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Neurocognitive Brain Changes in Hearing Loss: Effects of Early Intervention with Hearing Aids

Anu Sharma, PhD

University of Colorado, Boulder



Anu Sharma, PhD

Anu Sharma, Ph.D. is Professor in the Department of Speech Language and Hearing Science and Fellow in the Institute for Cognitive Science and Center for Neuroscience at University of Colorado, Boulder, USA. Her research focuses on examining brain and behavioral outcomes in children and adults with hearing loss who are fitted with hearing aids and/or cochlear implants. She has over 75 scientific publications and she has given over 200 invited and keynote addresses on her research including the Carhart Memorial Lecture at the American Auditory Society the Marion Downs lecture twice at the American Academy of Audiology and the Ted Evans Lecture at the British Academy of Audiology. Her research is funded by the United States National Institutes of Health.

Disclosures

- **Presenter Disclosure:** Financial: Dr. Sharma is employed by the University of Colorado at Boulder. Dr. Sharma's research has been funded by the National Institutes of Health and the Hearing Industry Research Consortium. She received an honorarium for this course. Non-financial: Dr. Sharma has no relevant non-financial relationships to disclose.
- **Content Disclosure:** This learning event does not focus exclusively on any specific product or service.
- **Sponsor Disclosure:** This course is presented by the American Auditory Society in partnership with AudiologyOnline.

Learning Outcomes

After this course, participants will be able to:

- Describe cortical neurophysiological changes in hearing loss.
- Describe cognitive changes in hearing loss.
- Discuss the cortical changes from early intervention with hearing aids and cochlear implants in persons with hearing loss.

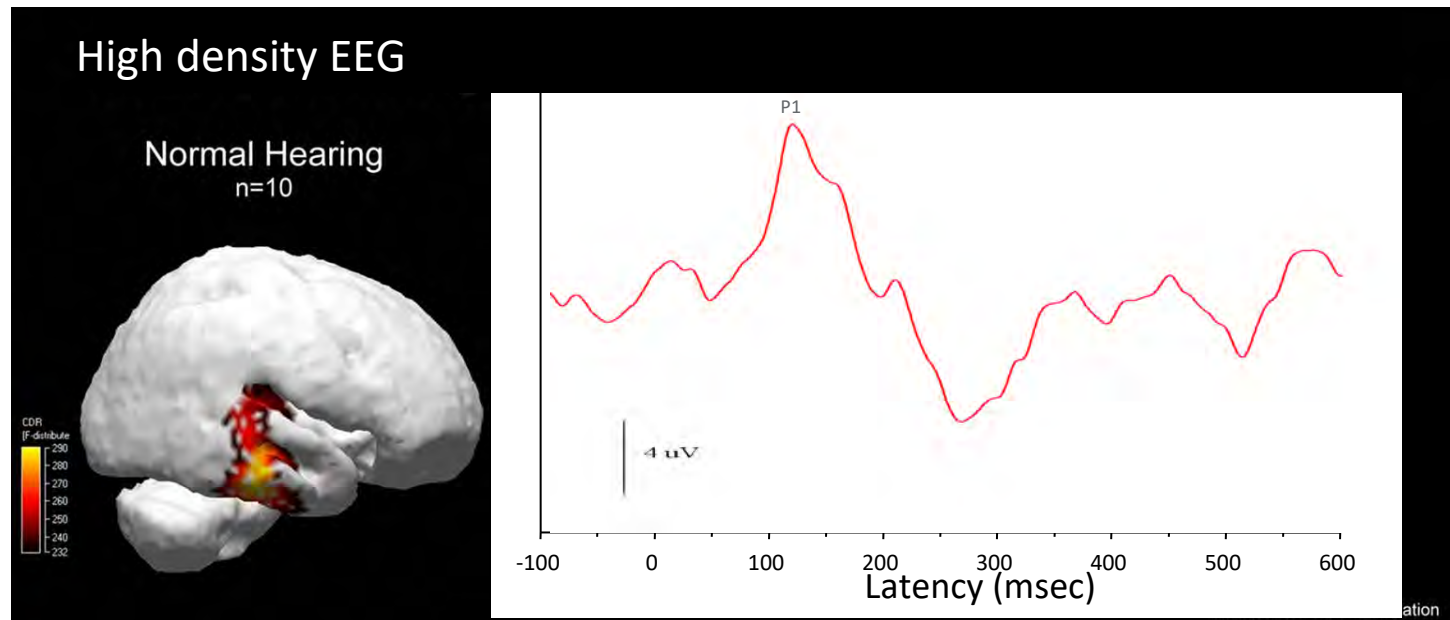
What are the central consequences of hearing loss?

Developmental Neuroplasticity

Neuroplasticity in age-related
hearing loss

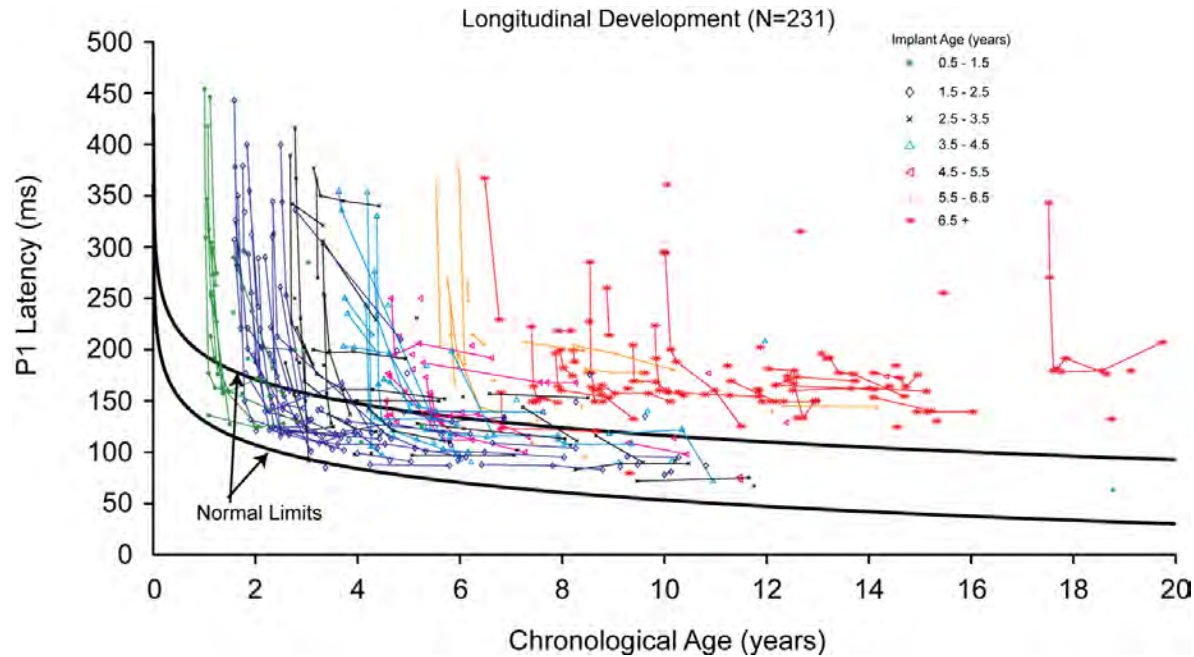
A basic tenet of neuroplasticity is that the brain will change or re-organize following sensory deprivation.

P1 Cortical Auditory Response Biomarker

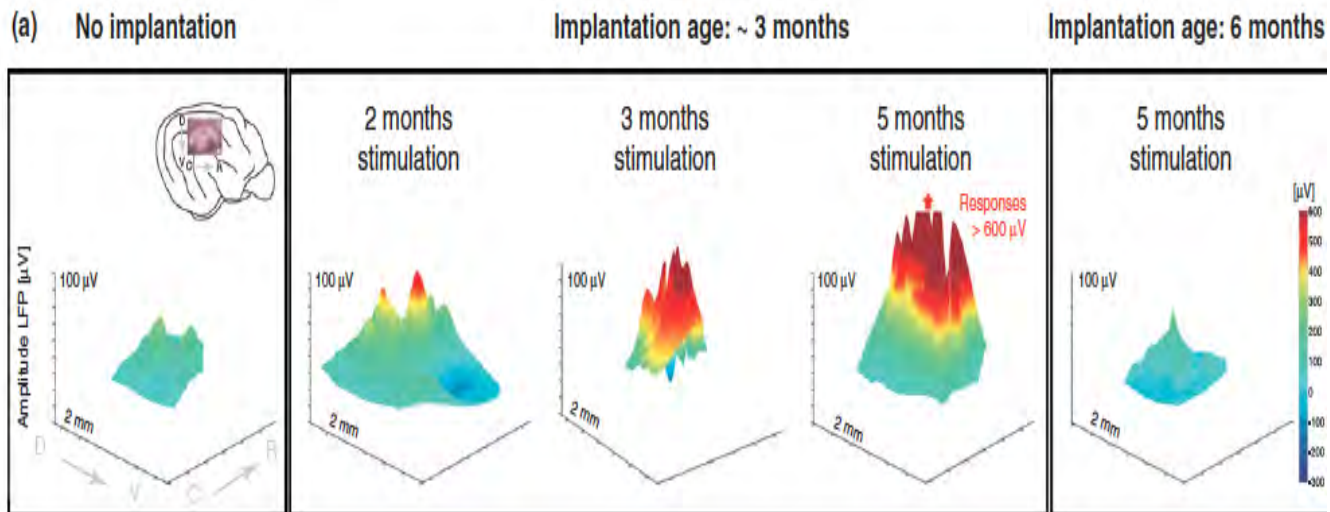


P1 has input from primary auditory cortex and cortico-cortical loops.

Individual Developmental Trajectories n=(231)

