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Low frequency noise effects on the periodontium of the Wistar rat - a light microscopy study

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SUMMARY

Long-term low-frequency noise (LFN) (≤500Hz, infrasound included) exposure is known to cause extracellular matrix proliferation with fibrosis, in the absence of inflammatory signs. The aim of this work was to study the morphological alterations to the periodontium of Wistar rats exposed to LFN. 10 rats were exposed to LFN for 2184 consecutive hours and another 10 were kept in silence. The mandibles were removed, kept in 10% buffered formalin, sectioned sagitally, stained with haematoxylin-eosin (HE) and Masson's trichromic solution (TCM) and observed with light microscopy (LM).

The results revealed a disappearance of the cementum, irregular erosion of surface alveolar bone, and signs of bone necrosis, with detached bone particles. The periodontal ligament was disorganized and had deficient anchorage of the fibers.

These findings may be due to a direct effect of noise and vibration impinging on the structures, to stress, to vascular alterations or to a combination of these factors. They may also explain the alterations in alveolar bone, reported by other authors.

Key words: Low frequency noise – Periodontium – Vibration

INTRODUCTION

Low-frequency noise (LFN) (≤ 500Hz) is present in the everyday environment of industrialized nations (Alves-Pereira, 1999) and low-frequency noise-induced pathology has been studied in Portugal since the early 80's (Gimogma, 1984).

Noises rich in low-frequency components are issued by an increasing number of technical sources. Airplanes, helicopters, trains, ventilation equipment are, among others, common sources of noise and vibration. Moreover, because of its band width LFN can spread across large distances with low attenuation, passing through walls and windows and making protection very difficult.

Aircraft maintenance technicians, cabin crew members and pilots have often been studied as regards the deleterious effects of LFN on their organs and systems, namely cardiovascular, CNS, and respiratory (Mauricio et al., 1999; Reis-Ferreira et al., 1999).

Morphologically, the most frequent by reported alterations consist of a progressive fibrosis of tissues, due to extracellular matrix proliferation, and cellular death in the absence of inflammatory process (Castelo-Branco et al., 1999a, 1999b). The cardiovascular system has been extensively studied and the most striking alterations consist of a thickening of

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the heart valves and of the pericardium, documented echographically (Marciniak et al., 1999). Arterial and lymphatic lesions consist of a thickening of the vascular walls and distortion (Martins dos Santos et al., 2002a, 2004b). The lung parenchyma also shows a marked thickening caused by fibrosis (Grande et al., 1999; Sousa Pereira et al., 1999; Oliveira et al., 2002; Castelo Branco et al., 2003, 2004). Gastric and duodenal lesions have also been reported (Da Fonseca et al., 2005, 2006).

There are few studies concerning the oral cavity and its structures. Morphofunctional alterations in the parotid gland have been reported (Oliveira et al., 2007). In 1975, Haskell studied a group of aircraft pilots and concluded that alveolar bone loss was strongly correlated with flight hours and noise levels (Haskell, 1975). In 1998, Pimentel reported increased rates of alveolar bone osteolysis in individuals subjected to LFN.

The objective of the present study was to evaluate the morphological alterations of the alveolar bone of Wistar rats exposed to LFN.

Material and Methods

All animal experiments were performed on isolated mandibles taken from 20 adult (250-350 g) male and female, (in equal numbers) CD strain Wistar rats with ages ranging from 8 to 12 months.

The animals used in this study were randomly divided into 2 groups. 10 rats were assigned to group 1 (LFN subjected group) and subjected to 2184 h (13 wk) of continuous LNF (Fig. 1) while the other half, assigned to group 2 (Control group), were kept in similar laboratory conditions but in silence. The rats were kept in cages with 2 or 3 animals per cage, with no limits to their movements, and were exposed to cycles of 12 hours light/dark. All animals were fed with standard rat chow, had unrestrained access to water, and were treated according to the EU directive on Animal Protection for Experimental and Scientific Purposes (86/609/CE) and also according to the Portuguese laws in that regard.

Animals were sacrificed with a lethal intraperitoneal injection of ketamine (4.0-8.0 mg/Kg). Mandibles were removed, kept in 10% buffered formalin, sectioned sagitally, stained with haematoxylin-eosin (HE) and Masson's trichromic solution (TCM).

