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## Hearing loss is associated with delayed neural responses to continuous speech

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Marlies Gillis, Decrui L., Jonas Vanthornhout, Tom Francart

**Institutions:** Katholieke Universiteit Leuven, University of Maryland, College Park

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9 **Abstract**

10 We investigated the impact of hearing loss on the neural processing of speech. Using a forward modelling  
11 approach, we compared the neural responses to continuous speech of 14 adults with sensorineural hearing  
12 loss with those of age-matched normal-hearing peers.

13 Compared to their normal-hearing peers, hearing-impaired listeners had increased neural tracking and  
14 delayed neural responses to continuous speech in quiet. The latency also increased with the degree of  
15 hearing loss. As speech understanding decreased, neural tracking decreased in both population; however,  
16 a significantly different trend was observed for the latency of the neural responses. For normal-hearing  
17 listeners, the latency increased with increasing background noise level. However, for hearing-impaired  
18 listeners, this increase was not observed.

19 Our results support the idea that the neural response latency indicates the efficiency of neural speech  
20 processing. Hearing-impaired listeners process speech in silence less efficiently than normal-hearing  
21 listeners. Our results suggest that this reduction in neural speech processing efficiency is a gradual effect  
22 which occurs as hearing deteriorates. Moreover, the efficiency of neural speech processing in hearing-  
23 impaired listeners is already at its lowest level when listening to speech in quiet, while normal-hearing  
24 listeners show a further decrease in efficiency when the noise level increases.

25 From our results, it is apparent that sound amplification does not solve hearing loss. Even when  
26 intelligibility is apparently perfect, hearing-impaired listeners process speech less efficiently.

27 **Key words:** neural tracking, hearing loss, speech, EEG

## 28 Introduction

29 It is widely known that hearing loss alters the brain (Eggermont, 2017; Peelle and Wingfield, 2016). To study  
30 the functional neural changes, several studies focussed on cortical auditory evoked potentials (CAEP) using  
31 electroencephalography (EEG). CAEPs reflect the cortical responses evoked by repetitions of simple sounds  
32 such as syllables, tone pips, or clicks. These responses represent the detection and/or discrimination of a  
33 sound. The CAEP-response is characterized by a first positive peak (P1) around 50 ms, a first negative peak  
34 (N1) around 100 ms and a later positive peak (P2) around 180 ms (Burkard et al., 2007). Harkrider et al.  
35 (2009) and Campbell and Sharma (2013) reported increased P2-latencies in hearing impaired listeners (HI  
36 listeners) compared to normal hearing listeners (NH listeners). Interestingly, Campbell and Sharma (2013)  
37 reported that P2-latency was also correlated with the person's speech perception ability in noise. Although  
38 changes in latency are often not reported, in most studies HI listeners showed increased amplitudes compared  
39 to NH listeners (Tremblay et al., 2003; Harkrider et al., 2006; Bertoli et al., 2011; Alain, 2014; Maamor  
40 and Billings, 2017) while Billings et al. (2015) and Koerner and Zhang (2018) did not observe differences  
41 between these two populations or others attributed these differences to decreased audibility of the stimulus  
42 (Oates et al., 2002; Van Dun et al., 2016; McClannahan et al., 2019). No consensus has been reached on  
43 the impact of hearing loss on the P1-N1-P2-complex. The use of continuous speech as the stimulus can be  
44 key to characterize the neural differences between these two populations as it requires more in-depth neural  
45 processing of the stimulus to understand the speech.

46 A limited number of studies has been conducted to study the effect of hearing loss on the neural responses to  
47 continuous speech. In these studies, the amount of neural tracking, i.e. to what extent speech is tracked by  
48 the brain, has been investigated in a two-talker scenario: an attended speaker and an ignored one (Petersen  
49 et al., 2017; Mirkovic et al., 2019; Presacco et al., 2019; Decruy et al., 2020; Fuglsang et al., 2020). In all these  
50 studies, both NH listeners and HI listeners, showed a higher neural tracking of the attended speech stream  
51 than that of the ignored speech stream. Petersen et al. (2017) reported that adults with a higher degree of  
52 hearing loss showed a higher neural tracking of the ignored speech and no change in the attended stream,  
53 suggesting that they experience more difficulties inhibiting irrelevant information. Although Mirkovic et al.  
54 (2019) and Presacco et al. (2019) did not report a neural difference between the two populations, Decruy  
55 et al. (2020) and Fuglsang et al. (2020) observed, in contrast to Petersen et al. (2017), an enhanced neural  
56 tracking in HI listeners for the attended-speech compared to their normal-hearing peers. This enhancement  
57 can indicate a compensation mechanism: HI listeners need to compensate for the degraded auditory input  
58 and therefore show enhanced neural tracking.

59 The difficulties of researching HI listeners are twofold. First, most HI listeners are older, and ageing also has  
60 an impact on brain responses (Tremblay et al., 2003; Harkrider et al., 2006; Burkard et al., 2007; Harkrider  
61 et al., 2009; Decruy et al., 2019; Presacco et al., 2016). Therefore, it is important to compare HI listeners to  
62 age-matched normal-hearing peers. Second, audibility of the stimulus must be taken into account: sound  
63 presented at the same intensity can be less audible for HI listeners than for NH listeners.

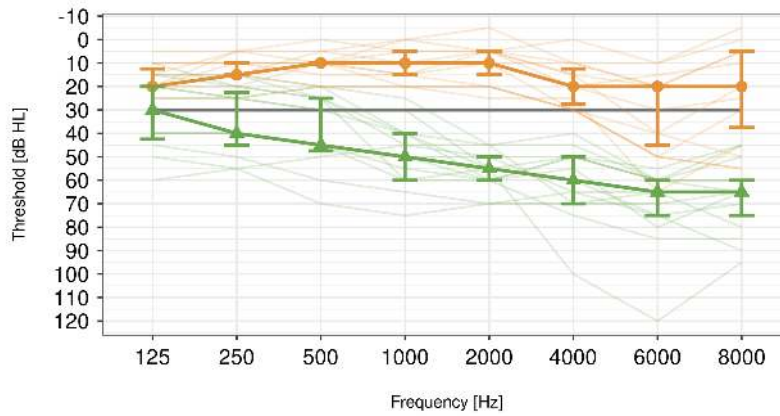
64 Previous studies which reported the differences between HI listeners and NH listeners, focused on differences  
65 in neural tracking. In the current study, we investigated whether the characteristics of the neural responses  
66 to continuous speech (e.g., latency and topography) differed. Here, we showed that there are differences in  
67 the neural responses to continuous speech between HI listeners and their age-matched normal-hearing peers.  
68 Our results showed delayed neural responses to continuous speech in HI listeners. We hypothesized that HI  
69 listeners recruit more brain regions to understand speech, which is reflected in enhanced neural tracking of  
70 speech as well as a delay of neural responses.

## 71 **Materials and Methods**

### 72 **Participants**

73 We used a dataset containing EEG of 14 HI listeners (8♀) with sensorineural hearing loss and 14 aged-matched  
74 normal-hearing peers (13♀) (between 21 and 82 years old). The data were collected in a previous study by  
75 Decruy et al. (2020) (medical ethics committee of the University Hospital of Leuven approved the experiment  
76 (S57102); all participants signed an informed consent form). Inclusion criteria were: (1) having Dutch  
77 as a mother tongue, (2) having symmetrical hearing and (3) absence of medical conditions and learning  
78 disorders. A cognitive screening, the Montreal Cognitive Assessment (Nasreddine, 2004), was performed for  
79 all participants to ensure the absence of cognitive impairment. Hearing thresholds were determined using  
80 pure tone audiometry (125 to 8000 Hz). Normal hearing was defined for all participants where the hearing  
81 threshold did not exceed 30 dB HL for frequencies 125 to 4000 Hz (average of hearing thresholds within this  
82 frequency range in the stimulated ear is denoted as the pure-tone average (PTA)). The hearing thresholds  
83 and PTA are shown in Figure 1 (NH listeners: average PTA=  $13.27 \pm 5.60$  dB HL, HI listeners: average PTA  
84 =  $44.46 \pm 10.54$  dB HL).

