Distribution of Vitamin B₆ Deficiency in University Students¹

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Summary The basal specific activities (S.A.; μmol of pyruvate/hr/ 10^8 erythrocytes) and the % deficiencies of activity of the glutamic oxaloacetic transaminase of the erythrocytes (EGOT) of 174 university students was 0.28 ± 0.05 and $33 \pm 9\%$, respectively. There was a negative correlation, $r = -0.65$ ($p < 0.001$), between the mean basal S.A. and the mean % def. (i.e., the lower the S.A., the higher the % def.). There were students with low basal S.A.'s who showed symptoms of carpal tunnel syndrome. On the basis of these data, 93% of 174 students had deficiencies of 20% and higher which was potentially correctable by oral pyridoxine; these students had B₆-deficient diets. On the basis that a normal basal S.A. may be 0.7, and that the maximum S.A. (0.45) for all 174 students is about 65% of 0.7, all 174 students had varying vitamin B₆ deficiency, and their diets provided inadequate amounts of this vitamin.

Key Words vitamin B₆, deficiency, transaminase (GOT)

In studies of human deficiencies of vitamin B₆, notwithstanding the assays being based upon the determinations of a member of the vitamin B₆ group as a chemical substance or upon determinations of the activities of vitamin B₆ enzymes, there is always the need for a control group of individuals presumed to be normal. Ideally, the normal control group would consist of individuals who have a zero deficiency of vitamin B₆.

A normal control group may consist of relatively young individuals who have no known illness and are a judged "healthy." University students are relatively young and are generally "healthy," and students in Nutrition and Home Economics are particularly desirable as controls, because of their training in nutrition and vitamins.

Over about two years, we have analyzed blood samples for detection and measurement of human deficiencies of vitamin B₆ from 174 students consisting of

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158 females and 16 males. The predominance of females was because of the background of Nutrition and Home Economics.

The detection and accurate measurement of deficiencies of vitamin B₆ was done by a dual enzymic assay based upon the principle of the unsaturation and saturation of the apoenzyme of a transaminase with the coenzyme (pyridoxal 5'-phosphate). In 1976, Azuma *et al.* (1) used this enzyme methodology to measure levels of vitamin B₆, but only for a group of 21 students and older adults. Since this group perhaps consisted of too few individuals for proper evaluation, the control group in this study has been extended to 174 university students.

It was not the purpose of this study to control or record the dietary background of such students, but rather to appraise the nutritional status of vitamin B₆ while living and serving as controls.

METHODS

Ten-ml blood samples were drawn from the cubital vein of the subjects into "lavender" vacutainers containing 14 mg of EDTA. The differential enzyme assay to measure the specific activities of the glutamic oxaloacetic transaminase (GOT), including washing of the erythrocytes to minimize contamination by plasma and leucocytes, was conducted as described by Kishi *et al.* (4). The specific activity (S.A.) is expressed as μ moles of pyruvate formed per hour/ 10^8 erythrocytes. The % deficiency of the activity of EGOT was calculated by subtracting the basal S.A. from the S.A. determined in the presence of pyridoxal 5'-phosphate, and dividing by the S.A. in the presence of pyridoxal phosphate, times 100. The data were calculated as mean values $\pm$ standard deviation.

Fig. 1. The distribution of basal specific activities of EGOT.

RESULTS

Figure 1 shows the distribution of the basal specific activities of EGOT of all 174 students. The "bell-shape" of the distribution is evident, as expected. The range of specific activity for the 174 students was from 0.17 to 0.45, and the mean value was 0.28 ± 0.05 .

The data in Fig. 2 show that the % deficiency of activity of EGOT ranged from 10 to 58%. The mean value of the % deficiency was $33 \pm 9\%$.

A negative correlation between the basal specific activity and the % deficiency is evident from the data as plotted in Fig. 3. The correlation is significant; $r = -0.65$ ($p < 0.001$); $y = -108x + 63$. The lower the S.A., the higher the % deficiency becomes.

Fig. 2. The distribution of per cent deficiencies of EGOT.

Fig. 3. Correlation between % deficiency and basal S.A.

$$r = -0.65 \ (p < 0.001); \ y = -108x + 63$$

DISCUSSION

All 174 students reported that no vitamin formulations were being taken, vitamin B₆ status thus being determined solely by diet.

Figure 1 reveals that 53/174 students, or about 30%, had a basal specific activity which ranged from the lowest value of 0.17, to 0.25.

Shizukuishi *et al.* (7) reported that two of eight university students (included in this group of 174) having a mean basal specific activity of 0.25 ± 0.03 had mild but distinct symptoms of carpal tunnel syndrome. One male student had nocturnal paresthesia in an arm and one female student had periodic tingling in the fingers; these symptoms characterize the carpal tunnel syndrome as evaluated by Phalen (6) and reviewed by Taylor (8). The established association of a deficiency of vitamin B₆ with carpal tunnel syndrome led to the conclusion that this syndrome is a deficiency disease of vitamin B₆, which may be effectively treated by oral pyridoxine; Ellis, Folkers *et al.* (2). On this basis, it is believed that some of the 63 students whose basal specific activity is 0.25 or lower may develop overt and painful symptoms of carpal tunnel syndrome by the age of 40, which a common age for symptoms of this neurological disease to increase to a painful state (9).

A mean value of 0.28 ± 0.05 for the basal S.A. of all the students is scarcely above an S.A. of 0.25 at which or below, symptoms of the carpal tunnel syndrome may become evident in due time. These values may be compared with those for a group of patients with severe carpal tunnel syndrome, which consisted of 5 males and 3 females ranging in age from 25 to 53 (7). The mean basal S.A. of these eight patients with carpal tunnel syndrome was 0.24 ± 0.04 .

It may be concluded that at least 30% of these 174 students had a deficiency of vitamin B₆. The maximum value of the basal specific activity for these 174 students was 0.45. Only 5/174, or 3%, of all students had a basal S.A. of 0.40–0.45.

The crucial question is what the normal value of the S.A. of EGOT for a state of zero deficiency of vitamin B₆ is. The data in Fig. 2 show that 163 of the 174 students had deficiencies of >20% (21–58%); consequently, the diets of 93% of the students caused a state of B₆ deficiency in their metabolism. Previous studies (1) have shown that oral supplementation of pyridoxine corrects within a few days the unsaturation by pyridoxal 5'-phosphate of the receptors of the apoenzyme. Of greater significance was the observation that the presence of the coenzyme over and above that required for saturation of the apoenzyme, causes a continuing increase over 6 weeks to a level of S.A. approaching 0.7. Since these are natural events in young and presumably "healthy" individuals, it seems that *ca.* 0.7 is the normal value of the S.A. of EGOT for a state of zero deficiency of vitamin B₆ in an individual.

Other data supporting the significance of 0.7 as a normal value stems from the study of the administration of 50–300 mg of pyridoxine to 12 individuals for *ca.* 1–15 years; the mean value of S.A. was 0.69 ± 0.09 ($n=36$) (3). Related data stem from the analysis of cord blood of newborn infants; the mean value of S.A. was 0.71 ± 0.21 ($n=49$) (5). The S.A. of EGOT for adults on pyridoxine supplemen-

tation was identical to that of the cord blood of newborn infants.

On the basis of 0.7 for the normal value of S.A. of EGOT for a human state of zero deficiency of vitamin B₆, in conjunction with the observation that the maximum S.A. (0.45) of all 174 students is only about 65% of 0.7, every one of the 174 students had a deficiency of vitamin B₆.

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