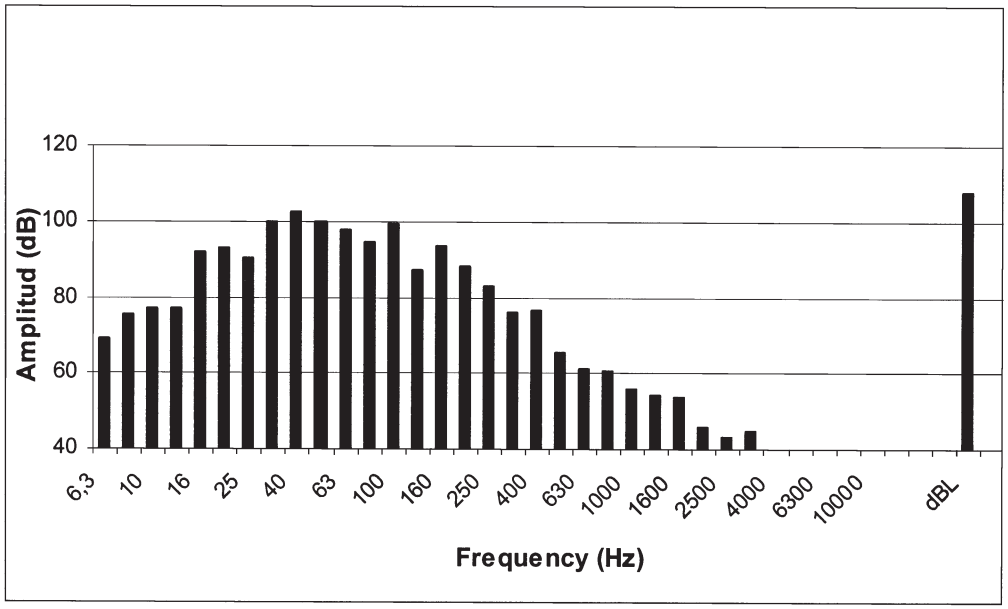
Results

Control animals presented full integrity of the periodontal structures, as well as tooth structures (pulp and dentin) (Fig. 2).

LFN-exposed animals showed periodontal alterations; namely, in alveolar bone and in the periodontal ligament.

We observed irregular erosion of the bone surface and signs of bone necrosis, with detached bone particules in the periodontal ligament (Figs. 3 and 4).

Fig. 1. Frequency distribution of the acoustic environment to which the rats were exposed.



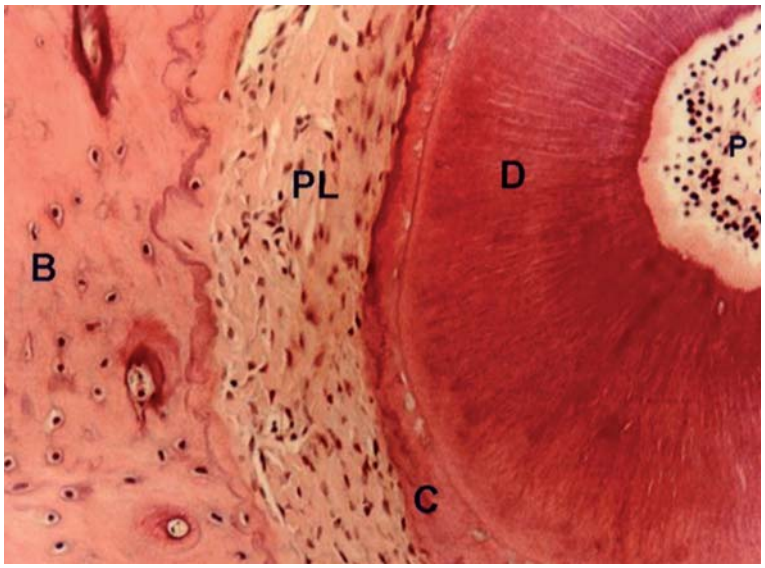


Fig. 2. Periodontium structures (HE) with normal definition. B- alveolar bone, PL- periodontal ligament, C- cement, D- dentin, P- pulp. x 200.

