
By: Fowler, Kat

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Fisher was born on 19 August 1970 in London, England, to Susan Fisher and Colin Fisher. In 1988 he began his undergraduate studies in natural sciences at Trinity Hall College at Cambridge University in Cambridge, England. During his time at Trinity Hall, Fisher participated in undergraduate research related to genetics and techniques used to describe the parts of DNA sequences.

After graduating in 1991, Fisher began his graduate studies under the direction of geneticist Ian Craig at the University of Oxford [6] in Oxford, England. In Craig's lab, Fisher studied how developmental disorders were determined by the location of genes [4] on chromosomes. Fisher used a technique called linkage scanning, which uses the location of a gene to estimate the unknown location of the gene of interest. In 1995, Fisher published his doctorate research on the location of a gene, CLCN5, that correlated with Dent's disease, a kidney disorder that first appears in early childhood and can result in end-stage renal disease by adulthood. In a 1996 paper, Fisher and colleagues described the specific mutations in the CLCN5 gene that caused Dent's disease and two other kinds of renal tubular disease. That discovery led to earlier diagnosis and treatment of the diseases.

While working in Craig's lab, Fisher also studied the connection between genetics and language. Through The Language Instinct, by linguist Steven Pinker at the Massachusetts Institute of Technology in Cambridge, Massachusetts, Fisher learned about a family in London that shared a language disorder across multiple generations. Called the KE family, affected individuals had difficulty processing grammar and coordinating the complex facial movements required to speak clearly.

In 1996, when Fisher began a postdoctoral position at the Wellcome Trust Center for Human Genetics at the University of Oxford [6], geneticist Anthony Monaco mentioned the KE family as a potential research project for Fisher. Under the direction of Monaco, Fisher used linkage scanning to find a genetic mutation that might be responsible for the family's language disorder. However, when the paper was published in 1998, Fisher did not know the specific location of the mutation within a region of approximately a hundred genes [4].

According to Fisher, after the 1998 publication, he learned about a five-year old boy with similar speech defects to the KE family. The boy, referred to as CS, had a mutation on the same chromosome as the KE family, enabling Fisher and his colleagues to find the location of a gene, called CAGH44, that could be responsible for the disorders in both the KE family and CS. Fisher and graduate student Cecilia Lai then found the specific mutation in the CAGH44 gene that caused the disorder in the KE family. Instead of linkage scanning, the two used genomic sequencing, a technique that involves describing the sequence of nucleotides that comprise the gene and noting how the sequence changes, or mutates, in family members with the disorder. In the resulting 2001 publication, Fisher and his colleagues renamed the gene FOXP2 and concluded it was involved in the developmental process of speech and language. Additionally, Fisher and his colleagues determined that the FOXP2 gene was a transcription factor, which produces a protein that affects the expression of other genes [6], and thus could affect many areas of development.

In 2002, Fisher completed his postdoctoral work but remained at the Wellcome Trust Center for Human Genetics to lead his own lab. Fisher continued to focus on the FOXP2 gene by researching how the FOXP2 protein regulated and affected other genes [4], in humans [5] and other animal species. Among the genes [4] affected was CNTNAP2, which affects language use and is linked to developmental disorders like autism. Fisher and neuroscientist Daniel Geschwind at the University of California, Los Angeles, in Los Angeles, California, showed that FOXP2 gene mutations, like in the KE family, led the CNTNAP2 gene to produce more proteins than normal. That in turn led to the development of language disorders. In 2009, Fisher and Geschwind published a paper highlighting the CNTNAP2 gene as a target of the FOXP2 protein.

Fisher left Wellcome Trust Center for Human Genetics in 2010 to become director of the Max Planck Institute for Psycholinguistics in Nijmegen, the Netherlands. There, Fisher founded the Language and Genetics department, one of the first to focus on the connections between genetics, brain development, speech, and language. Fisher also continued to collaborate with...
In 2012, in addition to his directorship at the Max Planck Institute, Fisher began teaching language and genetics at the Donders Institute for Brain, Cognition, and Behavior in Radboud University in Nijmegen. Between his duties at the Max Planck Institute and his lecturing responsibilities at the Donders Institute, Fisher also participated in public outreach projects, appearing in several film clips, discussion panels, and media interviews, including the documentary The Human Spark with actor Alan Alda. By 2017, Fisher continued to oversee student research, collaborating with researchers worldwide, and speaking at international conferences. Fisher received numerous awards, including the Royal Society Fellowship and the Eric Kandel Young Neuroscientists Prize. He lives in Nijmegen with his wife, Victoria, and his two children.

Sources

Simon Edward Fisher studied the genes that control speech and language in England and the Netherlands in the late twentieth and early twenty-first centuries. In 2001, Fisher co-discovered the FOXP2 gene with Cecilia Lai, a gene related to language acquisition in humans and vocalization in other mammals. When damaged, the human version of the gene leads to language disorders that disrupt language and speech skills. Fisher's discovery validated the hypothesis that genes influence language, resulting in further investigations of language disorders and their heritability. Fisher's research enabled scientists to better study how genetics play a role in speech, language, and human behavior.