**(A) Hearing thresholds for stimulated ear**



**(B) Distribution of age and PTA**

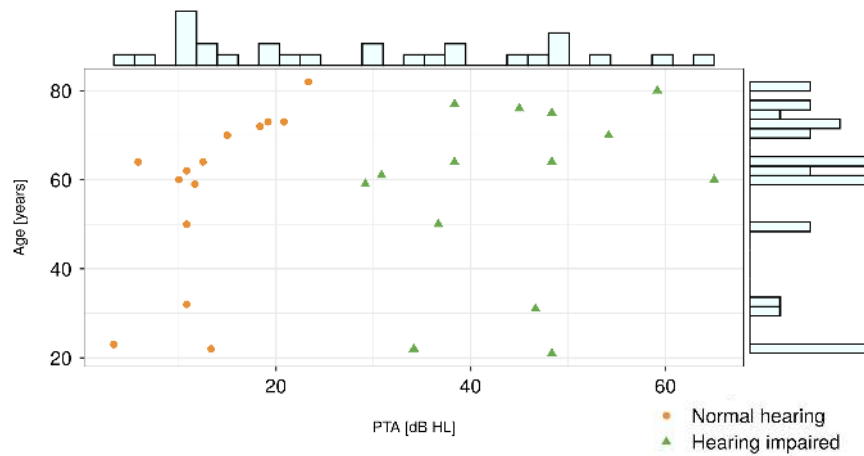


Figure 1: The hearing thresholds for the stimulated ear (panel A) and PTA as a function of age (panel B) for NH listeners (orange) and HI listeners (green).

## 85 **Experimental Procedures**

### 86 **Behavioural Experiment: Flemish Matrix sentence test**

87 The Matrix sentence test was performed to determine the participant's Speech Reception Threshold (SRT) in  
88 speech weighted noise (SWN). These Matrix sentences have a standard grammatical structure, consisting of a  
89 name, a verb, a numeral, a colour and an object (Luts et al., 2014). The SRT represents the signal-to-noise  
90 ratio (SNR) at which 50% of the presented words are recalled correctly.

### 91 **EEG Experiment**

92 **Data acquisition** A BioSemi ActiveTwo system (Amsterdam, Netherlands) was used to measure EEG  
93 signals during stimuli presentation. This system uses 64 Ag/AgCl electrodes placed according to the 10-20  
94 system (Oostenveld and Praamstra, 2001). The EEG signals were measured with a sampling frequency  
95 of 8192 Hz. All recordings were carried out in a soundproof booth with Faraday cage at ExpORL (Dept.  
96 Neurosciences, KU Leuven).

97 **Stimuli presentation** The speech stimuli were presented monaurally through ER-3A insert phones  
98 (Etymotic Research Inc, IL, USA) using the software platform APEX (Dept. Neurosciences, KU Leuven)  
99 (Francart et al., 2008). The stimuli were presented to the right ear unless the participant preferred the left  
100 ear ( $n = 3$ ; 1 NH; 2 HI). All stimuli were set to the same root mean square level and were calibrated.

101 For all NH listeners, the speech stimuli' intensity was fixed at 55 dB SPL (A-weighted). To ensure audible  
102 stimuli for HI listeners, the stimuli were linearly amplified based on the participant's hearing thresholds  
103 according to the National Acoustics Laboratory-Revised Profound (NAL) algorithm (Byrne et al., 2001).  
104 To ensure a comfortable level, the overall level was adjusted on a subject-specific basis in addition to the  
105 linear amplification so that the stimulus was minimally effortful and comfortable to listen to. The individual  
106 presentation levels are reported by Decruy et al. (2020).

107 During the EEG recording, 2 Dutch stories were presented: (1) "Milan", a 12-minute long story narrated by  
108 Stijn Vranken ( $\sigma$ ) presented in quiet and (2) "De Wilde Zwanen" narrated by Katrien Devos ( $\varphi$ ) presented  
109 in 5 different levels of background speech-weighted noise (each lasted around 2 minutes). The duration of  
110 silences was limited to 200 ms.

111 The levels of background noise for the second story depended on the participant's speech-in-noise performance.  
112 Using an adapted version of the self-assessed Békesy procedure (Decruy et al., 2019), the SRT of the Matrix  
113 sentences was adjusted to obtain a SRT of a story (Decruy et al., 2018, 2019). The noise conditions were

114 calculated on the participant's story adjusted SRT, namely: SRT - 3 dB, SRT, SRT + 3 dB, SRT + 6 dB and  
115 a condition without noise, which approximate speech understanding levels of 20%, 50%, 80%, 95% and 100%.  
116 A subjective rating of the participant's speech understanding was obtained after each condition (details are  
117 described in Decruy et al. (2018, 2019)).

## 118 **Signal Processing**

### 119 **Processing of the EEG signals**

120 The EEG recording with a sampling frequency of 8192 Hz was downsampled to 256 Hz to decrease processing  
121 time. To remove artefacts of eye blinks, we applied multi-channel Wiener filtering to the EEG data to remove  
122 artefacts of eye blinks (Somers et al., 2018). Then we referenced the EEG data to the common-average and  
123 filtered the data between 0.5 and 25 Hz using a zero-phase Chebyshev filter (Type II with an attenuation of  
124 80 dB at 10% outside the passband). Additional downsampling to 128 Hz was performed.

### 125 **Extraction of the speech features**

126 In this study, we used 2 speech features: spectrogram and acoustical onsets. Both speech features are  
127 continuous features which represent the acoustical properties of the speech stimulus.

128 To create the spectrogram, the speech stimulus (without amplification) was low-pass filtered below 4000 Hz  
129 (zero-phase low-pass FIR filter with a hamming window of 159 samples) because the ER-3A insert phones  
130 also low-pass filter at this frequency. A spectrogram representation was obtained using the Gammatone  
131 Filterbank Toolkit 1.0 (Heeris, 2014) (centre frequencies between 70 and 4000 Hz with 256 filter channels and  
132 an integration window of 0.01 second). This toolkit calculates a spectrogram representation based on a series  
133 of gammatone filters inspired by the structure of the human auditory system (Slaney, 1998). The resulting  
134 256 filter outputs were averaged into 8 frequency bands (each containing 32 outputs). Additionally, each  
135 frequency band was downsampled to the same sampling frequency as the processed EEG, namely 128 Hz.  
136 The NAL filtering introduced a delay of 5.334 ms, which was compensated for. The acoustical onsets were  
137 calculated as a half-wave rectification of the spectrogram's derivative.

### 138 **Prediction accuracies, temporal response function & peak picking method**

139 In this study, we focused on a linear forward modelling approach that predicts the EEG based on a linear  
140 combination of speech features of the presented speech. This forward modelling approach results in 2  
141 outcomes: (a) a temporal response function (TRF) and (b) a prediction accuracy. (a) A TRF is a linear  
142 approximation of the brain's impulse response. It is a signal over time that describes how the brain responds



143 to the speech features. (b) TRFs can be used to predict the EEG by convolving it with the speech features.  
144 The predicted EEG is then correlated with the actual EEG to obtain a prediction accuracy. Prediction  
145 accuracy is considered a measure of neural tracking: the higher the prediction accuracy, the better the brain  
146 tracks the stimulus.

147 (a) To estimate TRFs, we used the Eelbrain toolbox (Brodbeck, 2020). The toolbox estimates TRFs using  
148 the boosting algorithm by David et al. (2007) (using a fixed step size of 0.005; stopping criteria based on  
149  $\ell_2$ -norm; kernel basis of 50 ms). We used 4-fold cross-validation (4 equally long folds; 3 folds used for training,  
150 1 for validation) and an integration window between 0 and 700 ms. The estimated TRFs, averaged across  
151 folds and frequency bands, were used to determine the peak latencies.

152 (b) To calculate the prediction accuracy, the TRF is applied to left-out EEG to allow a fair comparison  
153 between models with a different number of speech features. We used the boosting algorithm with a testing  
154 fold. This implies a 4-fold cross-validation with 2 folds for training, 1 fold for validation and 1 fold for testing,  
155 which is left-out during training and validation. Each estimated TRF was used to predict the EEG of the  
156 left-out testing fold. The predicted EEG of all left-out segments are correlated, using Pearson correlation,  
157 with the actual EEG to obtain a prediction accuracy per EEG-electrode. The prediction accuracies were  
158 averaged across EEG-electrodes and denoted as neural tracking. Similarly, as Decruy et al. (2020), we  
159 calculated the neural tracking of the second story, presented in different level of background noise, using the  
160 TRFs estimated on the story in quiet.

