

# Trajectories of Hearing From Childhood to Adulthood

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**Objectives:** The Dunedin Multidisciplinary Health and Development Study provides a unique opportunity to document the progression of ear health and hearing ability within the same cohort of individuals from birth. This investigation draws on hearing data from 5 to 13 years and again at 45 years of age, to explore the associations between childhood hearing variables and hearing and listening ability at age 45.

**Design:** Multiple linear regression analyses were used to assess associations between childhood hearing (otological status and mid-frequency pure-tone average) and (a) age 45 peripheral hearing ability (mid-frequency pure-tone average and high-frequency pure-tone average), and (b) age 45 listening ability (listening in spatialized noise and subjective questionnaire on listening experiences). Sex, childhood socioeconomic status, and adult IQ were included in the model as covariates.

**Results:** Peripheral hearing and listening abilities at age 45 were consistently associated with childhood hearing acuity at mid-frequencies. Otological status was a moderate predicting factor for high-frequency hearing and utilization of spatial listening cues in adulthood.

**Conclusions:** We aim to use these findings to develop a foundational model of hearing trajectories. This will form the basis for identifying precursors, to be investigated in a subsequent series of analyses, that may protect against or exacerbate hearing-associated cognitive decline in the Dunedin Study cohort as they progress from mid-life to older age.

**Key words:** Childhood, Life course, Listening ability, Mid-life, Peripheral hearing, Trajectory.

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## INTRODUCTION

Changes in auditory function and perception occur across the life course through congenital and genetic disorders, with additional influences of trauma, diseases, and environmental factors (World Health Organization 2021). Much can be learned about the causative and preventative factors underlying changes

in hearing or development of hearing loss from longitudinal studies (Russ et al. 2018).

Little is known about the long-term effects of childhood hearing and ear health on adulthood. Only a handful of studies have tracked the same individuals across the life course from birth to adulthood. One example of such a study, the Trøndelag Health Study, measured the hearing of 32,786 children at primary school, and then again in the same individuals at 20 to 56 years of age (Aarhus et al. 2015, 2020). Hearing loss associated with recurrent ear disease in childhood showed subsequent negative associations with hearing ability in middle adulthood between the ages of 40 and 56 years (more so than in young adulthood, aged 20 to 40 years), and marginally increased the risk of developing adult hearing loss. There was a deterioration in hearing thresholds of 1.1 dB per year (95% confidence interval [CI] = 0.7 to 1.6) for chronic middle ear infections, and deterioration of 0.9 dB per year in hearing thresholds (95% CI = 0.2 to 1.7) for acute middle ear infections (Aarhus et al. 2015). In the same cohort (Aarhus et al. 2020), associations were found between chronic suppurative otitis media in childhood, and sensorineural hearing loss (5.58 fold increased risk; 95% CI = 3.78 to 8.22) and tinnitus (2.62 fold increased risk; 95% CI = 1.07 to 6.41) in mid-adulthood (mean age = 52 years), along with other diseases like chronic sinusitis (3.13 fold increased risk) and cardiovascular disease (1.38 fold increased risk). Another study (Pearson et al. 2015) used retrospective data on childhood infections and pure-tone audiometry results collected from 333 members (aged 61 to 63 years) of the original Newcastle Thousand Families Study cohort in the UK. A range of common infections (such as tonsillitis, bronchitis, otorrhea, and severe respiratory infections) that occurred in the first year of life were associated with increased likelihood of impaired hearing function in adulthood.

According to the Developmental Origins of Health and Disease hypothesis (Barker 2007) there is evidence suggesting that a myriad of early life factors can impact cognitive (Grove et al. 2017) and hearing functioning (Barrenäs et al. 2003) in adulthood. Identifying potentially modifiable early childhood factors could therefore be crucial in changing the trajectory of hearing decline across mid-life through to older age (Dawes et al. 2022).

Throughout the early developmental period, recurrent otitis media in childhood can also impact on central auditory processes, especially around the recovery of binaural integration capabilities (Hall et al. 1995) and functional aspects of spatial processing (Tomlin & Rance 2014). This subsequently can lead to listening difficulties (Gravel et al. 1996; Klausen et al. 2000), behavioral challenges like hyperactivity and inattention (Silva et al. 1986; Bennett et al. 2001; Welch & Dawes 2007), and educational disadvantages in reading and verbal comprehension and expression (Silva et al. 1986; Schilder et al. 1993; Klausen et al.

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2000; Bennett et al. 2001; Welch & Dawes 2007; Williams & Jacobs 2009). The Developmental Origins of Health and Disease hypothesis suggests a further link to age-related hearing decline and its secondary effects on, for example, understanding speech in quiet and noisy environments, depression, poorer balance, falls, hospitalizations, and earlier mortality, as well as social challenges, such as social isolation, loss of autonomy, impaired driving ability, and financial decline (Davis et al. 2016).

The Dunedin Multidisciplinary Health and Development Study (“the Dunedin Study”) provides a unique opportunity to address the gaps in the literature by documenting the progression of ear health and hearing ability from early childhood to mid-life within a longitudinal cohort with a high retention rate. In line with models of life-course health development (Halfon et al. 2014, 2017; Russ et al. 2018) it is important to understand changes in hearing ability across the lifespan (World Health Organization 2021) and the burden of poor hearing over time (Wilson & Tucci 2021) in a context that simultaneously considers age, hearing, lifestyle, cognitive, and psychosocial factors (Halfon & Hochstein 2002; Pronk et al. 2019).

Findings from the Dunedin Study cohort can shed light on the biological and environmental factors influencing trajectories of hearing health and “pace of aging” (Elliott et al. 2021; Wertz et al. 2021), as well as the secondary consequences of uncorrected hearing impairment, which for example, contribute to a reported 8.2% risk for dementia (weighted fraction attributable to the population) (Livingston et al. 2020). The importance of tackling ear and hearing health is gradually being acknowledged as a public health problem (Monasta et al. 2012; World Health Organization 2021).

The Dunedin Study has hearing data from 5 to 13 years and again at 45 years of age. Hearing measures were included in the data collection at age of 45 years to enable an investigation of the relationship between sensory and cognitive health as the longitudinal study progresses. It is an ongoing study, thus results reported here will form the foundation for future explorations of childhood hearing and associated factors influencing adult hearing trajectories. It is acknowledged that minimal conclusions on longer-term outcomes can be drawn, as much of the cohort at age 45 is yet to show signs of presbycusis (age-related hearing loss). However, we aim to capitalize on the availability of hearing data at mid-life, which is sparse in the literature. Moreover, most studies are concerned with “clinical” hearing loss rather than hearing ability, and here we aim to consider peripheral hearing ability, the detection of sound; and listening ability, which engages higher-order cognitive processes. It is hypothesized that adult peripheral hearing and listening abilities will be strongly associated with childhood hearing acuity, but potentially mediated by otological status. These findings will enable the narrowing of dependent variables down to a few that can form the basis for future analyses into other life-course factors that contribute to hearing acuity in mid- and later-life.

## MATERIALS AND METHODS

### Sample

Participants are members of the Dunedin Multidisciplinary Health and Development Study (“Dunedin Study”), a longitudinal investigation of health and behavior in a population-representative birth cohort of 1037 individuals (91% of eligible births; 52% male) born between April 1, 1972 and March 31,

1973 in Dunedin, New Zealand (NZ). The longitudinal study was established at age 3 years based on residence in the Otago province (Poulton et al. 2015, 2022). Assessments were conducted at birth and at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and most recently at age 45, when 94% (938 of the surviving 997) participants still alive took part. Each study member was brought to the research unit for a day of interviews and examinations. Study participants are primarily of New Zealand European ethnicity (approximately 93%). Written informed consent was obtained from participants, and the study was approved by the New Zealand Health and Disability Ethics Committee.

A total of 908 study members (91% of the remaining surviving members of the cohort,  $N = 997$ ) participated in the collection of objective hearing data at age 45, which took place at the Dunedin Multidisciplinary Health and Development Research Unit facility at the University of Otago, Dunedin, New Zealand. A total of 938 study members (94%) participated in the collection of subjective hearing data at age 45, most of which were collected at the research unit, but some of which were collected in the field (due to exceptional circumstances that warranted personal visits from a study investigator). Missing data (0.8 to 1.5%) were attributed to the inability to achieve a response due to equipment malfunction and declining to answer questions, for objective and subjective measures, respectively. The total numbers of final valid data points are reported below for each measure.

Attrition analyses were conducted using childhood IQ, childhood socioeconomic status (SES), a psychopathology “*p*-factor” (using ages 18 to 45 data), Adverse Childhood Events, and a polygenic score for educational attainment, to determine whether participants in the phase 45 data collection were representative of the original cohort. No significant differences were found between those who were seen at age 45 and the full cohort, including data from those who are deceased, for all measures; except childhood IQ and *p*-factor where those who were deceased (by age 45) had significantly lower and higher scores, respectively. These results strongly suggest that the current Dunedin Study cohort is still representative of the original members.

All independent and dependent variables included in the statistical analyses are described later and are detailed in Table 1 and in the Supplemental Digital Content, <http://links.lww.com/EANDH/B439>. These consist of peripheral hearing ability and listening ability measures from childhood (ages 5, 7, 9, and 11 years) and adulthood (age 45 years).

