No otoacoustic evidence for peripheral basis underlying absolute pitch

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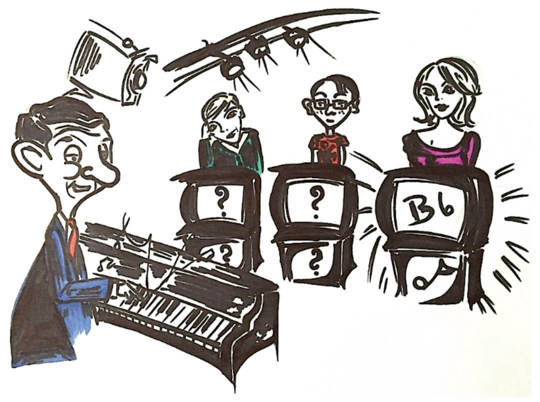
Background

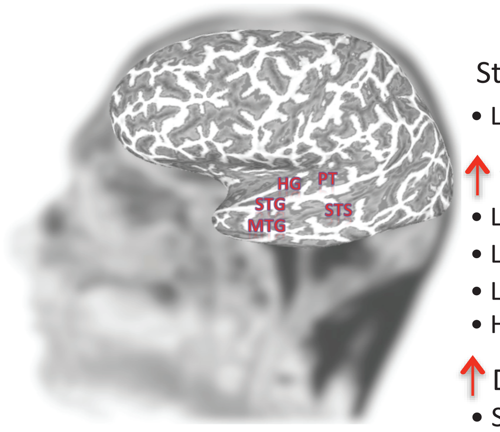


Absolute Pitch (AP) - The ability to identify or recreate a note or a collection of notes in the absence of a reference note

Prevalence: 1 in 10,000
(Profita and Bidder, 1988)

Debate on how it arises:
Genetic? Environmental?
Neural? Biomechanical?

"Central" AP Studies

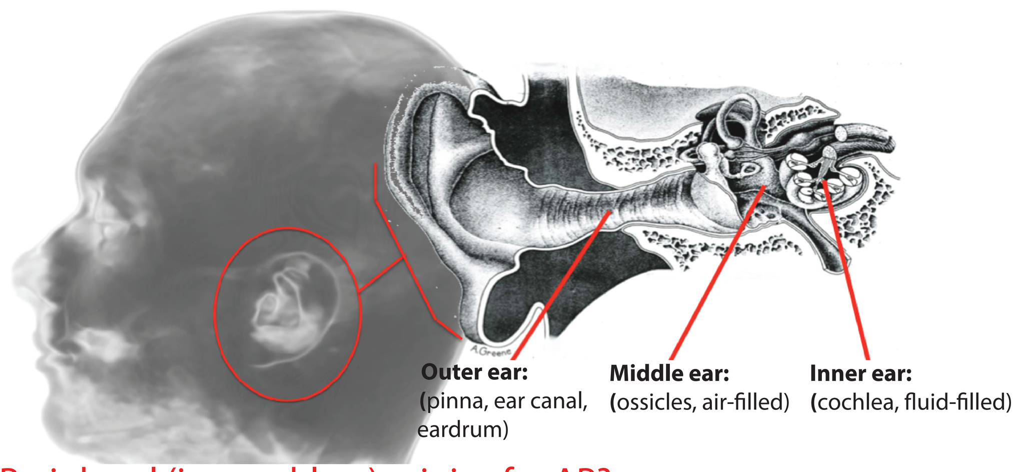


Structural MRI: leftward volume asymmetry
- Left planum temporale (PT) (Keenan et al, 2001)

↑ fMRI activation:
- Left superior temporal gyrus (STG)
- Left superior temporal sulcus (STS)
- Left middle temporal gyrus (MTG)
- Heschl's gyrus (HG) (Shulze et al, 2009; Loui et al, 2012)

↑ DTI connectivity/white matter volume
- STG and MTG (Loui et al, 2011)

Motivation



Peripheral (i.e., cochlear) origins for AP?

