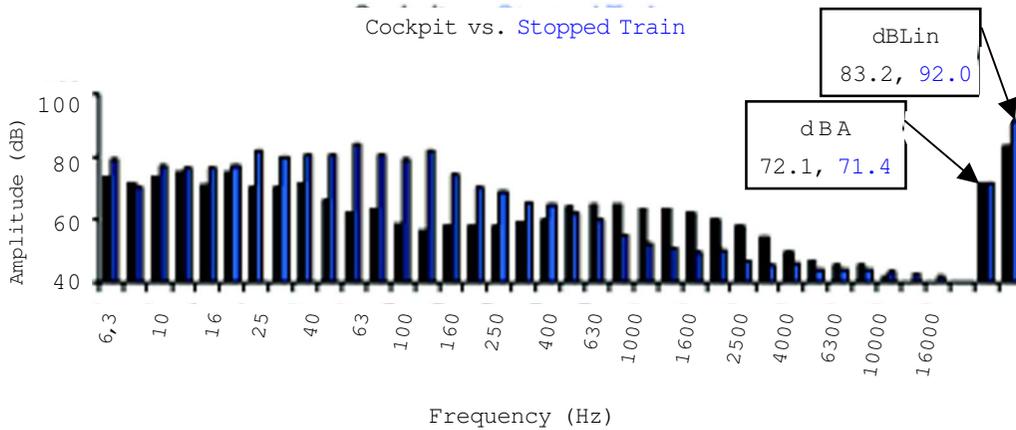
Fig. 2 — Comparison between the frequency distributions obtained within the Airbus-340 cockpit (black) and within an electric commuter train stopped at a station (blue). dBA levels are comparable (72.1 vs. 71.4). dBLin levels are higher than dBA levels: 83.2 vs. 92.0. In both locations, a substantial part of the acoustic energy is concentrated within the lower frequency bands (<500Hz). At the more audible frequencies (>1000 Hz), the amplitude is lower, and above 6300 Hz, it is ≤ 45 dB.
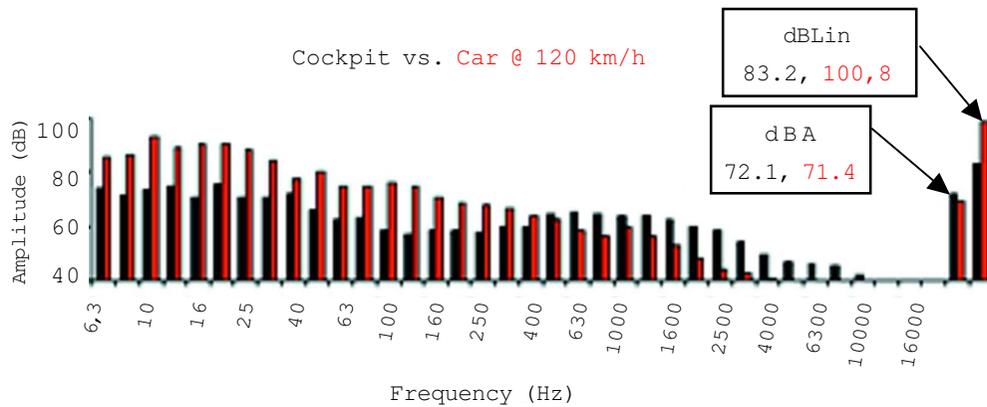

Fig. 3 — Comparison between the frequency distributions obtained within the Airbus-340 cockpit (black) and within an common passenger vehicle, travelling at 120 km/h, alone on a highway (at 3 a.m.), and with windows closed and radio off (red). dBA levels are comparable (72.1 vs. 71.2). dBLin levels are higher than dBA levels and the difference between both environments is more substantial (83.2 vs. 100.8). In both locations, most of the acoustic energy is concentrated within the lower frequency range (<500Hz). At the more audible frequencies (>1000 Hz), the amplitude is lower, and above 4000 Hz, it is below 40 dB.

frequency distributions are usually unknown (because they are not required by legislation), equal biomedical studies that use comparable dB-level acoustic environments may unknowingly yield different results, due to the differences in frequency distributions. The issue of LFN contamination is, therefore, non-trivial because if LFN is not being assessed (particularly the non-audible frequencies <20 Hz), then biomedical studies that impute some result to a certain cause may be erroneous.

As a consequence of neglecting to provide information on the frequency content, parallel but non-comparable studies proliferate among the

scientific literature[9]. For example, Resi and colleagues have been performing biochemical and ultrastructural studies among noise-exposed animal models[9,10]. However, noise was only described in terms of a dB-level measurement. Thus, it would not be surprising to see similar studies, with the same level of noise exposure, but with distinctly different results. These are the main reasons why non-auditory, noise-induced pathology is still considered a controversial, contradictory, and inconclusive subject.

