



Cochlear influences on the timing of the speech-ABR

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The speech-ABR as a correlate of temporal processing

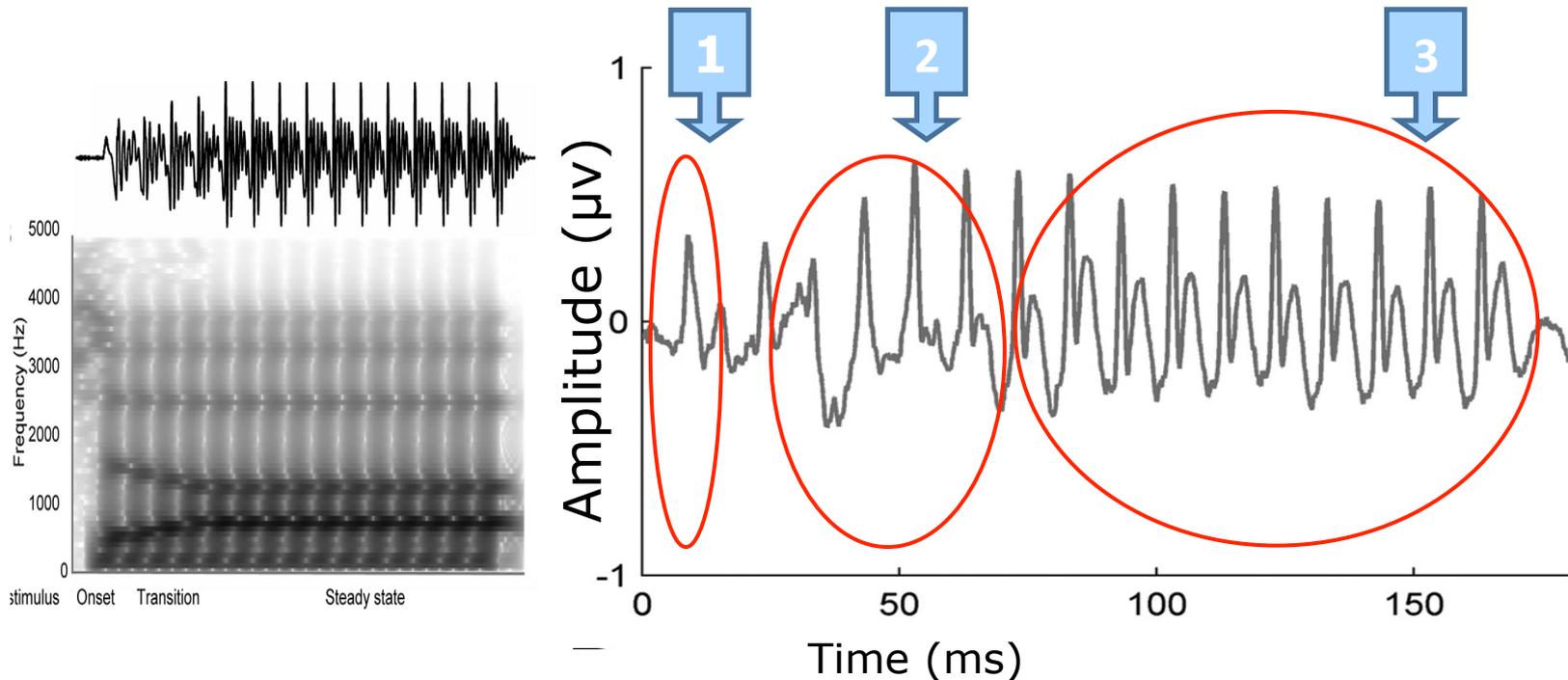
- It has been suggested that temporal processing deficits in the central auditory pathway may be implicated in:
 - difficulties perceiving speech-in-noise (Pichora-Fuller & Souza, 2003)
 - language and literacy difficulties (Cohen-Mimran & Sapir, 2007)
- A neurophysiological correlate of this temporal processing deficit has been proposed in the speech-evoked auditory brainstem response (speech-ABR).
- The speech-ABR has been hailed as a 'biomarker' for indexing temporal processing at the brainstem (Johnson et al., 2007).