161 From the TRF, we aimed to identify the amplitude and latency of 3 peaks: P1, N1 and P2. As the EEG  
162 data contains 64 different channels, 64 different TRFs were estimated, which made peak picking more  
163 complex. Therefore we applied principal component analysis (PCA), a dimensionality reduction method. The  
164 PCA-method results in (a) signals in component space and (b) corresponding spatial filters which describe  
165 the linear combinations of EEG channels to obtain these components. In our analysis, the first component  
166 was used. Adding more components up to 4 did not change the findings of this study. In addition to the time  
167 course of the component, we also investigated the corresponding spatial filter. As the sign of this spatial filter  
168 is arbitrary, we forced the average of occipital and parietal channels (P9, P7, PO7, O1, Oz, O2, PO8, P8, Iz,  
169 P10) to be negative by multiplying the spatial filter with -1 when needed. The PCA-method was applied to  
170 the data per story for each participant.

171 To identify the different peaks, we performed a z-score normalization of the TRF in component space and  
172 determined the maximal or minimal amplitude for positive and negative peaks in different time regions (P1:  
173 30 to 110 ms, N1: 70 to 210 ms, P2: 110 to 270 ms), respectively. The overlap of these time regions is not an

174 issue as we identified either the maximal or minimal amplitude to determine the peak latency of a positive or  
175 negative peak, respectively. To only identify prominent peaks, a peak was discarded from the analysis if the  
176 amplitude of the normalized TRF was smaller than the threshold of 1.

## 177 **Statistical analysis**

178 We used the R software package (version 3.6.3) (R Core Team, 2020). We used the Buildmer toolbox, which  
179 allows identifying the best linear mixed model (LMM) or linear model (LM) given a series of predictors and  
180 all their possible interactions based on the likelihood-ratio test (Voeten, 2020). Depending on the analysis,  
181 we used the following predictors: (a) hearing status (NH or HI) or the PTA depending on whether we were  
182 interested in the group effect or the effect of the degree of hearing loss, (b) age and (c) peak type (P1,  
183 N1, P2). To observe an effect of model choice on prediction accuracy, we also included the predictor (d)  
184 model type (Spectrogram, Acoustic onsets, Acoustic onsets + Spectrogram) in the statistical analysis. The  
185 analysis over different noise conditions also included the predictor (e) speech understanding. For the analysis  
186 with regard to peak latency, all continuous predictors were z-scored to minimize effects due to differences in  
187 scale. A matching factor indicated the participants belonging to the same age-matched pair. We included  
188 a nested random effect: participant nested inside match, as each match contained a pair of participants,  
189 and each participant had multiple dependent observations. The models' assumptions were checked with a  
190 visual inspection of the residual plots to assure homoscedasticity and normality. The models' outcomes were  
191 reported with the unstandardized regression coefficient ( $\beta$ ) with standard error (SE), t-ratio and p-value per  
192 fixed effect. If significant interaction effects were found or if we aimed to identify differences between different  
193 levels of a factor, additional Holm-adjusted post-hoc tests were performed by looking at the estimates of the  
194 estimated marginal means or estimated marginal means of linear trends or pairwise comparisons of these  
195 estimates, implemented by the Emmeans toolbox (Lenth, 2020). A significance level of  $\alpha = 0.05$  was used.

196 To compare differences in spatial filters or topographies of the peaks between the 2 groups, we used a related  
197 cluster-based permutation test proposed by Maris and Oostenveld (2007) to determine whether the topography  
198 differs between NH listeners and HI listeners, using the Eelbrain implementation (Brodbeck, 2020). For these  
199 related cluster-based permutation tests the age-matching was preserved. For instance to test whether the  
200 topography differed between HI listeners and NH listeners, only the peak topographies of the age-matched  
201 participants were considered if both participants showed a prominent peak. A significance level of  $\alpha = 0.05$   
202 was used.

## Results

### Neural differences when listening to speech in quiet

#### Hearing-impaired listeners show higher neural tracking

We identified the speech feature(s) that resulted in the highest neural tracking: acoustic onsets, spectrogram or a combination of both speech features. As shown in Figure 2 and verified by the statistical analysis, the highest neural tracking was obtained with a combination of both speech features (analysis using LMM: Table 1). Additionally, HI listeners showed higher neural tracking compared to the group of NH listeners (on average 0.012 higher; SE = 0.0054, df = 26, t-ratio = 2.2715, p = 0.0316). Age did not have a significant effect on the neural tracking of speech. A Holm-adjusted pairwise comparison confirmed that the highest neural tracking was obtained with a combination of both speech features which was higher compared to the model using just the acoustic onsets (on average the prediction accuracy of the combined model is 0.003 higher; SE = 0.000651; df = 54, t-ratio = 4.521, p = 0.0001) and higher compared to the model using the spectrogram variable (on average the prediction accuracy of the combined model is 0.005 higher; SE = 0.000651; df = 54; t-ratio = 7.230; p < 0.0001).

Table 1: Linear mixed model: the effect of hearing status and model type on neural tracking. Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect. Formula: neural tracking  $\sim$  1 + hearing status + model type + (1 | match/participant)

fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for NH / Spectrogram)	0.0252	0.0039	26.4961	6.5042	p < 0.001
Hearing status: HL	0.0124	0.0054	26	2.2715	p = 0.0316
Model type = Acoustic onsets	0.0018	7e-04	54	2.7088	p = 0.0090
Model type = Acoustic onsets + spectrogram	0.0047	7e-04	54	7.2301	p < 0.001

#### Delayed peak latencies for hearing-impaired listeners

In Figure 5.A, all TRFs in component space are shown for both populations and speech features. The TRFs of HI listeners show delayed neural responses to speech compared to those of NH listeners. Additionally, the average TRF for each speech feature show 2 prominent peaks: P1-peak of acoustic onsets ( $P1_{AO}$ ) and N1-peak of acoustic onsets ( $N1_{AO}$ ) for the acoustic onsets, N1-peak of spectrogram ( $N1_S$ ) and P2-peak of spectrogram ( $P2_S$ ) for the spectrogram. The best LMM predicting latency included a main effect of the considered peak, hearing status and age (Table 2). Adults with hearing loss showed later peak latencies (an increase of 23 ms, SE = 6.7665, df = 24.0459, t-ratio = 3.3822, p = 0.0025). The effect of age depended

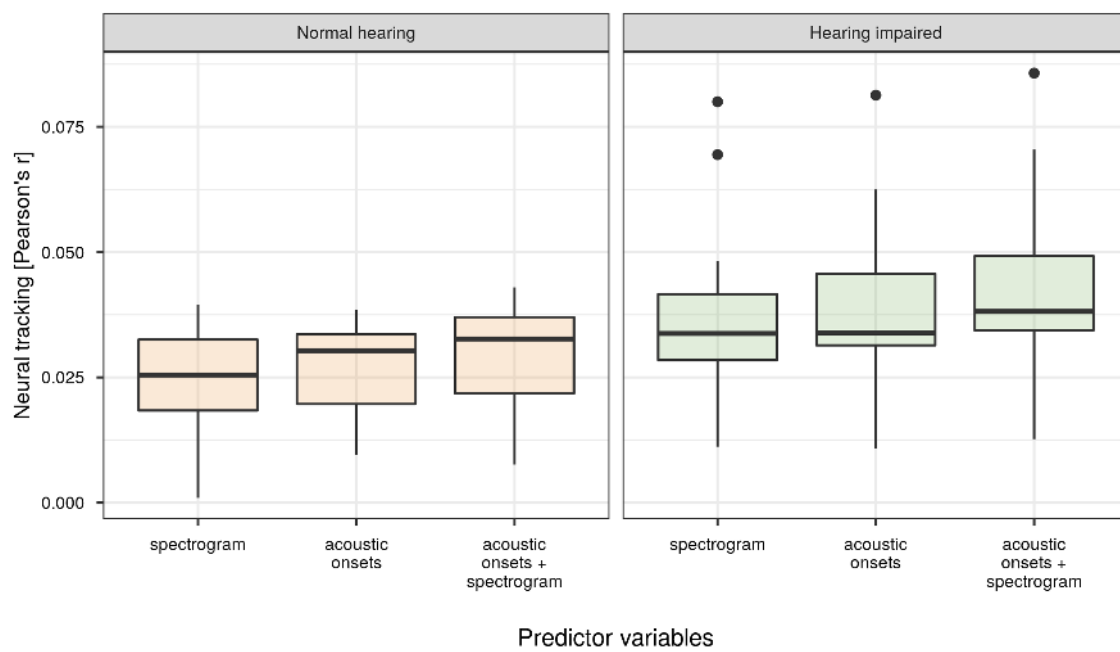


Figure 2: Neural tracking (Pearson's  $r$ ) as a function of different combinations of speech features ('spectrogram', 'acoustic onsets' and 'acoustic onsets + spectrogram', respectively) for both NH listeners (left; orange) and HI listeners (right; green).

