BIOCHEMICAL LETTERS

Nomenclature for mammalian glucokinase

The major hexokinase of the mammalian liver, hexokinase D or type VI, is commonly referred to as 'glucokinase'. This enzyme, however, does not display an absolute substrate specificity for glucose, but is actually able to phosphorylate fructose when the latter is supplied at high concentration [1]. The enzyme is therefore not a glucokinase in the strict sense of the term, but rather a hexokinase. In 1984, the IUB-IUPAC Joint Commission on Biochemical Nomenclature [2] issued the following recommendation: (i) the name ATP: D-glucose 6-phosphotransferase and EC number 2.7.1.2 should be reserved for enzymes highly specific for glucose such as those found in bacteria and lower eukaryotes, and (ii) 'glucokinase' of vertebrate liver should be assigned the name ATP: D-hexose 6-phosphotransferase and EC number 2.7.1.1. The second point would evidently apply also to the enzyme of the islet of Langerhans, closely related to hepatic 'glucokinase' and expressed from the same gene [3,4].

Unfortunately, these recommendations are widely ignored. In a recent paper from our laboratories published in this journal [5], the improper name and EC number were assigned to rat liver 'glucokinase'. This occurred as a result of an editorial change to our manuscript that we did not recognize until after publication. We regret this oversight and wish to point out that, for our part, we fully endorse the IUB-IUPAC recommendation cited above. Indeed, recent cDNA sequence data support this recommendation, since they unequivocally establish an evolutionary relationship between hepatic 'glucokinase' and the hexokinase family of enzymes [6,7]. It would be desirable that all authors adopt the recommended nomenclature and designate the mammalian 'glucokinase' as an ATP: D-hexose 6-phosphotransferase (EC 2.7.1.1).

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The *N*-acetylglutamate content and *N*-acetylglutamate synthase activity of human liver

In a recent article [1], Tuchman & Holzknicht present data on the *N*-acetylglutamate content and *N*-acetylglutamate synthase activity of human liver, pointing out that detailed studies of these variables in human liver had not been reported. They briefly discuss the limitations of previously used methods for the measurement of *N*-acetylglutamate and the advantages of their new method, described in [2].

Using their method, Tuchman & Holzknicht [1] found that the *N*-acetylglutamate content of 16 human livers varied over an 8.8-fold range, between 6.8 and 59.7 nmol/g wet wt. The *N*-acetylglutamate synthase activity varied similarly, between 44.5 and 374.5 nmol/min per g wet wt. (see Table 1 of [1]). These authors state "No correlation was found between hepatic *N*-acetylglutamate concentrations and the respective maximal enzymic activities of *N*-acetylglutamate synthase *in vitro*" [1]. On this basis they propose "The marked variability in this system among individual livers may reflect its regulatory role in ureagenesis" and later conclude "This finding suggests that the regulation of *N*-acetylglutamate synthase activity may be post-translational, involving activators and/or inhibitors" [1].

The method of Tuchman & Holzknicht [1,2] appears to be very good for the measurement of *N*-acetylglutamate in simple solutions. The values for this compound in liver given in [1], however, are open to question because the authors have not reported a proper study of the recovery of *N*-acetylglutamate from tissue samples (see [1,2]), e.g. of the recovery of *N*-acetylglutamate added to the tissue samples themselves, at a proper range of concentrations, and carried over the entire procedure.

Good methods for the measurement of *N*-acetylglutamate in simple solutions and in extracts of tissue or of subcellular components have been available for some time. We have used extensively [3,4] two methods based on kinetic assays of *N*-acetylglutamate [3,5,6], and the method of Alonso & Rubio [7] with minor modifications (C.-W. Cheung & L. Rajman, unpublished work); these have the advantage that they do not require gas chromatography-mass spectrometry [1], a technology not available to all. Nonetheless, a new, sensitive, method based on different analytical properties of the compound will be a welcome addition once the applicability of the method to the analysis of tissue samples is established.

We are compelled to question the significance of the data in [1] on additional grounds, and some of the authors' interpretations as well. Assuming for a moment that the data are valid, we think that the 'lack of correlation' between the *N*-acetylglutamate content and the activity *in vitro* of *N*-acetylglutamate synthase of liver reported in [1] does not necessarily suggest a post-translational regulation of this enzyme, as the authors propose; it is equally possible that the degradation of *N*-acetylglutamate is regulated, and this should not be excluded *a priori*.

Received 3 December 1990