Casual Friday Series

Thyroid-HPA Crosstalk: Part 2

A Biogenetix Clinical Presentation

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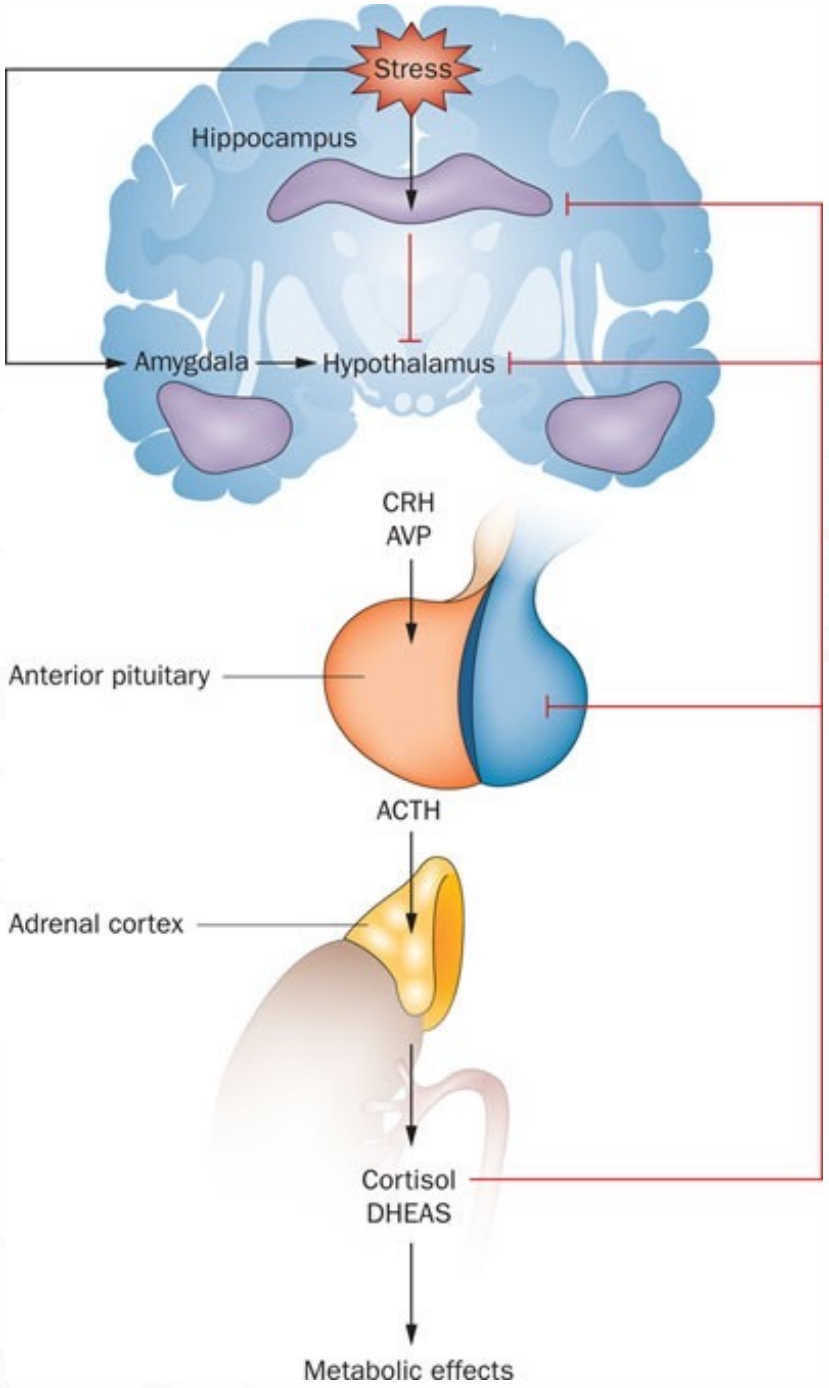
- *Information in this presentation is not intended, in itself, to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

