

## JOURNAL WATCH

Our panel of experts highlight the most important research articles across the spectrum of topics relevant to the field of diabetes management

**Expert panel:** X Charlie Dong, Department of Biochemistry & Molecular Biology, Indiana University School of Medicine, Room 1021D, 635 Barnhill Drive, IN, USA; xcdong@iupui.edu; Braxton D Mitchell, Division of Endocrinology, Diabetes & Nutrition, University of Maryland School of Medicine, Room 492, 660 West Redwood Street, MD, USA; bmitchel@medicine.umaryland.edu

---

**Marselli L, Suleiman M, Masini M et al.** Are we overestimating the loss of  $\beta$  cells in Type 2 diabetes? *Diabetologia* 57(2), 362–365 (2013).

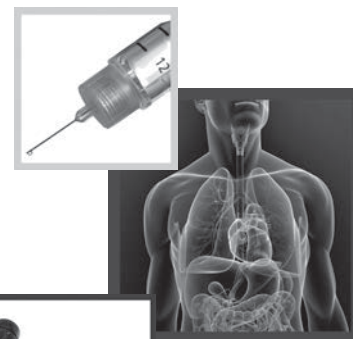
The aim of this study was to assess pancreatic  $\beta$  cell mass and function in Type 2 diabetics. The authors obtained pancreases from heart-beating organ donors, including ten nondiabetics (five males and five females at an average age of 68 years) and ten diabetics (four males and six females with a mean age of 65). Pancreatic islet mass was analyzed by immunocytochemistry and electron microscopy and islet functions were examined by glucose-stimulated insulin secretion analysis. The major findings from this study are the followings: insulin-positive area was significantly lower ( $54.9 \pm 6.3\%$  vs  $72.1 \pm 8.7\%$ ) between diabetic and nondiabetic groups; the proportion of  $\beta$  cells was decreased by only 10% ( $p = 0.051$ ) in the diabetics compared with the nondiabetics, but the diabetics had fewer insulin granules per  $\beta$  cell ( $3.1 \pm 0.2\%$  vs  $5.6 \pm 0.3\%$ ); and glucose-stimulated insulin secretion was decreased 40–50% in the diabetic islets as compared with the nondiabetic islets. From these data, it was concluded that at least in subgroups of Type 2 diabetics the loss of pancreatic  $\beta$  cells might be over estimated.

– Written by X Charlie Dong

---

**Ilegems E, Dicker A, Speier S et al.** Reporter islets in the eye reveal the plasticity of the endocrine pancreas. *Proc. Natl Acad. Sci. USA* 110(51), 20581–20586 (2013).

The objective of this research was to test an optically accessible indicator of pancreatic islet plasticity in an obese mouse model. To do so, the authors transplanted a few reporter islets from a donor mouse into the anterior chamber of the eye of the recipient leptin-deficient ob/ob mouse. Engrafted reporter islets were monitored longitudinally using confocal microscopy. With this new approach, the investigators could observe an increase in islet mass and intra-islet blood vessel diameters from the transplanted islets in the ob/ob mice. By using backscatter imaging, they could also analyze the islet morphology and insulin secretion status: the engrafted islets in the eye of ob/ob mice exhibited a degranulated and uneven pattern. The transplanted islets also showed donor strain-independent growth in the recipient ob/ob mice, and the proliferation rate was not significantly different from *in situ* pancreatic islets. After the leptin treatment in the sham and islet-transplanted ob/ob mice was performed between 3 and 4 months of age, the engrafted islets appeared to be normalized in morphology and function. In the end, the authors concluded that reporter islets could be a useful tool



## News & Views

News

---

**Journal Watch**

---

Conference Scenes

---

Interview

---

Future  
Medicine part of **fsg**

for monitoring islet functions in the pancreas, and may be used for evaluation of treatment efficacy targeting islet plasticity *in vivo*.

– Written by X Charlie Dong

---

**Moran C, Phan TG, Chen J *et al*.  
Brain atrophy in Type 2 diabetes:  
regional distribution and influence  
on cognition. *Diabetes Care* 36(12),  
4036–4042 (2013).**

Type 2 diabetes (T2DM) is associated with brain atrophy, cerebrovascular disease, and cognitive decline. To better understand potential causal links between T2DM and dementia, Moran and colleagues obtained brain scans using MRI from more than 700 people with and without T2DM

and compared brain scan characteristics between the two groups. As expected, subjects with diabetes performed less well on multiple cognitive tests compared with age-matched controls. Diabetic subjects also had more shrinkage in certain areas of the brain, particularly in areas related to cognitive function and in patterns seen in preclinical Alzheimer's disease. Adjusting for these brain atrophy characteristics in multivariate models markedly attenuated the associations between T2DM and cognition, implying that the cognitive decline accompanying T2DM is mediated in part by brain atrophy. By contrast, although subjects with T2DM also had more strokes and more cerebral infarcts, adjusting for these did not attenuate associations between T2DM and cognition. Taken together, these results imply that

brain atrophy, rather than cerebrovascular lesions, is likely the primary reason for cognitive impairment associated with T2DM and suggest that strategies to slow brain atrophy may preserve cognitive function in subjects with T2DM.

– Written by Braxton D Mitchell

---

**Financial & competing interests  
disclosure**

*The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.*

*No writing assistance was utilized in the production of this manuscript.*