Noise-induced non-auditory pathology

Numerous authors have referred to non-auditory pathology[8], 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"[11]. Regarding non-auditory effects of noise, the 4th edition of Industrial Hygiene, published in 1996 by the United States National Safety Council 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."[12]

According to OSHA[13] 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"[14]. 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"[15]. Controversial, contradictory, and hence, inconclusive is still the mainstream belief regarding noise-induced, non-auditory pathology[8,16].

LOW FREQUENCY NOISE-INDUCED PATHOLOGY

Studies in Portugal

In Portugal, research into the effects of low frequency noise (LFN) (≤500 Hz, including infrasound) are ongoing since 1980[17,18]. As a consequence, vibroacoustic disease (VAD) has been identified as a systemic pathology, characterized by an abnormal proliferation of the extra-cellular matrices, and caused by long-term exposure to LFN[19-21].

The initial triggering factor that justified the undertaking of a thorough neuropsychological evaluation, of aircraft technicians employed at OGMA[22], was the observation that 10% of these workers had been previously diagnosed with late-onset epilepsy. The expected rate for the Portuguese population is 0.2%[23]. The following section will chronologically trace the interest taken in the respiratory pathology associated with VAD and LFN exposure.

From pleural effusion to focal fibrosis

Four VAD patients had atypical cases of pleural effusion that persisted in spite of therapy. Three of these cases were of unknown origin, although the fourth may have been caused by diphenylhydantoin[24]. The follow-up recovery periods were very prolonged, even in the case where diphenylhydantoin was suspended. Treatment took several months, and recovery was not only slow and irregular, but no conclusion was ever reached about the aetiology or choice of treatment.

In 1987, the autopsy performed on a deceased VAD patient (one of the 4 cases of pleural effusion), marked a drastic shift in concepts. LFN-induced pathology was not limited to the neuropsychological realm. This individual exhibited

TABLE II
Data from a group of 140 aircraft technicians (selected from an initial group of 306 workers), occupationally exposed to LFN. Exposure time (in years) refers to the amount of time it took for 70 individuals (50%) to develop the corresponding sign or symptom[19].

Clinical Stage	Sign/Symptom
Stage I- Mild (1-4 years)	Slight mood swings, Indigestion & heart-burn, Mouth/throat infections, Bronchitis
Stage II-Moderate (4-10 years)	Chest pain, Definite mood swings, Back pain, Fatigue, Fungal, viral and parasitic skin infections, Inflammation of stomach lining, Pain and blood in urine, Conjunctivitis, Allergies
Stage III – Severe (> 10 years)	Psychiatric disturbances, Haemorrhages of nasal, digestive and conjunctive mucosa, Varicose veins and haemorrhoids, Duodenal ulcers, Spastic colitis, Decrease in visual acuity, Headaches, Severe joint pain, Intense muscular pain, Neurological disturbances

two tumors (Grawitz and malignant glioma), as well thickened cardiovascular structures throughout the body, particularly the pericardium. Focal lung fibrosis was also identified, however no importance was attributed to this finding since chemicals, fumes and dusts were assumed to be present in this man's occupational environment[24]. The cause of death was cardiac tamponade due to a perforated infarct, and his heart presented an additional 11 scars of previous, silent ischemic events[24].

In 1992, still concerned about the enigmatic cases of pleural effusion, animal models were used to study the respiratory tract under the effect of LFN exposure. The plethora of information yielded by these studies is still today the object of intense study. In LFN-exposed rodents, the amount of tracheal cilia was visibly reduced[25], and subsequent formal morphometric studies confirmed this feature[26]. Tracheal subepithelial fibrosis was also identified[25]. Structural changes of the lung parenchyma included irregular distribution of thickened alveolar walls, dilated alveoli, and irregularly distributed fibrous foci[27]. Pleural cells lost their phagocytic ability, and the pleural parietal leaflet had a marked reduction in the amount of microvilli per mesothelial cell [28].

The existence of fibrosis in the trachea and parenchyma was indeed a surprising finding, and taken together with the lung fibrosis observed in autopsy, it seemed to be an important finding. Subsequently, respiratory functional tests as well as high resolution CT scan of the lung were administered to LFN-exposed workers, with and without respiratory symptoms. Focal lung fibrosis and air-trapping was identified in these workers, independent of the existence of respiratory complaints[29]. Today, bronchitis is among the first symptoms to appear in LFN-exposed workers, smokers and non-smokers alike (See Table II). In the severe stages, many (non-smoker) patients develop respiratory insufficiency.

Genotoxicity and squamous cell carcinomas

To date, in a universe of 945 individuals, there are 41 reported cases of malignancies, of which 9 are multiple. There seems to be a pattern to the histological type of tumors associated with VAD. They occur in hollow organs or cavities, such as bladder, intestine, kidney, and lung. In the CNS all tumors in these patients are malignant gliomas (10 cases), where the skull functions as yet another cavity[20].

