Diabetologia 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 stand out in some regard and are very 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!

Hindrik Mulder, Editor

Environmental risk factors of type 2 diabetes—an exposome approach

Joline W. J. Beulens, Maria G. M. Pinho, Taymara C. Abreu, Nicole R. den Braver, Thao M. Lam, Anke Huss, Jelle Vlaanderen, Tabea Sonnenschein, Noreen Z. Siddiqui, Zhendong Yuan, Jules Kerckhoffs, Alexandra Zhernakova, Milla F. Brandao Gois, Roel C. H. Vermeulen

A major part of the burden of type 2 diabetes is attributed to environmental risks and modifiable risk factors such as lifestyle. The environment we live in, and changes to it, can therefore contribute substantially to the prevention of type 2 diabetes at a population level. In this issue, Beulens et al (https://doi.org/10.1007/s00125-021-05618-w) summarise the evidence on the role of the food-, built-, physico-chemical- and social environment in the development of type 2 diabetes. The authors discuss the established associations of air pollution, residential noise and area-level socioeconomic deprivation with an increased risk of type 2 diabetes, and highlight that neighbourhood walkability and green space are associated with a reduced risk of type 2 diabetes. The contribution of the food environment, along with other aspects of the social environment and outdoor temperature are less clear. The authors suggest that these environmental factors affect type 2 diabetes risk mainly through mechanisms that incorporate lifestyle factors, the microbiome, inflammation or chronic stress. The figures from this review are available as a downloadable slideset.

Prediabetes and risk of mortality, diabetes-related complications and comorbidities: umbrella review of meta-analyses of prospective studies

Sabrina Schlesinger, Manuela Neuenschwander, Janett Barbaresko, Alexander Lang, Haifa Maalmi, Wolfgang Rathmann, Michael Roden, Christian Herder

The number of people with prediabetes is increasing. Prediabetes is defined here as a condition in which glucose metabolism is impaired but the diagnostic criteria for type 2 diabetes are not currently met. However, it is often a precursor for the development of type 2 diabetes. Whilst it does not usually exhibit symptoms, studies have reported that prediabetes is associated with comorbidities that are traditionally considered as diabetes-related complications. In this issue, Schlesinger et al (https://doi.org/10.1007/s00125-021-05592-3) summarise and evaluate the existing evidence on prediabetes and health outcomes in an umbrella review of meta-analyses. The authors found that prediabetes is associated with an increased risk for all-cause mortality, cardiovascular diseases, chronic kidney disease, different types of cancer, and dementia, compared with people without prediabetes. They conclude that these observations emphasise the need for early prevention and management of prediabetes, for example by lifestyle interventions, to prevent diabetes-related complications.

Impact of insufficient sleep on dysregulated blood glucose control under standardised meal conditions

Neli Tsereteli, Raphael Vallat, Juan Fernandez-Tajes, Linda M. Delahanty, Jose M. Ordovas, David A. Drew, Ana M. Valdes, Nicola Segata, Andrew T. Chan, Jonathan Wolf, Sarah E. Berry, Matthew P. Walker, Timothy D. Spector, Paul W. Franks

Small in-patient studies and larger observational studies suggest that features of how we sleep may affect our metabolic health. In this issue, Tsereteli et al (https://doi.org/10.1007/s00125-021-05608-y) report data from the largest experimental study to date focusing on objectively assessed sleep and its impact on postprandial blood glucose following standardised breakfast meals. The authors show that poor sleep efficiency and later bedtime routines worsen blood glucose responses overall. The authors also show that at an individual level, sleep matters, as person-specific deviations from normal sleep patterns also impact the blood glucose response to breakfast. This was especially true when an oral glucose load was given as the breakfast meal, suggesting that the popular practice of consuming energy drinks after a poor night's sleep may be particularly detrimental for blood glucose regulation. The authors conclude that these findings underscore the importance of sleep in the optimal regulation of human metabolic health.

Relative leucocyte telomere length is associated with incident end-stage kidney disease and rapid decline of kidney function in type 2 diabetes: analysis from the Hong Kong Diabetes Register

Feifei Cheng, Andrea O. Luk, Hongjiang Wu, Claudia H. T. Tam, Cadmon K. P. Lim, Baoqi Fan, Guozhi Jiang, Luke Carroll, Aimin Yang, Eric S. H. Lau, Alex C. W. Ng, Heung Man Lee, Elaine Chow, Alice P. S. Kong, Anthony C. Keech, Mugdha V. Joglekar, Wing Yee So, Anandwardhan A. Hardikar, Juliana C. N. Chan, Alicia J. Jenkins, Ronald C. W. Ma

Telomere length shortening, representing reduction in the protective caps at the ends of our chromosomes, is known to be associated with biological ageing and different cardiomet-abolic diseases, although it is unclear whether it has prognostic significance for predicting kidney disease in diabetes. In this issue, Cheng et al (https://doi.org/10.1007/s00125-021-05613-1) report that in a large cohort of people with type 2 diabetes from Hong Kong, reduced telomere length in white blood cells was an independent predictor for decline in kidney function and future risk of kidney failure. The authors suggest that this effect was independent of other established risk factors for kidney dysfunction, and improves prediction beyond that provided by clinical factors alone. The authors conclude that these findings indicate telomere length shortening may be helpful in stratifying the future risk of kidney disease in people with diabetes.

Upregulation of HLA class II in pancreatic beta cells from organ donors with type 1 diabetes

Estefania Quesada-Masachs, Samuel Zilberman, Sakthi Rajendran, Tiffany Chu, Sara McArdle, William B. Kiosses, Jae-Hyun M. Lee, Burcak Yesildag, Mehdi A. Benkahla, Agnieszka Pawlowska, Madeleine Graef, Susanne Pfeiffer, Zbigniew Mikulski, Matthias von Herrath

For many decades, the question of whether HLA class II can be expressed by pancreatic beta cells has been controversial. In this issue, Quesada-Masachs et al (https://doi.org/10.1007/s00125-021-05619-9) report that HLA class II is upregulated in islets of pancreatic tissue sections from organ donors with type 1 diabetes: 28% of the beta cells from the patients with type 1 diabetes expressed HLA class II. Immunofluorescence microscopy was used to thoroughly quantify HLA class II in situ, using a machine learning approach. Furthermore, the authors report that healthy human islets stimulated with proinflammatory cytokines upregulate HLA class I and class II, as measured by quantitative fluorescent microscopy and RNA sequencing. The authors suggest that a crosstalk could exist between beta cells and CD4[+] T cells in type 1 diabetes, although further research is necessary to demonstrate this cellular communication and elucidate its biological role in disease initiation and progression.

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