COMPARISON BETWEEN RADIOIMMUNOASSAY (RIA) AND CYTOCHEMICAL BIOASSAY (CBA) OF THYROID STIMULATING HORMONE (TSH).

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Recently a highly sensitive cytochemical bioassay (CBA) for the determination of human TSH has been developed. We could show that this assay is specific for TSH and measurements done on plasma of normal euthroid persons agree very well with radioimmunological findings. Due to the extreme sensitivity of the CBA we were able to detect low but measurable TSH levels in patients with primary hyperthyroidism, which were not increased by TRH treatment before therapeutic treatment. After therapeutic treatment, TRH application was able to stimulate additional biologically active TSH release, which however, barely reached the lowest limit of detection by RIA. In certain pathological cases we were able to detect elevated plasma TSH levels, which were active immunologically but inactive biologically.

We demonstrated there was a remarkably good agreement between the radioimmunologically determined activities, even of the guanidine-treated "big'-TSH fraction, and the cytochemically measured diological activities, respectively.

STUDIES ON RADIORECEPTOR ASSAY OF TSH: PROPERTIES OF TSH-BINDING INHIBITOR IMMUNOGLOBULINS (TBII) IN PATIENTS WITH HASHIMOTO'S THYROIDITIS

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By using the radioreceptor assay of TSH, TSH-binding inhibitor immunoglobulins (TBII) were detected in 60% of 31 patients with untreated Graves' disease. In patients who had been treated by ^{131}I 5 to 17 years before, the incidence of TBII was 20% (10/51). And all except two cases having TBII were found to be still thyrotoxic. Further TBII activities in untreated Graves' patients significantly correlated with human thyroid adenyle cyclase (AC) stimulating activities ($p < 0.01$). These data suggested the thyroid stimulating nature of TBII in Graves' disease. But TBII were detected in two (7%) of 29 patients with Hashimoto's thyroiditis. Both patients were untreated and hypothyroid. The potent TBII detected in one of them did not contain anti-TSH antibody, LATS, LATS-protector or human thyroid AC stimulating activity. The IgG inhibited both human thyroid AC stimulation and c-AMP increase in mouse thyroid induced in vitro by TSH or LATS. The serum inhibited LATS-stimulated ^{131}I-release in McKenzie bioassay. These data clearly indicated that TBII detected by the radioreceptor assay of TSH were not always thyroid-stimulating and contained heterogeneous population of TSH receptor related antibodies.