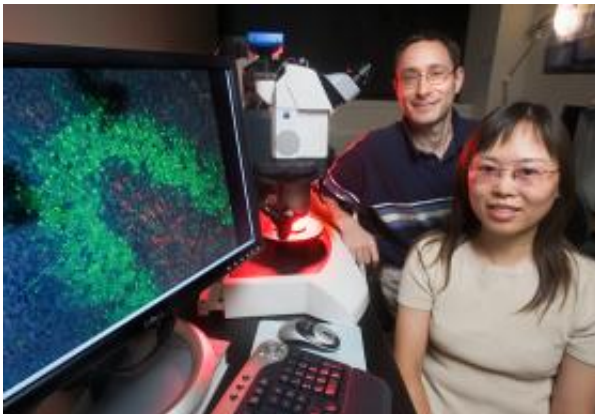


Scientists identify gene involved in stem cell self-renewal in planaria

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New research has identified a protein, produced by a gene found in both planarians and humans, that plays a vital role in maintaining the stem cell population in planarian. Phillip A. Newmark, U. of I. professor of cell and developmental biology, and graduate student Tingxia Guo were the Illinois co-authors of the paper to appear in the August issue of the journal *Developmental Cell*. Photo by L. Brian Stauffer

No matter how you slice it, the freshwater planarian possesses an amazing ability to regenerate lost body parts. Chop one into pieces, and each piece can grow into a complete planarian. The flatworm relies upon a population of stem cells to accomplish this remarkable feat; recent work sheds light on how planarians maintain these stem cells throughout their lives.

In a paper to appear in the August issue of the journal *Developmental Cell*, scientists show that a member of the Bruno-like family of RNA binding proteins – produced by a gene found in both planarians and humans – plays a vital role in maintaining the stem cell population in the planarian *Schmidtea mediterranea*. The work could lead to a better understanding of the fundamental mechanisms by which stem cells are regulated; such basic understanding is required for the successful therapeutic application of stem cells in humans.

“One of the defining characteristics of stem cells is their ability to self-renew – that is, to make more stem cells in addition to differentiating into multiple cell types,” said Phillip A. Newmark, a professor of cell and developmental biology at the University of Illinois at Urbana-Champaign and corresponding author of the paper. “We found that in the absence of this protein, the stem cells could respond to wound stimuli, proliferate, and differentiate, but they were unable to self-renew. As a result, the regeneration process failed and the animals died.”

Using a technique called RNA interference, Illinois graduate student Tingxia Guo and Newmark first eliminated most of the Bruno-like protein (Bruli) from a number of planarians. Then they amputated a small piece from each flatworm.

In the usual manner, the planarian stem cells migrated to the site of the wound, sensed what was missing and began rebuilding. Regeneration ceased, however, when the stem cell population became depleted.

“Had Bruli protein been present, the regeneration process would have continued to completion,” Newmark said. “What may be happening is that when this protein is eliminated, RNAs that are supposed to be turned off (that is, not made into proteins) are now turned on and made into proteins. Those proteins may then cause the stem cells to differentiate, instead of also producing new stem cells to maintain the

population.”

While there is still much to be learned about stem cell self-renewal, the researchers’ results suggest that Bruli protein is required for stem cell maintenance in planarians.

“The next steps are to see if the gene that makes this protein in planarians plays a similar role in stem cells in other organisms and to identify possible RNA targets of this protein,” Newmark said.

Source: University of Illinois at Urbana-Champaign

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