Speech-ABR to [da] stimulus

Onset response
~6 ms

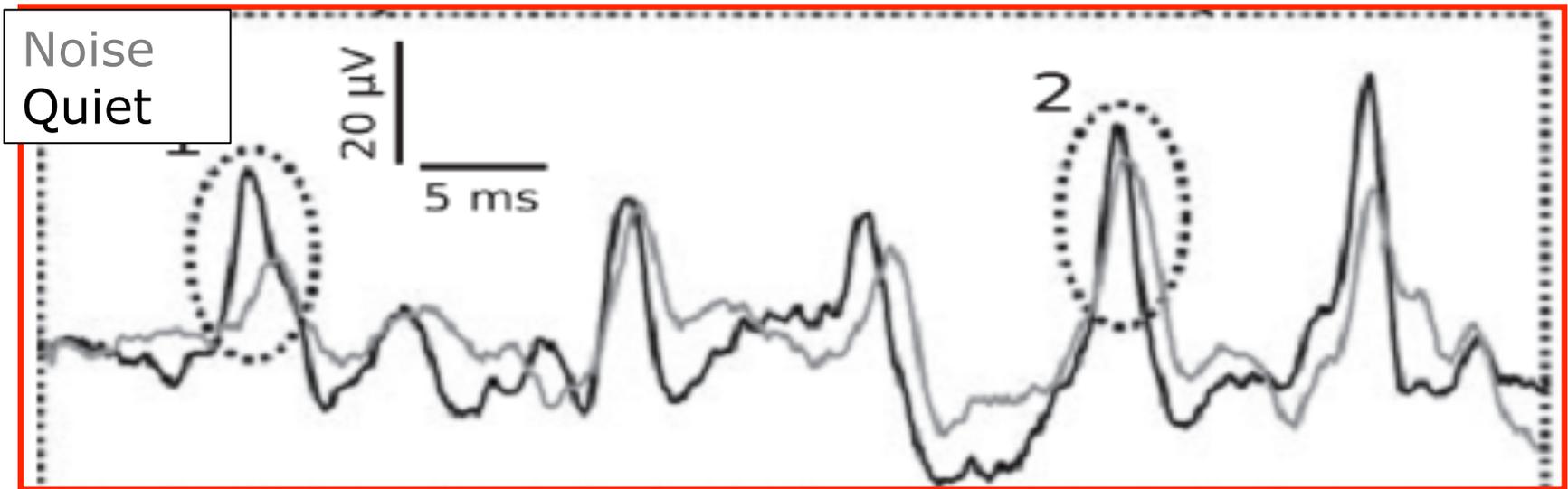
Formant transition
~20-60 ms

Steady-state
~60-170 ms



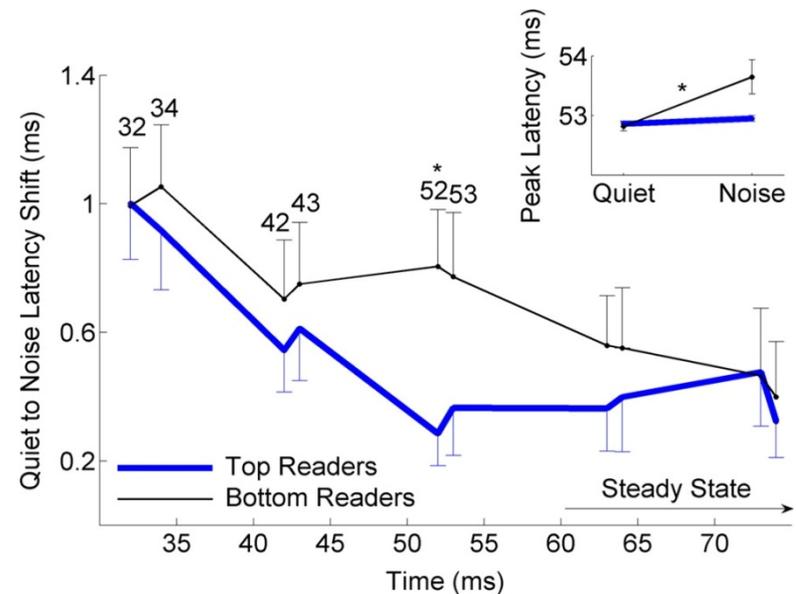
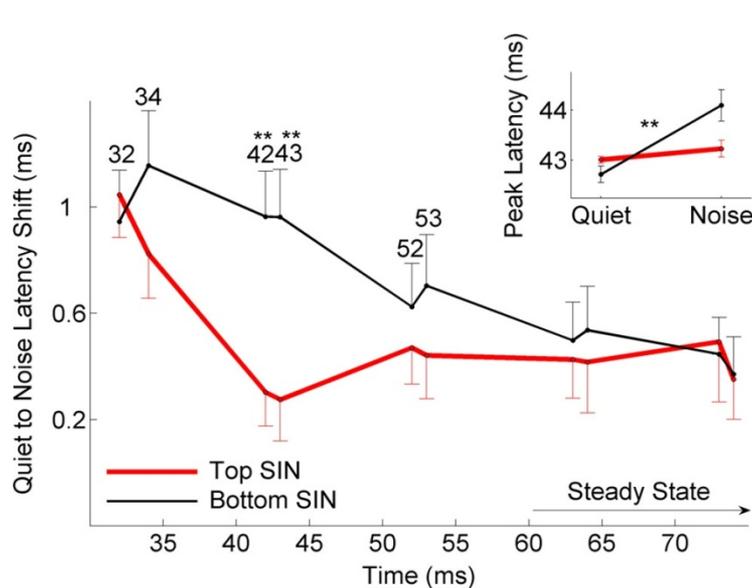
The speech-ABR reflects neural phase-locking to the envelope of the stimulus.

Speech-ABR to [da] stimulus



Background noise causes reductions in peak amplitude and increases in peak latency (Parbery-Clark et al., 2011). Interpreted as a loss of neural synchrony.

Delayed speech-ABR timing relates to poor reading ability and SIN perception



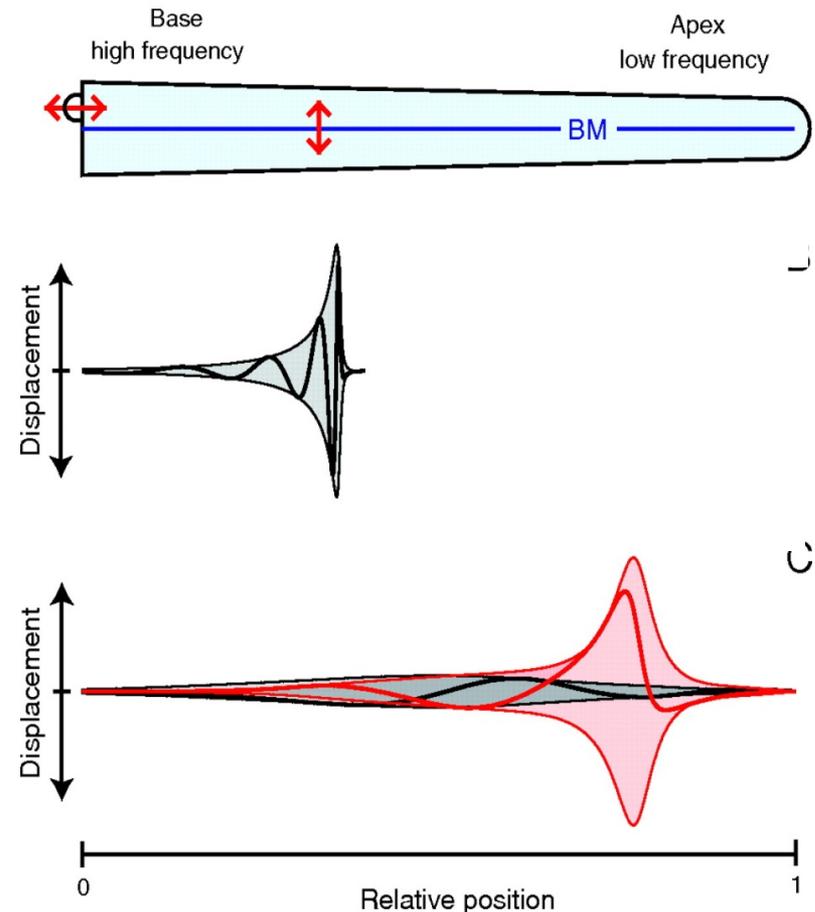
- In certain groups, despite normal audiograms, the timing of the speech-ABR is delayed (Anderson et al., 2010).
- This delay has been interpreted to reflect reduced neural synchrony and timing precision at the brainstem.

Normal audiograms do not tell us everything about cochlear processing

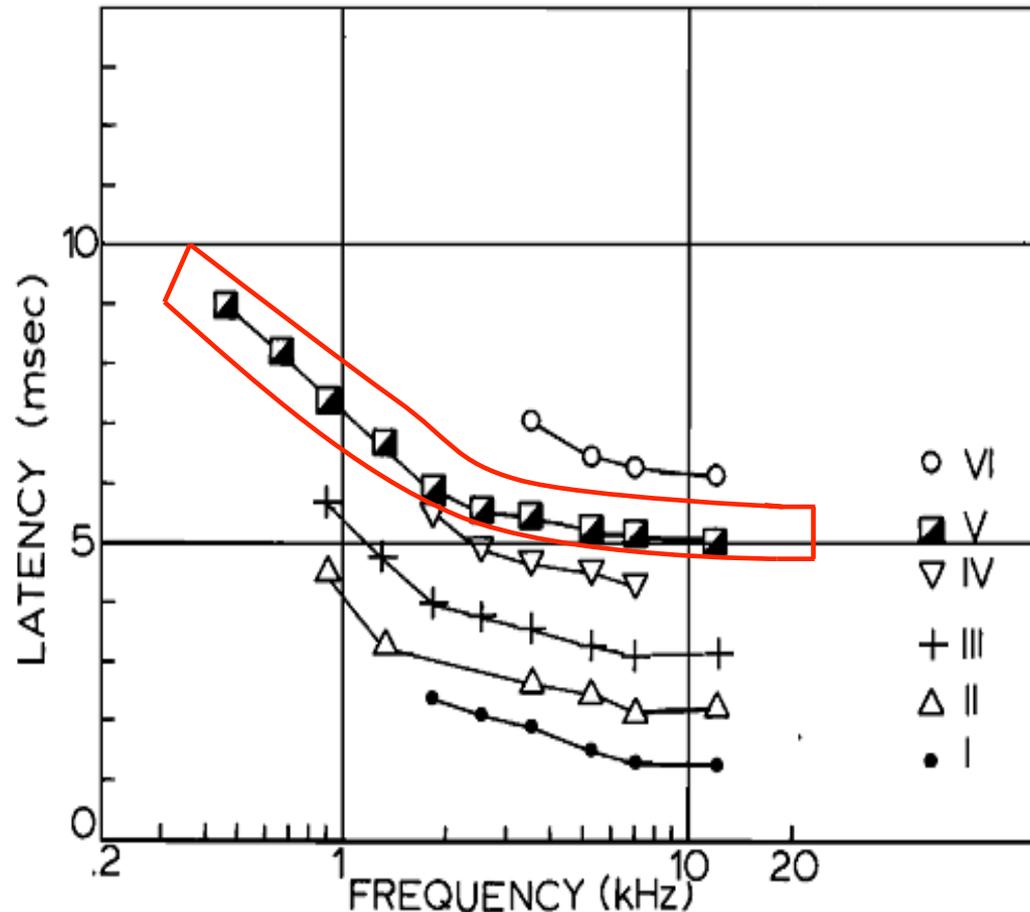
- Although a neural basis for these differences is assumed, no neural mechanisms have been directly linked to human ABR timing.
- A peripheral basis is ruled out due to the presence of normal hearing.
- Evidence suggests that suprathreshold differences in cochlear processing exist despite normal hearing (Ruggles et al., 2011; Kujawa & Liberman, 2009).
- Outer hair cell status shows considerable variability in normally-hearing listeners and is associated with speech-in-noise perception differences (Dubno et al., 2007; Sommers & Gehr, 2010).

