



Neurobiology of speech motor control

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Faculty research talk

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Who am I?

Lynn Kurteff, PhD, CF-SLP

- Multidisciplinary clinician-scientist
 - Linguistics; Psychology; Neurosurgery; Speech, Language, & Hearing Sciences; Cognitive Sciences; Neurology
- My work focuses on:
 - **Clinical translation** of basic science research
 - The **neurobiological foundation** of communication disorders
 - Integrating communication disorders with **theoretical cognitive-neuroscientific models** of speech



2012-2015



2016-2018



2018-2024



2024-2025



2025-



Outline

- What is speech motor control?
- The brain, speech, and language
- Mapping brain networks important for apraxia of speech
- Auditory-motor monitoring of vocal feedback during speech
- Future directions



image credit: Wildrose Hamilton



Learning outcomes

After this talk, audience members should be able to...

- Describe the difference between **feedforward** and **feedback** motor control of speech
- Identify **brain regions** associated with feedforward and feedback control
- Explain how a **disordered speech motor control** system results in different **communication disorders**

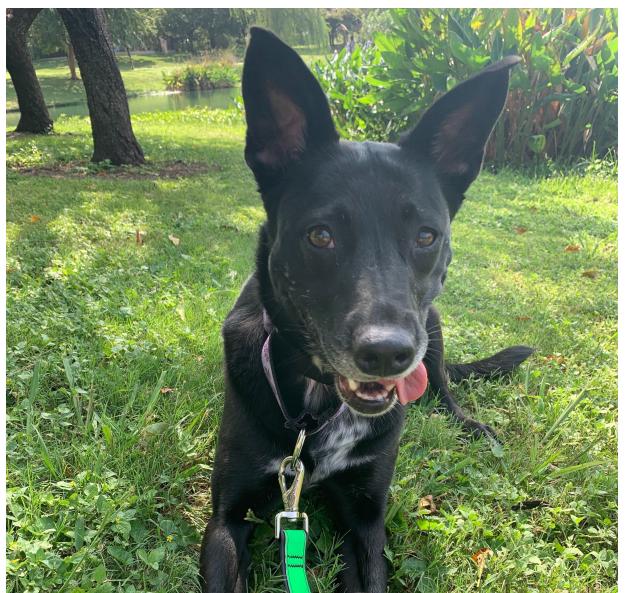
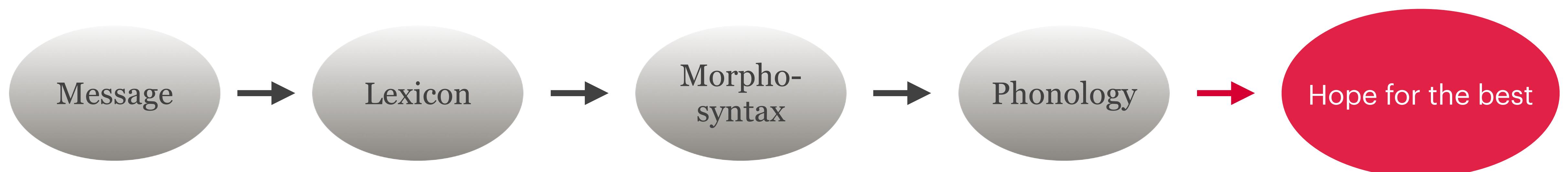


What is speech motor control?

"The hearing ear is always found close to the speaking tongue"



The stages of speech production



dog
want
you
can

CAN 1sg PET 2sg.POSS DOG

want
pet
me

/kæn aɪ pɛt jə· dag/



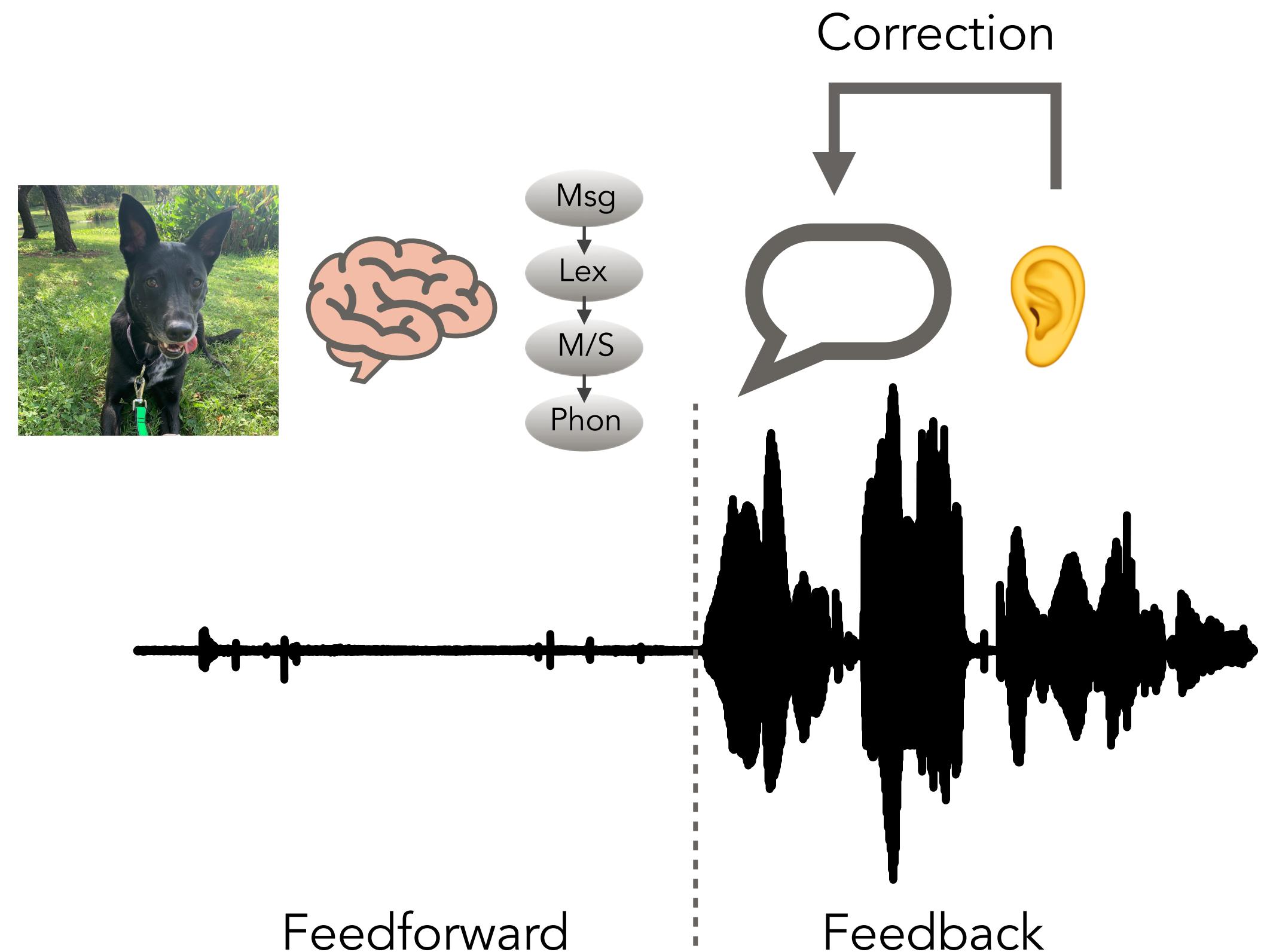
: *What a cute dog!*



What is “speech motor control” ?

A series of processes for ensuring accurate speech production in real time [2]

- **Feedforward control:** The generation of a motor plan and a set of expectations regarding what will happen before we speak
- **Feedback monitoring:** Tracking the sensory consequences of our own speech while we speak to determine when we've made a mistake





Disorders of speech motor control

Stuttering

- Theorized to involve disordered feedforward and disordered feedback control
- Individuals who stutter have limited pre-speech auditory modulation [3]
- Disruptions in fluency arise from an over-corrective feedback correction system
- Experimental research on delayed auditory feedback in a therapeutic context, but the jury is out

Speech sound disorders

- Children with SSD show reduced compensatory responses to feedback perturbation [4]

Schizophrenia

- Faulty feedback control results in auditory hallucinations [5]

[3] Max & Daliri, 2019

[4] Terband et al., 2014

[5] Heinks-Maldonado et al., 2007



Disorders of speech motor control

Apraxia of speech

- A motor disorder affecting speech coordination
 - Selective deficit in **feedforward control**
 - Abnormal **feedback control** emerges as a compensatory strategy [6]
- Assessment, treatment, and neurobiology are ... messy [7]
 - Disagreement about hallmark symptoms (inconsistent speech errors) [8]
 - Disagreement about anatomical correlate (insula vs. Broca's area) [9, 10]
 - Disagreement about whether or not subtypes of AOS exist [11]
- What I study!

[6] Jacks & Haley, 2015

[7] Ziegler et al., 2012

[8] Haley et al., 2021

[9] Dronkers, 1996

[10] Hillis et al., 2004

[11] Mailend & Maas, 2020



The brain, speech, and language



The Classic Model

- **Broca's area** controls speech **production**
- **Wernicke's area** controls speech **perception**
- Broca and Wernicke are connected by the **arcuate fasciculus**

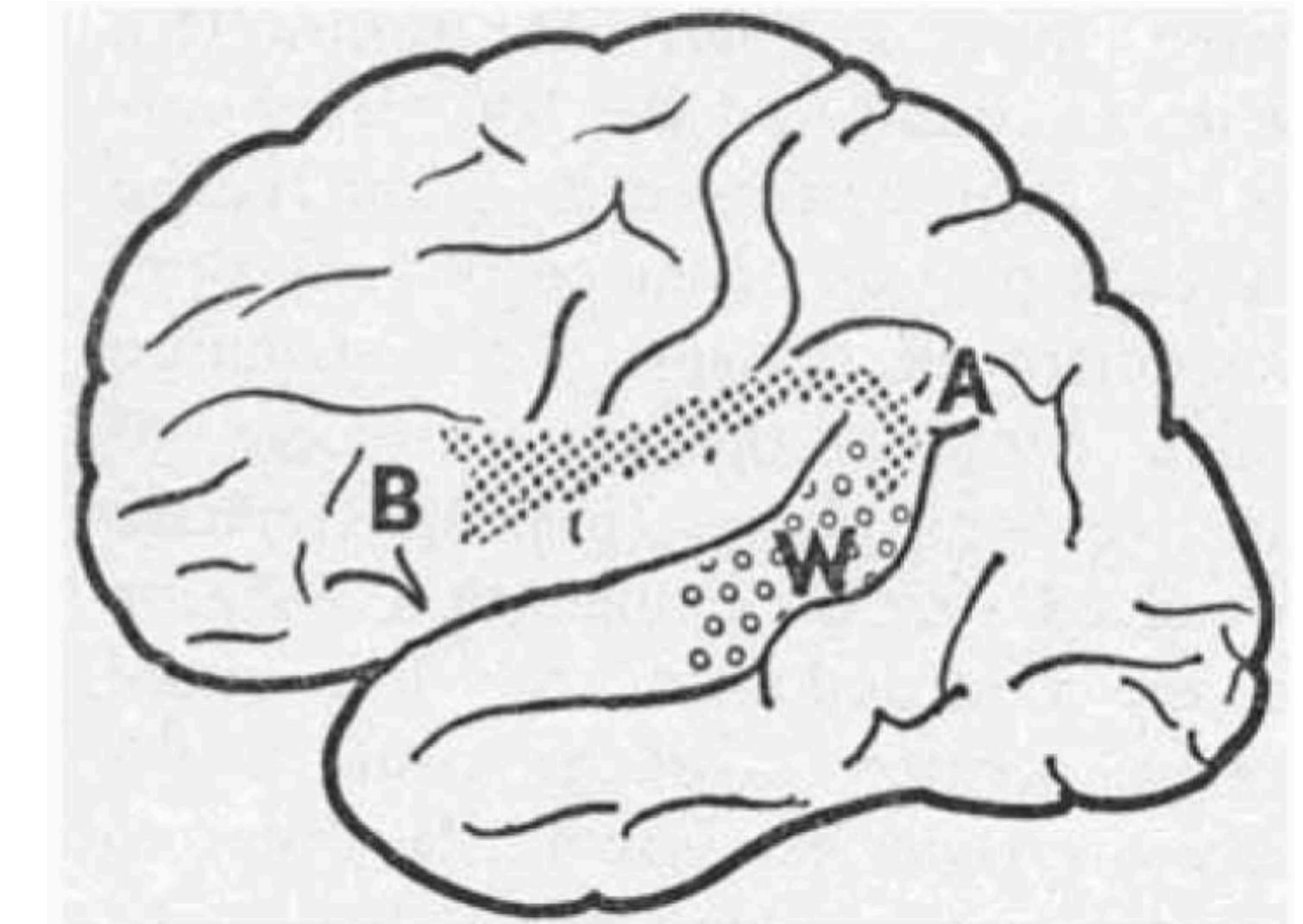


Fig. 2. Lateral surface of the left hemisphere of the human brain. *B*, Broca's area, which lies anterior to the lower end of the motor cortex; *W* (open circles), Wernicke's area; *A* (closed circles), arcuate fasciculus, which connects Wernicke's to Broca's area. (See text.)



“Broca and Wernicke are dead”

- The Classic Model still prevails in modern medical textbooks, but the real picture is much, much more complicated.
- “All models are wrong; some are useful.”