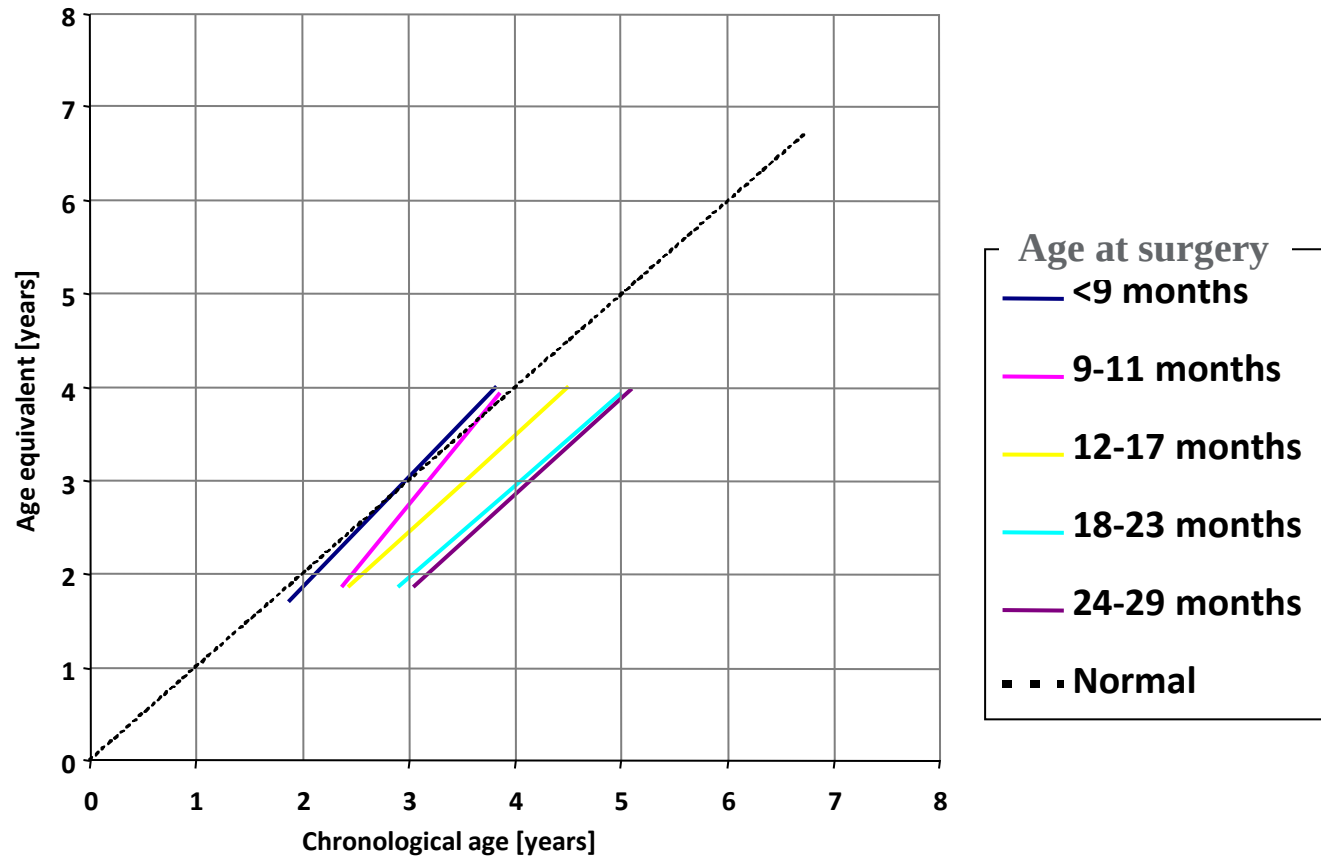


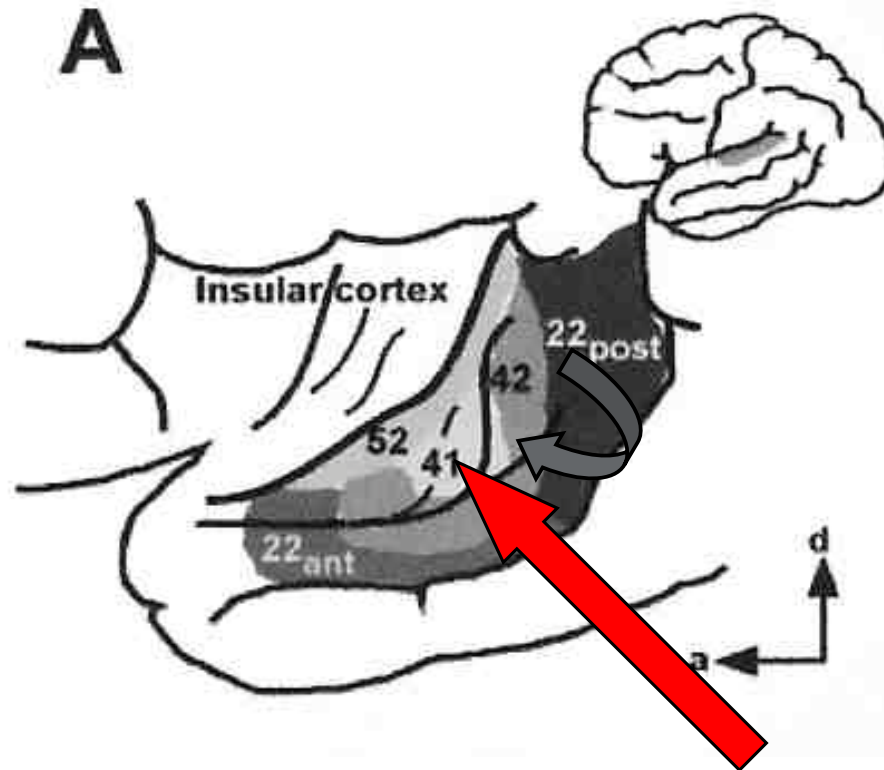
Cortical Activation Map in Primary Auditory Cortex: Deaf Cats



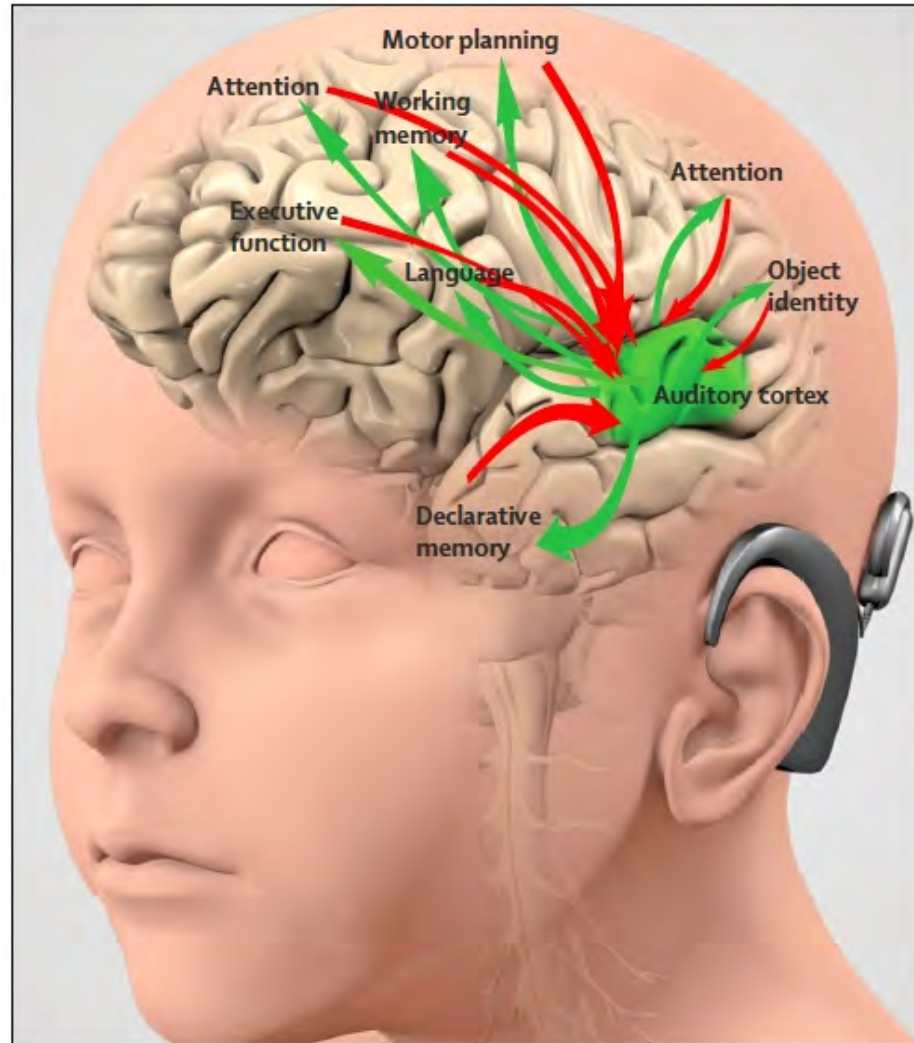
Implantation early within an early sensitive period (*best by age 1yr*) allows for the most favorable outcomes for children with cochlear implants.

Spoken Language Progression: Reynell III



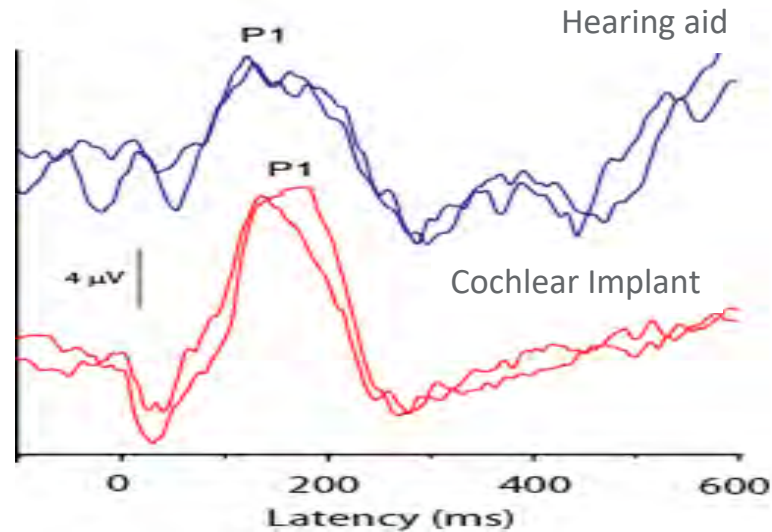


Hearing loss
affects
downstream
cognitive
function.

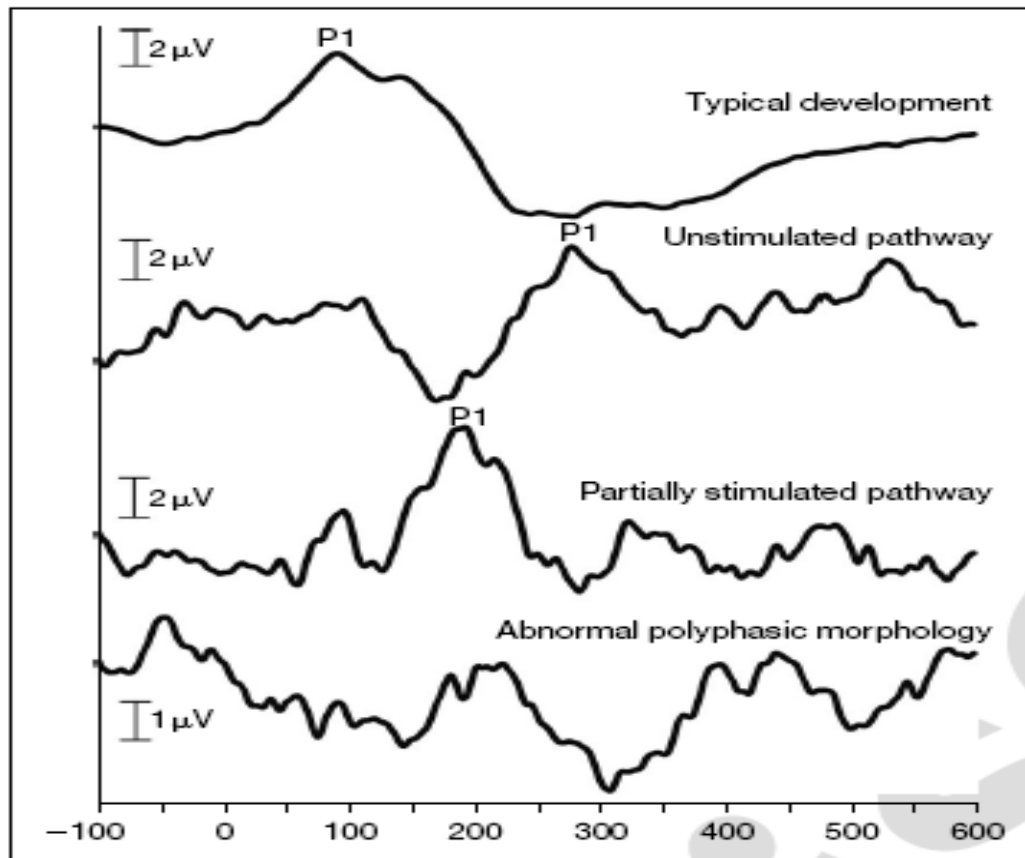


Can we use central auditory system
neuroplasticity to help with clinical
decision making in newborns and infants?

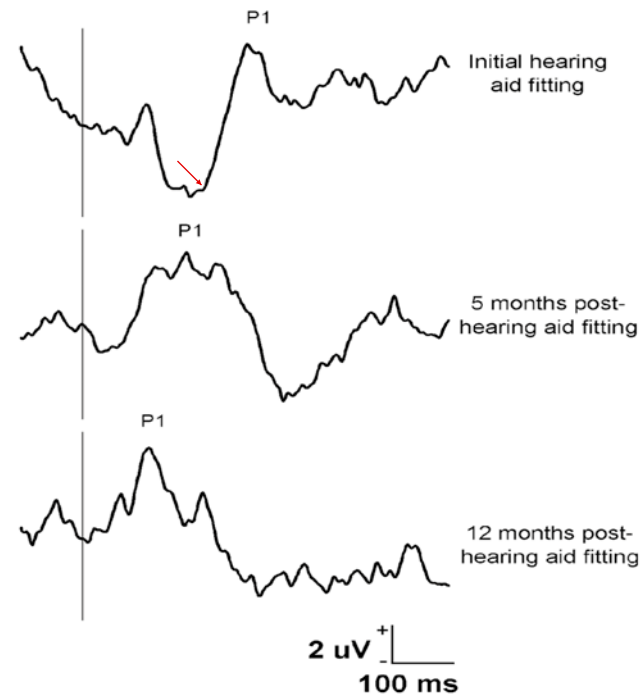
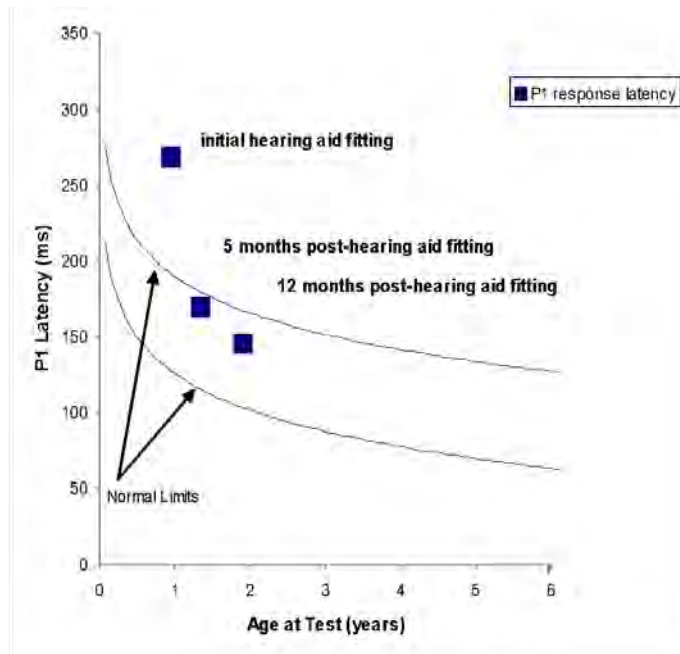
P1 biomarker of auditory cortical maturation



