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$1.3M NIH grant funds brain development, cancer research

By Krishna Ramanujan

Researchers will seek to uncover fundamental processes in brain development and their links to brain cancers with a new grant.

Richard Cerione, Cornell professor of molecular medicine and chemistry and chemical biology in the colleges of Veterinary Medicine and Arts and Sciences, has received a four-year, $1.3 million grant from the National Institutes of Health (NIH) to investigate the roles of two closely related signaling proteins in the growth and development of nerve cells (neurogenesis) and brain cancer.

The grant renews NIH funding for a project that began some 25 years ago when Cerione first became a faculty member at Cornell and a graduate student in his lab discovered a gene and the protein it expresses, called Cdc42, while searching for novel participants in growth factor-signaling pathways.

The human Cdc42 protein is virtually the same as its yeast homolog, showing that it has been conserved throughout evolution and is important in fundamental signaling processes related to cell shape and cell movement. After discovering the human Cdc42 protein, Cerione and researchers in his lab spent a number of years investigating how it is regulated and interacts with its signaling partners. In many cases, this involved understanding the three dimensional X-ray crystal structures of different Cdc42 complexes, taking advantage of the Cornell High Energy Synchrotron Source (CHESS).

More recently, a research associate in the Cerione laboratory, Makoto Endo, discovered that a variant of this gene, called Cdc42b, which is only found in the brain, plays a distinct role in neurogenesis. It turns out the more ubiquitous form of the gene and its protein, Cdc42, is essential for stimulating the transition of an embryonic stem cell into a neuroprogenitor cell, the progeny of stem cells that can differentiate into neurons. At this stage, the Cdc42b protein steps in and causes neuroprogenitor cells to become neurons.

“We’ve been trying to understand how the more widely distributed ubiquitous form, Cdc42, causes neuroprogenitors to form,” Cerione said, “and then, what does the brain-specific member, Cdc42b, do to cause them [neuroprogenitors] to become neurons. We try to map those pathways.”

The lab members have also been studying links between brain development, stem cells and the process of developing brain cancers. They found that only the ubiquitous form, Cdc42, is expressed in these brain cancers, and not the brain-specific form, Cdc42b, which supports specialized functions of differentiated cells and does not support growth. The team has been investigating why the Cdc42b protein is not expressed in brain cancers and what role the Cdc42 protein plays in cancer stem cells in the brain.

“If we can understand more about what regulates the lack of expression of Cdc42b in these cancer stem cells and get around that regulation so that it does become expressed, we think then maybe Cdc42b could cause the cancer stem cells to undergo differentiation and thus block tumor progression,” Cerione said.

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