### Childhood Hearing Measures

**Otological Status** • Otological and audiological examinations were conducted at ages 5, 7, and 9 years on each child according to an assessment protocol, using otomicroscopy, impedance audiometry, and pure-tone audiometry.

- a. Otological examinations were performed by a medical practitioner specifically trained in the use of an otomicroscope. Based on the otoscopy results the condition of each ear was categorized as: (i) normal, (ii) fluid present, (iii) ventilation tubes present, (iv) acute otitis media, or (v) other abnormalities.
- b. Impedance audiometry (tympanometry) was performed by an audiometrist trained specifically for the study.

**TABLE 1.** List of variables included in the analyses, the number of available data points (% of cohort at the time), and the assessment ages that contributed to the data marked by “x”

Variable	N (%)	Phase (Age)									
		Birth	3	5	7	9	11	13	15	38	45
Childhood hearing (independent) variables											
Childhood otological status	961 (92.7%)			x	x	x					
(Ln) Childhood mid-frequency pure-tone average	926 (89.3%)				x	x	x				
Adult hearing and listening (dependent) variables											
(Ln) Mid-frequency pure-tone average	891 (89.4%)										x
(Ln) High-frequency pure-tone average	896 (89.9%)										x
LiSN-S low-cue speech-reception threshold	902 (90.5%)										x
LiSN-S high-cue speech-reception threshold	902 (90.5%)										x
LiSN-S talker advantage	902 (90.5%)										x
LiSN-S spatial advantage	902 (90.5%)										x
LiSN-S total advantage	902 (90.5%)										x
Speech, spatial, and qualities of hearing	923 (92.6%)										x
Control variables											
Childhood socioeconomic status	1030 (99.3%)	x	x	x	x	x	x	x	x		
Adult IQ (processing speed)	904 (90.7%)										x

Percentage of cohort size at the time was calculated out of  $N = 1037$  for childhood data, and  $N = 997$  for adulthood data.

LiSN-S, Listening in Spatialized Noise-Sentences test. (Ln), transformed using a natural logarithm

This was performed on each ear and classified into three basic types of pressure-compliance functions (types A, B, C) (Jerger 1970).

- c. Hearing thresholds—obtained by pure-tone audiometry performed by the audiometrist—were collected at 500, 1000, 2000, and 4000 Hz. A mean hearing threshold was calculated across these frequencies for each ear at each age. Those meeting criteria for surgical treatment received ventilation tubes, and this was incorporated as a factor in the otological status classification.

These three sources of information (otoscopy, impedance audiometry, pure-tone audiometry) across three occasions (time points) were combined to provide an objective measurement of the cumulative history of otitis media with effusion (OME)—referred to as childhood otological status. This otological status variable (available for 962 study members) consists of seven classification categories ranging from severe to healthy (Share et al. 1986; Bennett et al. 2001; Welch & Dawes 2007; Dawes & Welch 2010).

- Classification 1 ( $n = 53$ , 5.5%) described children with bilateral ventilation tubes for OME with proven hearing loss, observed on one or more occasions.
- Classification 2 ( $n = 45$ , 4.7%) described study members with persistent bilateral OME (with or without ventilation tubes), but who did not have bilateral hearing loss exceeding 25 dB on at least one occasion.
- Classification 3 ( $n = 26$ , 2.7%) had the same criteria as classification 2, except that study members had persistent unilateral OME.
- Classification 4 ( $n = 118$ , 12.3%) described study members with transient uni/bilateral OME measured on at least one occasion.
- Classification 5 ( $n = 138$ , 14.3%) described study members with no evidence of OME but with scar tissue present, indicative of some degree of perforation to the tympanic membrane between ages 5 and 9 years.
- Classification 6 ( $n = 449$ , 46.7%) made up the majority of the study members who had type-C tympanograms on at least one occasion, but no OME or B-type tympanograms.

- Classification 7 ( $n = 133$ , 13.8%) described study members who always had type-A tympanograms in both ears.

**Pure-Tone Audiometry** • A separate variable called the childhood mid-frequency pure-tone average (MF-PTA) was calculated by averaging thresholds for frequencies at 1000, 2000, and 4000 Hz, across the ages 7, 9, and 11 years. Pure-tone audiometry was conducted, for the majority of the study members, in a quiet room at the Dunedin Study offices. A small proportion had their hearing tested at school, at home, at a psychologist service, or overseas. Pure tones were presented through an Interacoustics AS7 portable audiometer via a pair of TDH39 headphones.

A number of factors, including different testing protocols and equipment (Universal UAL 10 audiometer), led to the exclusion of hearing data at age 5 from subsequent analyses. The 500 Hz frequency measurements were also excluded due to high levels of variability.

As the data were analyzed to describe the hearing loss at age of 45 years and to look at the relationship between childhood ear disease and hearing loss, a detailed investigation of the childhood hearing levels was undertaken to exclude children with substantial congenital hearing losses. This revealed three study members with elevated bilateral pure-tone hearing thresholds across the ages of 7, 9, and 11 years. Elevated thresholds for two of these children were related to middle ear pathology (43 dB associated with persistent OME; and 46 dB associated with a type-C tympanogram). These were considered to be transient conductive hearing losses, and thus these participants were retained in the final sample. One study member had profound hearing loss in childhood (86 dB HL), and thus their data were subsequently excluded from all analyses. Participant numbers for each assessment are reported with this participant excluded.

#### Adult Hearing Measures at Age 45 (Outcome Measures)

Hearing assessments at age 45 years encompassed a combination of measures that were generally considered as indications of peripheral hearing ability or acuity (pure-tone audiometry) and listening or auditory processing ability, which engages higher-order cognitive processes; each is described in detail



later. Study members were asked if they wore hearing aids, and if so, for which ear and for how long. Those with hearing aids ( $n = 6$ ) were asked to remove them for audiometric testing but were asked to wear their hearing aids for the listening in spatialized noise test, and for completing the questionnaire. Hearing assessments were completed in a sound-treated room at the research unit.

**Otoscopy** • Otoscopic examination was conducted using a Heine Light emitting diode otoscope with disposable specula. The amount of wax occlusion was observed. Note “occlusion” refers to the occluded view of the tympanic membrane through the otoscope, and not the degree of wax impaction or functional occlusion. Any abnormalities observed in the study member’s outer or middle ear were also noted.

**Pure-Tone Audiometry** • Pure-tone audiometry was conducted in a sound-attenuating booth (350 Series Maxi Audiology Booth, IAC Acoustics) which meets the standard for maximum permissible ambient sound pressure levels (Technical Committee ISO/TC 43 2010). Pure-tones were presented via Sennheiser HAD 300 headphones, connected to the Interacoustics Callisto Suite (configured to the Interacoustics OtoAccess database on an HP Envy laptop). Pure-tone thresholds were determined for frequencies ranging from 500 to 12,500 Hz. The starting presentation intensity was at 40 dB HL for normal hearing study members, and 60 dB HL for hearing aid users. A “No Response” was noted if the maximum intensity for a particular frequency was reached and this level failed to elicit a response. A “No Attempt” was noted if study members did not complete the task due to equipment failure. Maximum intensity thresholds set by the Interacoustics Callisto software to align with safety requirements (Interacoustics A/S 2020) were: 120 dB HL for 500, 1000, 2000, and 4000 Hz; 105 dB HL for 8000 Hz; 95 dB HL for 12,500 Hz.

A mid-frequency PTA (MF-PTA) was calculated by averaging thresholds for three frequencies: 1000, 2000, and 4000 Hz. There had to be at least two available threshold measurements to calculate the average. A high-frequency PTA (HF-PTA) was calculated by averaging 8000 and 12,500 Hz. There had to be two available threshold measurements, or at least one available threshold and one “no response,” for a valid data point. If the second threshold was a “no attempt,” the data point was invalid. The beginning of a degree of decline in high-frequency hearing is likely in mid-life, thus a measurement of “no response” (at the intensity limits of the audiometer) was considered a valid data point.

PTAs calculated for the right and left ears separately were further averaged to form a single MF-PTA and a single HF-PTA. There were no instances where study members had only one valid data point for one of their ears. Thus, valid MF-PTAs and HF-PTAs included study members with average thresholds available for both ears; and missing data were recorded for those who did not have available thresholds for either ear. Of the 908 study members assessed on pure-tone audiometry, there were 891 valid data points for MF-PTAs, and 896 valid data points for HF-PTAs (Table 1).

**Listening in Spatialized Noise** • Central auditory processing was evaluated using the Australian version of the listening in spatialized noise-sentences test (LiSN-S) (developed by the National Acoustics Laboratories, and distributed by Phonak, Switzerland) (Cameron & Dillon 2007). The LiSN-S (software on Microsoft Surface Pro tablet) produces a three-dimensional

auditory environment through headphones (Sennheiser 215 headphones configured to Mini PCM2704 external sound card). The test consists of target sentences superimposed with distractor (masker) stories. Maskers differed in perceived spatial location ( $0^\circ$  or  $\pm 90^\circ$  azimuth), and speaker identity (same as or different from the target speaker, both female). There are four different test conditions: (1) different speaker at  $\pm 90^\circ$  azimuth; (2) same speaker at  $\pm 90^\circ$  azimuth; (3) different speaker at  $\pm 0^\circ$  azimuth; and (4) same speaker at  $\pm 0^\circ$  azimuth. Of the 908 study members assessed on the LiSN-S, there were 902 valid data points (Table 1).

