Faye Rogers, PhD, was puzzled, a fruitful state for a scientist. The Associate Professor of Therapeutic Radiobiology knew that cells respond to DNA damage by altering a network of pathways to manage and thus preserve genomic integrity. “One of the foundational questions of my lab,” said Dr. Rogers, “is how do these pathways talk to each other? And, if too much damage occurs, and the DNA can’t be repaired efficiently, how do cells determine to activate apoptosis?”

The answer she found to those quintessential questions to new possibilities for cancer treatment. Dr. Rogers and her team have discovered a way to turn on the apoptotic pathway in cancer cells, tricking them into killing themselves while leaving normal cells unscathed. Their research was published in October 2021 in Nature Biotechnology.

Working with a model of HER2-positive breast cancer, the scientists had been studying nucleotide excision repair (NER). NER is one of the main pathways for removing damaged strands of DNA and replacing them with healthy ones, forming a double helix. Instead of forming oligonucleotide (TFO) that binds to the site, then inserted a three-stranded structure using a triplex-forming oligonucleotide (TFO) that binds to the site. They watched how the NER pathway responded. Instead of forming oligonucleotide (TFO) that binds to the site, the TFOs offered a major advantage over drugs that work by inhibiting the overexpressed protein driving the cancer. Gene amplification often allows cancer cells to figure out ways to sabotage the inhibitor and resume growth. This drug resistance has proven to be the Achilles heel of many cancers that lack targeted therapies, such as ovarian cancer. Dr. Rogers’s research. HER2 is just the beginning. More than 460 amplified genes have been identified in 14 cancer subtypes. All these genes are potentially vulnerable to specific TFOs. Currently, Dr. Rogers and her team are focusing on cancers that lack targeted therapies, such as ovarian cancer. As the top of her lab’s most-recent list is 400, an oncogene amplified in up to 70 percent of human cancers, including ovarian.

“At the top of our wish list are patients who have never been treated HER2,” said Dr. Rogers. Her lab is also exploring different ways to deliver the TFOs, from nanoparticles to antibodies.

“We’re really excited about this work,” she added. “I think it has the potential to serve as a foundation for a platform that can be beneficial for the next generation of precision medicine for a wide range of patients who suffer from many different cancers.”