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UNIVERSITY · CANADA

Tutorial 8
Sections 009/010

TA: Greydon Gilmore

Physiology 2130

Oct 29th, 2019

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Your TA reminding you…

- **2nd Peerwise assignment** (1.5%)
  - Post 2 MC questions: due Nov 27th @ midnight
  - Answer 5 MC questions: due Nov 29th @ midnight
- **2nd Quiz** (1%)
  - Opens: Dec 2nd @ 4pm
  - Closes: Dec 3rd @ 4pm

Today

- Group work activity
- Learning Catalytics Question
- Thyroid Gland and Adrenal Gland

Group Work

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Diagnose me!

All of the following patients have conditions that affect various hormones in their body. Your job is to diagnose them and explain the physiological reasons for each symptom and hormone imbalance.

Patient #1: Miranda C.

As a hospital physiotherapist, you get a call to see a new patient who needs assistance with her mobility. When you check her chart, you read that she has very thin arms and legs and an enlarged abdomen. She is complaining of severe muscle weakness. You are told that she has been taking high doses of hydrocortisone (synthetic cortisol) orally for 6 months for her Crohn's disease. Her blood tests show normal TSH levels, low CRH, low ACTH, and normal T3/T4.

What would you diagnose her with and why? What other symptoms might your find in her chart? What caused her condition?

Learning Catalytic Question

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Patient #1: Miranda C. Answer

Diagnosis:

- The low CRH and ACTH suggests it is some abnormality with cortisol
- She is experiencing symptoms of Cushing's disease: thin arms and legs, enlarged abdomen (weight gain), severe muscle weakness.

Other symptoms:

- Recurring infections due to immune suppression, pronounced stretch marks from tearing of the skin, a moon face, possible osteoporosis, and high blood sugar.

What is the cause:

- Cushing's is due to high levels of cortisol, which would explain why CRH and ACTH are low (negative feedback is "on")
- These symptoms would be caused by the hydrocortisone. While her body isn't making its own due to negative feedback, each time she takes the drug, her blood cortisol levels rise.

Patient #2: Reginaldo D.

You start a new practice as a recent graduate of medical school. Reginaldo is one of the first patients you see, who was transferred to your practice. On his physical exam you notice he has a slow heart rate. You ask if he's tired a lot and he confirms he has. When you feel his neck, you discover a large growth in the front, which worries you. You send him off for some specialized blood work and book a follow up consult.

Based on what you observed in the clinic, what would you suspect his diagnosis to be? What would his blood work show for ACTH, CRH, cortisol, TSH, TRH, and T3/T4? What treatment would you recommend?

Learning Catalytic Question

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Patient #2: Reginaldo D. Answer

Diagnosis:

- He is showing signs of a goiter (enlarged/hypertrophy of the thyroid). This could be hyperthyroidism OR hypothyroidism. However, the slowed heart rate and fatigue would suggest it is hypothyroid (low T3/T4).

Blood work for TSH, TRH, and T3/T4 , ACTH, CRH, cortisol:

- The goiter suggests the thyroid is still being stimulated by TSH. So TSH and TRH levels are likely high.
- But how can T3/T4 be low? He is unable to make thyroid hormone.
    - Negative feedback is not “on”, raising the levels of TSH and TRH to increase production BUT if he is experiencing iodide deficiency (or low thyroglobulin/tyrosine which we never discussed in class), he can’t make the hormone and the vicious cycle continues.
- We would expect CRH and ACTH to likely be normal since this is a thyroid issue.

Treatment:

- He would be treated with synthetic thyroid hormone: clinically we use thyroxine. The reason we don’t use T3 is that T4 remains in the blood longer (longer ½ life) and can be converted into T3, the more potent hormone.

Endocrine: Thyroid Gland and Adrenal Gland

Chapter 3: Dr. Beye

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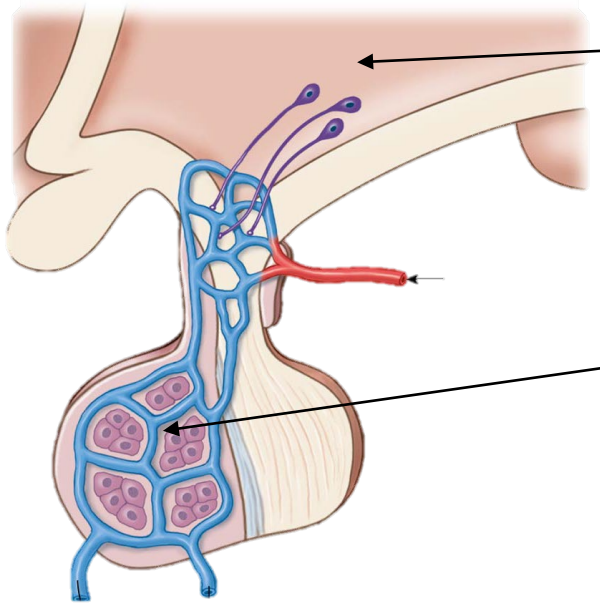
Adrenal Gland: Layers