➤ Where/when/how does AP "start"? At the cochlea, where sound is transduced to the central nervous system (CNS)?
➤ **Your ears emit sound!** As part of a (cellular-based) amplification process, healthy ears emits sounds known as otoacoustic emissions (OAEs)
- Spontaneous emissions (SOAEs)
  emitted in the absence of a stimulus
- Stimulus frequency emissions (SFOAEs)
  generated in response to an acoustic stimulus (akin to an echo)

➤ OAEs can be used to infer properties of cochlear functionality (e.g., SOAEs correlated to dips in audiometric thresholds, delays associated with SFOAEs tell you about frequency selectivity)

Specific aims of present study:

1. Are SOAEs more prevalent in AP subjects? Both AP and SOAEs appear to have genetic associations, such as higher prevalence in certain populations (e.g., Asians)

2. Using SFOAE delays as a proxy measure of frequency tuning, do AP subjects exhibit differences in selectivity (i.e., sharper tuning)?

Methods

- We examined SOAEs and SFOAEs in both control (N=21) and AP (N=13) normal hearing subjects. Date were collected at both UWO and York (only UWO data included here)
- Standardized AP test given to assess/confirm AP (Loui et al. 2011)
- Normal audiometric thresholds were confirmed for all subjects
- OAE measurements were taken in a double-walled acoustic chamber using an insert probe (Etymotic ER-10C)
- Canal earphone calibration performed in-situ
- SFOAE measured using a swept-tone paradigm [Kalluri & Shera, 2013] using a variety of probe levels: Lp=20-50 dB SPL [Ls=Lp+15 dB, fs=fp+40 Hz]
- SFOAE phase-gradient delays (Nsf) extracted using a "peak-picking" algorithm [Shera & Bergevin, 2012] (see Figure 6)

Results - SOAEs

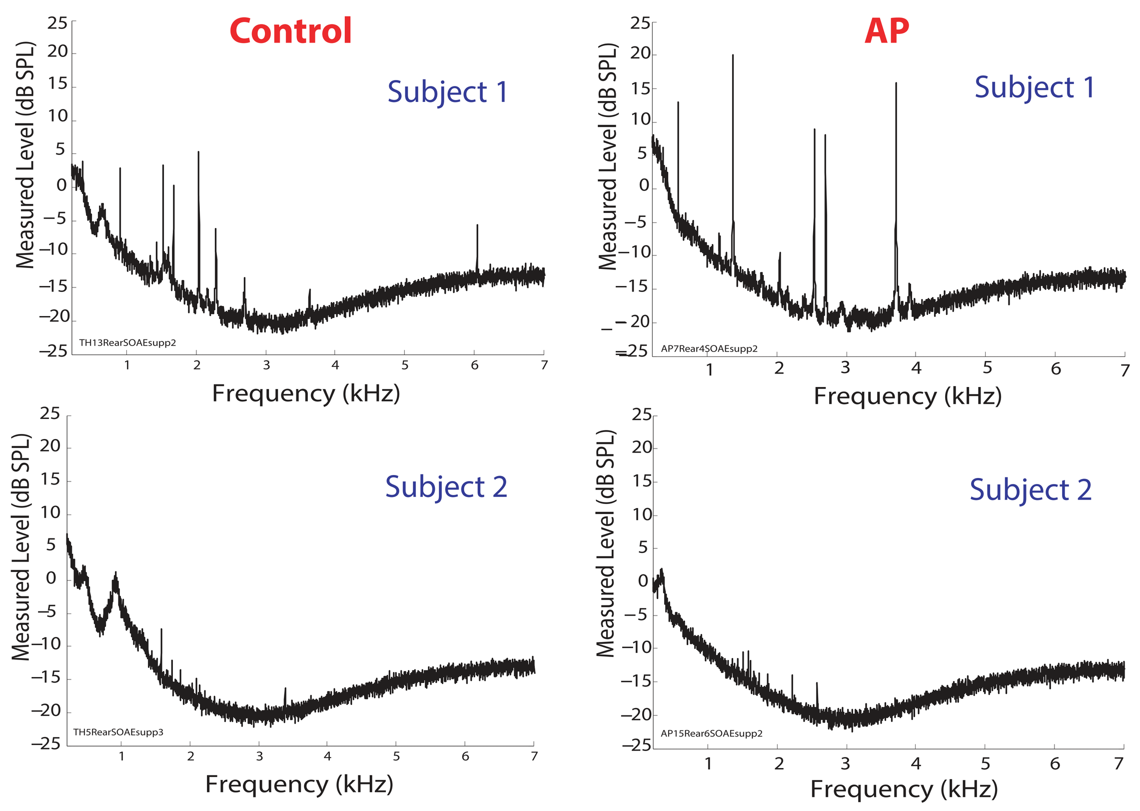


Figure 1: SOAE differences in representative control and AP subjects. Panel 1.) illustrates robust SOAE peaks, whereas Panel 2.) illustrates no SOAE peaks present in both control and AP subjects. Note: some 60 Hz noise present.