225 on the considered peak. No significant interaction between age and hearing status was observed. Post-hoc  
226 testing showed a significant decrease in latency with increasing age for the  $N1_S$ -latency (estimate of marginal  
227 trend:  $-0.793$ ,  $SE = 0.271$ ,  $df = 68.8$ ,  $t\text{-ratio} = -2.926$ ,  $p = 0.0186$ ) while no significant trend was observed for  
228 the other peak latencies.

229 We did not observe a significant difference between the spatial filters of HI listeners and NH listeners. HI  
230 listeners showed a significantly different topography for  $N1_S$  compared to NH listeners (Figure 3). HI  
231 listeners showed a more prominent central negativity and a higher occipital positivity which was slightly  
232 left-lateralized.

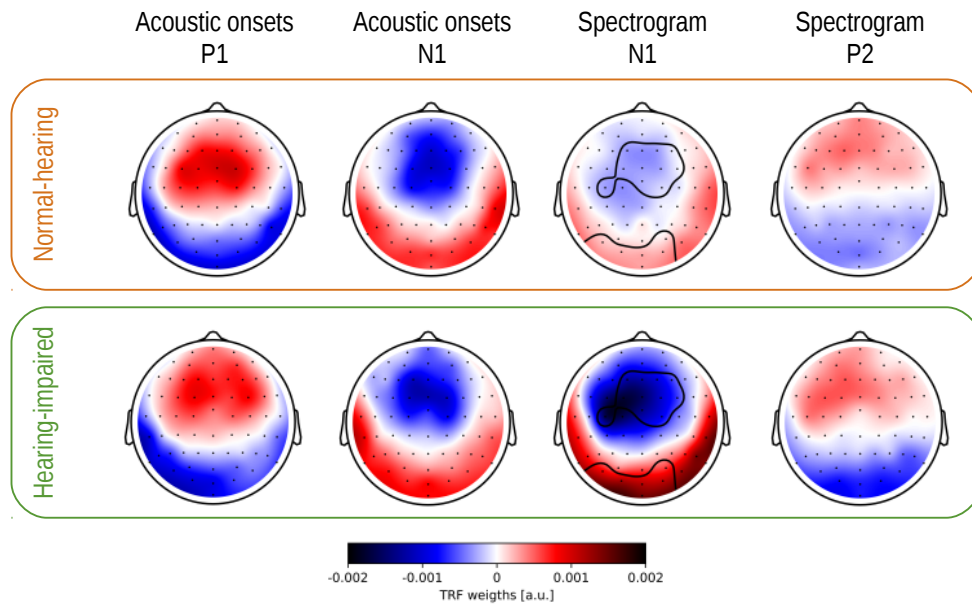


Figure 3: Visualization of the topographies of the peaks in the TRFs in sensor space for both speech features, spectrogram and acoustic onsets, and for NH listeners and HI listeners.

### 233 **Longer latencies are associated with higher degrees of hearing loss**

234 As significant differences in peak latencies were observed between NH listeners and HI listeners, we hypothesized  
235 that a higher degree of hearing loss is associated with increased latency of the peaks. Similarly as above, we  
236 identified the LMM which explains the variance in the latency. However, instead of using the factor hearing  
237 status, we used the continuous variable describing the degree of hearing loss. We justify this approach because  
238 the degree of hearing loss (represented by the PTA) is rather continuously distributed across the participants  
239 (Figure 1).

240 The latency depended on the considered peak, the degree of hearing loss and age of the listener (Table 3;  
241 using scaled predictors). The effect of degree of hearing loss on the latency depended on the considered

Table 2: Results of the linear mixed model in order to assess peak type, hearing status and age on the peak latency of  $P1_{AO}$ ,  $N1_{AO}$ ,  $N1_S$ ,  $P2_S$ . Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect.

Formula: latency  $\sim 1 + \text{peak} + \text{hearing status} + \text{age} + \text{peak:age} + (1 | \text{match}/\text{participant})$

fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for NH / for P1 - acoustic onsets)	65.5364	13.9654	49.1829	4.6928	p < 0.001
peak = N1 - acoustic onsets	73.013	17.5116	57.2985	4.1694	p < 0.001
peak = N1 - spectrogram	74.7323	17.7336	58.4244	4.2142	p < 0.001
peak = P2 - spectrogram	105.822	16.1937	56.478	6.5348	p < 0.001
Hearing status: HL	22.8857	6.7665	24.0459	3.3822	p = 0.0025
Age	-0.3274	0.2266	52.8304	-1.4449	p = 0.1544
peak = N1 - acoustic onsets:age	-0.0341	0.2805	56.788	-0.1214	p = 0.9038
peak = N1 - spectrogram:age	-0.4657	0.2781	57.2401	-1.6747	p = 0.0994
peak = P2 - spectrogram:age	0.7456	0.2649	56.0189	2.8147	p = 0.0067

242 peak and age of the listener. The Holm-adjusted estimates of the marginal trend showed that the trend  
 243 of increasing latency with increasing degree of hearing loss is only significant for older adults for the peak  
 244 latency of  $N1_{AO}$  (estimate of trend = 21.0, SE = 6.44, df = 61.0, t-ratio = 3.260, p = 0.0146) and  $N1_S$   
 245 (estimate of this trend = 20.4, SE = 6.38, df = 60.5, t-ratio = 3.192, p = 0.0157). However, this trend did  
 246 not significantly differ between the different peaks nor between younger and older adults.

247 Looking at Figure 1, age is not evenly distributed. Therefore, the age effects in the above-mentioned analysis  
 248 might be biased towards 3 younger age-matched pairs. We replicated the above-mentioned analysis using only  
 249 participants above 40 years old. Indeed, in this analysis no interaction was found between degree of hearing  
 250 loss and age nor degree of hearing loss, age and the considered peak. The latency of the peaks depended on  
 251 the considered peak, age and the degree of hearing loss and the effect of age depended on the considered  
 252 peak (Table S.1; using scaled predictors). The peak latency increased with increasing degree of hearing loss  
 253 independent of the considered peak (estimate = 12.8954, SE = 3.5184, df = 17.8724, t-ratio = 3.6652, p =  
 254 0.0018). The Holm-adjusted estimates of the effect of age on the peak latency were not significant for any of  
 255 the peak latencies.

256 The results of the above analysis suggest that the effect of age on the peak latencies is not robust. Therefore,  
 257 this effect was not visualized in Figure 5. With increasing degree of hearing loss, the latency of neural  
 258 responses increased (estimate = 14.4906, SE = 3.9794, df = 53.1663, t-ratio = 3.6414, p < 0.001; Table 3;  
 259 Figure 5.B).

260 For each peak latency, we identified whether the variance in latency is explained by age and/or degree of  
 261 hearing loss when only considering HI listeners. Although this reduced the statistical power, we observed a

262 significant effect of degree of hearing loss on the  $N1_{AO}$ -latency: HI listeners with a more severe hearing loss  
 263 showed an increased latency (analysis using LM and scaled predictors; Table S.2; estimate = 2.0006, SE =  
 264 0.6996, t-ratio = 2.860,  $p = 0.0188$ ).

Table 3: Results of the linear mixed model in order to assess the effects of degree of hearing loss (PTA) and age on the peak latency of  $P1_{AO}$ ,  $N1_{AO}$ ,  $N1_S$ ,  $P2_S$ . Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.