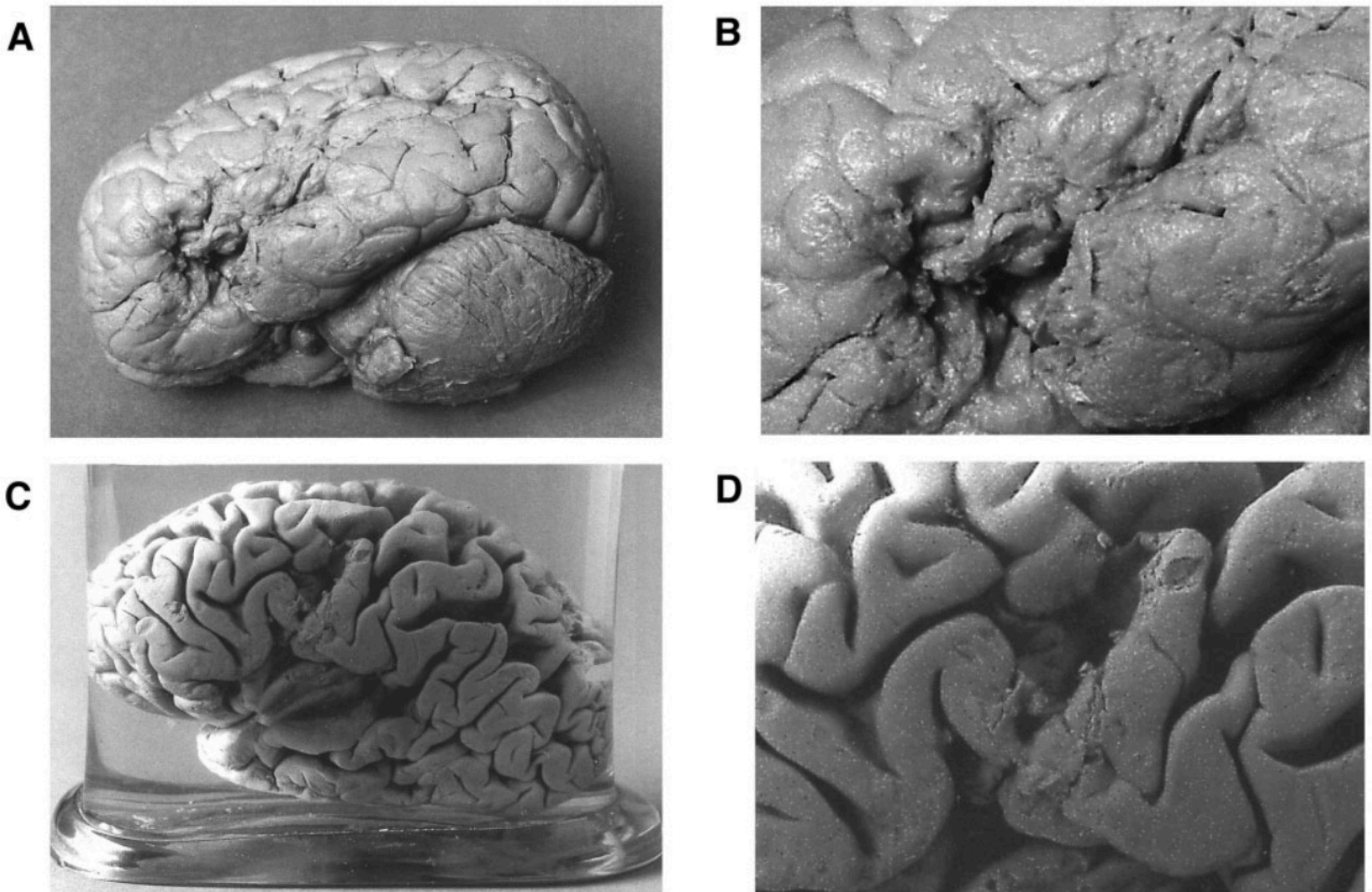
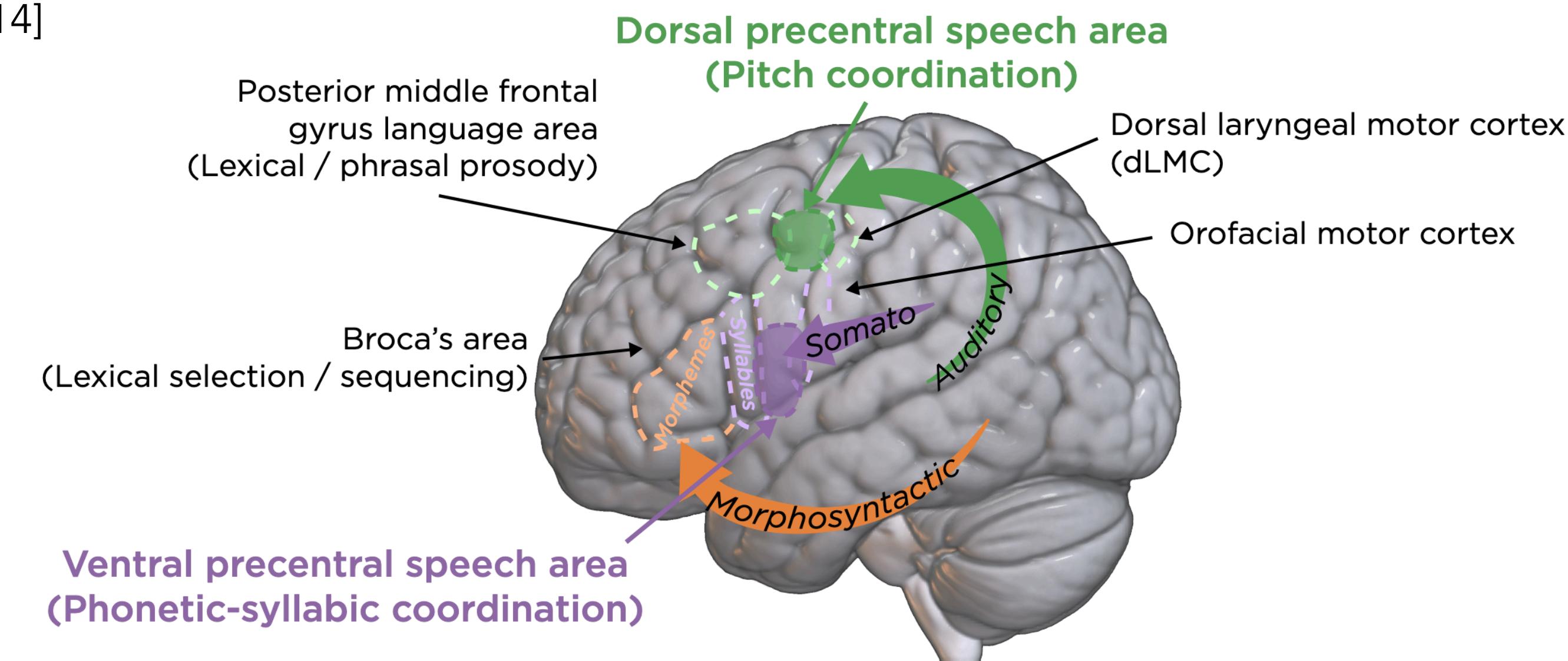


Fig. 3 Photographs of the brains of Leborgne and Lelong, Paul Broca's first two aphasic patients. **(A)** Lateral view of the brain of the first patient, Leborgne. The external lesion is clearly visible in the inferior frontal lobe. The softening in the area superior and posterior to the lesion suggests further cortical and subcortical involvement. **(B)** Close-up of the visible lesion in Leborgne's brain. **(C)** Lateral view of Broca's second patient, Lelong. The frontal, temporal and parietal lobes have retracted due to severe atrophy, exposing the insula. **(D)** Close-up of the visible lesion in Lelong's brain. Note that only the most posterior part of what is currently called Broca's area is infarcted; the anterior portion is completely spared.



“Beyond Broca”

- A 21st century model of the neurobiology of speech motor control [14]
- Two important sub-regions in the precentral gyrus for different aspects of motor speech coordination:
 - **Dorsal precentral speech area (dPCSA)** controls prosody
 - **Ventral precentral speech area (vPCSA)** controls phonetics
 - Alternatively: dPCSA is **laryngeal** and vPCSA is **supra-laryngeal**.





Mapping brain networks important for apraxia of speech

AKA, my research on feedforward speech motor control



Mapping the brain?

Lesion-symptom mapping (LSM): technique for associating brain & behavior

- **Step 1**: Segment the lesion
- **Step 2**: Break the brain down into tiny parts (usually **voxels**)
- **Step 3**: Perform a statistical test at each voxel to see if damage to that voxel correlates with behavior deficits
- **Step 4**: Visualize all statistically significant voxels at once on the brain; that's your map!

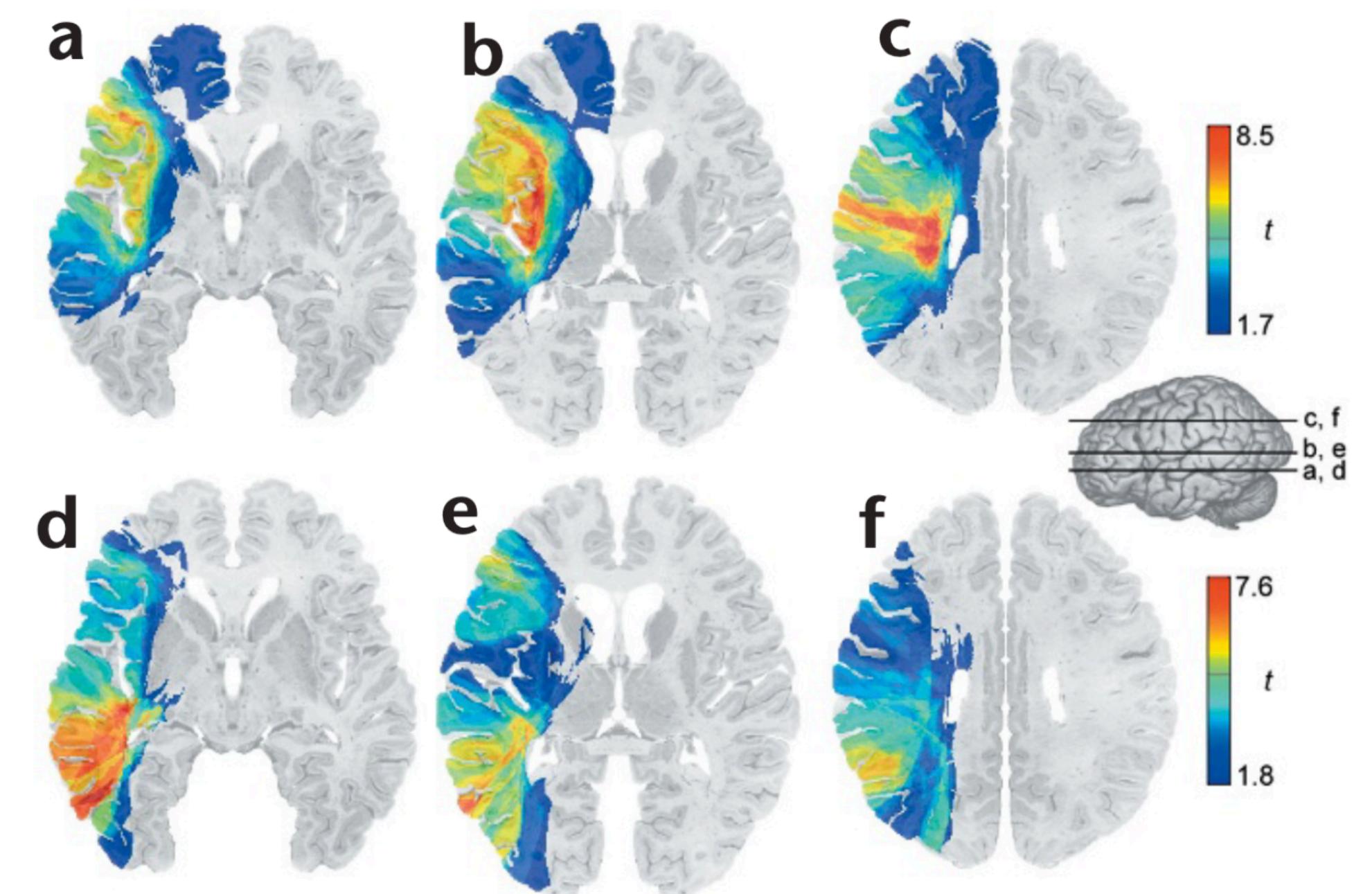


Fig. 1. Representative slices from VLSM maps computed for fluency and auditory comprehension performance of 101 aphasic stroke patients.



Issues with conventional VLSM

- Assumes independence (each voxel is its own p value)
- Heavy statistical correction required
- Only one solution presented
- “Researcher degrees of freedom”

What would a better technique look like?

- Treats brain as a **connected network** rather than independent parcels of tissue
- Describes **multiple plausible brain-behavior relationships**
- Limit need for statistical correction and parameter-tuning; keep it **simple & interpretable**



Critical Network Lesion-Symptom Mapping

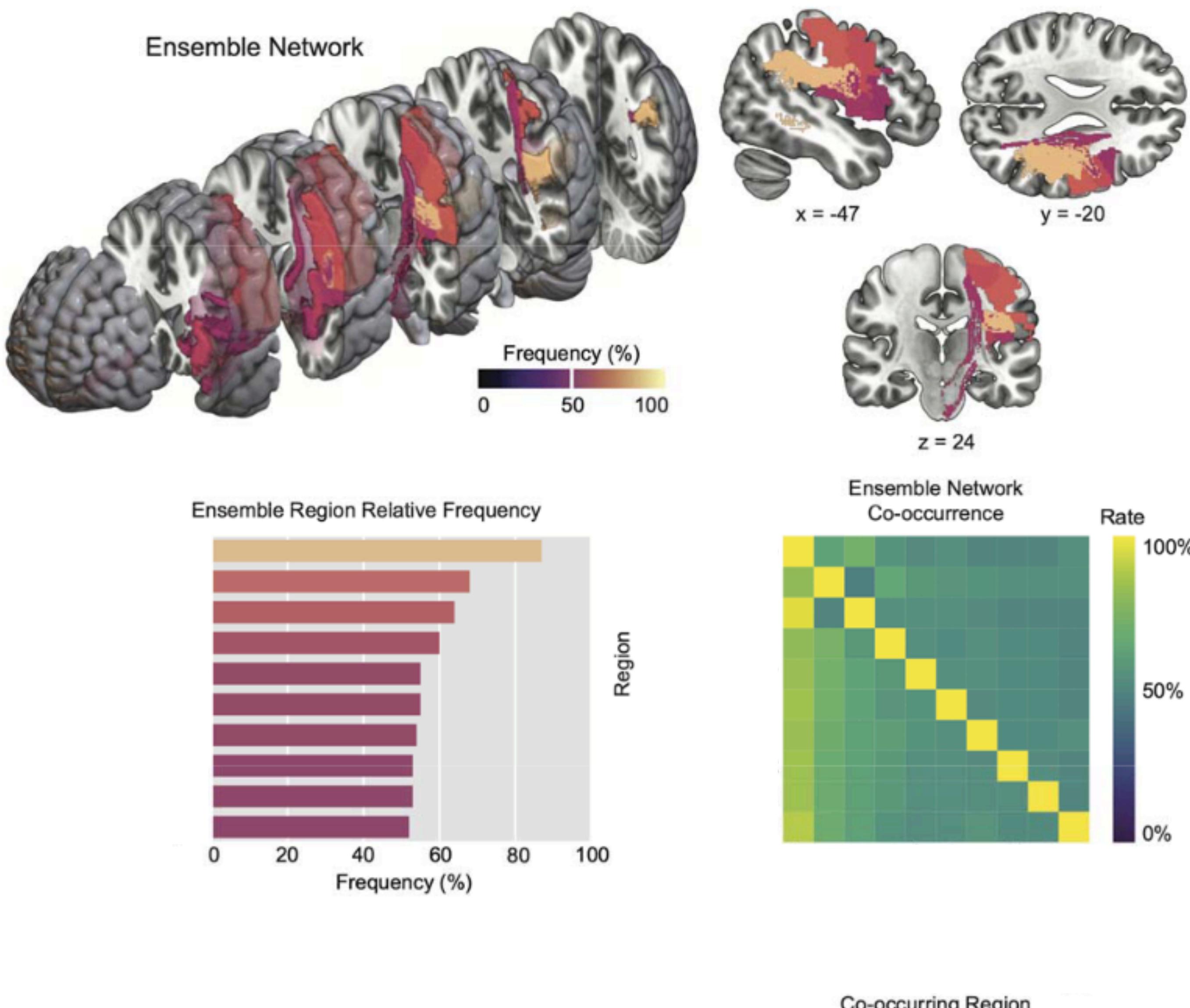
Collaboration with Grant Walker & Akbar Hussain

- Instead of voxelwise statistics, map brain-behavior region-by-region
 - Step 1: Calculate significance for each brain region separately
 - Step 2: Test different combinations (*networks*) of regions' ability to predict behavior
 - Step 3: Generate report of all critical networks that model behavior well (there can be many!)
- Used prediction-based statistics instead of conventional t tests
- Statistical model is kept small/simple compared to fancy machine learning models





adapted from Kurteff et al., 2025 [16]



Critical Network Lesion-Symptom Mapping



The POLAR cohort

Predicting Outcomes of Language Rehabilitation, a large cohort of stroke survivors

- Collected in collaboration with UCI at the Center for the Study of Aphasia Treatment & Recovery (**C-STAR**) at U. South Carolina
- 57% (61/127) of participants have AOS
- AOS assessed using the Apraxia of Speech Rating Scale (**ASRS**^[16, 17])





Apraxia of Speech Rating Scale

Score	0	1	2	3	4
Description	Not observed in any task	Infrequent	Frequent but not pervasive	Very often evident but not marked in severity	Nearly always evident and/or marked in severity
Guidelines (estimate of feature prevalence)	No more than one occurrence	Noted more than once (but less than about 20% of words)	Noted in about 20–50% of words	Noted in majority of words	Noted in nearly all words
Exceptions	<ul style="list-style-type: none"> • Score no higher than “2” if present only during repetition tasks. • Score a “4” if intelligibility is more than mildly reduced by the feature. • Performance on AMRs and SMRs should be considered only for items 9–11 				

Phonetic Features*

1 Sound distortions (excluding distorted substitutions or distorted additions)
 2 Distorted sound substitutions
 3 Distorted sound/syllable additions (including intrusive schwa)
 4 Increased sound distortions or distorted sound substitutions with increased utterance length or increased syllable/word articulatory complexity

Prosodic Features*

5 Syllable segmentation within words > 1 syllable
 (Brief silent interval between syllables &/or inappropriate equalized stress across syllables)
 6 Syllable segmentation across words in phrases/sentences
 (Increased inter-word intervals &/or inappropriate equalized stress across words)
 7 Slow overall speech rate (apart from pauses for word retrieval and/or verbal formulation)
 8 Lengthened vowel &/or consonant segments independent of overall slow speaking rate

vPCSA?

dPCSA?

