UPCOMING EVENTS

LENS

Laboratory, Endocrine, & Neurotransmitter Symposium

February 13 - 14, 2026 | Las Vegas, NV

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Navigating the Doctor's Data GI Assessment Menu

Presented by Brandon Lundell, DC
February 18th, 2026 at 11 AM Pacific

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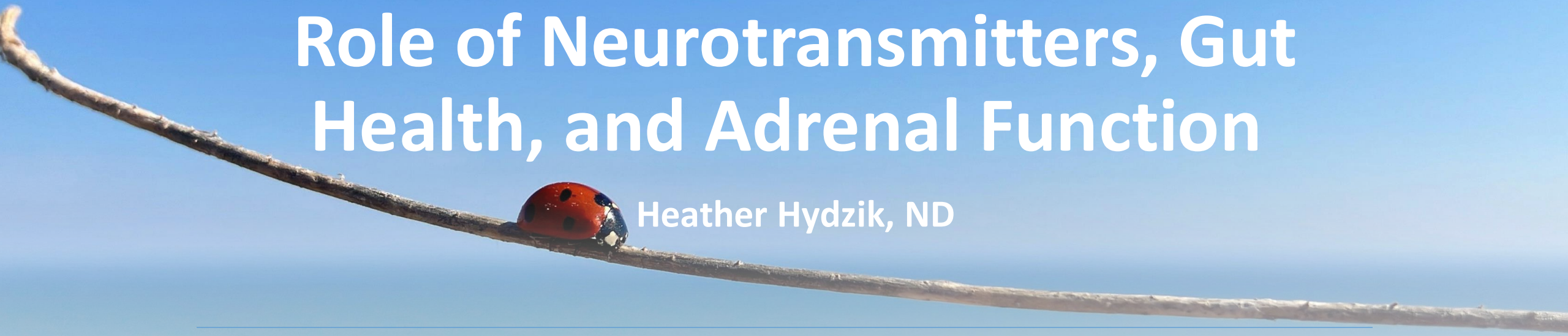
Wellness Wednesday

WILL BEGIN SHORTLY

MORE WEB EVENTS

Balancing the HPA Axis: The Critical Role of Neurotransmitters, Gut Health, and Adrenal Function



Heather Hydzik, ND

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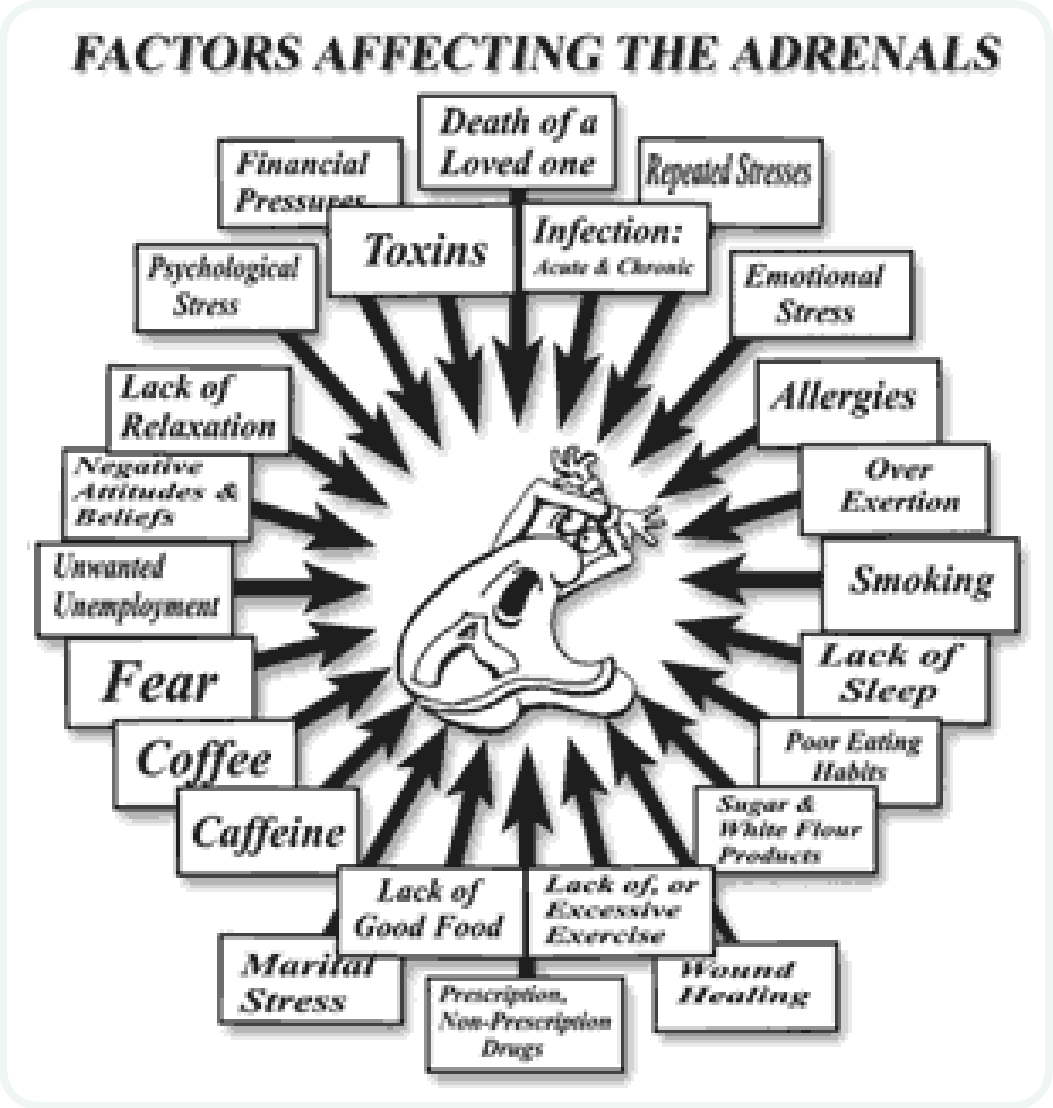
Learning Objectives

1. Explore adrenal anatomy, physiology, and the key mechanisms that regulate a healthy HPA axis.
2. Explore how the HPA axis adapts to stress and learn to identify key dysfunction patterns at different stages.
3. Evaluate advanced assessment tools, including diurnal cortisol patterns and the cortisol awakening response (CAR).
4. Discover how neurotransmitters and gastrointestinal health influence stress tolerance and HPA axis balance.
5. Implement proven strategies to rebalance the HPA axis and strengthen patient resilience to stress.



Why has “Stress” become “Adrenals”?

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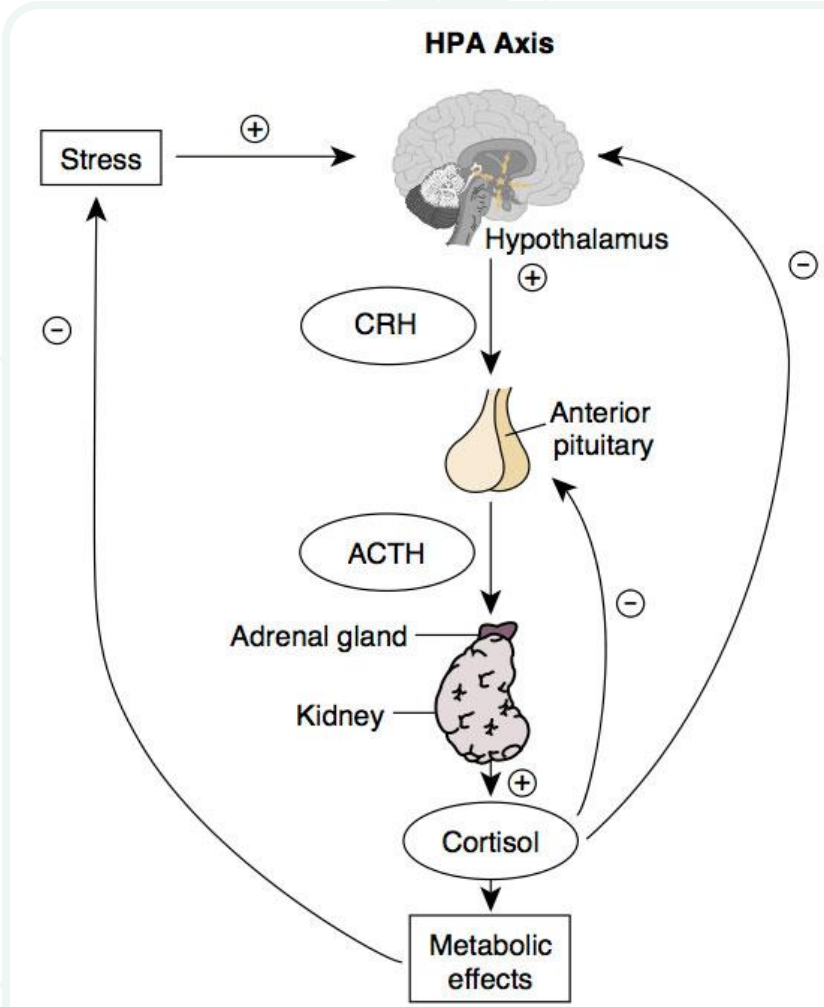

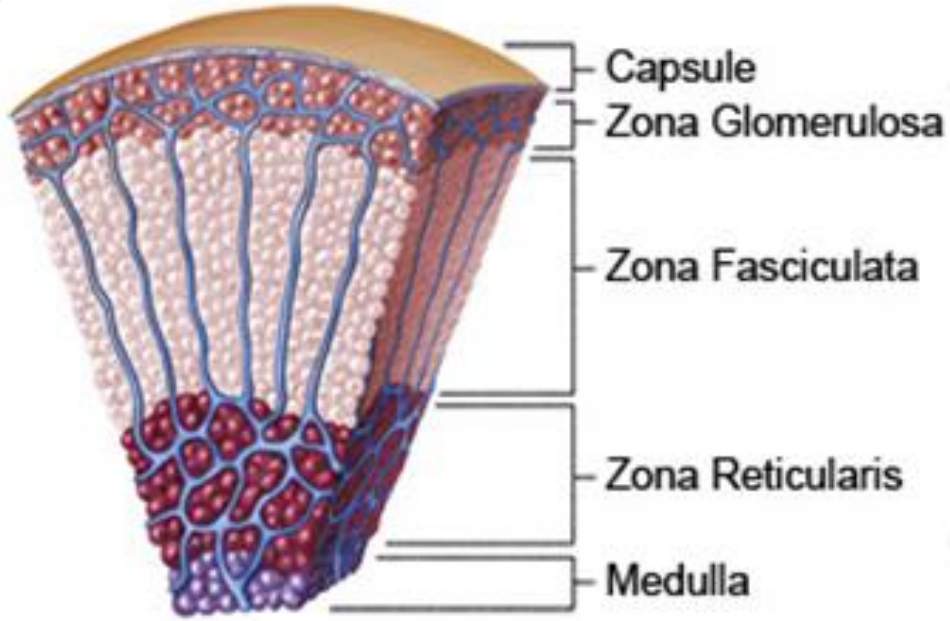

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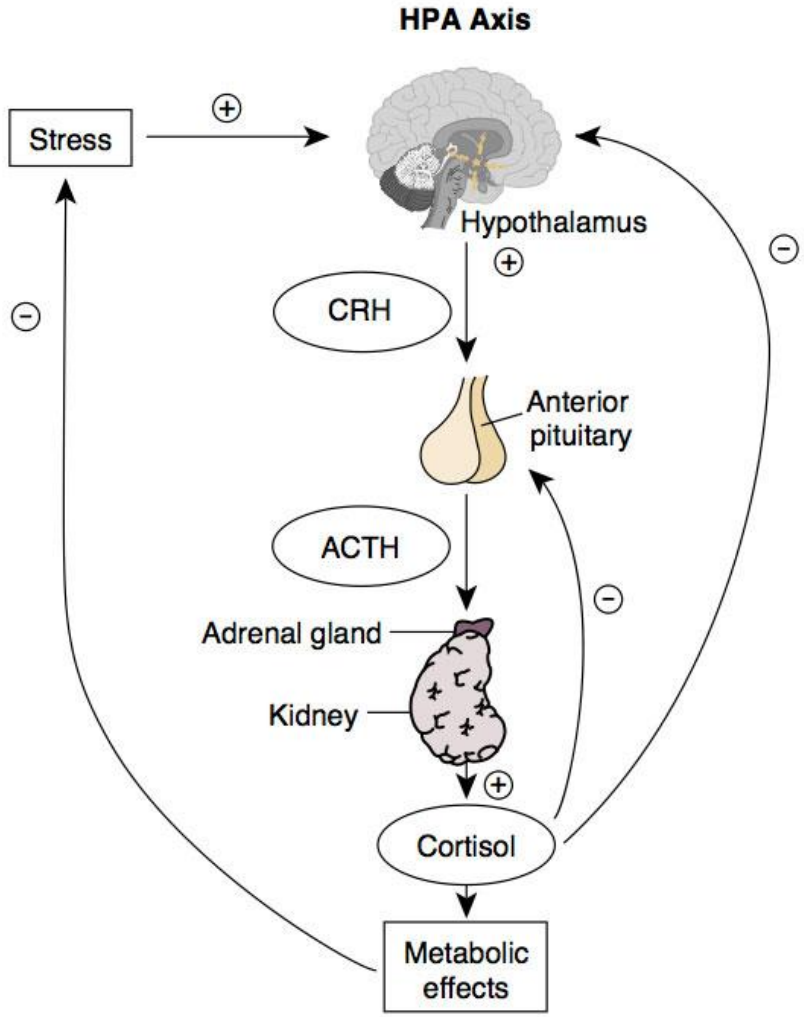
What are we really trying to measure or understand?

