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Neural encoding of speech is interrupted by premature birth

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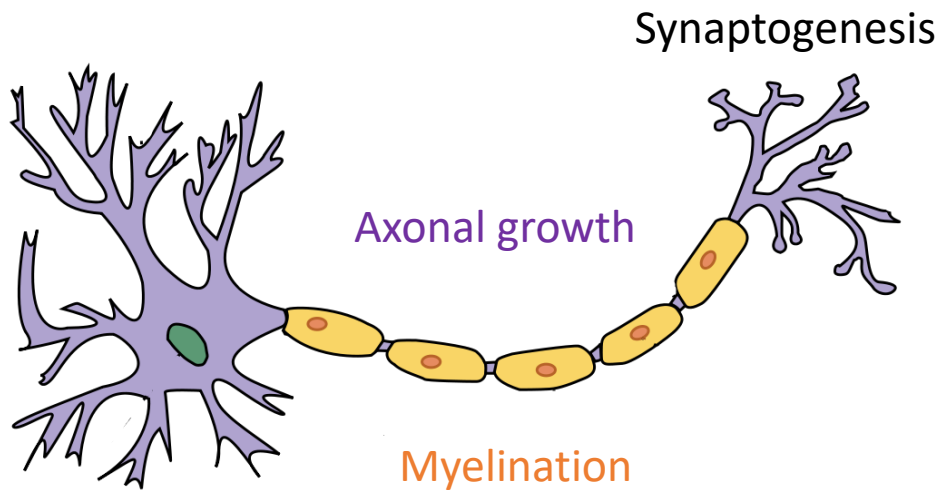
Background: The perinatal period

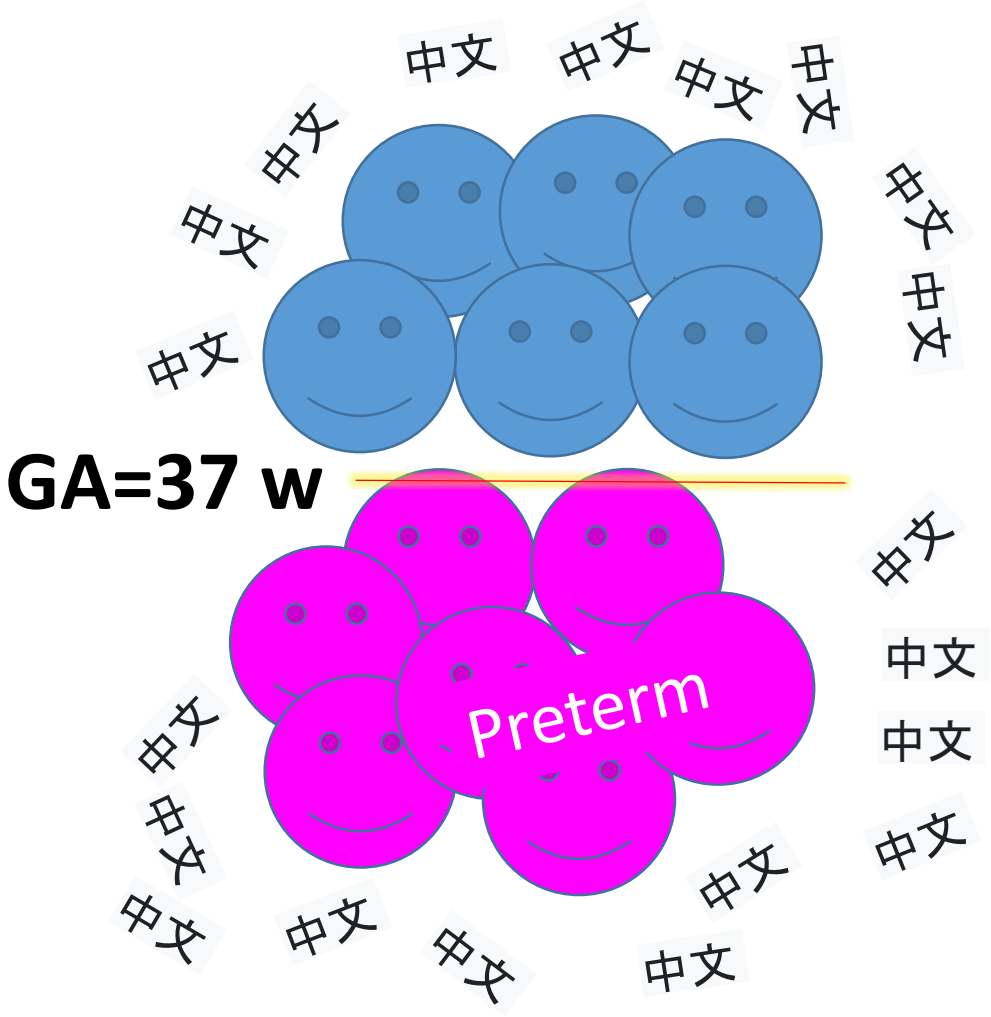


- Extensive growth of axonal connections, synaptogenesis and myelination in the brainstem and cortex.
- Preterm birth leads to smaller brain sizes and slower brain growth.