Five possible outcome scores are listed and described later.

1. Speech-reception thresholds from a low-cue condition, which represents performance in the most difficult auditory environment, where the masker speaker was the same as the target speaker, and the masker was presented at  $0^\circ$  azimuth, in the same location as the target speaker.
2. Speech-reception thresholds from a high-cue condition, which represents performance in the easiest listening environment, where the masker speaker was different from the target speaker, and the masker was presented at  $90^\circ$  azimuth.
3. “Talker advantage” score measures the benefit gained when the masker sentences are spoken by a different person than the target.
4. “Spatial advantage” score measures the benefit gained when the masker is presented from a different direction than the target.
5. “Total advantage” score is a combination of talker and spatial advantages.

Figure 1 (Cameron & Dillon 2007) illustrates the four test conditions and details how spatial, talker, and total advantage scores are calculated. A full description of the test can be found in the original paper by Cameron and Dillon (2007). The LiSN-S has good reliability. Test-retest data from an Australian sample aged 12 to 60 years of age (Cameron et al. 2011) showed small improvements between repeat tests for the four conditions ( $p = 0.01 - < 0.001$ ), but no significant changes were shown for advantage scores. Test-retest data also did not differ as a function of age ( $p = 0.178 - 0.980$ ). Similar results were found with a North American sample of adolescents and young adults who were given the North American version of the LiSN-S (Cameron et al. 2009), with a significant difference in test-retest scores for only the low-cue SRT. The three “advantage” measures make use of difference scores to minimize the effects of between-listener variation (such as in linguistic skills and general cognitive ability) on LiSN-S performance (Cameron & Dillon 2007), thus giving a better reflection of within-listener variation as individuals are tracked longitudinally. The five LiSN-S scores are analyzed for between-sex differences and overall descriptive results, but only the three “advantage” scores are analyzed as part of the childhood-to-adulthood association.

**Questions on Hearing** • Lastly, the study members were asked three key questions from the 12-item Spatial and Qualities of Hearing Scale (SSQ12) (Noble et al. 2013); only three items were used due to time constraints. These three questions cover different and complex listening conditions (in noise, multiple speakers, and localization of sound), and were selected from the 12-item scale based on our own unpublished data, as well as other studies, showing that older

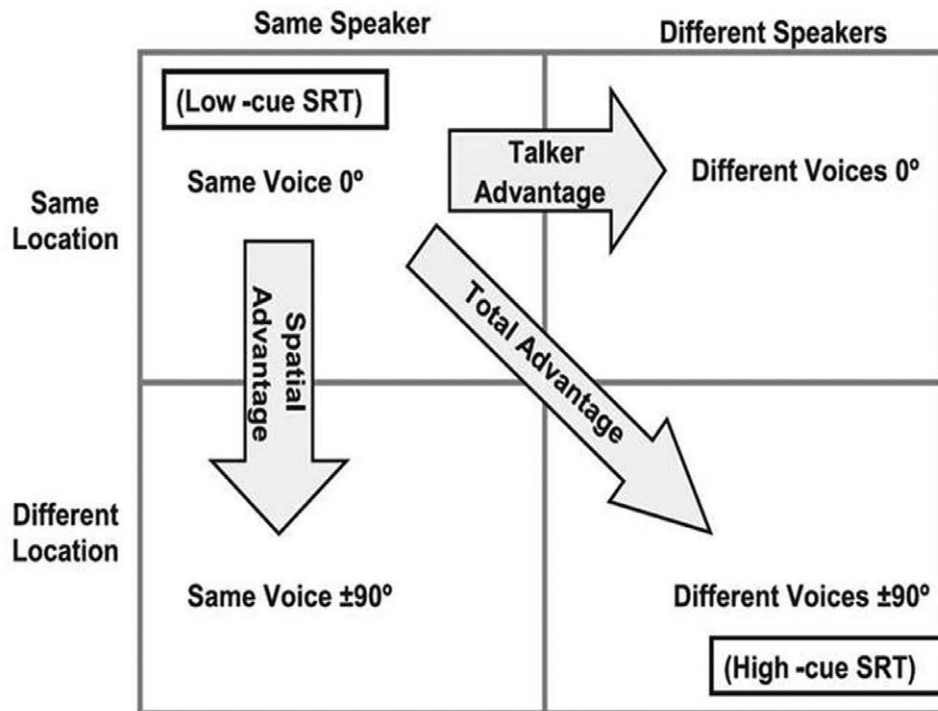


Fig. 1. Illustration of LiSN-S speech-reception threshold and advantage measures. “LiSN-S SRT and advantage measures” from Cameron and Dillon (2007), development of the LiSN-S (*Ear & Hearing*, 28(2)), page 199, Figure 1. Permission granted by H. Dillon on December 10, 2021. LiSN-S indicates listening in spatialized noise-sentences test.

individuals with mild cognitive impairment or the beginnings of subjective memory complaints (preclinical dementia) demonstrate significant difficulties in these areas compared with their healthy peers (Idrizbegovic et al. 2011; Rahman et al. 2011; Ghannoum et al. 2018; Sardone et al. 2019; Humes 2020; Jayakody et al. 2020). This item selection was made with the continuity of the Dunedin Study in mind, assuming that repeated administration of these questions might reveal changes in subjective listening experiences associated with cognitive decline as the study members age.

On a scale of 0 (not at all) to 10 (perfectly),  $N = 938$  study members were asked if they could: (1) follow a conversation in a group in a busy restaurant, (2) follow a conversation in a group where the conversation switches between speakers, and (3) tell immediately where a dog is by hearing its bark. Cronbach  $\alpha$  showed strong ( $\alpha = 0.813$ ) internal reliability between the three questions; thus, an average subjective listening score was calculated (Table 1). Valid data points ( $N = 923$ ) included study members who answered all three questions.

### Control Variables

**Childhood Socioeconomic Status** • Childhood SES was calculated as the average of the highest SES level of either parent of each study member, assessed repeatedly across the eight-time points from birth to 15 years old. This method was used because measurement of SES at a single point early in life does not reflect cumulative exposure to low SES during childhood. Standard New Zealand occupationally-based indices were used to classify SES (Elley & Irving 1985). By controlling for childhood SES, we aimed to mitigate potential bias in the data associated with the effects of socioeconomic disparities on accessing hearing healthcare (Boss et al. 2011).

**Adult Intelligence** • The Wechsler Adult Intelligence Scale (Fourth Edition; WAIS-IV) (Wechsler 2008) was administered to participants at age 45 years, yielding four IQ indices (verbal comprehension, perceptual reasoning, working memory, and processing speed) as well as an overall IQ score. Each of the indices is standardized to a mean of 100 with  $\pm 15$  SD.

The “adult IQ” variable included in these analyses is specifically the processing speed index, which is a measure of mental speed, and is sensitive to changes in other cognitive factors. Developmental changes in processing speed have been shown to mediate working memory, and together account for half of age-related developmental increases in fluid intelligence (Fry & Hale 1996). By controlling for processing speed, we aimed to reduce the impact of cognitive factors on LiSN-S task performance, so that the results more accurately reflect central auditory processing skills without the influence of varying cognitive abilities. In the Wechsler Adult Intelligence Scale, processing speed is comprised of visual-based subtests: coding and symbol searching. The rationale for using a visual-based IQ index is to remove the confounding bias of hearing ability which may be present in indices that include verbal-based subtests.

### Statistical Analyses

The three PTA variables (one mid-frequency in childhood; and one mid- and one high-frequency in adulthood) were highly skewed toward lower values (i.e., better hearing), as is expected for generally healthy hearing thresholds that do not indicate clinical hearing loss. Natural log transformations were applied to all the pure-tone outcome variables to normalize their distributions, and these are used in all subsequent analyses. All other variables were normally distributed and hence were not transformed before analyses.

Independent samples *t* tests were conducted to investigate between-sex differences in all adult hearing and listening ability outcome measures. Multiple linear regression analyses were used to assess associations between childhood hearing (two independent variables) and (a) age 45 peripheral hearing ability (two dependent variables, separately), and (b) age 45 listening ability (four dependent variables, separately). Sex, childhood SES, and adult IQ were included in the model as covariates. IBM SPSS Statistics, v28 was used. Effect sizes were reported as the square of the correlation coefficient (*R*<sup>2</sup>), due to the inclusion of standardized and nonstandardized normally distributed variables in the regression models. *R*<sup>2</sup> indicates the proportion of variance shared by the two independent variables, and size interpretations are based on guidelines relevant to psychological research (Funder & Ozer 2019).

## RESULTS

### Overview of Hearing and Listening at Age 45

Peripheral hearing ability at age 45 is illustrated in the audiogram and accompanying box plots illustrating the distribution of thresholds in Figure 2. On average, study members were still within the normal range for hearing at mid-frequencies (1000, 2000, and 4000 Hz) (*M* = 11.44, *SD* = 7.75), however, early stages of decline in hearing acuity were evident at higher frequencies (8000 and 12,500 Hz) (*M* = 26.51, *SD* = 15.41). Variation in hearing ability increased markedly for these higher frequencies,

as some study members retained their high-frequency hearing, while others showed some hearing loss, especially at 12,500 Hz. There was also a significant difference in hearing acuity at mid- and high-frequencies between males and females (Table 2).

