Hearing in old age – epidemiological and etiological aspects

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Introduction
The number of elderly persons is increasing all over the world. This strong demographic trend will affect our societies profoundly. Since the largest relative increase is expected to occur in the group of very old persons, over 80 years of age, the demands on the society and on relatives in terms of health- and social care are considerable. The influence of ageing on the auditory function is pronounced. Age-related hearing loss (ARHL), or presbyacusis, belongs to one of the three most frequently reported chronic health problems in old age, and is also the most prevalent cause of hearing loss. Communication with other people, and the auditory system is the most important link in communication. ARHL has often a devastating effect on the social contacts and quality of life of many elderly people.

Audiometric features of ARHL
In presbyacusis the pure tone audiogram shows a high frequency, symmetrical, sensorineural hearing loss in a vast majority of the cases. The configuration is either gently sloping or steeply sloping. In one audiometric variety the pure tone loss is relatively flat over the entire frequency range. Speech perception is in most elderly persons fairly well preserved. The single most important factor causing impaired speech perception and a reduction of the ability to understand speech, is peripheral hearing loss [¹]. Central auditory dysfunction and cognitive problems can explain pronounced auditory problems in relatively small groups of elderly persons [², ³]. It is difficult to distinguish the separate roles of cognitive and perceptual processes in speech recognition in older people [⁴, ⁵].

Epidemiological aspects of ARHL
The extent of ARHL, its progress with increasing age, its distribution within different populations, and calculations of rehabilitative needs can be studied using epidemiological methods. There are many epidemiological studies emanating from post-industrialized countries describing the auditory function in various ages, including mapping ARHL among elderly people. There are many examples of epidemiological studies, published during the last decade or slightly more. Many studies have been carried out in the Nordic countries [⁶-¹¹]. Other important studies come from other European countries [¹², ¹³], from North America [¹⁴, ¹⁵], Australia [¹⁶], and Japan [¹⁷], just to mention some investigations. The results of the investigations from western countries coincide reasonably well. Prevalences of hearing loss of different extent (according to pure tone averages across the frequencies 0.5 to 4 kHz) among adults and elderly persons are presented in table 1. Hearing loss is uncommon among young adults, mild hearing loss is common among middle-aged persons, and moderate to
severe hearing loss is common in old age.

High frequency hearing in different studies, not screened for otological disease and exposure to noise, are fairly similar, but a difference of up to 10 dB or more at some frequencies exists. Threshold values of the frequencies 2, 4, and 6 kHz, selected from seven epidemiological studies, are presented in table 2. Screened studies, consisting of subjects without otological disease and exposure to noise, also coincide rather well, but with some variability. Unscreened populations have 5–15 dB poorer threshold values than screened. Reports from longitudinal studies on the decline of hearing with increasing age are somewhat contradictory. The deterioration is more pronounced in the high frequency range (1–2 dB per year) compared to the low frequency range (1 dB per year or less). In some studies there is a tendency that men have the most pronounced decline in the mid frequencies (1–2 kHz per year).

**ARHL in a global perspective**

There is a wealth of information regarding hearing in elderly people in western countries, but the phenomenon of increasing elderly populations is worldwide. A majority of the world’s population lives in countries that are in a process of rapid industrialization and economical development. Some of these countries have very large populations, like China, India and Brazil. In spite of the fact that a large proportion of the world’s population lives in these countries, information of many health sectors, like audiology services, is

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**Table 1.** Percentages of hearing loss of different severity in the adult general population, a compilation of two epidemiological studies (Davis, 1995; J?nsson & Rosenhall, 1998). The hearing capacity is defined according to the pure tone average across the frequencies 0.5 across 4 kHz. Severe to profound hearing loss: 65 dB HL; Moderate hearing loss: 40–64 dB HL; Mild hearing loss: 20–39 dB HL.