Lifestyle + Genetics = Chronic Health Condition



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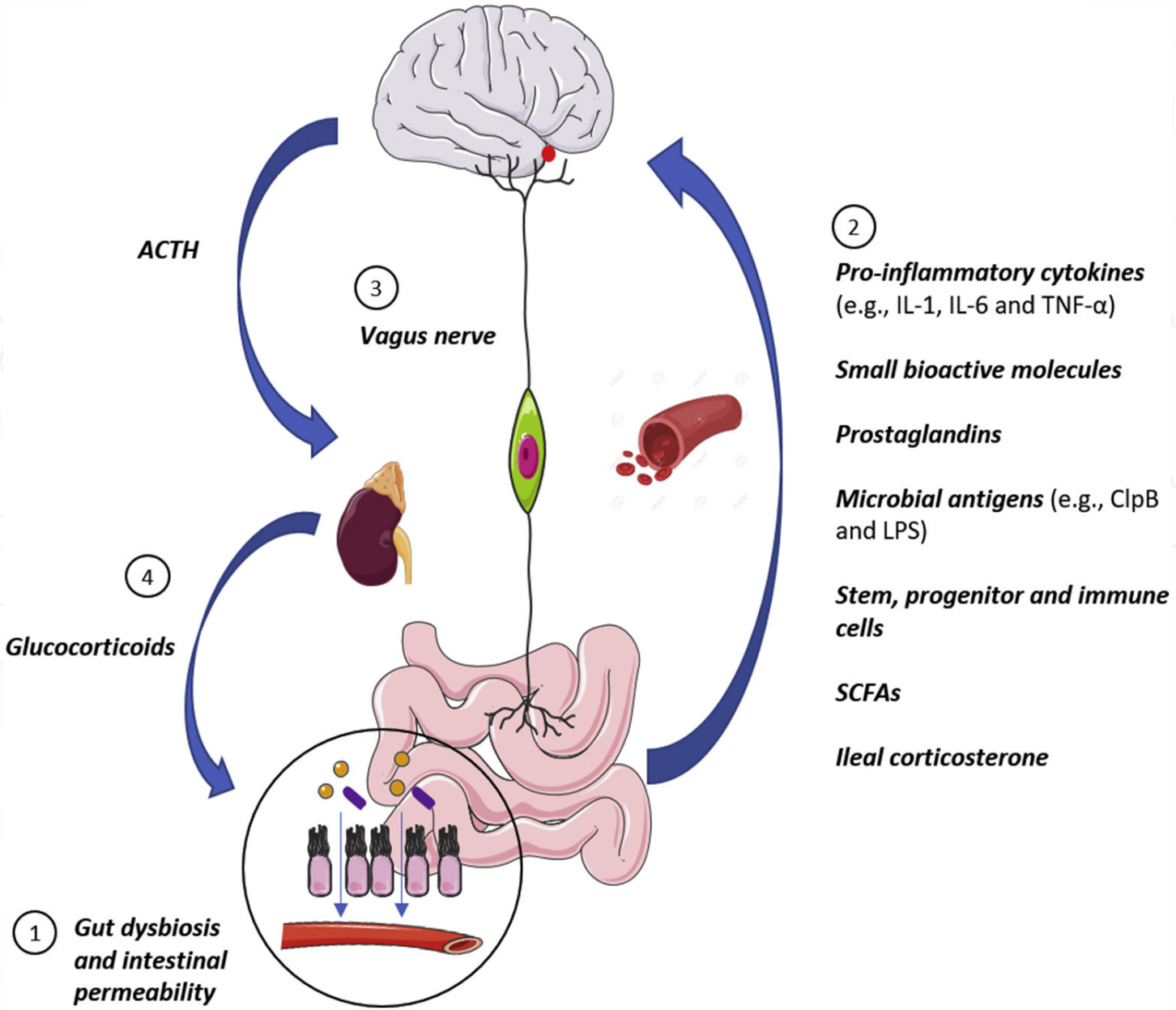