- The Relationship between a Stressor and its **Metabolic** Effects
- **Reserve** capacity
- How Acute and Chronic Stressors have affected the **circadian** nature of the HPA axis
- What **therapies** are most likely to help.



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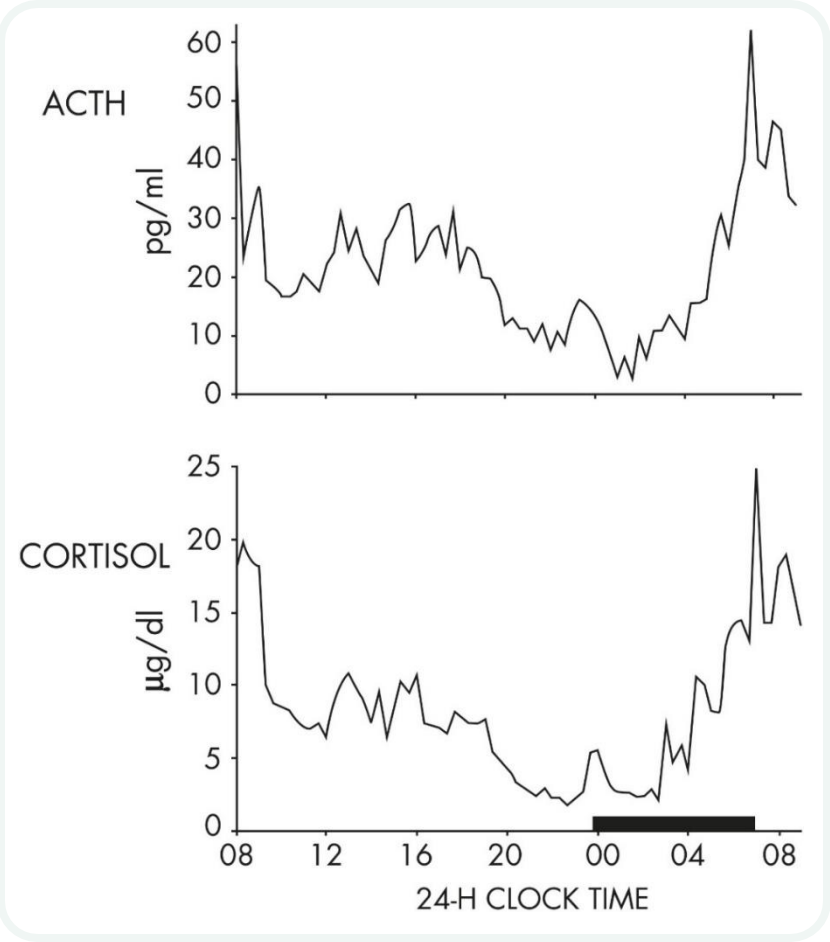


Cortisol as a surrogate for ACTH

- Plasma ACTH and Cortisol measured every 15 minutes in a healthy young man under normal (basal) conditions.
- Note the Ultradian Rhythm of ~60-90 minutes of both ACTH and Cortisol

Endocr Rev, Volume 38, Issue 1, 1 February 2017, Pages 3–45

What tells us someone is on “overload”?

- Overeating, comfort foods
- Sleep Deprivation
- Anxious or depressed
- Taking medications—anxiolytics, sleep-promoting agents—to help cope
- Excessive alcohol/nicotine/drug use
- Neglecting to see friends or take time off
- Quitting regular physical activity
- Staying at a computer far too long
  - to try to get out from under the burden of too much to do.
  - or to distract and divert from feelings of overload

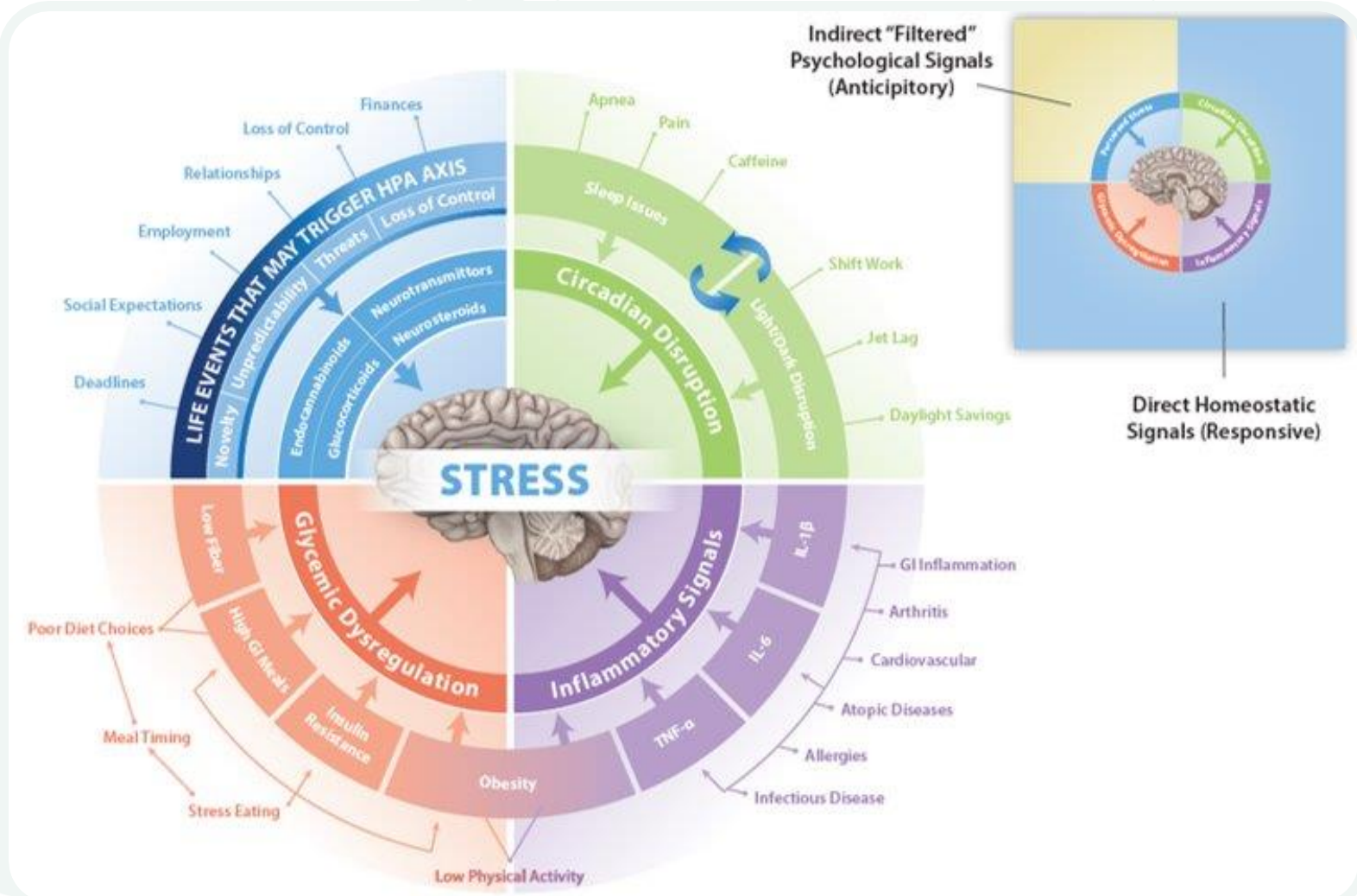
Allostatic Load

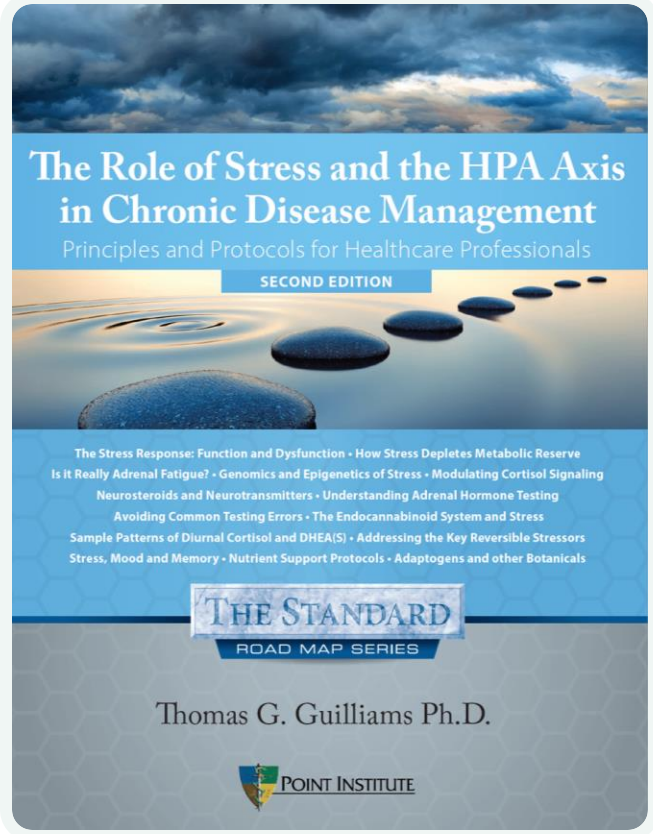
- Maintain effective blood supply (O_2/nutrition) to brain, heart, skeletal muscle for immediate survival
- Increase energy production by recruiting substrates (glucose, FA, AA) from body stores and enhance gluconeogenesis
- Optimize ATP production for vital short-term needs
- Achieving physiological reliance in the moment

Allostatic Overload
= Increased Cost (inflation) in the Now

- Allostatic Overload: The wear and tear that results from excessive stress or inefficient management of allostatic load
- Essentially, not turning off the response when it is no longer needed (cortisol, sympathetic activity, and proinflammatory cytokines, with a decline in parasympathetic activity).
  - Ex. Short term solution for immediate need for energy (glucose for the brain and muscle)
  - Leads to insulin resistance, and other insulin-linked metabolic disorders such as abdominal obesity and type 2 diabetes

Gui T. The Role of Stress and the HPA Axis in Chronic Disease Management, Principles and Protocols for Healthcare Professionals 2nd edition. 2020





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Guilliams, 2023 LENS conference, The HPA axis and Chronic Disease Management

