**How would you explain the main findings of your paper in lay terms?**

*Schizosaccharomyces pombe* is a rod-shaped organism that grows through elongation of its cell tips and divides through medial division. *S. pombe* initially exhibits monopolar growth from the old cell tip, which previously belonged to the mother cell. As the cell progresses through the cell cycle, it transitions to a bipolar growth state where the new cell tip, generated from the most recent division, also begins to grow. The transition from monopolar to bipolar growth relies on the successful completion of cell division in the previous cell cycle. Specifically, loss of the cytokinetic protein Fic1 prevents cells from completing this monopolar to bipolar growth transition successfully. In this study we reveal that Fic1 is modified by an unidentified signaling pathway. Disrupting the sites on Fic1 modified by this pathway produces daughter cells that exhibit invasive growth. These Fic1 mutants produce independent cells that grow in chains extending into the agar medium, unlike wild-type cells, which grow on the surface of the agar. Additionally, these Fic1 mutants are deficient in the monopolar to bipolar growth transition compared to wild-type cells. These findings highlight the impact that signaling pathways involved in cell division can exert on cell growth polarity.

**Were there any specific challenges associated with this project? If so, how did you overcome them?**

The most challenging aspect of this project was completing the screen to identify the kinases responsible for phosphorylating Fic1. Initially, we expected Cdk1 or Orb5 to be responsible for Fic1 phosphorylation, as these kinases could phosphorylate Fic1 *in vitro*. However, inhibiting these kinases did not alter the phospho-state of Fic1 *in vivo*. We then screened for deletions of polarity kinases, given the polarity defects observed in the Fic1 phosphomutants; however, none of these kinases altered the phospho-state of Fic1 either. We ultimately screened the effects of 111 protein kinase deletions on Fic1 phosphorylation, only to conclude that no single kinase regulates its phospho-state. During the screening of analog-sensitive kinases, we noted that control cells, with no analog-sensitive mutations, exhibited loss of Fic1 phosphorylation when treated with 50 µM 1-NM-PP1. This finding led us to determine that multiple kinases are involved in regulating the phospho-state of Fic1 and explained why no single kinase deletion led to the loss of Fic1 phosphorylation.

**When doing the research, did you have a particular result or ‘eureka’ moment that has stuck with you?**

During the screen to identify the kinases responsible for Fic1 phosphorylation, I was fortunate to determine that a high dose (50 µM) of 1-NM-PP1 resulted in the loss of phosphorylation at one of the two Fic1 phosphorylation sites. When I was able to repeat this result, I was relieved because I had spent a substantial amount of time trying to find a condition that altered the phospho-state of Fic1 to reveal the signaling pathway upstream of Fic1 and to understand how Fic1 phosphorylation is regulated. Finding even just one condition that altered Fic1 phosphorylation was satisfying and provided much-needed answers. Specifically, this finding provided evidence that at least two groups of kinases were phosphorylating Fic1 and that phosphorylation of either site was not dependent on phosphorylation of the other.

**What motivated you to pursue a career in science, and what have been the most interesting moments on the path that led you to where you are now?**

Curiosity has motivated my pursuit of a career in science. I always want to know more about how our world works and how we can leverage our understanding of these processes for our own benefit. My career in scientific research started by studying host–pathogen interactions...
interactions between Pseudomonas syringae and tomato plants in Dr Melanie Filiatrault’s lab at Cornell University. I spent the summer between my sophomore and junior years of college infecting tomato seedlings with P. syringae to understand how the metabolic processes of P. syringae influence its virulence.

The following summer, I was fortunate enough to have the opportunity to work with deep sea microbes in Dr Terry Hazen’s lab, and identify species capable of digesting hemicellulose to improve the conversion of corn stover to ethanol. Lastly, I had the good fortune of being mentored by Dr Melanie Styers at my alma mater, Birmingham-Southern College, where I used S. pombe to study membrane trafficking. While I am grateful for all of these opportunities, it was my time with Dr Styers that influenced me the most to continue scientific research and pursue a PhD.

What’s next for you?
When I complete my PhD, I plan to pursue a career where I can pair my scientific knowledge with my passion for brewing. Securing a career at a commercial brewery as a microbiologist would be my dream job. Working for a commercial brewery would allow me to implement my research abilities and creative thinking to produce innovative quality products for the general public to enjoy.

Tell us something interesting about yourself that wouldn’t be on your CV
My fascination with yeast extends beyond my work in the lab. On the weekends, I employ Saccharomyces cerevisiae to make beer. I am constantly experimenting with different and new yeast, malt, and hops to create interesting and delicious beer. I enjoy sharing my brews with my friends and family.

Reference