Formula: latency  $\sim 1 + \text{peak} + \text{PTA} + \text{age} + \text{peak:age} + \text{peak:PTA} + \text{peak:PTA:age} + (1 | \text{match}/\text{participant})$

fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	57.5671	4.1993	57.7904	13.7087	$p < 0.001$
peak = N1 - acoustic onsets	71.513	4.9762	50.0303	14.371	$p < 0.001$
peak = N1 - spectrogram	45.1675	5.0515	50.412	8.9414	$p < 0.001$
peak = P2 - spectrogram	150.0076	4.7256	47.5163	31.7433	$p < 0.001$
Degree of hearing loss (PTA)	14.4906	3.9794	53.1663	3.6414	$p < 0.001$
Age	-8.1202	3.8447	52.9895	-2.1121	$p = 0.0394$
peak = N1 - acoustic onsets:age	-2.2935	5.2705	53.8308	-0.4352	$p = 0.6652$
peak = N1 - spectrogram:age	-9.1196	4.9517	51.9372	-1.8417	$p = 0.0712$
peak = P2 - spectrogram:age	14.7078	4.721	50.1196	3.1154	$p = 0.0030$
Degree of hearing loss (PTA):age	1.1324	3.7797	52.3099	0.2996	$p = 0.7657$
peak = N1 - acoustic onsets:PTA	2.0488	5.0542	51.1439	0.4054	$p = 0.6869$
peak = N1 - spectrogram:PTA	-11.919	5.3951	51.9705	-2.2092	$p = 0.0316$
peak = P2 - spectrogram:PTA	-2.2075	4.4718	46.5795	-0.4936	$p = 0.6239$
peak = N1 - acoustic onsets:PTA:age	3.3177	5.2119	55.1995	0.6366	$p = 0.5270$
peak = N1 - spectrogram:PTA:age	16.6746	5.7976	55.4212	2.8761	$p = 0.0057$
peak = P2 - spectrogram:PTA:age	-0.5236	4.3161	47.3511	-0.1213	$p = 0.9040$

## 265 Neural differences when speech understanding decreases

### 266 Increased neural tracking with increased speech understanding

267 The effect of increased neural tracking for HI listeners was robust over different levels of background noise  
 268 (estimate = 0.0154, SE = 0.0046, df = 25.1566, t-ratio = 3.3594,  $p = 0.0025$ ); Table 4; Figure 4). Additionally,  
 269 higher neural tracking was observed with increasing age (estimate =  $3e-04$ , SE =  $1e-04$ , df = 24.9812, t-ratio  
 270 = 2.2452,  $p = 0.0339$ ); Table 4; Figure 4) and with increasing speech understanding (estimate =  $2e-04$ , SE  
 271 = 0, df = 115.3178, t-ratio = 5.6547,  $p < 0.001$ ; Table 4; Figure 4). No significant interaction effect was  
 272 observed between hearing status and speech understanding.

### 273 Normal hearing listeners show a prominent increase in latency when speech understanding 274 decreases, while this is less prominent for hearing-impaired listeners

275 We analysed the effects of speech understanding, age and degree of hearing loss on the peak latencies for the  
 276 second story presented in different levels of background noise. The latency of the neural responses depends

Table 4: Linear mixed model: the effect of hearing status and speech understanding on neural tracking. Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in match was included as a random effect. Formula: neural tracking  $\sim 1 +$  hearing status + age + speech understanding + (1 | match/participant)

fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for NH)	-0.0068	0.0081	29.4306	-0.8397	p = 0.4078
Hearing status: HL	0.0154	0.0046	25.1566	3.3594	p = 0.0025
Age	3e-04	1e-04	24.9812	2.2452	p = 0.0339
Speech understanding	2e-04	0	115.3178	5.6547	p < 0.001

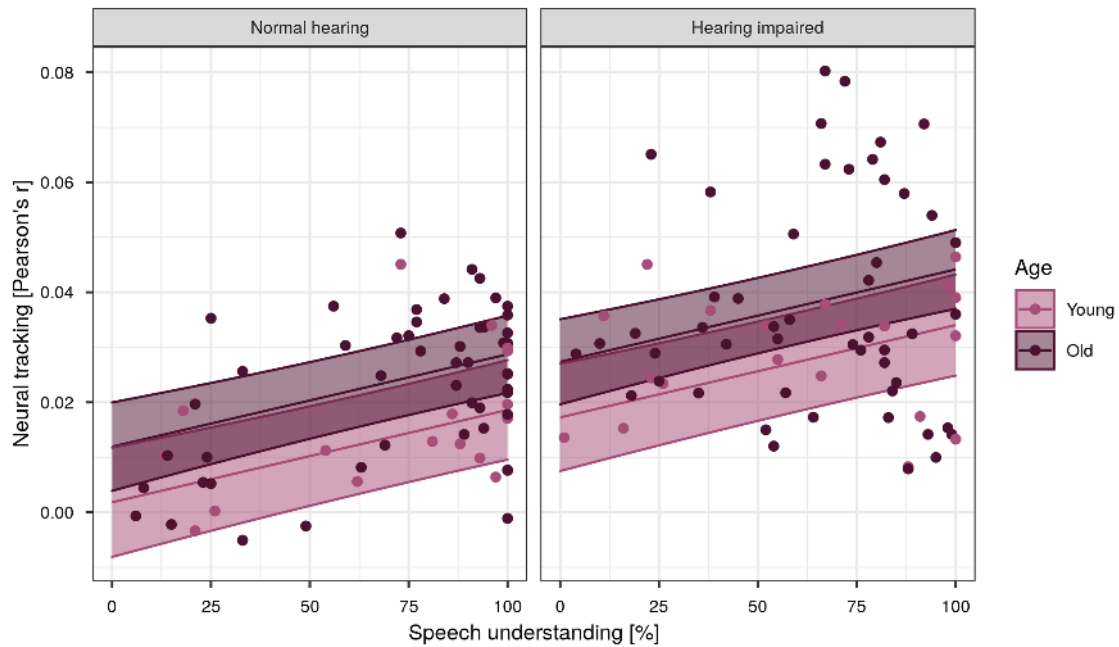


Figure 4: Neural tracking (Pearson's  $r$ ) as a function of speech understanding. The effect of age was discretized with 2 levels: the average average age of participants younger than 50 years (level young; 31 years; pink) and participants with hearing loss (level old; 68 years; purple) and average average age of participants older than 50 years



277 on the considered peak, age and degree of hearing loss (analysis using LMM with scaled predictors; Table 5;  
 278 Figure 5.C). The trends of age and speech understanding on peak latency depended on the considered peak.  
 279 However, post-hoc tests did not show a significant effect of age on any of the peak latencies. Therefore this  
 280 effect is not visualized in Figure 5.C.

281 Interestingly, a significant interaction effect between speech understanding and degree of hearing loss was  
 282 found (estimate = 4.441, SE = 1.071, df = 396.822, t-ratio = 4.148,  $p < .001$ ). Post-hoc testing showed that  
 283 NH listeners showed a significant increase in latency when speech understanding decreased (estimate = -10.5,  
 284 SE = 1.42, df = 390, t-ratio = -7.401,  $p < 0.0001$ ) while no significant increase was observed for HI listeners.  
 285 This trend significantly differed between the NH listeners and HI listeners (estimate = -1.6, SE = 1.68, df =  
 286 399, t-ratio = -0.955,  $p = 0.3402$ ).

Table 5: Results of the linear mixed model in order to assess the effects of degree of hearing loss, speech understanding and age on the peak latency of  $P1_{AO}$ ,  $N1_{AO}$ ,  $N1_S$ ,  $P2_S$ . Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.