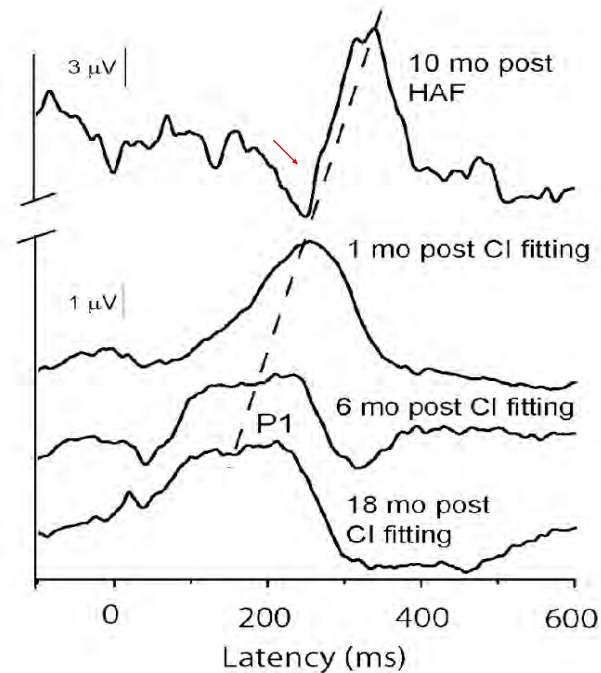
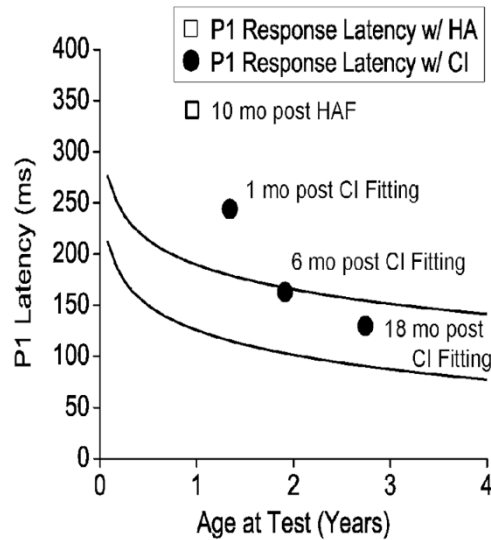
Auditory cortical responses examined in several hundred children with normal hearing and hearing loss



Neuroplastic changes following hearing aid fitting in infants



Neuroplastic changes following cochlear implant fitting in infants



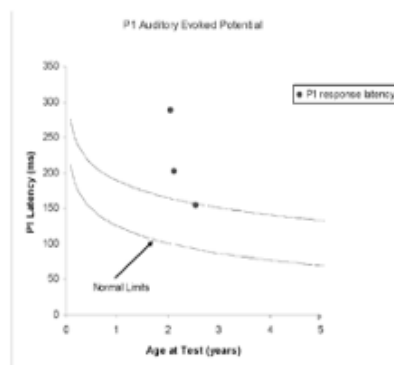
Name: 68280
CCCD#: 3-5-2002
Date of Birth: 3-25-2004, 4-22-2004, 9-24-2004
Date(s) of Test: 3-25-2004
Date of Cochlear Implant Initial Stimulation: 3-25-2004
Implant Type: Med-El
Approx. Implant Experience at Test: Initial stimulation, 1 month, 6 months

P1 Auditory Evoked Potential Testing

Auditory evoked potentials reflect EEG activity in response to sound stimulation. The latency of the P1 auditory evoked potential reflects synaptic propagation through the thalamo-cortical portions of the central auditory pathways. P1 latencies are considered to be an index of the maturation of the central auditory pathways.

The P1 response was recorded non-invasively using a 5-electrode montage with Cz as the active electrode and the mastoid serving as the reference. A separate eye channel (lateral outer canthus referenced to superior outer canthus) was used for eye-blink rejection and a common ground electrode was placed on the forehead. The response was elicited using a "ba" speech stimulus presented through a speaker. A's cochlear implant speech processor was set at a level through which the sound could be heard at a comfortable loudness level.

Results



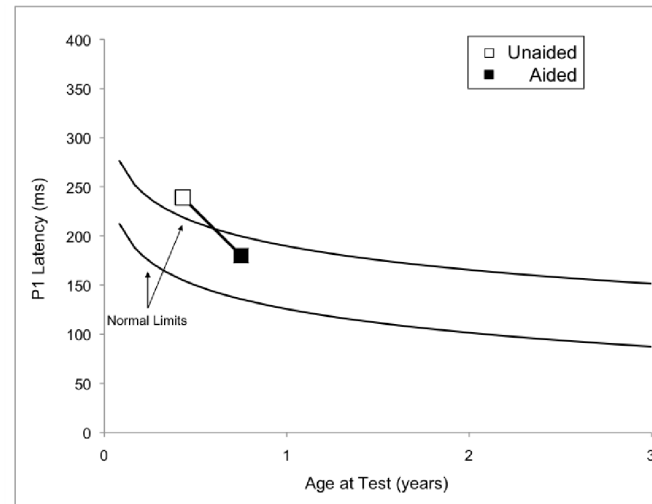
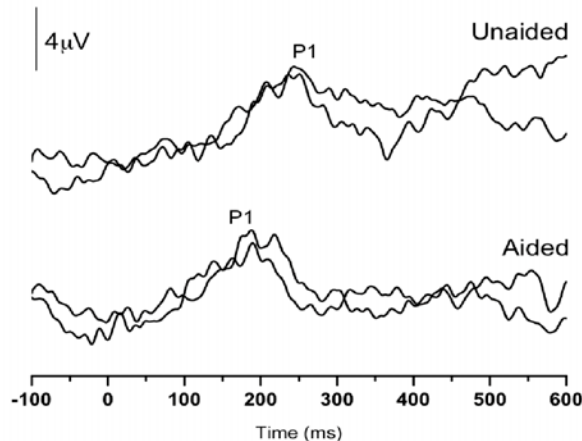
Impressions

Abigail has shown a 124 msec decrease in P1 latency since the time of her initial cochlear implant fitting in March 2004. This decrease in P1 latency has moved Abigail just within the expected latency range for a normal hearing child of a similar age. An age-appropriate P1 response latency suggests intact development of auditory thalamo-cortical areas.

Recommendations

- 1) Abigail should continue to be monitored electrophysiologically at regular intervals (i.e. every 6-9 months), and behaviorally to assess acquisition of auditory and speech skills.
- 2) Abigail should continue to wear her cochlear implant speech processor for all waking hours, except when bathing or swimming.

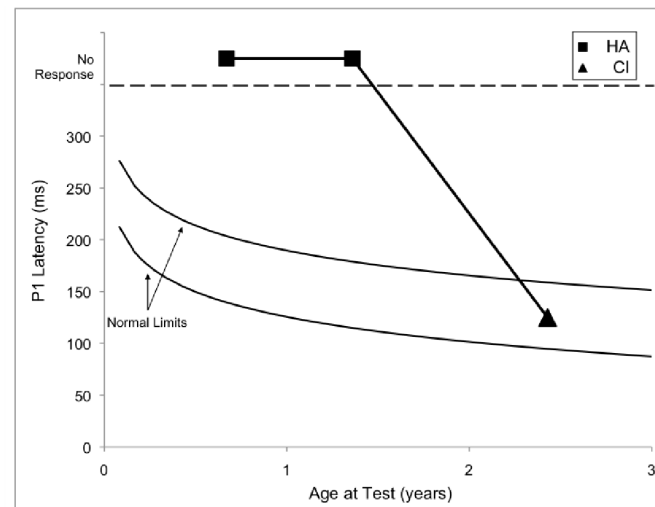
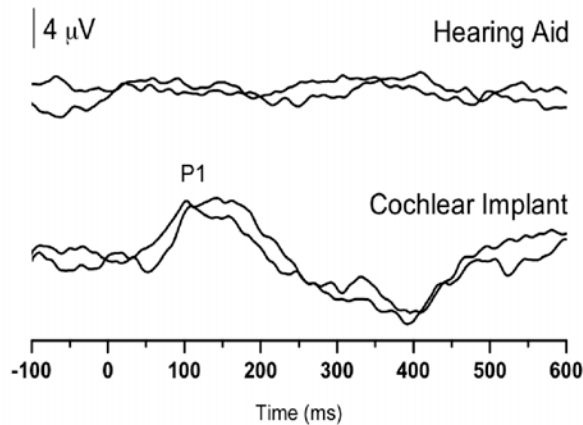
Auditory Neuropathy Spectrum Disorder Case A



IT MAIS: 39/40 with hearing aid experience

Performing well with hearing aids, will continue to monitor

Auditory Neuropathy Spectrum Disorder Case B



P1 was absent with hearing aids and normal with cochlear implants.

SUMMARY

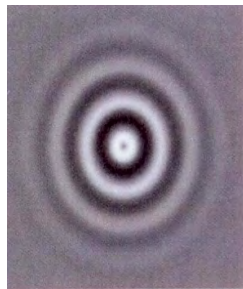
Cortical biomarkers are useful in assessing neuroplastic changes in the clinical management of children with hearing loss who have multiple disabilities and ANSD.

Compensatory Plasticity

A form of neuroplasticity that occurs with sensory deprivation is compensatory cross-modal plasticity.

For example, in deafness, auditory cortical areas can be recruited by the visual modality for visual processing.

Visual cross-modal plasticity in cochlear implanted children

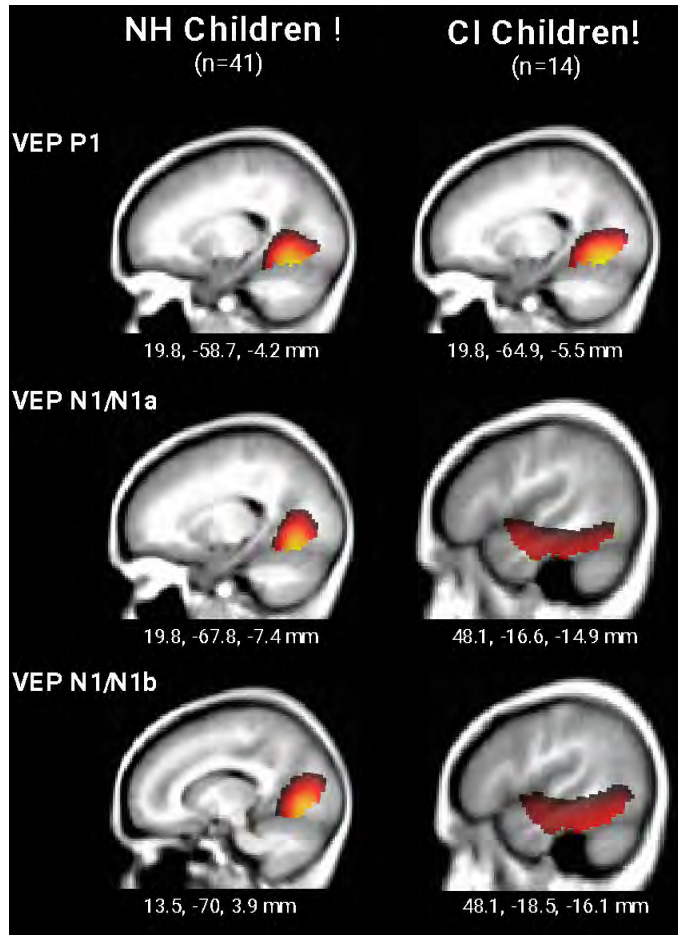


500 ms



500 ms

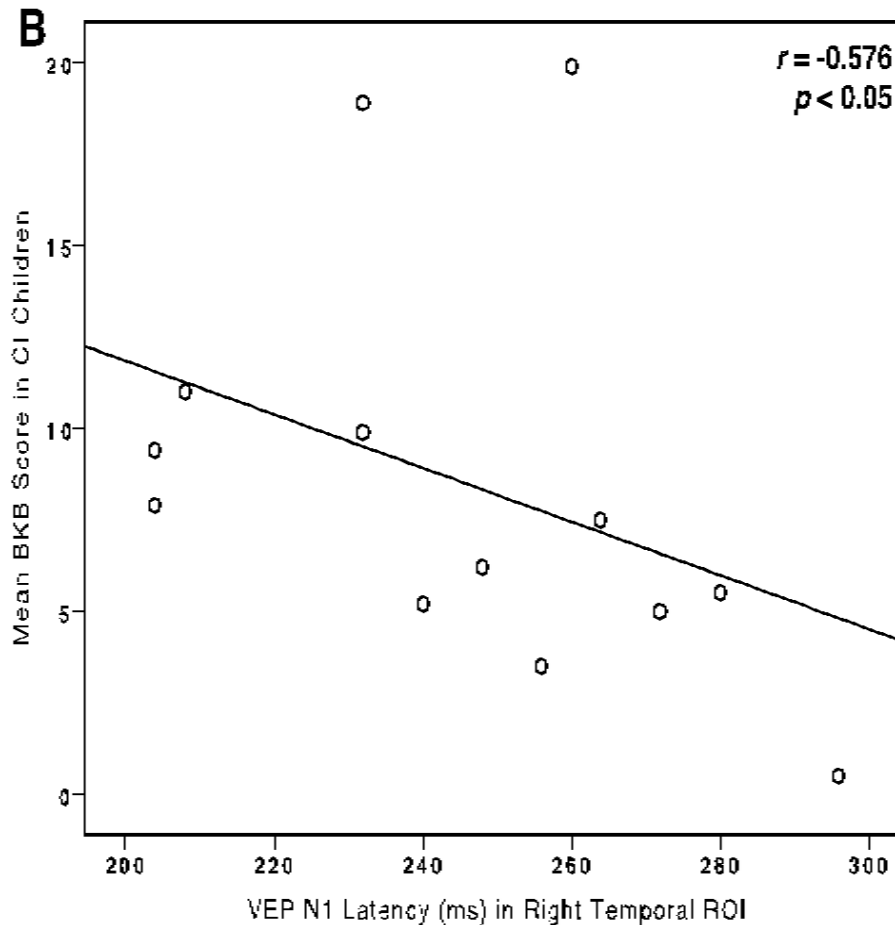




Current Density Reconstructions

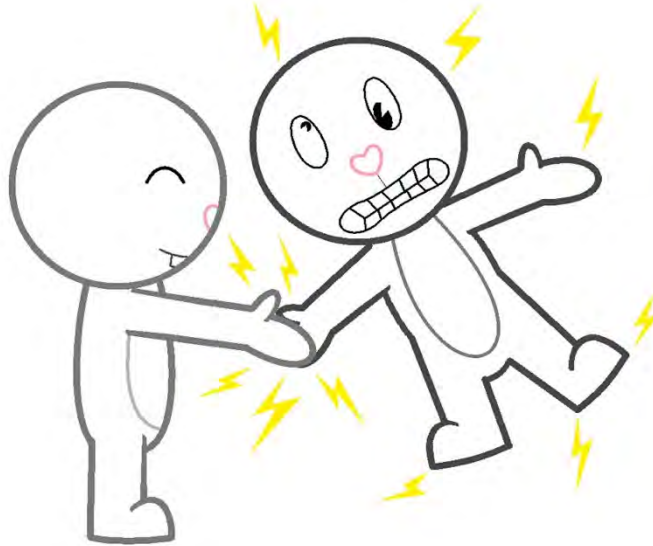
Suggestive of cross-modal plasticity between visual and auditory systems.

