



February 2, 2010
RIKEN

HMGB1 protein triggers islet cell rejection

Researchers at RIKEN and Fukuoka University have pinpointed the mechanism responsible for early rejection of transplanted pancreatic islet cells in the treatment of type 1 diabetes, also known as juvenile diabetes. A new system based on this mechanism has been shown to vastly increase transplant efficiency, setting the stage for the development of powerful new treatment techniques.

Currently, the most widely-used treatment for type 1 diabetes is the regular injection of insulin, a burdensome task for patients. Islet cell transplantation, whereby insulin-producing cells from a donor pancreas are transplanted into the patient's liver, is a promising alternative approach. However, it has achieved limited success due to a strong and rapid immune-mediated rejection of the transplanted islets.

With their discovery, the researchers have demonstrated that HMGB1 (high-mobility group box 1), a nuclear protein whose precise function has heretofore remained elusive, is in fact produced by the islet cells and directly triggers their early rejection. Based on this finding, they developed a system to measure the level of HMGB1 in the blood and determine the onset of rejection, information which they used to establish a treatment four times more effective than earlier islet transplantation protocols.

While shedding light on a previously-unknown function of a major nuclear protein, the discovery of the HMGB1-mediated pathway also represents a breakthrough in diabetes research. For millions of diabetes sufferers around the world, its application to islet transplantation promises great improvements in this technique, bringing dreams of insulin independence one step closer to reality.

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