Formula: latency  $\sim 1 + \text{peak} + \text{SI} + \text{degree of hearing loss} + \text{speech understanding} + \text{degree of hearing loss} + \text{peak:speech understanding} + \text{age} + \text{degree of hearing loss:age} + \text{peak:age} + \text{speech understanding:age} + (1 | \text{match}/\text{participant})$

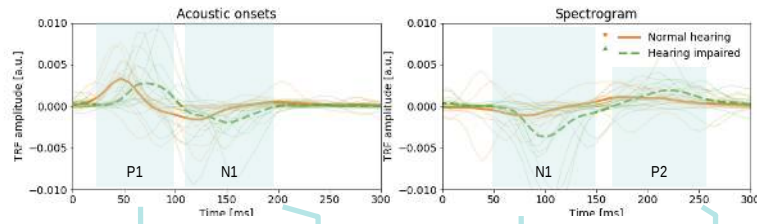
fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	65.523	3.149	69.524	20.808	$p < .001$
peak = N1 - acoustic onsets	71.012	3.015	384.446	23.557	$p < .001$
peak = N1 - spectrogram	39.875	3.203	385.734	12.449	$p < .001$
peak = P2 - spectrogram	144.887	3.097	385.778	46.783	$p < .001$
Speech understanding	-2.46	2.336	388.475	-1.053	$p = 0.293$
Degree of hearing loss (PTA)	5.489	2.324	23.74	2.362	$p = 0.027$
Age	-6.421	2.882	59.27	-2.228	$p = 0.03$
Speech understanding:Degree of hearing loss (PTA)	4.441	1.071	396.822	4.148	$p < .001$
peak = N1 - acoustic onsets:speech understanding	-4.487	2.968	381.779	-1.512	$p = 0.131$
peak = N1 - spectrogram:speech understanding	-1.148	3.163	382.021	-0.363	$p = 0.717$
peak = P2 - spectrogram:speech understanding	-8.703	3.066	383.334	-2.839	$p = 0.005$
Degree of hearing loss (PTA):Age	4.797	2.232	24.283	2.149	$p = 0.042$
peak = N1 - acoustic onsets:age	4.906	2.832	379.405	1.732	$p = 0.084$
peak = N1 - spectrogram:age	0.94	3.196	384.574	0.294	$p = 0.769$
peak = P2 - spectrogram:age	13.103	2.897	384.584	4.523	$p < .001$
Speech understanding:Age	-2.121	1.082	390.971	-1.96	$p = 0.051$

287 The effect of the degree of hearing loss on the peak amplitude was not consistent for all peaks.

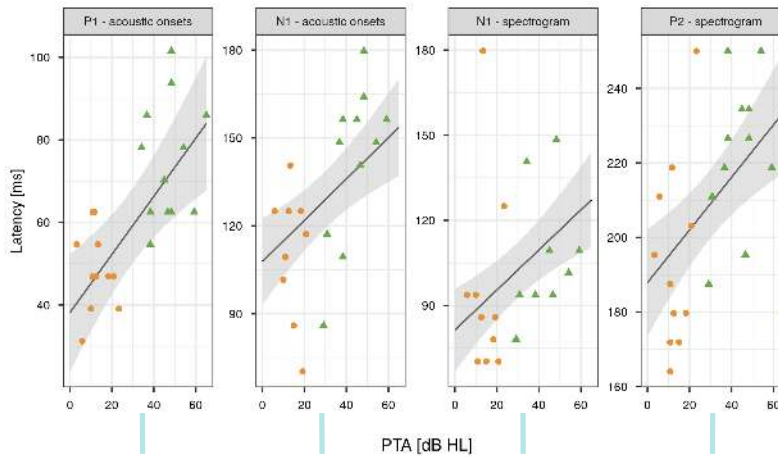
## 288 Discussion

289 We compared the neural responses to continuous speech of adults with a sensorineural hearing loss with those  
 290 of age-matched normal-hearing peers. We found that HI listeners show higher neural tracking and increased

**(A) Delayed peak latencies for hearing-impaired listeners**



**(B) Longer latencies are associated with higher degrees of hearing loss**



**(C) Normal hearing listeners show a prominent increase in latency when speech understanding decreases, while this is less prominent for hearing-impaired listeners**

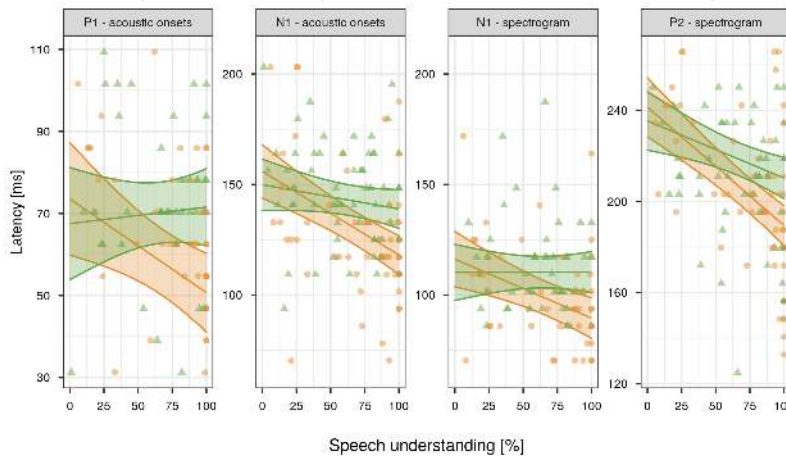


Figure 5: An overview of the neural responses of HI listeners (HI; striped line; green, triangle) and NH listeners (NH; orange, dot). Panel A: TRF in component space when listening to a story in quiet for both speech features evaluated for both HI listeners and NH listeners. The thick line represents the average TRF over participants. The lighter lines represent the subject-specific TRFs. Panel B: The peak latency in function of the degree of hearing loss (PTA) derived from the neural responses when listening to a story in quiet. Panel C: The peak latencies in function of speech understanding derived from the neural responses when listening to a story presented in multiple levels of background noise. The effect of degree of hearing loss was made discrete at 2 levels, the average hearing thresholds of all NH listeners (level NH listeners: 13 dB HL) and subjects with hearing loss (level HI listeners: 44 dB HL) and is represented by the regression lines with confidence intervals (shaded area).

291 peak latencies in their neural responses. Across noise conditions, NH listeners showed increased latencies  
292 as speech understanding decreased. However, for adults with hearing loss, this increase in latency was not  
293 observed.

## 294 **Higher neural tracking of speech in hearing-impaired listeners**

295 By evaluating neural tracking, we concluded that (1) higher neural tracking is observed for a combination of  
296 the spectrogram and acoustic onsets compared to the speech features individually and (2) HI listeners show  
297 enhanced neural tracking compared to normal-hearing peers.

298 The combination of speech features results in higher neural tracking, which implies that both speech features  
299 encode unique information. Following Hamilton et al. (2018) and Brodbeck et al. (2020), both speech features  
300 allow a differentiation between sustained activity, represented by the spectrogram, and transient activity,  
301 represented by acoustic onsets.

302 Using a forward modelling approach (instead of backward), we observed enhanced neural tracking in HI  
303 listeners. This agrees with results of Decruy et al. (2020) and Fuglsang et al. (2020) who also reported that  
304 HI listeners have higher neural tracking than NH listeners of the attended speaker. Nevertheless, Presacco  
305 et al. (2019) did not find a difference in neural tracking between the two populations. However, in their study,  
306 the populations were not closely age-matched, while ageing is known to increase neural tracking (Presacco  
307 et al., 2016; Decruy et al., 2019).

308 Like previous literature, we also observed that neural tracking decreases with decreasing speech understanding  
309 (Vanthornhout et al., 2018; Lesenfants et al., 2019; Decruy et al., 2020). Even when the speech is presented  
310 with background noise, HI listeners showed enhanced neural tracking of speech. This suggests evidence for  
311 a compensation mechanism: higher neural tracking indicates more neural activity to compensate for the  
312 degraded auditory input (Eggermont, 2017; Fuglsang et al., 2020). Although the enhanced neural tracking  
313 of speech in HI listeners, the effect of speech understanding on the neural tracking was similar for both  
314 populations.

## 315 **Hearing-impaired listeners process speech less efficiently**

316 HI listeners showed significantly increased latencies compared to their age-matched normal-hearing peers  
317 when they listened to a story presented in quiet (Figure 5.A). Additionally, the delay in neural responses  
318 increased with a higher degree of hearing loss (Figure 5.B).

319 Investigating the CAEP-response, Campbell and Sharma (2013) and Bidelman et al. (2019b) have reported an

320 increased P2 latency with worse speech perception in noise but not with the degree of hearing loss. However,  
321 in both studies, the same intensity was presented to both HI listeners and NH listeners. McClannahan  
322 et al. (2019) remarked that differences in the audibility of the stimulus might explain the differences in  
323 neural response latency. Indeed, Verschueren et al. (2020) observed that reduced audibility increases the  
324 latency of the neural responses to continuous speech. However, at a comfortable loudness (at intensities of 60  
325 dB or higher in a NH population), the latency reaches a plateau. Our stimulus is amplified based on the  
326 participants' hearing thresholds and is presented at a subject-specific intensity to assure comfortable listening  
327 for HI listeners, therefore, we minimized the effects of differences in audibility of the stimulus.