Worse performance with the cochlear implant is associated with greater cross-modal plasticity from vision.

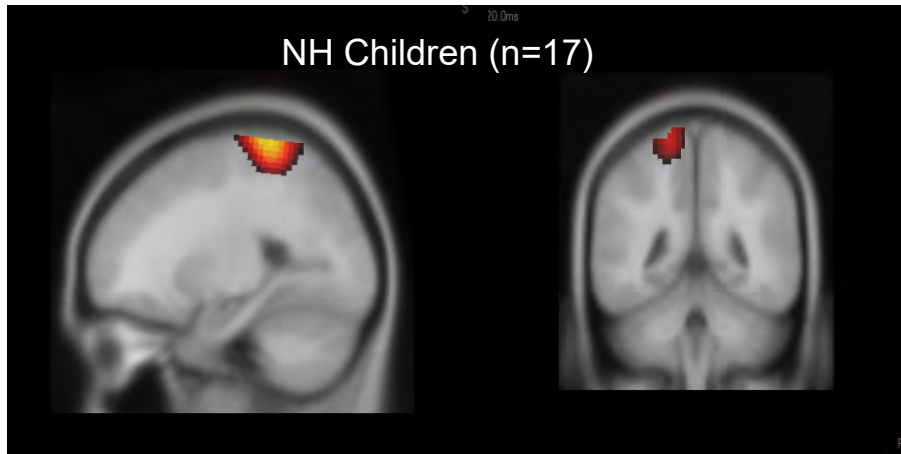


Somatosensory Stimulation

Vibrotactile stimulation of right hand



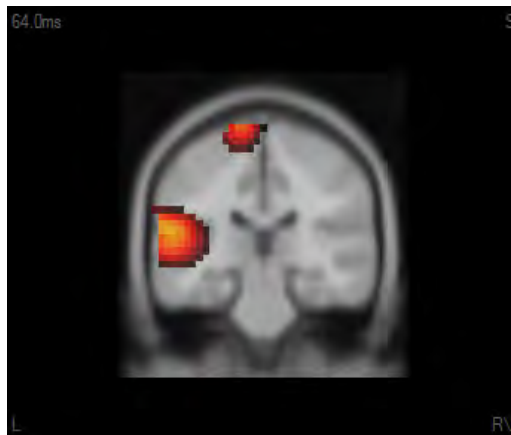
Cortical Activations to Somatosensory Stimulation



Areas Activated

Post-central Gyrus (BA 1-3, 5)
Pre-central Gyrus (BA 4)

CI Children (n=12)

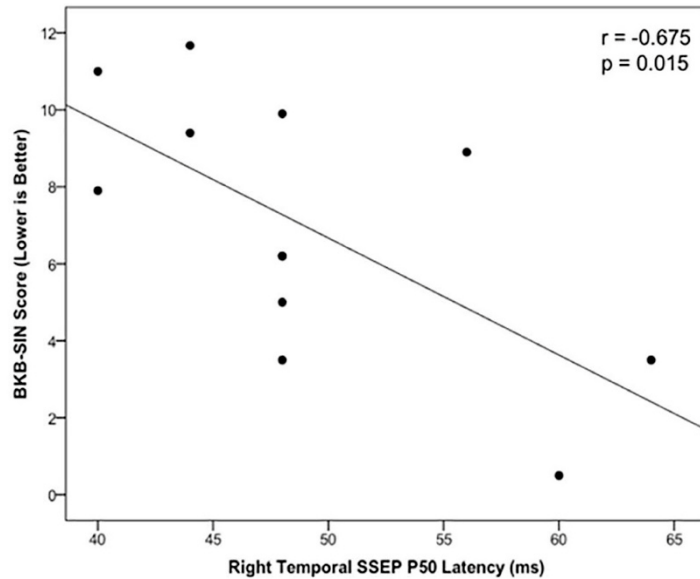


Evidence of
temporal
activation by
somatosensory
stimuli in CI
children.

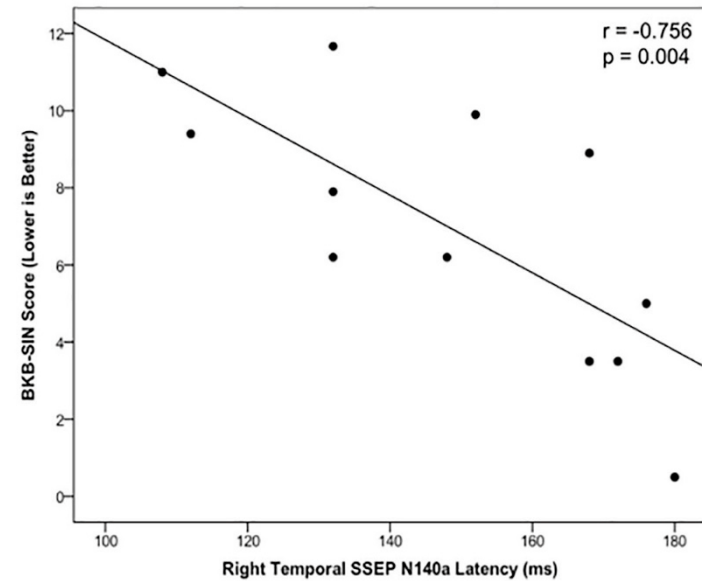
Superior Temporal Gyrus (STG; BA 22)
Middle Temporal Gyrus (MTG; BA 21, 39, 19)
Supramarginal Gyrus
Superior and Inferior Parietal Lobules (BA 40)
Post-central Gyrus (BA 1-3 & 7)
Pre-central Gyrus (BA 4)

Worse performance with the cochlear implant is associated with greater somatosensory cross-modal plasticity.

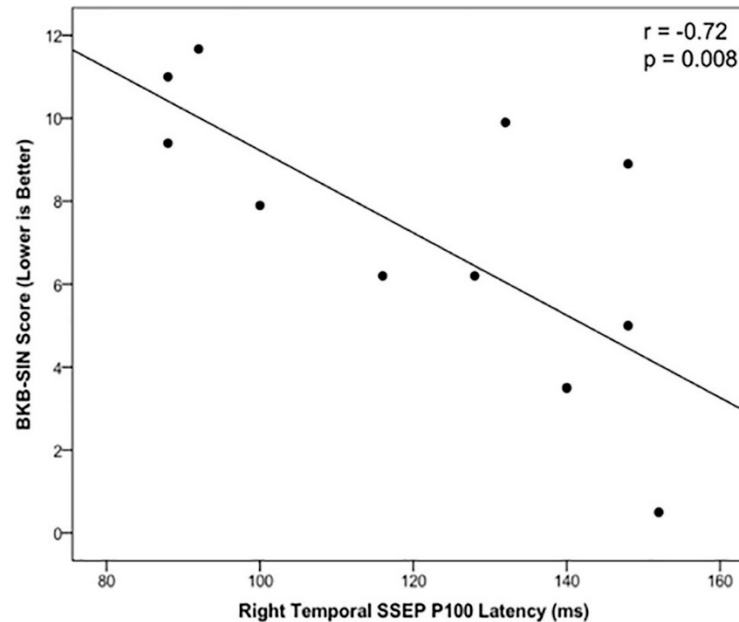
A



C

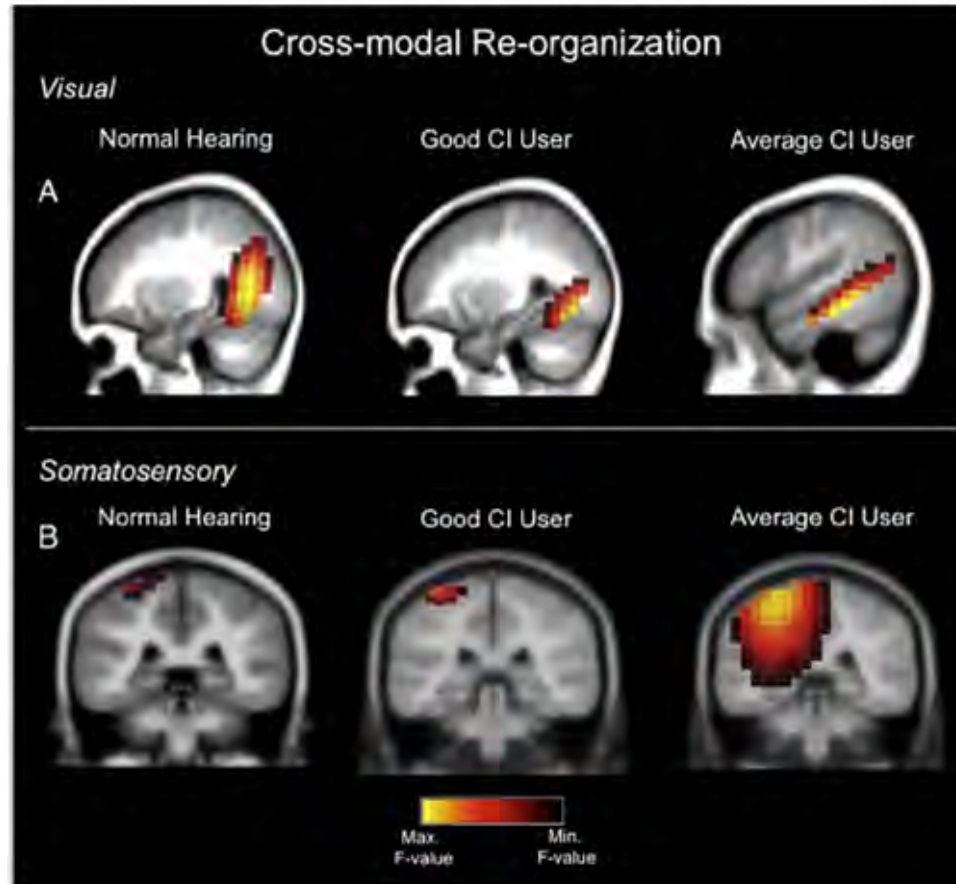


B



Cardon and Sharma
(2019) Frontiers in
Neuroscience

Cross-modal plasticity in individual children



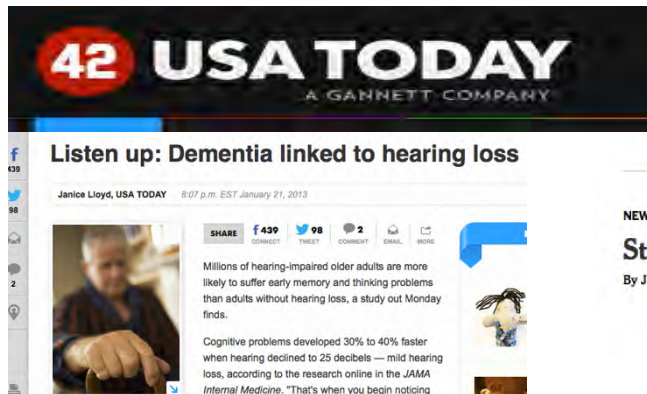
So far, studies have shown evidence of cross-modal plasticity *only* in congenitally and profoundly deaf individuals.

Could it be possible that cortical re-organization starts in much earlier stages of hearing loss?

If so, at what degree of hearing loss would cross-modal plasticity apparent?

