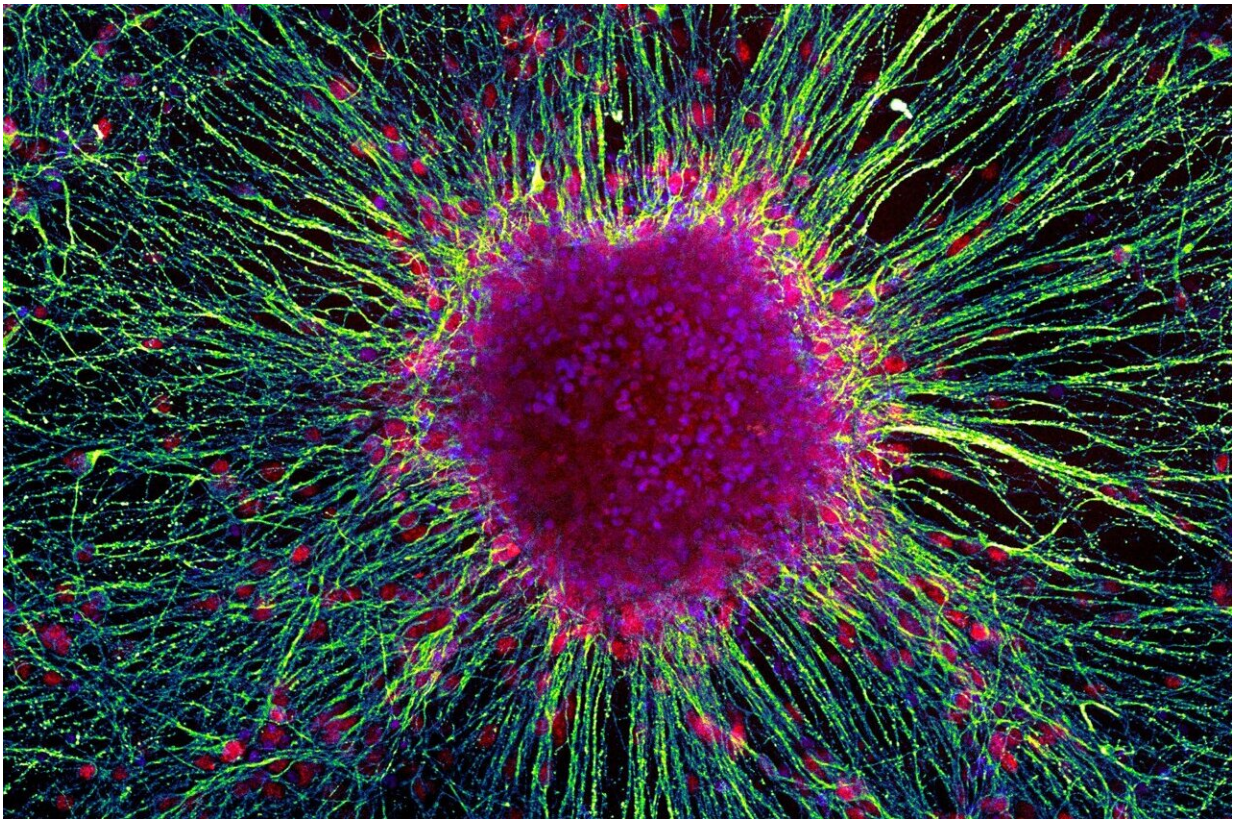


# A dimmer switch for human brain cell growth

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Human neurons derived from urine allow students in the Ernst lab to model neurodevelopmental disease such as FOXP1 syndrome. This image shows a large cluster of neuronal cells that are colored for genes known to be expressed in brain cells. Once created, these neurons can be used to study developmental processes, test drugs, or genetically engineer changes to gene products that may be deficient in diseases such as FOXP1 syndrome. Credit: Nuwan Hettige

Controlling how cells grow is fundamental to ensuring proper brain development and stopping aggressive brain tumors. The network of molecules that control brain cell growth is thought to be complex and vast, but now McGill University researchers provide striking evidence of a single gene that can, by itself, control brain cell growth in humans.

In a paper published recently in *Stem Cell* reports, Carl Ernst, an Associate Professor in the Department of Psychiatry at McGill University and his team have shown that the loss of the FOXP1 gene in [brain cells](#) from patients with severe microcephaly—a disease where the brain does not grow large enough—reduces brain cell growth. Using [genetic engineering](#), they turned on FOXP1 in cells from a microcephaly patient to different levels and showed corresponding increases in brain cell growth. They have uncovered a remarkable dimmer switch to turn brain cell growth up or down.

Their research indicates that a [single gene](#) could potentially be targeted to stop brain tumor cells from growing. Or that future gene therapy might allow this same gene to be turned up in patients with microcephaly or other [neurodevelopmental disorders](#).

"FOXP1 dose tunes cell proliferation dynamics in human forebrain progenitor cells" by Nuwan C. Hettige et al. was published in *Stem Cell Reports*.

**More information:** Nuwan C. Hettige et al, FOXP1 dose tunes cell proliferation dynamics in human forebrain progenitor cells, *Stem Cell Reports* (2022). [DOI: 10.1016/j.stemcr.2022.01.010](https://doi.org/10.1016/j.stemcr.2022.01.010)

Provided by McGill University

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