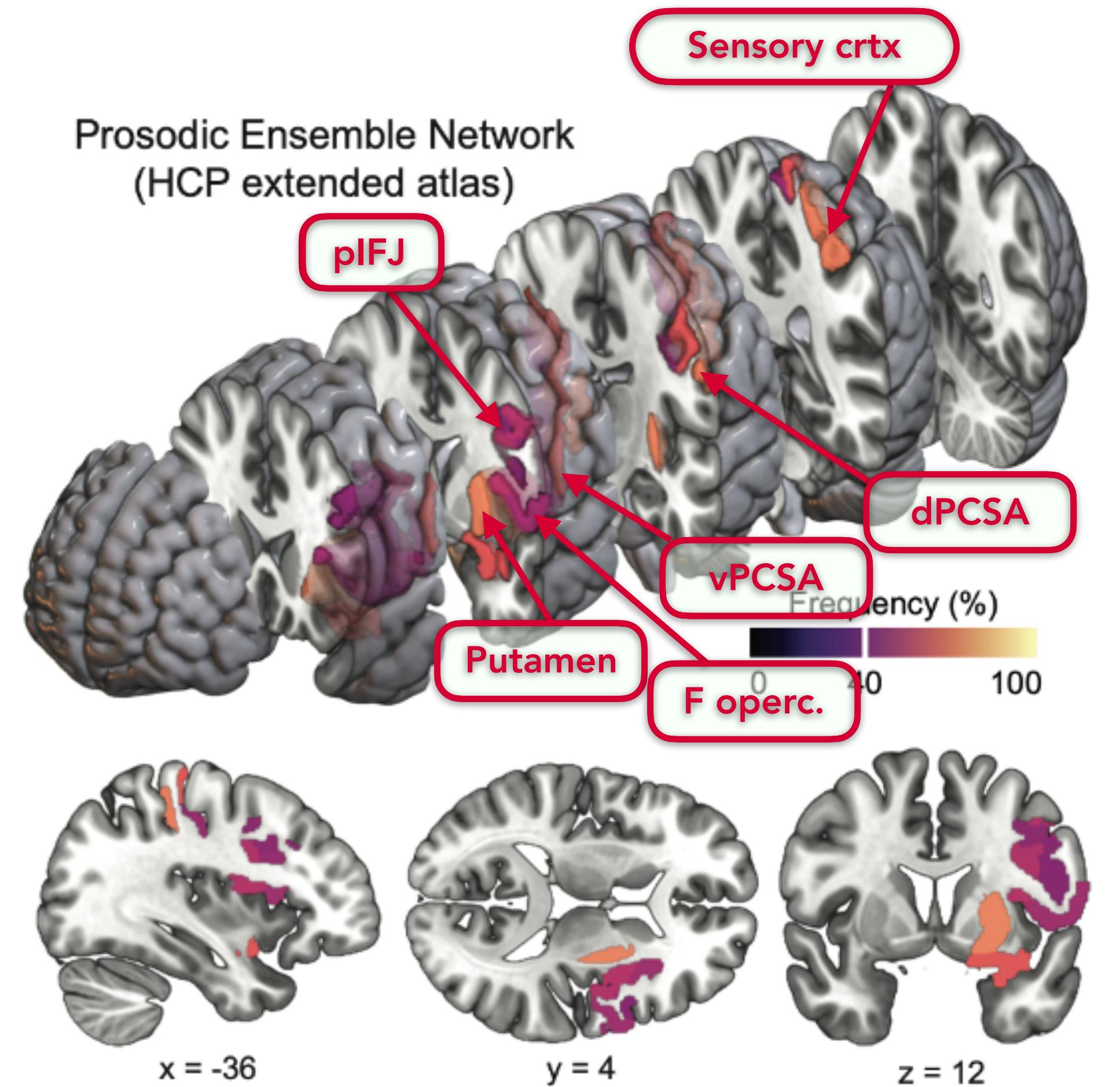
Duffy et al., 2023 [18]



Does prosody map onto dPCSA?

Yes, and also ...

- Inferior frontal junction (directly anterior to dPCSA)
- Frontal operculum (bottom “fold” of frontal lobe, near insula)
- Putamen (subcortical nucleus involved in motor function; part of basal ganglia)
- Sensorimotor cortex (including dPCSA and... vPCSA?!)

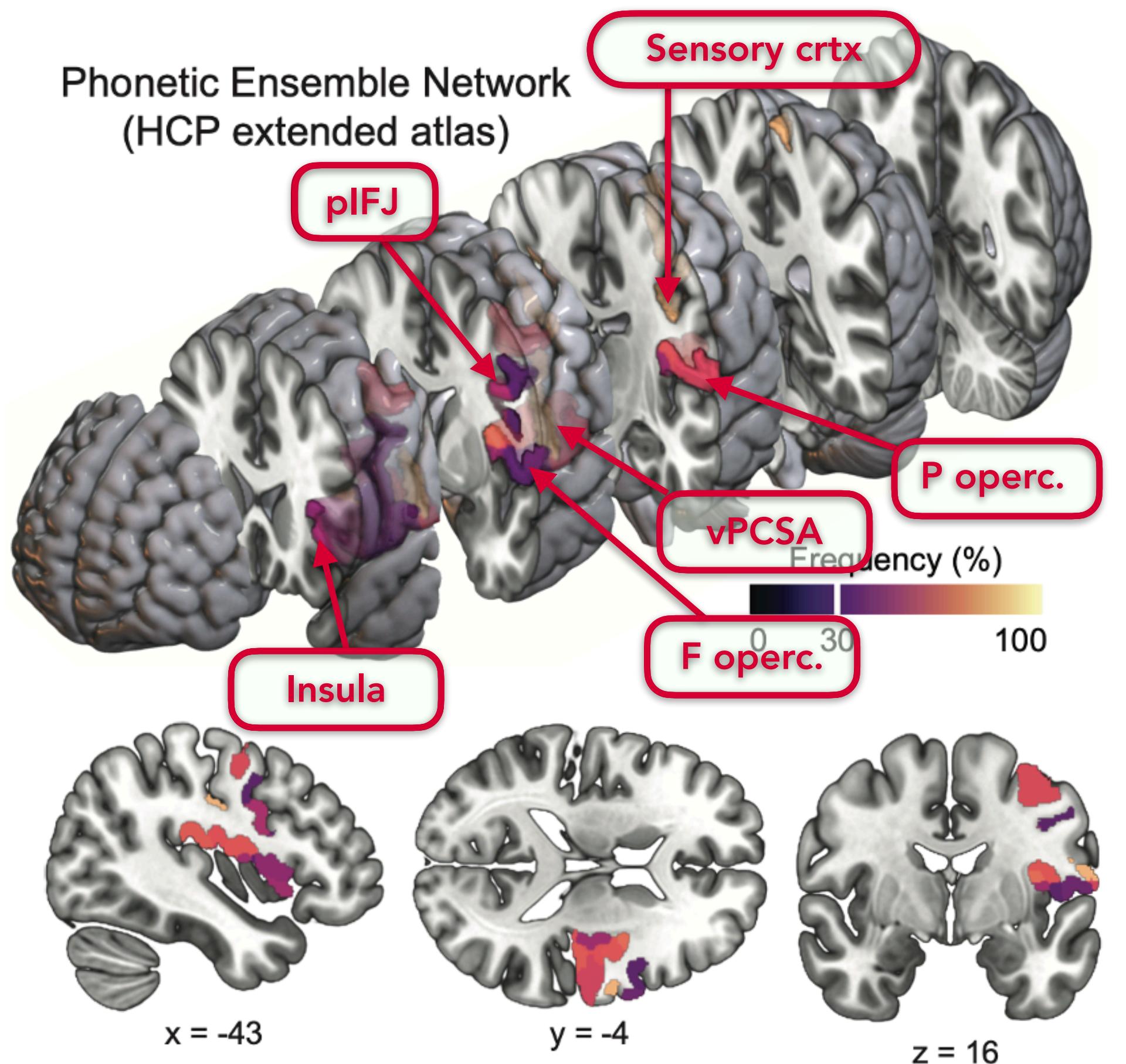




Does phonetics map onto the vPCSA?

Again, **Yes**, but also ...

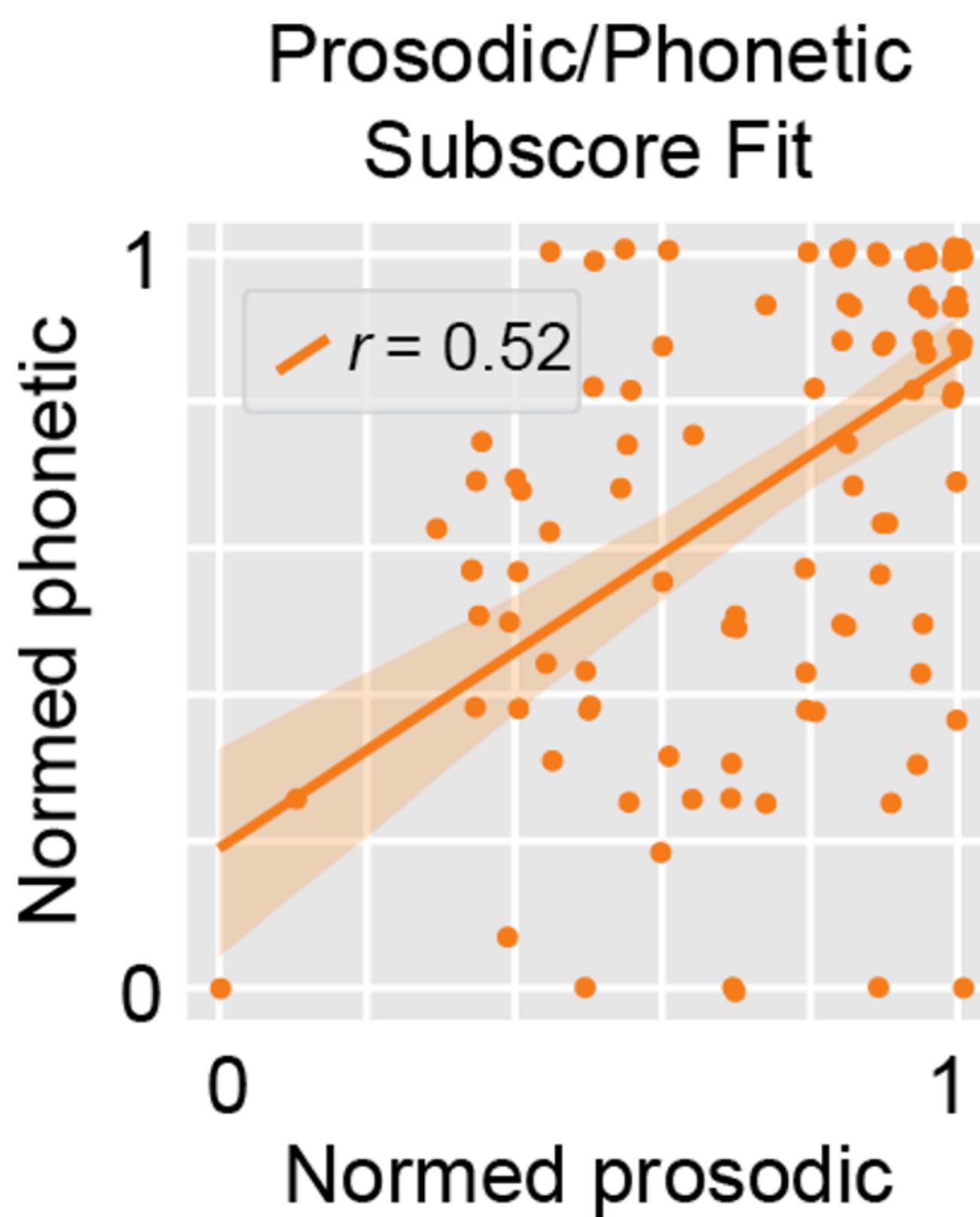
- Partial overlap with prosodic subscore map (IFJ, sensorimotor, frontal operculum)
- Insula (middle/granular aspect; classic AOS area)
- Parietal operculum (bottom “fold” of parietal lobe)





What gives?

- dPCSA and vPCSA are clearly important brain regions in AOS, but they **aren't alone**
 - General trend: prosody is frontal, sensorimotor & subcortical motor; phonetics is frontal, sensorimotor, parietal, & insula
- Partial overlap in networks could be due to **collinearity** in severe cases
- If many regions are involved, perhaps mapping **white matter** can tell us more

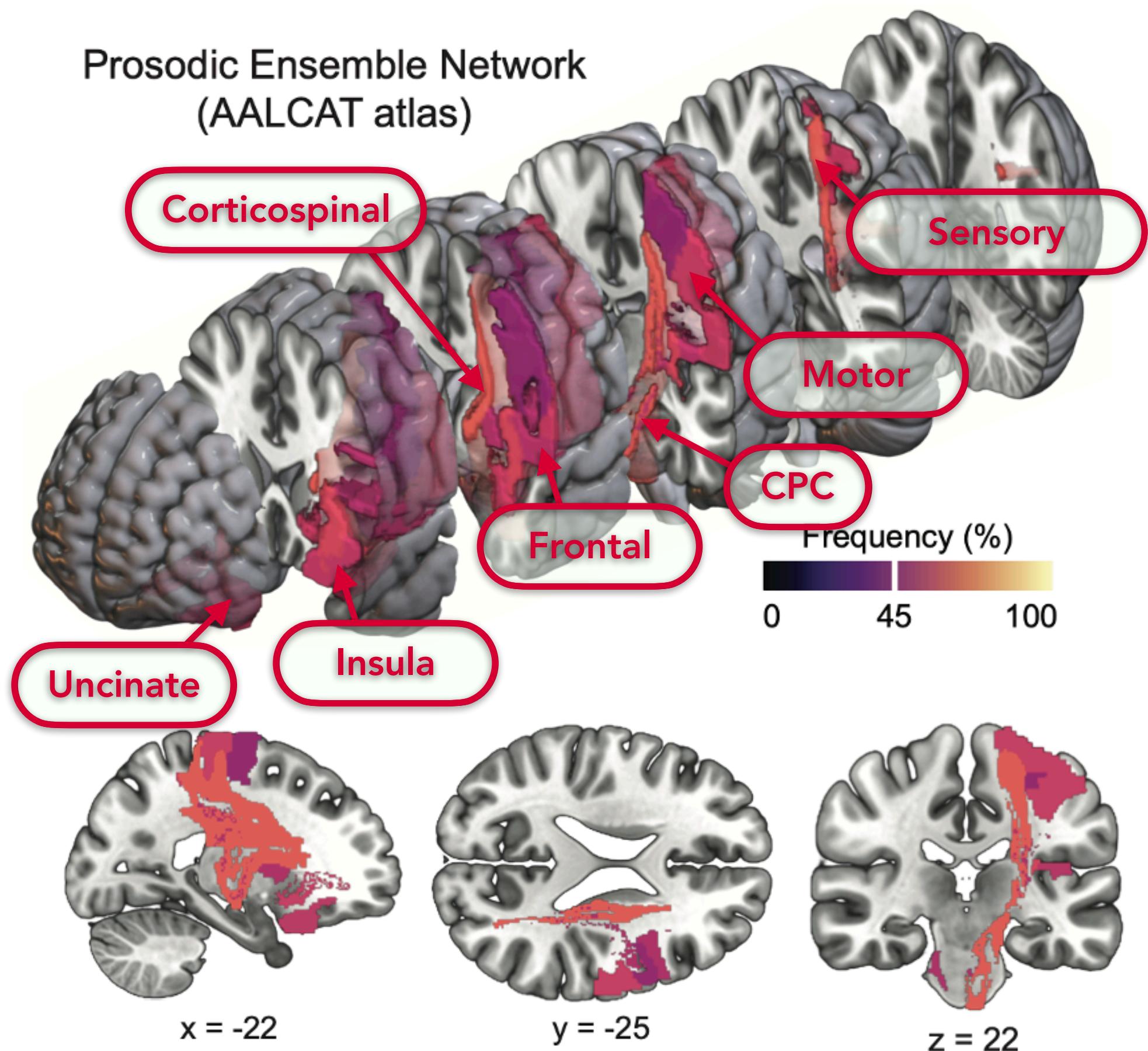




White matter supporting prosodic function

White matter pathways associated with the **prosodic** subscore of the ASRS included:

- Corticospinal tract (efferent motor fiber pathway, including larynx)
- Cortico-ponto-cerebellar tract (cerebellum important for motor coordination)
- Uncinate fasciculus (connects anterior temporal lobe to inferior frontal)
- Intra-region fibers of insula, sensorimotor, & frontal