Cochlear processing is important for the formation of brainstem responses

- The importance of cochlear processing on brainstem responses has been well-documented (Dau, 2003).
- Of particular importance for the click-ABR is the cochlear response time (CRT).
- A response at low frequencies will occur several milliseconds after a response at high frequencies.



Click-ABR latency as a function of cochlear frequency origin



- ABR peak latency increases with decreasing centre frequency (Don & Eggermont, 1978).
- This reflects the increase in CRT from high to low frequencies.

The impact of frequency-specific CRT differences on ABRs

CRT changes are due to the narrowing of cochlear filters from high to low frequencies, which leads to increasing filter build-up time (Don et al., 1998).

1. These cochlear delays are preserved in the latency of click-ABR wave V, which can increase by over 3 ms (Burkard & Hecox, 1983).
2. Activity is less synchronous towards lower frequencies and responses from neighbouring regions cancel out in the ABR.
3. As a result, wave V mainly reflects activity from mid-high frequency regions of the cochlea, even when the stimulus contains lower frequencies (Dau et al, 2003).

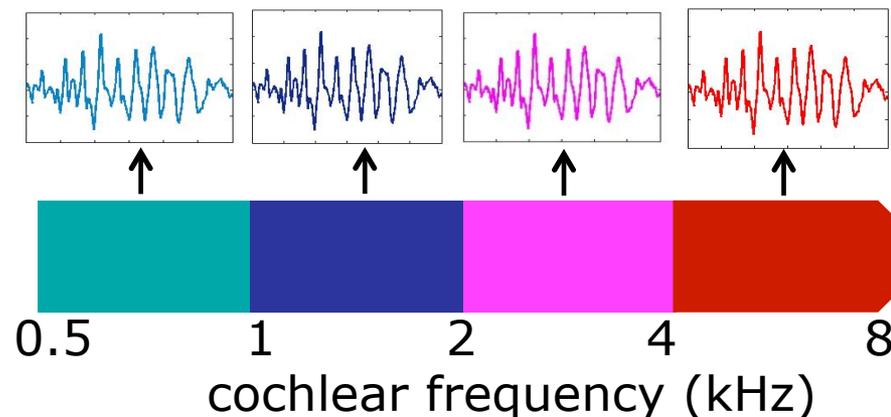
Experiment 1: Aim & Hypotheses

Aim: To test if the speech-ABR shows a similar dependence on CRT as the click-ABR.

1. Speech-ABR latency increases with decreasing cochlear centre frequency according to a non-linear function derived for click-ABR wave V data (Strelcyk et al, 2009).
2. The speech-ABR is a linear sum of contributions from all frequency regions, but low frequency contributions will be attenuated as a result of phase cancellation.
3. Latency of frequency-delimited speech-ABRs is correlated to filter bandwidth at the corresponding frequency.

Experiment 1: Method

- **Participants:** 26 normally-hearing adults (aged 18-39) with normal click-ABRs and no known language or learning difficulties.
- **Stimuli:** Synthetic [da] syllables presented in alternating polarity.
- **Electrophysiology:** Speech-ABRs recorded in quiet and with high-pass masking noise to record responses from cochlear regions **0.5-1**, **1-2**, **2-4**, and **4-8** kHz (centre frequencies of **0.7** kHz, **1.4** kHz, **2.8** kHz and **5.7** kHz).

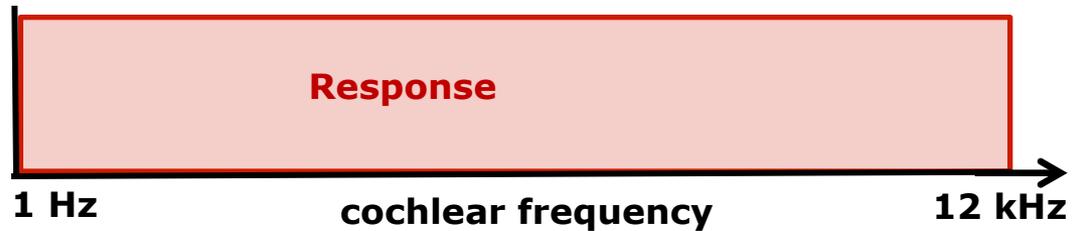


Experiment 1: Method

- **Auditory filter bandwidths:** Measured at 0.5, 1, 2 and 4 kHz using a modified version of the simultaneous notched noise method (Glasberg & Moore, Cambridge). Notch widths = 0.0, 0.1, 0.2, 0.3.
- **Data analysis:** Broadband response cross-correlated with frequency-specific responses; time point at max correlation taken as the latency difference. Cross-spectrum used for amplitude.
- Non-linear mixed effects modelling to compare latencies to non-linear click-ABR latency function derived by Strelcyk et al (2009) $t(i,f) = a + bc^{0.93-i} f^{-d}$. Linear mixed effect modelling for amplitude data.
- Correlations performed between frequency-specific speech-ABR latencies and auditory filter estimates at the corresponding frequency.