<table>
<thead>
<tr>
<th>Age, years</th>
<th>Percentages with hearing loss according to pure tone averages 0.5 – 4 kHz</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>65 dB HL</td>
</tr>
<tr>
<td>18 – 30</td>
<td>0.0</td>
</tr>
<tr>
<td>31 – 40</td>
<td>0.7</td>
</tr>
<tr>
<td>41 – 50</td>
<td>0.3</td>
</tr>
<tr>
<td>51 – 60</td>
<td>0.9</td>
</tr>
<tr>
<td>61 – 70</td>
<td>2.3</td>
</tr>
<tr>
<td>71 – 80</td>
<td>4.0</td>
</tr>
<tr>
<td>≥81</td>
<td>13.0</td>
</tr>
<tr>
<td>All, 18 – &gt; 80 y</td>
<td>1.6</td>
</tr>
</tbody>
</table>

**Table 2.** Approximate pure tone levels in dB HL at 2, 4, and 8 kHz, collected from seven epidemiological studies, at 70 – 80 years of age, and 80+ years

<table>
<thead>
<tr>
<th>Age, years</th>
<th>2 kHz, dB HL</th>
<th>4 kHz, dB HL</th>
<th>8 kHz, dB HL</th>
</tr>
</thead>
<tbody>
<tr>
<td>70 – 80 years</td>
<td>Men</td>
<td>35 – 40</td>
<td>60 – 65</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>30</td>
<td>40 – 45</td>
</tr>
<tr>
<td>80+ years</td>
<td>Men</td>
<td>50 – 55</td>
<td>70 – 75</td>
</tr>
<tr>
<td></td>
<td>Women</td>
<td>40 – 45</td>
<td>55 – 60</td>
</tr>
</tbody>
</table>

**Table 3.** Prevalence of hearing loss in the Sichuan province in China (Liu et al, 2001), after screening for hearing loss by a questionnaire (left columns). Right columns: results from a national survey of self-assessed hearing in Sweden, 2005 (Statistics Sweden).

<table>
<thead>
<tr>
<th>Sichuan province China</th>
<th>National survey Sweden</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>Prevalence %</td>
</tr>
<tr>
<td>15 – 29</td>
<td>1.34</td>
</tr>
<tr>
<td>30 – 44</td>
<td>3.6</td>
</tr>
<tr>
<td>45 – 59</td>
<td>6.1</td>
</tr>
<tr>
<td>60+</td>
<td>12.8</td>
</tr>
</tbody>
</table>
insufficient. Interesting results from countries undergoing rapid industrialization at present have been presented recently.

Liu et al.\textsuperscript{18} have studied the hearing in the Sichuan province in China. In the first stage all inhabitants of the referral sites answered a questionnaire, and the presence of hearing loss was established (Table 3). In the later stages those who had reported hearing loss were examined by otologists and tested with pure tone audiometry.

The results presented by Liu et al.\textsuperscript{11,18} are compared with questionnaire results from Sweden in 2005 (Statistics Sweden). The results from China cannot be compared directly with those from Sweden as well as from other western countries. The questionnaires are different, and the composition of the age groups is not the same. However, the prevalence of hearing loss in China seems to be lower than self-assessed hearing problems in Sweden. At least, it does not exceed that of Europe. It would be of great interest to compare audiometric results from China with those performed in other countries. Results regarding elderly persons from Brazil indicate the same tendency; hearing in elderly people is equal to, or even better, in Rio de Janeiro compared to Sweden.\textsuperscript{11,19}

Gender differences in hearing in old age

There is a clear tendency that elderly women have better high frequency hearing than men of the same age. The difference between the genders is apparent at the frequencies 2 – 8 kHz, and is most pronounced at the frequency 4 kHz. For 70 – 80 year-olds the male excess of hearing loss at 4 kHz is about 10 – 20 dB according seven investigations. For those who are 80 years or older the gender difference is somewhat smaller, 5 to 15 dB. In the low frequency area there is a tendency for women to present with somewhat poorer thresholds than men.\textsuperscript{20} Exposure to noise differs between genders, and is believed to be responsible for a substantial part of the difference in the high frequency region. It has also been suggested that a biological difference between men and women can explain some of the difference in hearing acuity. Cruickshanks et al.\textsuperscript{15} reported that the gender difference remained after adjusting for age, education, noise exposure, and occupation. However, the controversy over the gender difference is not entirely solved. No significant gender difference has been found in animal studies.\textsuperscript{21} Studies of isolated, relatively noise–free populations are also contradictory. In Easter Island hearing is, in general, well preserved, and there were no significant differences in hearing between men and women.\textsuperscript{22} The hearing of Amerindians in Surinam was similar to that seen in industrial societies, and there was a clear gender difference.\textsuperscript{23}

