uing acarbose, when serum fructosamine levels and 24-h urinary glucose excretion did not change compared with that during acarbose therapy. 1,5-AG is actively absorbed in the intestine (5) and actively transported in the renal reabsorption process (6). Daily 1,5-AG balance in nondiabetic subjects is constant (7), but serum 1,5-AG concentration is dominantly affected by the amount of urinary glucose (1). On the basis of the above information, and as mentioned by Sakane et al., different effects of acarbose on serum 1,5-AG may be due to reduction of absorption of 1,5-AG in the intestine via inhibiting α-amylase, although this point remains to be clarified. On the other hand, it is unlikely that metabolites of acarbose might inhibit reabsorption of 1,5-AG in renal tubules because only 1–2% of the orally administered dose of acarbose is absorbed in active form (8). Serum 1,5-AG is useful for evaluating current glycemic control in short-term response to voglibose (3). During acarbose therapy, however, it is underestimated for monitoring the glycemic status and should be used in cooperation with other markers, such as fructosamine.

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# Revised Etiologic Classification of Diabetes

The recently published comprehensive Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (1) is most welcome and timely because it reflects progress in the understanding of the etiology and pathogenesis of diabetes. I would like to comment on section III.A. of Table 1, which addresses other specific types of diabetes and the genetic defects of β-cell function, and the accompanying text on p. 1187. While correct, the statement in the second sentence that "these forms of diabetes are frequently characterized by onset of mild hyperglycemia at an early age (generally before age 25 years)" may perpetuate the misconception that maturity-onset diabetes of the young (MODY) is generally characterized by mild hyperglycemia. This pertains to the nonprogressive diabetes or impaired glucose tolerance associated with mutations of the glucokinase gene on chromosome 7 (MODY2) (2). On the other hand, diabetes associated with either mutations of hepatocyte nuclear factor (HNF)-1α on chromosome 12 (MODY3) or mutations of HNF-4α on chromosome 20 (MODY1) are forms of the disease associated with fasting hyperglycemia at diagnosis or on follow-up in up to 80% of patients, with insulin requirement in up to 30% of patients, with need for oral hypoglycemic agents in the majority of the remaining patients, and with microvascular complications in a frequency similar to that seen in type 2 diabetes (3–5). If "mild hyperglycemia" refers to the state at "onset," the same pertains to type 2 diabetes if the diagnosis is made at an early stage in the natural history of the disease.

In an etiologic classification, the use of chromosome number and gene give scientific precision to the loci of the mutations. In referring to the phenotypic expression of the various forms of these genetic defects of β-cell function, or to the group of these disorders (excluding mitochondrial DNA or other defects), the identification of the specific types by the repetitive use of chromosome and gene may be cumbersome, while the present designations of MODY1, MODY2, MODY3, etc. . . ., or MODY, respectively, may be more convenient. Thus, in Table 1, elimination of "formerly" within the parentheses would be appropriate.

Similarly, the designation of "type 1 diabetes," as used in Table 1 and text, is more convenient than the more exact scientific and etiologic term "Diabetes due to immune-mediated β-cell destruction."

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