High-pass subtractive-masking technique

1. Unmasked response =



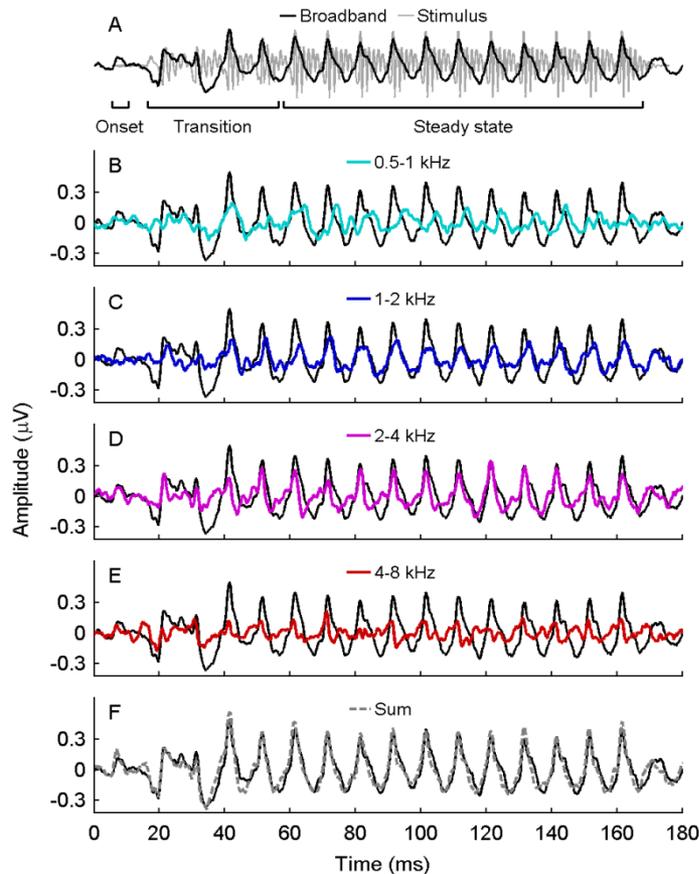
2. Response masked by A =



3. Response masked by B =

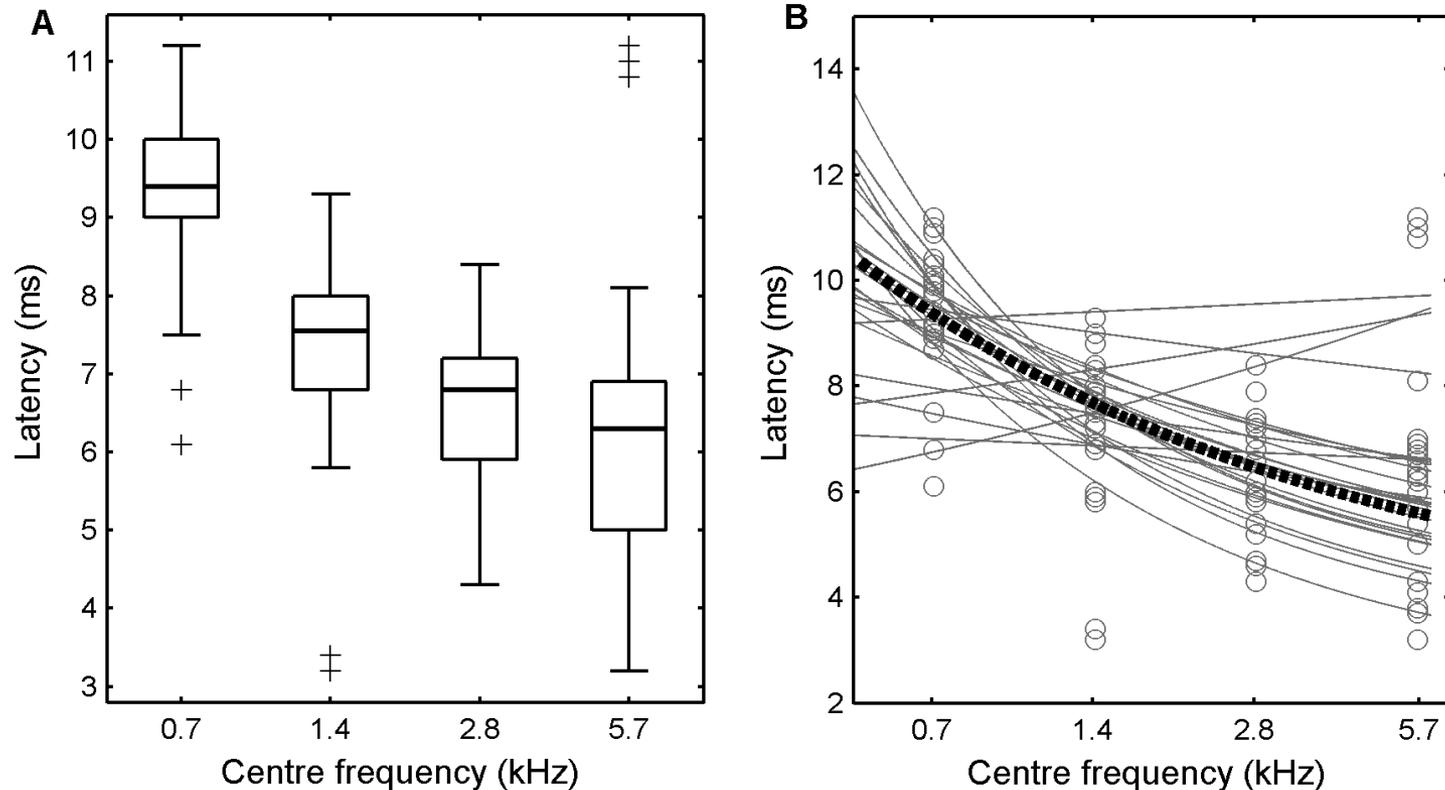


Results: Derived-band grand averages



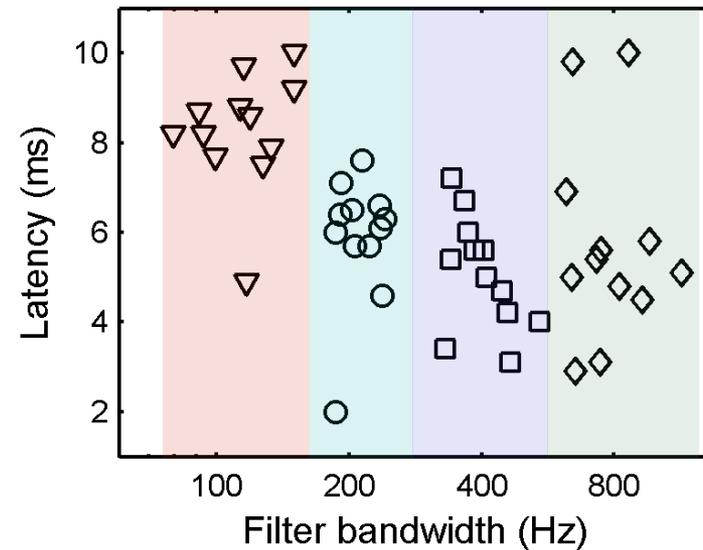
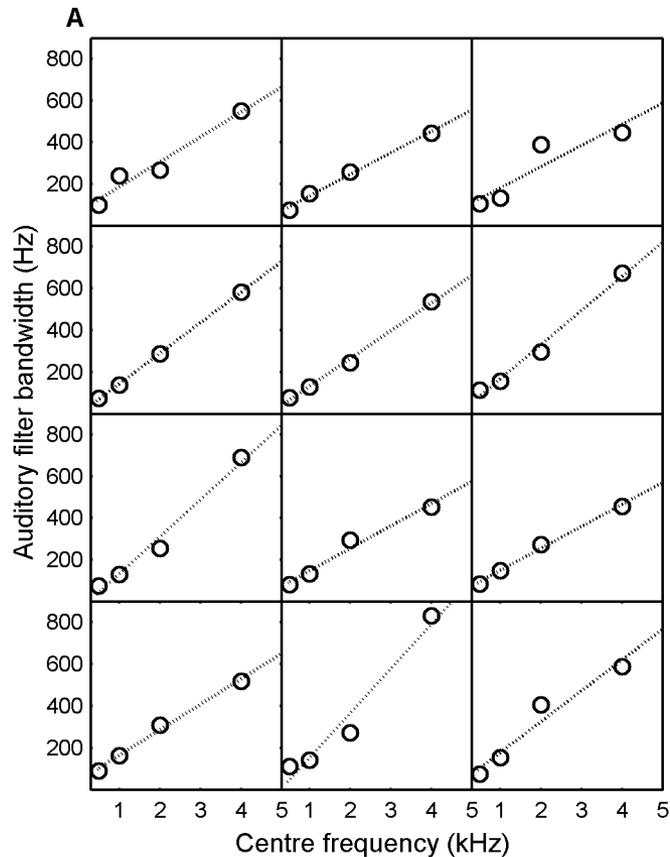
- All derived-bands showed evidence of a speech-ABR.
- Response latencies increased with decreasing derived-band frequency.
- Derived-band responses summed together to recreate the overall response.

