JIM Introduction

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Obesity and type 2 diabetes: understanding the role of ethnicity

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The prevalence of type 2 diabetes varies considerably throughout the world. The causes of this variation are incompletely known but include ethnic differences in genetic susceptibility, obesity, body fat distribution, lifestyle and socio-economic/environmental conditions. The Journal of Internal Medicine symposium entitled 'Obesity and type 2 diabetes: Understanding the role of ethnicity' was held in Umeå, Sweden, on 11–12 September 2019 in order to present an up-to-date view on obesity and type 2 diabetes from varying perspectives and different geographical/ethnic populations. In addition, the role of genetics and interactions between genes and environment was discussed. The presentations related to Indigenous populations in Australia, sub-Saharan Africa and East Asia; Greenland Inuits, African Americans and Pima Indians in the United States; and African migrants in Europe. The meeting was attended by researchers from 12 different countries including delegates from South Korea, Australia, South Africa, Zimbabwe, Congo, the United States and the UK, as well as from many of the Nordic countries. The symposium included an interactive poster session, with presentations from research groups from different parts of the world.

The role of specific gene variants with profound impact on metabolic dysfunction, including insulin secretion, was clearly demonstrated by studies on Greenland Inuits (Torben Hansen, Copenhagen) and East Asians (Kyong Soo Park, Seoul). The studies of isolated populations are likely to identify common high-impact gene variants that are easy to investigate functionally and thereby provide novel biological insights that are generally relevant, even if the identified variants are population-specific [1–3]. In contrast, Hanieh Yoghootkar (Exeter/Luleå) presented data on 'favourable adiposity alleles' associated with higher subcutaneous fat but lower visceral fat in both South Asians and Europeans. Notably, South Asians have a lower number of favourable alleles, which may explain why South Asians have a higher risk of disease at lower body mass index (BMI) cut-offs (see Ref. [4] in this issue). Paul Franks (Lund) reviewed the evolutionary arguments and recent empirical data elucidating the role of human biological variation and interactions with the environment as driving forces behind the current obesity and diabetes epidemics. He also addressed the important topic of individual variation in risk factor susceptibility and possibilities for precision prevention in diabetes and obesity [5].

The development of obesity and body composition and its relation to the pathophysiology of type 2 diabetes in populations of Black African descent compared to those of European descent were explored in detail in two presentations by Barbara Gower (Birmingham, USA) and Julia Goedecke (Cape Town). Gower presented data to show that the predisposition to obesity within African Americans is closely related to their unique phenotype characterized by relative hyperinsulinaemia, in combination with normal insulin sensitivity and a high-glycaemic-load diet (see Ref. [6] in this issue). Further, she showed that African American women in particular may be prone to obesity due to a combination of hyperinsulinaemia and elevated estradiol levels. Goedecke discussed the pathophysiology of type 2 diabetes in Black Africans, highlighting the following conundrums: (i) Black Africans may to be more sensitive to the effect of

ectopic fat disposition on insulin sensitivity, or alternatively that ectopic fat is not an important mediator of type 2 diabetes in Black Africans; and (ii) hyperinsulinaemia that is observed early during the development of metabolic dysfunction eventually leading to type 2 diabetes is a predisposing factor for insulin resistance or is rather a compensatory mechanism secondary to insulin resistance (see Ref. [7] in this issue). The answers to these conundrums remain to be established and are currently active areas of research.

Importantly, a combination of genetic, socio-economic and lifestyle factors plays an important role in the development of obesity and type 2 diabetes. This was well illustrated by the data presented by Knowler, Agyemang and Brown. Studies based on data from the Pima Indians in Arizona, USA (Willian C Knowler, Phoenix), have given a sound basis for interventions aimed at addressing multiple risk factors for diabetes (mainly diet and physical activity) in large randomized controlled trials [8, 9]. Charles Agyemang (Rotterdam) clearly demonstrated the need for further research into factors that are driving the migrant health disadvantage in order to provide solid evidence for targeted culturally sensitive interventions [10]. Aboriginal and Torres Strait Islander inhabitants experience profound health disparities compared to the remainder of the Australian population, with a life expectancy 12–15 years less. Type 2 diabetes and cardiovascular diseases are the leading causes of death and account for almost 50% of this differential, as reported by Alex Brown (Adelaide). Genetic and environmental factors may well play a significant role in this development. Notably, cultural differences in diet, physical activity, perceptions of body size and body image are of major importance when designing lifestyle interventions in specific populations, as shown by Goedecke et al (see Ref. [7] in this issue).

In summary, although ethnicity affects the risk for both obesity and type 2 diabetes, there is no single common physiological basis for this. It is clear that genetic mutations in specific populations (e.g. South Korea, Greenland) contribute to specific parts of the pathophysiology in diabetes development (e.g. beta cell responsiveness). It is critical to consider ethnicity when evaluating data or planning an intervention in lifestyle-related disorders. In particular, research in a range of Indigenous and other ethnic minority populations across the globe will continue to add significantly to our understanding of the susceptibility and pathophysiology of obesity and type 2 diabetes. In this, we need to embrace new research models such as the role of context and the role of migration illustrated by the RODAM study and pay more attention to other relevant factors such as early-life programming/epigenetics. The results of these studies need to be translated into strategies for the effective prevention and management of these diseases.

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Conflict of interest statement

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