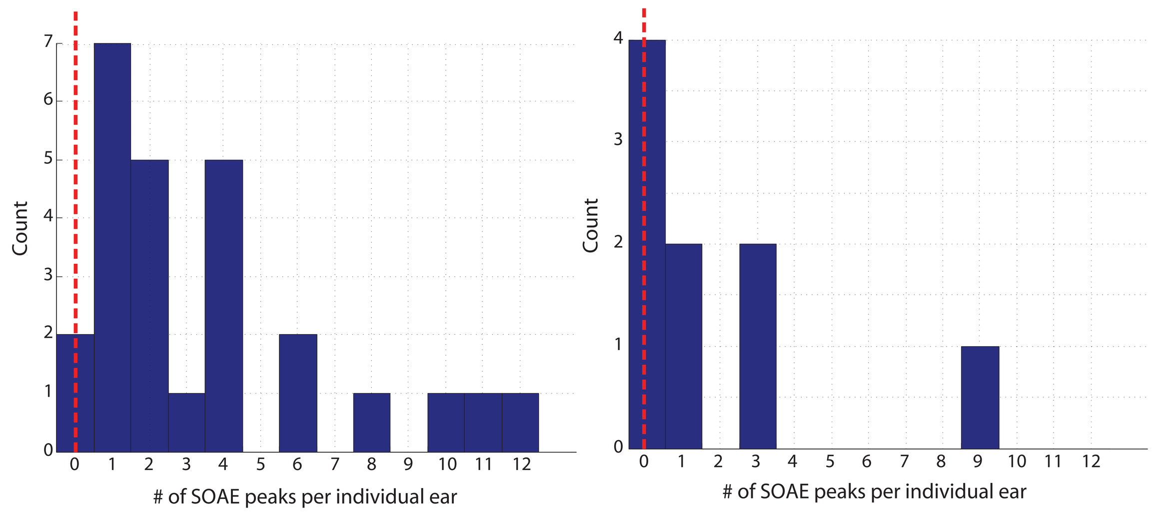


Figure 2: Histogram of SOAE count differences in control (N=21) and AP (N=13) subjects. Qualitatively, SOAE incidence was less prevalent in AP subjects. More rigorous analysis required to better quantify peak existence (e.g., Talmadge et al, 1993) and associated dynamics.

Summary: SOAEs are not more prevalent in AP subjects. Thus no evidence that SOAEs can act as a cue or reference for AP.