Listening ability at age 45 was measured via performance on the LiSN-S, as well as subjective reports of everyday listening abilities. Figure 3 illustrates the means and SDs of LiSN-S performance from the Dunedin Study cohort (*N* = 902) in comparison with extrapolated 45-year-old normative data from a cohort of 18 to 60-year-olds (*N* = 96) (Cameron et al. 2011). These projected values were provided by the developers of the LiSN-S test (Cameron & Dillon, National Acoustics Laboratory, Australia, 22nd January 2018). They fitted a three-segment piecewise linear function to their data and pinpointed 45 years.

Table 2 summarizes the independent *t* test results for differences between male and female study members' hearing and listening abilities at age 45. Figures 4A–F specifically illustrate the distribution of listening ability scores between males and females. Aside from the LiSN-S speech-reception thresholds in the low-cue test condition (Fig. 4A), and the LiSN-S talker advantage score (Fig. 4D), all other measures of adult hearing and listening abilities showed significant sex differences (Figs. 4B, C, E, F).

The many significant differences in hearing acuity and listening between males and females led to adjusting for sex (along with childhood SES and adult IQ) in subsequent regression analyses. Sex differences were regarded as a confounding

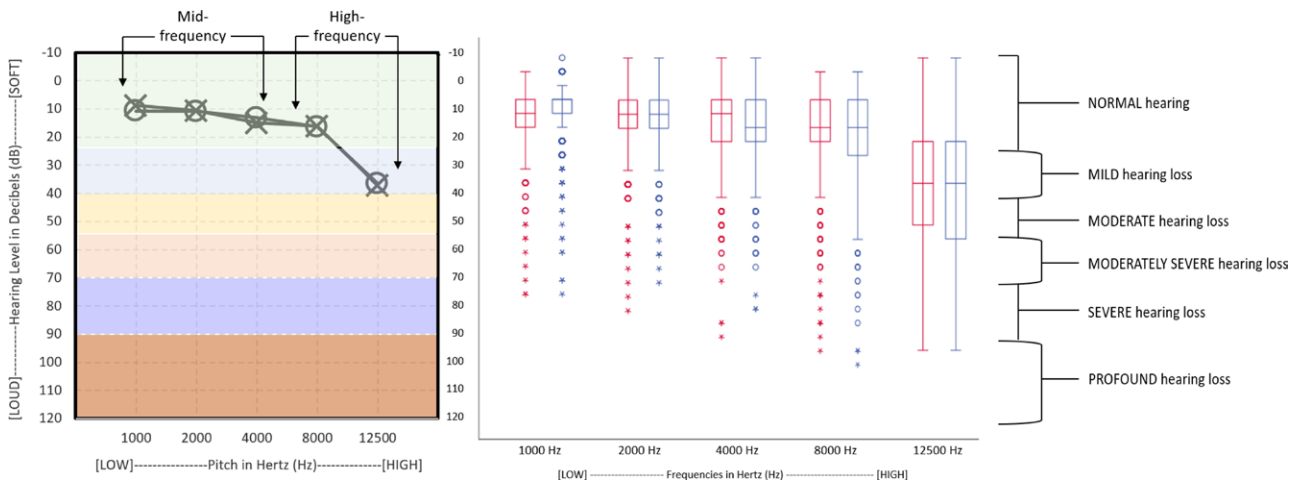


Fig. 2. Pure-tone audiogram (left) and box plots (right) illustrating mean hearing thresholds at age 45 for right (O and red) and left (X and blue) ears separately, across frequencies 1000, 2000, 4000, 8000, and 125,000 Hz.

**TABLE 2. Summary of independent *t* test results for differences between male and female study members' hearing and listening abilities at age 45**

Adult Hearing and Listening (Dependent) Variables	Male, M (SD); N	Female, M (SD); N	<i>t</i> (df)	<i>p</i>	<i>d</i>
(Ln) Mid-frequency pure-tone average	2.31 (0.68); 449	2.22 (0.57); 442	-2.24 (865.9)	0.026	0.143
(Ln) High-frequency pure-tone average	3.16 (0.67); 453	3.01 (0.70); 443	-3.35 (894.0)	<0.001	0.022
LiSN-S low-cue speech-reception threshold	-1.11 (1.44); 454	-1.11 (1.31); 448	0.05 (900.0)	0.480	0.000
LiSN-S high-cue speech-reception threshold	-14.52 (3.16); 454	-15.33 (2.79); 448	-4.03 (900.0)	<0.001	0.272
LiSN-S talker advantage	5.18 (2.14); 454	5.24 (2.00); 448	0.47 (900.0)	0.318	0.029
LiSN-S spatial advantage	12.28 (2.67); 454	13.00 (2.31); 447	4.34 (885.5)	<0.001	0.288
LiSN-S total advantage	13.40 (2.84); 454	14.20 (2.48); 448	4.53 (900.0)	<0.001	0.300
Speech, spatial, and qualities of hearing	7.28 (1.74); 463	7.50 (1.69); 460	2.01 (921.0)	0.022	0.128

*d*, Cohen *d* effect size (mean difference/pooled SD); *M*, mean; *N*, sample size; *p*, statistical significance value; *t* (df), *t*-statistic (degrees of freedom).

LiSN-S, listening in spatialized noise-sentences test. (Ln), transformed using a natural logarithm

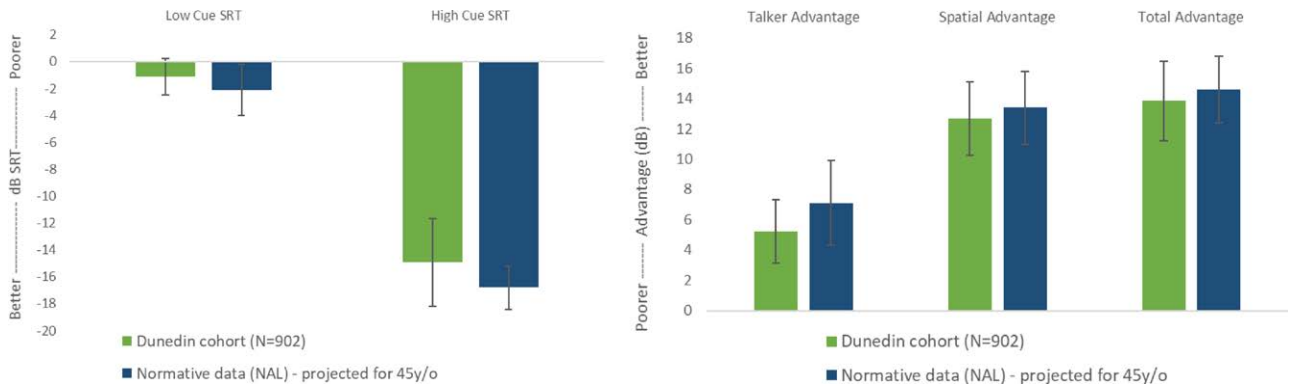


Fig. 3. Bar charts illustrating the means and SDs of LiSN-S performance from the Dunedin Study cohort (N = 902) in comparison with extrapolated 45-yr-old normative data from a cohort of 18 to 60-yr-olds (N = 96) (Cameron et al. 2011), as provided by the developers of the LiSN-S test from the National Acoustics Laboratory, Australia. LISN-S indicates listening in spatialized noise-sentences test.

