Adaptation of a Journal Article

I rewrote part of Shannon Maude's article, Chimeric antigen receptor T cells for sustained remissions in leukemia, to target a more general audience, specifically adults interested in science. Since Maude's study was conducted at the Children's Hospital of Philadelphia, my adaptation might be a good article for the science section of a Philadelphia newspaper. People reading a science section are somewhat educated, but not necessarily in biology. Overall, both texts have similar content, but my adaptation uses simpler grammar and vocabulary, plus adaptive techniques.

Both texts begin with the current status of leukemia treatments, but my adaptation uses simpler language to explain it. For example, I mentioned that the disease "can be difficult to treat," but the original article specified "relapsed and refractory" ALL. My adaptation excludes the very scientific description of how the modification works (the CD3-zeta domain and the CD137 [4-1BB] domain), and instead focuses on comparison.

The original article and my adaptation were written for readers with drastically different background knowledge. Readers of *The New England Journal of Medicine* are likely familiar with how chemotherapy works, and would therefore understand why this new treatment is beneficial. However, I made the connection clearer for general readers. Many science concepts can be abstract to a general audience, so I needed to make the concepts easier to understand. I compared the cell harvesting process to blood donation because even most general readers are familiar with that. I also used a hunter metaphor for the cells. These types of comparisons are not included in the article for a scientific audience.

I included definitions for things such as T cells, which did not need to be defined for readers of *The New England Journal of Medicine*. I defined T cells as a type of white blood cells, but I did not provide a complicated definition for what chimeric antigen receptor means. Instead, I focused on what they do.

Adaptation: Modified T cells provide new hope for leukemia patients

Acute lymphoblastic leukemia (ALL) can be a difficult cancer to treat, but a new targeted therapy offers hope for the future. Hospitals around the country, including the Children's Hospital of Philadelphia, have seen success in early patient trials.

The targeted treatment relies on T cells, which are a type of white blood cell. Patients' own T cells are harvested in a process similar to blood or platelet donation. In a lab, the T cells are then modified to include a chimeric antigen receptor (CAR). These receptors become cancer hunters – able to find and kill the leukemia cells. Unlike chemotherapy, which kills good and bad cells, the modified hunter cells look specifically for CD19, a marker common on leukemia cells.

Original: Chimeric antigen receptor T cells for sustained remissions in leukemia

Engineered T-cell therapy is a new strategy for the treatment of relapsed and refractory acute lymphoblastic leukemia (ALL), which is associated with an extremely poor prognosis in adults and remains a leading cause of death from childhood cancer.1-3 In initial proof-of-principle clinical trials involving patients with chronic lymphocytic leukemia (CLL), chimeric antigen receptor–modified T cells that target CD19 produced a durable complete remission in a small number of patients.4-6 Our group and others then extended these findings to relapsed and refractory B-cell ALL, and we found profound responses in a small number of children and adults.7,8

Chimeric antigen receptors are genetically engineered receptors that couple an anti-CD19 singlechain Fv domain to intracellular T-cell signaling domains of the T-cell receptor, thereby redirecting cytotoxic T lymphocytes to cells expressing this antigen. With the use of lentiviral-vector technology for gene transfer and permanent T-cell modification, CTL019 (formerly known as CART19)-engineered T cells express a chimeric antigen receptor in which the T-cell activation signal is provided by the CD3-zeta domain, and the costimulatory signal is provided by the CD137 (4-1BB) domain.4

Reference:

Maude, S. L., et al. (2014). Chimeric antigen receptor T cells for sustained remissions in leukemia. *The New England Journal of Medicine,* Vol. 371 Issue 16, 1507-17.