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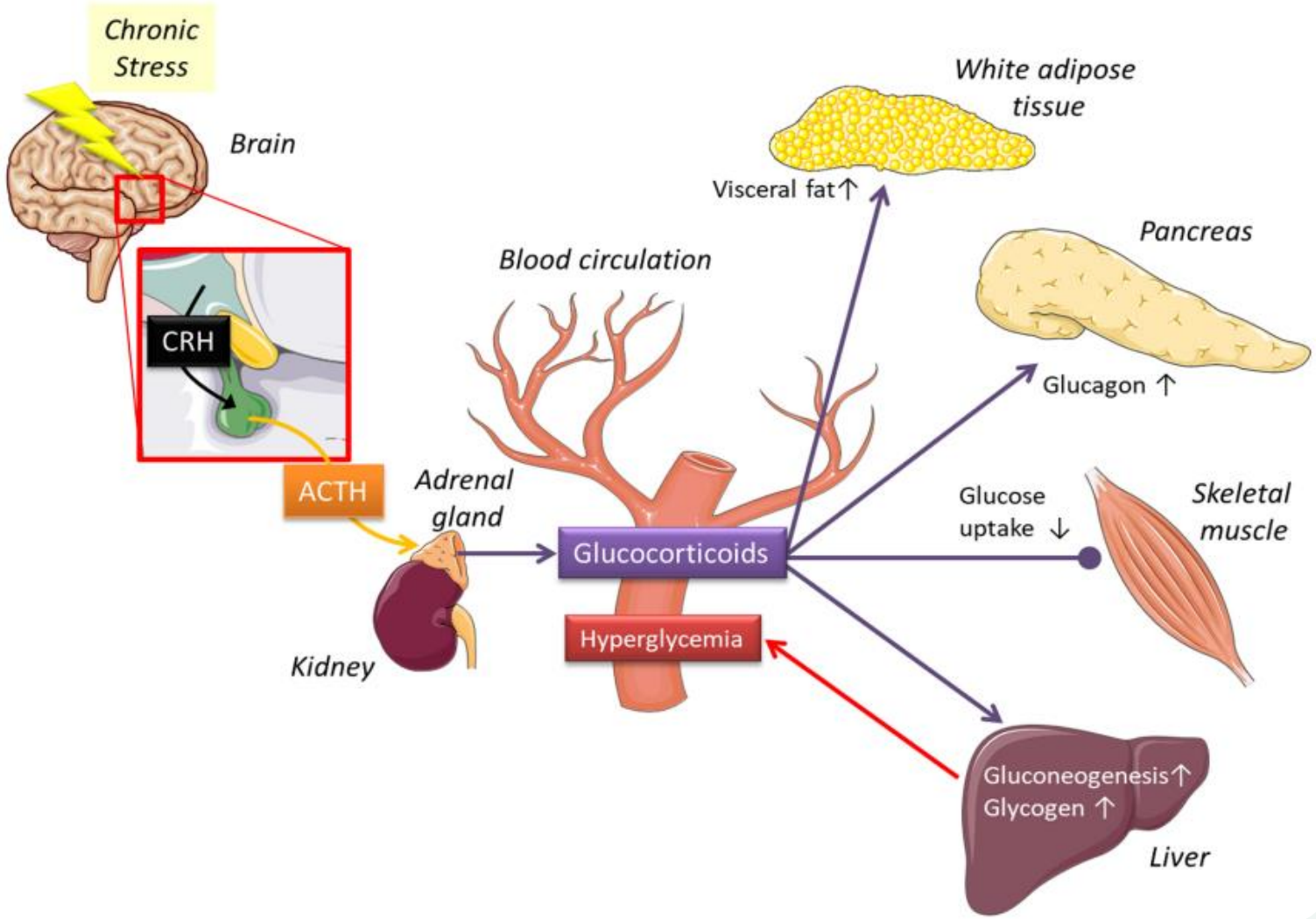
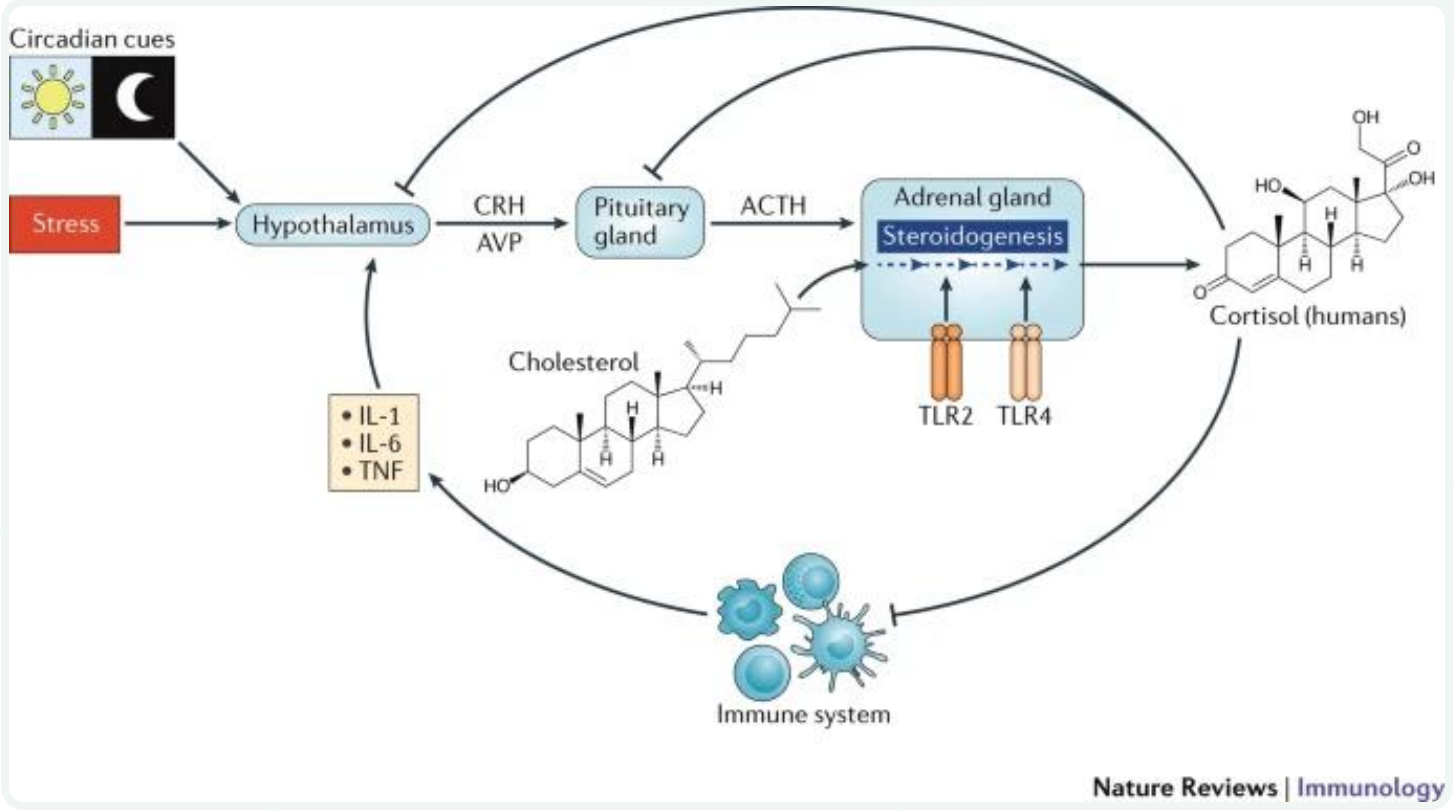
The Impact of Stress on Energy Metabolism - 2020



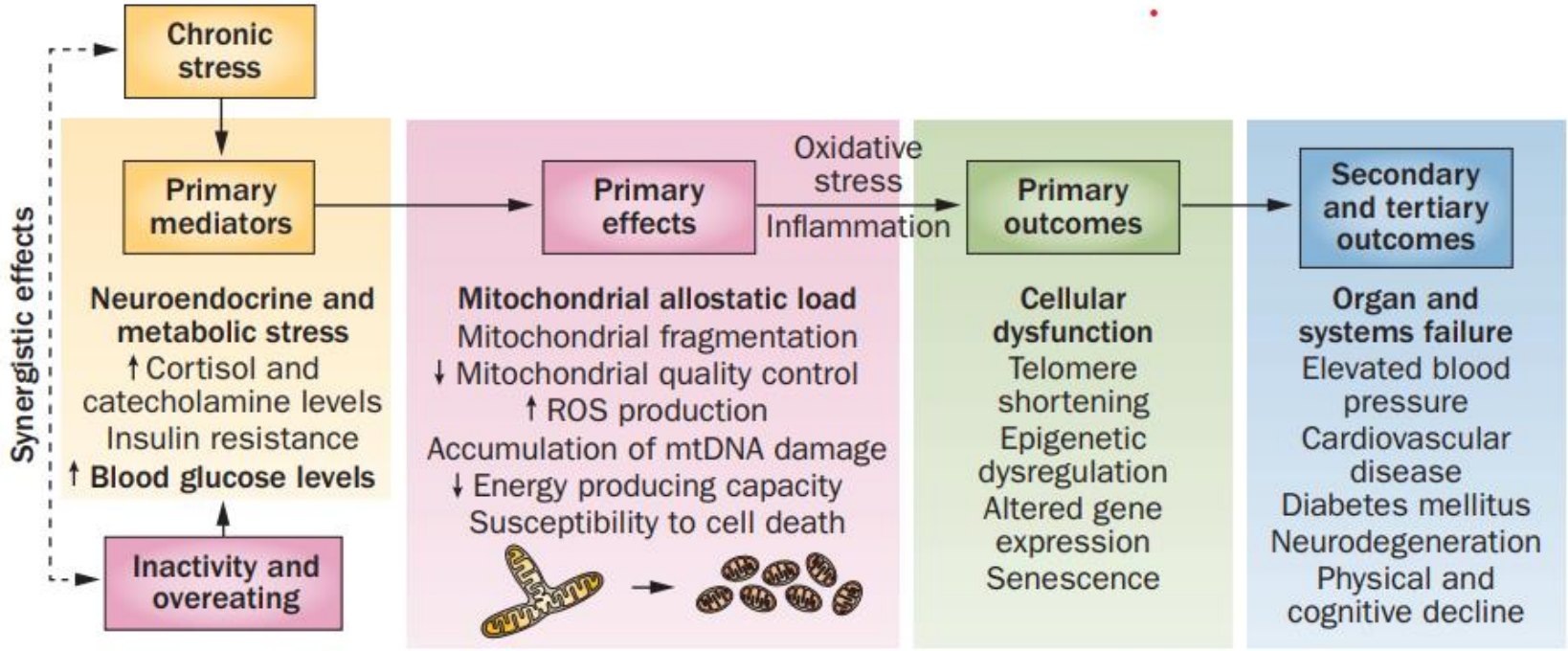
Doi: 10.1016/j.mcn.2020.103525



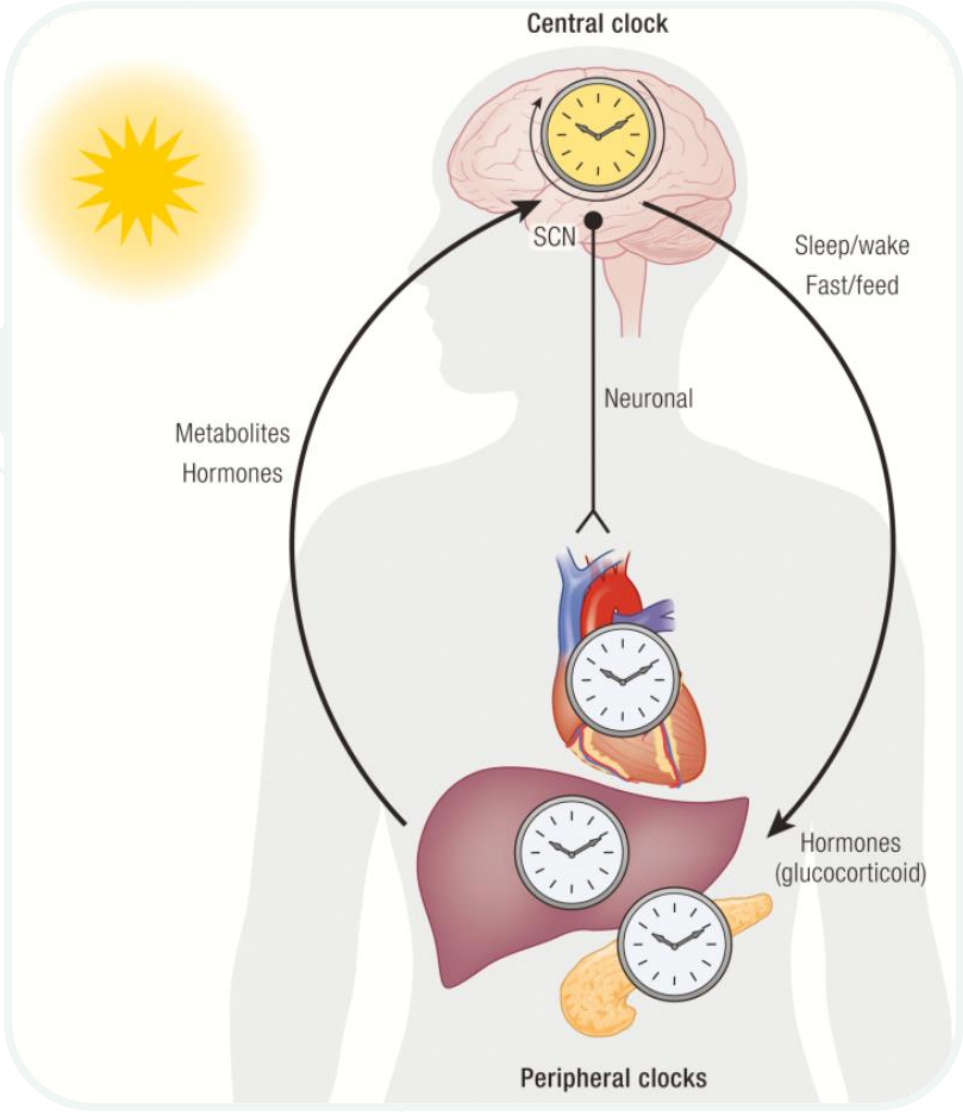
Inflammation affects cortisol secretion

“Mitochondrial allostatic load puts the gluc back in glucocorticoids”