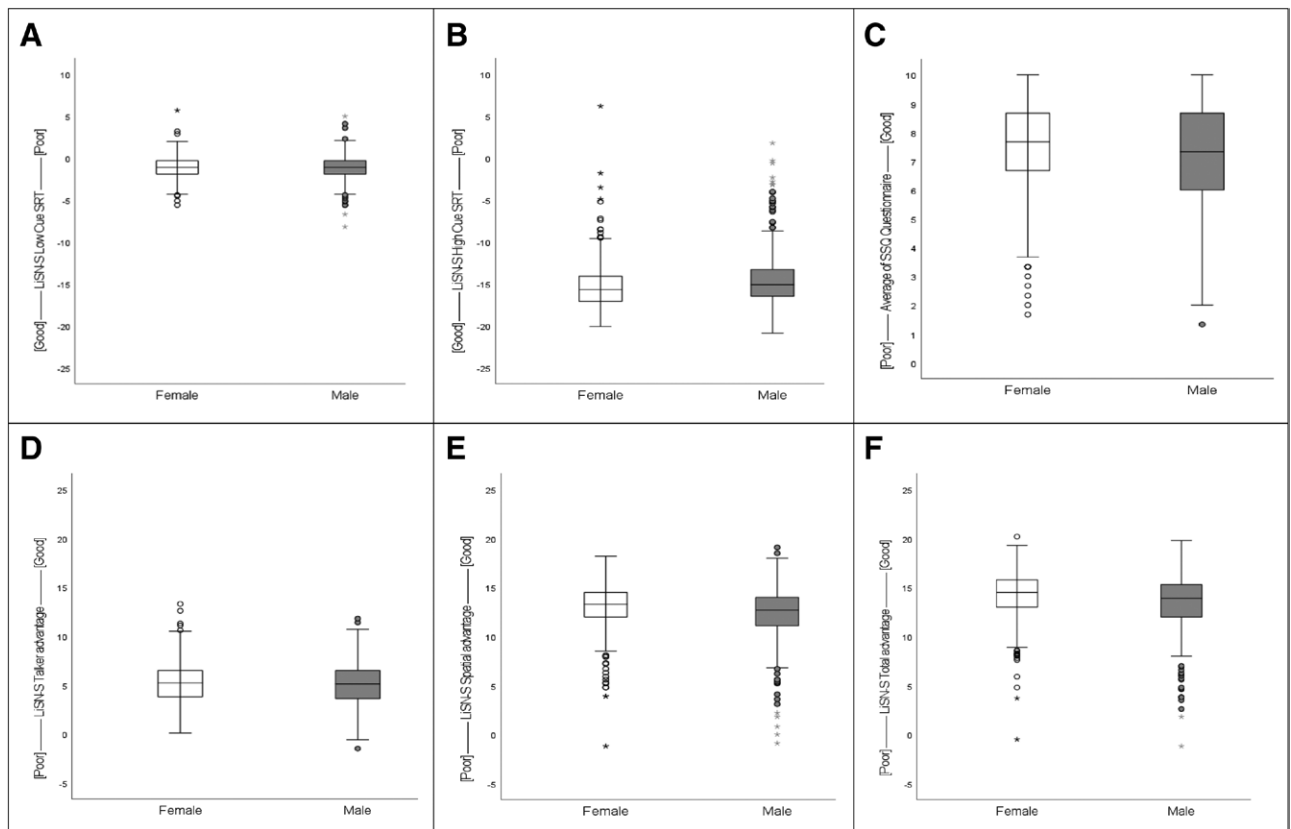


Fig. 4. Box plots illustrating the distribution of data from females and males separately. A-E for the five LiSN-S scores, and F for the average score from the SSQ subjective listening questionnaire. Taken together, these measures represent listening ability at age 45. LiSN-S, Listening in Spatialized Noise-Sentences test; SSQ, Spatial and Qualities of Hearing Scale.

factor for the purposes of keeping in line with the focus on hearing trajectories from childhood to adulthood. However, future investigations into modifiable protective and risk factors will take hearing and listening differences between males and females into consideration.

**Associations Between Childhood Hearing and Peripheral Hearing Ability at Age 45**

A MF-PTA (of 1000, 2000, and 4000 Hz), and an HF-PTA (of 8000 and 12,500 Hz), were used as measures of peripheral

hearing abilities at age 45. As mentioned, these two outcome variables were highly skewed, as the majority of study members did not yet show (age-related) hearing decline at age 45. Natural log transformations were applied, and  $\beta$  coefficients were exponentiated and converted into percentage changes for interpretation.

Results from multiple linear regressions revealed that poorer peripheral hearing abilities at age 45 were associated with poor pure-tone hearing thresholds in childhood (Table 3). Lower PTAs indicate better hearing. A unit decrease in childhood mid-frequency hearing thresholds (better hearing) was associated

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**TABLE 3. Associations between childhood hearing and peripheral hearing ability at age 45**

Models of Peripheral Hearing Ability (Adjusted for Sex, Childhood SES, and Adult IQ)		n	$\beta$ (95% CI)	*Percentage Change (95% CI)	p
(Ln) Adult mid-frequency pure-tone average	Childhood otological status	799	-0.044 (-0.045 to 0.010)	-4.30 (-4.40 to 1.01)	0.209
	(Ln) Childhood mid-frequency pure-tone average	799	0.356 (0.316–0.469)	42.76 (37.16–59.84)	<0.001
$F_{(5)} = 30.23; p < 0.001; R^2 = 0.160$					
(Ln) Adult high-frequency pure-tone average	Childhood otological status	805	-0.166 (-0.104 to -0.042)	-15.30 (-9.88 to -4.11)	<0.001
	(Ln) Childhood mid-frequency pure-tone average	805	0.132 (0.074–0.247)	14.11 (7.68–28.02)	<0.001
$F_{(5)} = 19.17; p < 0.001; R^2 = 0.107$					

Results are presented from multiple linear regression models, adjusted for sex, childhood socioeconomic status, and adult IQ. Model statistics, including effect size  $R^2$  with interpretation, are reported. Standardized  $\beta$  coefficients and 95% CIs are reported. Effect sizes ( $R^2$ ) are considered small-medium (0.1–0.2) for both models (Funder & Ozer 2019).

\*Percentage increase or decrease is calculated by the formula:  $(\exp(\beta) - 1) \times 100$ —to account for the natural log transformations of the peripheral hearing ability variables.

CIs, confidence intervals; LiSN-S, Listening in Spatialized Noise-Sentences test; (Ln), transformed using a natural logarithm; SES, socioeconomic status.

**TABLE 4. Associations between childhood hearing and listening ability (LiSN-S Advantage Scores and SSQ) at age 45**

Models of Listening Ability (Adjusted for Sex, Childhood SES, and Adult IQ)		n	$\beta$ (95% CI)	p
Adult LiSN-S talker advantage	Childhood otological status	810	-0.028 (-0.133 to 0.060)	0.457
	(Ln) Childhood mid-frequency pure-tone average	810	-0.114 (-0.689 to -0.147)	0.003
$F_{(5)} = 6.10; p < 0.001; R^2 = 0.037$				
Adult LiSN-S spatial advantage	Childhood otological status	810	0.087 (0.028–0.250)	0.014
	(Ln) Childhood mid-frequency pure-tone average	810	-0.261 (-1.463 to -0.843)	<0.001
$F_{(5)} = 23.94; p < 0.001; R^2 = 0.130$				
Adult LiSN-S total advantage	Childhood otological status	810	0.084 (0.025–0.261)	0.018
	(Ln) Childhood mid-frequency pure tone average	810	-0.265 (-1.584 to -0.921)	<0.001
$F_{(5)} = 24.41; p < 0.001; R^2 = 0.132$				
Adult SSQ	Childhood otological status	813	-0.005 (-0.081 to 0.071)	0.895
	(Ln) Childhood mid-frequency pure tone average	813	-0.160 (-0.682 to -0.257)	<0.001
$F_{(5)} = 14.01; p < 0.001; R^2 = 0.080$				

Effect sizes ( $R^2$ ) are considered very small (<0.1) for the LiSN-S talker advantage, and the SSQ. Effect sizes are considered small-medium (0.1–0.2) for the LiSN-S spatial advantage and total advantage (Funder & Ozer 2019). Results are presented from multiple linear regression models, adjusted for sex, childhood socioeconomic status, and adult IQ. Model statistics, including effect size  $R^2$  with interpretation, are reported. Standardized  $\beta$  coefficients and 95% CIs are reported.

CIs, confidence intervals; LiSN-S, Listening in Spatialized Noise-Sentences test; (Ln), transformed using a natural logarithm; SES, socioeconomic status; SSQ, Spatial and Qualities of Hearing Scale.

with (i) 42.76% better hearing (decrease of 1.64 dB) in adult mid-frequency hearing; and (ii) 14.11% better hearing (decrease of 1.18 dB) in adult high-frequency hearing. Although poorer otological status—the objective measurement of the study member's cumulative history of OME—did not significantly predict peripheral mid-frequency hearing ability in adulthood, it did significantly predict adult peripheral hearing ability for the higher frequencies. Each unit of increase in OME severity was associated with 15.30% worsening (increase of 1.21 dB) of high-frequency pure-tone thresholds at age 45.

Childhood hearing variables accounted for 16.0% of the variance in adult mid-frequency peripheral hearing ability, although mostly this was held up by childhood mid-frequency hearing ability. In comparison, childhood hearing variables accounted for 10.7% of the variance in adult high-frequency peripheral hearing ability. The small-to-medium effect sizes of both regression models suggest there are other childhood and life-course factors that may affect hearing acuity in mid-life, to be explored in future analyses.

### Associations Between Childhood Hearing and Listening Ability at Age 45

Listening abilities at age 45 were measured using the LiSN-S test and average response across the three questions regarding speech, spatial, and quality of listening experiences in everyday life. These outcome variables were normally distributed, thus no transformations were applied to the data. Table 4 presents outcome scores of the SSQ and the LiSN-S advantage scores only, allowing for the individual differences in linguistic and

cognitive abilities to be controlled for, and for a clearer evaluation of the study members' abilities to use spatial and voice cues to aid speech understanding central auditory processing).

Results from multiple linear regressions revealed that childhood hearing variables accounted for the most variance in the LiSN-S spatial total advantage scores (13.0% and 13.2%, respectively). These two models of listening ability showed small-to-medium effect size and narrow 95% CIs. The models were relatively weaker for the LiSN-S talker advantage and the SSQ ( $R^2 < 0.1$ ) (Table 4), and thus significant associations are interpreted with caution.

Poorer otological status in childhood and poorer childhood mid-frequency hearing thresholds were significantly associated with the ability to utilize cues, specifically spatial cues, to aid task performance on the LiSN-S. The results indicate that the LiSN-S spatial advantage measure may be the most informative for future analyses—using childhood hearing variables as mediators to explore other life-course factors that may affect hearing acuity in mid-life.

## DISCUSSION

The Dunedin Multidisciplinary Health and Development Study provides a unique opportunity to analyze childhood ear health and hearing acuity data against adult abilities from the same cohort. The key findings from this analysis highlighted that measures of peripheral hearing and listening abilities at age 45 were consistently associated with childhood hearing at mid-frequencies. Otological status was predictive of high-frequency hearing and select listening abilities, specifically the use of



spatial cues to aid listening to competing information in a three-dimensional auditory environment.

