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科目：英文科普文章測驗 共3頁

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Human cells reprogrammed to create insulin (From NEWS 13 FEBRUARY 2019)

Pancreatic cells that don't normally produce insulin can be modified to do so, and to help control blood sugar levels in diabetic mice.

The destruction of a single kind of insulin-producing cell in the pancreas can lead to diabetes — but a study suggests that other cells could be modified to take its place and help to control blood sugar levels.

The results raise hopes that 'reprogrammed' insulin-producing cells could be used as treatment for diabetes, but the approach has so far only been tested with human cells in mice studies.

In a study published on 13 February in *Nature*[1], researchers report coaxing human pancreatic cells that don't normally make insulin, a hormone that regulates the amount of glucose in the blood, to change their identity and begin producing the hormone.

When implanted in mice, these reprogrammed cells relieved symptoms of diabetes, raising the possibility that the method could one day be used as a treatment in people.

"I think this has got huge potential," says Terence Herbert, a biologist at the University of Lincoln, UK. But it is still early days, he says, with several hurdles to overcome before the technique can be used in the clinic.

System breakdown

When blood sugar levels rise after eating, cells in the pancreas called β-cells normally respond by releasing insulin, which in turn stimulates cells to start absorbing sugars. In people with diabetes, this system breaks down, leading to high blood sugar levels that can damage the body and cause illness.

In type 1 diabetes, the immune system attacks and destroys β-cells; in type 2, the β-cells do not produce enough of the hormone, or the body becomes resistant to insulin.

Scientists have previously shown in mouse studies that if β-cells are destroyed, another type of pancreatic cell, called α-cells become more β-like and start making insulin[2]. These α-cells normally produce the hormone glucagon, and are found alongside β-cells in clumps of hormone-secreting cells called pancreatic islets or islets of Langerhans. Previous studies showed that two proteins that control gene expression seemed to have an important role in coaxing α-cells to produce insulin in mice: Pdx1 and MafA.

The human factor

So Pedro Herrera at the University of Geneva, Switzerland, and colleagues wondered whether producing more of these proteins in human α-cells would have a similar effect.

They first took islet cells from human pancreases, and separated out the individual cell types. They then introduced DNA that encoded Pdx1 and MafA proteins into the α-cells, before clumping them back together.

After one week in culture, almost 40% of the human α-cells were producing insulin, whereas control cells that hadn't been reprogrammed were not. The reprogrammed cells also showed an increase in the expression of other genes related to β-cells. "They have a hybrid personality," says Herrera.

The team then implanted the mass of cells into diabetic mice, which had their β-cells destroyed, and found that blood-sugar levels went down to normal levels. When the cell grafts were removed, the mice's blood sugar shot back up.

Switching identity

Herrera says that if α-cells — or other kinds of islet cells — could be made to start producing insulin in this way in people with diabetes, their quality of life might be greatly improved. The dream, Herrera says, is to find a drug that can switch the identity of α-cells.

But he acknowledges that any kind of treatment is still far away. First, his team will need to work out what is going on at the molecular level when α-cells become more β-like.

Other teams are also trying to create new insulin-producing cells in the pancreas: some have sought to generate β-cells from stem cells. But in type 1 diabetes, the immune system attacks β-cells, posing a challenge for such strategies.

Herrera and his team present some evidence that their hybrid cells are less prone to this kind of attack, notes Herbert, suggesting that their method could be a more feasible way of generating β-cells than the stem-cells approach.

But Herbert adds that, before the authors can draw strong conclusions about the efficacy of their approach, they will need to test the hybrid cells with other antibodies present in type-1 diabetes that could potentially attack those cells.