Study in mice may identify new ways to treat immune thrombocytopenia

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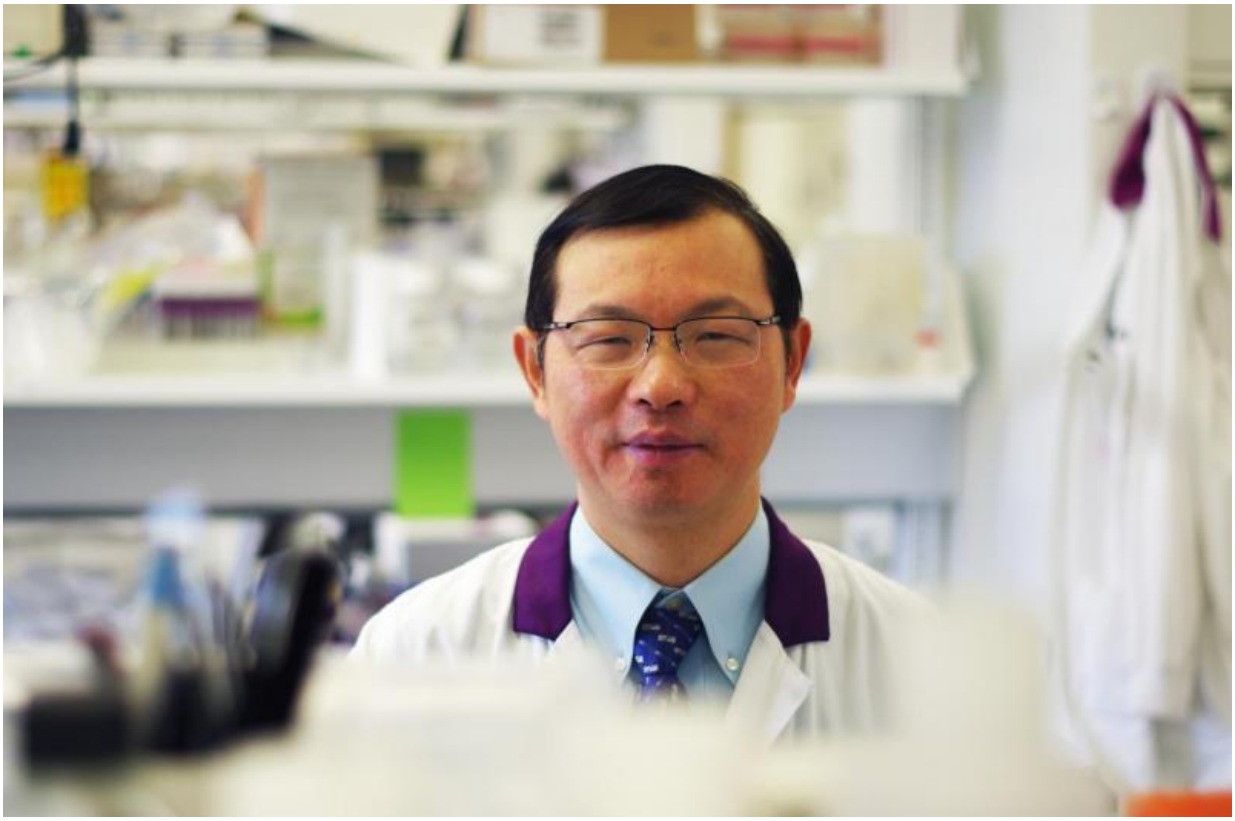

A new study by Dr. Heyu Ni, a scientist in the Keenan Research Centre for Biomedical Science of St. Michael's Hospital, may identify new ways to treat immune thrombocytopenia. Credit: St. Michael's Hospital

Immune thrombocytopenia, or ITP, is an autoimmune disease whereby the immune system sends antibodies to attack and destroy the body's

platelets—blood cells responsible for controlling bleeding.

Stopping ITP is important because if platelet counts in a body are low, simple cuts could bleed for hours and more traumatic injuries could be fatal. The body's inability to control bleeding can also lead to stroke.

ITP affects one in 10,000 people in Canada and accounts for 0.18 per cent of all hospital admissions. Most cases are spontaneous and without any clear cause. ITP's severity and the effectiveness of its treatment vary from patient to patient. A new study, published today in *Nature Communications*, may explain why there is so much variance in symptoms and response to treatment.

The surface of every platelet is covered with thousands of different proteins and each type of antibody targets a specific protein on the platelet. The first antibody to find a platelet latches on and leads the platelet to an organ where it will be destroyed.

It's always been thought that all ITP antibodies lead platelets to the spleen for destruction.

"Every existing treatment for ITP has been dedicated to stopping antibodies from destroying platelets in the spleen, but we've discovered that some antibodies actually destroy platelets in the liver," said Dr. Heyu Ni, a scientist in the Keenan Research Centre for Biomedical Science of St. Michael's Hospital.

The discovery was made by looking mice treated with two different types of antibodies most common to ITP. Each of these two antibodies targets a different protein on the surface of platelets—either GPIb or GPIIbIIIa. The researchers found that antibodies targeting GPIb lead to platelets to be destroyed in the liver, whereas those targeting GPIIbIIIa caused platelet destruction in the spleen.

"By detecting the specific antibodies present in someone with ITP, we may be able to detect where and how the immune system will attack," said Dr. Heyu Ni, who is also a scientist with Canadian Blood Services and a professor at the University of Toronto. "And because we now know the liver's immune response destroys platelets covered with GPIb, we may be able to design new therapies to stop this type of platelet destruction."

Dr. Ni said that there are drugs, such as Tamiflu, which may be able to inhibit the liver's immune response to the platelets.

While much of this study's research was conducted in mice, researchers used some human blood samples to test whether Tamiflu might inhibit antibodies targeting GPIb.

"Using healthy blood samples and ITP antibodies in a test tube, we showed that Tamiflu may impede platelet destruction for those with antibodies that target GPIb," said Dr. Ni.

Based on an early abstract of this research study, some individuals around the world with ITP have been given Tamiflu to treat the disease. These people were in life-threatening condition and extremely drug-resistant to existing treatments targeting the spleen. Although these instances of experimental treatment have been successful, Dr. Ni said more research is needed to verify the safety and efficacy of this approach.

Provided by St. Michael's Hospital