LUND UNIVERSITY

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

Schölin, Anna; Sundkvist, Göran

Published in:
Journal of Internal Medicine

DOI:
[10.1111/j.1365-2796.2004.01385.x](https://doi.org/10.1111/j.1365-2796.2004.01385.x)

2004

[Link to publication](#)

Citation for published version (APA):
Schölin, A., & Sundkvist, G. (2004). Reply to: What is the mechanism behind the association between autoantibodies against GAD65 and high body mass index? *Journal of Internal Medicine*, 256(3), 264-264. <https://doi.org/10.1111/j.1365-2796.2004.01385.x>

Total number of authors:
2

General rights

Unless other specific re-use rights are stated the following general rights apply:
Copyright and moral rights for the publications made accessible in the public portal are retained by the authors and/or other copyright owners and it is a condition of accessing publications that users recognise and abide by the legal requirements associated with these rights.

- Users may download and print one copy of any publication from the public portal for the purpose of private study or research.
- You may not further distribute the material or use it for any profit-making activity or commercial gain
- You may freely distribute the URL identifying the publication in the public portal

Read more about Creative commons licenses: <https://creativecommons.org/licenses/>

Take down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00

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 ≥ 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.

ANNA SCHÖLIN
GÖRAN SUNDKVIST
Department of Endocrinology,
University of Lund,
Malmö, Sweden

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.

Correspondence: Professor Göran Sundkvist, Department of Endocrinology, University of Lund, 205 02 Malmö, Sweden.
(fax: +46-40-33 62 01; e-mail: goran.sundkvist@endo.mas.lu.se).