We should note that the exploratory nature of this paper's objectives meant that we decided to investigate what we term "peripheral hearing ability" and "listening ability" as separate factors to try to tease apart processes that required either minimal (e.g., pure-tone threshold detection) or substantial higher-order cognitive input, respectively. We recognize that pure-tone thresholds can involve cognitive processes and thus correlate with aspects of auditory cognition (Lin et al. 2011, 2013), but this changes with age as other factors can influence listening and auditory ability. By including adult IQ as a covariate, we aimed to adjust for effects of individual cognitive differences on hearing performance. Pure-tone audiometry may well predict listening ability, at least at mid-life, but it will be interesting to see if this correlation changes as the cohort ages.

Given that this is an ongoing longitudinal study, the reported associations are not meant to substantiate any definitive effects. Instead, we will further discuss the results within the context of how these preliminary analyses may be applied to further investigations of the impact of hearing and listening on trajectories of cognitive decline as the Dunedin Study progresses from mid-life to older age.

### The Dunedin Study Cohort at Age 45—Peripheral Hearing and Listening Abilities Compared With Other Populations

The Dunedin Study cohort had slightly poorer mid-frequency hearing thresholds, yet slightly better high-frequency hearing thresholds, compared with the international standard statistical distribution of hearing thresholds related to age and gender (ISO 7029, 2017). According to this standard, the expected median for an MF-PTA (1000, 2000, 4000 Hz) for males and females aged 50 years are 7 and 5.3 dB HL, respectively. Mean mid-frequency thresholds for the Dunedin cohort were 12.2 and 10.6 dB HL, for males and females respectively. The expected median for a high-frequency average (8000, 12,500 Hz) for males and females aged 50 years are 29.5 and 27 dB HL, respectively (ISO 7029, 2017). Mean values for the cohort were 28.5 and 24.5 dB HL, for males and females respectively.

In comparison with the Dunedin Study cohort, the Baltimore Longitudinal Study of Aging cohort reported a poorer MF-PTA of 20.1 dB HL for their 50-year-old male population (Brant & Fozard 1990). A large Japanese study reported MF-PTAs of 11.3 dB HL and 9.67 dB HL for males and females aged 40 to 49 years, respectively (Wasano et al. 2021). In both studies, pure-tone measurements on frequencies as high as 12,500 Hz were not conducted. Across all comparisons, the pattern of sex differences was consistent with that observed within the Dunedin cohort—where thresholds in women were on average close to 3 dB better (lower) than in men, for mid-frequency hearing acuity at middle age. Subsequent investigations into potential preventative and causative factors may shed more light onto what underlies these gender differences, may they be biological, occupational, or lifestyle-related factors.

Figure 3 illustrates the LiSN-S performance of the Dunedin Study cohort in comparison with extrapolated 45-year-old normative data from a cohort of 18 to 60-year-olds from Australia

(Cameron et al. 2011). The Dunedin cohort consistently performed slightly poorer than the estimated Australian 45-year-old values for both low- and high-cue LiSN-S test conditions; this difference was also evident for their advantage scores based on utilizing talker and/or spatial differences to aid listening. It is possible that the extrapolation approach utilized by Cameron and Dillon over-estimated LiSN-S performance at mid-life by extrapolating linearly based on a limited range of ages present in the Australian cohort and the relatively small sample sizes within each age bracket.

### Hearing Influences From Childhood

The effect sizes, while consistent with Aarhus et al. (2015) and Pearson et al. (2015), show that the influence of childhood ear and hearing status on adult hearing levels was quite small (<2 dB effects on PTA). It is acknowledged that these effects are not large enough to be detected by a clinical audiogram (which has a test-retest reliability range of  $\pm 5$  dB), as much of the Dunedin cohort at age 45 are yet to show signs of presbycusis (age-related hearing loss). However, it is still relevant to note that the strongest and most consistent associative factor with adult peripheral hearing ability (at both mid- and high-frequencies) in the present study was mid-frequency hearing acuity in childhood. Otological status had an additional small-to-medium-sized effect on high-frequency hearing only at age 45. This is consistent with research showing that all forms of otitis media cause high-frequency hearing loss that persists beyond recovery of the disease (Hunter et al. 1996; Margolis et al. 2000; Ryding et al. 2002). High-frequency hearing is also more susceptible to the effects of aging, with and without the compounding influences of ototoxic drugs and noise exposure (Lough & Plack 2022). This also aligns with evidence that adult hearing thresholds significantly reduce across the entire auditory frequency range only if there is hearing loss in childhood caused by OME, chronic suppurative otitis media, or after recurring acute otitis media (Aarhus et al. 2015, 2020). Together, these results indicate that poor otological health to the point when it is compounded by measurable, clinical hearing loss, can have a negative effect on adult hearing thresholds, emphasizing the importance of prioritizing the prevention and treatment of poor otological health in childhood (Browning et al. 2010; World Health Organization 2021).

As seen in the handful of studies that have looked at ear and hearing health longitudinally, there are numerous childhood health risk factors that contribute to hearing decline in mid-life, ranging from recurrent/chronic suppurative otitis media (Aarhus et al. 2015, 2020), to infections (Pearson et al. 2015), to birth weight (Barrenäs et al. 2003). It was also interesting to see that both otological status and childhood mid-frequency hearing acuity were associated with listening abilities specifically related to spatial cues, to a moderate extent. There have been reports from the Trøndelag Health Study of adults diagnosed with childhood chronic suppurative otitis media and/or childhood hearing loss after recurrent acute otitis media, who were 2.1 fold significantly more likely to have increased risk of reported dizziness (Aarhus et al. 2016). The suggested permanent effect of inflammatory mediators on the vestibular system is just one possible explanation for the link between ear infections and spatial awareness. The Dunedin Study is in a unique position to extend this exploration into other potentially

modifiable early childhood factors, such as perinatal complications, nutrition, environmental, and other respective health indices, to contribute to knowledge on different trajectories of hearing decline from mid-life through to older age (Dawes et al. 2022). Overall, these findings highlight the importance of acknowledging the complexities of tackling ear and hearing health holistically and “beyond the audiogram” (World Health Organization 2021), and that while screening for ear disease is important at specified time points across the lifespan, it is also crucial to follow-up and address persistent issues that might result in permanent hearing impairment and longer-term consequences (Wilson & Tucci 2021).

### **Impact of Hearing and Listening on Cognition: The Future Contribution From the Dunedin Study**

The importance of considering hearing and its contribution to brain health and aging, from a life-course perspective, is a strong recurring theme in the literature (Davis et al. 1991; World Health Organization 2021). Recently published analyses of data from the Dunedin Study cohort highlight significant associations between faster “Pace of Ageing” and reduced hearing acuity across frequencies and reduced listening performance on the LiSN-S (Elliott et al. 2021). The Pace of Ageing algorithm quantifies and tracks the decline of 19 biomarkers that index cardiovascular, metabolic, renal, immune, dental, and pulmonary systems from ages 26 to 45 years, and can reflect accelerated anatomical and physiological deterioration that is not proportional to chronological age (Belsky et al. 2015). Aside from hearing, increased Pace of Ageing was also linked to a myriad of other factors including cognitive difficulties, signs of advanced brain aging, motor and visual functions, older appearance, and more pessimistic perceptions of aging (Elliott et al. 2021). Significant associations remained after adjusting for sex, body-mass index, smoking, and diagnoses of cancer, diabetes, or heart attack. This highlights the interplay between different lifestyle factors across the lifespan that influence long-term hearing ability, and ultimately, trajectories of age-related cognition (Abel et al. 1990; Sardone et al. 2019; Livingston et al. 2020).

There is accumulating evidence showing that peripheral hearing loss is a substantial indicator for cognitive decline (Lin et al. 2011), with up to 24% increased risk for incident cognitive impairment (Lin et al. 2013), and a reported 8.2% risk for dementia (weighted fraction attributable to the population) (Livingston et al. 2020). Several large-scale studies show associations between hearing acuity loss and increased rates of cognitive decline and risk of cognitive impairment (Humes et al. 2013; Lin & Albert 2014; Wayne & Johnsrude 2015; Peelle & Wingfield 2016; Thomson et al. 2017; Strutt et al. 2020). In adults over 55 years of age (Baltimore Longitudinal Study of Aging, United States), poorer hearing thresholds were significantly associated with lower performance on the Mini-Mental State Exam, free recall memory tasks, and executive functioning (Lin et al. 2011). In older populations aged 70 to 90 years (Sydney Memory and Aging Study, Australia), moderate-to-severe hearing difficulties were associated with poorer cognitive performance specifically in domains of attention, processing speed, and visuospatial ability; as well as an increased risk in developing neurocognitive disorders within a 6-year follow-up period (Strutt et al. 2020). Results from a meta-analysis of 33 studies within the last decade demonstrated that hearing

impairment affected all domains of cognition; the degree of cognitive deficit was significantly associated with degree of hearing impairment and whether or not treatment was provided and accepted (Nixon et al. 2019).

