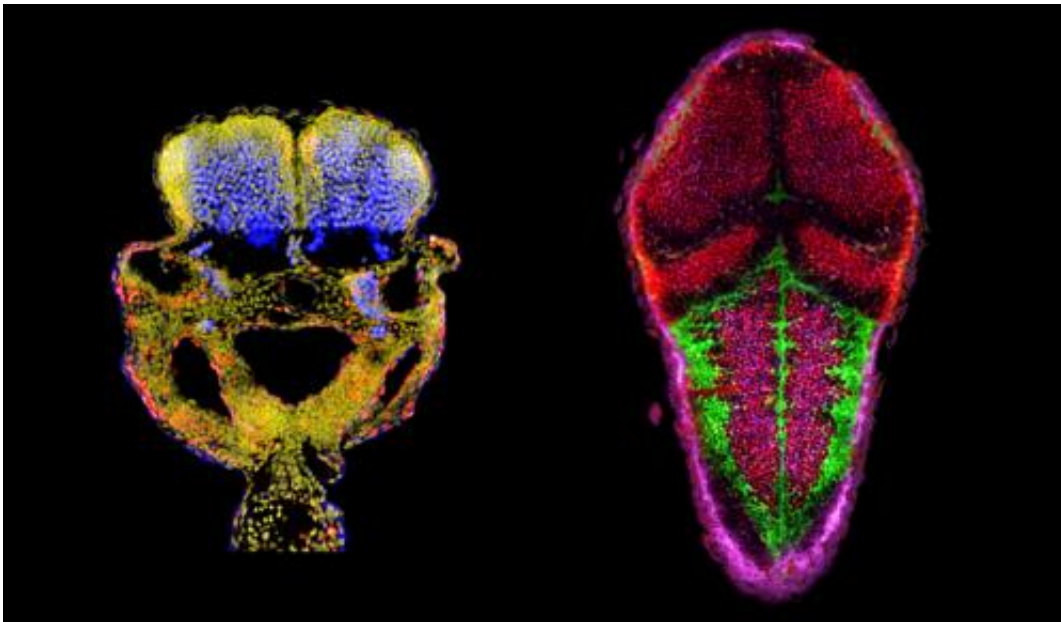


Research team identifies key process in embryonic neurogenesis

February 6 2015, by Ziba Kashef



Left, hindbrain of zebrafish embryo at 48 hours post fertilization show increase of neurogenesis. Right, dorsal view of a Zebrafish embryo treated for miR-107. Credit: Emma Ristori

MicroRNA are the tiny non-coding RNA molecules that help determine whether genes are expressed or silenced. One particular microRNA—miR-107—plays a key role in early brain development, and perhaps in the development of brain-related disease, a Yale School of Medicine team has found.

Experts have long known that miRNAs are involved in brain development but not precisely how. Assistant professor of medicine Stefania Nicoli and her team set out to describe the process by studying miR-107 in zebrafish. They discovered that miR-107 regulates the levels of an enzyme known as dicer, which in turn regulates another RNA molecule, miR-9, that is essential to normal brain development. This regulatory loop was previously unknown.

Though miR-107 has been studied previously in cancer progression and diabetes, the Yale team observed how it impacts brain development in an animal for the first time. The loss of miR-107 in the zebrafish led to an excessive amount of neurons (nerve cells) and neural progenitors.

"This is the first paper showing that if you lose miR-107, you have abnormal embryonic hindbrain development," said Nicoli.

Understanding this miRNA's functions may be essential to understanding the genetic and cellular basis of neural disorders that can occur during [brain development](#), she notes.

The study findings may also point to future research into treatment for adult neurological disorders involving miR-107, the expression of which is dramatically lost in the brain of early Alzheimer's patients, the paper noted.

The study was published early online in the journal *Developmental Cell*.

More information: "A Dicer-miR-107 Interaction Regulates Biogenesis of Specific miRNAs Crucial for Neurogenesis." [DOI: 10.1016/j.devcel.2014.12.013](https://doi.org/10.1016/j.devcel.2014.12.013)

Provided by Yale University

Citation: Research team identifies key process in embryonic neurogenesis (2015, February 6) retrieved 22 May 2024 from <https://medicalxpress.com/news/2015-02-team-key-embryonic-neurogenesis.html>

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