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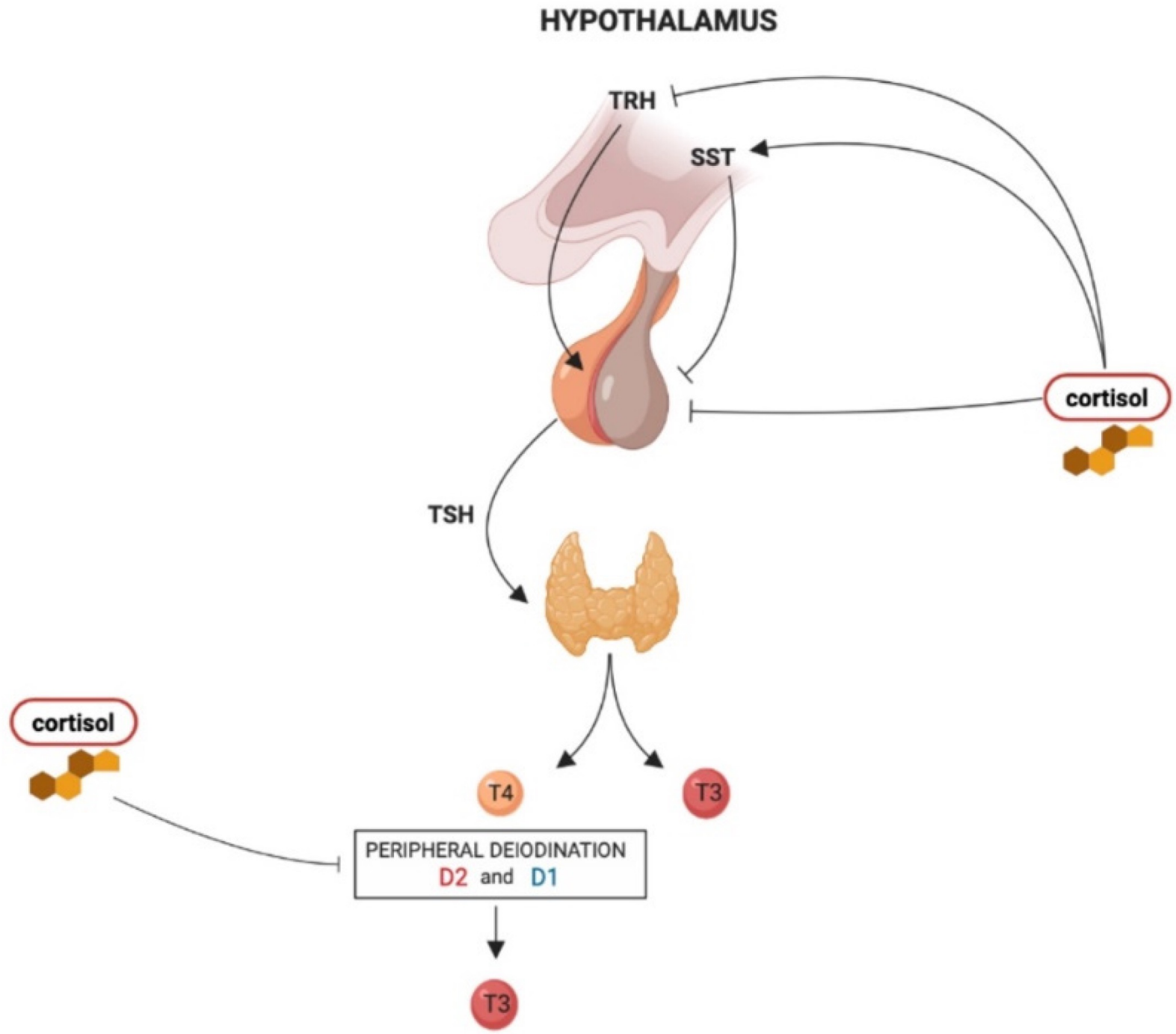
HYPOTHALAMUS
TRH
SST
cortisol
TSH
cortisol
T4
T3
PERIPHERAL DEIODINATION
D2 and D1
T3

Thyroid Res. 2012; 5: 13.
Published online 2012 Oct 30. doi: 10.1186/1756-6614-5-13
PMCID: PMC3520819
PMID: 23111240