Results - SFOAEs

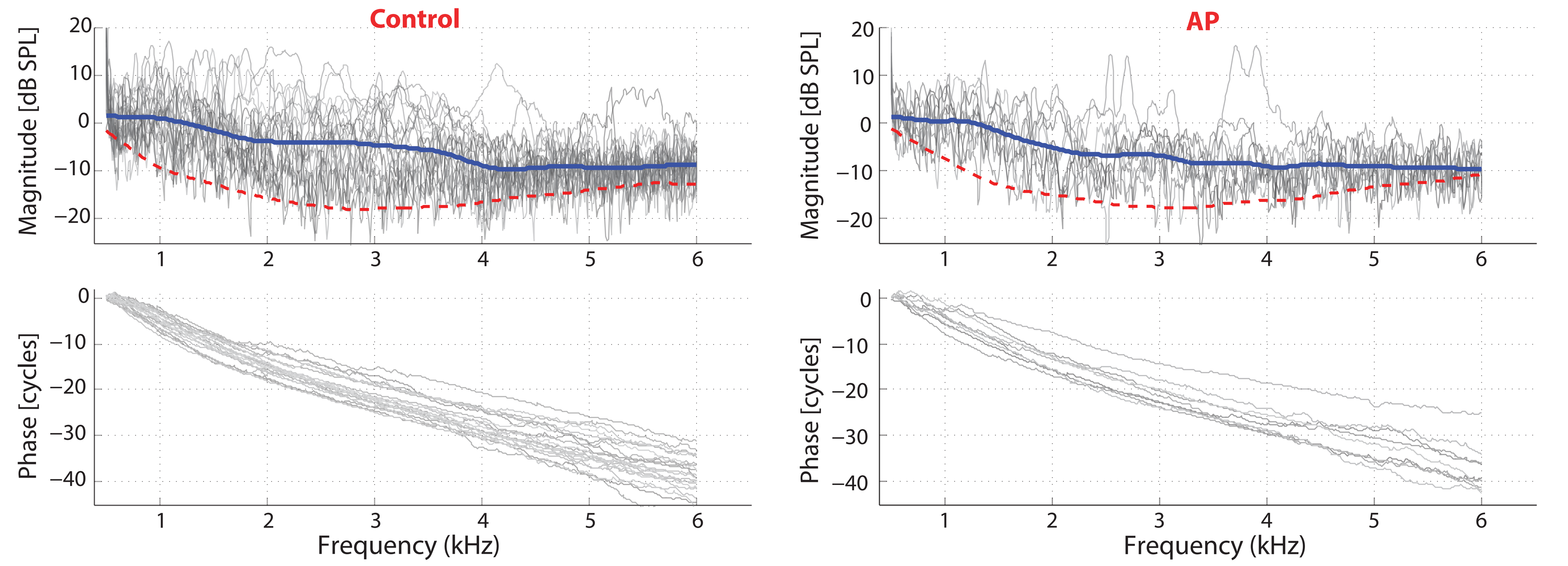


Figure 4: SFOAE differences in control (N=23) and AP (N=9) subjects. Thick lines are locally-weighted regression (loess) trends pooled across subjects.

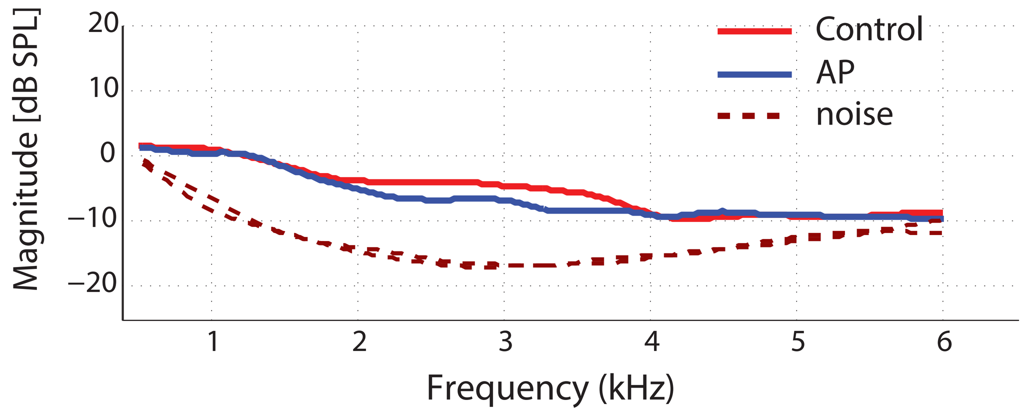


Figure 5: No significant differences in SFOAE magnitudes (Lp=30 dB SPL) between pooled groups.

