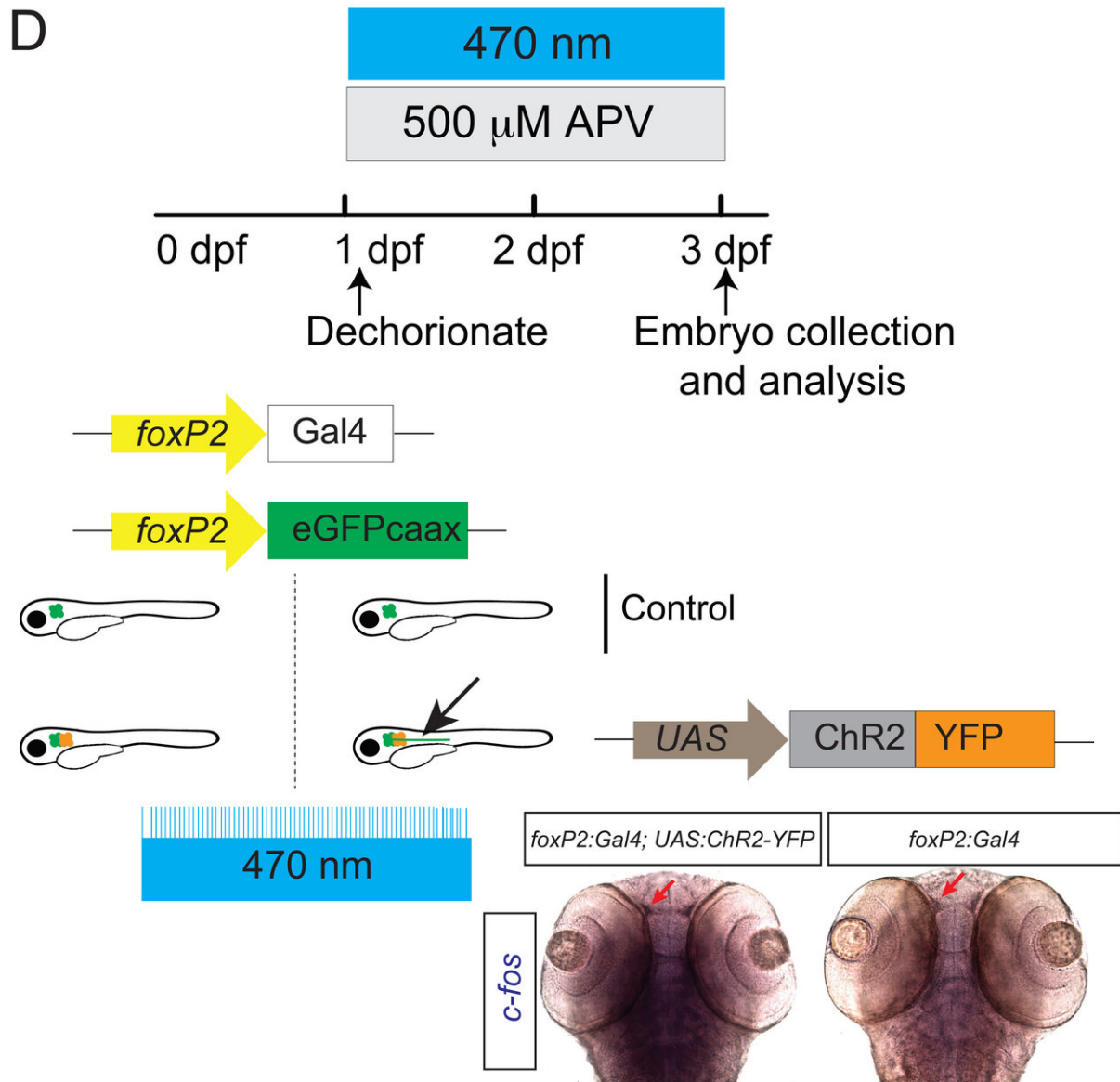


Early environment may shape axon pathfinding

April 16 2018



Schematic workflow of optogenetic analysis. Embryos were dechorionated and exposed to APV and 470 nm light from 1 to 3 dpf. Embryos were double-transgenic for foxP2:egfp-caax; foxP2:Gal4. Some embryos carried UAS:ChR2-YFP (determined by post-hoc genotyping). If neuronal activity could rescue pathfinding, only triple-transgenic embryos would have normal midline pathfinding (arrow). Bottom pictures show in situ c-fos analysis of experimental embryos after 470 nm light exposure. Embryos carrying ChR2-YFP have increased c-fos expression (red arrow in region of foxP2.A.2 neurons). Credit: Gao et al., *eNeuro* (2018)

A new mechanism regulating the early development of connections between the two sides of the nervous system has been identified in a paper published in *eNeuro*. The work demonstrates that neuronal activity is required for this process, a finding that may provide new insight into brain connectivity disorders such as autism.

The paths of axons in the central nervous system are laid down during [embryonic development](#). Most of them will cross the middle of the organism, while some will not. The resulting framework is critical for the connectivity that will later give rise to cognitive functions. Although axon pathfinding is under tight genetic control, the extent to which it can be influenced by environmental factors is unclear.

Addressing this question in transparent zebrafish embryos, Josh Bonkowsky and colleagues found that optogenetic stimulation of an inhibited N-methyl-D-aspartate receptor (NMDAR) was necessary for axons to properly cross the midline. Regulation of neuronal activity by the NMDAR may act through a gene implicated in brain development and neurological diseases.

This research suggests that, in addition to genetic control, [neuronal activity](#) in response to environmental factors—for example, the low

oxygen levels experienced by premature babies—can influence the development of brain connectivity.

More information: The Midline Axon Crossing Decision is Regulated Through An Activity-Dependent Mechanism by the NMDA Receptor, *eNeuro*, [DOI: 10.1523/ENEURO.0389-17.2018](https://doi.org/10.1523/ENEURO.0389-17.2018)

Provided by Society for Neuroscience

Citation: Early environment may shape axon pathfinding (2018, April 16) retrieved 1 May 2024 from <https://medicalxpress.com/news/2018-04-early-environment-axon-pathfinding.html>

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