

# Genes versus environment in diabetes

*This short feature is based on a thought-provoking debate that took place at the 6<sup>th</sup> Scottish Conference of the Primary Care Diabetes Society, 30 October 2013, Crowne Plaza, Glasgow*

Professor Naveed Sattar was assigned the task of arguing the case that diabetes is more about genes than the environment.

Professor Sattar's argument opened with a comparison of rates of obesity (which can be viewed as the intermediate outcome of the obesogenic environment) and diabetes. He observed that the top-10 list for obesity (which is headed by the US, Mexico and Scotland) shared not a single country with the equivalent list for diabetes. At the top of the latter sits Tokelau, the Federated States of Micronesia, and Kiribati ([www.idf.org/diabetesatlas](http://www.idf.org/diabetesatlas) [accessed 03.12.13]).

Professor Sattar next turned to examine how BMI cut-offs for equivalent metabolic risk vary dramatically between ethnic groups. Assessed against the metabolic risk indicated by a BMI of 30 kg/m<sup>2</sup> in a person of European ethnicity, in relation to glucose control, equivalent BMIs have

been calculated as 20.6 and 21.0 kg/m<sup>2</sup> in people of Chinese and South Asian ethnicity, respectively (Razak et al, 2007).

Various other analyses were reviewed that point to the large bearing that ethnicity, and thus genetic background, has on metabolic risk.

Professor Sattar went on to remind the audience that over half of the elements contained within standard type 2 diabetes risk scores have a strong genetic basis (including ethnicity, family history of diabetes and gender). He then concluded with a personal example to illustrate the impact of genetics on diabetes risk. Based on his own calculations, as a person of South Asian ethnicity he has an approximately 10-fold higher risk than friends of his who have the same BMI but are of European ethnicity. ■

Razak F, Anand SS, Shannon H (2007) Defining obesity cut points in a multiethnic population. *Circulation* **115**: 2111–8



**Naveed Sattar**

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Dr Jason Gill made the case for the importance of environment. He began with a summary of the argument he was going to present: in a “good environment”, “good genes” generally equate to “low risk” and “bad genes” to “low-ish risk”, while in a “bad environment”, “good genes” equate to “increased risk” and “bad genes” to “high risk”. Expressed differently: “changing the environment can overcome the adverse effect of ‘bad genes.’”

A key paper that he cited in support of the importance of environment was a systematic review by Bao et al (2013), in which it was concluded that “the addition of genome-wide association markers into conventional risk models produced little improvement in predictive performance.”

Dr Gill next explored how prevalence of diabetes in each State in the US closely tracked rising levels of obesity, according to data from the Centers for Disease Control and Prevention (<http://apps.nccd.cdc.gov/DDTSTRS/default.aspx> [accessed 03.12.13]).

Additional data were provided on the extent of variation in diabetes prevalence between, for instance, rural India and urban India, as an indication of the role of environmental factors in the epidemiology of the condition (Hall et al, 2008).

Dr Gill finished his talk with a number of further key points supporting the importance of the role of the environment. These included that the dramatic increase in diabetes prevalence over the past half-century must be down to changes in environmental factors, rather than genetic aspects, as well as a reassuring reminder that diabetes prevention is in the control of the individual (irrespective of his or her genes) if a favourable lifestyle is adopted. ■

Bao W, Hu FB, Rong S et al (2013) Predicting risk of type 2 diabetes mellitus with genetic risk models on the basis of established genome-wide association markers: a systematic review. *Am J Epidemiol* **178**: 1197–207

Hall LM, Sattar N, Gill JM (2008) Risk of metabolic and vascular disease in South Asians: potential mechanisms for increased insulin resistance. *Future Lipidology* **3**: 411–24



**Jason Gill**

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