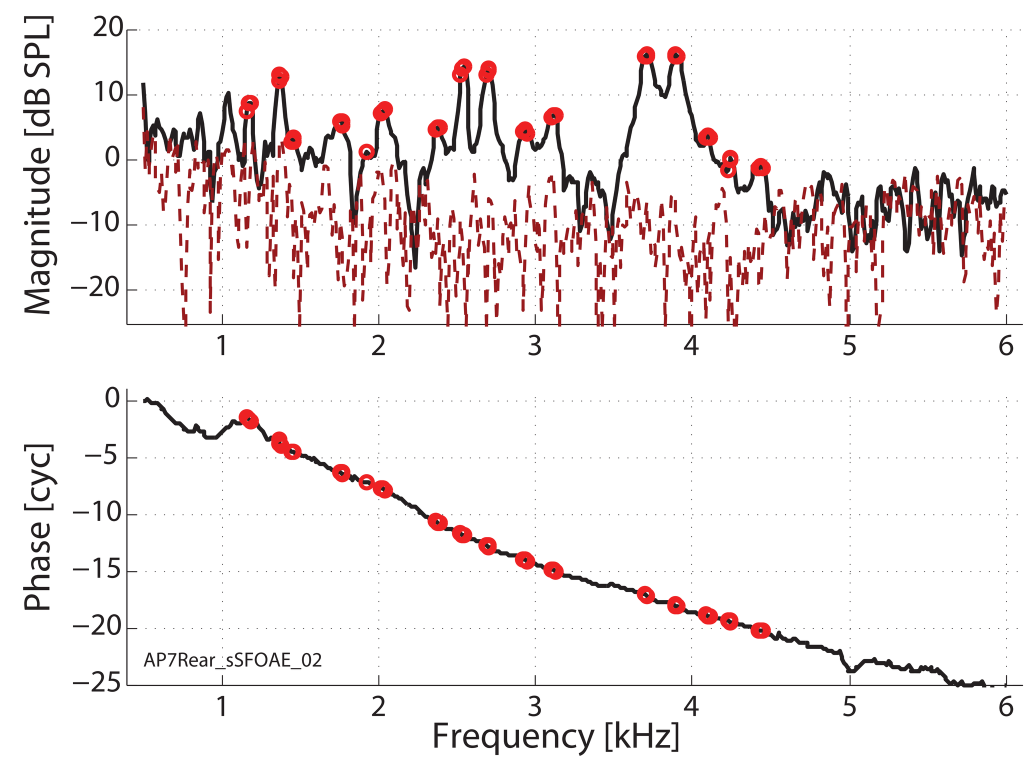


Figure 6: Representative SFOAE data from a single ear showing the "peak-picking" algorithm used for extracting SFOAE phase gradient delays [denoted as open red circles].