328 Even though the sound was amplified, HI listeners showed increased latencies. Therefore, we hypothesize  
329 that there are some intrinsic differences in neural speech processing between HI listeners and NH listeners.  
330 A possible explanation may be that HI listeners process speech less efficiently, as proposed by Bidelman  
331 et al. (2019a). Using functional connectivity analysis, Bidelman et al. (2019a) showed that HI listeners have  
332 (a) more extended communication pathways and therefore (b) less efficient information exchange among  
333 these brain regions. (a) More extended communication paths may reflect a form of compensation in which  
334 additional brain regions are recruited to understand the degraded auditory input. This is supported by  
335 increased frontal activation in HI listeners in the neural responses to simple sounds (Campbell and Sharma,  
336 2013; Bidelman et al., 2019b). Similarly, using continuous speech rather than simple repeated sounds, we  
337 showed that HI listeners have a significantly different  $N1_S$  peak topography which suggests the recruitment  
338 of additional and/or different underlying neural sources (Figure 3). (b) When more or different brain regions  
339 are involved to process the speech, it causes longer communication pathways in the brain and therefore  
340 decreases the neural speech processing efficiency (Bidelman et al., 2019b). Here, we propose the neural  
341 response latency as a marker for the efficiency of neural processing of continuous, natural speech: less efficient  
342 speech processing is reflected by increased neural response latency as information exchange is hampered due  
343 to more involved brain regions and longer communication pathways.

344 When the intensity of the background noise increases in which speech is presented, NH listeners showed  
345 a prominent increase in latency when speech understanding decreases, while this was less prominent for  
346 HI listeners. In several studies, it has been shown that NH listeners show an increased neural response  
347 latency with increasing task demand due to lower stimulus intensity, increasing background noise or stimulus  
348 vocoding. This is the case for neural processing of continuous speech (Mirkovic et al., 2019; Verschueren  
349 et al., 2020; Kraus et al., 2020) as well as simple sounds (Billings et al., 2015; Van Dun et al., 2016; Maamor  
350 and Billings, 2017; McClannahan et al., 2019). Our results show that this increase in latency is absent for  
351 adults with a higher degree of hearing loss (Figure 5.C). This can explain why Mirkovic et al. (2019) did not

352 find a difference in latency between NH listeners and HI listeners as they presented only two noise conditions.  
353 As the noise level increases, the difference in latency between the two populations becomes smaller, which  
354 reduces the likelihood of a statistical difference between the two populations.

355 Bidelman et al. (2019b) did not report an effect of noise on the P2-latency. However, investigating the  
356 functional brain connectivity in the same data, Bidelman et al. (2019a) reported that as noise was added  
357 to the stimulus, NH listeners showed more long-range neural signalling whereas this was not seen for HI  
358 listeners (Bidelman et al., 2019a). The latter finding is supported by our data: in NH listeners the neural  
359 response latency increases as the speech understanding decreases due to increasing level of background noise  
360 while this was less prominent for HI listeners. This suggests that NH listeners process speech in noise less  
361 efficiently: more processing time is required to attend the speech stream and ignoring the noise. However, for  
362 HI listeners, this is not the case: when background noise increases, processing efficiency does not decrease.  
363 If longer latencies are a marker for less efficient neural processing and thus the number of recruited brain  
364 regions, our results in noise suggest that HI listeners recruit already recruit a maximum number of brain  
365 regions in the speech network to understand speech in quiet as their neural response latency does not increase  
366 with increasing amount of background noise.

367 Finally, we would like to highlight the difference in the trend of neural tracking and neural response latency.  
368 As speech understanding decreases, neural tracking decreases for both NH and HI listeners while the neural  
369 response latency remains constant (HI) or increases (NH). This difference in trend suggests that both measures  
370 represent different underlying neural processes for speech comprehension.

## 371 **Conclusion**

372 In this study, we compared the neural responses to continuous speech of adults with a sensorineural hearing  
373 loss with those of age-matched normal-hearing peers. HI listeners showed increased peak latencies of their  
374 neural responses. Interestingly, the latency increases as the degree of hearing loss increases. Across noise  
375 conditions, latency generally increases as the listening conditions become more difficult. However, for HI  
376 listeners, this increase in latency is not observed. We here suggest latency as a marker for the efficiency of  
377 neural processing to understand continuous, natural speech.

## References

- 378
- 379 Alain, C. (2014). Effects of age-related hearing loss and background noise on neuromagnetic activity from  
380 auditory cortex. *Frontiers in systems neuroscience*, 8:8.
- 381 Bertoli, S., Probst, R., and Bodmer, D. (2011). Late auditory evoked potentials in elderly long-term  
382 hearing-aid users with unilateral or bilateral fittings. *Hearing research*, 280(1-2):58–69.
- 383 Bidelman, G. M., Mahmud, M. S., Yeasin, M., Shen, D., Arnott, S. R., and Alain, C. (2019a). Age-related  
384 hearing loss increases full-brain connectivity while reversing directed signaling within the dorsal–ventral  
385 pathway for speech. *Brain Structure and Function*, 224(8):2661–2676.
- 386 Bidelman, G. M., Price, C. N., Shen, D., Arnott, S. R., and Alain, C. (2019b). Afferent-efferent connectivity  
387 between auditory brainstem and cortex accounts for poorer speech-in-noise comprehension in older adults.  
388 *Hearing research*, 382:107795.
- 389 Billings, C. J., Penman, T. M., McMillan, G. P., and Ellis, E. (2015). Electrophysiology and perception of  
390 speech in noise in older listeners: effects of hearing impairment & age. *Ear and hearing*, 36(6):710.
- 391 Brodbeck, C. (2020). Eelbrain 0.32. <http://doi.org/10.5281/zenodo.3923991>.
- 392 Brodbeck, C., Jiao, A., Hong, L. E., and Simon, J. Z. (2020). Neural speech restoration at the cocktail party:  
393 Auditory cortex recovers masked speech of both attended and ignored speakers. *bioRxiv*, page 866749.
- 394 Burkard, R. F., Eggermont, J. J., and Don, M. (2007). *Auditory evoked potentials: basic principles and*  
395 *clinical application*. Lippincott Williams & Wilkins.
- 396 Byrne, D., Dillon, H., Ching, T., Katsch, R., and Keidser, G. (2001). Nal-nl1 procedure for fitting nonlinear  
397 hearing aids: characteristics and comparisons with other procedures. *Journal of the American academy of*  
398 *audiology*, 12(1).
- 399 Campbell, J. and Sharma, A. (2013). Compensatory changes in cortical resource allocation in adults with  
400 hearing loss. *Frontiers in systems neuroscience*, 7:71.
- 401 David, S. V., Mesgarani, N., and Shamma, S. A. (2007). Estimating sparse spectro-temporal receptive fields  
402 with natural stimuli. *Network: Computation in neural systems*, 18(3):191–212.
- 403 Decruy, L., Das, N., Verschueren, E., and Francart, T. (2018). The self-assessed Békésy procedure: validation  
404 of a method to measure intelligibility of connected discourse. *Trends in hearing*, 22:2331216518802702.
- 405 Decruy, L., Vanthornhout, J., and Francart, T. (2019). Evidence for enhanced neural tracking of the speech  
406 envelope underlying age-related speech-in-noise difficulties. *Journal of neurophysiology*, 122(2):601–615.