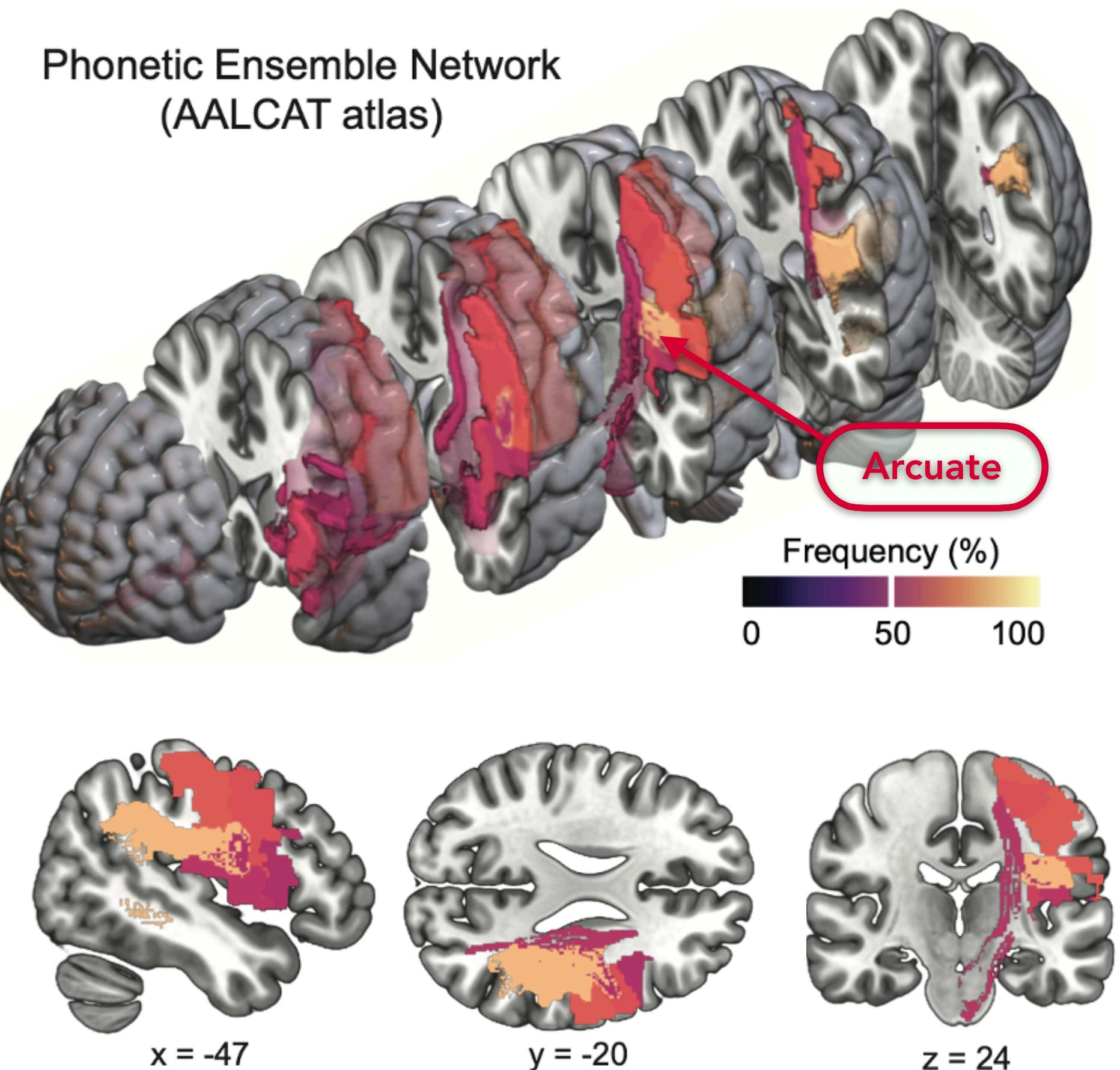


White matter supporting phonetic function

White matter pathways associated with the **phonetic** subscore of the ASRS included:

- Anterior segment of the arcuate fasciculus (connects inferior frontal/insula to inferior parietal)
- Long segment of the arcuate fasciculus (connects inferior frontal/insula to posterior temporal)

...that's pretty much it!





The prosodic/phonetic networks

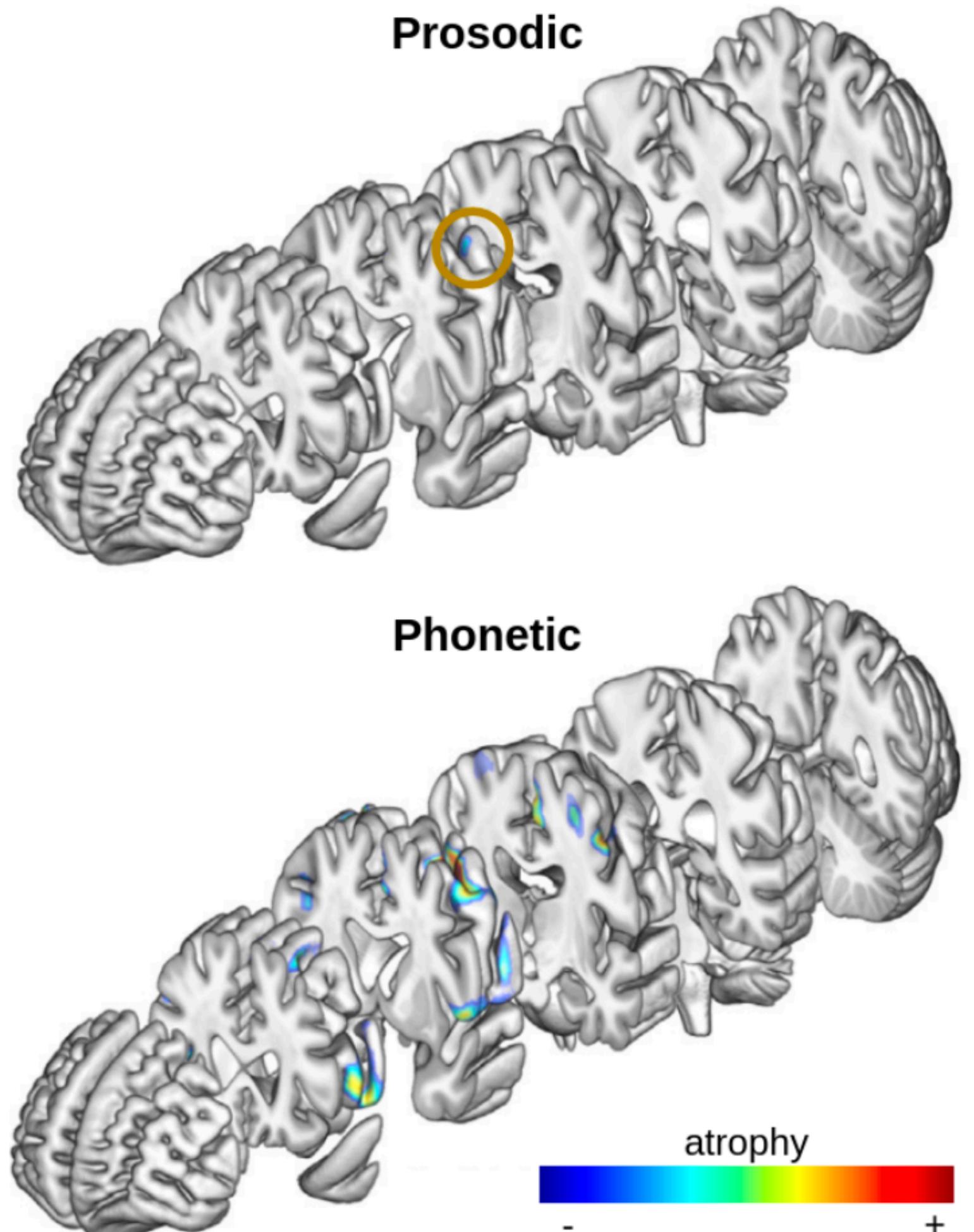
	Prosodic	Phonetic
Regions in network	<ul style="list-style-type: none"> - Sensorimotor cortex - Inferior frontal cortex - Subcortical motor nuclei - Long distance motor fibers - Fronto-temporal white matter 	<ul style="list-style-type: none"> - Sensorimotor cortex - Inferior frontal cortex - Inferior parietal cortex - Anterior/middle insula - Arcuate fasciculus
Network function	<ul style="list-style-type: none"> - Laryngeal motor initiation, control, and coordination - Auditory-motor connectivity 	<ul style="list-style-type: none"> - Transformation of phonological-linguistic information (parietal) into articulatory commands (frontal) - Motor coordination

Takeaway: it's not just the dPCSA and vPCSA that are important!



Prosodic & phonetic progressive AOS

- In neurodegenerative AOS, a split between prosodic & phonetic subtypes has been proposed [19]
- **Prosodic** AOS: Slow rate, syllable segmentation difficulties, poor speech-breath coordination
 - Prosodic atrophy localizes pretty perfectly to dPCSA, in line with "Beyond Broca"
- **Phonetic** AOS: Distorted sound substitutions, difficulty with more complex syllables
 - Phonetic atrophy is a little more distributed across frontal/parietal cortex

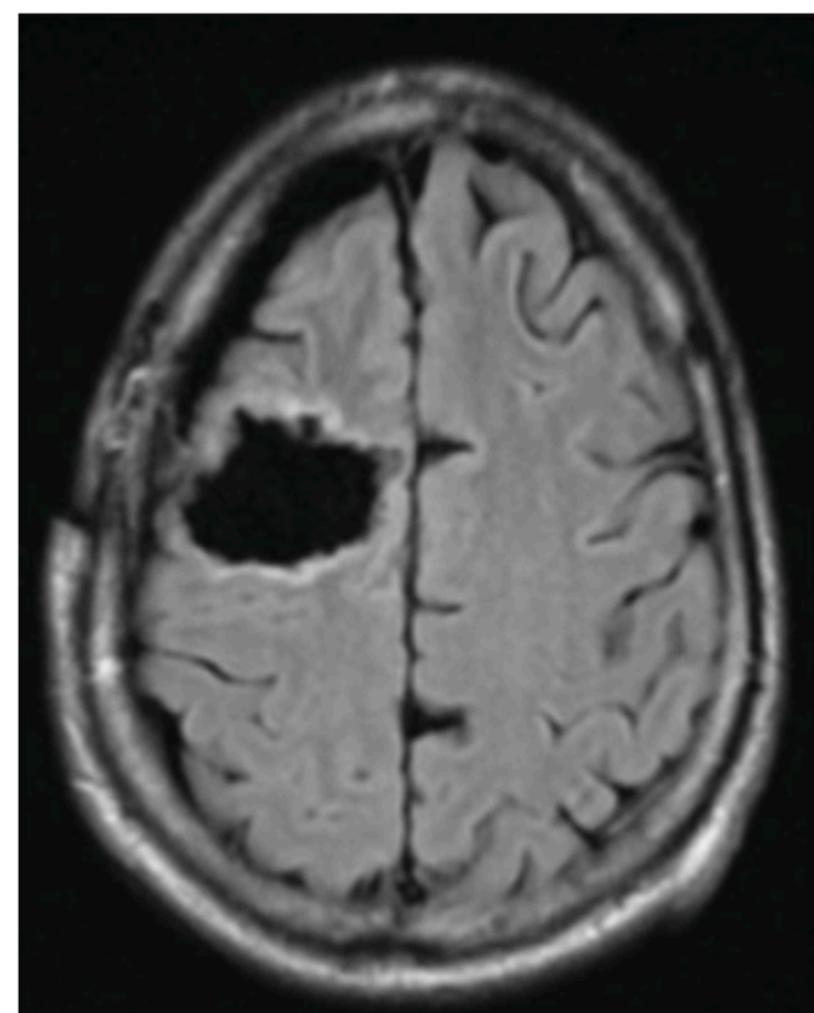




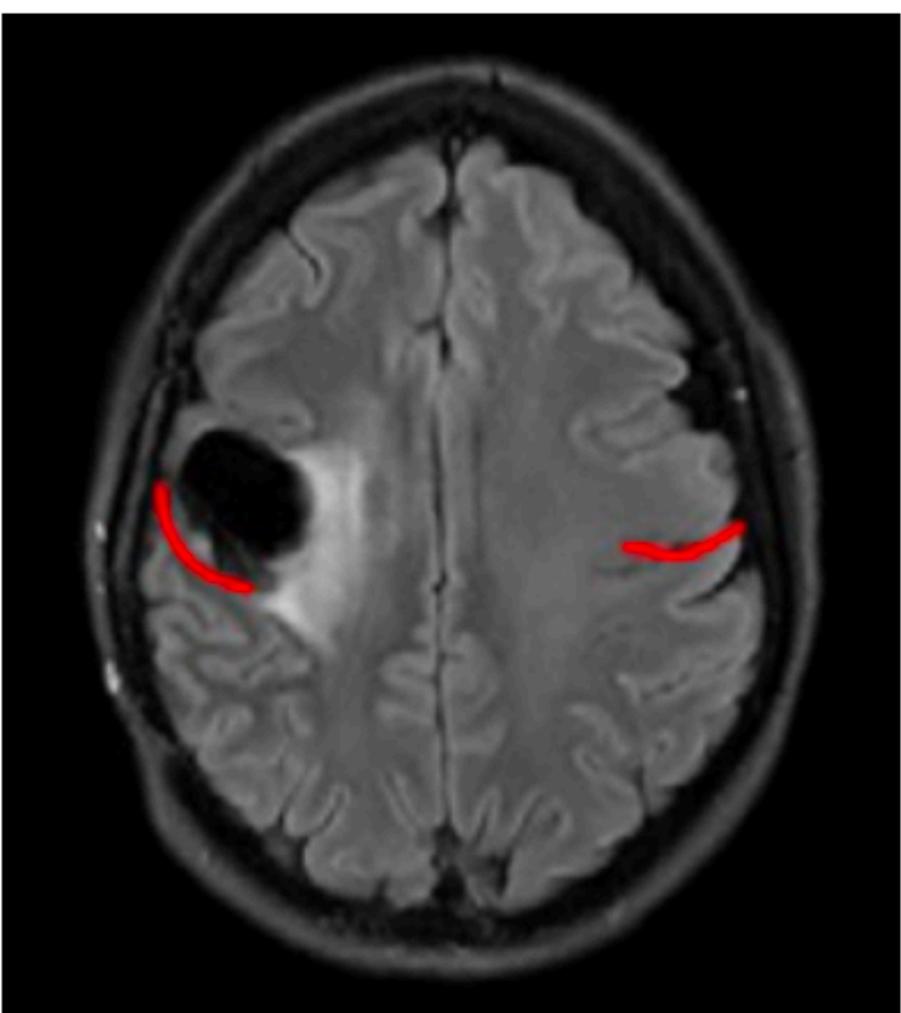
Case studies of AOS after dPCSA damage

Two different case studies showing relatively isolated AOS after damage to dPCSA and adjacent regions

- **Case 1:** AOS with prosodic deficits & without aphasia after surgical resection of dPCSA [13]
- **Case 2:** AOS with prosodic deficits, dyslexia, & dysgraphia after surgical resection of dPCSA and posterior middle frontal gyrus [34]



Case 1



Case 2



Is prosody harder to pin down?

Overall, the prosody network was more distributed than the phonetic one

- Prosody is more of a “footnote” in classic pathology of AOS
 - Mentioned in classic AOS textbooks, but more emphasis placed on motor coordination and speech sound distortion [22, 23]
- Many characteristics of prosodic AOS overlap with dysarthria and even dysphonia
- Remember: **stroke data is messy!**

“The characteristics of [phonetic AOS] generally seem to be emphasized in the AOS diagnosis. In contrast,... [prosodic AOS] appears to be underemphasized or not recognized, perhaps because the characteristics are more subtle or are considered to reflect dysarthria.”

from Josephs et al., 2013 [24]

[22] Darley, Aronson, & Brown, 1975

[23] Wertz, LaPointe, & Rosenbek, 1984



Clinical management of prosodic and phonetic deficits

Cliché, but ... **more work is needed.**

- Neuroscience separates prosodic from phonetic function at this point
- Clinically though, most patients are a mixture of both
 - **Evaluation:** It could be worth assessing function in prosodic/phonetic domains separately
 - **Treatment:** ...but, interventions that target both will likely be as effective as ones that separate the two
- Academic and clinical benefits of prosodic/phonetic subtyping may not always align



Auditory-motor monitoring of vocal feedback during speech

AKA, my research on feedback speech motor control



Neural biomarkers of feedback control

My PhD research, in part, focused on two auditory responses in the brain:

1. **Speaker-induced suppression** (our brain responds less to our own voice)
2. **Auditory onset responses** (some auditory neurons are active only at the beginning of a sound, while others are active throughout)