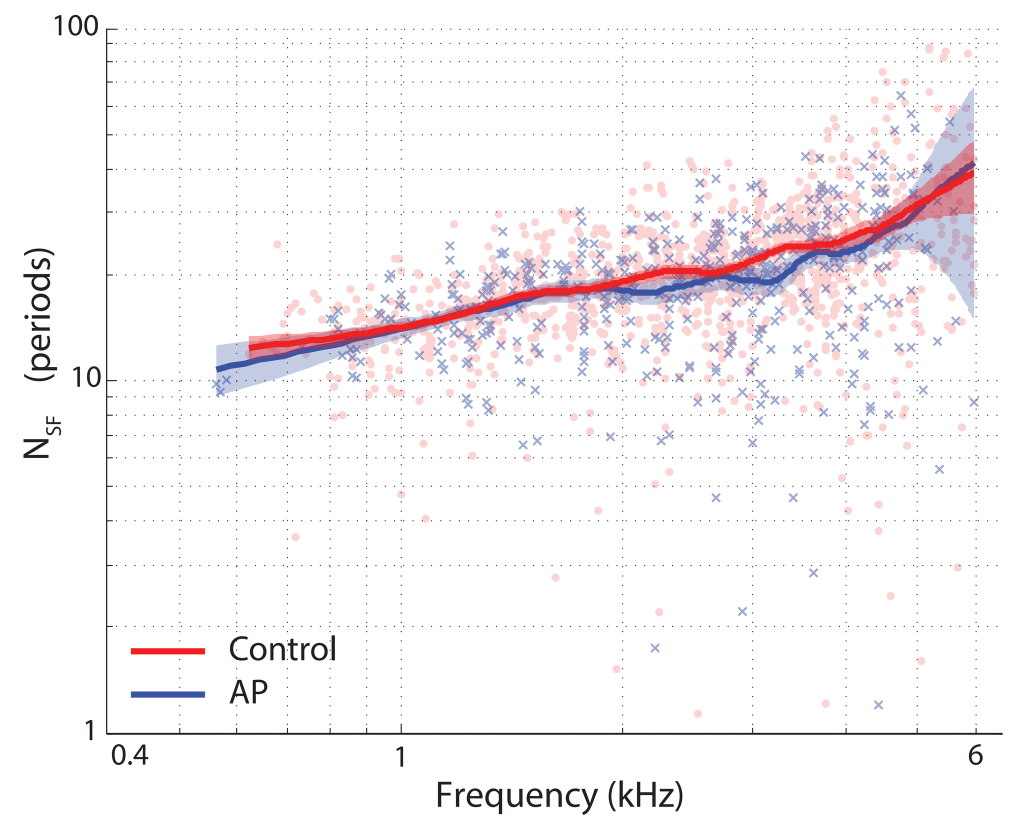


Figure 7: Plot showing number of stimulus periods of delay (Nsf) extracted from phase-gradient delays in Control (N=23) and AP (N=9) subjects, Lp= 30 dB SPL. Shaded areas indicate a 95% confidence interval (CI) via bootstrapping. Results suggest no improved frequency selectivity in AP subjects. Similar relationship exists at other stimulus levels, although delays get progressively longer for lower stimulus levels.

Summary: SFOAEs are not "stronger" in AP subjects, nor indicative of sharper peripheral tuning. Thus, no evidence for improved sensitivity or frequency selectivity in AP subjects at level of the cochlea.

Conclusions

➤ No obvious SOAEs to act as a "reference" and no indication of sharper frequency tuning

➤ OAE data thus suggest no clear peripheral differences between AP and non-AP individuals

➤ We therefore conclude that the mechanisms arise at the central/neural level

Future Work

➤ What is the central basis in AP?

➤ How is the auditory (sub)cortex organized in AP vs. non-AP?

➤ Does tuning "sharpen" as one ascends the CNS?

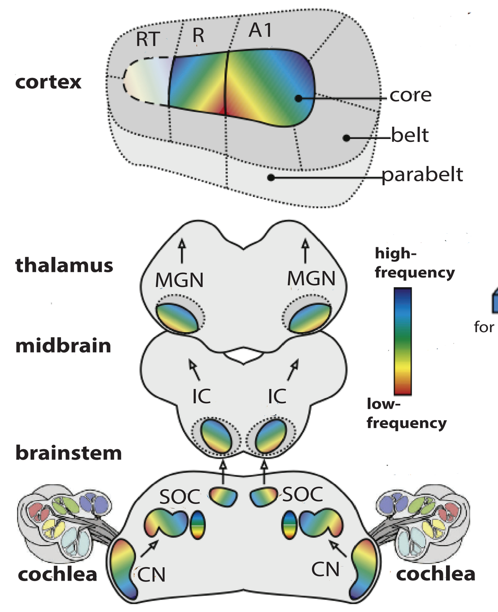


Figure 7: Tonotopic organization of central auditory pathways (based on human and non-human primate models). Modified from (Saenz and Langers 2013)

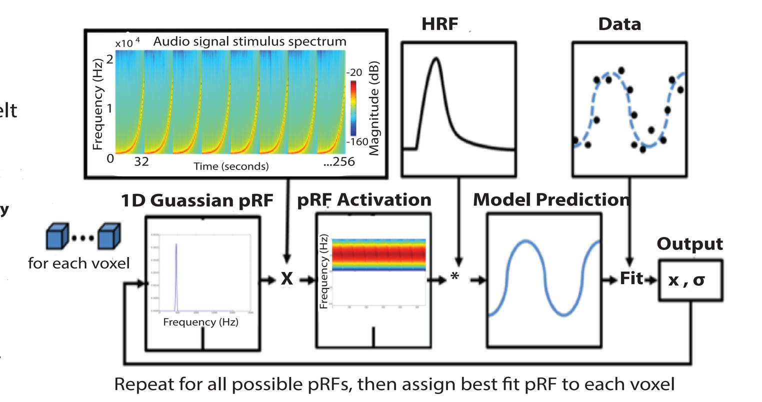


Figure 7: Population receptive field (pRF) model used in fMRI data collection to establish the tonotopic mapping of cortex and subcortex. Provides an estimated sensitivity function for each voxel with a given center, or preferred frequency, and standard deviation, or tuning bandwidth.