Elevated thyroid stimulating hormone is associated with elevated cortisol in healthy young men and women

Kimberly N Walter,[1] Elizabeth J Corwin,[2] Jan Ulbrecht,[1] Laurence M Demers,[1,3] Jeanette M Bennett,[1]

Recent attention has been given to subclinical hypothyroidism, defined as a TSH elevation with T4 and T3 levels still within the normal range. Subclinical hypothyroidism is a common disorder; two large population-based studies revealed that 4% to 8.5% of individuals without known thyroid disease actually have subclinical hypothyroidism as evidenced by a mildly elevated TSH (i.e., 4.5-10 uIU/L) [2,3]. Complicating matters is the current controversy about the proper lower limit of TSH that defines patients in the subclinical hypothyroidism range (in other words, the upper limit of the normal reference range for TSH) [4-8]. In apparently healthy populations, the TSH distribution is skewed towards the lower end of the reference range, with the mean value typically being around 1.5 uIU/L, but with the range extending from 0.5–4.5 uIU/L [8]. Therefore it is possible that seemingly healthy individuals with TSH levels in the upper end of this range may in fact have elevated TSH in response to early thyroid gland failure and also should be considered to have subclinical hypothyroidism. This possibility is further supported by the fact that many patients with TSH levels in the 3.0–4.5 uIU/L range are positive for antithyroid antibodies [6,8].

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We examined the relationship between TSH levels and cortisol in a preliminary study of young, healthy adults without known thyroid disease or other underlying health conditions. The positive relationship between serum TSH and cortisol levels in a healthy population is a compelling new finding that is consistent with and extends the observation that frankly hypothyroid patients have frankly elevated cortisol levels [11].

These preliminary results raise important questions – such as whether this relationship is pathologic or phy siologic and what the mechanism(s) involved in this relationship may be. While in frank hypothyroidism, it is hypothyroidism that causes elevation of cortisol by reducing peripheral disposal and blunting feedback of cortisol on the hypothalamic-pituitary-adrenal axis [11], our cross sectional data do not elucidate whether the same mechanisms hold true for TSH levels in the high normal and low elevated range. Thus more definitive population-based and intervention studies are now needed to confirm this finding and answer these questions.

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Another potential explanation for the positive TSH-cortisol relationship is that hypothyroidism - subclinical or clinical - is associated with subtle metabolic stress. Metabolic stress could be imposing an effect on the adrenocorticotropin hormone-adrenal axis leading to an increase in stress hormone (i.e., cortisol) release and production. This hypothesis should be confirmed through the measurement of other stress hormones including the catecholamines, norepinephrine/epinephrine, and/or prolactin.

Although limited in sample size, our findings demonstrate that a positive relationship exists between TSH and cortisol that is maintained down to a TSH level of 2.5 uIU/L (but not below). This observation raises the possibility that negative health effects of mild, subclinical hypothyroidism with mild to modest elevations in TSH may begin at levels much lower than those currently considered abnormal based on assigned normal reference range values with an upper reference level of 4.5 uIU/L.

