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 the 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*

### The pancreas in health and disease

The focus of this year’s special issue is ‘The pancreas in health and in diabetes’, introduced in an editorial (https://doi.org/10.1007/s00125-020-05235-z). Atkinson et al (https://doi.org/10.1007/s00125-020-05203-7) begin by reviewing the current evidence on human pancreatic anatomy and the changes that occur in individuals with type 1 or type 2 diabetes. Jennings and colleagues (https://doi.org/10.1007/s00125-020-05161-0) go on to discuss how alterations in important steps in the development of the mammalian pancreas may contribute to the abnormalities seen in diabetes. Abnormal insulin production by the pancreas underlies the pathogenesis of diabetes, and Vasiljević and coauthors (https://doi.org/10.1007/s00125-020-05192-7) outline the post-transcriptional mechanisms regulating insulin production and how their deficits can cause diabetes. The sole source of circulating insulin is the pancreatic beta cell. In their review, Rutter et al (https://doi.org/10.1007/s00125-020-05205-5) discuss the specialisations that allow this cell to perform its unique role. Autoimmune beta cell destruction results in type 1 diabetes and Mallone and Eizirik (https://doi.org/10.1007/s00125-020-05176-7) consider why beta cells are vulnerable autoimmune targets. Esser et al (https://doi.org/10.1007/s00125-020-05245-x) go on to describe models to explain the pathogenesis of hyperglycaemia in type 2 diabetes. Age is one of the main risk factors for developing type 2 diabetes and Cristina Aguayo-Mazzucato (https://doi.org/10.1007/s00125-020-05185-6) summarises the functional changes that occur in ageing beta cells. Diabetes can also occur as a consequence of a wide variety of exocrine pancreas diseases, as discussed by Rickels et al (https://doi.org/10.1007/s00125-020-05210-8). Redondo et al (https://doi.org/10.1007/s00125-020-05211-7) propose a paradigm to help explain the heterogeneity within and between diabetes types, concluding that this new approach may facilitate personalised medicine in diabetes. In their review, Bellin and Dunn (https://doi.org/10.1007/s00125-020-05184-7) detail the variety of transplant strategies available for individuals with type 1 diabetes. Looking more closely at the islet itself, Mark Huising (https://doi.org/10.1007/s00125-020-05213-5) describes the cellular components of this special structure that are important to its role. Moede et al (https://doi.org/10.1007/s00125-020-05196-3) focus specifically on the importance of islet alpha cell–beta cell communication for glucose homeostasis, while Almaça et al (https://doi.org/10.1007/s00125-020-05186-5) summarise how different cellular and acellular components of the islet microenvironment can have an impact on endocrine cell function. Faber et al (https://doi.org/10.1007/s00125-020-05204-6) describe both the anatomy of autonomic nervous system input to the islet and the functional importance of autonomic nervous system outflow to the islet across a variety of physiological challenges to glucose homeostasis. Finally, Arrojo e Drigo et al (https://doi.org/10.1007/s00125-020-05159-8) conclude by discussing approaches to connect molecular profiles with functional properties underlying insulin secretion.
Diabetes is associated with an elevated risk of mortality in most infectious diseases. In this issue, Wang et al (https://doi.org/10.1007/s00125-020-05209-1) performed a retrospective study of the relationship between hyperglycaemia and 28-day mortality in coronavirus disease 2019 (COVID-19) patients not previously diagnosed with diabetes. They report that, of 605 COVID-19 patients at two hospitals based in Wuhan, China, 176 (29.1%) had hyperglycaemia (≥7.0 mmol/l) at admission without a previous diagnosis of diabetes. These patients had more than twice the risk of death as those with normal blood glucose. Even patients with a CRB score (an effective measure for assessing the severity of pneumonia) that was low or zero were more likely to die if they had increased fasting blood glucose levels. The authors conclude that most COVID-19 patients are prone to glucose metabolic disorders, and that glycaemic testing and addressing elevations in fasting blood glucose at an early stage might help improve the overall outcomes.