Picard, Juster and McEwen - 2014

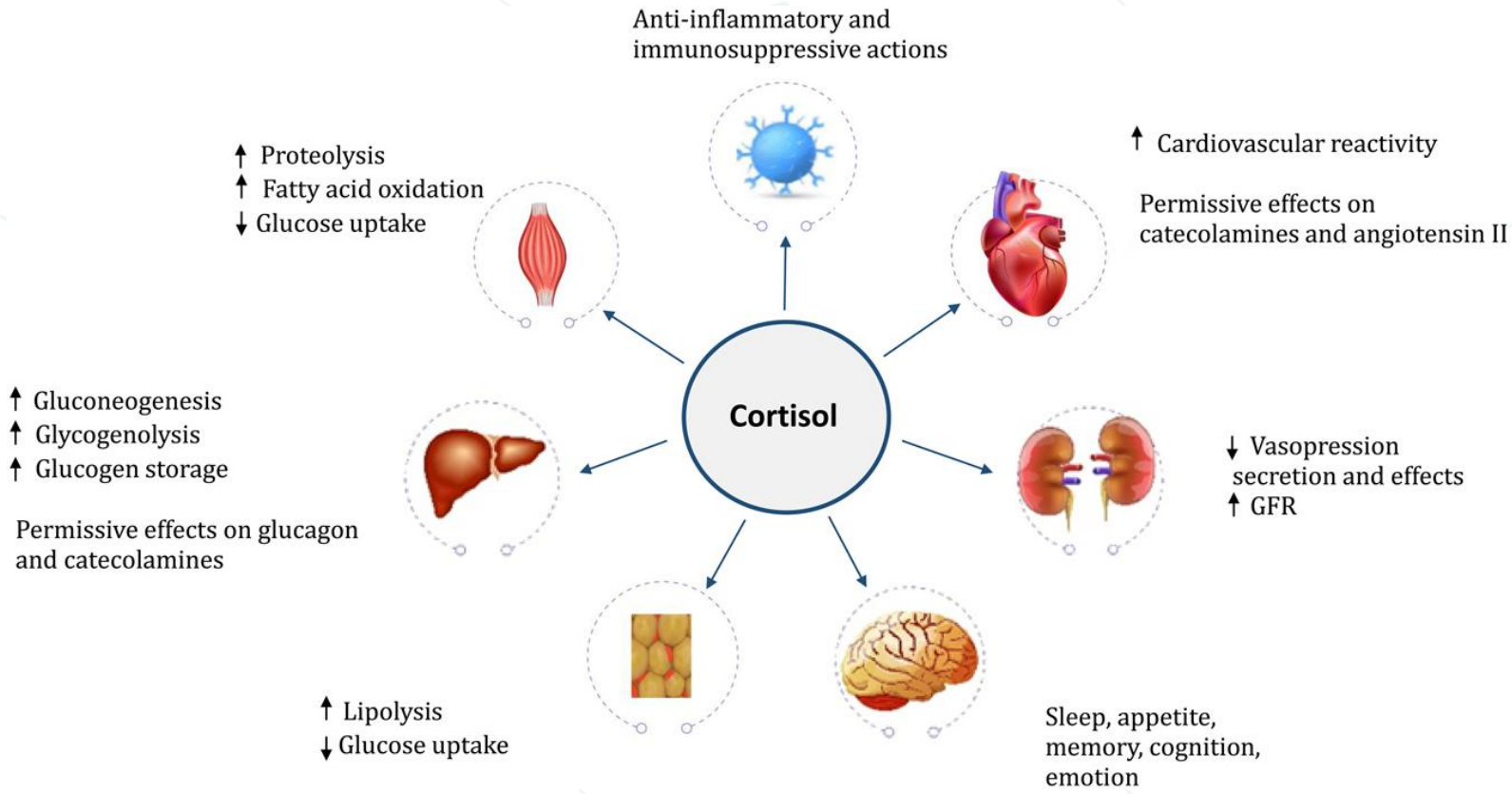


Circadian Rhythms and Cortisol



Endocrine Reviews, 01 Oct 2020, 41(5):bnaa014

Cortisol: multisystem effects



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European Journal of Endocrinology 184, 4; 10.1530/EJE-20-1199

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HPA axis testing salivary testing options

- Adrenal Function Profile:
  - Diurnal Cortisol Profile= 4 cortisols (AM30, noon, evening, PM)
  - DHEA

- CAR (Cortisol Awakening Response*) = immediate upon waking, AM30, AM60

*Can add to any adrenal profile

ADRENAL PROFILE

DIURNAL CORTISOL

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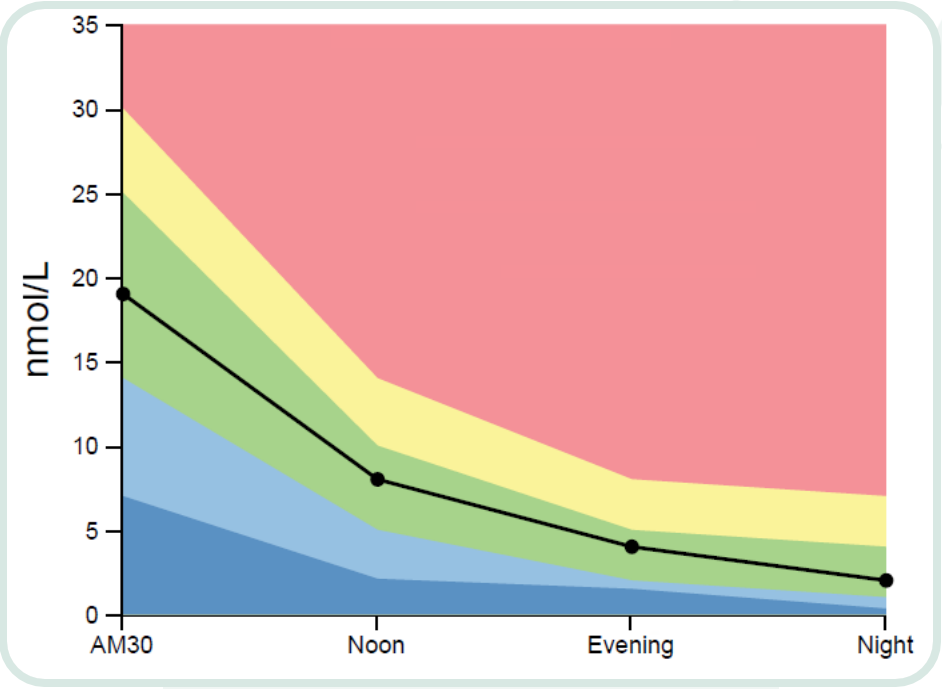
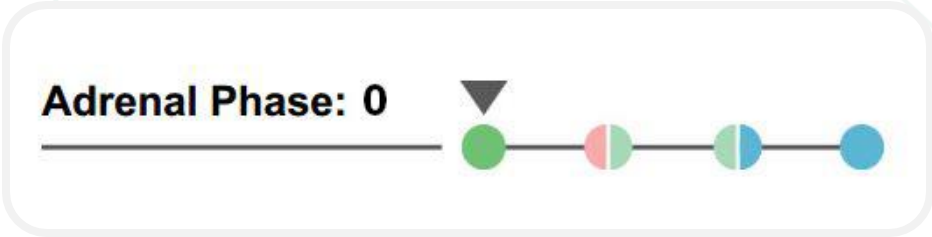
Phases of HPA axis (adrenal gland) dysfunction

Healthy adrenal response (Cortisol levels within range with desired rhythm)	Phase 0
Acute fight or flight (Increased HPA tone)	Phase 1
HPA axis dysfunction (Zig Zag patterns)	
Early adrenal dysfunction (elevated/high range AM with HPA blunting thereafter)	
Evolving adrenal dysfunction (N or low AM cortisol with HPA blunting thereafter)	Phase 2
Hypoadrenia (hypofunction HPA axis)	Phase 3

