18 - C. Adrenal Cortex

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© SPMM Course B. Thyroid gland TRH from the hypothalamus stimulates the secretion of TSH from the pituitary. TSH in turn stimulates the thyroid gland to synthesize and release thyroxine T4 and triiodothyronine T3. T4 is the predominant form of thyroid hormone, but T3 is biologically more potent. T4 is converted into T3 by target organs as well as the brain. Exogenous administration of TRH produces a brisk response by increasing TSH concentration. In patients with depression, a blunted response to TRH administration is seen. Mania, alcohol withdrawal and anorexia can also cause blunted TRH response. The addition of T3 and T4 as supplements to antidepressant treatment has been shown to accelerate response in some patients, particularly women. Exogenous administration of thyroid hormones (e.g. in resistant depression) increases serotonergic transmission with decreased 5-HT1A sensitivity and increased 5-HT2A sensitivity Nerve growth factor genes are activated by T3 during early development but not in the adult's brain. Lithium produces hypothyroidism especially in middle-aged women who are predisposed to carry antithyroid autoantibodies. Hypothyroidism is sometimes implicated in rapid cycling mood pattern in previously stable bipolar patients. Hyperthyroidism is associated with symptoms of generalized anxiety disorder. Hyperthyroidism Hypothyroidism Physical symptoms: Tachycardia, weight loss, heat intolerance, sweating Physical symptoms: Fatigue, weight gain, cold intolerance, dry skin Mental symptoms: Anxiety, irritability, poor concentration, agitation, emotional lability. Mental symptoms: Depression, reduced activity (psychomotor retardation), reduced libido and poor memory

C. Adrenal Cortex CRH from the hypothalamus stimulates ACTH release from the anterior pituitary. ACTH in turn stimulates the release of cortisol from the adrenal cortex. Cortisol thus produced in turn inhibits both CRH and ACTH in a negative feedback loop to maintain homeostasis. This is called Hypothalamic-PituitaryAdrenal (HPA) axis. HPA axis is involved in regulation of stress response. With chronic stress the HPA feedback fails and continuous excess of cortisol is produced with deleterious consequences to the hippocampus where glucocorticoid receptors are abundant. Decreased hippocampal neurogenesis with atrophy of hippocampal dendrites results in shrinkage of the hippocampus. This disrupts long-term potentiation (LTP) and leads to impaired memory performance. A compensatory increase in dendritic arborization of

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