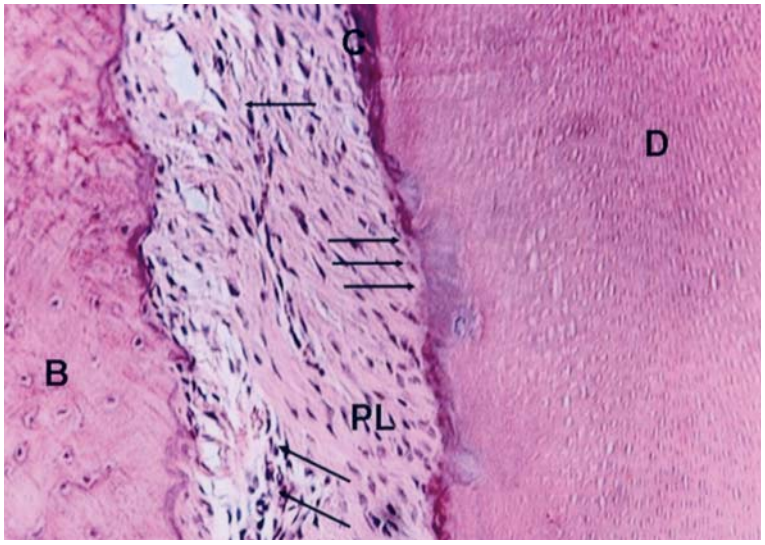


Fig. 3. Periodontal structures (HE) of a LFN-exposed animal with the observed irregular erosion of the bone surface (arrow), irregular teeth surface (triple arrow), and deficiency anchorage of periodontal ligament fibers, more evident on alveolar side bone (double arrow). B- alveolar bone, PL- periodontal ligament, D- dentin, C-cement. x 400.

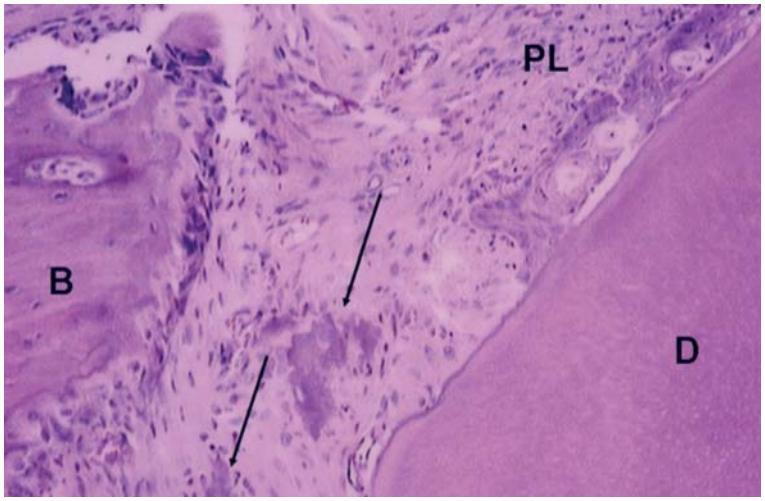


Fig. 4. Periodontal structures (HE) with the presence of necrosis (arrow), the presence of bone particles detached from the bone surface. B- alveolar bone, PL- periodontal ligament, D- dentin. x 400.

A disappearance of the cementum and surface erosions on the root side were also observed (Fig. 3).

The periodontal ligament showed a deficient anchorage of the fibers, more visible on the alveolar bone side of the ligament (Fig. 3). Some spatial disorganization of fibroblasts was also found.

Discussion

The most striking alterations observed in LFN-exposed animals were alveolar bone necrosis and irregular erosion on the bone surface, probably due to increased osteoclastic activity.

In a radiological study conducted in aircraft pilots, Haskel (1975) concluded that alveolar bone loss had occurred; this was more pronounced in propeller aircraft pilots then in jet fighter pilots.

This author found a high correlation between flight hours and the degree of bone loss.

He put forward the hypothesis that his observations were due to noise and vibration, to circulatory alterations, differences in atmospheric pressure or, less likely, to psychological stress.

In 1977, Carlson and Kjell, in a radiological study, compared a group of non-flying with jet flying personnel over 10 years. He concluded that there was a significant alveolar marginal bone loss of the mandible in the flying personnel. As etiological factors, those authors considered: stress, irregular habits, oxygen breathing, noise, vibration and pressure from the oxygen mask.

More recently, in 2004, Takada et al. studied the effects of restraint stress and ligation on the progression of periodontitis in rats. They reported a loss of alveolar bone with the concurrent effects of stress and ligation. These authors concluded that stress can lead to severe periodontitis and that this condition, together with ligature, causes the observed bone loss.

In previous studies, our group reported marked vascular alterations in small- and medium- sized arteries of LFN-exposed animals. (Martins dos Santos et al., 2002; Da Fonseca et al., 2005, 2006).

In our study we found alveolar bone necrosis; this may be responsible for the bone loss reported by Haskel (1975) and Carlson and Kjell (1977).

Bone necrosis may be caused by a direct effect of noise and vibration impinging on bone structures, stress, vascular alterations or a combination of all these factors.

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