Reply to: What is the mechanism behind the association between autoantibodies against GAD65 and high body mass index?

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LETTER TO THE EDITOR

Reply to: What is the mechanism behind the association between autoantibodies against GAD65 and high body mass index?

DEAR SIR,

Dr Rolandson infers that we reported an association between high body mass index (BMI) and autoantibodies against glutamic acid decarboxylase, isoform 65 (GADA) in our recent paper [1]. This was not the case. Actually, we reported the following. Amongst patients with islet antibodies [islet cell antibodies (ICA), protein tyrosine phosphatase-like protein antibodies (IA–2A) and/or GADA] higher BMI at diagnosis were noticed in those with preserved β-cell function 8 years after diagnosis when compared with those without [22.7 (15.8–35.1) vs. 20.5 (17.2–38.2) kg m^{-2}; $P = 0.0003$]. As the question has been raised, we have now in detail analysed the associations between GADA and BMI in the study. This analysis showed the opposite of that suggested by Dr Rolandson. Eight years after diagnosis, patients with GADA had a significantly lower mean BMI than those without GADA (24.0 ± 0.4 vs. 25.0 ± 0.2 kg m^{-2}; $P = 0.02$). At diagnosis, no significant correlations (significant r values) were found between GADA concentrations or GADA positive/negative versus BMI. In our paper we suggested two explanations for the observation that normal BMI at diagnosis of diabetes is associated with preserved β-cell function 8 years later. Low BMI at diagnosis identifies patients with more severe ketoacidosis and a higher loss of β-cells before diagnosis than those with a normal BMI. Another option is that patients with normal BMI primarily have a higher amount of β-cells than those with low BMI as seen in ob/ob mice [2]. Indeed, in a second and recently published study, we demonstrated a similar phenomenon. Compared with low BMI, normal weight (BMI $\geq$20 kg m^{-2}) at diagnosis promotes remission in islet antibody-positive patients with type 1 diabetes [3].

Conflict of interest statement

No conflict of interest was declared.

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References

1 Schölin A, Björklund L, Borg H *et al*. Diabetes Incidence Study in Sweden. Islet antibodies and remaining beta-cell function 8 years after diagnosis of diabetes in young adults: a prospective follow-up of the nationwide Diabetes Incidence Study in Sweden. *J Intern Med* 2004; **255:** 384–91.

2 Starich GH, Zafirova M, Jablenska R, Petkov P, Lardinois CK. A morphological and immunohistochemical investigation of endocrine pancreata from obese ob+/ob+ mice. *Acta Histochem* 1991; **90:** 93–101.

3 Schölin A, Törn C, Nyström L *et al*. Normal weight promotes remission and low number of islet antibodies prolong the duration of remission in Type 1 diabetes. *Diabet Med* 2004; **21:** 447–55.

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