Noise-Induced Hearing Loss and Tinnitus in Military Personnel

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ABSTRACT
Hearing loss is the most common form of sensory impairment in humans, affecting 5.3% worldwide population. Hearing is critical to the performance of military personnel and is integral to the rapid and accurate processing of speech information. Noise-induced hearing loss represents a severe impairment that reduces military effectiveness, safety, and quality of life. Military personnel work in high-noise environments, yet the Department of Defense cannot predict who is susceptible to noise-induced hearing loss and tinnitus. Of those exposed to noise, 80% may also suffer from chronic tinnitus. Despite its prevalence, there are no means to objectively measure the severity of tinnitus in those individuals. A fundamental understanding of the underlying mechanisms of tinnitus and its relation to noise-induced hearing loss is critical. Such an understanding may provide insight to who is at risk for each condition, allow aggressive hearing protection measures in those individuals most at risk, and create areas for treatment for those already suffering from the conditions. The current review addresses the scope of the problems of noise-induced hearing loss and tinnitus for the military, discuss the noise environments in which military personnel operate, and describe recent pharmacotherapy trials. Some recent breakthroughs in noise-induced hearing loss research are discussed along with some challenges and directions for future research on hearing loss and tinnitus.

KEYWORDS
Noise Exposure; Hearing loss; Military; Tinnitus.

COCHLEA AND HAIR CELLS
The mammalian cochlea is the sensory organ capable of perceiving sound over a range of pressure, and discriminating both infrasonic and ultrasonic frequencies in different species. The organ of Corti is located in the cochlea of the inner ear and is responsible for the detection of sound. This organ harbours the auditory sensory epithelium, which, in humans, contains approximately 16,000 hair cells that are patterned into three rows of outer hair cells (OHCs) and one row of inner hair cells (IHCs)[1, 2]. The cell bodies of hair cells form specialized adhesive contacts with supporting cells that adhere at their basolateral surfaces to the basilar membrane, an extracellular matrix assembly with a different molecular composition from the tectorial membrane [3, 4].

Hearing is initiated when sound waves that reach the outer ear travel through the ear canal to the tympanic membrane. Then, the sound energy is transferred, via the bony ossicles of the middle ear, to the oval window at the base of the fluid-filled cochlea. The motions of the oval window are converted into fluid pressure waves that induce vibrations in the basilar membrane. Then, the vibrations are transferred onto the hair cells, leading to the deflection of the hair cell stereocilia [5]. This deflection causes the opening of transduction channels leading to hair cell depolarization and to the release of neurotransmitters onto neurons, which form synapses with hair cells. The electrical signals are propagated through the nervous system and processed in the brainstem and auditory cortex [6, 7].
HEARING LOSS

Hearing loss is the most common form of sensory impairment in humans, affecting 360 million persons worldwide, with a prevalence of 183 million adult males and 145 million adult females. In nonsyndromic deafness, only hearing function is noticeably altered, whereas syndromic deafness is accompanied by other physiological defects.

Hearing loss can be caused by environmental factors, such as exposure to noise or ototoxic chemicals, or by aged related senescence. Traumatic injury, such as injury caused by exposure to an explosion or to the firing of a gun, can lead to sudden hearing loss. Sometimes this hearing loss is accompanied by the perception of a constant ringing noise called tinnitus [8]. Moreover, genetic factors as mutations in MT-TS1, MYO7A or ACTG1 genes [9-11], between many others, have already been linked to nonsyndromic hearing loss.

Noise exposure is responsible for approximately 10% of hearing loss in adults, in particular military veterans [12]. Short impulses of high intensity noise such as a gunshot or explosion can trigger sudden hearing loss, which is generally irreversible and associated with structural damage to the auditory system. Susceptibility to damaging effects of noise differs remarkably among individuals, which indicates that genetic factors might be important in disease development. Gene association studies using candidate-gene approaches have focused mostly on genes that are linked to oxidative stress, potassium recycling and the heat shock response [13].