Histopathological correlate to ARHL

Inner ear morphology–histopathology in old age has been studied in inner ears from humans as well as research animals. The most pronounced degeneration of the two types of cochlear hair cells is found in the basal coil.\textsuperscript{24} The outer hair cells (OHCs) show a patchy degeneration most pronounced in the apical and basal coils. The degeneration of inner hair cells (IHC), and also the nerve fibres, is predominantly confined to the basal coil.\textsuperscript{25} Over the age of 50 the degeneration of OHCs is more severe than the degeneration of IHCs. Atrophy of the spiral ganglion and the nerves in the osseous spiral lamina is another finding. ARHL is related to dysfunction on a cellular level. Derangement of the hair bundles, and formation of giant cilia are findings often seen in aged human inner ears. Lipofuscin, also called the pigment of ageing, is assumed to be waste products of lysosomal activity.\textsuperscript{26}

Histopathological studies also have demonstrated degenerative changes in the central auditory nervous system. Loss of neurons has been found in the cochlear nuclei and in the central auditory pathway with increasing age. This concept has, however, been challenged, and presbyacusis–related histopathological alterations have been focussed in the inner ear.\textsuperscript{27}

Schuknecht and Gacek\textsuperscript{28} described six types of ARHL, based on morphological findings correlated to audiometric measurements. Sensory presbyacusis exhibits a loss of sensory cells as well as supporting and neuronal cells mostly in the basal coil of the cochlea. The hearing impairment in this type of ARHL is a high frequency loss with relatively normal speech perception. Neural presbyacusis is characterised by a loss of neurons more than sensory cells. This neuronal loss is seen in the entire spiral ganglion but is more severe in the basal turn. The speech recognition is reduced in relation to the pure tone audiogram. In strial (or metabolic) presbyacusis a patchy atrophy of the stria vascularis is seen in the entire cochlea but most pronounced in the middle and apical turns. The audiometric pattern is a flat hearing loss. The
progression of the hearing loss is slow and the speech recognition ability good. Gates and Mills have suggested that strial presbyacusis is the type of cochlear degeneration has the strongest correlation to ageing, and thus represents “true” ARHL. Mixed presbyacusis includes combinations of the types described above. Cochlear conductive presbyacusis is a hypothetical disturbance of the mechanics of the spiral ligament, but like indeterminate presbyacusis, no morphological changes are detectable with light microscopy.

Causes of ARHL

ARHL is a very multifactorial process, and it is in general impossible to separate the different constituents from each other. There is a multitude of both intrinsic and extrinsic causes. Biological ageing, resulting in loss of hair cells and neurons that cannot be regenerated, is an obvious cause of intrinsic ARHL. It is conceivable that this process starts relatively late, and that part of ARHL in advanced age can be explained in this way. It has been proposed that the term presbyacusis should be restricted to the hearing loss caused by normal ageing processes.

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There is increasing evidence that genetic mechanisms are of great importance to determine the hearing in old age. Some persons have excellent hearing in advanced age, while others get ARHL–like, gently sloping hearing loss already when they are middle-aged. Genetic factors are apparent in familial ARHL, an entity with relatively early onset of hearing loss, and affecting many members of the same family. They reported that familial aggregations are correlated to both sensory and strial ARHL phenotypes, but the heritibility seemed to be greater for the strial than the sensory type. They also reported that the genetic effect was more pronounced for women than for men, who seem to have a more multifactorial ARHL, with mixed genetic and acquired ARHL. A similar effect has been described in a study of male twins. In their study the environmental effect became more pronounced with increasing age. Liu et al studied 4164 persons of all ages with hearing loss, living in the Sichuan province in China. In about 8% there was a genetic cause of the impaired hearing.

Erway et al identified and mapped the first gene for ARHL in the C57BL/6J inbred mice, the Ahl (Age–related Hearing Loss) gene. This recessive gene, located on chromosome 10, is a major contributor to early, progressive ARHL in many other inbred mouse strains. Mice with the Ahl gene were more sensitive to NIHL in young age, before the gene had an effect on hearing in old age. In a twin study of elderly men Garringer et al have identified another chromosomal region that could be linked to ARHL. A region suggestive of linkage was found on chromosome 3q, in the same region where the DFNA18 locus resides. This locus has earlier been shown to cause progressive hereditary hearing loss.

