

Influence of adrenalectomy on vitamin B₆ status

S. David and G.D. Kalyankar

Department of Biochemistry & Biophysics, St. John's Medical College, Bangalore-560034 (India), June 6, 1982

Summary. Adrenalectomy results in depletion of vitamin B₆ stores. This depletion is rapid and significant in liver, as compared to brain. When the adrenalectomized rats are fed with excess vitamin B₆ or injected with corticosterone, the normal vitamin levels are maintained. Thus, it is possible that adrenocortical hormones help in the retention of vitamin B₆ in the body.

It has been generally recognized that dietary deficiency of vitamin B₆ results in hypertrophy of the adrenals¹⁻³. Cytochemically, the zona fasciculata has been shown to undergo a progressive loss. Administration of vitamin B₆ produced rapid reversal of these cytochemical changes. Stebbins⁴ and Guggenheim⁵ demonstrated an impaired water balance in vitamin B₆-deficient animals. The abnormality was readily corrected by administering either vitamin B₆ or adrenal cortical hormones.

Greengard and Gordon⁶ have reported decreased tyrosine aminotransferase activity in adrenalectomized (Adx) animals, and that administration of large doses of vitamin B₆ caused restoration of the activity. Nakahara and Sakamoto⁷ observed decreased level of pyridoxine phosphate oxidase in Adx rats, and the activity was restored to normal after glucocorticoid injection. Studies from this laboratory⁸⁻¹⁰ have shown that many abnormalities seen in Adx animals, such as diuresis, negative nitrogen balance, increased ammonia and urea excretion, electrolyte imbalance, and decreased activity of liver aminotransferases, are minimized when the adrenalectomized rats are fed a diet with added vitamin B₆ (2 mg/rat/day). Thus, there seems to be a close association between the functions of adrenocortical hormones and vitamin B₆. In view of these observations, it was planned to study the levels of total vitamin B₆ in tissues such as liver and cerebral cortex in Adx rats.

Materials and methods. Bilateral adrenalectomy was carried out on adult male albino rats (200–225 g b.wt) under ether anesthesia; rats were maintained on saline for drinking and Hind Lever rat feed given ad libitum.

A group of Adx rats was injected i.p. with corticosterone acetate, 2.5 mg/rat, immediately after operation and at the end of 48, 96 and 144 h after Adx. Another group of Adx rats was fed with excess vitamin B₆ (2.0 mg/rat/day) applied as a solution to the food pellets. On the 8th day after the operation, the animals were sacrificed. Total vitamin B₆ content of liver and cerebral cortex was estimated according to the method described by Haskell and Snell¹¹ using *S. carlsbergensis* (ATCC 9080) as the test organism.

Table 1. Total B₆ content of liver

Group treatment	µg/g fresh tissue	µg/liver
1 Sham	10.09 ± 0.78	65.54 ± 7.46
2 Adx	7.45 ± 0.98*	39.54 ± 5.55*
3 Adx + corticosterone	9.47 ± 0.37	68.55 ± 6.05
4 Adx + vitamin B ₆	11.61 ± 0.98	53.50 ± 3.99

Values are mean ± SE from 6 animals in each group. *p < 0.001, compared to sham.

Table 2. Total B₆ content of cerebral cortex

Group treatment	µg/g fresh tissue
1 Sham	3.0 ± 0.62
2 Adx	2.86 ± 0.66
3 Adx + corticosterone	3.06 ± 0.24
4 Adx + vitamin B ₆	3.10 ± 0.88

Values are mean ± SE from 6 animals in each group.

Results and discussion. Comparative data on vitamin B₆ levels in liver, under various experimental conditions, are presented in table 1. There is a significant decrease in vitamin B₆ content in Adx rats. This is kept to normal level by either injecting the Adx animals with corticosterone or by feeding a diet with excess vitamin B₆ added. However, under the conditions studied, corticosterone seems to be superior to vitamin feeding. In the case of the cerebral cortex the vitamin content is marginally altered in different groups (table 2). From table 1, it seems possible that adrenocortical insufficiency results in the depletion of vitamin B₆ stores in liver. Ting-kai Li et al.¹² have shown that binding of pyridoxal-5'-phosphate to proteins protects the coenzyme from vitamin B₆ catabolizing enzymes. Further, Spector and Greenwald¹³, and Mulligan and Snell¹⁴ have shown that pyridoxal kinase plays a 'trapping' role in that the diffusible nonphosphorylated vitamin is converted intracellularly to a poorly diffusible phosphorylated form. Both pyridoxal kinase and pyridoxine-5'-PO₄ oxidase, the enzymes responsible for the formation of pyridoxal-5'-phosphate, under the influence of adrenocortical hormones, may play a role in the retention of vitamin B₆ in the body.

It is also known that the turnover of vitamin B₆ is considerably faster in the liver as compared to the brain^{15,16}, and possibly the longer retention of the steroids by the brain may have led to the differences observed in liver and brain of Adx rats.

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