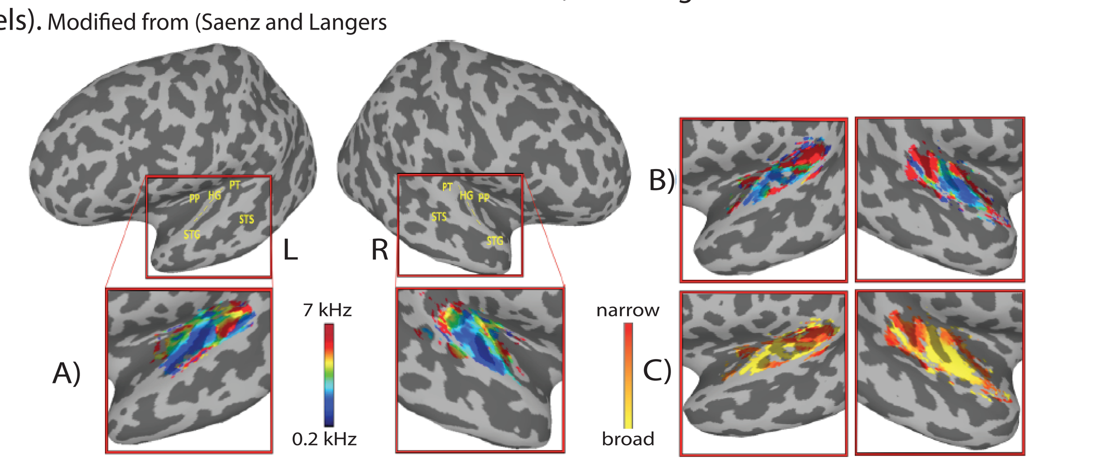


Figure 8: Tonotopic pRF and tuning curve map of the auditory cortex from two representative controls subjects. Center frequency gradient and tuning curve maps were plotted on the unfolded cortical surface for each hemisphere. Panel A shows the left and right hemisphere from control subject [female, 27] from 3 sessions worth of data (35 functional runs). Panel B shows pRF maps and C) tuning curve maps from a control subject [female, 23] from 1 sessions worth of data (10 functional runs).

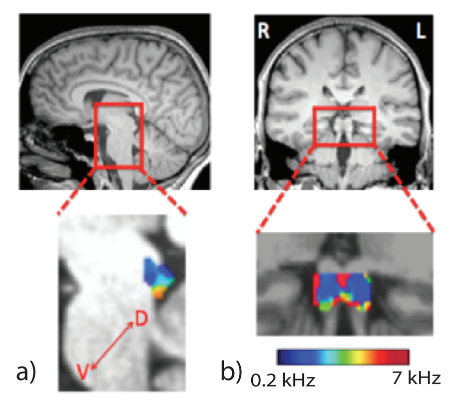


Figure 9: Tonotopic map of the Inferior Colliculus (IC) in a control. **a)** (Top) Sagittal, (bottom) zoomed in view of the IC displaying a high-to-low frequency gradient from the ventral (V) to dorsal (D) orientation. **b)** (Top) Coronal, (bottom) zoomed in view of the right and left tonotopic maps of the IC. [3 sessions worth of data]

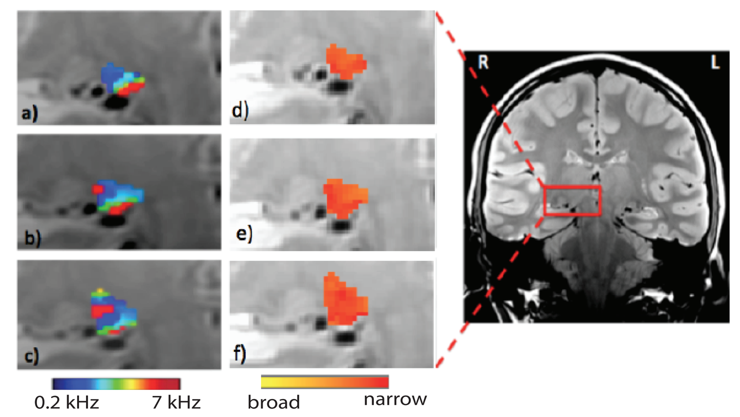


Figure 10: Tonotopic map of the Medial Geniculate Nucleus (MGN) in a control. **a)** (Top) Sagittal, (bottom) zoomed in view of the IC displaying a high-to-low frequency gradient from the ventral (V) to dorsal (D) orientation. **b)** (Top) Coronal, (bottom) zoomed in view of the right and left tonotopic maps of the IC. [3 sessions worth of data]

References & Acknowledgements

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