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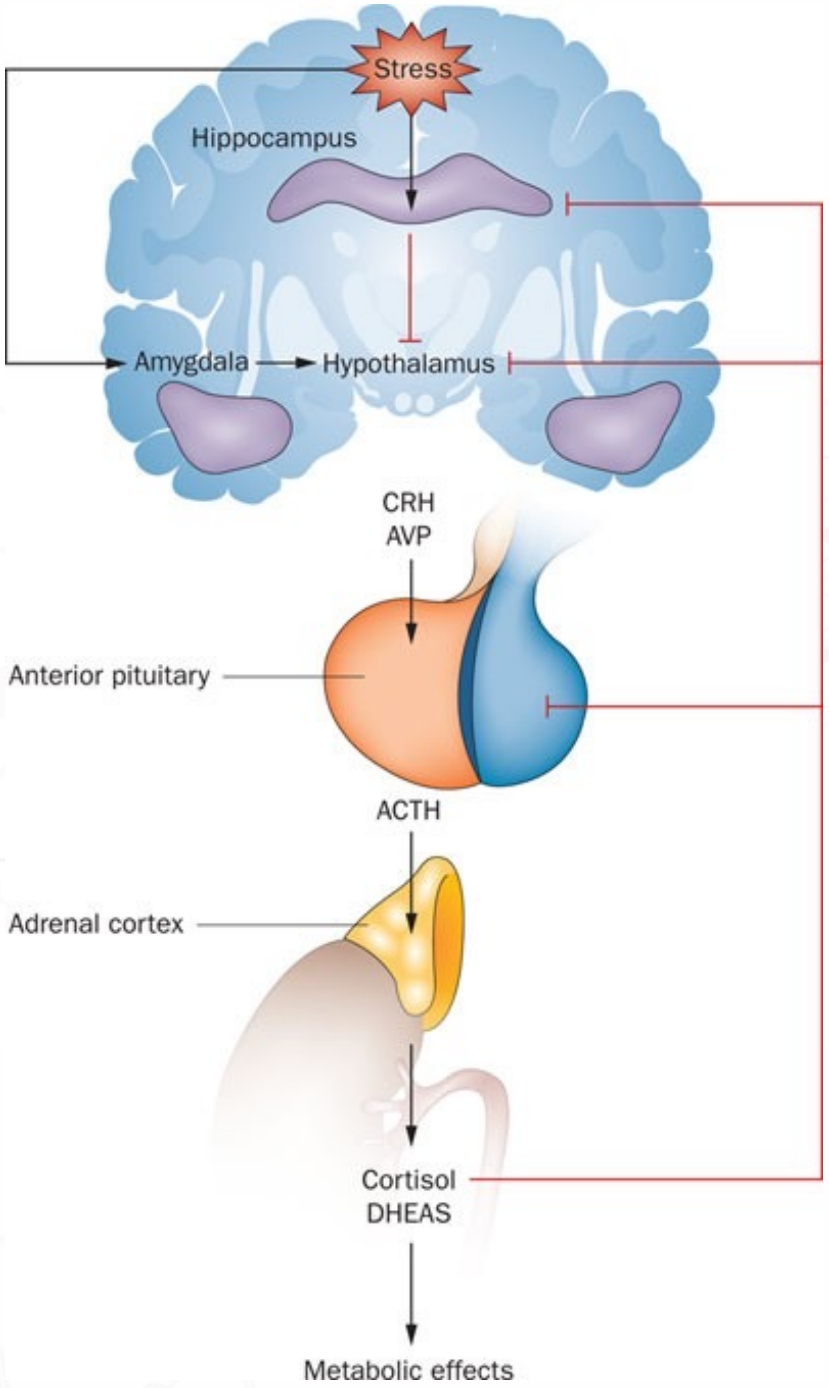
Chronic elevations in serum cortisol and hypothyroidism (including subclinical hypothyroidism) have been separately linked with increased rates of depression, anxiety, and poor cognitive functioning e.g., [15-17]. Thus, the association between TSH levels and cortisol suggests at least the possibility of a novel pathway through which hypothyroidism (both clinical and subclinical) may promote poor mental health; or hypothyroidism and an elevated cortisol level could be synergistic on mental health.

> J Clin Endocrinol Metab. 1990 Jan;70(1):155-61. doi: 10.1210/jcem-70-1-155.

Dynamics of 24-hour endogenous cortisol secretion and clearance in primary hypothyroidism assessed before and after partial thyroid hormone replacement

0.05); 3) mean prepeak nadir concentrations (298 vs. 221 nmol/L; P less than 0.05); and 4) mean half-life of cortisol disappearance (155 vs. 112 min; P less than 0.0019). In summary, the present study of cortisol secretory dynamics in hypothyroid men has shown elevated mean 24-h serum concentrations of cortisol with preserved circadian rhymicity and normal endogenous production rates, but prolonged half-lives of cortisol disappearance. In conjunction with normal serum cortisol-binding globulin concentrations, these largely reversible findings suggest that significant hypercortisolemia in primary hypothyroidism is primarily due to decreased metabolic clearance of cortisol and a presumptive decrease in the negative feedback effect of cortisol on the hypothalamo-pituitary axis.



