Immune protein could stop diabetes in its tracks

May 20 2013

Diabetes researcher Professor Len Harrison has identified that the immune protein CD52 protects the body against excessive or damaging immune responses, and could be used to prevent and treat type 1 diabetes and other autoimmune diseases.

Melbourne researchers have identified an immune protein that has the potential to stop or reverse the development of type 1 diabetes in its early stages, before insulin-producing cells have been destroyed.

The discovery has wider repercussions, as the protein is responsible for protecting the body against excessive immune responses, and could be used to treat, or even prevent, other immune disorders such as multiple sclerosis and rheumatoid arthritis.

Professor Len Harrison, Dr Esther Bandala-Sanchez and Dr Yuxia Zhang led the research team from the Walter and Eliza Hall Institute's Molecular Medicine division that identified the immune protein CD52 as responsible for suppressing the immune response, and its potential for protecting against autoimmune diseases. The research was published today in the journal *Nature Immunology*.

So-called autoimmune diseases develop when the immune system goes awry and attacks the body's own tissues. Professor Harrison said CD52 held great promise as a therapeutic agent for preventing and treating autoimmune diseases such as type 1 diabetes.

"Immune suppression by CD52 is a previously undiscovered mechanism that the body uses to regulate itself, and protect itself against excessive or damaging immune responses," Professor Harrison said. "We are excited about the prospect of developing this discovery to clinical trials as soon as possible, to see if CD52 can be used to prevent and treat type 1 diabetes and other autoimmune diseases. This has already elicited interest from pharmaceutical companies."

Type 1 diabetes is an autoimmune disease that develops when immune cells attack and destroy insulin-producing beta cells in the pancreas. Approximately 120,000 Australians have type 1 diabetes and incidence has doubled in the last 20 years. "Type 1 diabetes is a life-long disease," Professor Harrison said. "It typically develops in children and teenagers, and it really makes life incredibly difficult for them and their families. It also causes significant long-term complications involving the eyes, kidneys and blood vessel damage, and at great cost to the community."

Professor Harrison said that T cells that have or release high levels of CD52 are necessary to maintain normal balance in the immune system. "In a preclinical model of type 1 diabetes, we showed that removal of CD52-producing immune cells led to rapid development of diabetes. We

think that cells that release CD52 are essential to prevent the development of autoiummune disease, and that CD52 has great potential as a therapeutic agent," he said.

CD52 appears to play a dominant role in controlling or suppressing immune activity in the early stages of the immune response, Professor Harrison said. "We identified a specialised population of immune cells (T cells) that carry high levels of CD52, which they release to dampen the activity of other T cells and prevent uncontrolled immune responses," Professor Harrison said. "The cells act as an early 'braking' mechanism."

Professor Harrison said his goal is to prevent and ultimately cure type 1 diabetes. "In animal models we can prevent and cure type 1 diabetes," Professor Harrison said. "I am hopeful that these results will be translatable into humans, hopefully in the not-too-distant future."

This research was supported by the National Health and Medical Research Council of Australia and the Victorian Government.

The article is titled "T cell regulation mediated by interaction of soluble CD52 with the inhibitory receptor Siglec-10."

More information: www.nature.com/ni/journal/vaop … ent/abs/ni.2610.html

Provided by Walter and Eliza Hall Institute of Medical Research