New Drug = New Hope

Kevin Williams thought cancer was something that happened to other people. At 35, he thought he was too young to get cancer. He felt great, was active and lived a healthy lifestyle. Cancer, he thought, couldn't happen to him.

He was wrong.

At 35, Williams, a husband and father of two, was diagnosed with chronic myeloid leukemia (CML), a type of cancer that starts in the blood-forming cells of the bone marrow. He was playing racquetball and felt a stitch in his side. That stitch turned out to be an enlarged spleen caused by his excessively high white blood cell count.

"When I heard the word leukemia, I thought it was over," said Williams, now 42 and a Lakewood, Calif., resident. "I thought I was going to die. The thought that I might not be around for my family was incredibly painful. My kids were still very young and depended on me. I felt like I was letting them down."

Williams was put on Interferon, the standard treatment for CML at the time. The drug controlled his white blood cell count, but it made him feel more sick than the cancer did. He had chills, fevers, and he lost a significant amount of weight. It was, he recalls, like having the flu—and not just for a few days, but for months and months on end with no relief in sight.

Williams had heard about an experimental targeted therapy, now called Gleevec, being tested at UCLA's Jonsson Comprehensive Cancer Center in patients with CML. Renowned UCLA researcher Dr. Owen Witte had previously discovered the gene that causes CML, providing a target for new therapies that attempt to home in on what is broken in a cancer cell, leaving the healthy cells alone.

UCLA clinical researchers were among the first in the nation to test Gleevec in CML patients. Meanwhile, scientists on the Westwood campus were studying its biologic mechanisms in the laboratory. Gleevec was one of the first in a wave of molecularly targeted therapies that many researchers believe represent the future in cancer care. Its success in clinical trials garnered national headlines. It was touted as hope in a pill.

For Williams, the news reports were exciting, and torturous. He couldn't enroll in the clinical trial because the Interferon, technically, was still working for him.

"Interferon, basically, was a chemotherapy drug," Williams said. "It was pretty tough to take."

Then, in 2001, the U.S. Food & Drug Administration approved Gleevec for CML and Williams was able to take the drug to treat his cancer.

"It was like night and day," Williams said. "I took a pill and, literally, I had no side effects. I felt great."

Gleevec kept Williams's blood counts controlled, but it never fully eliminated the cancer. The mutated gene causing the CML was still active in his bone marrow. And while some patients experienced

a complete response to the drug, Williams was not one of them.

However, as long as his blood counts remained good and he suffered no debilitating side effects, the drug had transformed his cancer into a chronic condition, like diabetes or high blood pressure. His cancer, he found, was something he could live with — in fact, live well with.

But, unknown to Williams, inside his body the CML was silently and craftily mutating in ways that would make it resistant to Gleevec one day. A persistent enemy, his cancer was finding a way to get around the drug.

While this silent war raged within Williams's body, UCLA scientists were discovering how the cancer became resistant to Gleevec. They uncovered more than a dozen mutations that developed over time. That discovery provided potential novel targets for new therapeutics and renewed hope for patients like Williams.

Gleevec worked for Williams for about five years. When his white blood cell counts began to rise, his oncologist increased the drug's dosage. Then the higher doses became ineffective, and Williams again began to worry that he might not beat his cancer after all.

Then, in 2005, he was able to enroll in a UCLA study testing a drug that appeared to be effective in patients who became resistant to Gleevec, a drug that targeted the mutations discovered in the UCLA laboratory. The drug, now called Sprycel, also is taken in pill form. It gave Williams another shot at fighting his cancer.

"Nine months after I started taking Sprycel, the cancer was undetectable in all the tests they did," Williams said. "It's so nice now as I watch my children get older. I have hope for the future. It's great to know I'll be around for them."

The mutated genes in his bone marrow causing the CML have disappeared, something that Gleevec could not accomplish. He continues to get tests to monitor for the CML-causing mutation, but his bone marrow remains clear.

"My life is completely back to normal now," he said. "I feel like I did before I got diagnosed. I can do anything I want, go skiing, ride my motorcycle, be with my family. This drug really has given me my life back. For me, it's been a miracle.

"The work that the researchers at the Jonsson Cancer Center are doing is critical. They're looking at different ways to attack cancer and doing cutting-edge studies to find new and more effective therapies. They're why I'm here today. I'm living proof that cancer research can save lives." ✶

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Williams plays with his son, Brian, in the backyard of their Lakewood home.