Minimal conclusions on the relationship to cognitive decline can be drawn at this stage from the Dunedin Study, as much of the cohort at age 45 are yet to show signs of presbycusis (clinically relevant age-related hearing loss) and cognitive impairment. The Dunedin Study is unique in its coverage of both hearing acuity and listening ability in mid-life—an important distinction that has different underlying preventative and causative factors and different implications for healthy cognitive aging. Further tracking of peripheral hearing trajectories, listening ability, and cognitive functioning in this cohort as it ages will facilitate continued development of a model of change that includes a wealth of hearing information from childhood as well as other physical, social, and environmental factors, that truly covers the life course. It is currently unclear at what stage during one’s hearing aging trajectory intervention should be implemented. Existing evidence—underpinning the life course and multidisciplinary perspective on aging—supports the reasonable assumption that early treatment of ear health and hearing abilities contributes to better trajectories. However, as demonstrated in the Aging and Cognitive Health Evaluation in Elders study being conducted in the United States (Lin et al. 2023), the success of obtaining significant change in cognition scores as a result of receiving a hearing intervention (audiological counseling and provision of hearing aids), compared with a non-hearing-specific control intervention, very much depends on the number of preexisting risk factors for cognitive decline. Future directions along this line of investigation might consider whether the identification of significant contributing lifestyle factors can facilitate access to earlier points for intervention. Future directions might also consider what sensory-based screening tools could be incorporated into standard healthcare practices to detect initial markers of cognitive decline.

In this article, we have presented a foundational model of hearing trajectories from which to continue exploring and identifying precursors that may protect against, or exacerbate the risk of, hearing decline leading to cognitive decline. We have highlighted the effects of childhood hearing thresholds and otological status on hearing in mid-life. Following the Dunedin Study cohort as they move into more advanced age provides a solid foundation from which to understand the difference between being at-risk and not-at-risk, and how hearing impairment is just one component of the complexities of aging. It also allows for the exploration of approaches to reduce risk and inform rehabilitation strategies and public health interventions.

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## REFERENCES

- Aarhus, L., Homøe, P., Engdahl, B. (2020). Otitis media in childhood and disease in adulthood: A 40-year follow-up study. *Ear Hear*, *41*, 67–71.
- Aarhus, L., Tambs, K., Hoffman, H. J., Engdahl, B. (2016). Childhood otitis media is associated with dizziness in adulthood: The HUNT cohort study. *Eur Arch Otorhinolaryngol*, *273*, 2047–2054.
- Aarhus, L., Tambs, K., Kvestad, E., Engdahl, B. (2015). Childhood otitis media: A cohort study with 30-year follow-up of hearing (The HUNT Study). *Ear Hear*, *36*, 302–308.
- Abel, S. M., Krever, E. M., Alberti, P. W. (1990). Auditory detection, discrimination and speech processing in ageing, noise-sensitive and hearing-impaired listeners. *Scand Audiol*, *19*, 43–54.
- Barker, D. J. P. (2007). The origins of the developmental origins theory. *J Intern Med*, *261*, 412–417.
- Barrenäs, M.-L., Bratthall, A., Dahlgren, J. (2003). The thrifty phenotype hypothesis and hearing problems. *BMJ*, *327*, 1199–1200.
- Belsky, D. W., Caspi, A., Houts, R., Cohen, H. J., Corcoran, D. L., Danese, A., Harrington, H., Israel, S., Levine, M. E., Schaefer, J. D., Sugden, K., Williams, B., Yashin, A. I., Poulton, R., Moffitt, T. E. (2015). Quantification of biological aging in young adults. *Proc Natl Acad Sci U S A*, *112*, E4104–E4110.
- Bennett, K. E., Haggard, M. P., Silva, P. A., Stewart, I. A. (2001). Behaviour and developmental effects of otitis media with effusion into the teens. *Arch Dis Child*, *85*, 91–95.
- Boss, E. F., Niparko, J. K., Gaskin, D. J., Levinson, K. L. (2011). Socioeconomic disparities for hearing-impaired children in the United States. *Laryngoscope*, *121*, 860–866.
- Brant, L. J., & Fozard, J. L. (1990). Age changes in pure-tone hearing thresholds in a longitudinal study of normal human aging. *J Acoust Soc Am*, *88*, 813–820.
- Browning, G. G., Rovers, M. M., Williamson, I., Lous, J., Burton, M. J. (2010). Grommets (ventilation tubes) for hearing loss associated with otitis media with effusion in children. *Cochrane Database Syst Rev*, *10*, CD001801. doi:https://doi.org/10.1002/14651858.cd001801.pub3.
- Cameron, S., Brown, D., Keith, R., Martin, J., Watson, C., Dillon, H. (2009). Development of the North American listening in spatialized noise-sentences test (NA LiSN-S): Sentence equivalence, normative data, and test-retest reliability studies. *J Am Acad Audiol*, *20*, 128–146.
- Cameron, S., & Dillon, H. (2007). Development of the listening in spatialized noise-sentences test (LISN-S). *Ear Hear*, *28*, 196–211.
- Cameron, S., Glyde, H., Dillon, H. (2011). Listening in spatialized noise—Sentences test (LiSN-S): Normative and retest reliability data for adolescents and adults up to 60 years of age. *J Am Acad Audiol*, *22*, 697–709.
- Davis, A., McMahon, C. M., Pichora-Fuller, K. M., Russ, S., Lin, F., Olusanya, B. O., Chadha, S., Tremblay, K. L. (2016). Aging and hearing health: The life-course approach. *Gerontologist*, *56*, S256–S267.
- Davis, A. C., Ostri, B., Parving, A. (1991). Longitudinal study of hearing. *Acta Otolaryngol*, *111*, 12–22.
- Dawes, P., Newall, J., Graham, P. L., Osmond, C., Von Bonsdorff, M. B., Gunnar Eriksson, J. (2022). Early life influences on hearing in adulthood: A systematic review and two-step individual patient data meta-analysis. *Ear Hear*, *43*, 722–732.
- Dawes, P. J. D., & Welch, D. (2010). Childhood hearing and its relationship with tinnitus at thirty-two years of age. *Ann Otol Rhinol Laryngol*, *119*, 672–676.
- Elley, W. B., & Irving, J. C. (1985). The Elley-Irving socio-economic index 1981 census revision. *N Z J Educ Stud*, *20*, 115–128.
- Elliott, M. L., Caspi, A., Houts, R. M., Ambler, A., Broadbent, J. M., Hancox, R. J., Harrington, H., Hogan, S., Keenan, R., Knodt, A., Leung, J. H., Melzer, T. R., Purdy, S. C., Ramrakha, S., Richmond-Rakerd, L. S., Righthart, A., Sugden, K., Thomson, W. M., Thorne, P. R., Moffitt, T. E. (2021). Disparities in the pace of biological aging among middle adults of the same chronological age have implications for future frailty risk and policy. *Nature Aging*, *1*, 295–308.
- Fry, A. F., & Hale, S. (1996). Processing speed, working memory, and fluid intelligence: Evidence for a developmental cascade. *Psychol Sci*, *7*, 237–241.
- Funder, D. C., & Ozer D.J. (2019). Evaluating Effect Size in Psychological Research: Sense and Nonsense. *Adv Methods Pract Psychol Sci*, *2*, 156–168. https://doi.org/10.1177/2515245919847202.
- Ghannoum, M. T., Shalaby, A. A., Farghaly, M., Hamdy, M., Hamdy, H. S. (2018). Central auditory processing findings in a group of cognitively impaired individuals. *Hear Balance Commun*, *16*, 145–154.
- Gravel, J. S., Wallace, I. F., Ruben, R. J. (1996). Auditory consequences of early mild hearing loss associated with otitis media. *Acta Otolaryngol*, *116*, 219–221.
- Grove, B. J., Lim, S. J., Gale, C. R., Shenkin, S. D. (2017). Birth weight and cognitive ability in adulthood: A systematic review and meta-analysis. *Intell*, *61*, 146–158.
- Halfon, N., Forrest, C. B., Lerner, R. M., Faustman, E. M. (2017). Handbook of life course health development. In Neal Halfon, Christopher B. Forrest, Richard M. Lerner, Elaine M. Faustman. *Handbook of Life Course Health Development*. Springer International Publishing. https://doi.org/10.1007/978-3-319-47143-3.
- Halfon, N., & Hochstein, M. (2002). Life course health development: An integrated framework for developing health, policy, and research. *Milbank Q*, *80*, 433–479, iii.
- Halfon, N., Larson, K., Lu, M., Tullis, E., Russ, S. (2014). Lifecourse health development: Past, present and future. *Matern Child Health J*, *18*, 344–365.
- Hall, J. W., Grose, J. H., Pillsbury, H. C. (1995). Long-term effects of chronic otitis media on binaural hearing in children. *Arch Otolaryngol Head Neck Surg*, *121*, 847–852.
- Humes, L. E. (2020). Associations between measures of auditory function and brief assessments of cognition. *Am J Audiol*, *29*, 825–837.
- Humes, L. E., Kidd, G. R., Lentz, J. J. (2013). Auditory and cognitive factors underlying individual differences in aided speech-understanding among older adults. *Front Syst Neurosci*, *7*, 1–16.
- Hunter, L. L., Margolis, R. H., Rykken, J. R., Le, C. T., Daly, K. A., Giebink, G. S. (1996). High frequency hearing loss associated with otitis media. *Ear Hear*, *17*, 1–11.
- Idrizbegovic, E., Hederstierna, C., Dahlquist, M., Nordström, C. K., Jelic, V., Rosenhall, U. (2011). Central auditory function in early Alzheimer's disease and in mild cognitive impairment. *Age Ageing*, *40*, 249–254.
- Interacoustics A/S. (2020). *Instructions for Use - CallistoTM*. Interacoustics A/S.
- Jayakody, D. M. P., Menegola, H. K., Yiannos, J. M., Goodman-Simpson, J., Friedland, P. L., Taddei, K., Laws, S. M., Weinborn, M., Martins, R. N., Sohrabi, H. R. (2020). The peripheral hearing and central auditory