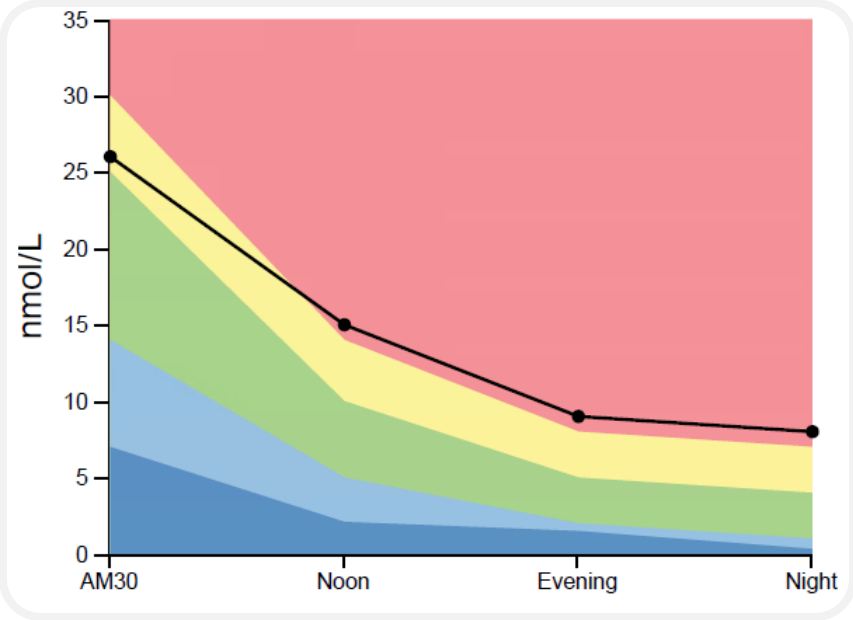
A healthy stress response

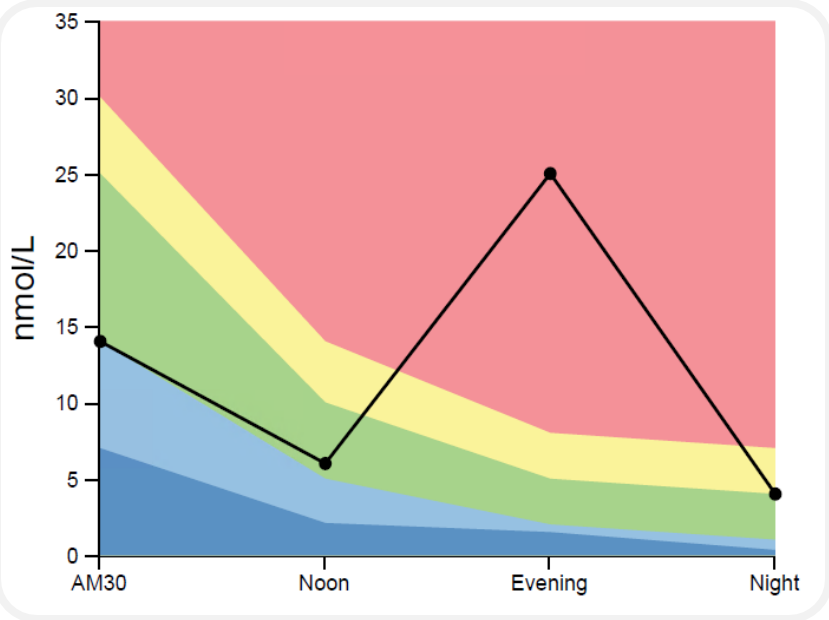


Salivary cortisol diurnal rhythm

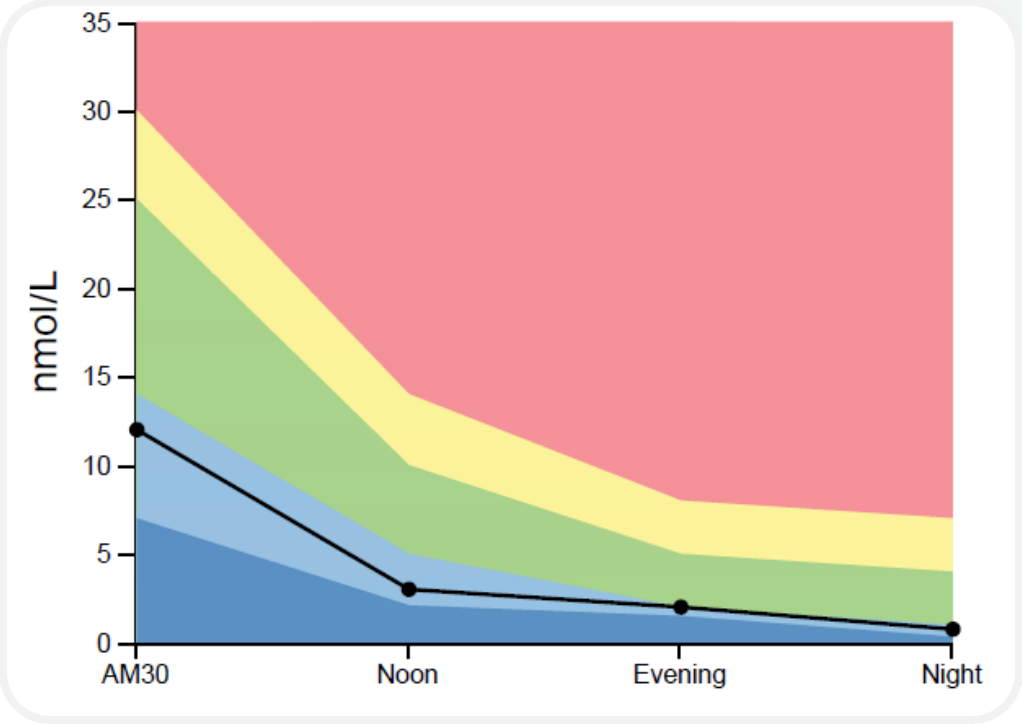


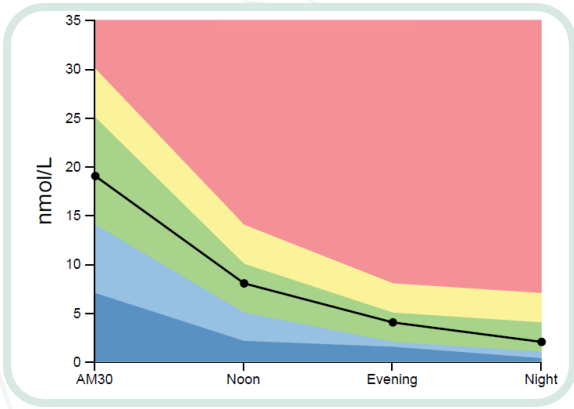
Adrenal Phase 1

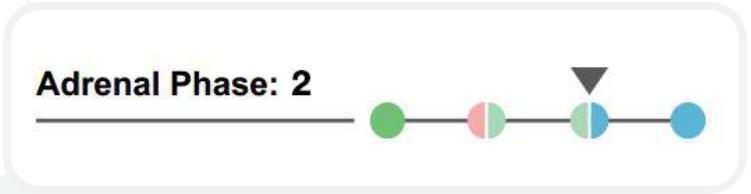




Adrenal Phase 2







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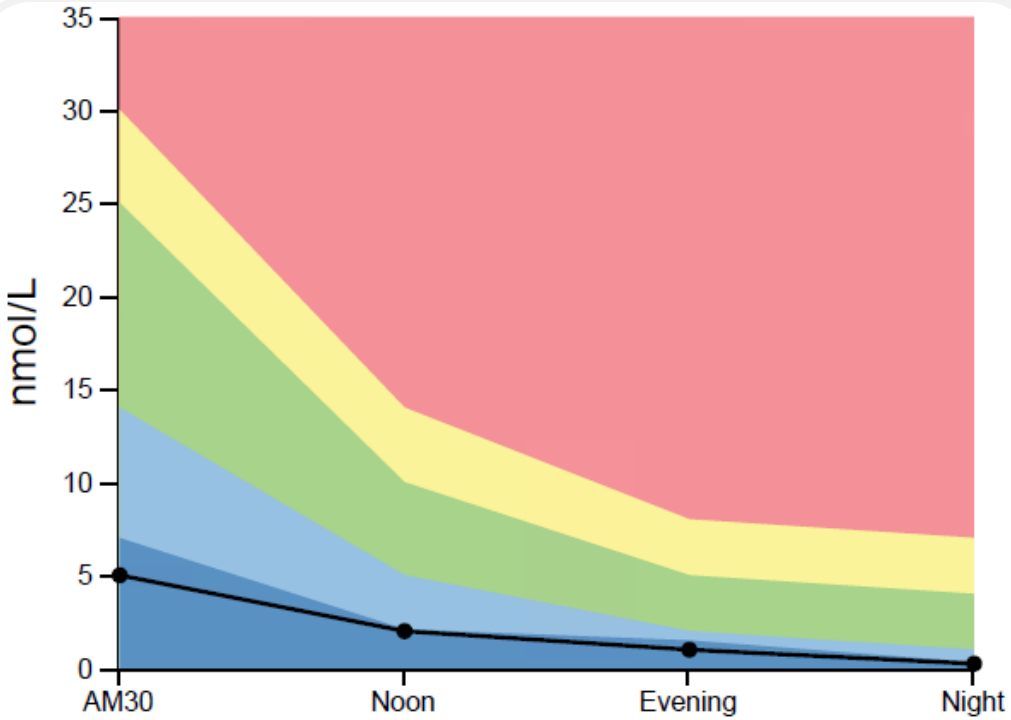
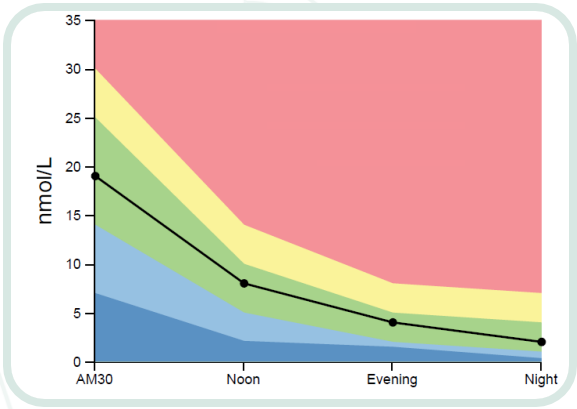
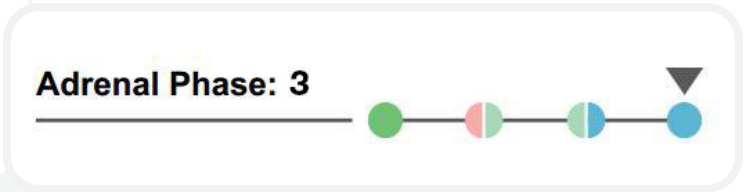
Adrenal Phase 3







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Brandtstädter J, et al. Developmental and personality correlates of adrenocortical activity as indexed by salivary cortisol: observations in the age range of 35 to 65 years. *J Psychosom Res*. 1991; 35(2-3): 173-185.

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ADRENAL TREATMENT CONSIDERATIONS

PHASE 0	• Multivitamin / Multi mineral • Omega 3 EFAs • Consider vitamin D, iodine and probiotics
PHASE 1	• Phosphorylated serine 100 mg corresponding to times with elevated cortisol • Vitamins B5 (500 mg), C (1000 mg) TID; B6 (100 mg) BID; E (800 i.u.) QD • Melatonin (0.5 - 3mg) qhs if cortisol levels elevated at night • Lifestyle modification: deep breathing, stress management, exercise, optimal diet, etc.
PHASE 2	• Vitamins B5 (500 mg), C (1000 mg) TID; B6 (100 mg) BID; E (800 i.u.) QD • Adrenal glandular and/or herbal adaptogens in morning and at noon • Lifestyle modification: deep breathing, stress management, exercise, optimal diet, etc.
PHASE 3	• Vitamins B5 (500 mg), C (1000 mg) TID; B6 (100 mg) BID; E (800 i.u.) QD • Adrenal glandular and/or herbal adaptogens in morning and at noon. • Cortef or hydrocortisone supplementation 5-10 mg in AM and 5-10 mg at noon • Lifestyle modification: deep breathing, stress management, exercise, optimal diet, etc.

But there is more to the story of evaluating the stress response…

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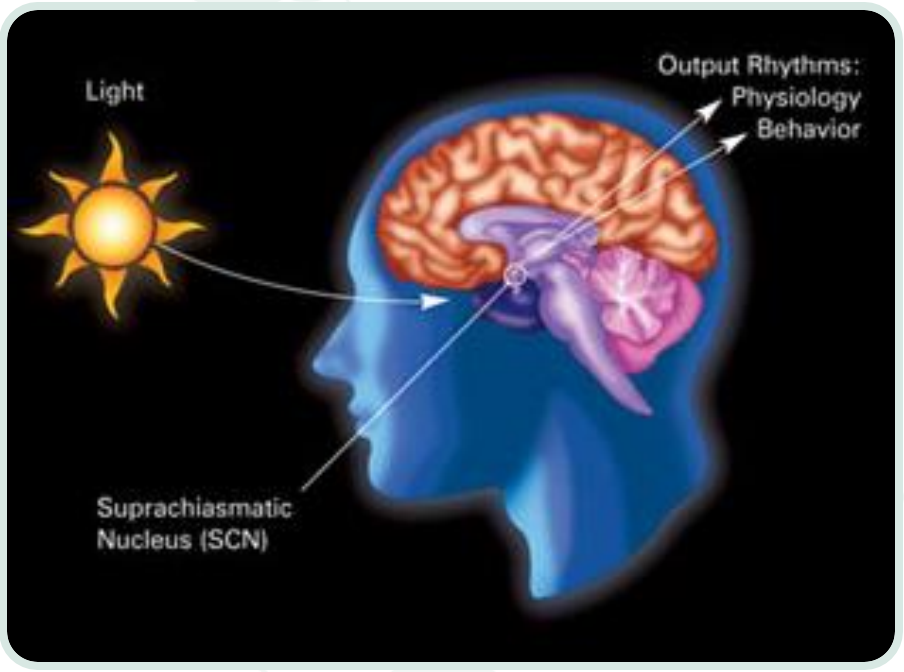


Waking and the Stress Response

Pre-awakening period:

- Cortisol is blunted
- Reduced sensitivity to ACTH, controlled by the SCN

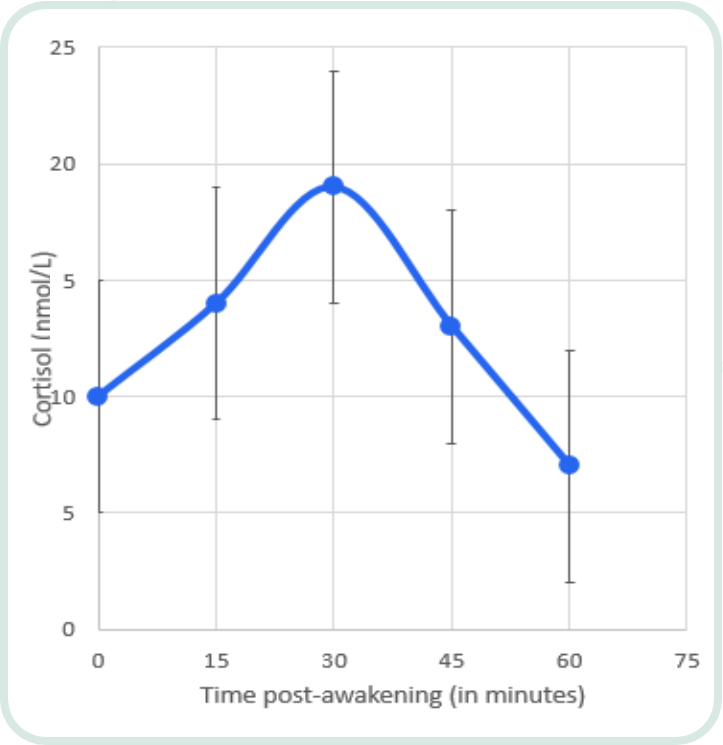
Post awakening period:

- Switch on of regional brain activity, return of consciousness
- HPA axis activation
- Release of reduced sensitivity to ACTH
- Enhanced adrenal sensitivity in the presence of light, via extra pituitary pathway from SCN.