LFN has already been identified as a genotoxic agent in both human[30,31] and animal models[32] where the frequency of sister chromatid exchanges is statistically significantly increased in the LFN-exposed populations. The synergy among vibration and low frequency noise aggravates this genotoxic effect[33].

To date, all respiratory tract tumors in VAD patients are squamous cell carcinomas: 11 cases (3 smokers) – 2 of the glottis and 9 of the lung[34]. All lung carcinomas were located in the upper right lobe. Squamous cell carcinomas account for 40% of all lung cancers in males[35].

Studies from abroad

Other authors have studied the effects of LFN the respiratory system. Most of these studies were conducted some decades ago but still provide valid evidence of the respiratory system as a target for this physical stressor. Some of these studies may have slight design flaws: insufficient population sample to render them statistically significant, inadequate description of the sound sources used, or even, perhaps, a different agenda; i.e., noise abatement or hearing conservation programs. But the information they provide is intriguing, somewhat consistent, and certainly warrants a closer look of the effects of noise on the respiratory tract.

Two interesting studies were conducted on human subjects by the same team of scientists in the mid-60's[36,37]. Unfortunately the number of subjects involved in these two studies (respectively, N=5 and N=4 military personnel) renders them statistically nonsignificant. However, the acoustic simulation facilities used were unique: they allowed for simultaneous subjective and objective stress responses during exposure to a well-defined acoustic environment, which provided detailed information on both amplitude *and* frequency *and* exposure time. The following year, a similar study was performed with the same acoustic source. Please see Tables III and IV. Subjective complaints involving the respiratory tract are abundant.

In 1969, Ponomarkov et al. explored the effects of wide-band noise at 105-155 dB on dogs[38]. After 1.5-2 h of exposure, the animals were sacrificed. Autopsy results revealed "hemorrhages up to 3 mm in diameter (...) in the lungs" of the animals exposed to about 126 dB, located "beneath the pleura in the form of convex vesicles." They were most common in the costal surface of the upper lobe of the right lung. The authors claim that as the dB level of the noise increased, the number of the hemorrhages increased, but they "never exceeded 3mm in diameter." Microscopic analysis of the hemorrhaged section of lung tissue revealed ruptured capillaries and larger vessels. Microscopic analysis of the emphysematous areas showed "focal enlargement of the alveoli, stretching of the connective-tissue structures of the alveolar walls, and compression of lung tissue." They conclude their report by giving a possible explanation for such pathology: "The lungs, as a system open to the external environment, are subjected to the greatest influence of changes in pressure in this medium. Depending on the magnitude of the pressure differential created by sound in the lungs during its transformation, and of the threshold of resistance of the lung tissue to pressure changes, the severity of the lung lesions gradually increases."

Table III
Summary of noise source description and subjective responses[1].

Noise Source	Frequency Range (Hz)[2]	dB Range[2]	Frequency @ max. dB (Hz)	Exposure Time	Subjective Response (No. of Subj) (N=5)
AMRL[3] High Intensity Noise Facility	8-1000	95-122	100	2 min	mild chest wall (5) & body hair vibration (5)
J57 Turbojet Aircraft Engine	6-1000	112-135	100	1 min	mild chest wall vibration (5); "awareness" of the respiratory action (1)
NASA-LRC[4] Thermal Structures Tunnel	3 - 10000	117-138	40	1 min	mild chest wall (2) & nasal cavity (2) vibration; perceptible throat fullness (1)
NASA-LRC Thermal Structures Tunnel	3-10000	102-135	80	25 sec	mild-moderate chest wall vibrations (5); interference with normal respiratory rhythm (3); throat pressure (2)
NASA-LRC Low Frequency Noise Facility	10-60	118-140	30	2 min minimum	moderate chest wall vibration (5); hypopharyngeal fullness (gagging) (5); perceptible visual field vibration (5)
USAF-RTD[5] Sonic Fatigue Facility	100 (discrete signal)	153	n/a	2 min minimum	coughing, severe substernal pressure, choking respiration, salivation, pain on swallowing, hypopharyngeal discomfort (5)

[1] As per Mohr et al.[36]; [2] Values are approximate; [3] Aerospace Medical Research Laboratories Wright-Patterson AFB; [4] Langely Research Center;
[5] Research and Technology Division

In 1976, Cohen evaluated 400 boiler plant workers before and 2 years after the implementation of a hearing conservation program[39]. Noise was described as 95 dBA or higher, and no frequency spectrum was provided. Job injuries, diagnosed disorders, symptomatic complaints, discrete absences and total absent days were registered before the program was established and

TABLE IV
Summary of noise source description and subjective responses[1].

