

A Study on the Role of Thyroid Hormone in Regulating Serum Sodium Concentrations

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Summary

Thyroid hormone regulates the metabolic activity of almost all body tissues. However, studies on its effect on electrolyte balance has been far less compared to reports on energy metabolism. Clinically some patients with hypothyroidism presents hyponatremia. Thus I attempted to study the role of thyroid hormone in electrolyte regulation with emphasis on sodium. Last year I reported an exaggerated response of antidiuretic hormone (ADH) to various stimuli in experimental hypothyroidism(#8918). In this paper I studied the role of thyroid hormone in the regulation of Na/K ATPase in kidney.

Thyroxine(T4) or 3,5,3'-L-triiodothyronine(T3) treatment increased and hypothyroidism decreased rat renal Na/K ATPase. Simultaneous injection of iopanoic acid completely abolished the effect of T4. Iopanoic acid is a competitive inhibitor of 5'-deiodinase, which converts T4 to T3. Current concepts hold that T4 is merely a prohormone and T3 is the active form. Most of circulating T3 is derived from conversion from T4 rather than secretion from thyroid gland. Thus above data shows the importance of 5'-deiodinase in inducing Na/K ATPase.

Activity of 5'-deiodinase seems to be under the control of other hormones and neural tones, which differ from one tissue to another. Curiously regulatory mechanism of renal 5'-deiodinase has been little studied.

Chronic sodium loading decreased and furosemide administration increased renal 5'-deiodinase. This decreased enzyme activity is not due to accompanied decreased food intake, because 3 days complete fasting decreased hepatic 5'-deiodinase but had no effect on renal 5'-deiodinase and sodium overload had no effect on 5'-deiodinase in liver.

Previous studies on the role of thyroid hormone in regulating Na/K ATPase activity does not take into account the importance of 5'-deiodination in expressing the hormone action. Current data, although a preliminary one, clearly demonstrates that renal 5'-deiodinase is controlled by a kidney-specific mechanism. Further study on the regulatory mechanism of 5'-deiodinase will give us better understanding of how thyroid hormone regulates Na/K ATPase and serum sodium concentrations.