Based on a temporal bone material, a mitochondrial genetic model of ARHL has been proposed. Mitochondrial DNA deletions(mtDNA) have been associated to ARHL. A majority of elderly subjects with hearing loss showed a 4977 bp deletion, a deletion that was present in only half of the subjects in a control group with normal audiograms. Production of reactive oxygen species has been shown to be one cause of this mtDNA damage.

Exposure to noise is the most important extraneous noxious factor that affects the hearing in old age, above all for men. Occupational exposure to extensive noise is of considerable importance, but also every–day noise exposure. Although the levels of environmental noise often are not directly harmful to hearing, the effects of such noise in the long run are not known. The effect of noise is equivocal. The interactions between noise–induced hearing loss (NIHL) and ARHL are complex, difficult to determine, and poorly understood. One important issue that has been discussed is if there is an increased or decreased sensitivity to noise with age. The traditional model to assess NIHL in older persons assumes that presbyacusis adds to permanent noise–induced threshold shift. This additive model is embraced by ISO 1999, and is favoured by earlier literature. The formula suggested by ISO means that the total hearing loss is the sum of age–related hearing loss and NIHL minus a compression factor that is used when
threshold shifts exceeds 20–25 dB. However, this basically additive model has been challenged. It has been proposed that the additive model overestimates the interaction between noise and ageing. In the subadditive model the deterioration caused by presbyacusis is reduced within the noise frequencies in noise-damaged ears. In this model, noise injury is less likely in the aged ear with presbyacusis. However, contradictory results have also been published. In some mouse models it has been found that ageing, with or without hearing loss, increased the sensitivity to NIHL by a genetic disposition, proposed that the additive model overestimates the interaction between noise and ageing. In the subadditive model the deterioration caused by presbyacusis is reduced within the noise frequencies in noise-damaged ears. In this model, noise injury is less likely in the aged ear with presbyacusis. However, contradictory results have also been published. In some mouse models it has been found that ageing, with or without hearing loss, increased the sensitivity to NIHL by a genetic disposition.

It has been proposed that the ageing process is different in a noise-damaged cochlea than in a "pristine" one. According to this hypothesis, the deterioration is less pronounced in the NIHL-frequencies (3–6 kHz) in a noise-damaged ear, than in an ear not influenced by noise. In adjacent frequencies (especially 2 kHz) there is an increased progress of ARHL. This could be explained by an increased vulnerability of the regions on the basilar membrane bordering to the NIHL-region. However, this model of a combined sub- and superadditive effect of noise has been challenged recently.

Hearing loss in elderly people can also be related to ototraumatic events other than noise. Such factors include influence from ototoxic agents and environmental ototoxic insults, smoking, and head trauma. Health factors e.g. otological disease and cardiovascular disorders have been related to the presence of ARHL. Other factors that interfere with the hearing capacity in old age are socio-economic state, level of education, and the state of health.

Hearing in old age–measures and prospects

Efforts to improve the auditory communication in old age are important and can be expected to result in better quality of life for elderly persons. The alleviation of age-related hearing handicap includes aural rehabilitation with hearing aid fitting and communication training programmes. Procedures and devices especially designed for elderly are needed for more efficient rehabilitation. The accessibility is important. ARHL is often combined with other handicaps, such as visual impairment, dementia, and immobility. The synergistic effect of multiple handicaps is important to notice. It has been reported in that elderly persons in institutions have poorer hearing compared to those living in their own homes. Is this a result of ailing health, or is the probability that elderly with hearing loss develop pseudodementia, or both?

Prevention and treatment of ARHL is a challenging issue. The most important measure of prevention involves noise reduction. Prevention of noise-induced hearing loss must start early in life and not shortly before retirement. It is, however, not an easy task to change attitudes of young persons for the benefit of better hearing, and less tinnitus, decades later. Other preventive measures are also possible. Suggestion of suitable diets, food additives (e.g. antioxidants), prevention of cardiovascular disease, changes of life-style e.g. smoking, are possible preventive measures. In the future there is an exciting possibility of gene therapy for individuals with genetic, early and progressive ARHL by gene-silencing techniques. A probable candidate is RNA interference a technique that was awarded the Nobel Price in Medicine and Physiology in 2006. The benefits of prevention, and eventually treatment, of ARHL are extraordinary both in humanitarian and economical terms.

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