On the other hand, various chemical agents as aminoglycoside antibiotics, platinum-containing chemotherapy agents and nonsteroidal anti-inflammatory drugs are ototoxic [14, 15]. For example, aminoglycosides, antibiotic with broad spectrum activity, cause significant hearing loss, with estimates of a 20–50% chance of incidence when treating acute infections [16, 17]. Hair cells are readily damaged by this compound probably due the similarity of hair cell mitochondrial ribosomes to their bacterial counterparts [18].

Finally, the most common form of sensory impairment in older people is the age-related hearing loss [19]. This disorder is characterized by symmetric sensorineural hearing loss that starts at high frequencies with a prevalence of 35% of individuals over 65 years of age [20]. Although hearing loss has been considered to be part of a natural ageing process, not all humans suffer from age-related hearing loss [21].

**Noise-induced hearing loss in military**

People serving in the military, especially those in areas of combat, are at some point exposed to high-intensity noise of various types. Two possible consequences of such exposures are the development of a hearing loss, most prominent for high-frequency sounds, and tinnitus [22, 23]. Depending on a variety of factors, these effects may be either temporary or permanent consequences of such an exposure.

If documentation of the existence of hearing loss or tinnitus at discharge from the military is missing, it is nearly impossible to determine whether hearing loss or tinnitus detected by audiometric testing later in life is the result of noise exposure during prior military service. However, several studies demonstrated that the two most prevalent service connected disabilities for veterans in the United States at the end of fiscal year 2012 remain tinnitus and hearing loss, with tinnitus affecting 115,638 veterans (9.7%) and hearing loss affecting 69,326 veterans (5.8%) [24]. Both noise and aging, for example, result in similar high-frequency hearing loss, although the specific patterns of hearing loss resulting from each are generally distinguishable until 60–70 years of age. This adds to the challenge of determining the cause of the hearing loss when the only existing documentation consists of hearing thresholds measured late in life and many years after military service. In addition, it is quite likely that an individual might have experienced other hazardous noise exposures subsequent to discharge from military service that could result in significant noise-induced hearing loss or tinnitus. After the fact, for example, there are no current available means to distinguish the hearing loss resulting from several years of military service from the noise-induced hearing loss resulting from subsequent work in a noisy industry or from participation in a wide variety of recreational activities. This serves to underscore the importance of measuring hearing thresholds at enlistment and at discharge, with annual measurements in between for those most at risk for noise-induced hearing loss and tinnitus.

These uncertainties regarding noise-induced hearing loss and tinnitus have placed the Department of Veterans Affairs (VA) in a quandary. Frequently, VA personnel are called on to determine whether the hearing loss measured in a 70- or 80-year-old veteran is due to this individual’s prior military service. Furthermore, this assessment frequently must be done in the absence of documentation of the measurement of hearing thresholds at or around the time of military service as mentioned before.

Moreover, information on noise sources and levels in the military environment is not easily summarized. Sound levels vary depending on the distance from the sound source and the conditions under which the sound is being generated. Important characteristics of impulse noise include not only the peak sound pressure level, but the time pattern of the impulses and the frequency spectrum [25-28]. The Table 1 provides examples of some of the measurements made since the 1950s of average sound levels found in ground vehicles and aircraft and
peak sound pressure levels generated by certain weapons. On aircraft carriers, flight operations create an environment with combinations of aircraft noise, mechanical noise, and impact noise [29]. Below the flight deck, sound levels have been measured at 106 dBA during aircraft launches. Exposure to high sound levels has also been reported for military personnel in positions such as radio operators [30] in the Navy and cryptolinguists in the Air Force [31]. In addition, military personnel may encounter potentially damaging noise from equipment and activities comparable to those found in industrial settings, such as the operation of heavy equipment. The examples of noise levels associated with equipment and weaponry in the military included in Table 1 clearly demonstrate that there are many sources of high sound pressure levels in the military environment that exceed criteria for safe exposure. Data on sound pressure levels, however, are not sufficient by themselves to determine the noise dose received by an individual.

