Understanding the biological responses elicited by low frequency noise exposure: contributions to vibroacoustic disease research.

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BACKGROUND. Low frequency noise (LFN, ≤500 Hz) is a physical agent of disease that can lead to the development of vibroacoustic disease (VAD) [1]. Some acousticians and physicians strongly disagree with this statement based, however, on pre-existing acoustical and biological assumptions that can no longer be upheld. For acousticians, the assumption is: what you can't hear won't hurt you. For physicians, the assumption is that collagen production needs to occur with an associated inflammatory process. In VAD, acoustical phenomena that do not cause pain to the ear can trigger morphological rearrangements in organs and tissues. These rearrangements include the production of functionally (not randomly) organized units of collagen fibers, in the absence of an inflammatory process.
PURPOSE. To propose hypotheses for the mechanisms responsible for the biological and clinical responses to LFN exposure, as seen in both human and animal models.
METHODS. Drawing upon 29 years of research, the biological and clinical outcomes of occupational LFN exposure are explored in the light of mechanical cellular signaling.
RESULTS. It is well-known that pressure is communicated throughout tissues and organs through mechanical cellular signaling, or mechanotransduction. In VAD, the agent of disease is a pressure wave. It is proposed that VAD is a mechanotranduction disease *par excellence* because immediate effects of LFN exposure are initially mediated through mechanical cellular signaling, and long term effects require an understanding of cellular tensegrity structures. Most medical diagnostic procedures are not based on mechanobiological features of disease. Hence, what is analyzed, quantified and tested are usually parameters that depend on biochemical pathways. In the first stages of VAD (1-10 years of occupational exposure to LFN), patients exhibit normal routine tests, such as blood chemistry analysis, EKG, and EEG, for example. With echo-imaging, however, where structural components can be observed, Stage 1 and 2 VAD patients disclose peculiar pericardial and cardiac valve thickening, mainly due the production of collagen. In autopsy and through electron microscopy studies of biopsy material, blood and lymphatic vessel walls are found to be thickened with collagen. Sub-basal layers of respiratory epithelia, in both VAD patients and LFN-exposed animals, also disclose thickened structures due to the presence of abnormal amounts of collagen.
Collagen has been likened to the "steel" of the human body because of its specific biomechanical properties. The elevated amount of collagen produced in response to LFN exposure, can be viewed as an attempt by the body to reinforce structural integrity. In effect, the biomechanical features of the morphologically "new" pericardium seen in VAD patients, suggests the formation of a pneumatic-like structure, presumably to maintain the cardiac cycle intact. Cell-matrix connections between the pericardial mesothelium and sub-mesothelial layer are often accomplished through invaginations, or herniations, and a peculiar structure with anti-seismic-like structures.
CONCLUSIONS. Excessive LFN exposure causes disease that can only be understood if mechanotransduction cellular signaling is considered as a mediating pathway, and when tensegrity architecture of cells and tissues is considered an important factor.

1. Alves-Pereira M, Castelo Branco NAA. Vibroacoustic disease: Biological effects of infrasound and low frequency noise explained by mechanotransduction cellular signaling. Prog Biophy Molec Biol, 93:256-279, 2007.