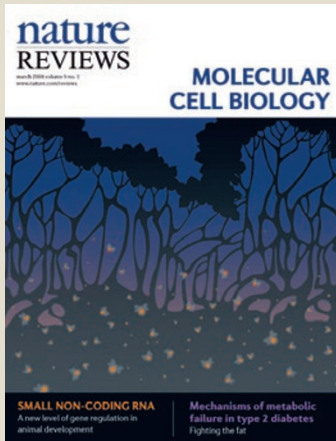




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► **COVER:** 'The synchronized 'flashing' of fireflies at night' by Vicky Askew, inspired by the Perspective on p255



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With the excesses of the Carnival season behind them, many people around the world are observing the traditional 40 days of Lent. For some, this means giving up their favourite food, while for others it means giving up meat. (Although the origin of the word 'carnival' has been disputed, it may come from the Latin *carne(m) levare*, which means 'to take away meat'.) All of us, however, might want to consider the benefits of permanently altering our diets to improve our health, especially given the well-documented link between obesity and type 2 diabetes.

Type 2 diabetes is projected to afflict 300 million people worldwide by 2020, so it is crucial to gain insight into the molecular and metabolic mechanisms of this disease. In the latest instalment of our [Mechanisms of Disease](#) series, Deborah M. Muoio and Christopher B. Newgard (page 193) discuss factors that cause impairment in insulin secretion and insulin action, which leads to increased concentrations of glucose and lipids in the blood and, ultimately, to type 2 diabetes. Overnutrition induces a high rate of insulin secretion by pancreatic β -cells, which results in cell stress and a loss of β -cell mass and contributes to development of the disease. The fact that type 2 diabetes affects different tissues, and that each of these responds in different ways to excess metabolic fuels, makes it difficult to find an effective and long-lasting therapy for this disease.

On a positive note, a recent report describes the identification of endogenous β -cell progenitors in the adult mouse pancreas, as discussed in a Research Highlight on page 188. These findings provide new hope for the therapeutic regeneration of β -cells in type 1 and type 2 diabetes.

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Printed in Wales by Cambrian Printers
on acid-free paper