**Table 1: Examples of sound levels associated with military equipment.**

<table>
<thead>
<tr>
<th>Aircraft in use in 1950s</th>
<th>Name</th>
<th>Location</th>
<th>Sound Level (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Douglas Skyraider</td>
<td>A-1J (AD-7)</td>
<td>Cockpit</td>
<td>118-132</td>
</tr>
<tr>
<td>Douglas Skyraider</td>
<td>A-1J (AD-7)</td>
<td>Passenger area</td>
<td>106-111</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Military Equipment in use in 1960s</th>
<th>Name</th>
<th>Location</th>
<th>Sound Level (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NATO rifle</td>
<td>M-14</td>
<td>Operator</td>
<td>159</td>
</tr>
<tr>
<td>Helicopter (Shawree)</td>
<td>H-21C</td>
<td>Crew chief</td>
<td>110</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Military equipment in use in 2005</th>
<th>Army vehicles</th>
<th>Model</th>
<th>Location</th>
<th>Sound Level (dB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>High-mobility multipurpose wheeled vehicle (HMMWV), non-heavy variants</td>
<td>M996, M997, M998, etc</td>
<td>Crew positions</td>
<td>78 - 94</td>
<td></td>
</tr>
<tr>
<td>HMMWV heavy variant</td>
<td>M1097</td>
<td>Crew positions</td>
<td>85-100</td>
<td></td>
</tr>
<tr>
<td>Abrams tank</td>
<td>M1A2</td>
<td>In vehicle</td>
<td>93-117</td>
<td></td>
</tr>
<tr>
<td>Army Helicopters</td>
<td>Model</td>
<td>Location</td>
<td>Sound Level (dB)</td>
<td></td>
</tr>
<tr>
<td>Blackhawk</td>
<td>UH-60A</td>
<td>Pilot</td>
<td>106</td>
<td></td>
</tr>
<tr>
<td>Apache</td>
<td>YAH-64</td>
<td>Pilot</td>
<td>104</td>
<td></td>
</tr>
<tr>
<td>Weapons</td>
<td>Model</td>
<td>Location</td>
<td>Sound Level (dB)</td>
<td></td>
</tr>
<tr>
<td>9mm pistol</td>
<td>M9</td>
<td>Shooter</td>
<td>157</td>
<td></td>
</tr>
<tr>
<td>Grenade</td>
<td>M26</td>
<td>At 50 ft</td>
<td>164</td>
<td></td>
</tr>
<tr>
<td>MAAWS recoiless rifle</td>
<td>M3</td>
<td>Gunner</td>
<td>190</td>
<td></td>
</tr>
</tbody>
</table>

NOTES: In flight, helicopter crews wear helmets with integral hearing protectors.

HMMWV, high-mobility multipurpose wheeled vehicle; MAAWS, multi-role anti-armor anti-personnel weapon system.

Source: Noise and military service. The national academies press. Washington

We can conclude that hazardous noise levels have been and are present in many military settings and that certain military personnel from World War II to the present have exhibited hearing thresholds while in the military that are typical of noise-induced hearing loss. Extensive collections of data on sound pressure levels produced by equipment and activities in military settings are available from World War II to the present. However, because of the changing nature of assignments in the military, the unpredictable aspects of military training and combat, the intermittent nature of many military noise exposures, and the sporadic use of hearing protection while in the military, these data do not provide a sufficient basis to estimate cumulative noise exposures over the course of military service. Nowadays, we are not able to determine the probability of acquiring noise-induced hearing loss associated with service in the military, or in specific branches of the military, for a given individual. The probability of acquiring noise-induced hearing loss can only be determined precisely with well-controlled, longitudinal epidemiological studies.

**Tinnitus in Military**

Tinnitus is the hearing of sound when no external sound is present. While often described as a ringing, it may also sound like a clicking, hiss or roaring. Tinnitus may mainly occur following a single exposure to high-intensity impulse noise, long-term exposure to repetitive impulses, long-term exposure to continuous noise, or exposure to a combination of impulses and continuous noise [32]. Other reasons such as ototoxic compounds administration, diseases or external traumatic factors could also lead to tinnitus disorder. Persistent tinnitus causes anxiety and depression. Moreover, psychological problems such as depression, anxiety, sleep disturbances and concentration difficulties are directly linked to tinnitus disorder [33].