Additionally, I wanted to study these phenomena in a **naturalistic setting**





Speaker-induced suppression

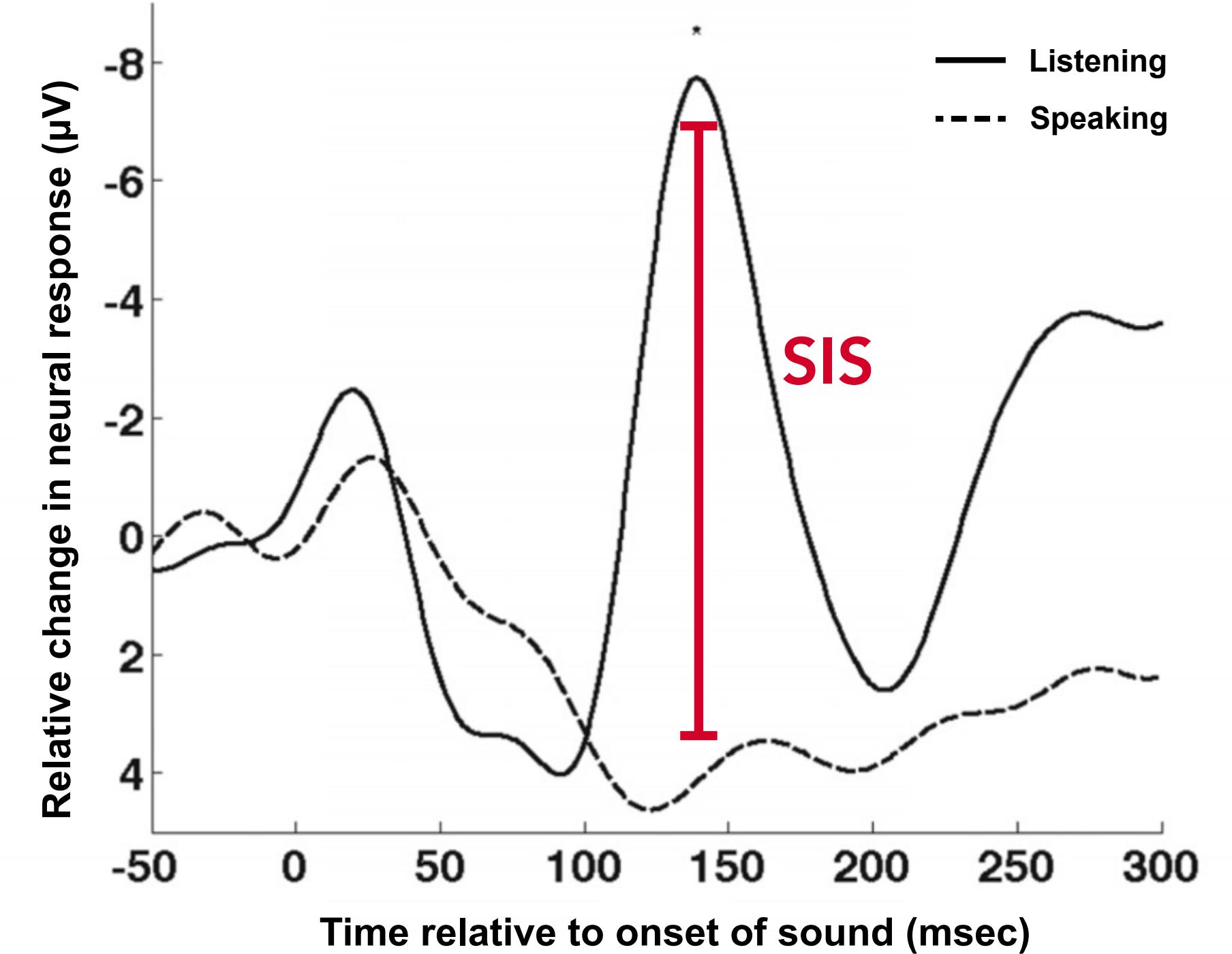
Speaker-induced suppression (SIS): Neural responses to your own voice are suppressed while speaking

- More brain activity when participants listen to recordings of their voice vs. speaking

SIS is a biomarker of the **feedback monitoring** system

- Less SIS when participants make errors

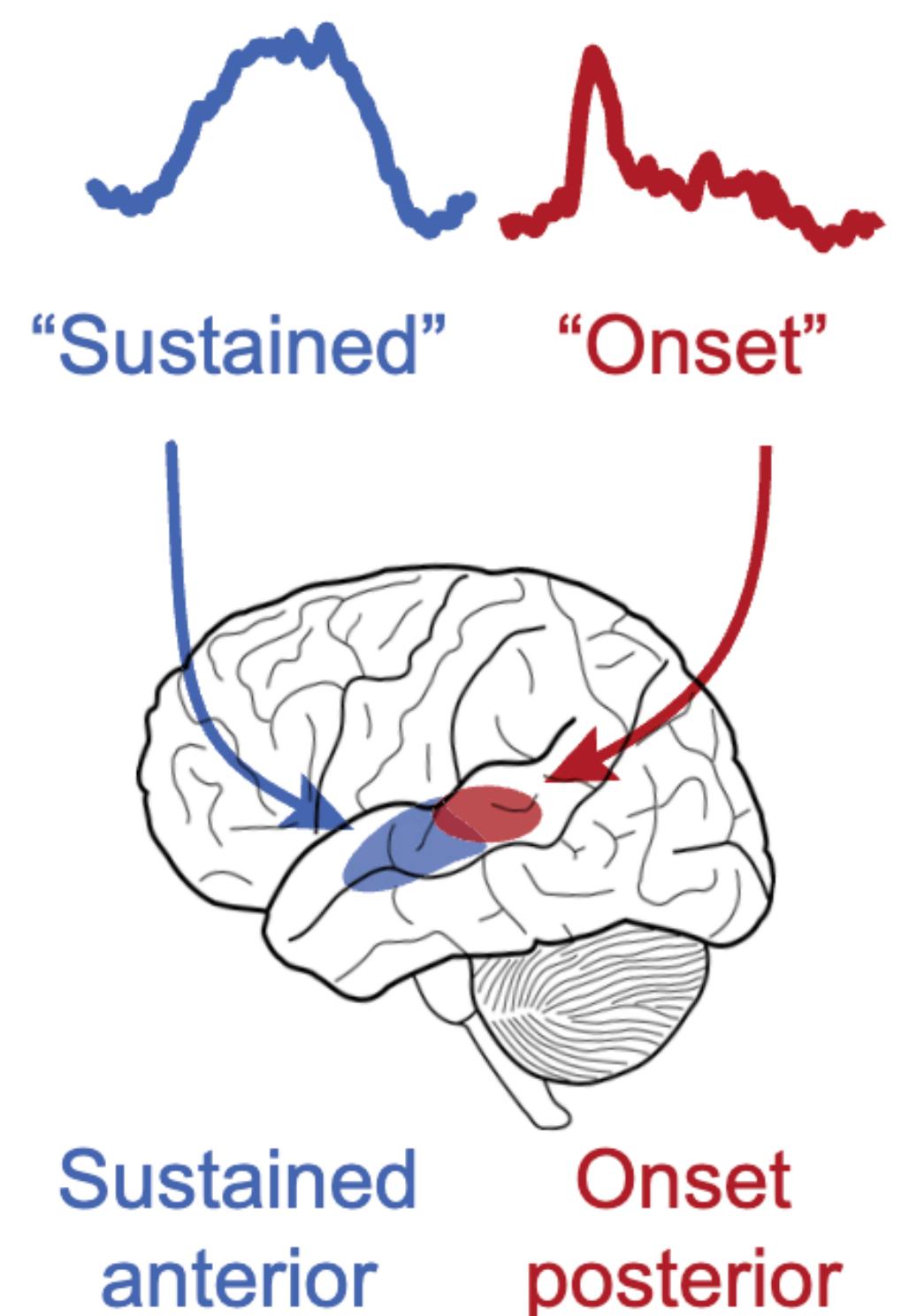
Most research on SIS takes place in heavily constrained experimental paradigms





Onset and sustained response profiles

- Two anatomically distinct patterns of temporal responses in auditory cortex:
 - **Onset** responses happen after >200 ms of silence
 - **Sustained** responses can happen anywhere in a speech stimulus
- What is the relationship between **onset responses** and speaker-induced suppression?



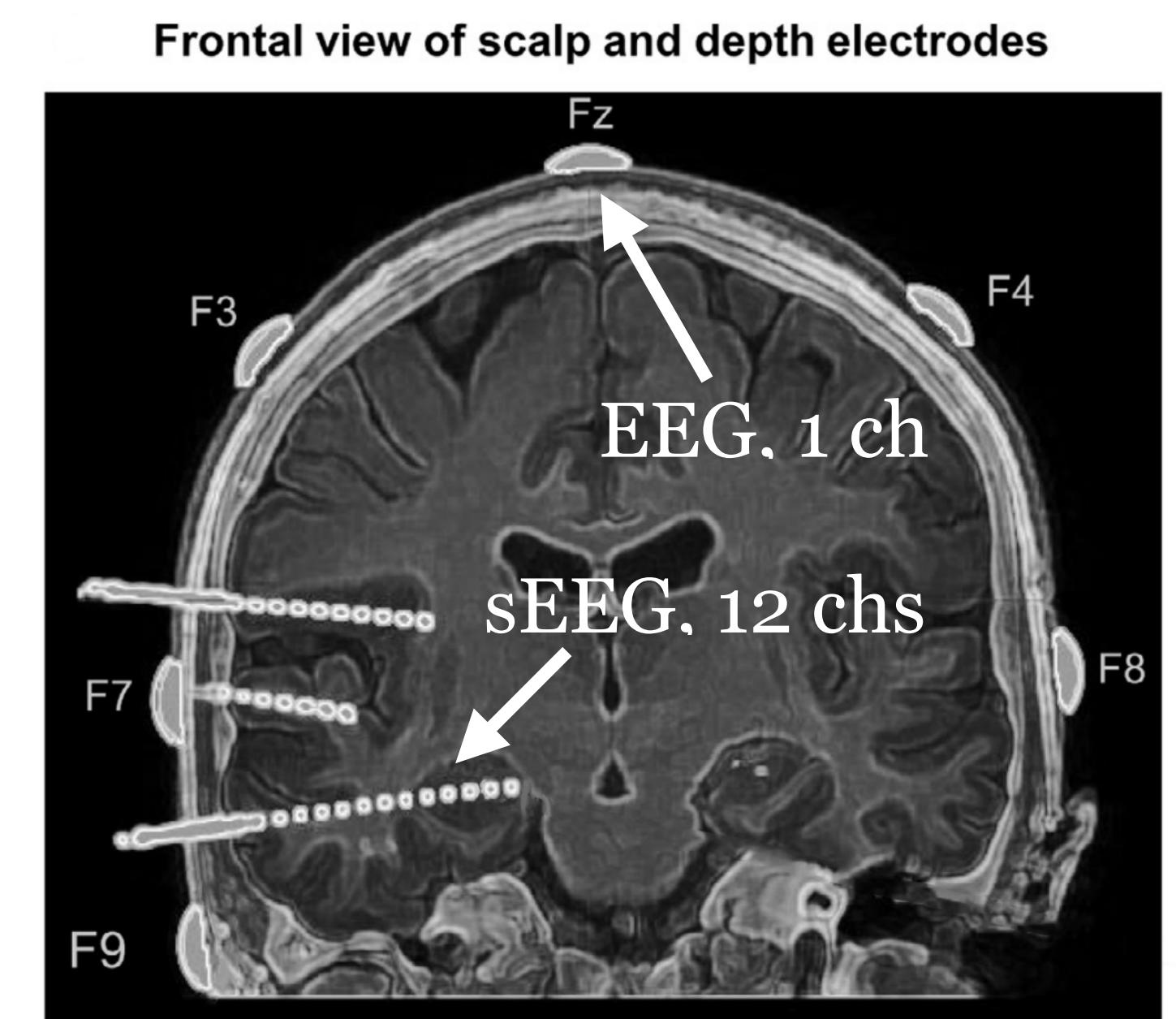
Sustained
anterior Onset
posterior





Two studies, one task

1. "Speaker-induced suppression in EEG during a naturalistic reading and listening task" [28]
 2. "Spatiotemporal mapping of auditory onsets during speech production" [29]
-
- Both studies use a **similar task**, but **different neuroimaging technique**
 - **Study 1:** Scalp EEG collected from UT undergrad/grad students
 - **Study 2:** Surgically implanted sEEG collected from epilepsy patients in Texas hospitals



from Ramantani, Maillard, & Koessler, 2016 [30]

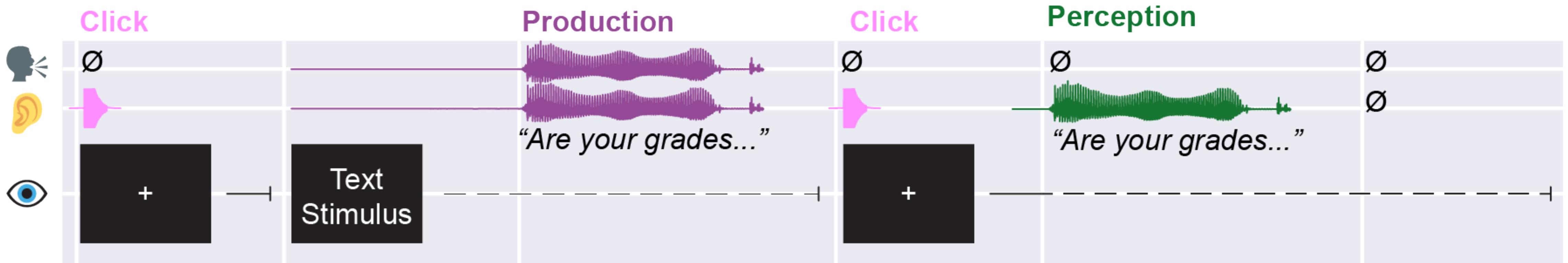




Reading and listening task

Participants read sentences aloud, then listened to playback

- Audio for **perception** trials generated from **production** trials, controlling for acoustics
- **Study 1:** $n=21$ UT students, EEG
- **Study 2:** $n=17$ epilepsy patients, sEEG



Trial #1:

Production (overt reading)

followed by

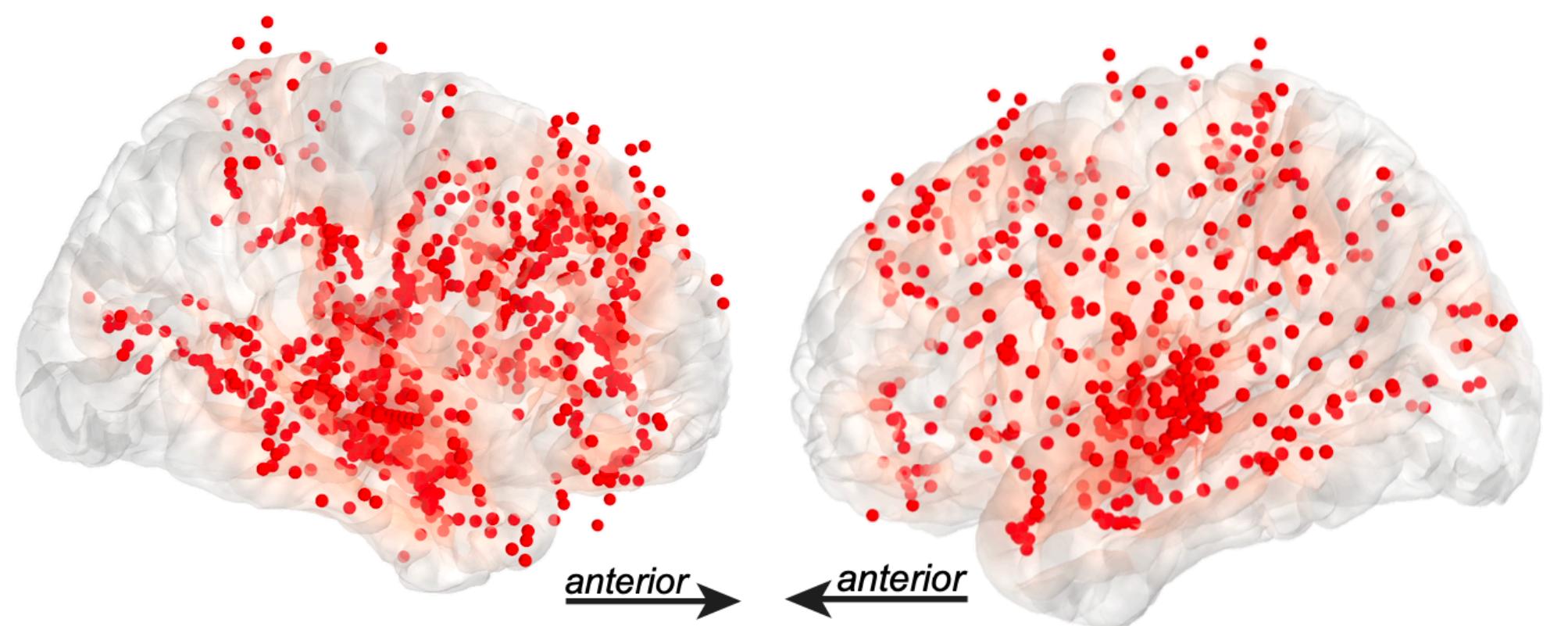
Perception (passive listening)

Task video



EEG vs. sEEG

	EEG	sEEG
Placement	On scalp	In brain
Requires surgery?	No	Yes
Population	Almost anyone	Medically necessary epilepsy cases
Electrode spacing	>5cm	<5mm
Frequency range	<30 Hz	<150 Hz
Sampling rate	10,000+ Hz	10,000+ Hz
Auditory biomarker	N1-P2 complex	High gamma activity (70-150 Hz)



top image credit: Lynn Kurteff
bottom: adapted from Kurteff et al., 2024 [27]

