

CLASSES 23–24: TOWARDS A GENETICS OF LANGUAGE

FOXP2 & SLI

- language is complicated — it can go wrong in a lot of ways
 - phenotype (and genotype, presumably) varies widely — like autism, not Down's
 - gSLI: problems marking tense, understanding passives, forming questions
 - similar problems w/ morphology seen in aphasics and L2 acquirers
 - atypical lateralization of phonological processing
- different hypotheses about SLI
 - auditory/phonological memory or processing speed
 - but auditory deficits are NOT correlated with any form of SLI, are not heritable
 - seems more related to dyslexia, not problems with morphosyntax problems
 - oro-facial dyspraxia (seems to be true of KE family)
- KE family: 15 individuals over 3 generations
 - problems with phi-marking
 - moderate nonverbal IQ reduction in affected individuals
- FOXP2: forkhead box gene on the long arm of chromosome 7 (7q31)
 - KE family has a missense point mutation
 - Fox genes seem to be related to body plan complexity
 - 4 in yeast, 15 in nematode, 20 in flies, 40+ in humans
 - down-regulates production of certain proteins expressed in various tissues
 - dozens of candidate genes associated with autism, Alzheimer's, schizophrenia
 - polymorphisms in a gene down-regulated by FOXP2 correlate with problems on non-word repetition (= phonological memory?); also associated with language delay in autism
 - Foxp2 highly conserved across species
 - 7 between finch & us, 3 between mice & us, 2 of those in the past 200K years
 - Neanderthals had same as us
 - suggests strong selectional pressure (for what?) recently
 - especially active in male birds during mating season; during song learning
 - mice: ultrasonic vocalizations disrupted/absent; dev. delay, motor ctrl. problems
 - knockout mice die in around 21 days because of lung underdevelopment
 - SLI mouse models show gender differences
 - question for the future:
 - How do we test for SLI in monkeys and other “abnormally developing children”?