Only a few studies reported on the prevalence of tinnitus in samples of military populations that might be considered representative, and information on noise exposures is limited. One of it presented a random sample of 2,200 Israeli soldiers, a 14 percent reported having tinnitus [34], but no information was available on individuals’ levels of noise exposure. Among
the 204 officers in a Swedish infantry regiment, a 17 percent reported experiencing tinnitus [35]. The rate ranged from a 11 percent among the officers 30 years of age and younger to a 24 percent among those over age 50. The prevalence of tinnitus was 26 percent among those who had been exposed to heavy-weapons fire and significantly higher than the rate of 5 percent among those exposed to gunfire from only smaller arms [35].

In an age-stratified random sample of 699 Finnish Army officers, a 34 percent reported experiencing “occasional” tinnitus and a 9 percent continuous tinnitus [36]. Tinnitus was significantly correlated with exposure to a greater number of weapons impulses and, in contrast to the Swedish study, more strongly correlated with exposure to impulses from small caliber than large-caliber weapons [36]. The scale of the exposure was not directly specified, but it was estimated to be more than 200,000 impulses for at least some officers.

Taken together, these data suggest that hearing loss is associated with a higher prevalence of tinnitus depending on noise doses. However, present data do not allow to reach conclusions regarding the specific number or proportion of service members, overall or in specific branches or occupational groups, who report that tinnitus began or was exacerbated by noise exposure during military service.

Pharmacotherapy for Noise-induced hearing loss

Currently there is no established treatment for patients and it is limited to prevention and follow-up. However, some clinical and military trials have been carried out for temporary threshold shift, in which administration of antioxidant nutritional supplements, such as magnesium, N-acetyl-cysteine (NAC) or vitamins, before moderate noise exposure showed some beneficial effects [37, 38].

Magnesium efficacy was tested in a double-blind study. Test subjects were given either 122 mg of magnesium or a placebo for 10 days and thereafter subjected monaurally to 90 dB SPL of white noise for 10 minutes. Increase of 20 dB threshold shifts was found in 28% of the placebo group compared to 12% in the magnesium supplemented group [39]. Moreover, Attias et al. conducted a double-blind placebo controlled study on army recruits and concluded that recruits who had magnesium supplementation had less frequent noise-induced hearing loss compared to the placebo group [40]. These 300 army recruits underwent basic military training where they were subjected to shooting range noises of an average peak level of 164 dB and <1 ms duration with the use of ear plugs which reduced noise level by about 25 dB. The hearing loss was defined as a threshold >25 dB hearing loss in at least 1 frequency and it was found that threshold shift was higher in placebo group (11.5%) as opposed to the participants in the magnesium group (1.2%).

NAC acts as a reactive oxygen species scavenger and is postulated to reduce noise-induced hearing loss by reducing the exposure of the cochlea to reactive oxygen species, as observed in some laboratory studies [41]. Clinical trials using NAC remain presently controversial and inconclusive. Whereas Kramer and collaborators published that NAC treatment didn’t protect temporary thresholds shifts after noise exposure [42], Kopke and collaborators demonstrated that NAC significantly reduced auditory threshold shifts and DPOAE changes in military subjects undergoing routine weapons training [43].

Finally, Ebselen is a potent glutathione peroxidase mimic and neuroprotectant. It also has strong activity against peroxynitrite, a super reactive oxygen species. It reduces cytochrome c release from mitochondria and nuclear damage during lipid peroxidation [44]. Since it acts as a catalyst, low does maybe sufficient to prevent or treat noise induced hearing loss [45]. Phase II trials are currently in progress to determine the efficacy of oral ebselen.

In conclusion, accumulating evidence demonstrated that antioxidants and free radical scavengers may serve as effective therapeutic agents to block the activation of death mechanisms induced by noise exposure.

COMPETING INTERESTS

Author declares no competing interests.

AUTHOR CONTRIBUTIONS

The author confirms being the sole contributor of this work and approved it for publication.

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