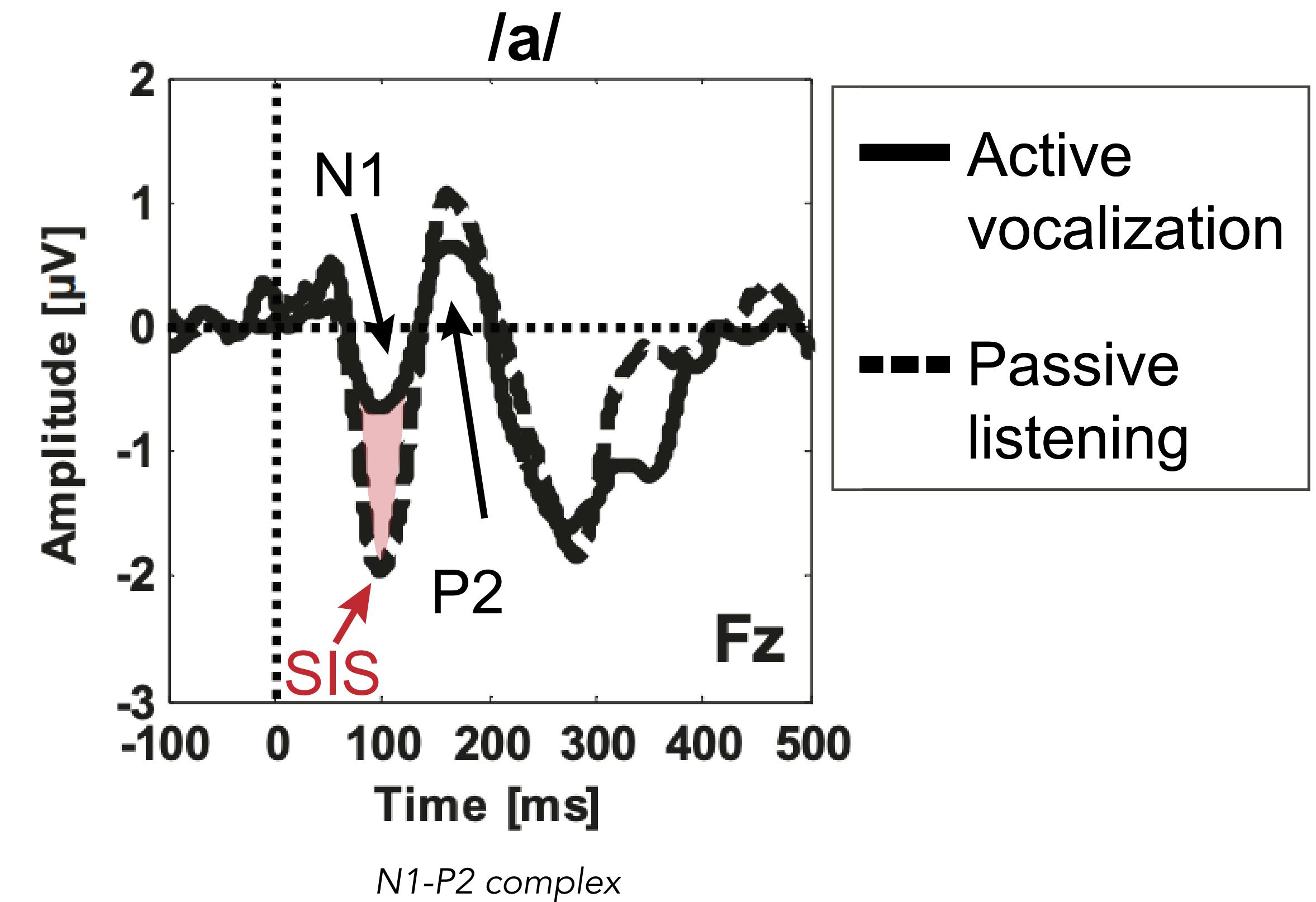


Outcome measures

EEG: The **N1-P2 complex**

- Biomarker of speaker-induced suppression
- sEEG: **High gamma** ($H\gamma$) activity
- $H\gamma$ activity correlates strongly with single-neuron responses to speech stimuli [25]
 - $H\gamma$ is also outcome measure for onset responses

Both are **event-related potentials**: activity over time, relative to sentence onset

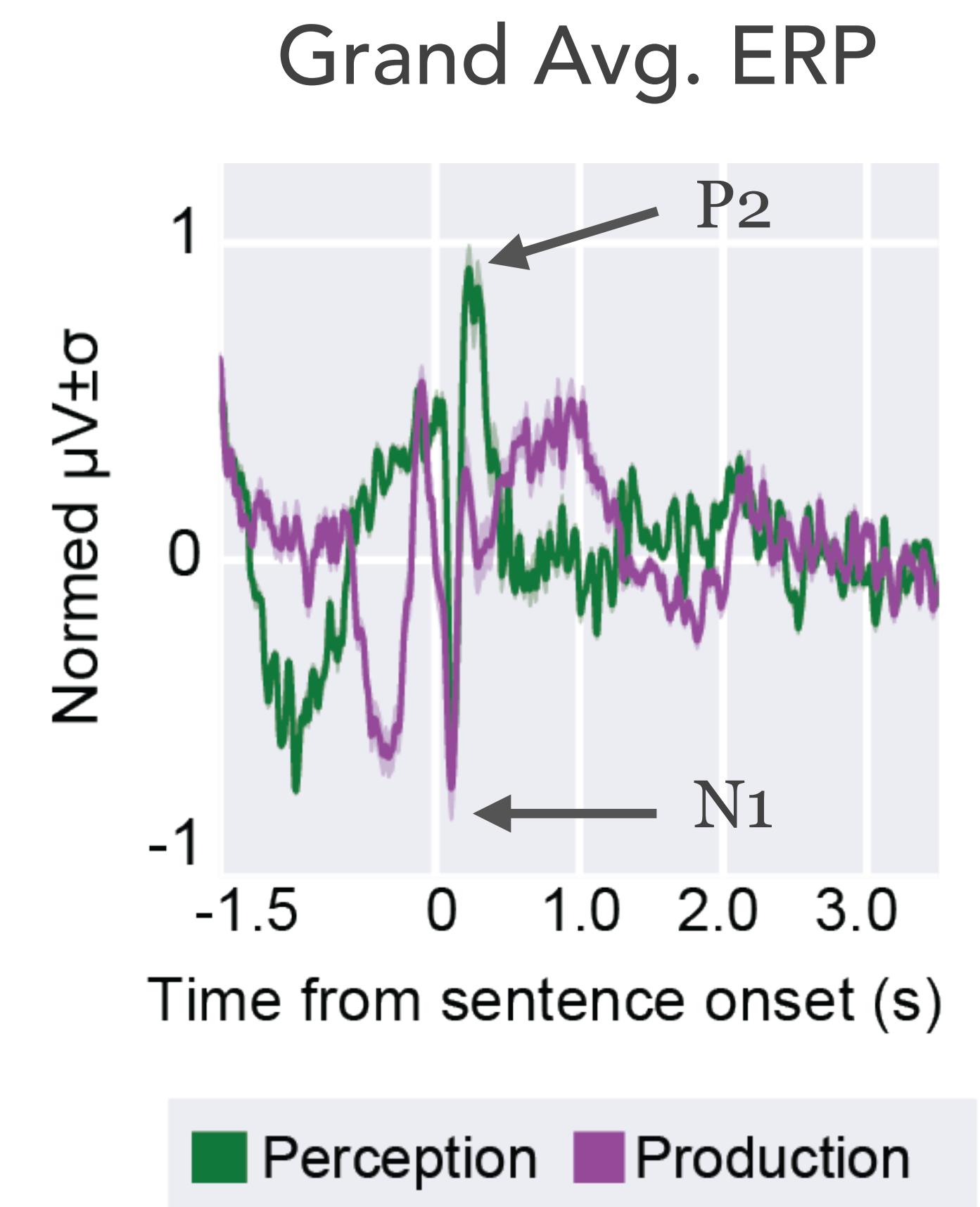




SIS is a naturalistic phenomenon

The N1-P2 complex is suppressed in naturalistic speech production

- ERPs show a difference in early components (N1/P2) between **perception** and **production** in frontal/central channels bilaterally
- Not an isolated “laboratory speech effect”

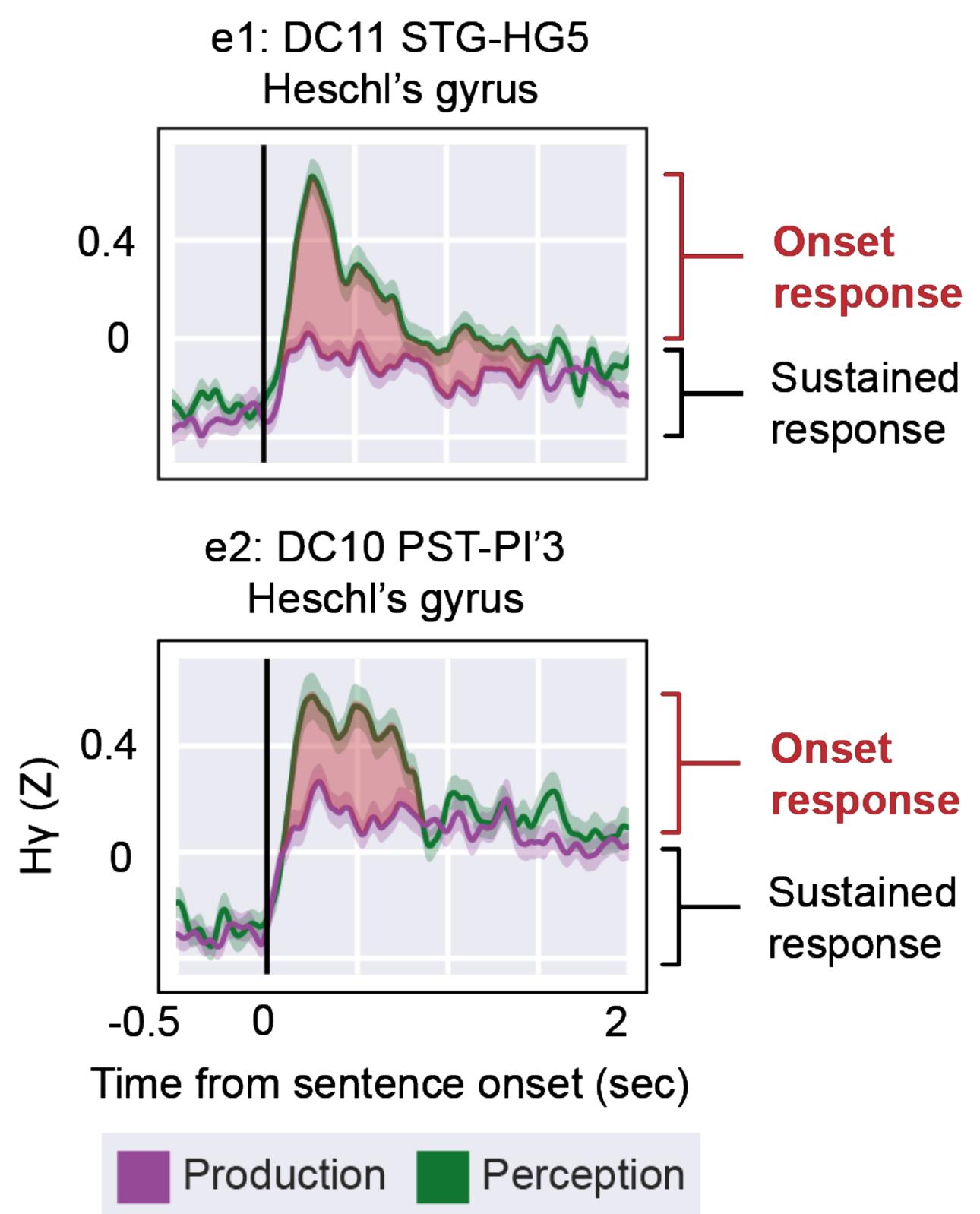
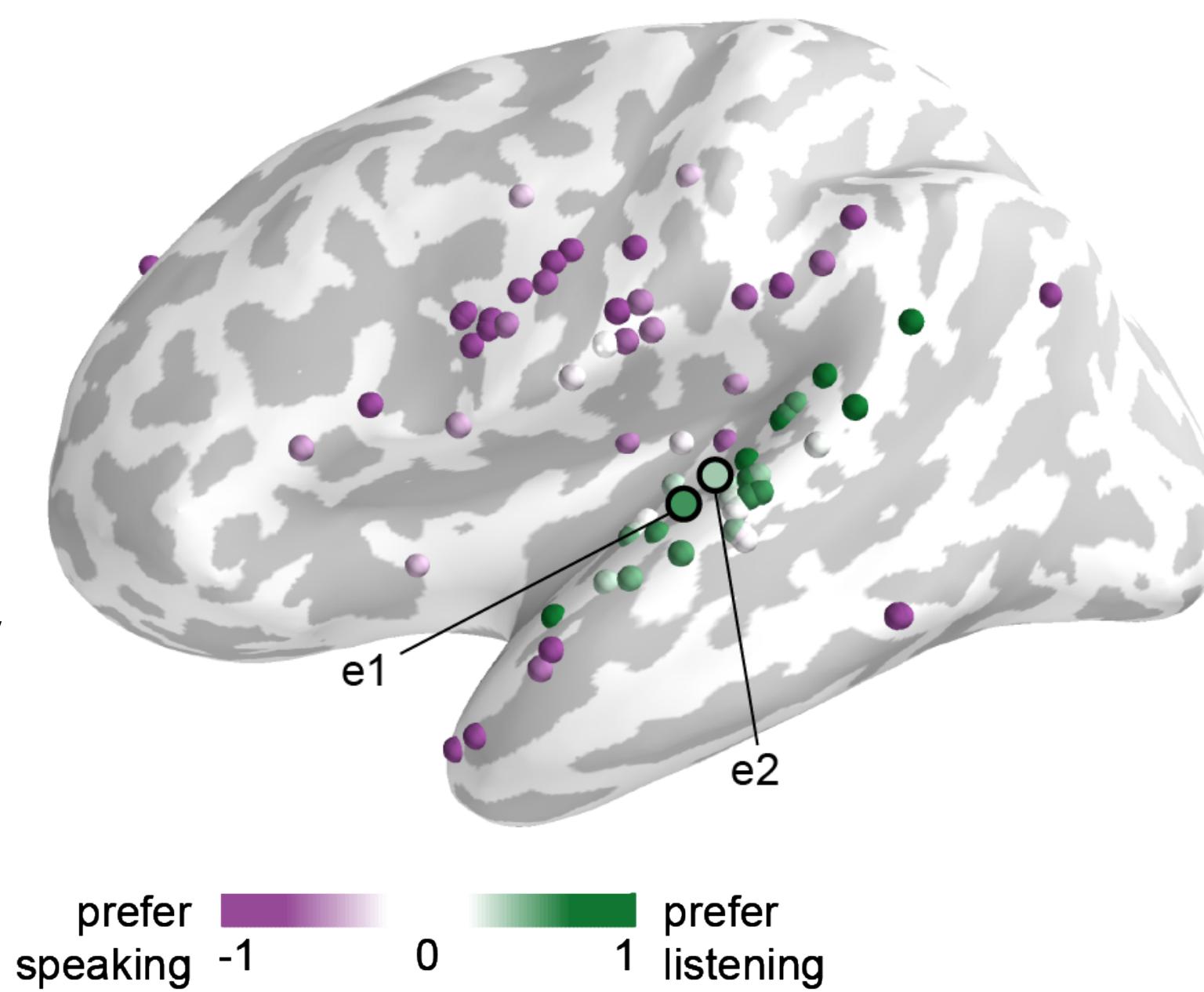




