Researchers discover rare leukemia-causing protein

July 2 2012

Researchers at the University of Cincinnati (UC) Hoxworth Blood Center have discovered a new gene target for leukemia therapy.

These findings, slated for the July 26, 2012 print issue of *Blood*, the journal of the American Society of Hematology, could lead to cellular targets for a patient population that otherwise may not have desirable outcomes and could potentially stop the onset of leukemia before it begins.

A team led by Jose Cancelas, MD, PhD, an associate professor of pediatrics at the UC College of Medicine and director of the research division at Hoxworth Blood Center, found that by inhibiting in animal models the protein Vav3, which controls cell signaling, the development of this leukemia—known as BCR-ABL lymphoid leukemia—is delayed.

"Despite advances in the treatment of this disease, the outcome of patients with this type of leukemia is very poor because it develops resistance to standard therapies," he says. "We found that the genetic deficiency of Vav3 delays the formation of leukemia by impairing the signals from BCR-ABL and the overproduction of leukemic cells. In doing this, it also allows the standardized therapies, or BCR-ABL inhibitors, to work."

Cancelas says this finding could lead to new multi-targeted therapies where Vav3 activity is related to the formation of leukemia.

"In collaboration with Dr. Nicolas Nassar, associate professor of pediatrics at UC and a physician in the division of experimental hematology at Cincinnati Children's Hospital Medical Center, we are now trying to find chemicals with Vav3 inhibitory activity," he says. "With this knowledge, we may be able to develop a therapy that can greatly improve the lives of patients facing leukemia."

Provided by University of Cincinnati Academic Health Center