Results: Derived-band latency functions



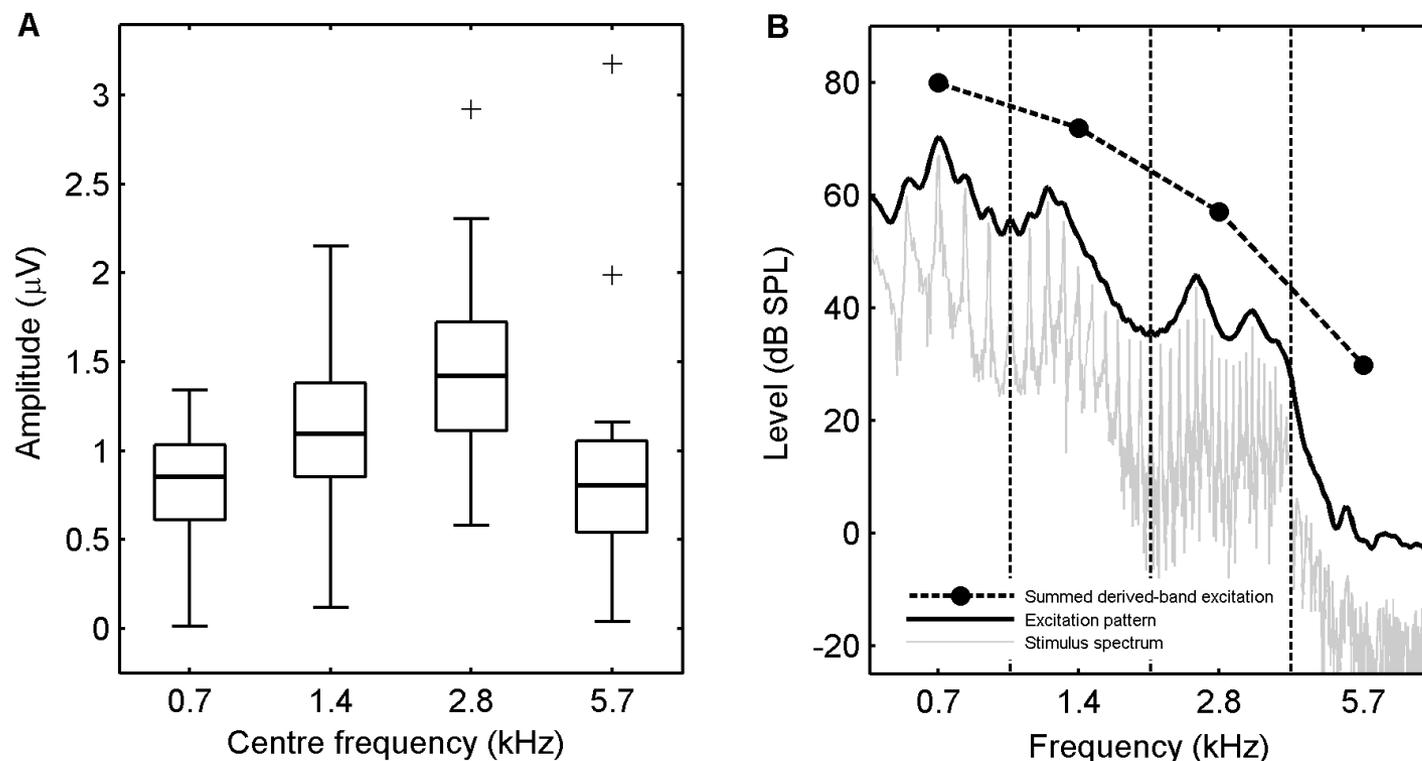
- Individual data (grey circles) and fits (grey lines) and mean population fit (black line) to Strelcyk et al. (2009) function. Frequency dependent parameter (d) estimated as 0.49, cf. 0.5 for Strelcyk et al. ($p < 0.001$).

Results: Relationship between derived-band latency and filter bandwidth



- No relationships observed between filter width and derived-band latency at the corresponding frequency ($p > 0.05$).

Results: Derived-band amplitude as a function of frequency



- Highest amplitude in 2.8 kHz region and smallest amplitudes at the lowest frequency (due to phase cancellation) and highest frequency (due to minimal stimulus energy). Significant effect of frequency ($p < 0.001$).

Experiment 1: Summary

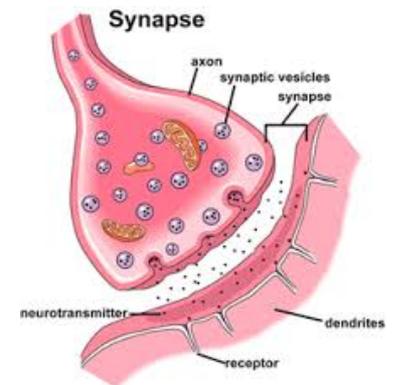
1. The speech-ABR reflects a sum of the responses from a broad range of cochlear regions, which each respond with their own delay.
2. Derived-band latency changes with cochlear frequency origin in a manner compatible with the click-ABR wave V.
3. Speech-ABR latency therefore depends on the relative contributions of different cochlear frequency regions.
4. Auditory filter bandwidth did not show a relationship with derived-band latency.

Experiment 2

The effect of within-band cochlear masking on the speech-ABR.

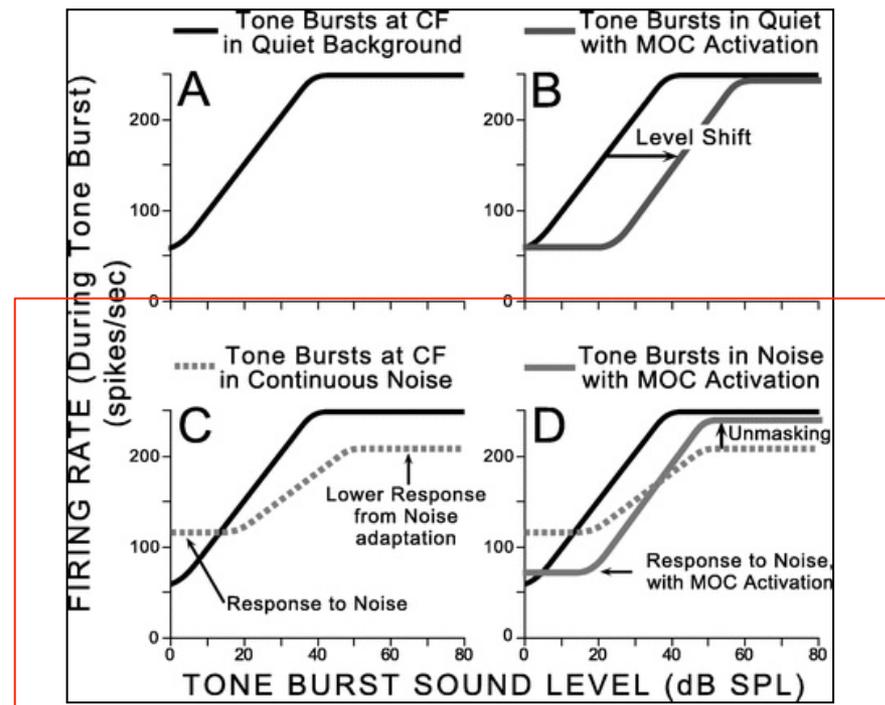
Neural adaptation influences brainstem response latency

- One neural mechanism known to lead to brainstem latency increases in noise is neural adaptation, particularly at the inner hair cell-auditory nerve (IHC-AN) junction.
- Adaptation is believed to result from synaptic depletion and somatic after-hyperpolarization (Hawkins & Kniazuk, 1950).
- This increases the threshold of receptor activation in the IHC-AN synaptic cleft, leading to a delayed auditory nerve response.
- This delay is then inherited by ABR latency (Kramer & Teas, 1982a,1982b; Krishnan & Plack, 2009).



Neural adaptation is influenced by the medial olivocochlear reflex

- Adaptation is affected by the medial olivocochlear reflex (MOCR), which via OHCs reflexively reduces cochlear gain.
- Noise-activation of the MOC may lead to the MOC attenuating the response to the background noise more than the response to the signal (Guinan, 2006).
- This reduces adaptation and restores the neurotransmitter supply in a process known as MOC 'anti-masking' (Guinan, 2006).



MOCR strength may relate to speech-ABR latency shifts in noise

- In this way, cochlear masking and cochlear amplification are under feedback control from the MOC system, which is activated in response to noise maskers.
- Noise-induced latency shifts at the brainstem caused by cochlear masking may therefore be associated with MOCR strength.
- The same groups with atypical speech-ABRs have been separately demonstrated to show abnormal MOCR function (Sanches & Carvello, 2006).
- A direct relationship between speech-ABR latency in noise and MOCR strength in normally-hearing listeners has also been demonstrated (de Boer & Thornton, 2008).