Noise Source	Frequency Range (Hz)*	dB Range*	Frequency at max. dB (Hz)	Exposure Time	Subjective Response (No. of Subj) (N=4)
AMRL[2] High Intensity Noise Facility	10-1000	75-105	60	1-5 min	vibration of body hairs (4); perceptible chest vibration (4)
J57 Turbojet Aircraft Engine	8-5000	80-122	80		tooth vibration (1); "awareness of respiratory excursions (1)

[1] As per Cole et al.[37]; [2] Aerospace Medical Research Laboratories Wright-Patterson AFB; [3] Values are approximate.

then, again, two years later. All of the above parameters registered a statistical decrease except the incidence of symptomatic complaints, which remained unaltered. Cohen states, "these general findings, with the exception of those for symptomatic complaints, uphold the hypothesis that there would be fewer reported extra-auditory problems for workers in high noise jobs subsequent to the establishment of a hearing conservation program." These symptomatic complaints arose from several organ systems and were identical to those found in VAD patients, including backaches and neckaches, dizziness, diarrhea and blood in urine, soreness in muscles, cramps and skin itching[19]. Respiratory system complaints were coughing, congestion in head and chest, shortness of breath, and hoarseness. See Table V.

In 1987 Svigovyi et al.[40] investigated the effects of infrasound on the pulmonary ultrastructure of white mice. Noise was characterized as 2, 4, 8, or 16 Hz, at 90-140 dB. The mice were exposed for 3 hr daily, for 1, 5, 10, 15, 24 and 40 days. After 3 h of exposure, "point, mosaic-type" hemorrhages were identified over the entire lung surface. These hemorrhages were observed beneath the pleura for exposures of 2-4 Hz at 92-100 dB, and large bleeding spots were identified in lung exposed to 8-16 Hz, at 120-140 dB. After 10-15 d of exposure, parts of the lung tissue are filled with blood and the walls between alveoli are swollen and thick. Dramatic morphological changes of alveolar, cellular and blood vessel structures, are described after exposures of 24-40 days, at 8-16 Hz and 120 dB. These included deformation of nuclei, and concentration of chromatin under the nuclear membrane in types I and II pneumocyte cells; breakage, and deformation of alveolar sacs, destruction of alveolar walls, and burst capillaries and larger vessels.

WHERE TO GO FROM HERE

The recognition of LFN as an agent of disease will require a major shift in noise-related policy making, and that usually takes a long time. In the meantime however, physicians and scien-

TABLE V
Some complaints of boiler-plant workers[1].

Organ System	Symptomatic Complaint
Allergenic and Dermatological	Skin Itching, Skin Burning
Respiratory	Coughing, Congestion in Head and Chest, Shortness of Breath, Hoarseness
Neurological	Headaches, Dizziness, Numbness
Digestive	Stomach Cramps, Nausea, Diarrhea, Heartburn
Urological	Irregular Urination, Pain in Bladder area, Blood in Urine
Muscular and Skeletal	Backaches and Neckaches, Soreness in Muscles, Cramps

[1] As per Cohen[39].

tists can begin to treat patients and conduct biomedical research keeping in mind that LFN can be a contaminating factor.

For example, in many institutions animal labs are kept in the basement where, depending on the type of ventilation (HVAC systems) and mechanical equipment (elevator machine rooms), LFN may be present in significant amounts. Within this context, what is a control animal? Similarly, in human studies that investigate pulmonary pathology, subjects are not questioned as to their LFN exposure – occupational, residential, leisurely or environmental. Thus, contamination by LFN is possible and even probable.

Environmental LFN is ubiquitous in urban settings and common in suburban settings. Trains, automobiles, buses, airplanes, ferries and subways, as well as highways, ventilation systems, and other commonly used mechanized technologies for the home, are all sources of LFN. Morning and afternoon rush hours can greatly increase the amount of LFN in surrounding residential areas. Leisurely LFN is an ever-growing indus-

try. Cinema, discotheques, automobile sound systems, motorbikes, and jet skis are but a few of the LFN sources to which everyone is exposed in modern society. In fact, a silent place is a luxury.

The need for further research using animal models is evident. LFN impinges on the whole body inducing a variety of pathologies in many organs and systems. In the real world, there are no pure models, and in addition to LFN, humans are exposed to a variety of other aggressors. Moreover, only through the use of animal models with controlled acoustic environments can specific frequencies be correlated with specific lesions. For the past decade, animal models have been yielding information not only related to LFN-induced pathology, but also associated to the nature of living tissue and cellular populations under stress. Within this context, classical concepts regarding the process of cell life and death are being challenged[41].

Given the information herein, it would seem that physicians and scientists should at least be giving noise-induced pulmonary pathology the

benefit of the doubt. Whether or not this is a politically convenient position is beyond the scope of this report. Of course, it is always possible to continue ignoring LFN as an agent of disease, although that would be highly undesirable and most certainly unethical.

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