	Layers	Categories of Hormones	Example	Stimulus	Effect
Cortex	Zona glomerulosa	Mineralocorticoids	Aldosterone	RAAS pathway (@ low BP)	Increase Na^+ reabsoprtion
Cortex	Zona fasciculata	Glucocorticoids	Cortisol	ACTH	-
Cortex	Zona reticularis	Androgens	DHEA	-	-
Medulla	Medulla	Catecholamines	Epinephrine	Sympathetic Nervous System	SNS response

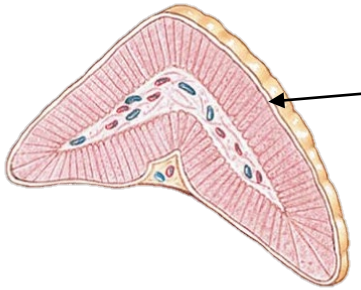
Three classes of steroids: Mineralocorticoids, Glucocorticoids and Androgens

Cortisol Feedback



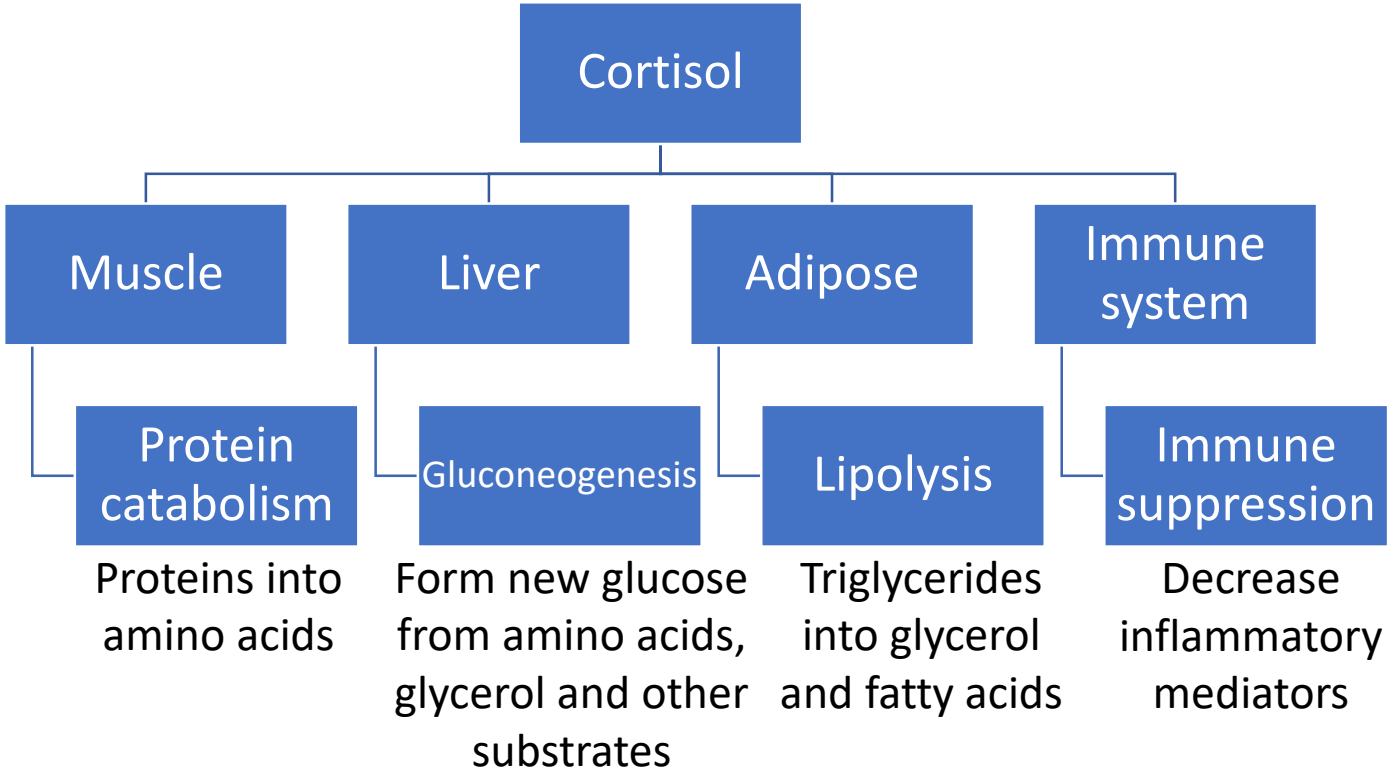
Hypothalamus
Cortisol Releasing Hormone (CRH)