Experiment 2: Aims

Aims

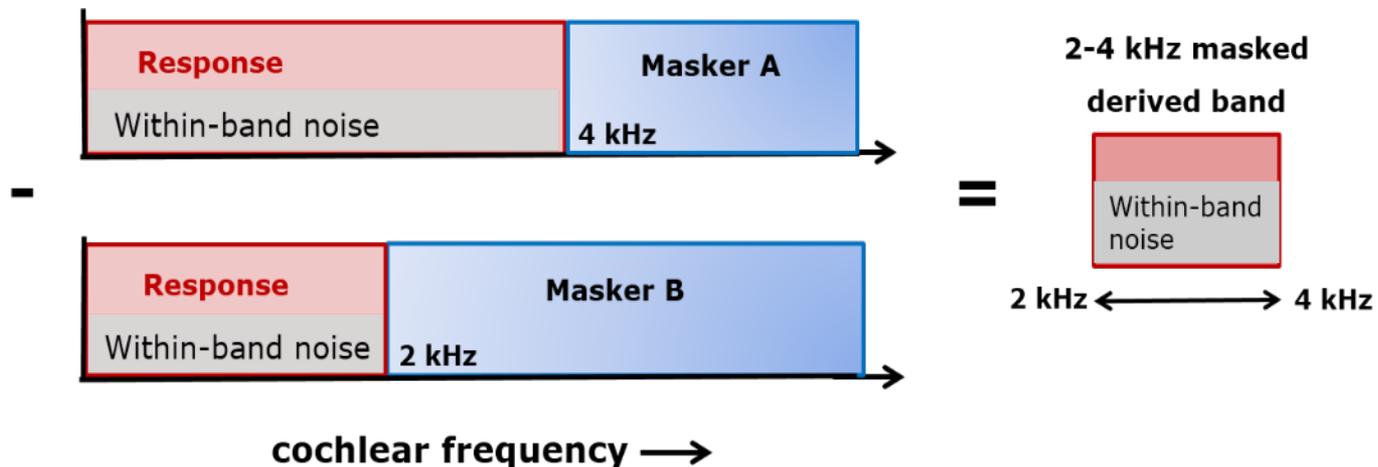
1. To assess the effect of neural adaptation on derived-band speech-ABRs in noise, without a cochlear place confound.
2. To determine the extent to which this contributes to the effects of noise-masking on the overall, broadband speech-ABR.
3. To test how the effects of background noise masking on the speech-ABR associate with MOCR strength.

Experiment 2: Method

Participants: 12 normally-hearing adult listeners (aged 18-40).

Stimuli: Synthetic [da] syllables as in Exp. 1.

Electrophysiology: Responses recorded using high-pass masking as in Exp. 1 with the addition of within-band noise masking (50 & 60 dB per ERB [N50 N60]).

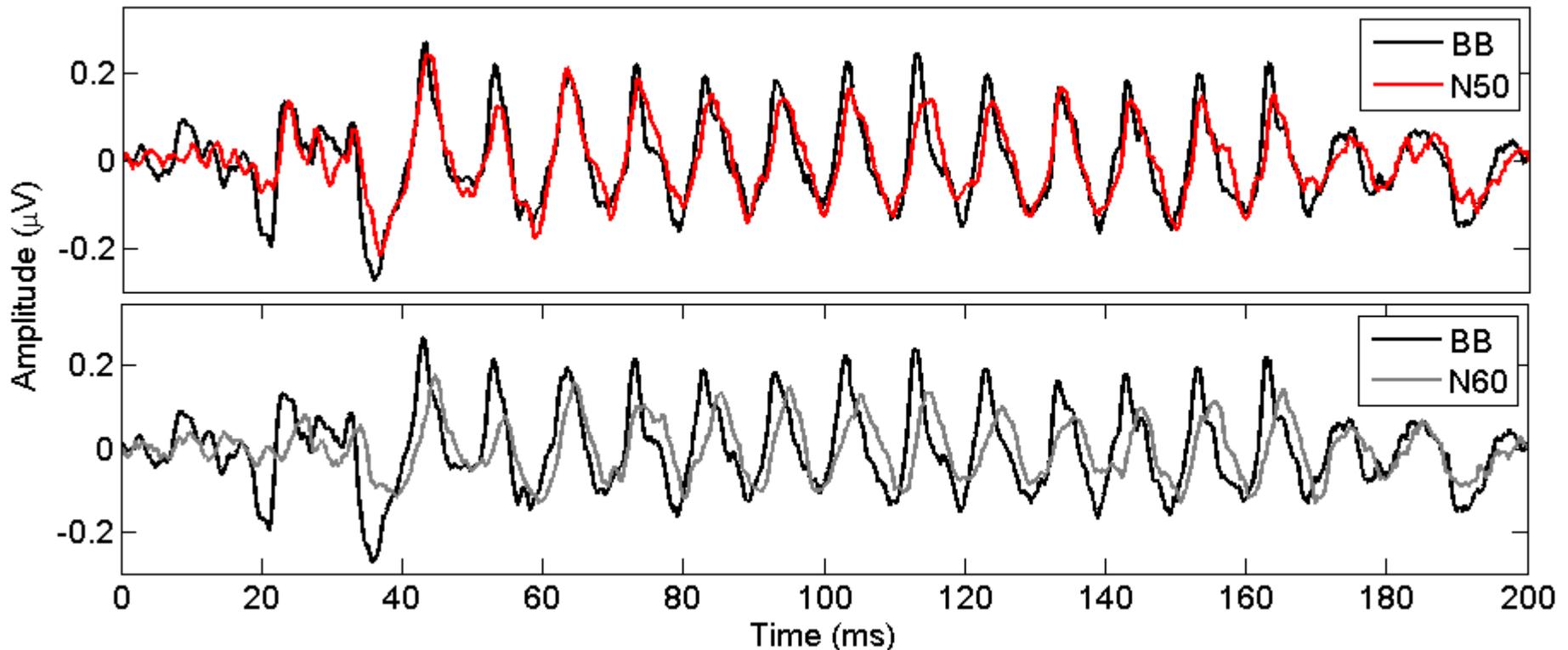


Experiment 2: Method

MOCR measurement:

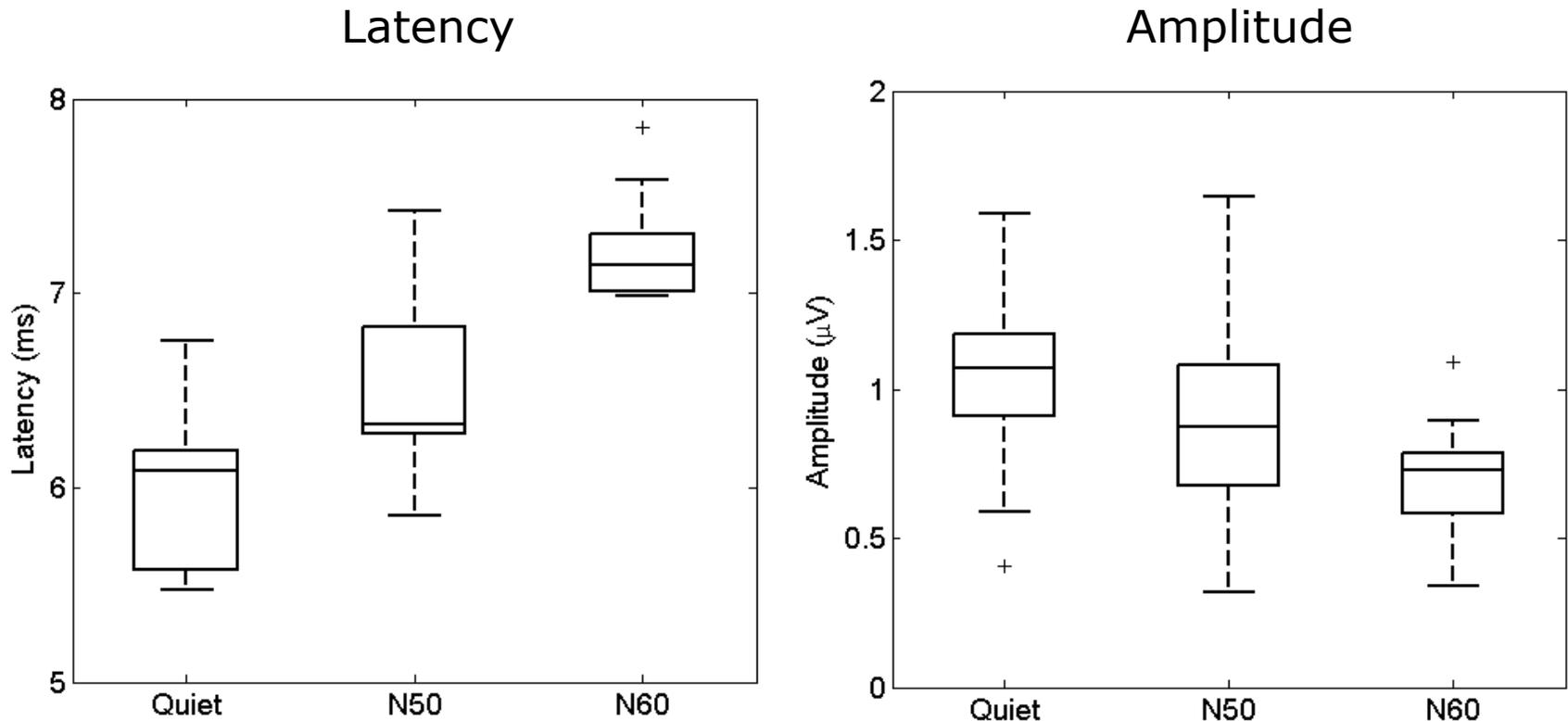
- Otoacoustic emissions (OAEs) measured to clicks (60 and 70 dB SPL) in quiet and contralateral broadband noise (70 dB SPL).
- MOCR measure: input-output (I/O) function slope =
OAE amplitude @ 70 dB – OAE amplitude @ 60 dB / 10 dB level difference.
- An increase in I/O slope in noise, referred to as I/O slope suppression, would be assumed to reflect a reduction in cochlear gain through noise-evoked MOC activation (de Boer et al., 2011; de Boer & Thornton, 2007; de Boer & Thornton, 2008).
- Therefore, a more negative difference between I/O slope in quiet – I/O slope in noise = stronger MOCR.

