I began my research career in 2007 with a Master’s thesis project at the M.S. University of Baroda, India. Under the supervision of Dr. Archana Gayatri, I investigated the diversity of uncultivable microbes near the Mahi river basin. Through this experience, I learned essential genetic and biochemical assays used to characterize microbes. After graduating in 2009 with an MSc, I was hired by a research-intensive biotech company named Intas Biotherapeutics Ltd, India. As a Research Associate I was involved in developing new analytical and bioanalytical techniques for characterization of biotherapeutic proteins including hematopoietic hormones and monoclonal antibodies. In 2010 I decided to return to academics and joined the Indian institute of Science (IISc) for graduate studies. For about two years I worked on a project that focused on DNA damage response using in vitro approaches. Meanwhile I came to learn about Dr. Ing Swie Goping’s research on breast cancer. I was fascinated by her approach of investigating abnormalities in patient derived tumors and dissecting out cellular pathways in the lab. I applied for a PhD position in her lab and was accepted in September 2012. I left IISc and came to Edmonton in November 2012. Since then I am investigating novel biomarkers of tumor pathogenicity on clinical samples and trying to understand their physiological context at the cellular level using cell biological and biochemical approaches.

In our Oncotarget publication, using gene and tissue microarray analyses, we show that Bcl-2 interacting killer (Bik) is overexpressed in breast cancer tumors and is associated with increased rate of disease recurrence and poor overall survival outcomes of patients. Bik is a pro-apoptotic protein involved in triggering cell death in response to cellular stress when studied under tissue culture (in vitro) conditions. The significance of our publication is that we demonstrated that the in vivo tumor response to Bik is much more complex. Our studies point to the possibility that within the tumor environment, Bik plays a cell survival role—a function completely opposite to what it is known for. Further, in cell culture studies shown by others, the apoptotic activity of Bik is kept in check by anti-apoptotic Bcl-2 family proteins. Interestingly, using breast cancer patient samples, our study makes the novel observation that the pro-survival function of Bik is independent of Bcl-2 mediated blockade of apoptosis. Additionally, we show that overexpression of Bik is also closely associated with upregulation of ATG5, a protein crucial for initiation of autophagy, which is a cellular pathway responsible for survival during cellular stress. We identified that combined high expression level of ATG5 and Bik was a stronger predictor of outcome than either alone. Thus, our study identifies Bik as a novel, independent prognostic biomarker for poor outcomes in breast cancer and suggests that Bik-mediated autophagy contributes to disease recurrence.

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