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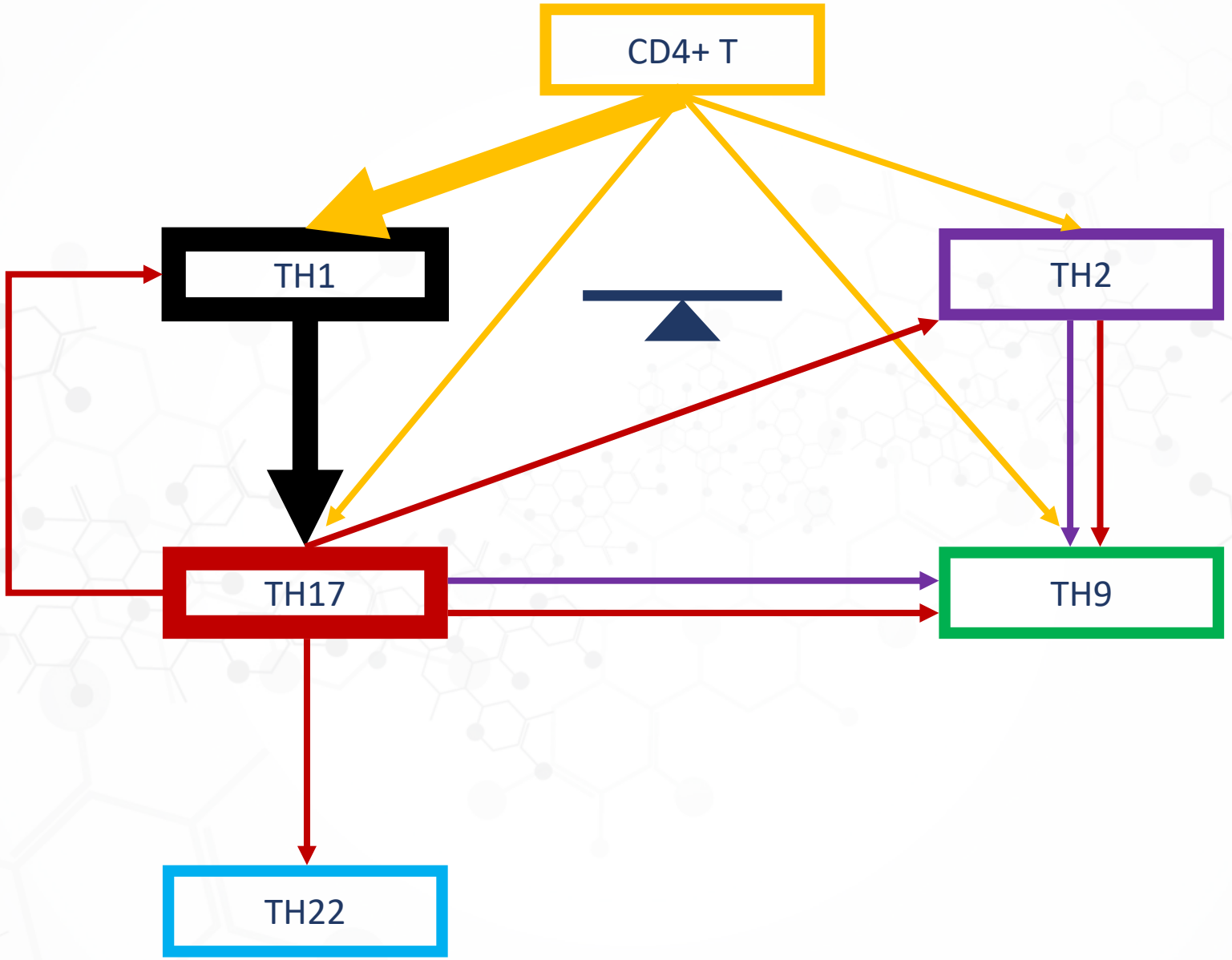
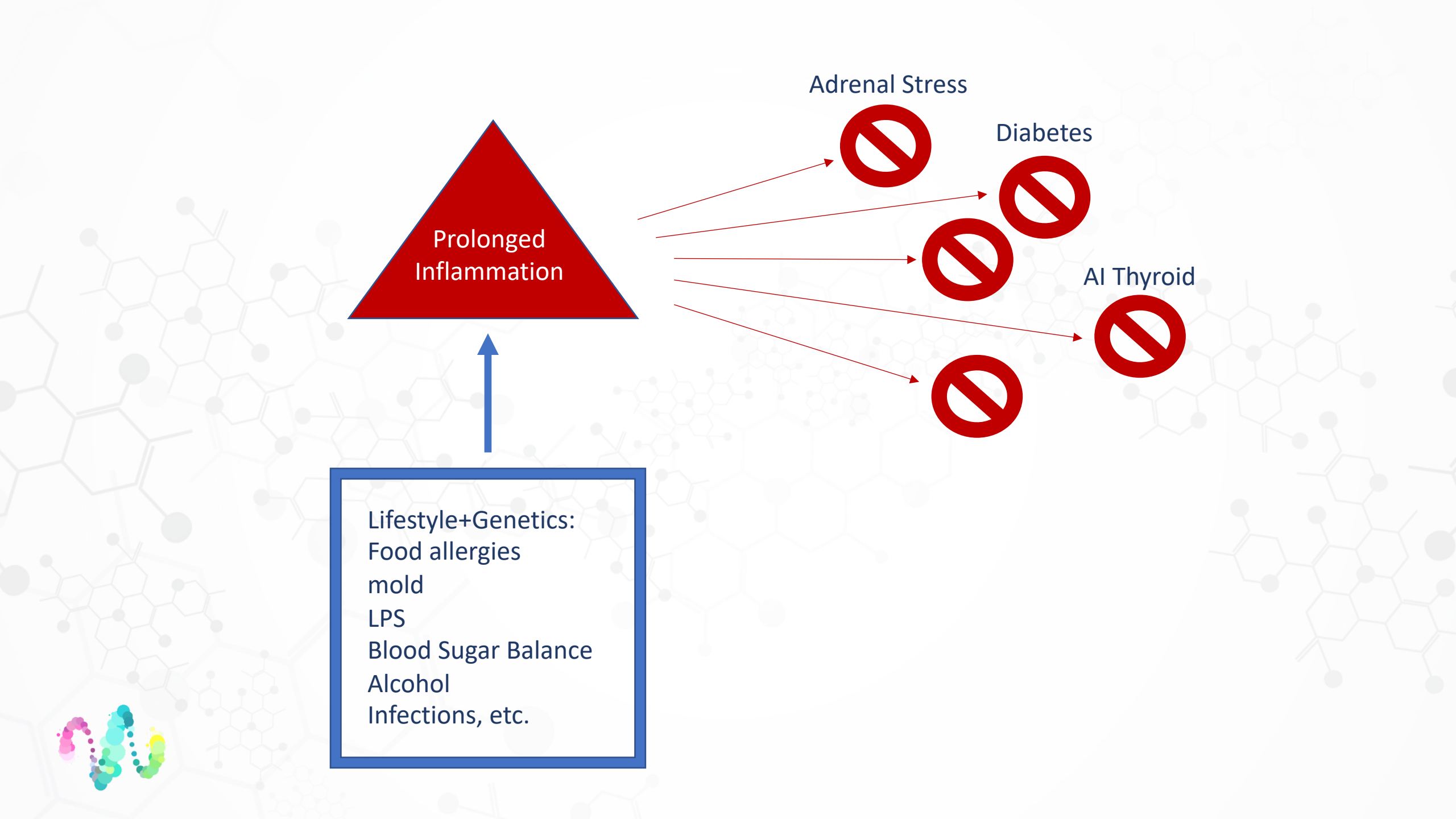
