Songbird genome provides clues about speech

For the first time, researchers have sequenced the genome of a songbird, reports an international consortium of researchers (*Nature* **464**, 757–762; 2010). This songbird, the zebra finch, is already an established animal model in several biological fields. Other than songbirds, only a few animals, such as whales and humans, can learn vocalizations. Researchers can now study this songbird's genome to better understand the development of learned vocalizations in songbirds and other animals.

Wesley Warren of the University of Washington in St. Louis, MO, and colleagues sequenced the genome of a male zebra finch (*Taeniopygia guttata*) and then compared this genome with that of a chicken (*Gallus gallus*), the only other bird whose genome has been sequenced to date. Chickens, whose lineage diverged from that of the zebra finch about 100 million years ago, cannot learn vocalizations. The researchers found that the genomes of the zebra finch and the chicken were generally similar in structure. However, these birds regulate their sex chromosomes

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in different ways. As only male zebra finches learn songs, this difference in sex chromosome regulation might be related to the zebra finch's singing behavior.

Further analysis of the zebra finch's genome showed that singing significantly altered the expression of about 800 genes in the zebra finch, causing these genes to be expressed at either higher or lower levels. Another recent research study that analyzed gene expression in the auditory forebrain of the zebra finch had shown that when a bird listened to a song, distinct sets of RNA were expressed in different ways. This RNA expression is involved in the bird's ability to learn. Warren and his team mapped these RNAs onto the zebra finch genome and found that many of these RNAs are non-coding. This suggests that some of these "song-responding" RNAs are modulating the expression of other genes instead of directly producing proteins. Based on phylogenomic comparisons between the genes of the chicken, the zebra finch and several different mammals, the team found evidence that some of the zebra finch genes that are regulated by singing or listening to song are rapidly evolving.

The authors explain that their results suggest that non-coding RNAs could be key links that help the zebra finch learn vocal communication. As the process by which male birds learn to sing is similar to how humans learn to speak, further study of the zebra finch genome could help elucidate the molecular mechanisms by which people learn to speak.

Kirsten Dorans

IS JUNK FOOD ADDICTIVE?

New work from Paul Johnson and Paul Kenny (Scripps Research Institute, Jupiter, FL) has elucidated the neurobiology underlying compulsive eating. Their results suggest that rats with easy access to high-fat diets may become addicted to the 'junk' food.

Johnson and Kenny used three groups of rats in their studies, all of which had unlimited access to standard rat chow. In addition to rat chow, one group also had limited access (1 hour per day) to junk food, and another group had almost unlimited access (18–23 hours per day) to junk food. The junk food included high-fat, high-calorie items such as bacon, sausage, cheesecake, pound cake, frosting and chocolate. After 40 days, the high-fat diet was removed and all the groups were given only standard rat chow. The researchers monitored the rats' body weight and caloric intake throughout the experiment, as well as the responsiveness of the rats' brain reward systems.

Rats given the most access to junk food consumed almost twice as many calories as control rats and quickly became obese (*Nat. Neurosci.* published online 28 March 2010; doi:10.1038/nn.2519). Overconsumption of the high-fat foods triggered a gradual increase in reward thresholds, such that the rats' brains required more of the foods in order to feel satisfied, indicating that the reward system had become unbalanced and unresponsive. The rats also developed compulsive, binge-like eating behavior, possibly as a result of the unresponsiveness of their brain reward systems. These rats would continue bingeing on the high-fat diet even when they knew they would receive a shock if they did; control rats stopped eating when they knew a shock would be coming.

The brain circuit changes in these rats were similar to those observed in rats allowed to self-administer cocaine or heroin, suggesting that overconsumption of junk food and drug addiction may share a similar neurological mechanism. To investigate this mechanism further, the researchers focused on dopamine D2 receptors, which are known to be involved in vulnerability to obesity and addiction. Dopamine is a neurotransmitter released by pleasurable experiences. They found that expression of D2 receptors was decreased in brains of obese rats. Additionally, when they used a lentivirus to reduce expression of D2 receptors in rats with extended access to the high-fat diet, development of compulsive eating and brain circuit changes was much faster.

Although these results can't be applied directly to humans, they suggest that common mechanisms may be involved in both obesity and addiction.

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