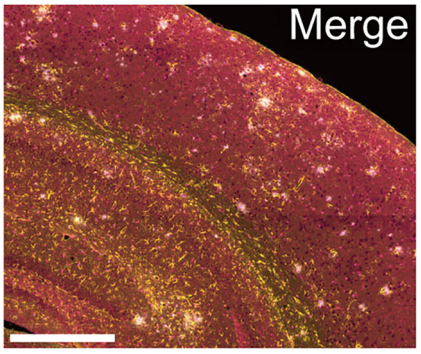
This Week in The Journal

Exploring How the Brain Perceives and Produces Speech

Garret Lynn Kurteff, Alyssa M. Field, Saman Asghar, Elizabeth C. Tyler-Kabara, Dave Clarke et al.

(see article e1109242024)

As we verbalize words, our auditory systems rapidly process our voices to detect and correct speech errors in real time. This voice processing requires perceiving auditory boundaries in continuous speech. Distinct neural populations in the auditory cortex respond to the onset of speech sounds to support boundary perception during speaking. However, the direct role of these speech onset-responding neurons in producing speech was not assessed until now. Kurteff et al. recorded neural responses in the auditory cortex of 17 study participants using intracranial EEG during a speaking and listening task. By comparing attributes of speech-driven neural activity, including temporal dynamics and speech sound encoding, the authors discovered fast responses to speech onset in the auditory cortex that were quickly suppressed during speaking. Kurteff et al. also observed sustained responses in the auditory cortex that may contribute to speech sound encoding and tuning. The authors suggest that onset responses are indicators for speech boundaries that may be quickly suppressed because they are redundant with forward prediction processes during speech. In their investigation, the authors discovered the posterior insula also responds to speech onset and contributes to speech sound tuning during speaking, showing a new role in speech perception and production for this region. Their work advances our understanding of how different brain regions contribute to feedback control during speech perception and production.



Representative image of the inflammatory response in mice with the Icelandic mutation. Displayed is the expression of amyloid-beta (gray), ionized calcium-binding adaptor molecule 1 (magenta), and glial fibrillary acidic protein (yellow-green). See Shimohama et al. for more information.

The Icelandic Mutation Protects against Alzheimer's Pathology In Vivo

Sho Shimohama, Ryo Fujioka, Naomi Mihira, Misaki Sekiguchi, Luca Sartori et al.

(see article e0223242024)

A poor understanding of how to target the mechanisms that drive Alzheimer's disease (AD) pathology hinders treatment development. AD is characterized, in part, by the buildup of amyloid plaques. Expression of the amyloid-beta precursor protein (APP) gene facilitates amyloid pathology and worsens plaque buildup. A mutation of the APP gene—referred to as the Icelandic mutation—protects against amyloid pathology ex vivo, but this has not been assessed in vivo. Shimohama and colleagues explored this using a genetic mouse model that mimics the amyloid pathology observed in humans. After inducing the Icelandic mutation on the APP gene in these mutant mice, the authors observed a reduction in amyloid plaques and other AD pathology such as neuroinflammation and neuritic alterations. They also uncovered a mechanism for the Icelandic mutation's protective effects involving inhibition of beta-cleavage. These mechanistic insights may inform pharmacological treatment interventions for AD, and, although the researchers did not use in vivo genome editing, this study may bring us closer to validating in vivo genome editing as a treatment intervention.

This Week in The Journal was written by Paige McKeon
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