- Clow A, Hucklebridge F, Stalder T, Evans P, Thorn L. The cortisol awakening response: more than a measure of HPA axis function. Neurosci Biobehav Rev. 2010;35(1):97-103.
- Image: https://www.nigms.nih.gov/education/fact-sheets/Pages/circadian-rhythms.aspx

Cortisol Awakening Response (CAR)

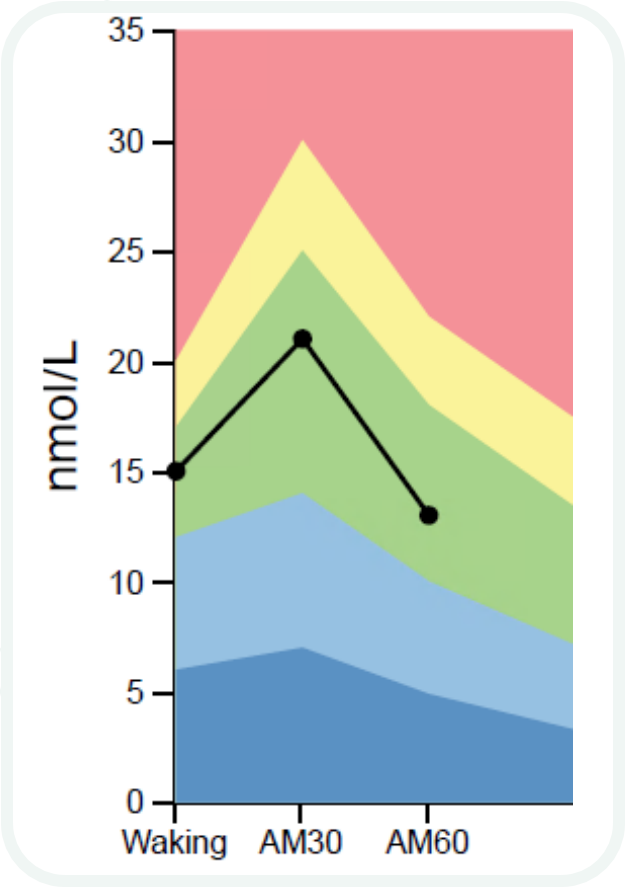
- **CAR:** the natural rise in cortisol that is seen 30 to 40 minutes after awakening followed by a noticeable drop by 60 minutes.
- CAR can be utilized as a biomarker for assessment of the HPA axis function in routine clinical practice.
- CAR is influenced by overall HPA reactivity, as well as a person's anticipation of stress



Clow A, Hucklebridge F, Stalder T, Evans P, Thorn L. The cortisol awakening response: more than a measure of HPA axis function. Neurosci Biobehav Rev. 2010;35(1):97-103.

Cortisol Awakening Response (CAR)

- Predictable rise of cortisol output (35-60%) 30 minutes post waking (AM30)
- Predictable decline(<33%) from waking to 60 minutes post waking (AM 60)



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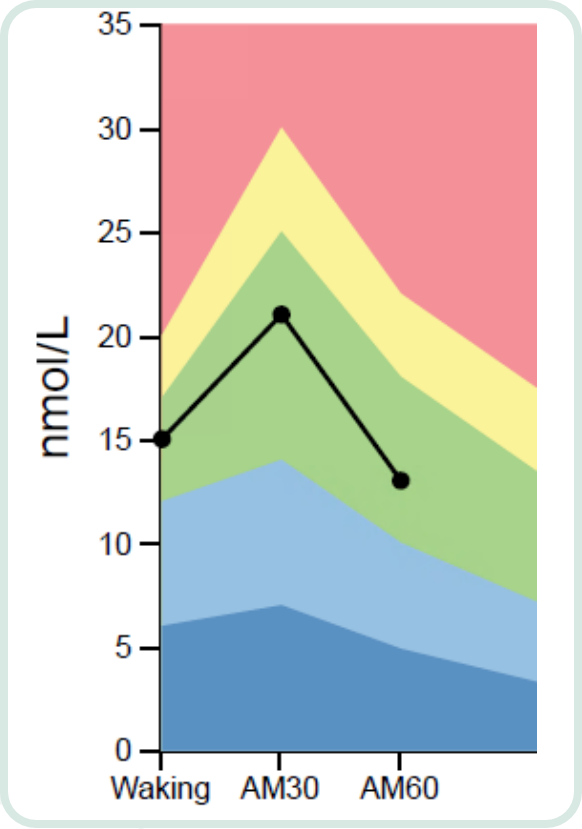
Sample CAR only report



Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol Waking	15	nmol/L		◆		12 – 17	6.0 – 20
Cortisol AM30	21	nmol/L		◆		14.0 – 25.0	7.0 – 30.0
Cortisol AM60	13	nmol/L		◆		10.0 – 18.0	4.9 – 22.0
CAR Rise *(Calculated)*	40.00	%		◆			35 – 60
CAR Decline *(Calculated)*	-13.33	%		◆			-33 – 0

- Predictable rise of cortisol output (35-60%): Waking to AM 30
- Predictable decline (<33%): Waking to AM 60

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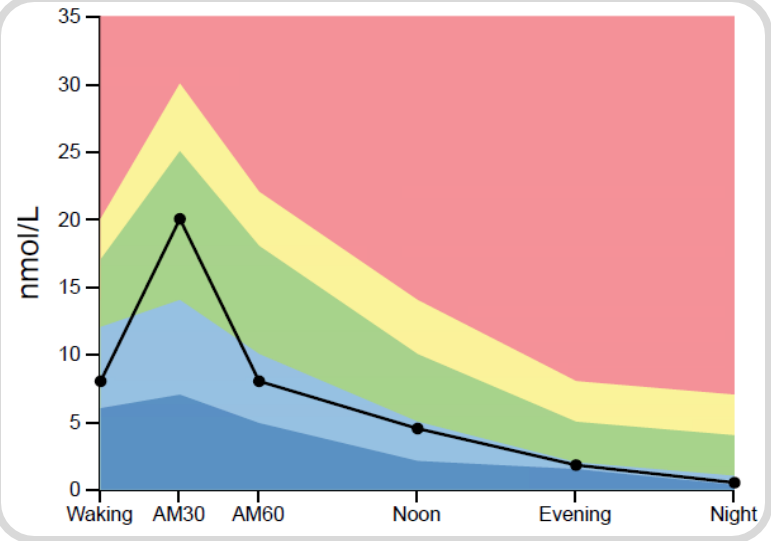
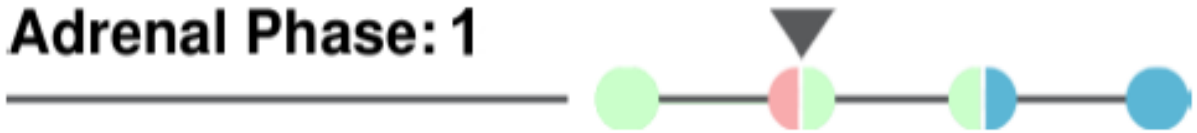
Elevated CAR



Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol Waking	8.0	nmol/L	◆			12 – 17	6.0 – 20
Cortisol AM30	20	nmol/L		◆		14.0 – 25.0	7.0 – 30.0
Cortisol AM60	8.0	nmol/L	◆			10.0 – 18.0	4.9 – 22.0
CAR Rise *(Calculated)*	150.0	%			▲		35 – 60
CAR Decline *(Calculated)*	<0.01	%			▲		-33 – 0
Cortisol Noon	4.5	nmol/L	◆			5.0 – 10.0	2.1 – 14.0
Cortisol Evening	1.8	nmol/L	◆			2.0 – 5.0	1.5 – 8.0
Cortisol Night	0.50	nmol/L	◆			1.0 – 4.0	0.33 – 7.0



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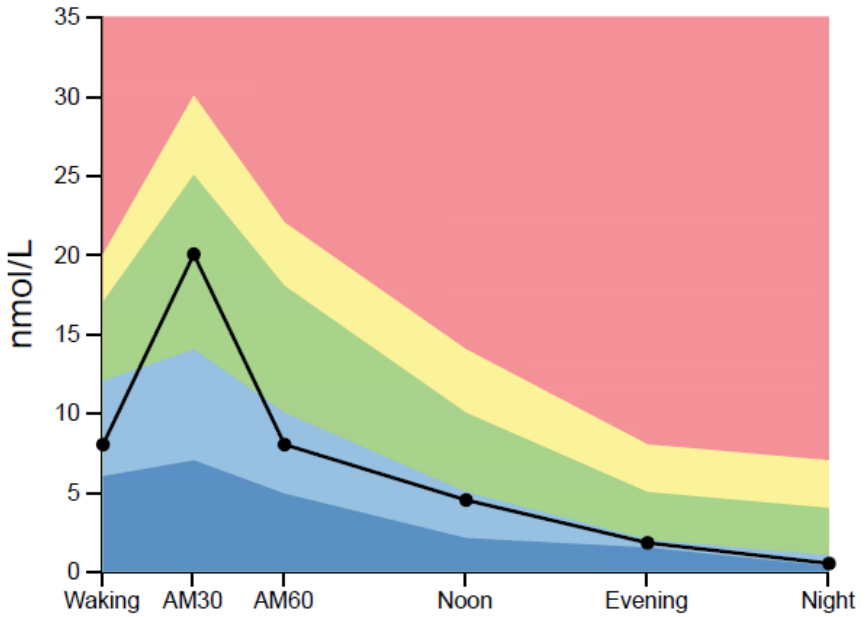
Elevated CAR

- Not necessarily maladaptive
- Ongoing job-related and perceived stress (CAR is significantly higher on work days)
- Immediate access to light upon awakening
- Depression
- Ovulation
- Sleep issues
- Older age



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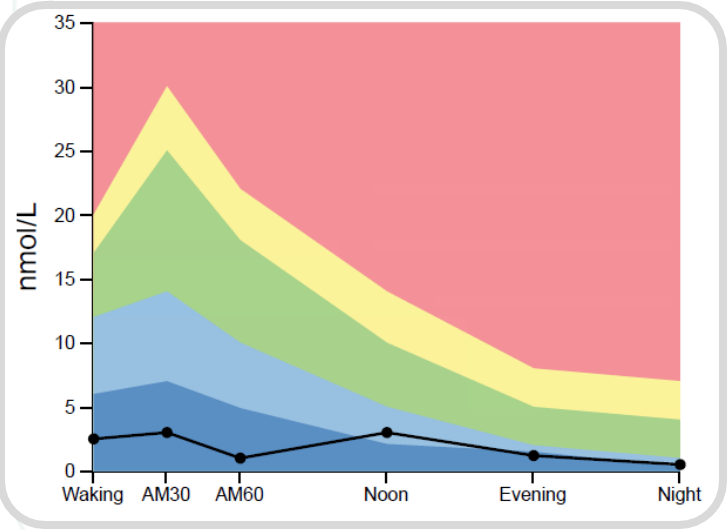
Blunted CAR



Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol Waking	2.5	nmol/L	↓			12 – 17	6.0 – 20
Cortisol AM30	3.0	nmol/L	↓			14.0 – 25.0	7.0 – 30.0
Cortisol AM60	1.0	nmol/L	↓			10.0 – 18.0	4.9 – 22.0
CAR Rise *(Calculated)*	20.00	%	↓				35 – 60
CAR Decline *(Calculated)*	-60.00	%	↓				-33 – 0
Cortisol Noon	3.0	nmol/L		◆		5.0 – 10.0	2.1 – 14.0
Cortisol Evening	1.2	nmol/L	↓			2.0 – 5.0	1.5 – 8.0
Cortisol Night	0.50	nmol/L		◆		1.0 – 4.0	0.33 – 7.0

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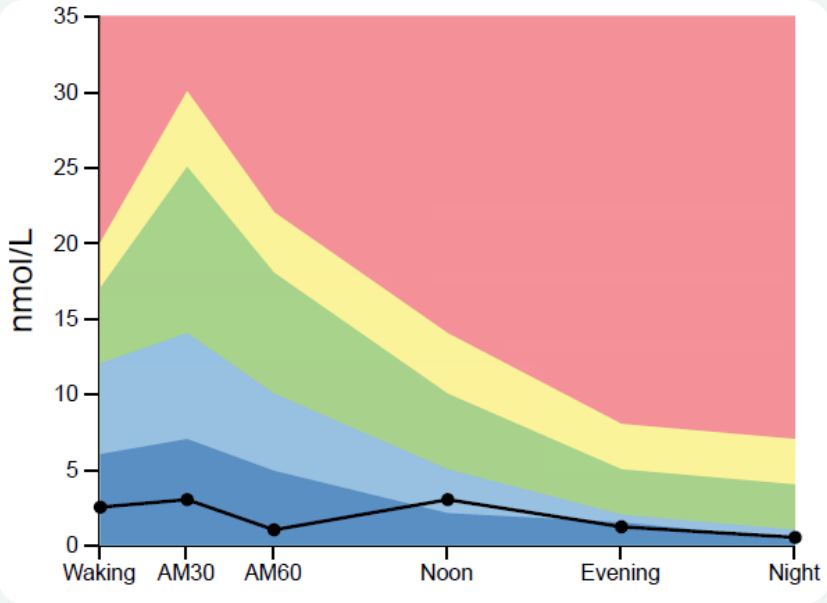
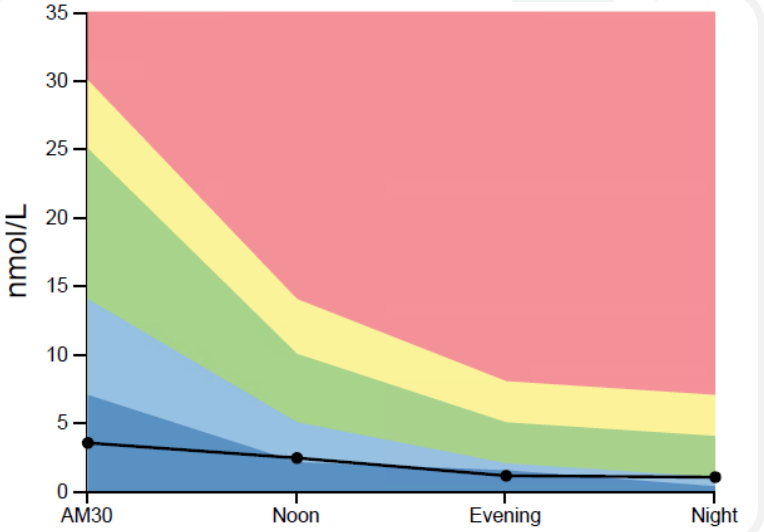
Blunted CAR

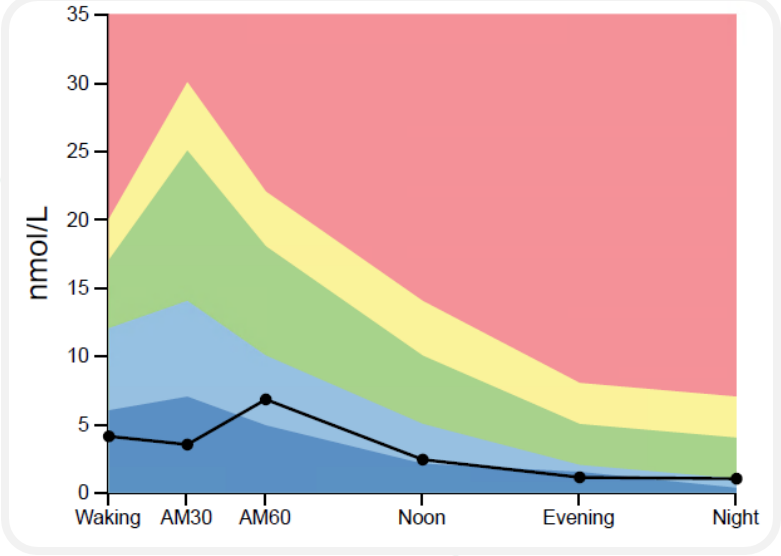
- Psychosocial burnout
- Chronic fatigue
- PTSD
- Seasonal affective disorder (during winter)
- Nightshift work schedules
- Sleep apnea
- Short sleep cycles
- Chronic inflammation
- Adrenal insufficiency
- Lack of morning sunlight exposure
- Hippocampal damage or atrophy
- Amnesia (due to temporal lobe damage)



Set Your Patients up for success: Tips for Capturing CAR

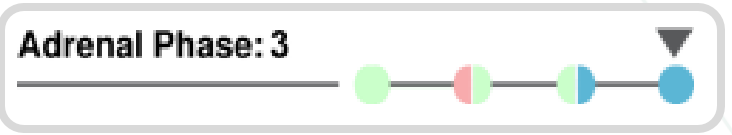
- Due to the critical timing of CAR testing, patients will need guidance
  - Set the waking collection tube on nightstand the night before.
  - Pen and paper for writing down the waking time
  - Set an alarm for 30 min and 60 minutes after the written down waking time to ensure proper sample collection times.





Case 2

Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol Waking	4.1	nmol/L	↓			12 – 17	6.0 – 20
Cortisol AM30	3.5	nmol/L	↓			14.0 – 25.0	7.0 – 30.0
Cortisol AM60	6.8	nmol/L		◆		10.0 – 18.0	4.9 – 22.0
CAR Rise *(Calculated)*	-14.63	%	↓				35 – 60
CAR Decline *(Calculated)*	65.85	%			↑		-33 – 0
Cortisol Noon	2.4	nmol/L		◆		5.0 – 10.0	2.1 – 14.0
Cortisol Evening	1.1	nmol/L	↓			2.0 – 5.0	1.5 – 8.0
Cortisol Night	1.0	nmol/L		◆		1.0 – 4.0	0.33 – 7.0
DHEA*	11	pg/mL	↓				106 – 300



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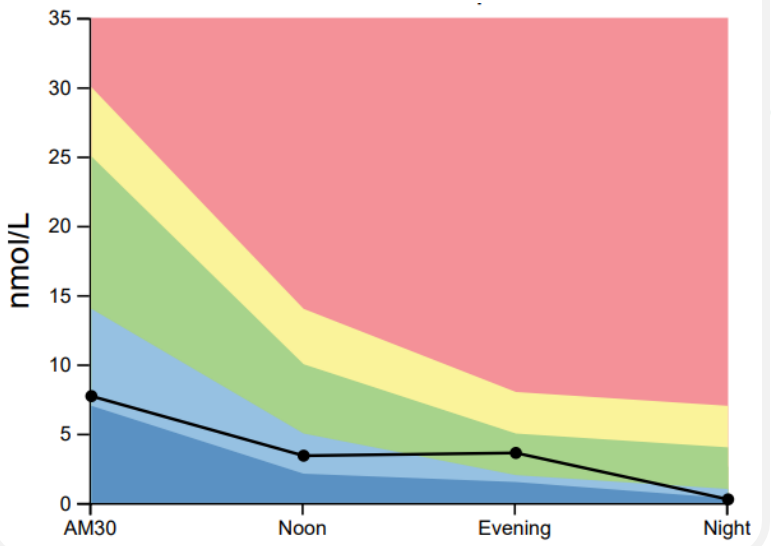
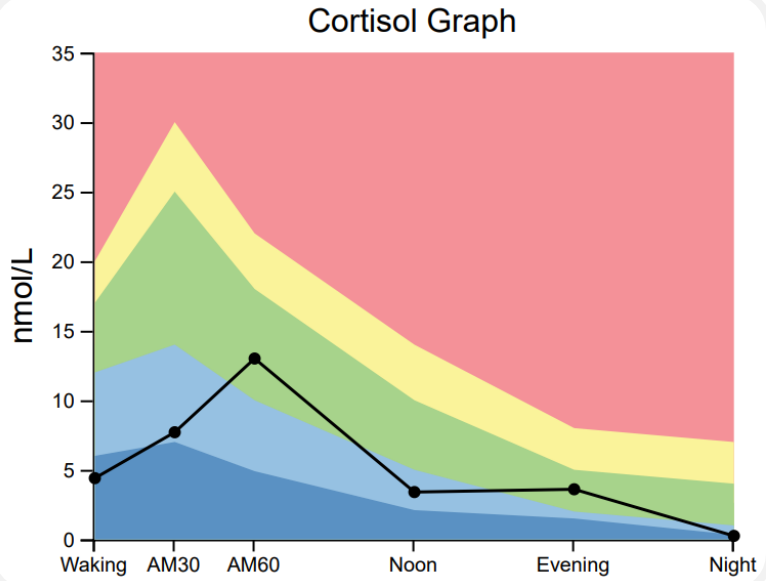
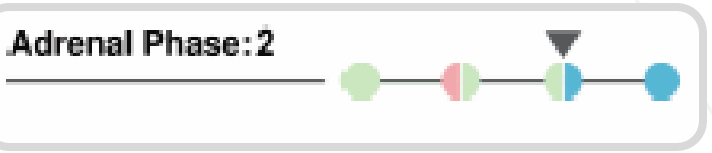




Case 3

Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol Waking	4.4	nmol/L	↓			12 – 17	6.0 – 20
Cortisol AM30	7.7	nmol/L		◇		14.0 – 25.0	7.0 – 30.0
Cortisol AM60	13	nmol/L		◇		10.0 – 18.0	4.9 – 22.0
CAR Rise *(Calculated)*	75.00	%			↑		35 – 60
CAR Decline *(Calculated)*	195.5	%			↑		-33 – 0
Cortisol Noon	3.4	nmol/L		◇		5.0 – 10.0	2.1 – 14.0
Cortisol Evening	3.6	nmol/L		◇		2.0 – 5.0	1.5 – 8.0
Cortisol Night	<0.33	nmol/L	↓			1.0 – 4.0	0.33 – 7.0



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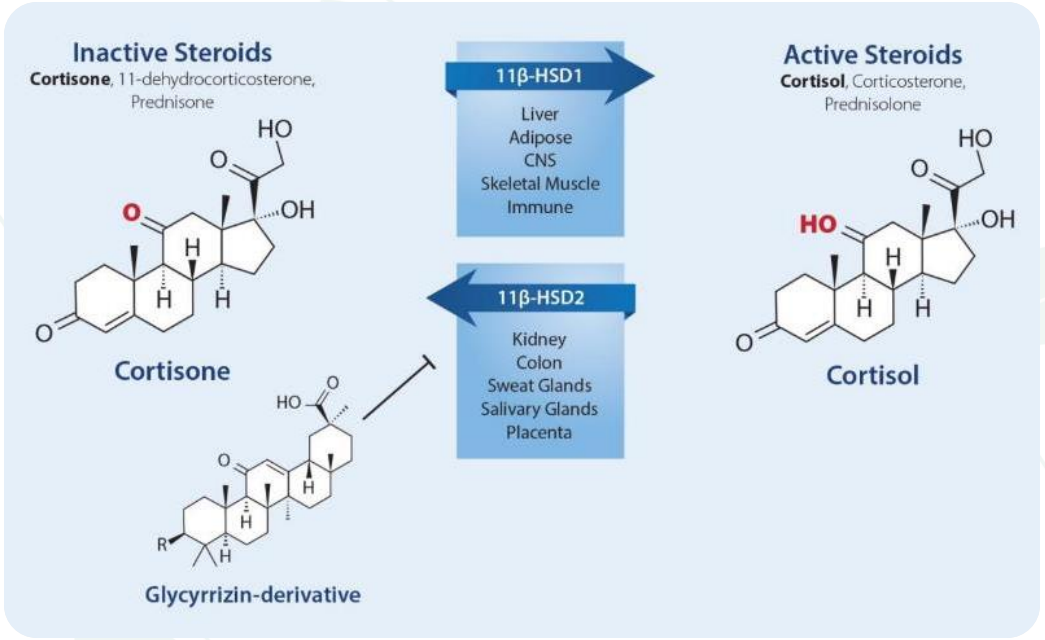
What can urinary testing add to the story?