**The metabolic syndrome in pregnancy and its association with child telomere length**

Dale McAninch, Tina Bianco-Miotto, Kathy L. Gatford, Shalem Y. Leemaqz, Prabha H. Andraweera, Amy Garrett, Michelle D. Plummer, Gus A. Dekker, Claire T. Roberts, Lisa G. Smithers, Jessica A. Grieger

The metabolic syndrome is a clustering of cardiovascular risk factors. In the adult population, the metabolic syndrome associates with increased risk for cardiovascular and related diseases. Telomere length is a biomarker of ageing and is associated with the development of future chronic diseases. Maternal exposures in utero impact offspring health. In this issue, McAninch et al (https://doi.org/10.1007/s00125-020-05242-0) investigated whether maternal metabolic syndrome associates with telomere length, a biomarker of ageing, in the child. The authors found that the 10-year-old children of mothers who had the metabolic syndrome in pregnancy had 14% shorter telomeres than children of mothers who did not have the metabolic syndrome during pregnancy. Interestingly, anthropometric measures were similar in children of mothers who did and did not have the metabolic syndrome in pregnancy. The authors conclude that, although further studies are warranted, early assessment of telomere length in children may provide insight into their potential future chronic disease risk.

**Type 1 diabetes in Africa: an immunogenetic study in the Amhara of North-West Ethiopia**

Shitaye A. Balcha, Abayneh G. Denisse, Rajashree Mishra, Tanwi Vartak, Diana L. Cousminer, Kenyaita M. Hodge, Benjamin F. Voight, Kim Lorenz, Stanley Schwartz, Samuel T. Jerram, Arla Gamper, Alice Holmes, Hannah F. Wilson, Alistair J. K. Williams, Struan F. A. Grant, R. David Leslie, David I. W. Phillips, Elisabeth R. Trimble

From sub-Saharan Africa, a region of enormous genetic diversity, reports of insulin-dependent diabetes associated with both a low autoantibody prevalence and a low incidence in childhood have raised questions about its relationship to classic type 1 diabetes. In this issue, Balcha et al (https://doi.org/10.1007/s00125-020-05229-x) performed an immunogenetic study of insulin-dependent diabetes in individuals from the Amhara, the second-largest ethnic group in Ethiopia. The authors found that, although the genomes of the Amhara were distinct from European and other African genomes, individuals with insulin-dependent diabetes (89.4% born in rural areas) had the same principal HLA-DRB1 allele associations as cases of European-background type 1 diabetes. In contrast to previous reports, at diagnosis, the age-related total autoantibody prevalence was similar to that in many industrialised countries. However, the autoantibody profile was different; this was most marked in childhood cases, where the majority had a single autoantibody (to GAD), and the prevalence of autoantibodies to ZnT8 and IA-2 was very low. The authors conclude that these Ethiopian cases have the immunogenetic characteristics of autoimmune type 1 diabetes associated with an autoantibody profile that differs somewhat from cases of European background.

**Multi-layered epigenetic regulation of IRS2 expression in the liver of obese individuals with type 2 diabetes**

Christin Krause, Cathleen Geißler, Heidi Tackenberg, Alexander T. El Gammal, Stefan Wolter, Joachim Spranger, Oliver Mann, Hendrik Lehnert, Henriette Kirchner

Epigenetic regulation of gene expression is altered in type 2 diabetes. However, epigenetic regulation of insulin signalling in key metabolic tissues in type 2 diabetes is largely unknown. In this issue, Krause et al (https://doi.org/10.1007/s00125-020-05212-6) analysed the epigenetic gene regulation of IRS2, which encodes a crucial mediator of insulin signal transduction, in liver biopsies of obese individuals with and without type 2 diabetes. They found that hepatic expression of IRS2 is
decreased in type 2 diabetes, which is accompanied by decreased intronic DNA methylation and enhanced binding of two transcriptional repressors. They also report that the microRNA let-7e-5p is increased in the liver in diabetes, correlates with the decreased IRS2 expression, and might represent a complementary mechanism of IRS2 regulation. The authors conclude that these findings contribute to an improved understanding of dysregulated pathways in the liver of individuals with type 2 diabetes and illustrate the complexity and multifactorial nature of mechanisms involved in the regulation of glucose homeostasis.

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