Neuroplasticity in age-related hearing loss

Recent research suggests that hearing loss is a significant risk factor for all-cause dementia (including Alzheimer's disease)^(Lin et al., 2011, 2012, 2014)



The New York Times



Risk of Dementia Increases with Hearing Loss (n=639)

Lin et al., 2011

Mild hearing loss: 2 fold increase
Moderate hearing loss: 3 fold increase
Severe hearing loss: 5 fold increase

Hearing loss may account for up to 9% of the
possibly modifiable risk for dementia
(Livingston et al., 2020)

Possible reasons for the link between hearing loss and cognitive decline

- *Common link in the aging process.*
- *Social Isolation.*
 - Untreated hearing loss in seniors has been linked to depression, social isolation.
- *Decreased Cognitive Reserve.*
 - Sensory deprivation taxes the brain by changing its normal resource allocation possibly affecting neural/cognitive reserve.

Neuroplasticity of Age-Related Hearing loss

How does the brain adapt, adjust and compensate for age-related hearing loss?

What does degraded auditory input sound like?

Aim

To explore cortical re-organization associated with mild-moderate age-related hearing loss.

Methods

To date, 96 adults with age-related hearing loss and NH controls.

Auditory, Visual and Somatosensory stimulation paradigms.

QuickSiN Clinical test of Speech Perception in Noise.

Tests of Global Cognitive Functioning and Executive Functioning (e.g., Working Memory, Processing Speed).

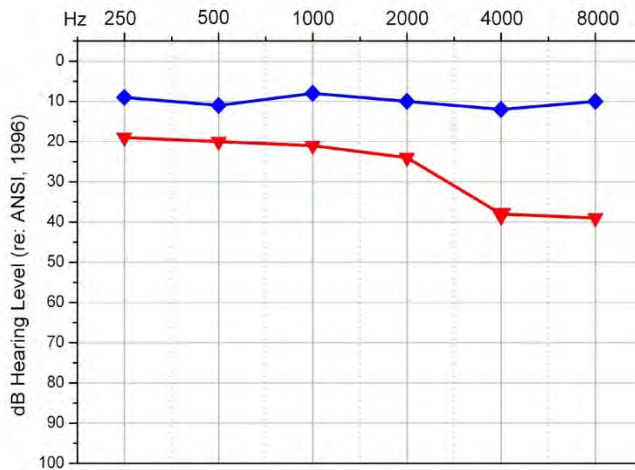
Depression Screener.