- processing skills of individuals with subjective memory complaints. *Front Neurosci*, 14, 888.
- Jerger, J. F. (1970). Clinical experience with impedance audiometry. *Arch Otolaryngol*, 92, 311–324.
- Klausen, O., Møller, P., Holmefjord, A., Reisæter, S., Asbjørnsen, A. (2000). Lasting effects of otitis media with effusion on language skills and listening performance. *Acta Otolaryngol*, 120, 73–76.
- Lin, F. R., & Albert, M. (2014). Hearing loss and dementia—Who is listening? *Ageing Ment Health*, 18, 671–673.
- Lin, F. R., Ferrucci, L., Metter, E. J., An, Y., Zonderman, A. B., Resnick, S. M. (2011). Hearing loss and cognition in the Baltimore Longitudinal Study of Aging. *Neuropsychology*, 25, 763–770.
- Lin, F. R., Pike, J. R., Albert, M. S., et al. ACHIEVE Collaborative Research Group. (2023). Hearing intervention versus health education control to reduce cognitive decline in older adults with hearing loss in the USA (ACHIEVE): A multicentre, randomised controlled trial. *Lancet*, 402, 786–797.
- Lin, F. R., Yaffe, K., Xia, J., Xue, Q. L., Harris, T. B., Purchase-Helzner, E., Satterfield, S., Ayonayon, H. N., Ferrucci, L., Simonsick, E. M.; Health ABC Study Group. (2013). Hearing loss and cognitive decline in older adults. *JAMA Intern Med*, 173, 293–299.
- Livingston, G., Huntley, J., Sommerlad, A., Ames, D., Ballard, C., Banerjee, S., Brayne, C., Burns, A., Cohen-Mansfield, J., Cooper, C., Costafreda, S. G., Dias, A., Fox, N., Gitlin, L. N., Howard, R., Kales, H. C., Kivimäki, M., Larson, E. B., Ogunniyi, A., Mukadam, N. (2020). Dementia prevention, intervention, and care: 2020 report of the Lancet Commission. *Lancet*, 396, 413–446.
- Lough, M., & Plack, C. J. (2022). Extended high-frequency audiometry in research and clinical practice. *J Acoust Soc Am*, 151, 1944–1955.
- Margolis, R. H., Saly, G. L., Hunter, L. L. (2000). High-frequency hearing loss and wideband middle ear impedance in children with otitis media histories. *Ear Hear*, 21, 206–211.
- Monasta, L., Ronfani, L., Marchetti, F., Montico, M., Brumatti, L., Bavarcar, A., Grasso, D., Barbiero, C., Tamburlini, G. (2012). Burden of disease caused by otitis media: Systematic review and global estimates. *PLoS One*, 7, e36226.
- Nixon, G., Sarant, J. Z., Tomlin, D., Dowell, R. (2019). The relationship between peripheral hearing loss and higher order listening function on cognition in older Australians. *Int J Audiol*, 58, 933–944.
- Noble, W., Jensen, N. S., Naylor, G., Bhullar, N., Akeroyd, M. A. (2013). A short form of the Speech, Spatial and Qualities of Hearing scale suitable for clinical use: The SSQ12. *Int J Audiol*, 52, 409–412.
- Pearson, F., Mann, K. D., Rees, A., Davis, A., Pearce, M. S. (2015). The effect of childhood infection on hearing function at age 61 to 63 years in the Newcastle thousand families study. *Ear Hear*, 36, 185–190.
- Peelle, J. E., & Wingfield, A. (2016). The neural consequences of age-related hearing loss. *Trends Neurosci*, 39, 486–497.
- Poulton, R., Guiney, H., Ramrakha, S., Moffitt, T. E. (2022). The Dunedin study after half a century: Reflections on the past, and course for the future. *J R Soc NZ*, 53, 446–465.
- Poulton, R., Moffitt, T. E., Silva, P. A. (2015). The Dunedin Multidisciplinary Health and Development Study: Overview of the first 40 years, with an eye to the future. *Soc Psychiatry Psychiatr Epidemiol*, 50, 679–693.
- Pronk, M., Lissenberg-Witte, B. I., van der Aa, H. P. A., Comijs, H. C., Smits, C., Lemke, U., Zekveld, A. A., Kramer, S. E. (2019). Longitudinal relationships between decline in speech-in-noise recognition ability and cognitive functioning: The longitudinal aging study Amsterdam. *J Speech Lang Hear Res*, 62, 1167–1187.
- Rahman, T. T. A., Mohamed, S. T., Albanouby, M. H., Bekhet, H. F. (2011). Central auditory processing in elderly with mild cognitive impairment. *Geriatr Gerontol Int*, 11, 304–308.
- Russ, S. A., Tremblay, K., Halfon, N., Davis, A. (2018). A life course approach to hearing health. In N. Halfon, C. B. Forrest, R. M. Lerner, E. M. Faustman (Eds.), *Handbook of Life Course Health Development* (pp. 349–373). Springer International Publishing. [https://doi.org/10.1007/978-3-319-47143-3\\_15](https://doi.org/10.1007/978-3-319-47143-3_15).
- Ryding, M., Kalm, O., Konradsson, K., Prellner, K. (2002). Auditory consequences of recurrent acute purulent otitis media. *Ann Otol Rhinol Laryngol*, 111, 261–266.
- Sardone, R., Battista, P., Panza, F., Lozupone, M., Griseta, C., Castellana, F., Capozzo, R., Ruccia, M., Resta, E., Seripa, D., Logroscino, G., Quaranta, N. (2019). The age-related central auditory processing disorder: Silent impairment of the cognitive ear. *Front Neurosci*, 13, 619.
- Schilder, A. G. M., Van Manen, J. G., Zielhuis, G. A., Grievink, E. H., Peters, S. A. F., Van Den Broek, P. (1993). Long-term effects of otitis media with effusion on language, reading and spelling. *Clin Otolaryngol*, 18, 234–241.
- Share, D. L., Chalmers, D., Silva, P. A., Stewart, I. A. (1986). Reading disability and middle ear disease. *Arch Dis Child*, 61, 400–401.
- Silva, P. A., Chalmers, D., Stewart, I. (1986). Some audiological, psychological, educational and behavioral characteristics of children with bilateral otitis media with effusion: A longitudinal study. *J Learn Disabil*, 19, 165–169.
- Strutt, P. A., Barnier, A. J., Savage, G., Picard, G., Kochan, N. A., Sachdev, P., Draper, B., Brodaty, H. (2020). Hearing loss, cognition, and risk of neurocognitive disorder: Evidence from a longitudinal cohort study of older adult Australians. *Neuropsychol Dev Cogn B Aging Neuropsychol Cogn*, 29, 121–138.
- Technical Committee ISO/TC 43, A. (2010). *ISO 8253-1 Acoustics-Audiometric test methods-Part 1: Pure-tone air and bone conduction audiometry*. <https://www.iso.org/standard/43601.html>
- Thomson, R. S., Auduong, P., Miller, A. T., Gurgel, R. K. (2017). Hearing loss as a risk factor for dementia: A systematic review. *Laryngoscope Invest Otolaryngol*, 2, 69–79.
- Tomlin, D., & Rance, G. (2014). Long-term hearing deficits after childhood middle ear disease. *Ear Hear*, 35, e233–e242.
- Wasano, K., Kaga, K., Ogawa, K. (2021). Patterns of hearing changes in women and men from denarians to nonagenarians. *Lancet Reg Health West Pac*, 9, 100131.
- Wayne, R. V., & Johnsrude, I. S. (2015). A review of causal mechanisms underlying the link between age-related hearing loss and cognitive decline. *Ageing Res Rev*, 23, 154–166.
- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV)* (4th ed.). APA PsycTests.
- Welch, D., & Dawes, P. J. D. (2007). Variation in the normal hearing threshold predicts childhood IQ, linguistic, and behavioral outcomes. *Pediatr Res*, 61, 737–744.
- Wertz, J., Caspi, A., Ambler, A., Broadbent, J., Hancox, R. J., Harrington, H., Hogan, S., Houts, R. M., Leung, J. H., Poulton, R., Purdy, S. C., Ramrakha, S., Rasmussen, L. J. H., Richmond-Rakerd, L. S., Thorne, P. R., Wilson, G. A., Moffitt, T. E. (2021). Association of history of psychopathology with accelerated aging at midlife. *JAMA Psychiatry*, 78, 530–539.
- Williams, C. J., & Jacobs, A. M. (2009). The impact of otitis media on cognitive and educational outcomes. *Med J Aust*, 191, S69–S72.
- Wilson, B. S., & Tucci, D. L. (2021). Addressing the global burden of hearing loss. *Lancet*, 397, 945–947.
- World report on hearing. Geneva: World Health Organization; 2021..