- 407 Decruy, L., Vanthornhout, J., and Francart, T. (2020). Hearing impairment is associated with enhanced  
408 neural tracking of the speech envelope. *Hearing Research*, page 107961.
- 409 Eggermont, J. J. (2017). Acquired hearing loss and brain plasticity. *Hearing Research*, 343:176–190.
- 410 Francart, T., van Wieringen, A., and Wouters, J. (2008). Apex 3: a multi-purpose test platform for auditory  
411 psychophysical experiments. *Journal of neuroscience methods*, 172(2):283–293.
- 412 Fuglsang, S. A., Märcher-Rørsted, J., Dau, T., and Hjortkjær, J. (2020). Effects of sensorineural hearing  
413 loss on cortical synchronization to competing speech during selective attention. *Journal of Neuroscience*,  
414 40(12):2562–2572.
- 415 Hamilton, L. S., Edwards, E., and Chang, E. F. (2018). A spatial map of onset and sustained responses to  
416 speech in the human superior temporal gyrus. *Current Biology*, 28(12):1860–1871.
- 417 Harkrider, A. W., Plyler, P. N., and Hedrick, M. S. (2006). Effects of hearing loss and spectral shaping on  
418 identification and neural response patterns of stop-consonant stimuli. *The Journal of the Acoustical Society  
419 of America*, 120(2):915–925.
- 420 Harkrider, A. W., Plyler, P. N., and Hedrick, M. S. (2009). Effects of hearing loss and spectral shaping on  
421 identification and neural response patterns of stop-consonant stimuli in young adults. *Ear and hearing*,  
422 30(1):31–42.
- 423 Heeris, J. (2014). Gammatone filterbank toolkit 1.0. <https://github.com/detly/gammatone>.
- 424 Koerner, T. K. and Zhang, Y. (2018). Differential effects of hearing impairment and age on electrophysiological  
425 and behavioral measures of speech in noise. *Hearing research*, 370:130–142.
- 426 Kraus, F., Tune, S., Ruhe, A., Obleser, J., and Woestmann, M. (2020). Unilateral acoustic degradation delays  
427 attentional separation of competing speech. *bioRxiv*.
- 428 Lenth, R. (2020). *emmeans: Estimated Marginal Means, aka Least-Squares Means*. R package version 1.4.8.
- 429 Lesenfants, D., Vanthornhout, J., Verschueren, E., Decruy, L., and Francart, T. (2019). Predicting individual  
430 speech intelligibility from the cortical tracking of acoustic-and phonetic-level speech representations. *Hearing  
431 research*, 380:1–9.
- 432 Luts, H., Jansen, S., Dreschler, W., and Wouters, J. (2014). Development and normative data for the  
433 Flemish/Dutch matrix test.
- 434 Maamor, N. and Billings, C. J. (2017). Cortical signal-in-noise coding varies by noise type, signal-to-noise  
435 ratio, age, and hearing status. *Neuroscience letters*, 636:258–264.

- 436 Maris, E. and Oostenveld, R. (2007). Nonparametric statistical testing of eeg-and meg-data. *Journal of*  
437 *neuroscience methods*, 164(1):177–190.
- 438 McClannahan, K. S., Backer, K. C., and Tremblay, K. L. (2019). Auditory evoked responses in older adults  
439 with normal hearing, untreated, and treated age-related hearing loss. *Ear and hearing*, 40(5):1106–1116.
- 440 Mirkovic, B., Debener, S., Schmidt, J., Jaeger, M., and Neher, T. (2019). Effects of directional sound  
441 processing and listener’s motivation on eeg responses to continuous noisy speech: Do normal-hearing and  
442 aided hearing-impaired listeners differ? *Hearing Research*, 377:260–270.
- 443 Nasreddine, Z. (2004). *Montreal cognitive assessment (MoCA)*. École des sciences de la réadaptation, Sciences  
444 de la santé, Université d’Ottawa.
- 445 Oates, P. A., Kurtzberg, D., and Stapells, D. R. (2002). Effects of sensorineural hearing loss on cortical  
446 event-related potential and behavioral measures of speech-sound processing. *Ear and hearing*, 23(5):399–415.
- 447 Oostenveld, R. and Praamstra, P. (2001). The five percent electrode system for high-resolution eeg and erp  
448 measurements. *Clinical neurophysiology*, 112(4):713–719.
- 449 Peelle, J. E. and Wingfield, A. (2016). The neural consequences of age-related hearing loss. *Trends in*  
450 *neurosciences*, 39(7):486–497.
- 451 Petersen, E. B., Wöstmann, M., Obleser, J., and Lunner, T. (2017). Neural tracking of attended versus  
452 ignored speech is differentially affected by hearing loss. *Journal of neurophysiology*, 117(1):18–27.
- 453 Presacco, A., Simon, J. Z., and Anderson, S. (2016). Evidence of degraded representation of speech in noise,  
454 in the aging midbrain and cortex. *Journal of neurophysiology*, 116(5):2346–2355.
- 455 Presacco, A., Simon, J. Z., and Anderson, S. (2019). Speech-in-noise representation in the aging midbrain  
456 and cortex: Effects of hearing loss. *PloS one*, 14(3):e0213899.
- 457 R Core Team (2020). *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical  
458 Computing, Vienna, Austria.
- 459 Slaney, M. (1998). Auditory toolbox. *Interval Research Corporation, Tech. Rep*, 10(1998).
- 460 Somers, B., Francart, T., and Bertrand, A. (2018). A generic eeg artifact removal algorithm based on the  
461 multi-channel wiener filter. *Journal of neural engineering*, 15(3):036007.
- 462 Tremblay, K. L., Piskosz, M., and Souza, P. (2003). Effects of age and age-related hearing loss on the neural  
463 representation of speech cues. *Clinical Neurophysiology*, 114(7):1332–1343.



- 464 Van Dun, B., Kania, A., and Dillon, H. (2016). Cortical auditory evoked potentials in (un) aided normal-hearing  
465 and hearing-impaired adults. In *Seminars in hearing*, volume 37, page 9. Thieme Medical Publishers.
- 466 Vanthornhout, J., Decruy, L., Wouters, J., Simon, J. Z., and Francart, T. (2018). Speech intelligibility  
467 predicted from neural entrainment of the speech envelope. *Journal of the Association for Research in*  
468 *Otolaryngology*, 19(2):181–191.
- 469 Verschueren, E., Vanthornhout, J., and Francart, T. (2020). The effect of stimulus intensity on neural  
470 envelope tracking. *bioRxiv*.
- 471 Voeten, C. C. (2020). *buildmer: Stepwise Elimination and Term Reordering for Mixed-Effects Regression*. R  
472 package version 1.6.

473 **Supplementary Material**

474 **Supplementary Statistical Material**

Table S.1: Results of the linear mixed model in order to assess the effects of degree of hearing loss (PTA) and age on the peak latency of  $P1_{AO}$ ,  $N1_{AO}$ ,  $N1_S$ ,  $P2_S$ . Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), degrees of freedom (df), t-Ratios and p-values are reported per fixed effect term. Participant nested in the matching factor was included as a random nested effect.

Formula: latency  $\sim$  1 + peak + PTA + age + peak:age + peak:PTA + peak:PTA:age + (1 | match/participant)

fixed effect term	$\beta$	SE	df	t-Ratio	p-value
Intercept (for P1 - acoustic onsets)	59.7545	5.7913	45.0463	10.318	p < 0.001
peak = N1 - acoustic onsets	63.1639	7.1839	46.8945	8.7925	p < 0.001
peak = N1 - spectrogram	32.1969	8.2213	48.341	3.9163	p < 0.001
peak = P2 - spectrogram	141.4255	6.1896	42.5149	22.8488	p < 0.001
Degree of hearing loss (PTA)	12.8954	3.5184	17.8724	3.6652	p = 0.0018
Age	-11.3602	9.4021	41.6263	-1.2083	p = 0.2338
peak = N1 - acoustic onsets:age	14.8838	11.4562	45.5116	1.2992	p = 0.2004
peak = N1 - spectrogram:age	18.3418	12.275	46.5449	1.4942	p = 0.1419
peak = P2 - spectrogram:age	32.7614	9.9271	41.8241	3.3002	p = 0.0020

Table S.2: Results of the linear model in order to assess the effect of degree of hearing loss on the peak latency of  $N1_{AO}$  when only HI listeners are taken into account. Estimates of the regression coefficients ( $\beta$ ), standard errors (SE), t-Ratios and p-values are reported per fixed effect term.

Formula:  $N1_{AO}$ -latency  $\sim$  1 + PTA

$R^2_{adj} = 0.4125$ ,  $F = 6.618$  on 1 and 7 df,  $p = 0.03688$

fixed effect term	$\beta$	SE	t-Ratio	p-value
Intercept	60.1035	31.0682	1.9346	0.0943
Degree of hearing loss (PTA)	1.8454	0.7174	2.5725	0.0369