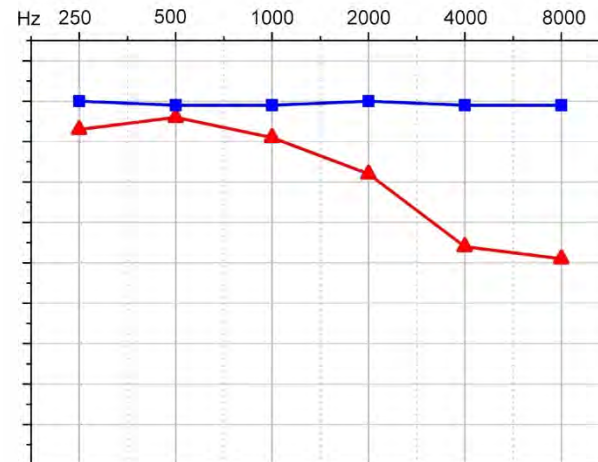
MEAN AUDIOGRAMS $n=17$

- ◆ Normal Hearing Group
- ▼ Hearing Loss Group

Right Ear



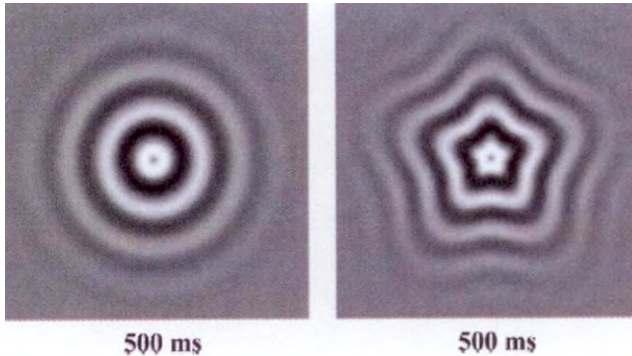
Left Ear



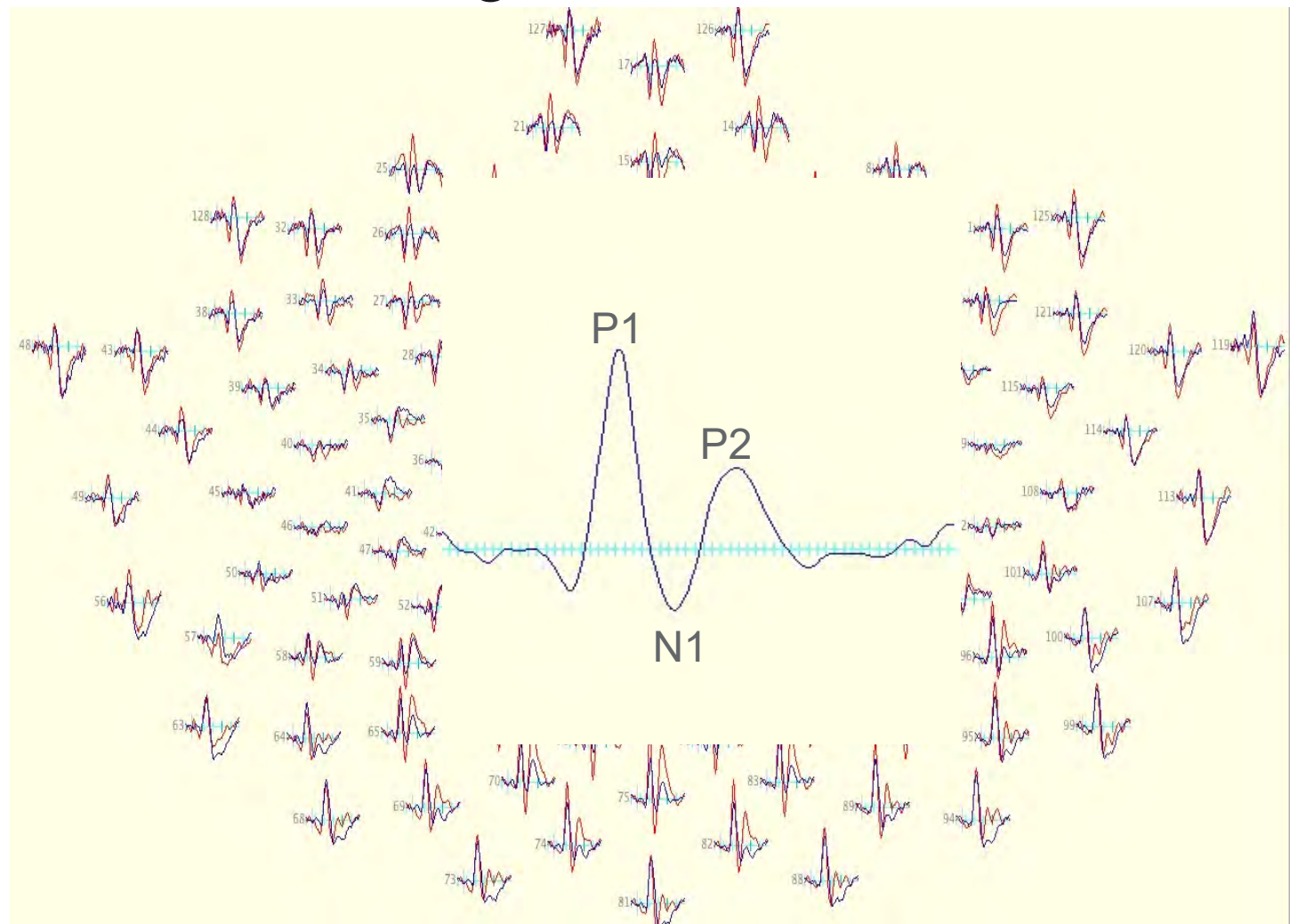
Visual stimulation

High-density EEG recordings

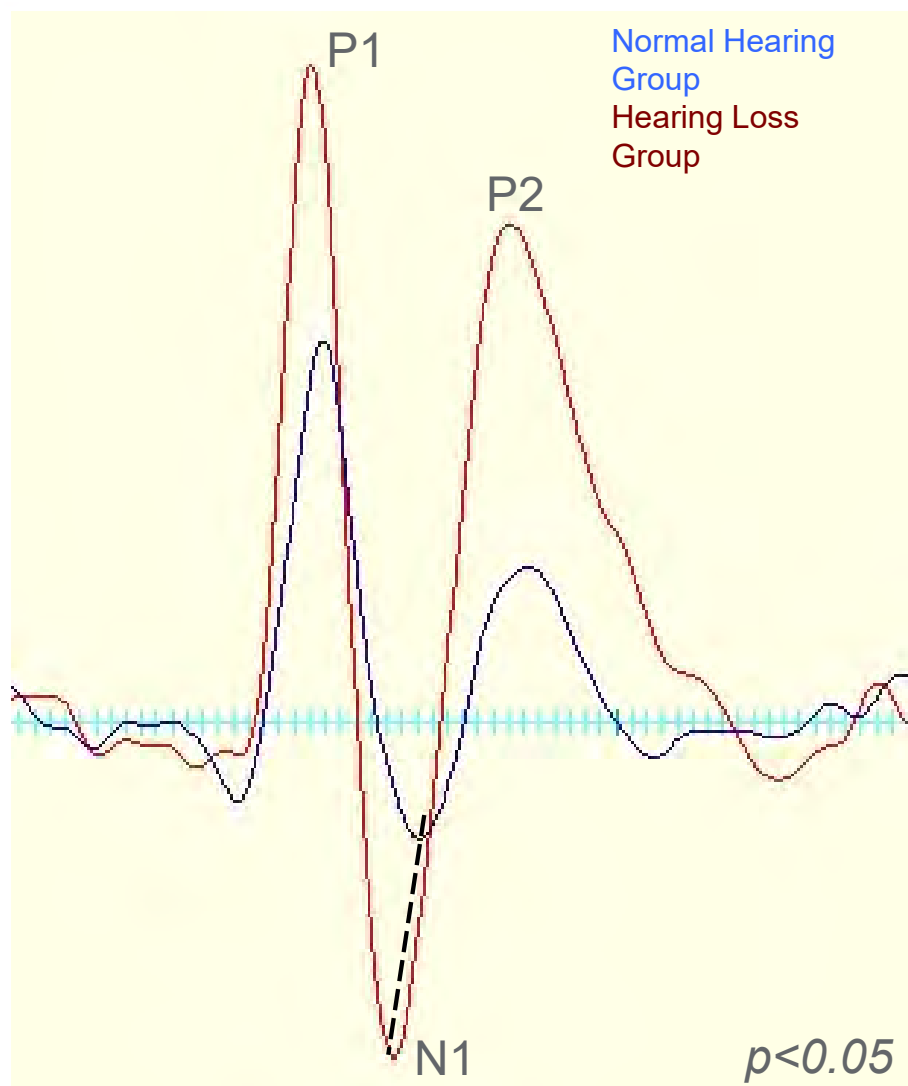
Visual gradient & motion stimuli

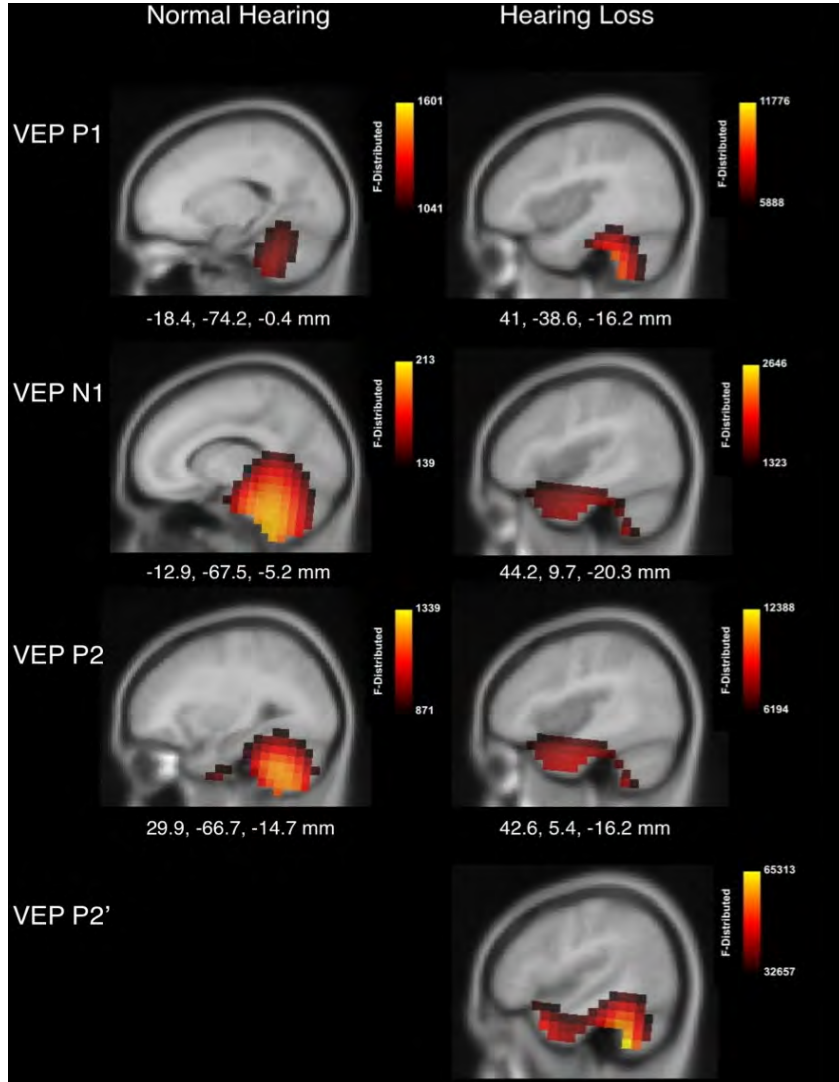


128 channel dense Array EEG Recordings to Visual Stimulation



Grand Average Visual Evoked Responses

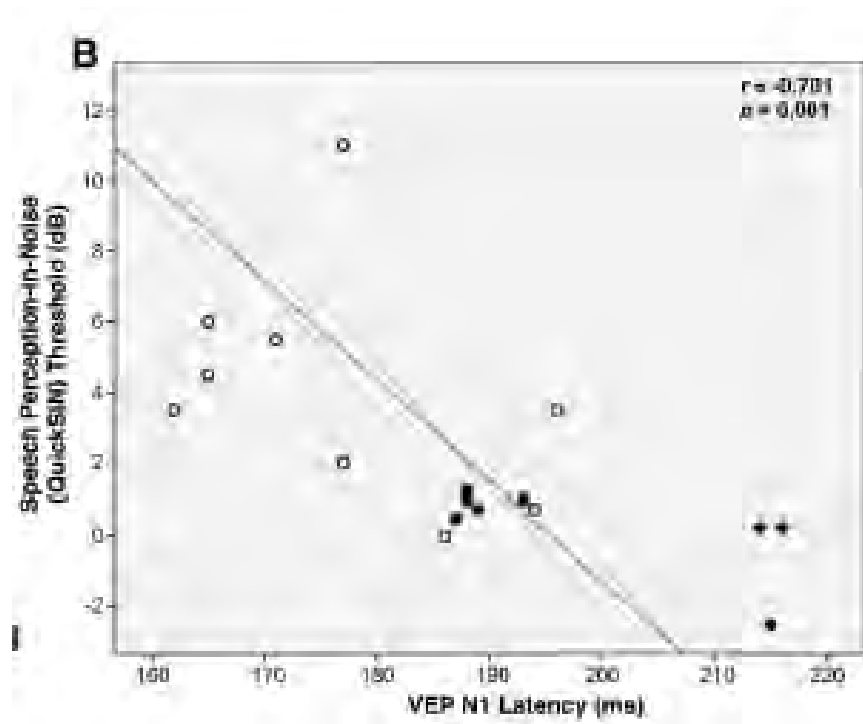




Visual cross-modal re-organization in mild-moderate age-related hearing loss

Relationship of visual cross-modal plasticity to speech perception?

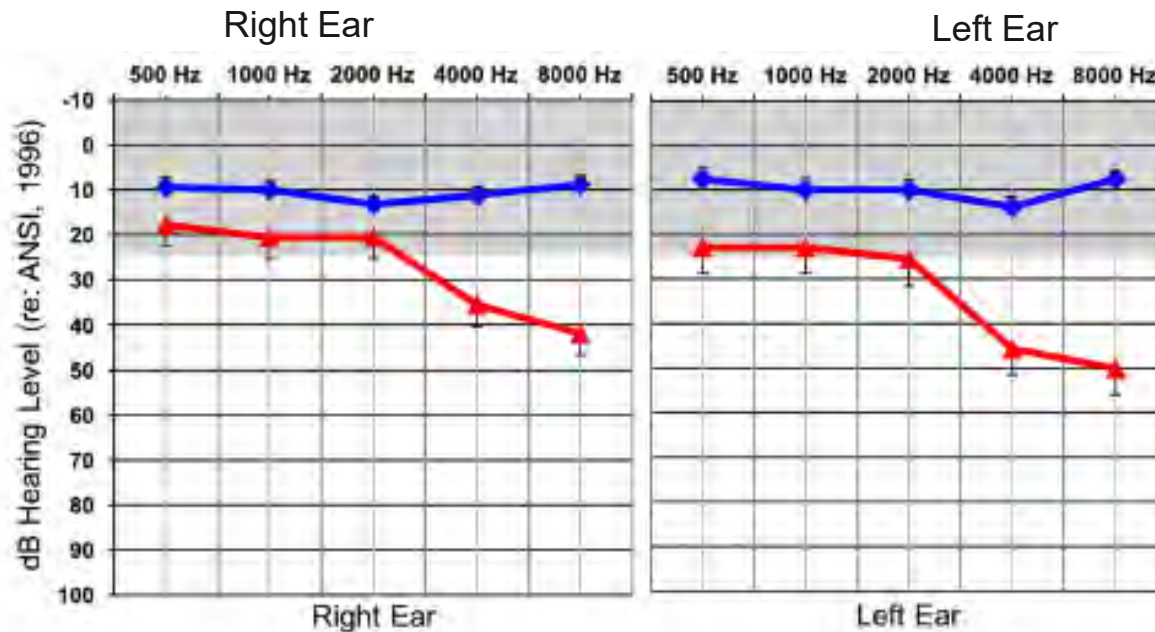
Worse speech perception-in-noise is associated with greater cross-modal plasticity



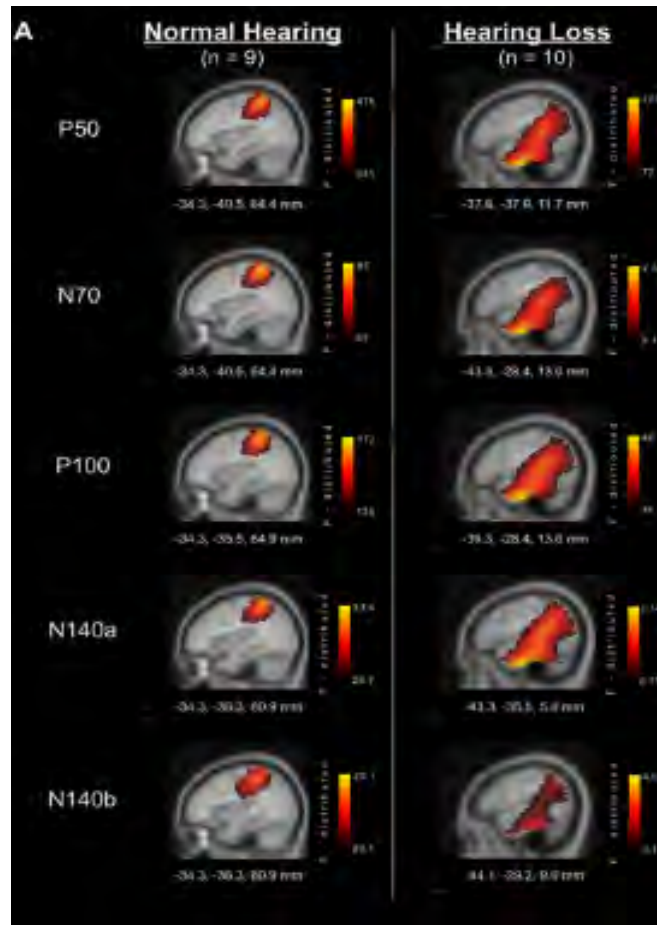
Somatosensory Stimulation

MEAN AUDIOGRAMS n=19

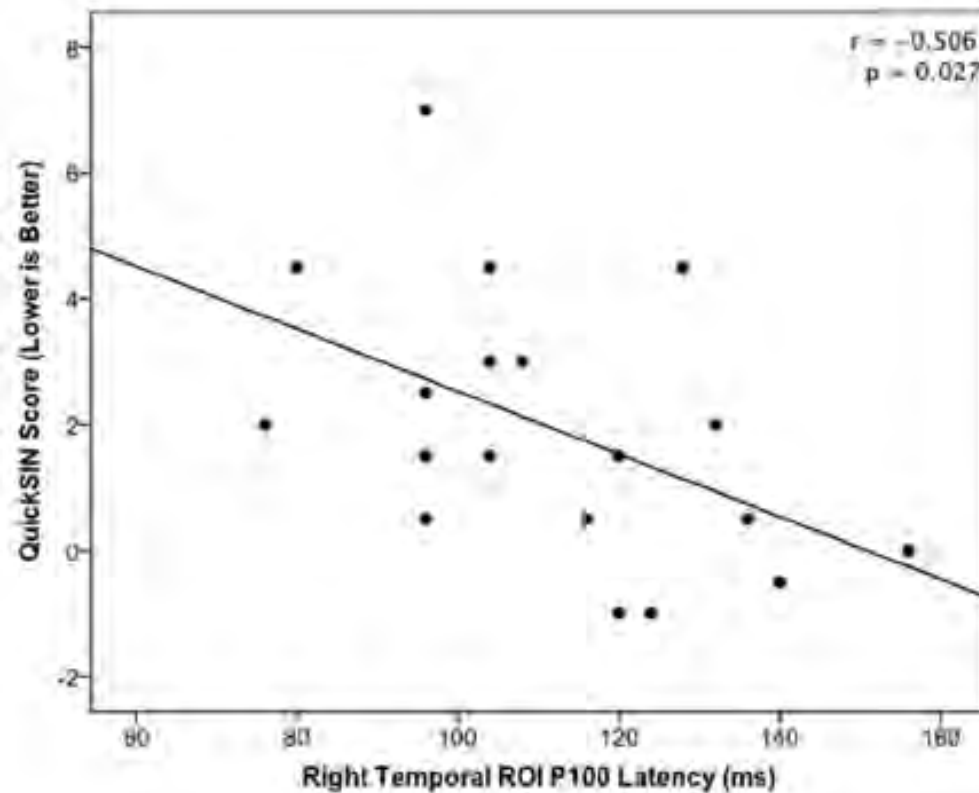
- ◆ Normal Hearing Group
- ▼ Hearing Loss Group



Somatosensory cross-modal re-organization in mild-moderate age-related hearing loss



Worse speech perception-in-noise is associated with greater cross-modal plasticity

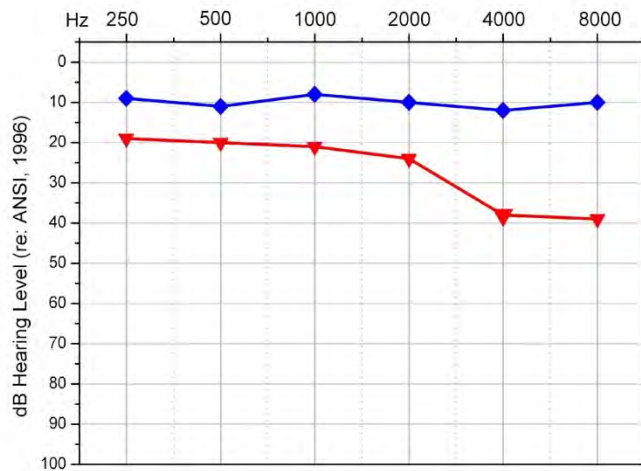


Auditory Stimulation

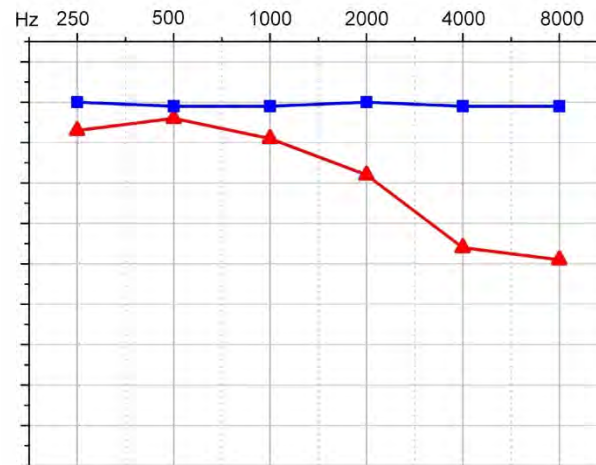
MEAN AUDIOGRAMS (n=17)

