Comment on: Keenan et al. (2010) Residual Insulin Production and Pancreatic β-Cell Turnover After 50 Years of Diabetes: Joslin Medalist Study. Diabetes 2010;59:2846–2853

Kristina I. Rother[1] and David M. Harlan[2]

We are pleased to find our previously published results (1,2) confirmed by the report of Keenan et al. (3). In their cohort of individuals with very long-standing type 1 diabetes, 67.4% of patients had residual endogenous insulin production, and in nine postmortem examinations of pancreatic tissue, insulin positive cells were found. However, we respectfully disagree that this is a surprising finding. We recently reported that many patients with long-standing type 1 diabetes maintain the capacity to secrete small amounts of insulin in a regulated manner (1,2). Their residual β-cells responded to both physiological (mixed meal) and pharmacological (arginine) stimuli. We further tested the hypothesis that expanding this residual β-cell mass might be a promising approach to treatment (1). Though our experimental conditions did not result in an improvement of β-cell mass or function, we strongly believe that expansion of remaining β-cells is a promising approach for future research.

Might the C-peptide results of the Joslin Medalists have been an underestimate of their true insulin secretory capacity? We had observed that the remaining β-cells' insulin secretion could be suppressed by exogenous insulin administration (1). Therefore, we wonder whether Keenan et al. would have found more C-peptide had the investigators tested the individuals under different conditions (some patients received insulin injections immediately before the mixed meals, which might have suppressed their C-peptide levels). The merit of the Joslin Medalist Study should, however, in no way be denigrated, especially because of its unique collection of clinical data and comparison with postmortem results.

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From the [1]Clinical Endocrinology Branch, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, Maryland; and the [2]Diabetes Center of Excellence, University of Massachusetts, Worcester, Massachusetts.
Corresponding author: Kristina I. Rother, kr58q@nih.gov.
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