SIS affects onset responses, but not sustained

Onset responses are **suppressed** during speech production

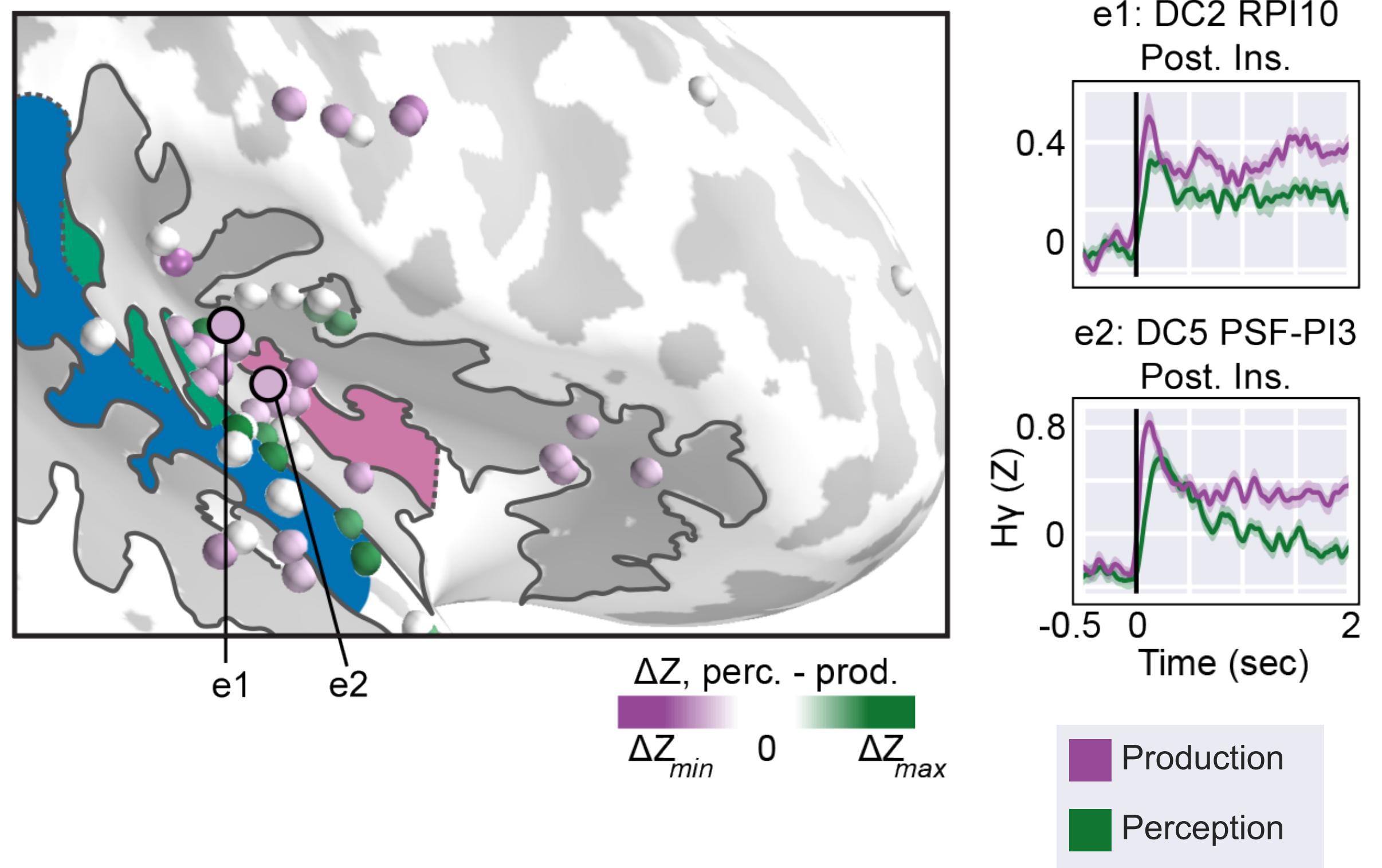
- In primary (HG, PT) and non-primary (STG, STS) auditory cortex, **onset responses** are only present during passive listening
- Sustained responses remain unsuppressed





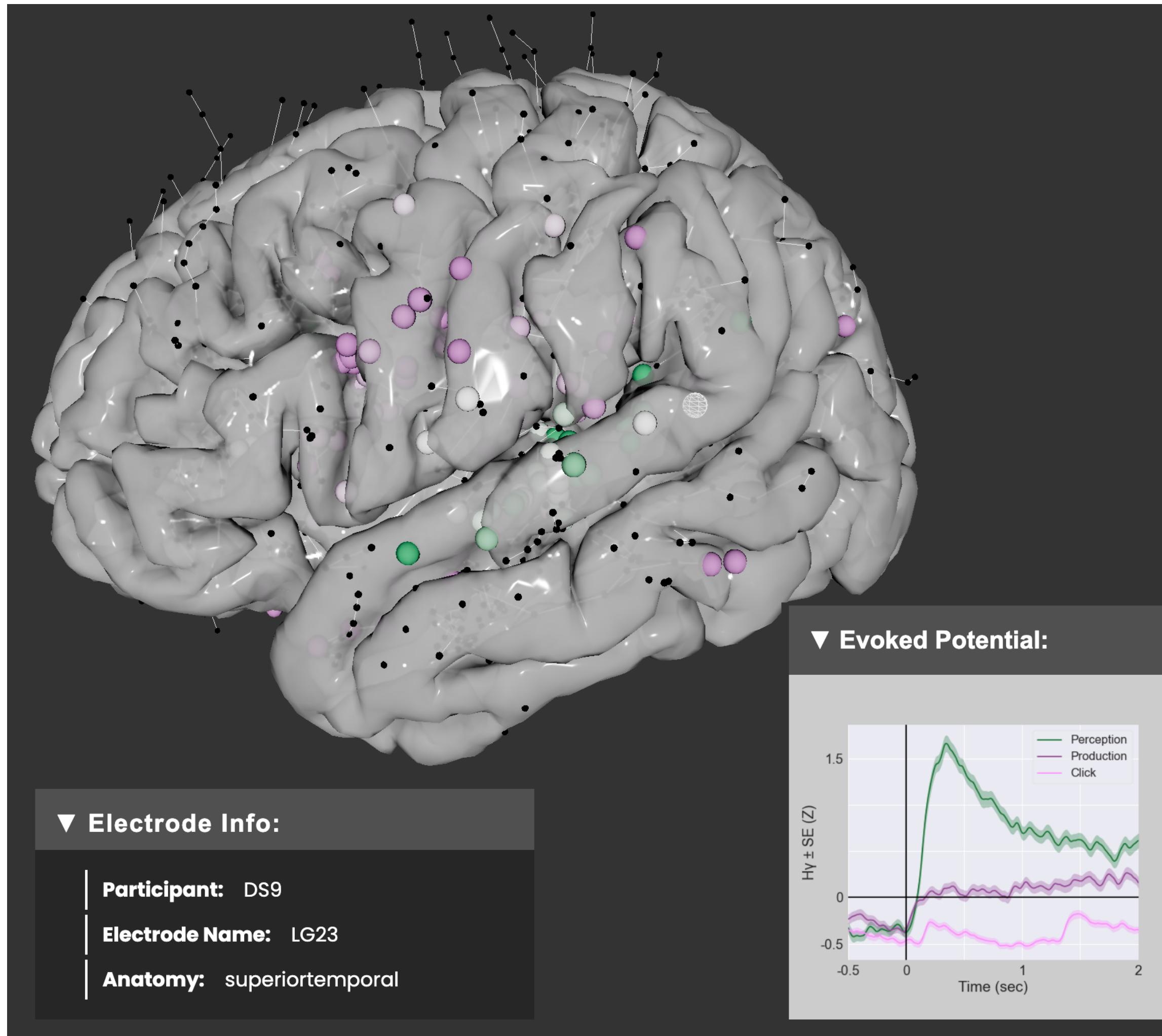
First report of a human insular auditory field

- In the posterior insula, onset responses remain **unsuppressed**
 - Posterior insula electrodes show auditory onset responses during **speaking** and **listening**
- Posterior insula and auditory cortex are **functionally distinct**
 - Auditory cortex: **onset suppression**
 - Posterior insula: **dual onset**
 - **Parallel processing:** PI auditory responses happen just as fast as, or even faster than, AC auditory responses





Explore the results yourself!



<https://hamiltonlabut.github.io/kurteff2024/>



SIS as a biomarker of feedback control

These studies replicated a **laboratory speech** phenomenon (speaker-induced suppression) in a **naturalistic context**

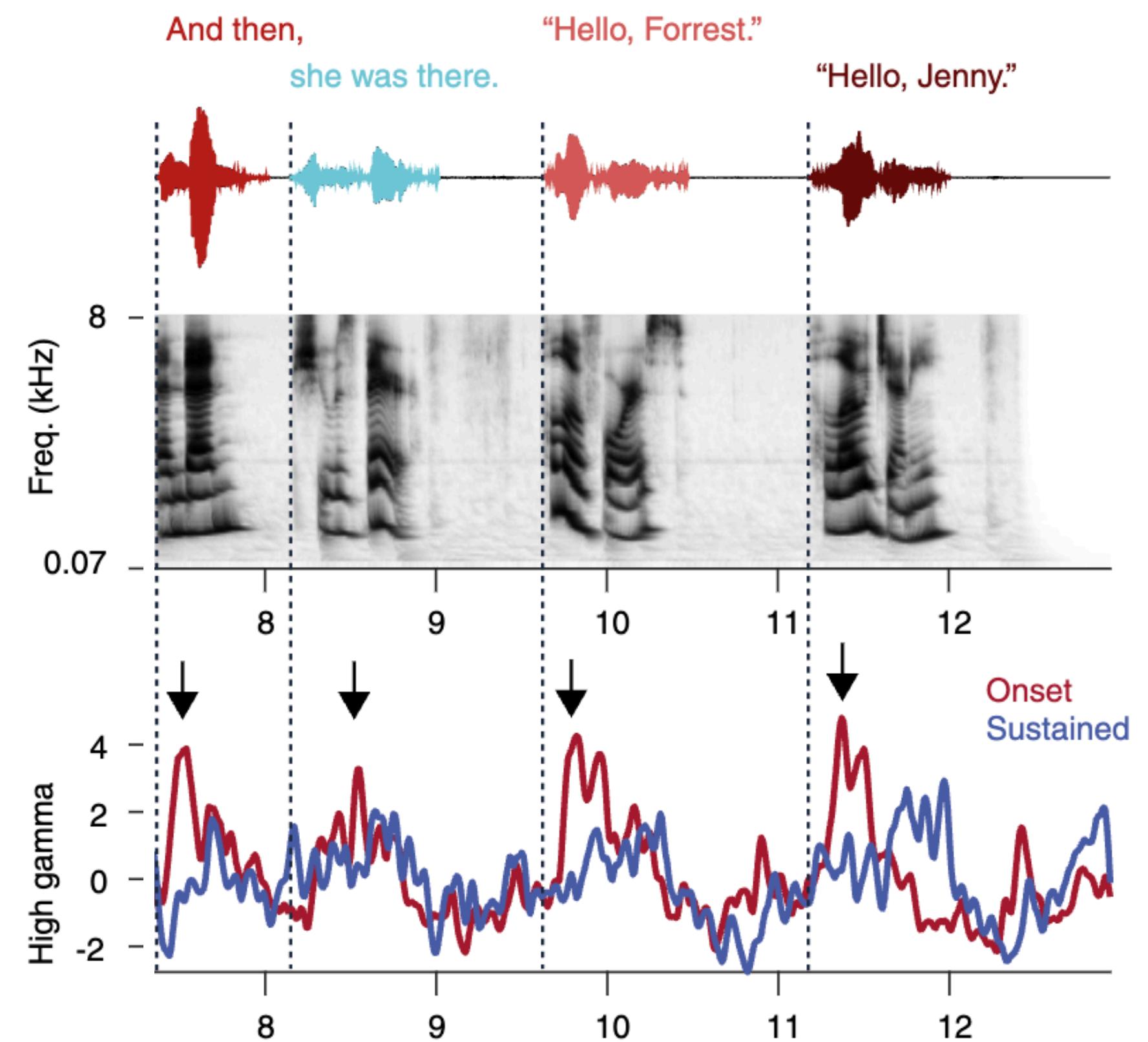
- Naturalistic stimuli facilitate **translational clinical research**
- SIS could be observed with portable EEG in the clinic to evaluate health of feedback control system (future direction!)
- EEG studies of naturalistic speech production are *extremely limited*





Onset responses in neural AAC

- The absence of **onset responses** in speech production trials supports a role in auditory landmark detection
- Decoding (the absence of) onset responses = decoding speech initiation attempts? (future direction!)
 - If yes: AAC/BCI applications





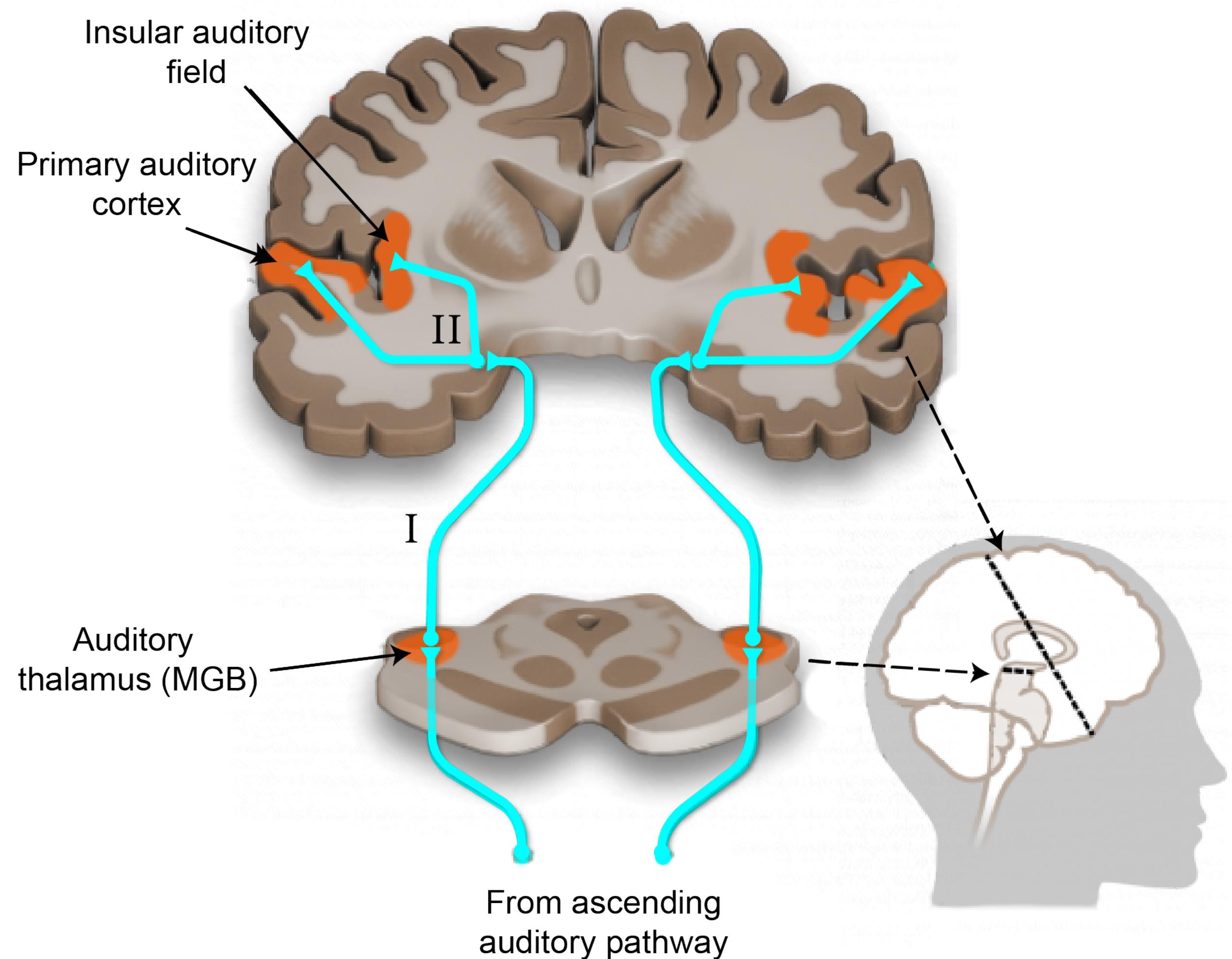
Evidence of a human insular auditory field

Insular auditory field is likely **parallel** to primary auditory cortex

- Parallel information is coming straight from auditory thalamus, not from primary auditory cortex
- A lack of onset suppression suggests a different role than primary auditory cortex, possibly multisensory integration [26]

Future direction: Investigate insular responses during speech errors

Theory 2: Parallel auditory processing





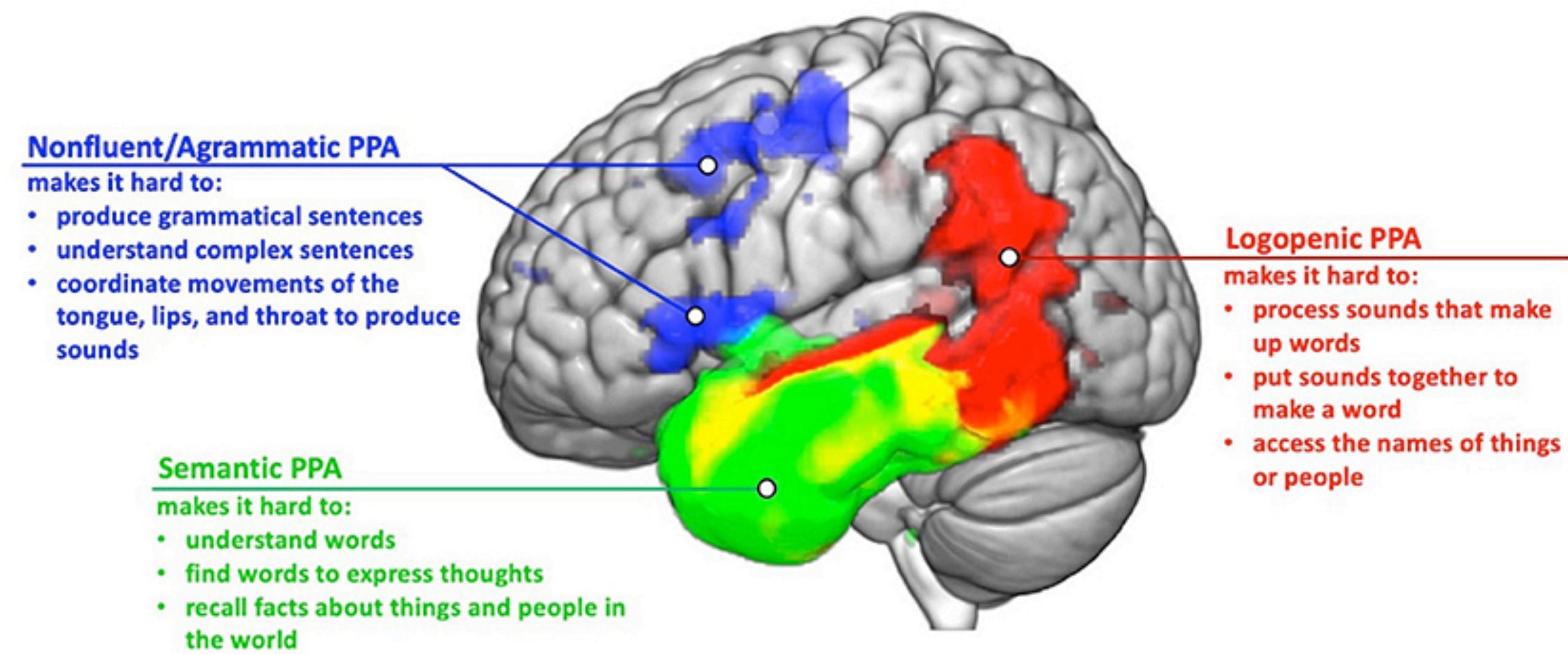
Future directions

AKA, what I'm doing now and what I plan to do at CSUEB!