- ◆ Normal Hearing Group
- ▼ Hearing Loss Group

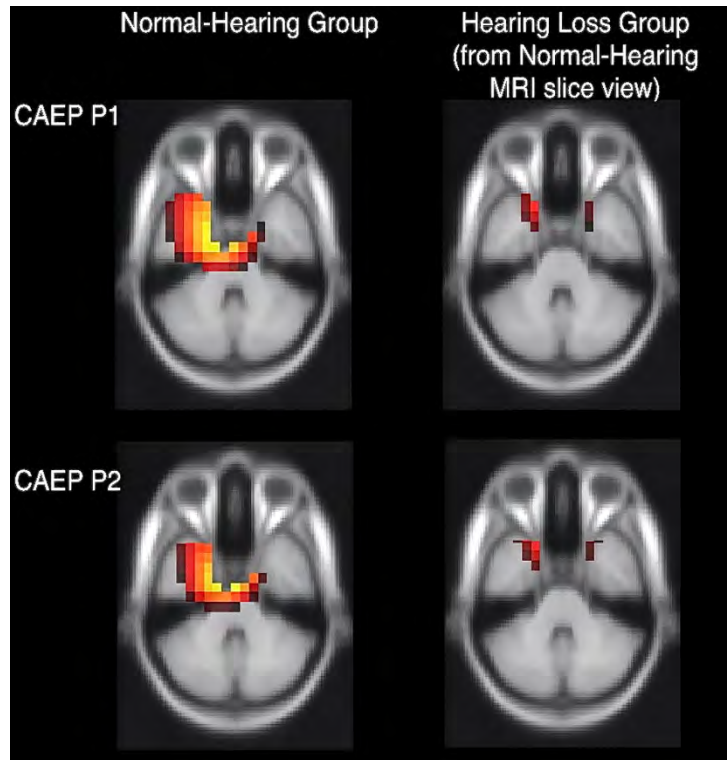
Right Ear



Left Ear

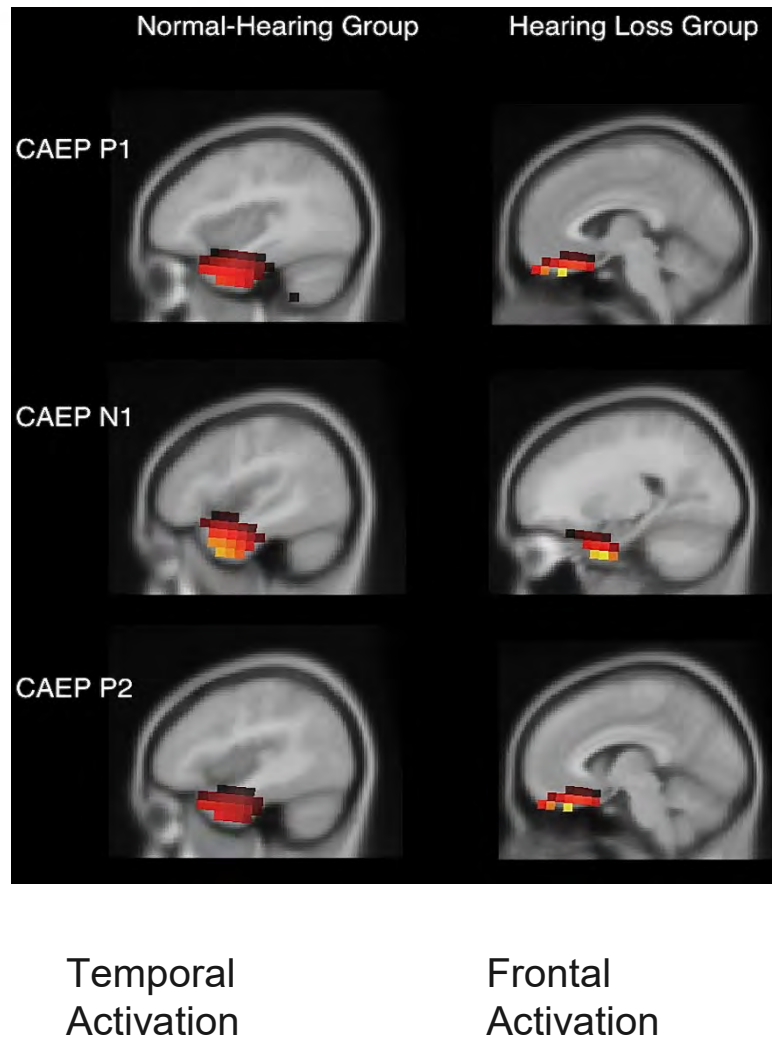


Auditory Stimulation: Cortical Activations



**Temporal
Activation**

**Limited Temporal
Activation**



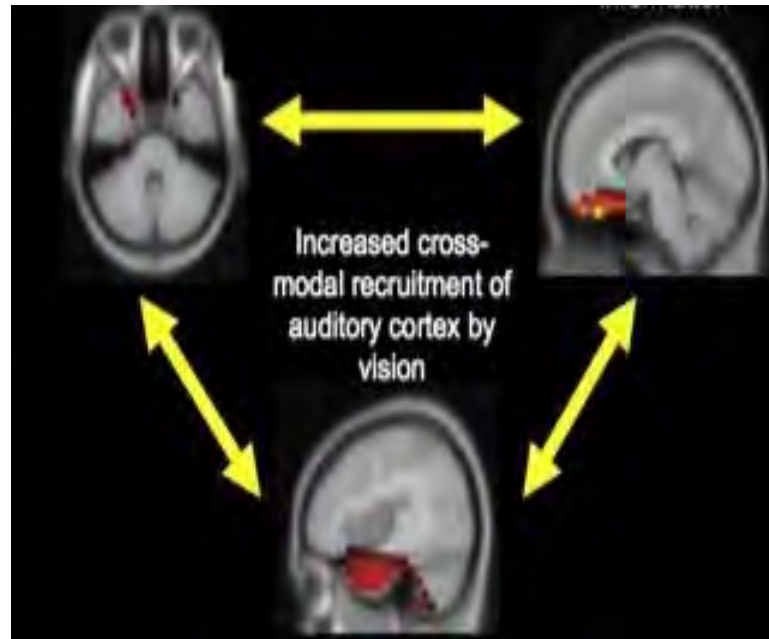
Auditory Stimulation:

Frontal and pre-frontal areas are involved in:
working memory & executive function, and
have been associated with increased
listening effort.

*(Fakhri et al., 2013, Ong et al., 2013, Obleser et al., 2011,
Vagharchakian et al., 2012, Bach et al., 2010, Takeuchi et
al., 2013, Talati and Hirsch 2005, Davis and Johnsru 2003, 2007;
Peele et al., 2010).*

Cortical Re-organization in Mild-Moderate Age-Related Hearing Loss

Reduced auditory cortical activation

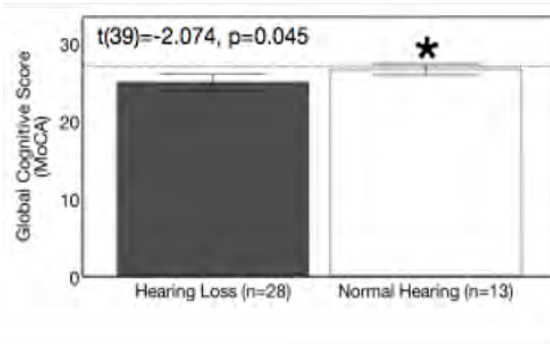


Cross-modal recruitment by vision

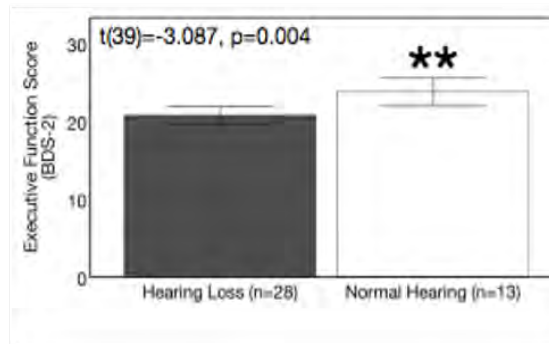
Cognitive function

Deficits in Cognitive Function in Early-Stage Mild-Moderate Hearing loss (n=28; mean age 65)

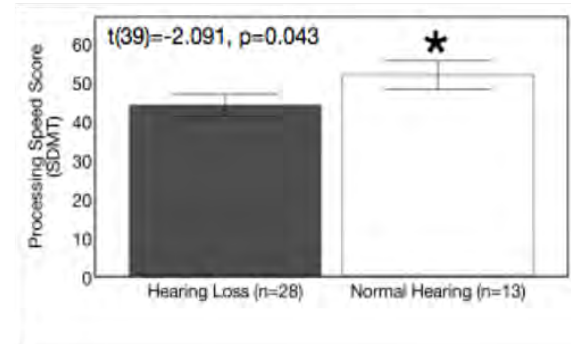
Global Cognitive Function-MoCA



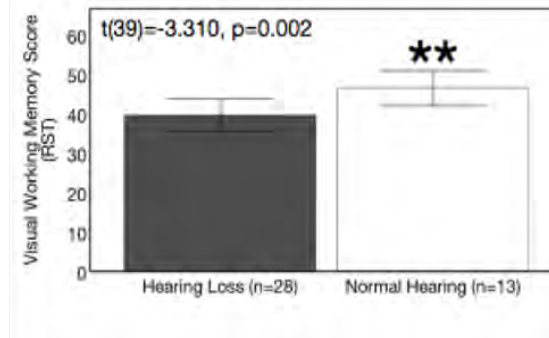
Executive Function (BDS)



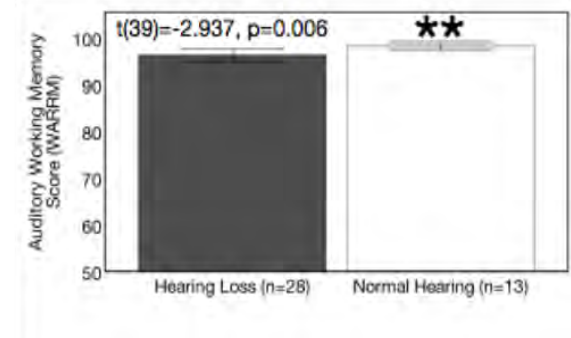
Processing Speed (SDMT)



Visual Working Memory (RST)



Auditory Working Memory (WARRM)



Sensory deprivation taxes the brain by changing its normal resource allocation possibly affecting cognitive spare capacity.

Decreased cognitive reserve may be a factor underlying the association between age-related hearing loss and cognitive decline (*Lin et al., 2011, 2012, 2013*)

**There is evidence of
deprivation-induced
neuroplastic changes in early
stage hearing loss.**

How rapidly do these changes
occur after HL onset?

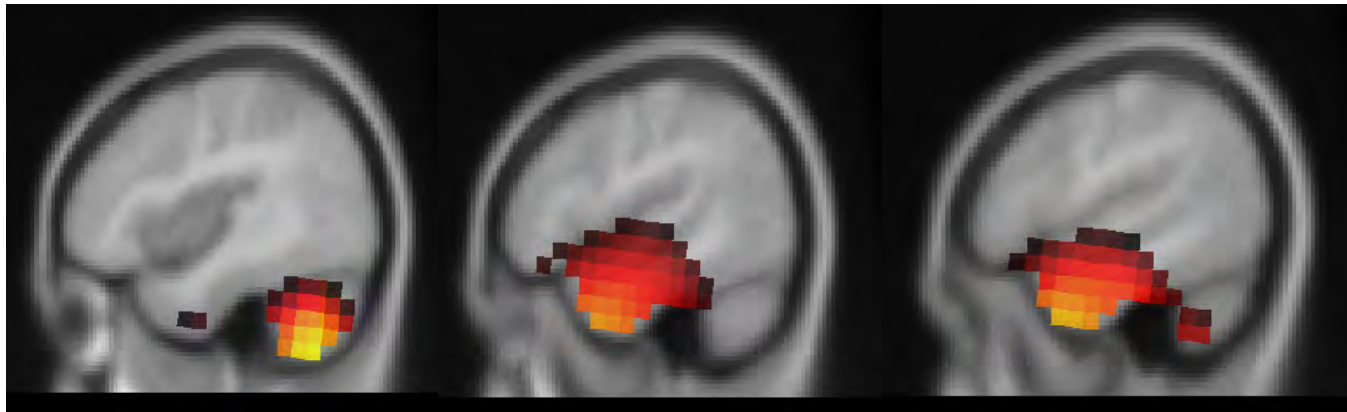
Visual cross-modal plasticity: Sudden SNHL case study

Mild sloping to moderate high frequency SNHL following viral infection.

Approx. onset of HL

3 months later

1 year later



Auditory-Visual
SpeechReading
in Noise

84%

85
%

100
%

Evidence of rapid short-term compensatory plasticity following SNHL

Neuroplasticity following intervention:

Can amplification reverse
compensatory cortical
changes?

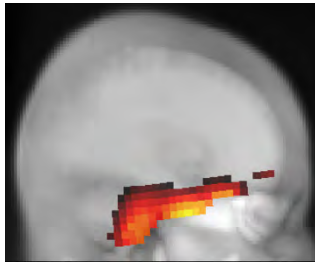
Six Month Clinical Trial with Hearing Aids for Mild-Moderate Hearing loss

- Bilateral receiver-in-the-ear HAs
- Fit and verified to ± 5 dB of NAL-NL2 prescriptive fitting targets for soft (55 dB SPL), medium (65 dB SPL) and loud (75 dB SPL) speech inputs.
- Limited advanced signal processing features (e.g. limited noise reduction, no feedback management) to promote generalizability across manufacturers and to ensure ideal frequency-gain characteristics.
- Inclusion criteria included hearing aid use for 6 hours+ daily.

