Finches with faulty talk gene fluff songs

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THE "language gene" is fundamentally important for many animals, not just humans. Like us, birds have problems communicating when the gene, *FoxP2*, isn't working as it should, showing that it is essential for normal everyday activity.

FOXP2 was discovered in the "KE" family, about half of whom are affected across three generations. They have great difficulty speaking fluently and pronouncing words. Similarly, when *FoxP2* is turned off in young zebra finches they find it difficult to learn and recite songs learned from an older tutor bird.

The *FoxP2* gene codes for a protein made of 715 amino acids. The gene is similar in all vertebrates – there are only two amino acid differences between chimps and humans. And the zebra finch and human versions of the gene differ by only seven amino acids, but are identical in the functional region.

To mimic the KE family mutation in zebra finches, the researchers injected the birds' brains with a virus carrying a snippet of RNA designed to selectively block functioning of the gene in "area X", the region required for song learning and recitation.

In this way, they stopped the gene working in young males just at the time when they would normally learn songs from an elder male. "That's the cool part of our study," says Constance Scharff of the Free University of Berlin, head of the team that carried out the experiments. "We knocked out *FoxP2* at the time the song learning happens, when the brain is already pretty much wired, at least at the gross anatomical scale."

As a result the affected birds failed to learn and recite songs properly. "You can definitely hear the difference," says Scharff, whose team analysed the song in great detail with software. "Instead of accurately copying the tutor song, *FoxP2*-knockdown pupils copied fewer syllables, and even those they did copy were less accurately imitated than they should be," she says.

The team found that the misrecited songs had features that matched speech deficits in members of the KE family, who mispronounce words, each time a little differently, as if the brain continues to search in vain for a perfect match to a sound it has heard but can't reproduce (*PLoS Biology*, DOI: 10.1371/journal.pbio.0050321).

"Beforehand, we knew from KE patients that *FOXP2* must be important for speech articulation and, less so, for language comprehension," says Scharff. "This could have been caused through mis-wiring during embryonic brain development, but now we know *FoxP2* is causally related to the function of brain circuits necessary for vocal learning," she says.

"The 'language' gene appears vital for everyday activities, not just for wiring up the brain during development"

Scharff now wants to see if the affected males were worse at attracting females.

Simon Fisher of the Wellcome Trust Centre for Human Genetics in Oxford, and co-discoverer of *FOXP2* in 2002, said Scharff's key finding is that the gene appears vital for everyday brain function, rather than for wiring up the embryonic brain as had previously been thought.