Anterior Pituitary
Adrenocorticotropic Hormone (ACTH)



Zona Fasciculata
Cortisol

Cortisol

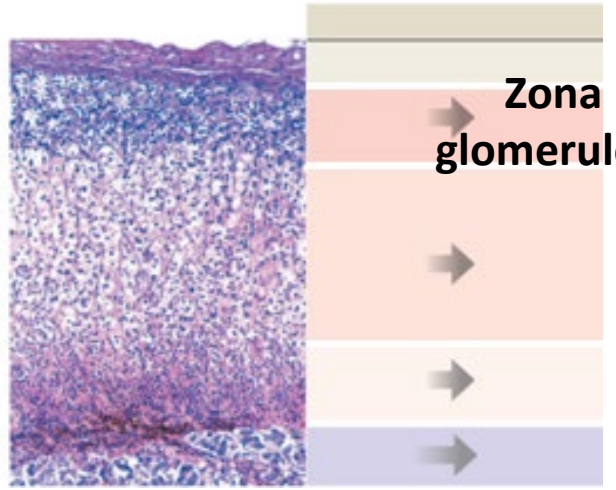


- Cortisol is catabolic: Break down larger molecules
- Cortisol levels peak early morning and decline throughout the day
- Cushing's disease (hypercortisolism): thinning skin, muscle wasting and weakness, stunted growth, increased infections, redistribution of fat tissue

Hormones

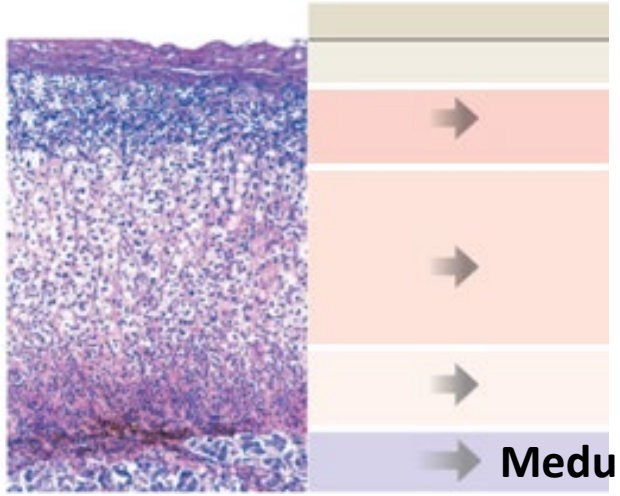
Aldosterone

- Released by the adrenal cortex and is a mineralocorticoid
- Angiotensin II hormone that stimulates aldosterone release
- Not from hypothalamus, comes from the renin-angiotensin-aldosterone system (RAAS)
  - RAAS upregulates when blood pressure low (sodium reabsorption)
  - More sodium within the blood would increase the osmolarity (thus more water would flow into the blood and increase pressure)



Epinephrine

- Released by the medulla and is a catecholamine
- Sympathetic nervous system triggers epinephrine for release
- Target effects: increase gluconeogenesis, increase heart rate, relaxes lung airw and increases blood pressure (constrict vessels)



Pancreas

	Glucagon	Insulin
Tissue	Islet of Langerhans	Islet of Langerhans
Made by	α- cells	β- cells
Stimulus	↓ Blood glucose Hypoglycemia	↑ Blood glucose Hyperglycemia
Effect	↑ Blood glucose (cells release glucose)	↓ Blood glucose (cells take up glucose)
Class	Peptide	Peptide

Antagonistic Effect

Next Tutorial (Nov 12th)

- Autonomic nervous system

What Questions Do You Have?

You can ask in the **Owl forums** as well!

Also anonymously ask questions in the **online dropbox**!!

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