Jon Gilbert Fox

ANNA HATCH

PhD candidate

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WHEN ANNA HATCH ENROLLED AS AN UNDERGRADUATE at the University of Wisconsin-La Crosse, science was pretty far off her radar. She wanted to be a physical therapist, but her plans changed when she started taking science courses. While working in a lab, she "fell in love" with getting results and figuring out what they mean.

She says her enthusiasm for science is largely the product of working with energetic mentors at Wisconsin-La Crosse. After several failed attempts while working on an experiment involving DNA hybrid molecules, she finally got the experiment to work. "I remember going to my advisor's office and he said, 'Oh my gosh! It happened!,'" she says. "He gave me this big high five . . . and ran down the hall with me and said, 'Ok, can you do it again?' . . . . That's one of the big points, that first high five!"

Now a graduate student in the lab of Henry Higgs, a Geisel professor of biochemistry, Hatch still feels that passion for science. She likes the lab because she and Higgs brainstorm a lot. "It becomes more like putting together pieces," she says. "Then the question becomes, 'How do you test this hypothesis?'"

Her current work is on mitochondrial fission—the splitting of mitochondria, which act as energy powerhouses inside the cell. It's a hot research topic, but despite its importance, many details in this process remain a mystery. At first glance, a diagram of mitochondrial fission looks jumbled and chaotic. Yet it is carefully choreographed, as Hatch is discovering.

Hatch likes to compare mitochondria to cars. Both are "machines that need maintenance to run efficiently," she says. "You want to give cars an oil change or tune-up every so many miles. Similarly, cells have these structures that also require maintenance; the mitochondrion is one of them. Fission is just one way for it to check itself." If there is damage, the mitochondrion will move the damaged part to one side, undergo fission, and eliminate the damaged portion. Fission also creates more mitochondria within a cell, giving the cell more sources of energy. And when mitochondria split, they are smaller and can travel more easily around the cell's cytoskeleton to carry out important functions.

During the process of fission, long strands called actin filaments work with a protein called myosin II to start constriction of the mitochondria. Another protein, named Drp1, concentrates at the fission site and helps to further induce fission. Hatch discovered that Drp1 binds directly to actin filaments. She hypothesizes that actin targets Drp1 to the site of fission, so that Drp1 is able to interact with receptors on the mitochondria and help bring about fission through constriction.

Mitochondrial fission is important in understanding neurodegenerative disorders, such as Alzheimer's disease. In a normal cell, fission takes care of removing damaged portions of the mitochondria. "But with patients with Huntington's disease, Parkinson's, and Alzheimer's you see defects in this fission," says Hatch. Through better understanding how fission works, scientists may be able to figure out what happens in the cells of an Alzheimer's patient, for example.

Hatch's research is supported by a prestigious National Science Foundation (NSF) Graduate Research Fellowship, which she was awarded in her first year at Geisel. She says her next step in her research is to figure out how Drp1 is interacting on actin filaments. "A lot of the biochemistry of this isn't hammered down completely," she says. "There's so much that we don't know about basic science that it's cool to figure it out. It's like a giant puzzle."

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