Apraxia of speech and neurodegeneration

- Neurodegeneration does not have some of stroke data's confounds (and vice versa), so they complement each other
- Neurogenic communication disorders are “stroke-centric”:
 - Boston classification system (Broca’s aphasia, Wernicke’s aphasia, etc.) is not applicable to PPA
 - Could DDx of dysarthria/aphasia also be “stroke-centric?”
 - Potential solution: feature-first classification of communication disorders



UCSF



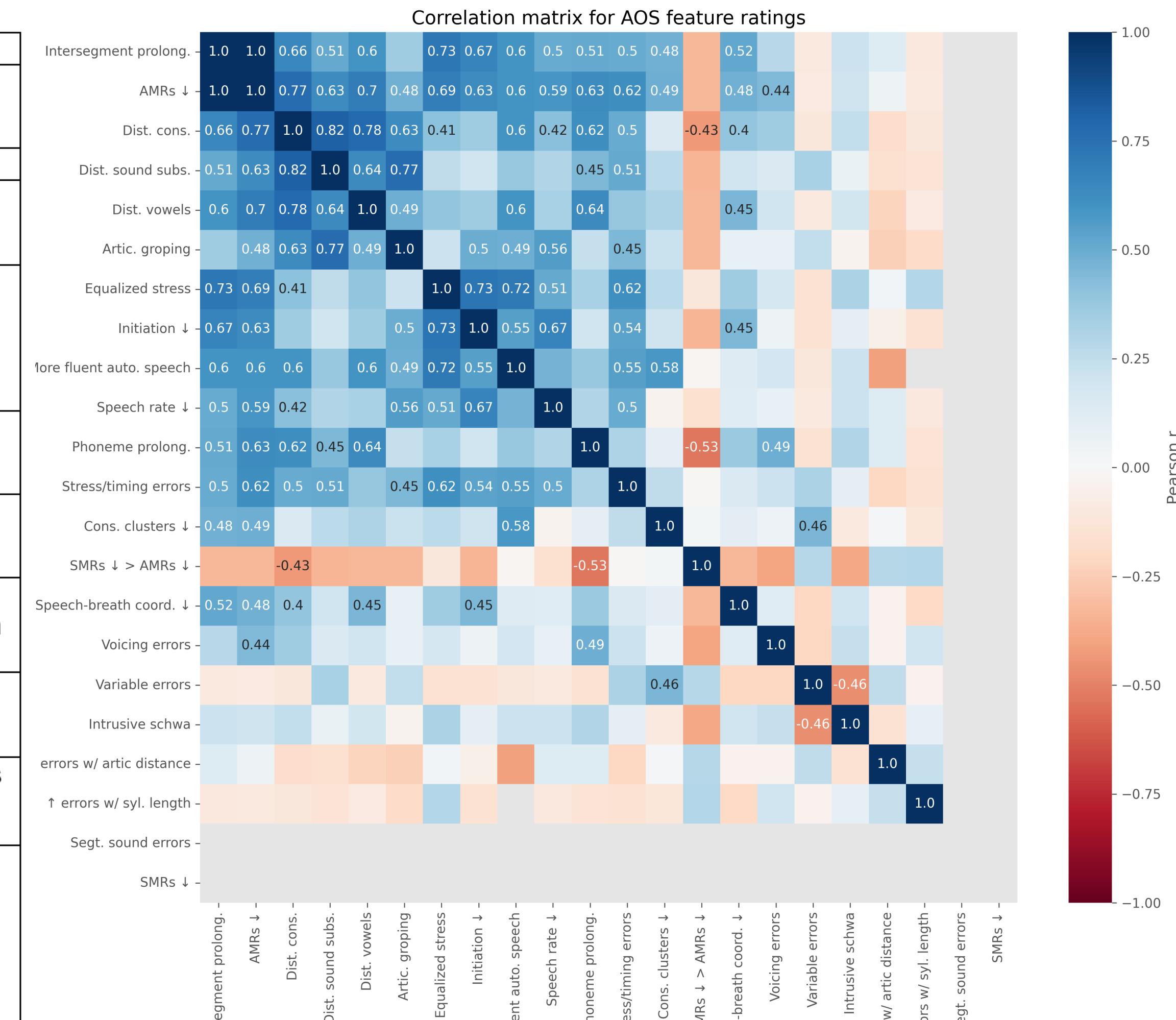


Feature-first apraxia of speech

Apraxia of speech – granular features

Scored as present/absent

#1 – Slowed rate of speech	#12 – SMRs more impacted than AMRs
#2 – Segmental sound errors <i>(i.e., substitutions, deletions, additions, transpositions, etc)</i>	#13 – Impaired initiation <i>(i.e., pauses at the beginning of words, phrases, sentences; visible difficulty with initiating speech)</i>
#3 – Distorted vowels	#14 – Visible and/or audible articulatory groping
#4 – Distorted consonants	#15 – Variable sound errors <i>(e.g., sounds in polysyllabic words vary with multiple attempts during MSE polysyllabic word repetition task)</i>
#5 - Distorted sound substitutions	#16 – Increase in segmental sound errors, distortions, or prolonged intersegmental durations as syllabic length increases <i>(e.g., performance on words of increasing length task during MSE)</i>
#6 – Intrusive schwa <i>(i.e., adding a schwa between syllables or within consonant clusters)</i>	#17 – Errors in timing and/or stress <i>(i.e., prosody)</i>
#7 – Voicing errors <i>(i.e., producing voiced consonants for voiceless consonants, vice-versa)</i>	#18 – Inappropriate equalized stress <i>(i.e., prosody)</i>
#8 – Prolongation of phonemes <i>(i.e., vowels and/or consonants)</i>	#19 – Improved fluency and/or fewer segmental sound errors with automatic/overlearned speech than with generative speech
#9 – Prolonged intersegmental durations <i>(i.e., between syllables, between sounds in consonant clusters, between words)</i>	#20 – Poor speech-breath coordination <i>(i.e., reduced words per speech breath group relative to maximum vowel duration)</i>
#10 – Deliberate, slow, segmented, and/or inaccurate speech AMRs <i>(i.e., alternating motion rates: e.g., /papapav/)</i>	#21 – Impaired production of consonant clusters <i>(e.g., "clean," "grow")</i>
#11 – Deliberate, slow, segmented, and/or inaccurate speech SMRs <i>(i.e., sequential motion rates: e.g., /patakan/)</i>	#22 – Increase in segmental sound errors, distortions, or prolonged intersegmental durations for words containing adjacent phonemes that require greater articulatory distance <i>(e.g., "poke," "cup")</i>



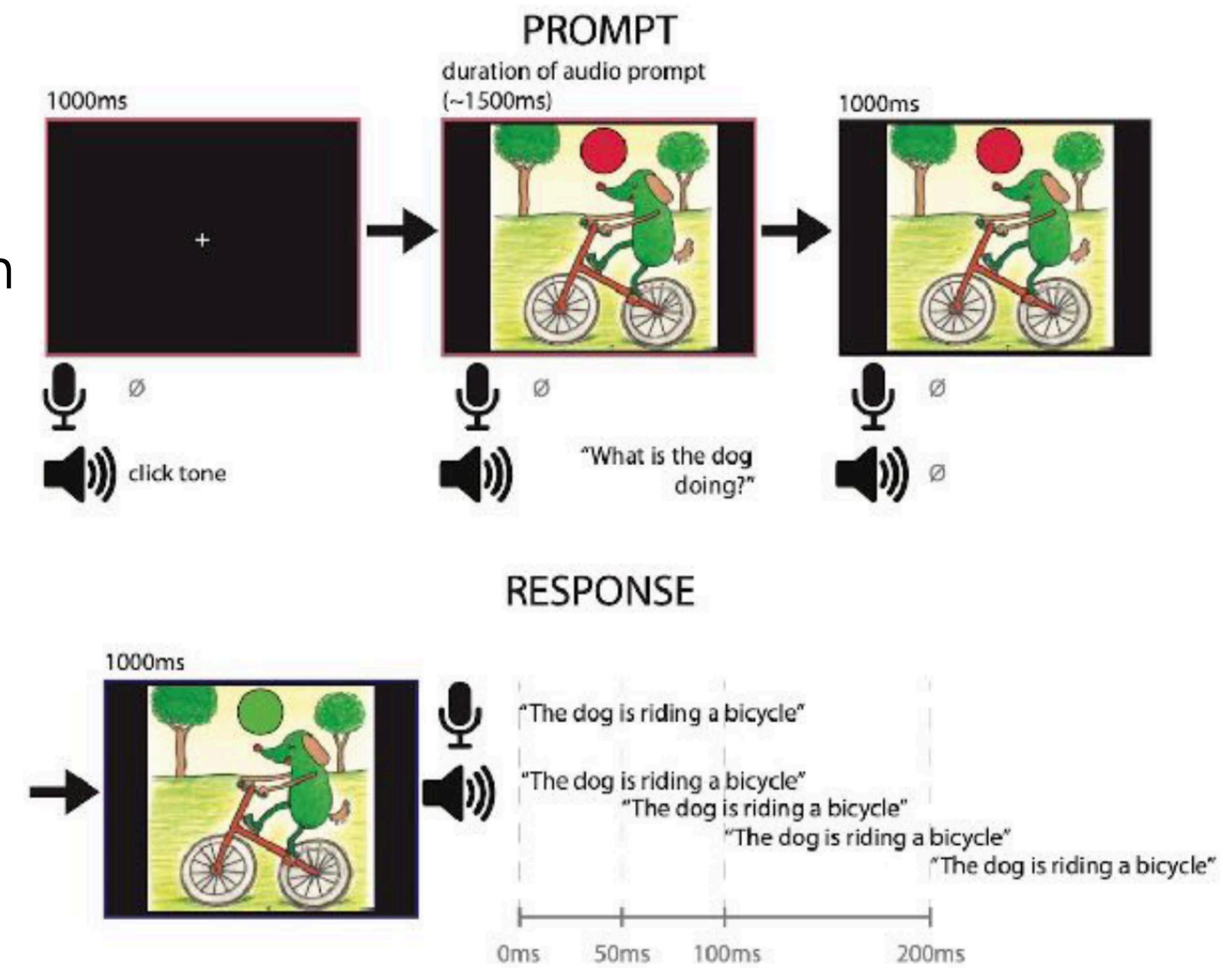
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Feedback processing as a biomarker of AOS

- Collected pilot error elicitation task data with sEEG (delayed auditory feedback)
- **Future direction:** continue SIS work in participants with AOS
 - Issue: Recruitment for experimental studies of AOS is challenging
 - Solution: Recruit from local cohorts (PPA-MEG @ UCSF; **Rees clinic @ CSUEB?**)



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Five year plan

A rough map of what I hope my early years as CSUEB faculty involve...

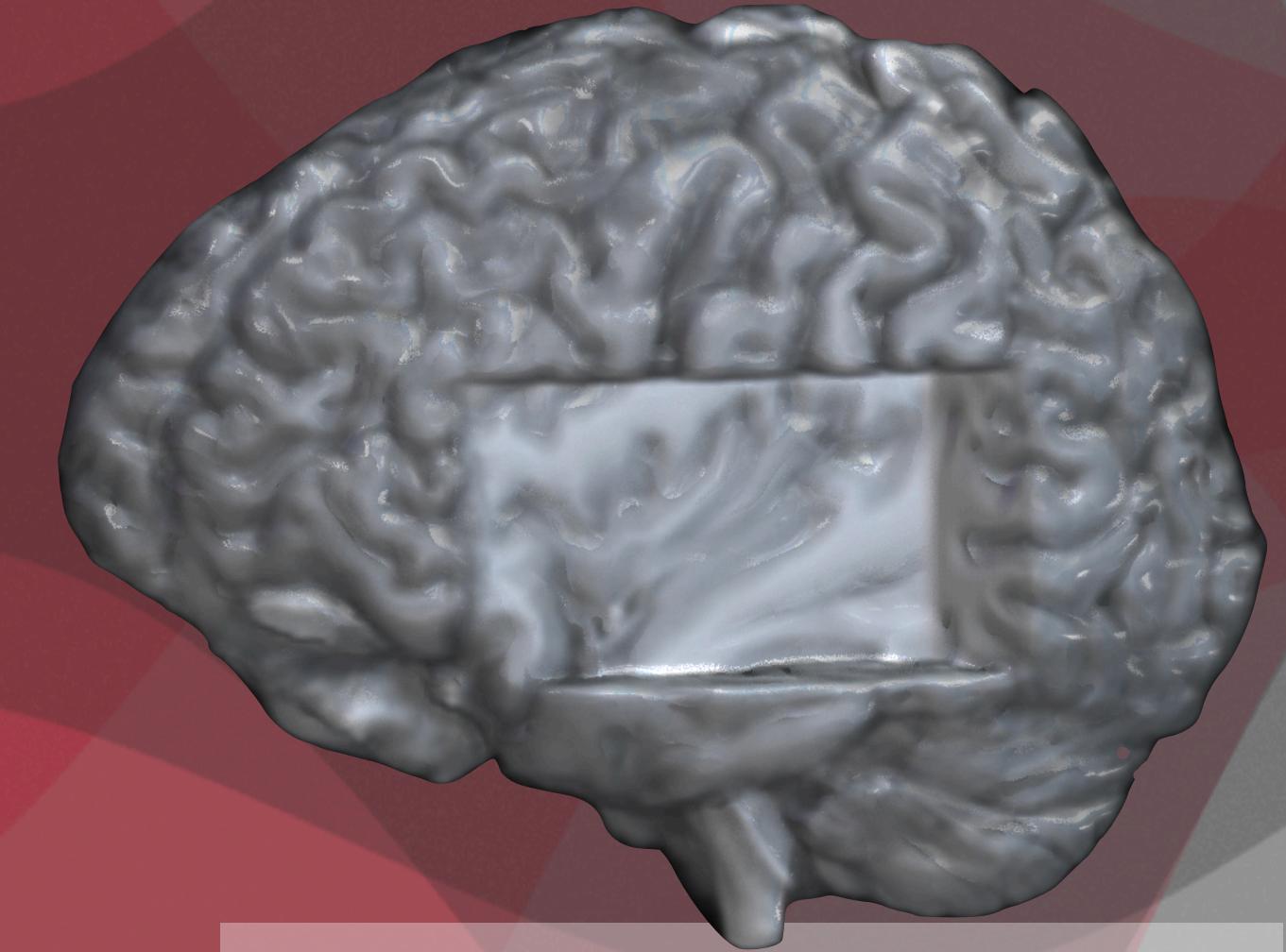
- Continue to **descriptively characterize motor speech disorders** to help disambiguate AOS from dysarthria and aphasia & **develop tools to help clinicians**
- Further **refine lesion-symptom mapping of AOS** in both stroke and neurodegenerative cohorts
- Begin **experimental neuroimaging studies in people with apraxia of speech**, leveraging local connections
 - Stroke-based AOS: recruit from CSUEB SLH clinic
 - Progressive AOS: recruit from UCSF Memory & Aging Center
 - Healthy controls: Start EEG/OPM lab for noninvasive recordings, collect invasive recordings with Hamilton Lab & Chang Lab
- Develop **theoretical model for role of the insula in speech motor control**

Thank you!

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(scan for references a/o slides)



My insula
(what are you up to, buddy?)