Results: The effect of noise masking on broadband speech-ABRs



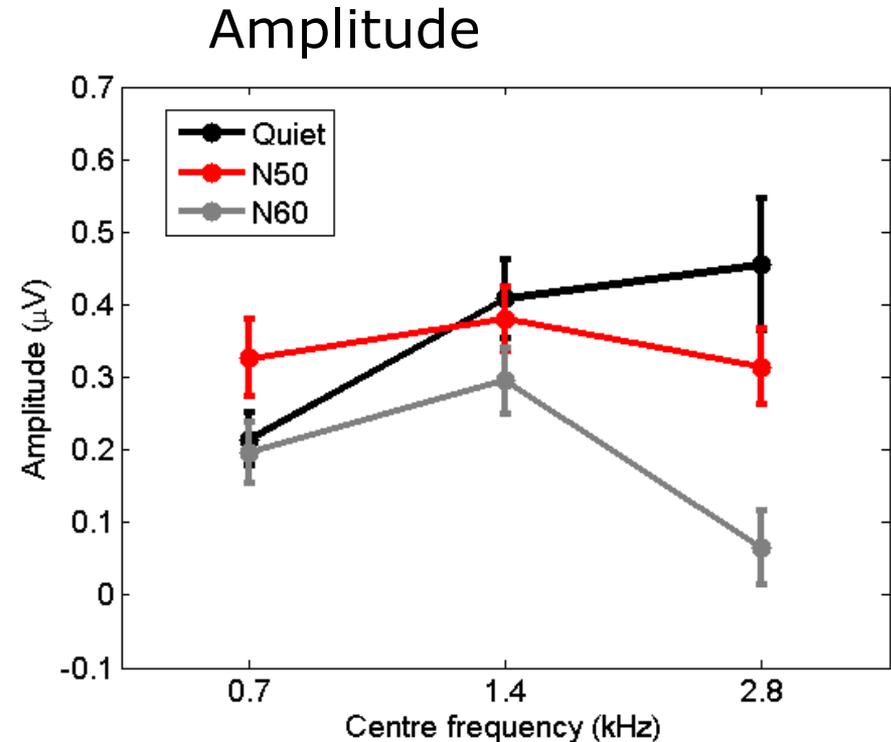
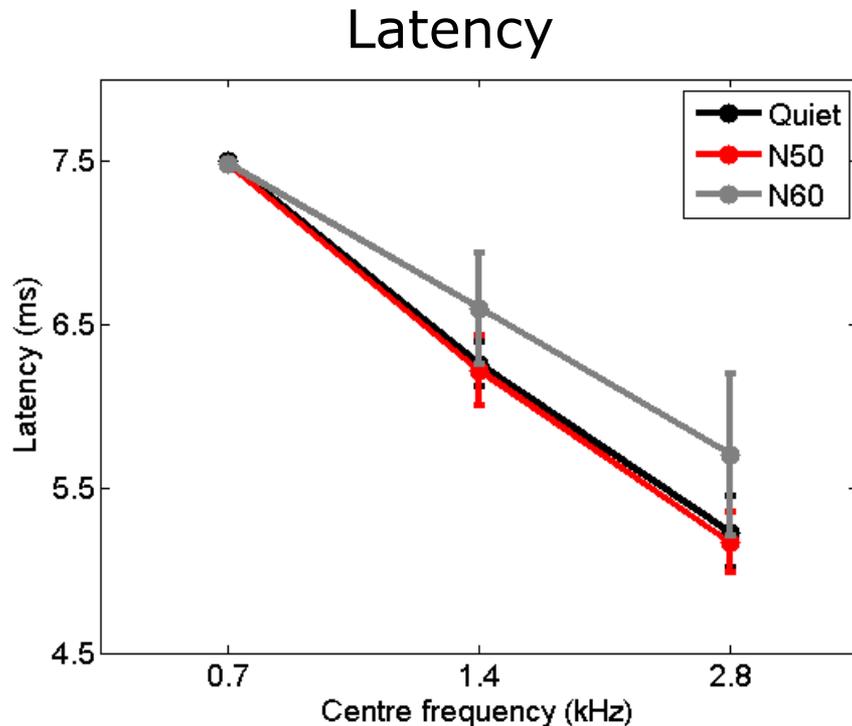
- Speech-ABR latency increases and amplitude decreases with increasing background noise level.

Results: The effect of noise masking on broadband speech-ABRs



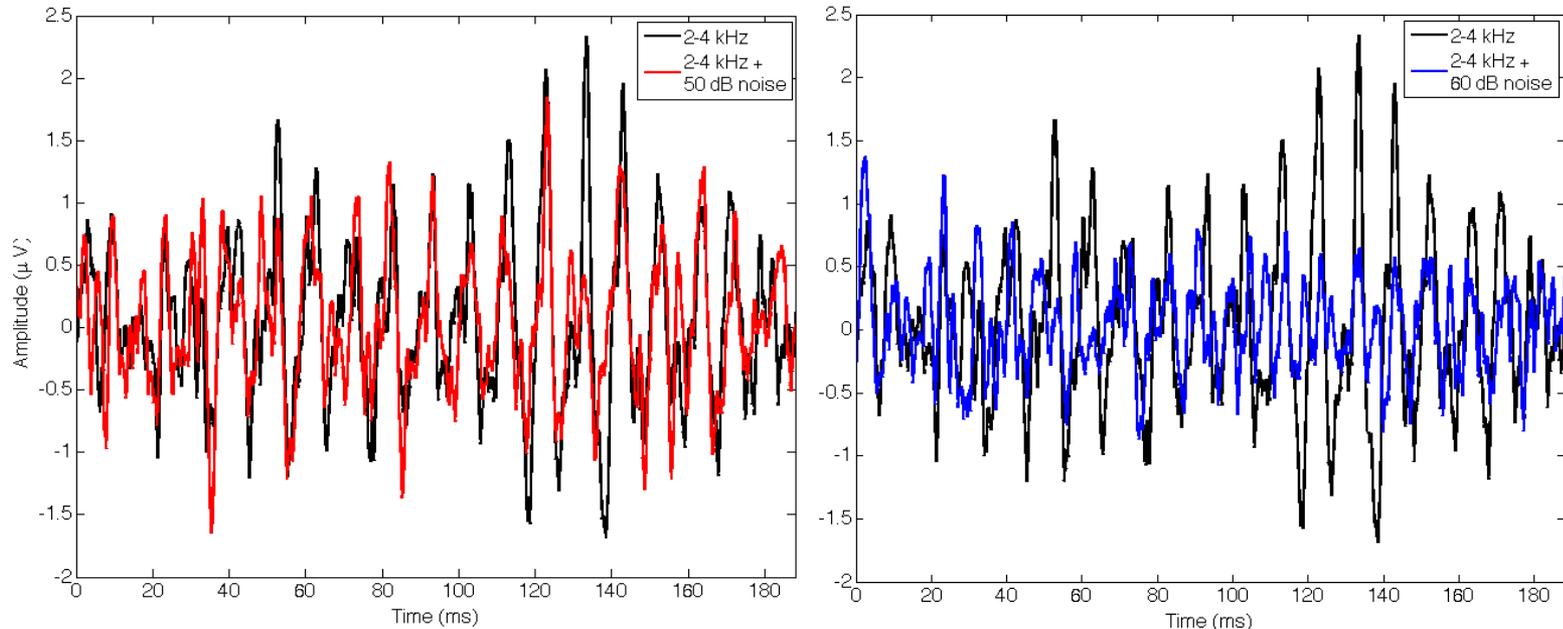
- Significant effect of noise on broadband speech-ABR latency ($F(2,22) = 65.5, p < 0.001$) and amplitude ($F(2, 22) = 30.5, p < 0.001$).