Mild-Moderate Hearing Loss Six Months After Hearing Aid Use (n=21)

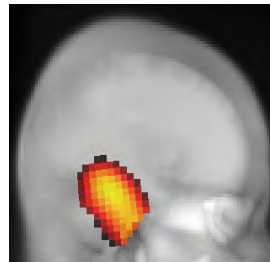
Visual

Before
Hearing Aid



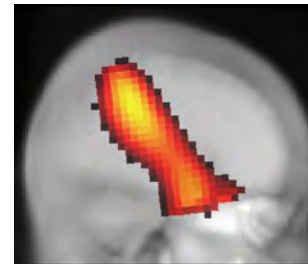
Cross-modal Re-Organization

After
Hearing Aid



Somatosensory

Before
Hearing Aid



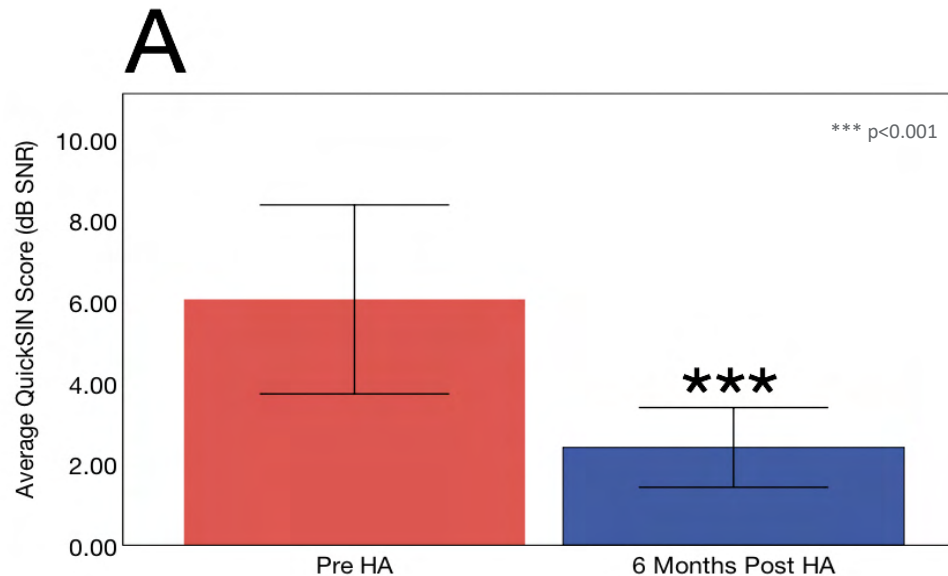
Cross-modal Re-Organization

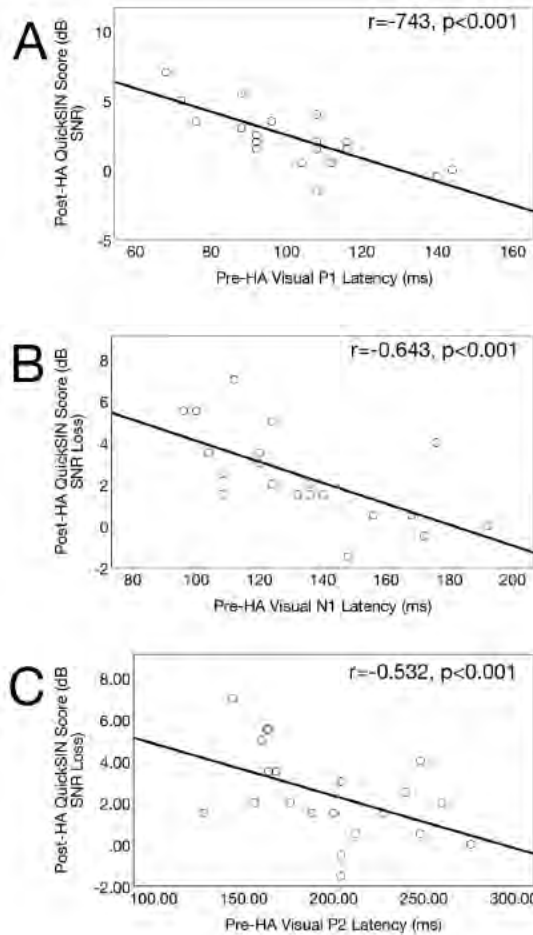
After
Hearing Aid



Cross modal plasticity reversed after 6 months of hearing aid use.

Improvement in Speech Perception in Noise (pre vs post hearing aid use n=21)



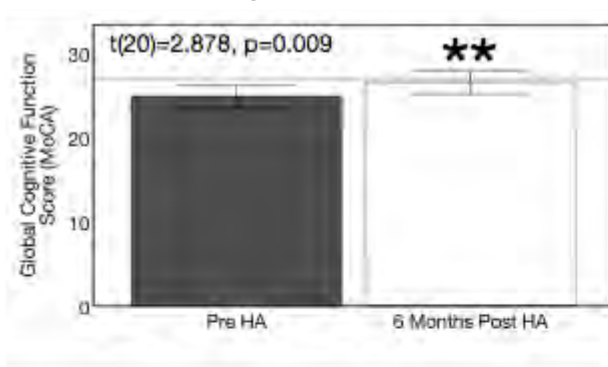


Cross-modal plasticity before hearing aid fitting is associated with worse speech perception-in-noise outcomes with hearing aid use.

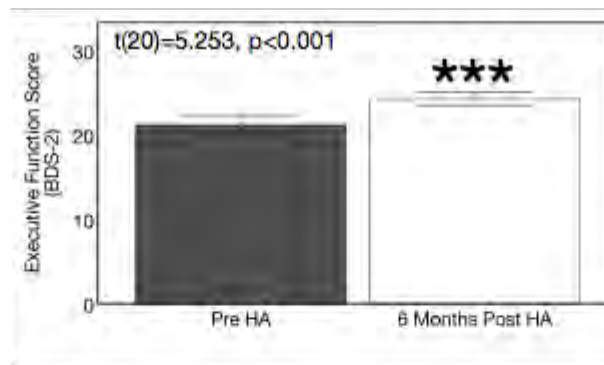
Are there improvements in
cognitive functioning after 6
months of hearing aid use?

Cognitive function after 6 months of hearing aid use (n=21)

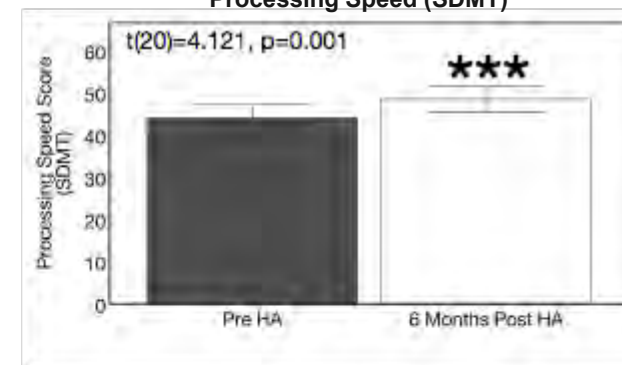
Global Cognitive Function (MoCA)



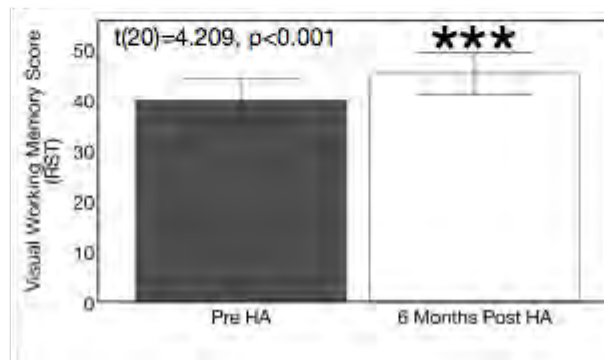
Executive Function (BDS-2)



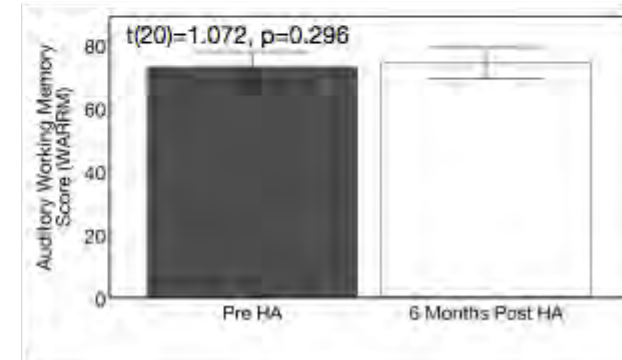
Processing Speed (SDMT)



Visual Working Memory (RST)

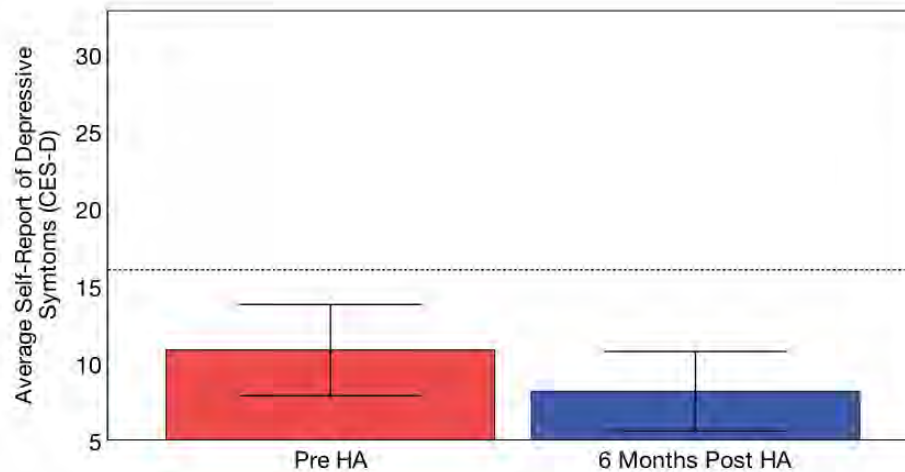


Auditory Working Memory (WARRM)



Significant improvements in global cognitive function, executive function, processing speed, and visual working memory in the hearing loss group

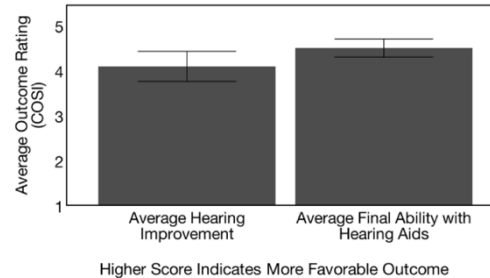
Average Depressive Symptoms (CES-D)



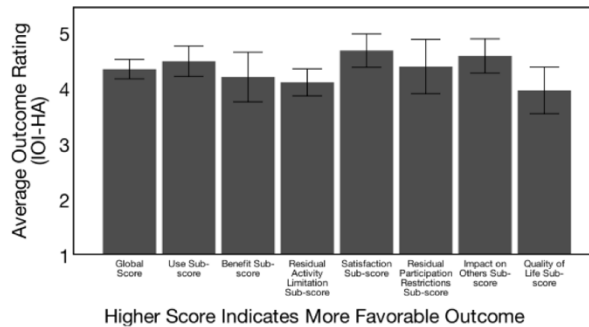
Trend for decrease in depressive symptoms
after hearing aid use

Hearing Aid Outcome Questionnaires

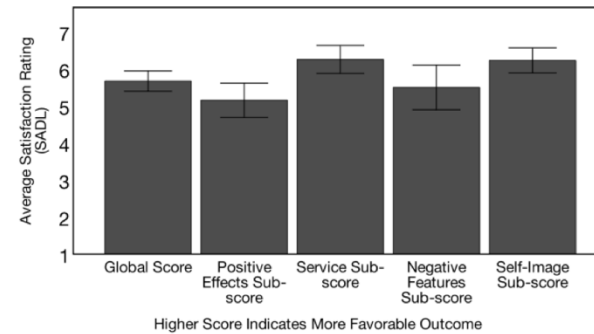
Client Oriented Scale of Improvement (COSI)



International Outcome Inventory for Hearing Aids (IOI-HA)



Satisfaction with Amplification in Daily Life (SADL)



Overall Improvement in Quality of Life with Hearing Aid Use

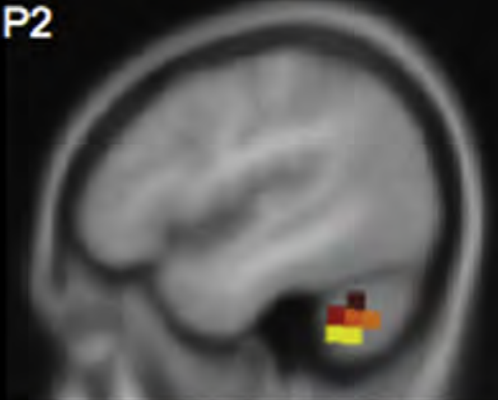
Early and appropriate intervention with amplification is needed in order to restore the gain to the auditory cortex, which likely reverses cortical reorganization and results in good cognitive outcomes.

Cortical Visual Evoked Potential

Good HA User

No evidence of cross-modal plasticity

P2

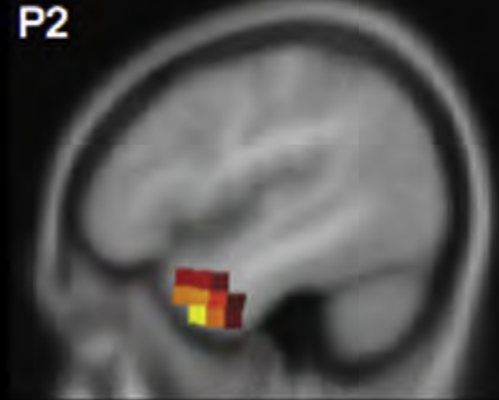


QuickSin Score: 1 dB SNR
(within normal limits)

Poor HA User

Evidence of cross-modal re-organization by vision

P2



QuickSin Score: 8 dB SNR
(moderate deficit in
background noise)

Min  Max
F-distribution

Thank you!

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