higher glycemic threshold), as recently suggested by Cryer (2) and others, seems to me to be both unnecessarily confusing and semantically incorrect. Lower may mean higher in golf and politics, but not in describing glycemic *thresholds* for counterregulatory responses.

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Reply

Blakiston's Gould Medical Dictionary defines *threshold* as "the lower limit of stimulus capable of . . . evoking a response" (1).

Thus, if a greater hypoglycemic stimulus (i.e., a lower plasma glucose concentration) is required to elicit a response, then the glycemic threshold for that response is higher or elevated. If a lesser hypoglycemic stimulus (i.e. a higher plasma glucose concentration) elicits a response, then the threshold is lower or reduced.

This is the rationale for my current use of the terms elevated or reduced glycemic thresholds (2). Unfortunately, it has not always been so (3). However, to paraphrase Confucius, a person who makes a mistake and fails to correct that mistake has made another mistake. The fundamental point, of course, is that all authors must define their terminology clearly when discussing glycemic thresholds.

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Difference Between Islet Cell Antibodies and Autoantibodies to Glutamic Acid Decarboxylase–Containing Purkinje Cells in IDDM

The occurrence of autoantibodies to GABA-ergic neurons and pancreatic β-cells was reported in stiff-man syndrome and in several patients with IDDM (1). They were directed against GAD in β-cells and in a subpopulation of nerve cells (i.e., Purkinje cells), and they have been found to be identical to the 64K antibodies related to the development of IDDM (2). A new question was raised regarding the correspondence of the autoantibody against GAD (the 64K antibody) with ICA, which is another marker of active pancreatic β-cell damage in IDDM (3,4). This is because immunofluorescence studies demonstrated that the GAD antibody reacted homogenously to β-cell cytoplasm with a similar pattern to ICA (2). In this study, the possible differences between the target antigens of ICA and the autoantibodies to GABA-ergic neurons in IDDM were examined.

Seventy-five subjects were studied, including 14 with recent-onset (<3 mo) IDDM (8 men, 6 women, mean age 23 yr), 11 with ICA^+ NIDDM, (8 men, 3 women, mean age 48 yr, duration 2.3 yr), 23 with ICA^- NIDDM, (12 men, 11 women, mean age 45 yr, duration 2.5 yr), 1 with stiff-man syndrome (1 male, mean age 61 yr, duration 1 yr), and 26 normal control subjects (16 men, 10 women, mean age 32 yr). The titers of autoantibodies to GABA-ergic neurons were assayed by reactivity to unfixed human cerebellar GAD-containing Purkinje cells using indirect immunofluorescence. ICA was assayed by the sensitive method previously reported (5).

Positive immunostaining of GAD-containing Purkinje cells was observed in 29% (4 of 14) of recent-onset IDDM patients and in 18% (2 of 11) of ICA^+ NIDDM patients, with a titer ranging from 1:20–1:40 (Fig. 1). Purkinje cell autoantibody was positive in the stiff-man syndrome patient with a titer of 1:80, but was negative in the 23 ICA^- NIDDM patients and the 26 normal control subjects. All recent-onset IDDM patients were positive for ICA, with a titer ranging from 5 to 10,240 JDF U. The stiff-man syndrome patient was negative for ICA. The titers of Purkinje cell auto-