an angiogram to confirm the presence or absence of an aneurysm. This patient was subsequently found to have a CSF infection with associated inflammation; no aneurysm was detected.

Because of the excellent correlation, we are now using the new method because it is simpler, thereby making the method potentially less prone to error. We have used the old method for >3 years now in >250 analyses.

Vitamin C and Glycohemoglobin Revisited

To the Editor:

Blood glucose (BG) concentrations averaged over a 2- to 3-month period are represented by blood glycohemoglobin (GHb) as a percentage of total hemoglobin. Strict glycemic control is important to the successful outcome of diabetic pregnancy and in avoiding development of many diabetic complications (1). Hyperglycemia in nondiabetes is a major risk factor in cardiovascular disease and cancer (2). Thus, knowledge of an individual’s true GHb value is vital. This need has prompted continuing investigations of potential interferences in GHb assays (3, 4), including conflicting reports on the effect of vitamin C on GHb (5–7).

As a part of our research on effects of dietary variables on tumor growth, two groups of 18 mice each were provided either ascorbic acid (AA; vitamin C) in drinking water (2.5 g/L) or unsupplemented water. Blood glucose was measured six times during the 2-month study (Glucometer; Bayer Diagnostics). We observed no differences in BG between the groups. At the beginning of the study, baseline BG concentrations (mean ± SD) for the non-AA and AA groups were 82 ± 16 and 79 ± 15 mg/100 mL, respectively. The BGs at the end of the study were, respectively, 90 ± 16 and 87 ± 18 mg/100 mL. Also at the end of the study, GHb by affinity chromatography (Glycostest; Pierce) was determined. For this variable, the AA group exhibited a significantly lower value (i.e., GHb, 4.39% ± 0.78% for non-AA mice and 3.39% ± 0.60% for the AA-supplemented mice; \( t = 4.324; P = 0.0001 \)).

AA may lead to interferences in GHb assays, particularly in some based on charge separation (e.g., electrophoresis and ion exchange), where a positive interference has been observed (6). The affinity-chromatography method we used, however, was not affected. This suggests that the AA-associated decrease in GHb reflected a genuine in vivo decrease in glycation.

In 1988, Ely et al. (5) reported antagonism of hemoglobin glycation by AA in animals and humans. Since then, two contradictory reports of the effect of AA supplementation (750–1500 mg/day) on GHb in humans have appeared (6, 7). Using affinity chromatography, Davie et al. (6) found an 18% decrease in GHb, whereas Weykamp et al. (7) found no significant change. Our data show a 23% reduction in GHb in mice consuming ∼7.5 mg of AA/day. Thus, the important question of whether GHb measurements accurately represent average BG in persons who take AA supplements remains unanswered.

AA is the most commonly consumed nutritional supplement after multivitamins (8), and in the western United States, >11% of adults take an AA supplement daily. Laboratory, epidemiologic, and intervention studies suggest that antioxidant vitamins, especially AA, have long-term benefits in attenuating the progression of diabetic complications, and diabetics are encouraged to take AA. In light of these facts and the importance of BG in other aspects of human health, including immunity and aging, the uncertainty regarding the influence of AA on GHb demands further investigation.

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References


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