- Is the preterm birth associated with neural functional deficits?
- Specifically, is there something wrong with the EEG responses to speech sounds, such as
  - frequency-following response (FFR)
  - long-latency responses (LLRs)

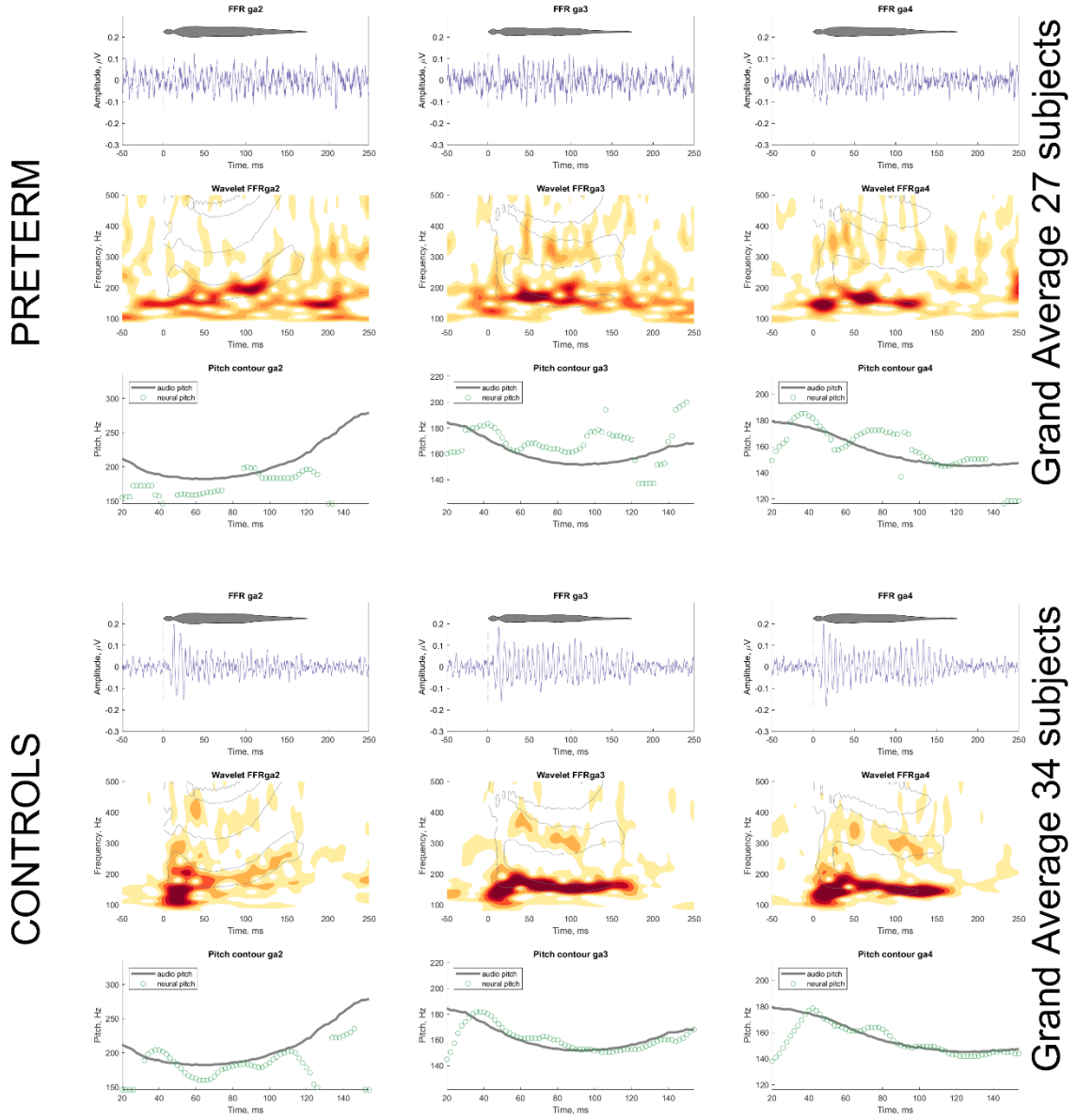
Methods I

- Twenty-seven preterm infants
- Preterm gestational age (GA)<37
- Corrected age (CA) 0-11 months
- Thirty four age- and sex-matched controls
- Equiprobable repetitions of three /ga/ syllables in two native Cantonese and one non-native Mandarin tones



