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# Up front

Competition for publication in *Diabetologia* continues to grow, and less than 20% of papers are accepted. Of all the high-quality papers that appear in this month's issue I want to draw your attention to five articles that I think are particularly interesting. The articles are summarised here. Our publisher, Springer, has kindly made the full text of each of these papers freely available.
I hope you enjoy reading them!

*Sally M. Marshall, Editor*

## Adipose tissue plasticity: how fat depots respond differently to pathophysiological cues

*Vanessa Pellegrinelli, Stefania Carobbio, Antonio Vidal-Puig*

Evidence for the existence of thermogenically active brown fat/beige cells in adult humans has fuelled new research aiming to cure obesity and related metabolic disorders. Strategies relying upon differentiation and activation of brown/beige fat are believed to halt the lipotoxic overspill resulting from dysfunctional white fat in obesity. In a review in this issue, Pellegrinelli et al summarise the current understanding of the specific origins, development and remodelling of different fat depots under physiological and pathophysiological conditions. Visceral and subcutaneous white fat depots differ at the structural and functional levels, particularly concerning progenitor number, adipocyte size and expandability capacity. Studies have also shed light on white/brown progenitor origins by identifying adipose niches adjacent to the vasculature. The integration of these recent reports with the current understanding of the impact of fibro-inflammation on adipogenesis promises to identify new molecular pathways for targeted strategies to improve brown/white fat plasticity, activation and global metabolic homeostasis.

## The fetal glucose steal: an underappreciated phenomenon in diabetic pregnancy

*Gernot Desoye, Christopher J. Nolan*

Despite intense efforts to achieve glycaemic control, frustratingly, macrosomia (excessive fetal growth) still commonly occurs in mothers with diabetes. In a review in this issue, Desoye and Nolan provide a potential explanation. They state that the fetus acts as a glucose sink that steals glucose from the mother. Importantly, this 'fetal glucose steal' effect can be exaggerated in diabetic pregnancy as a consequence of fetal hyperinsulinaemia and can occur even at times of maternal normoglycaemia. An exaggerated glucose steal will attenuate maternal hyperglycaemia, giving clinicians a false sense that all is well. For similar reasons, some mothers with fetuses suspected of being affected by diabetes late in pregnancy may have 'normal' glucose tolerance, a point in favour of early screening for gestational diabetes. To prevent an exaggerated glucose steal driven by fetal hyperinsulinaemia, the authors argue that tight glycaemic control from early in pregnancy is necessary.

### Recent trends in life expectancy for people with type 1 diabetes in Sweden

*Dennis Petrie, Tom W. C. Lung, Adin Rawshani, Andrew J. Palmer, Ann-Marie Svensson, Björn Eliasson, Philip Clarke*

Life expectancy estimates for those with type 1 diabetes allow gaps between them and the general population to be identified and improvements to be quantified. In this issue, Petrie et al report the results of their study in which they used health records from the National Diabetes Register and linked these with death records to examine mortality in those with type 1 diabetes in Sweden between 2002 and 2011. They found that for men the remaining life expectancy at age 20 increased significantly by about 2 years (from 47.7 in 2002–06 to 49.7 years in 2007–11), but for women there was no significant change, with a life expectancy at age 20 of 51.7 years in 2002–06 and 51.9 years in 2007–11. Recent gains have come from reductions in cardiovascular mortality; however, these gains were also seen in the general population, which meant that the life expectancy gaps between people with type 1 diabetes and the general population have stayed at about 11 years for men and 12 years for women over the last decade. More needs to be done to close this gap. This article is the subject of a commentary in this issue by Lars Stene.

### Life expectancy of type 1 diabetic patients during 1997–2010: a national Australian registry-based cohort study

*Lili Huo, Jessica L. Harding, Anna Peeters, Jonathan E. Shaw, Dianna J. Magliano*

There is limited information about the impact of type 1 diabetes on life expectancy in a contemporary population. In this issue, Huo et al report that Australians with type 1 diabetes had an estimated life loss of 11.6 years for men and 12.5 years for women compared with the general population. No narrowing of this gap was seen over a 7-year period. In addition to deaths from cardiovascular disease in older adults, deaths before the age of 60 years and mortality from endocrine and metabolic disease both contributed substantially to the years of life lost for type 1 diabetes. For improvements in life expectancy, greater attention must therefore be paid to both the acute metabolic and chronic cardiovascular complications of type 1 diabetes. A failure to address either one will continue to leave type 1 diabetic patients at risk of premature mortality. This article is the subject of a commentary in this issue by Lars Stene.

### Variants in the *FTO* and *CDKAL1* loci have recessive effects on risk of obesity and type 2 diabetes, respectively

*Andrew R. Wood, Jessica Tyrrell, Robin Beaumont, Samuel E. Jones, Marcus A. Tuke, Katherine S. Ruth, The GIANT consortium, Hanieh Yaghootkar, Rachel M. Freathy, Anna Murray, Timothy M. Frayling, Michael N. Weedon*

Genome-wide association studies have identified hundreds of common genetic variants associated with diabetes and obesity. Most studies have tested additive effects, assuming that the risk of heterozygous individuals lies halfway between the two homozygous groups. Few studies have performed genome-wide analyses of dominant or recessive effects for common variants. In this issue, Wood et al analysed data from the UK Biobank on 119,688 individuals with measured BMI and 4,040 individuals with type 2 diabetes status. The authors tested for deviation from additive effects and showed that two common variants in *CDKAL1* and *FTO* act partially recessively. These findings indicate that the large number of people carrying just one copy of the risk alleles at these loci are at little extra disease risk. The data also suggest that follow-up clinical or functional studies should focus on recessive mechanisms, and that non-additive effects of common variants only contribute a small amount to diabetes and obesity risk.

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