



sparked during my undergraduate degree has persisted and compounded until today.

After completing my PhD at Western, I began my postdoctoral fellowship at the Icahn School of Medicine at the Mount Sinai Hospital in New York City. I had the opportunity to work with Dr. Peter Palese, a world-acclaimed virologist and leading expert in the influenza virus. Upon completing my postdoctoral studies, the decision to remain in the U.S. or return to Canada was presented to me when looking for faculty positions. Even though there were a series of advantages to becoming a member of the vast and productive U.S. international research institutions, the Canadian research scene was more supportive to new investigators. McMaster University was the best fit in terms of resources, location, and faculty. What was important to me was to go to a place with excellent faculty and students. As faculty members, it is largely students that drive our research since we physically can't be in the lab at all times. My colleagues are also excellent researchers—they're all doing amazing work. I wanted to be in a place where everyone around you is great and where it isn't easy to be great. It was very motivating to come here, as it is easy to become complacent if you go somewhere and are immediately heads and tails above everyone else. McMaster is an institution where doing great things is the norm, pushing you to become really great.

■ IN YOUR OPINION, WHAT ARE THE HIGHLIGHTS OF YOUR RESEARCH?

A major highlight of my research began during the 2009 swine flu epidemic. At the time, scientists were investigating the antibody immune responses of those infected with swine flu versus those vaccinated against swine flu. They noticed that both populations were producing an antibody that recognizes a conserved component of the flu virus and as a result, could elicit a protective effect against a multitude of different flu strains. Thus, I investigated if this broad-spectrum antibody phenomenon was unique to swine flu or ubiquitous to all flus. My lab knew that induced systemic production of the swine flu antibody could become a viable universal flu vaccination strategy and therefore alleviate the need for revaccination each year. Ultimately, we discovered that this is a general phenomenon that can be exploited to “trick” the immune system into producing broad-spectrum flu antibodies. The resulting vaccine is now licensed and ready for clinical trial. Another major highlight relates to our research on neurodegenerative diseases, which is necessitated by the aging population and the fact that most of these diseases have few treatments and certainly no cures. We stumbled upon the observation that viral infections and their subsequent immune responses may trigger these neurodegenerative diseases. This is an exciting new frontier with a series of interesting findings that would make a big difference in a field that really needs it in the future.

■ RECENTLY, A SIGNIFICANT NUMBER OF MEDIA PUBLICATIONS HAVE BROADCASTED YOUR COLLABORATIVE RESEARCH IN CHARACTERIZING A NOVEL UNIVERSAL FLU VACCINE. CAN YOU EXPLAIN WHAT A UNIVERSAL VACCINE IS CAPABLE OF IN THE CONTEXT OF THE FLU AND THE IMMUNOLOGICAL MECHANISMS THAT MAKE YOUR PROPOSED STRATEGY POSSIBLE?

Most pathogens fortunately don't change much over time in structurally. The flu does. Flus evolve very rapidly to evade the preexisting immunity that is generated by either natural infection or vaccine. The protein on the outside of a flu agent that attaches to and infects our cells resembles a lollipop. It has a big, round, globular head and a skinny stalk. Like lollipops, for every flu, the head is either a different shape, colour, or flavour. Our immune system only recognizes one shape or colour typically, and preferentially generates antibodies against the head portion. But, the virus can change the head whenever it wants and the protein remains functional—it's a really unique property of the flu. Like a lollipop, the stem is conserved across all flus and never changes. That's partially because the virus requires the stem protein in order to infect the cell. The virus wants to mutate so that antibodies for the stalk can't bind either. However, it can't do that because then it becomes non-functional. So, by teaching the immune system to preferentially make antibodies to this unchanging portion,