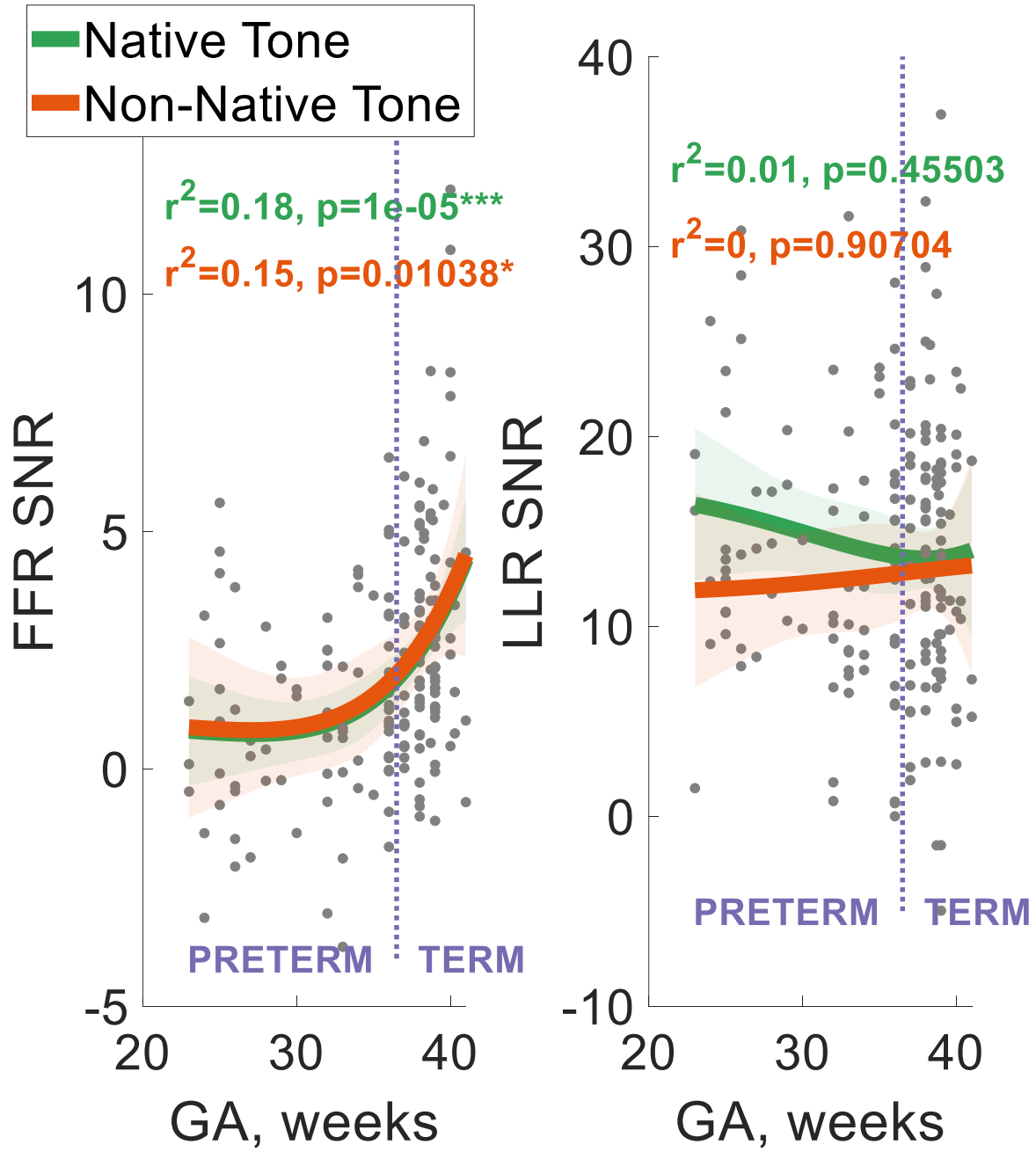
Methods II

- 3-channel EEG, 20 KHz s.rate
- Two parallel pipelines (at Cz):
  - FFR: 80-1500 Hz bandpass,
  - LLR: 0.1-40 Hz bandpass
- CA, GA, nativeness -> factors in a fixed-effect FDR-corrected ANOVAs



Results

- FFR SNR, Pitch Strength, Lower- and Middle-Frequency Spectral Power correlated with CA
- FFR SNR and Pitch Strength also increased with GA.
- No interaction CA-GA
- No effect of either age or prematurity on LLR was revealed.
- No effect of tone nativeness



Conclusions

- We conclude that neural processes that are principally driven by myelination (FFR SNR and Pitch Strength) are more sensitivite to prematurity than processes driven by axonal proliferation and synaptogensis (EEG power).