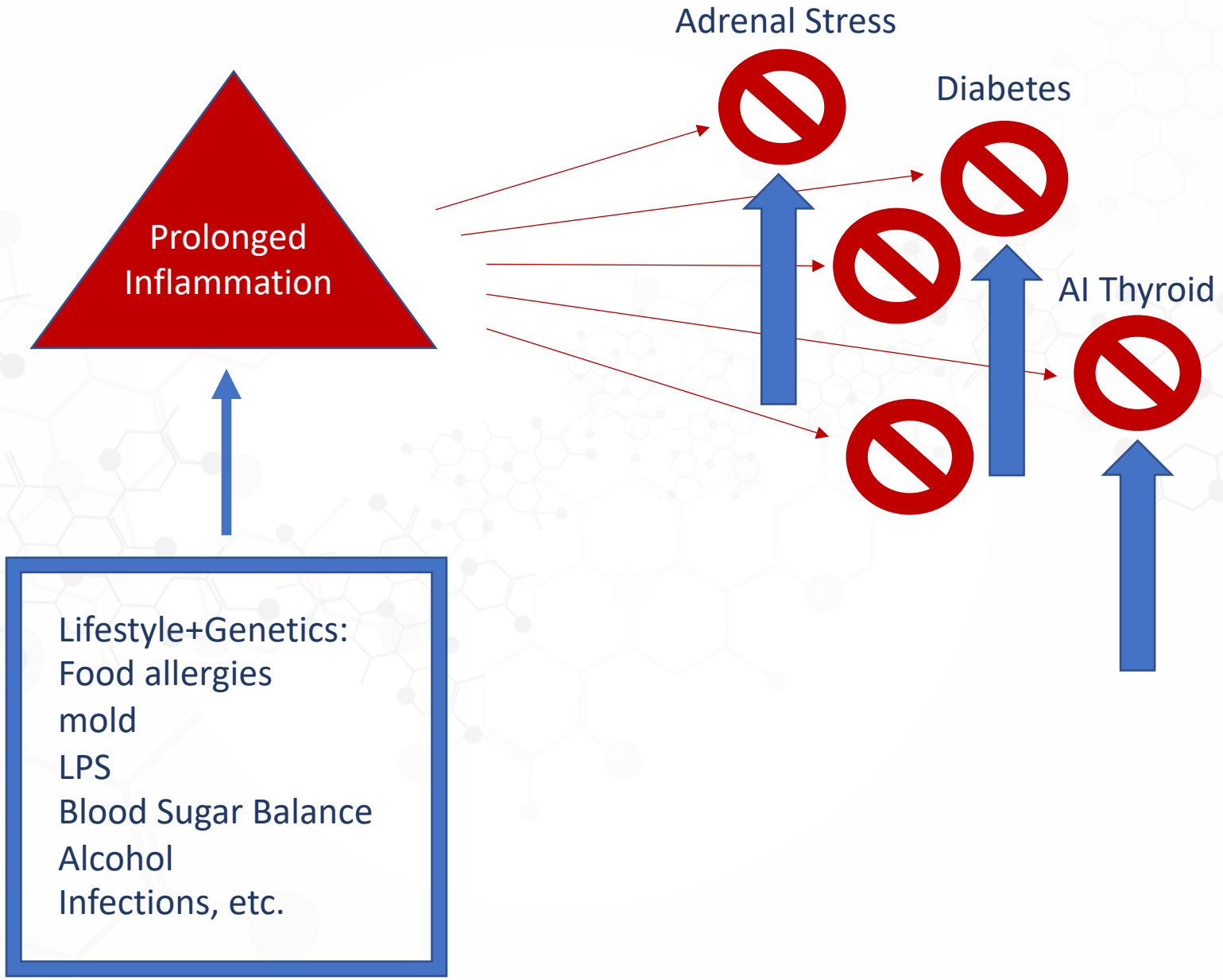

CD4+ T
TH1
TH2
TH17
TH9
TH22
(Dermatologic)


Adrenal Stress
Diabetes
Prolonged Inflammation
AI Thyroid
Lifestyle+Genetics:
Food allergies
mold
LPS
Blood Sugar Balance
Alcohol
Infections, etc.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