Results: The effect of noise masking on derived-band speech-ABRs



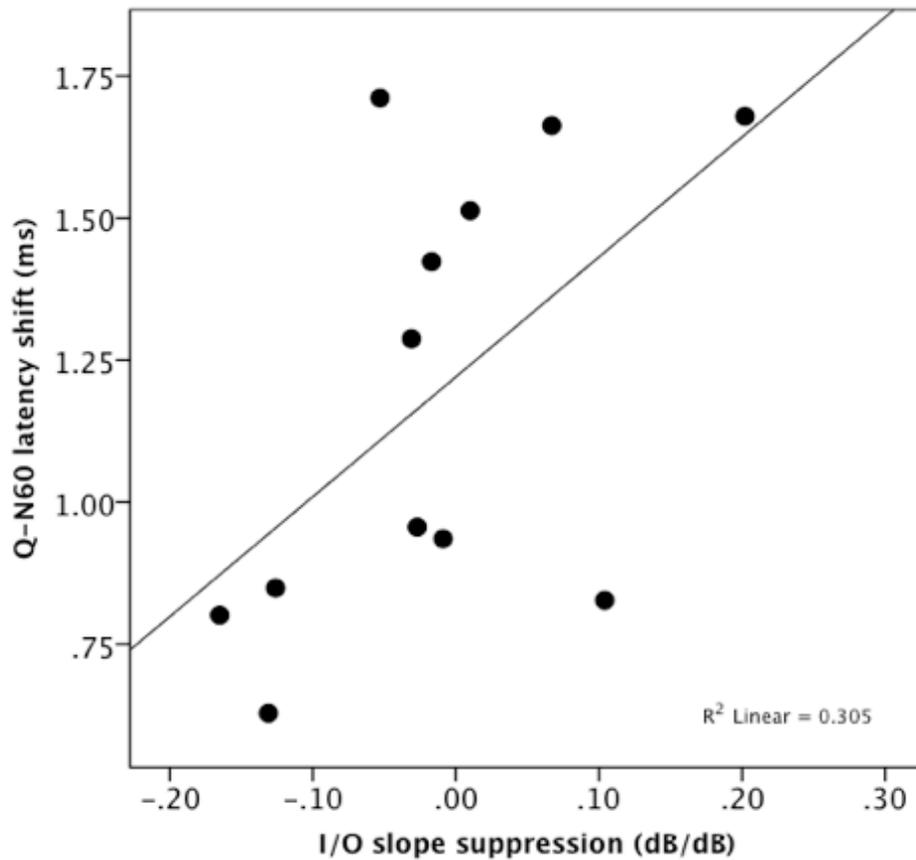
- No significant effect of noise on latency.
- Significant noise * frequency interaction on amplitude ($F(1,93)=8.72, p=0.004$) and main noise effect ($F(1,93)=11.85, p=0.009$).
- Related to a significant loss of amplitude at 2.8 particularly in N60.

Results: 2-4 kHz derived-band speech-ABRs in noise



- Significant reduction in amplitude from 2-4 kHz region in 60 dB noise (blue) compared to 2-4 kHz 50 dB noise (red).
- The speech-ABR is a linear sum of cochlear contributions. A reduction from 2-4 kHz would alter the relative balance and increase overall latency.

Results: Role of the MOCR in brainstem latency in noise

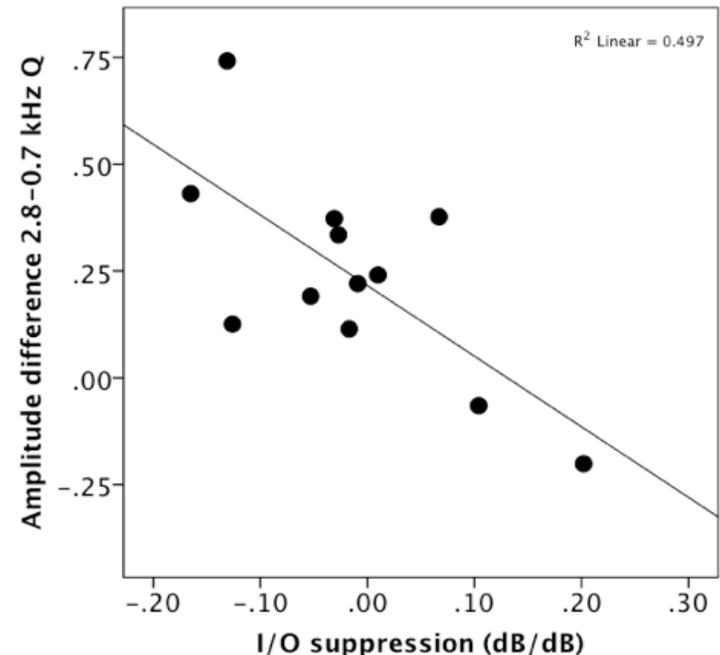


- Correlation between slope suppression and latency shift in highest level of noise ($r=0.55$, $p=0.063$).
- I/O slope suppression in derived-band amplitude LME did not improve the model.
- However, there was a trend towards a 3-way $\text{freq} \times \text{noise} \times \text{I/O slope suppression}$ interaction ($F(1,90)=3.20$, $p=0.08$)

Results: Role of the MOCR in brainstem latency in quiet

- To explore the 3-way interaction, the relationship between slope suppression and amplitude was assessed separately in N60 and Quiet:
 - No significant N60 derived-band amplitude * I/O slope suppression interaction.
 - Significant interaction between derived-band amplitude in quiet * I/O slope suppression ($F(1,22)=5.69$, $p=0.03$).

- Interaction based on greater differences between 0.5-1 and 2-4 kHz contributions to the speech-ABR in quiet associating with a stronger MOCR.



Experiment 1&2: Summary

1. Speech-ABR latency is dependent upon cochlear spectral processing.
2. After controlling for cochlear place changes, there was little evidence of neural adaptation affecting latency in noise.
3. Instead, data suggest that the amplitude and latency changes of masked speech-ABRs may result from a cochlear place mechanism.
4. In particular, a specific loss of contribution from the 2-4 kHz region in noise, which is the most dominant contribution in quiet (Exp 1).

Experiment 2: Summary cont.

5. Evidence of a correlation between overall speech-ABR latency shift in the highest level of noise and MOCR strength (stronger MOCR = less latency shift).
6. Correlation potentially associated with the difference between contributions from 0.5-1 kHz and 2-4 kHz regions in quiet.
7. This may reflect a shared dependency on third factor, such as outer hair cell status.

Implications

- Speech-ABR timing reflects both cochlear and neural influences.
- Differences in speech-ABR latencies may result from differences in cochlear contributions to the response.
- These differences may also be dependent upon individual differences in suprathreshold cochlear processing.
- It is important to consider these factors when interpreting speech-ABR latencies that vary between
 - i) experimental conditions
 - ii) populations
 - iii) different regions of the response

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