- Cortisol (active) vs. Cortisone (storage)
  - 11BHSD enzyme: most active in the kidney
    - 11BHSD1-pulls cortisol out of storage
      - Correlated with obesity, metabolic syndrome, inflammation, thyroid function.
    - 11BHSD2- protects the mineralocorticoid receptor from aldosterone effects (normal to see 2x more cortisone than cortisol)
- Because we are looking at the influence of the kidney, we aren’t necessarily seeing the HPA axis activity directly, rather the body's response to cortisol levels



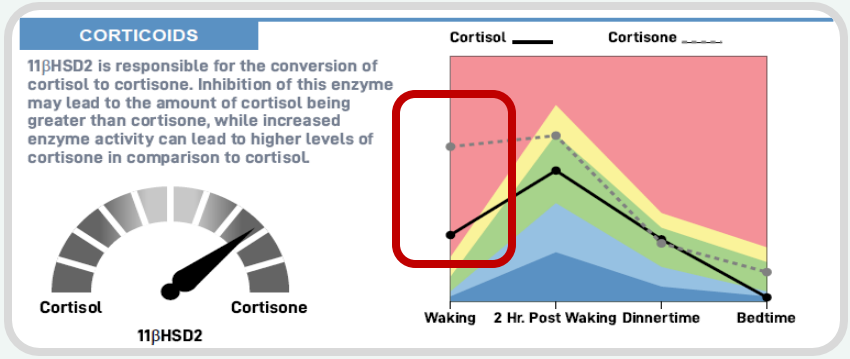
What can urinary testing add to the story?

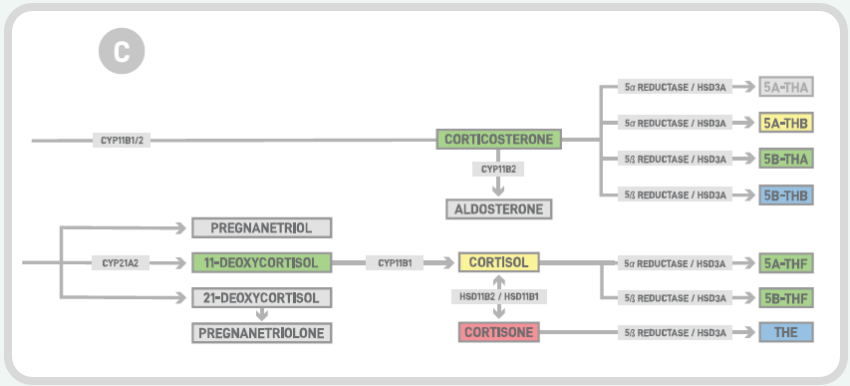
- Metabolized cortisol: (THE + 5αTHF + 5βTHF)
  - Measure of what the body has utilized
  - High metabolized cortisol: increased cortisol clearance due to over production of cortisol, also influenced by; obesity, IR, inflammation, hyperthyroidism
  - Low metabolized cortisol: decreased cortisol clearance which could also be due to obesity, inflammation, IR, hypothyroidism
- Cortisol / Cortisone metabolites: (5αTHF + 5βTHF) vs. THE
  - Because free levels of cortisol can be converted in the kidney before excretion, looking at the metabolites gives clinicians a better idea of overall preference within the body

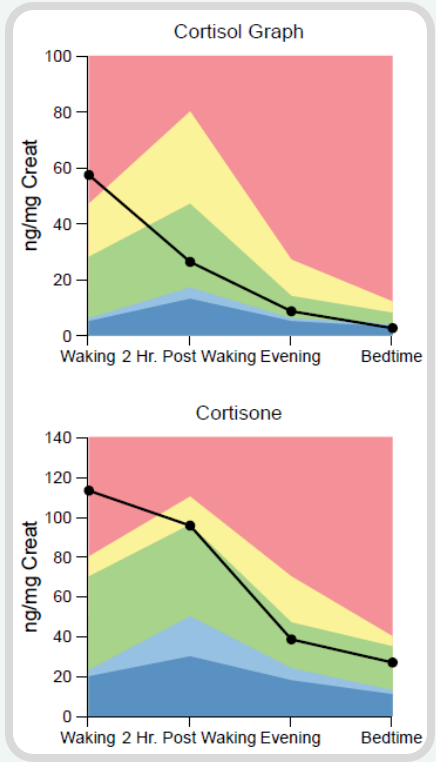
ADRENAL CORTICOIDS

HUMAP

Urinary Lab Results







THE+5A-THF+5B-THF‡	(Metabolized Cortisol)	3320	ng/mg Creat/Day	2500 – 7900
5A-THF+5B-THF/THE‡	(Cortisol/Cortisone Metabolites)	1.44		0.7 – 1.4
Cortisol/Cortisone‡	(11B HSD activity)	0.335		0.3 – 0.8

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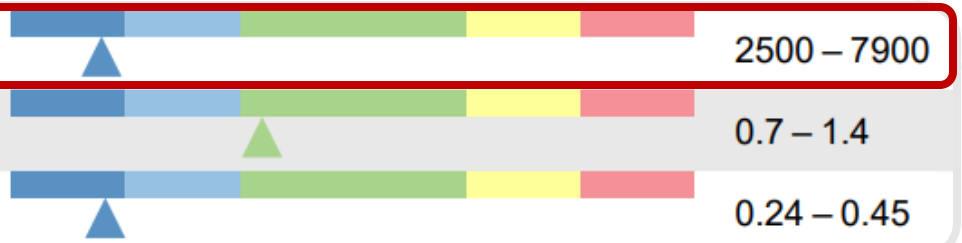
Low cortisol metabolites

- Don’t necessarily need to support cortisol creation-reflex to salivary testing
- Focus on potential cause (i.e. inflammation, hypothyroidism, obesity, insomnia)

THE+5A-THF+5B-THF‡	(Metabolized Cortisol)	1970	ng/mg Creat/Day	2500 – 7900
5A-THF+5B-THF/THE‡	(Cortisol/Cortisone Metabolites)	0.840		0.7 – 1.4
Cortisol/Cortisone‡	(11B HSD activity)	0.197		0.24 – 0.45



Cortisol Testing Summary

- Salivary testing
  - Diurnal Cortisol
    - Basal testing, good for baseline
    - Gold standard
    - Circadian compliance
  - CAR
    - Provocative testing
    - HPA axis function (plasticity / reactivity)
- Urinary testing
  - Cortisol vs. Cortisone: 11BHSD enzyme activity tells us if the body is activating or inactivating cortisol
  - Metabolized cortisol: Total measure of what the body has utilized (HPA axis output)
  - Cortisol vs. cortisone metabolites: metabolic preference
  - Morning cortisol sample representative of over night secretion

DIURNAL CORTISOL

CAR PROFILE

ADRENAL CORTICOIDS

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COMPREHENSIVE HORMONE

ADRENAL FUNCTION

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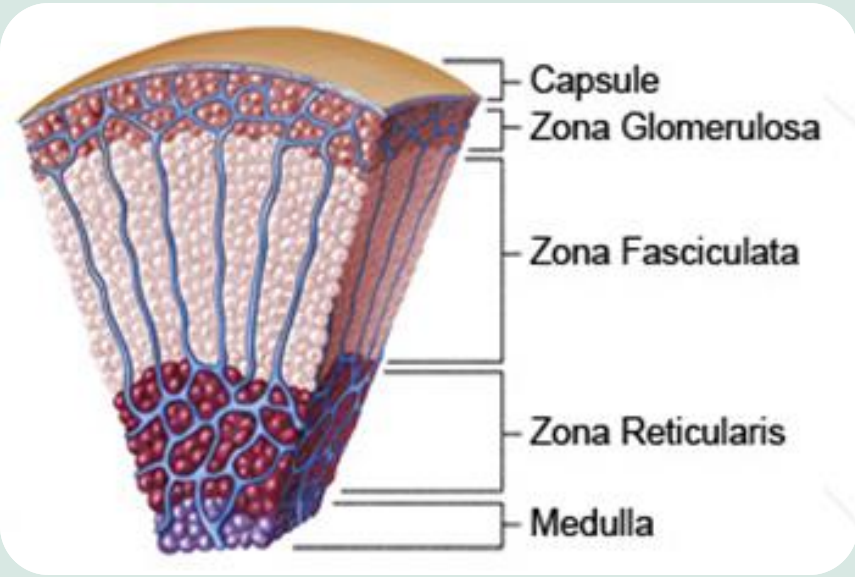
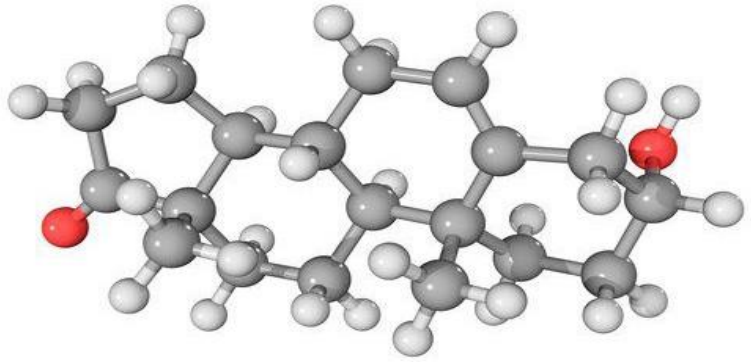
Zones and Hormones of the Adrenal Cortex

- **Zona Glomerulosa** – aldosterone
- **Zona Fasciculata** – "the glucocorticoids"
  - 11-deoxycorticosterone
  - Corticosterone
  - Cortisol
- **Zona Reticularis-**"androgens"
  - DHEA
  - DHEA sulfate
  - Androstenedione (precursor to T)



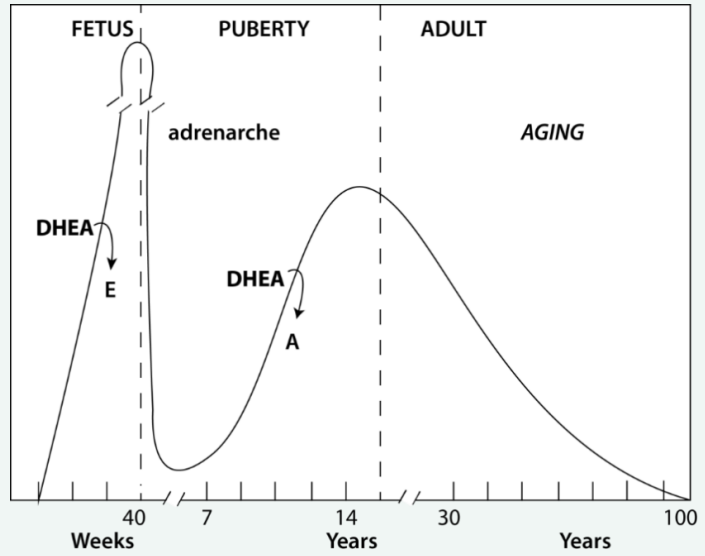
Role of DHEA



- Sexual development
- For many years scientists thought that DHEA production in adults functioned only as a precursor reservoir for testosterone and estrogens.
- However, it is now well understood that in adulthood DHEA can be secreted in response to ACTH stimulation just as cortisol is. Therefore, it also plays a role in the **stress response**.

Adrenopause

- DHEA levels from the adrenal gland begin to peak at around age 20-25 and then gradually begin to drop. This decline has been termed ADRENOPAUSE
- Levels of aldosterone and cortisol do not drop however and remain somewhat constant throughout life.



DHEA and health

Roth et al. showed that the lifespan of a person and the serum concentrations of DHEA are positively related.

- There are also reports suggesting DHEA has…
  - Anti-atherosclerotic
  - Anti-diabetic
  - Anti-inflammatory
  - Anti-osteoporotic
  - Anti-depressant
  - Anti-senility functions

Low Salivary DHEA

DHEA*	67	pg/mL	↓	106 – 300

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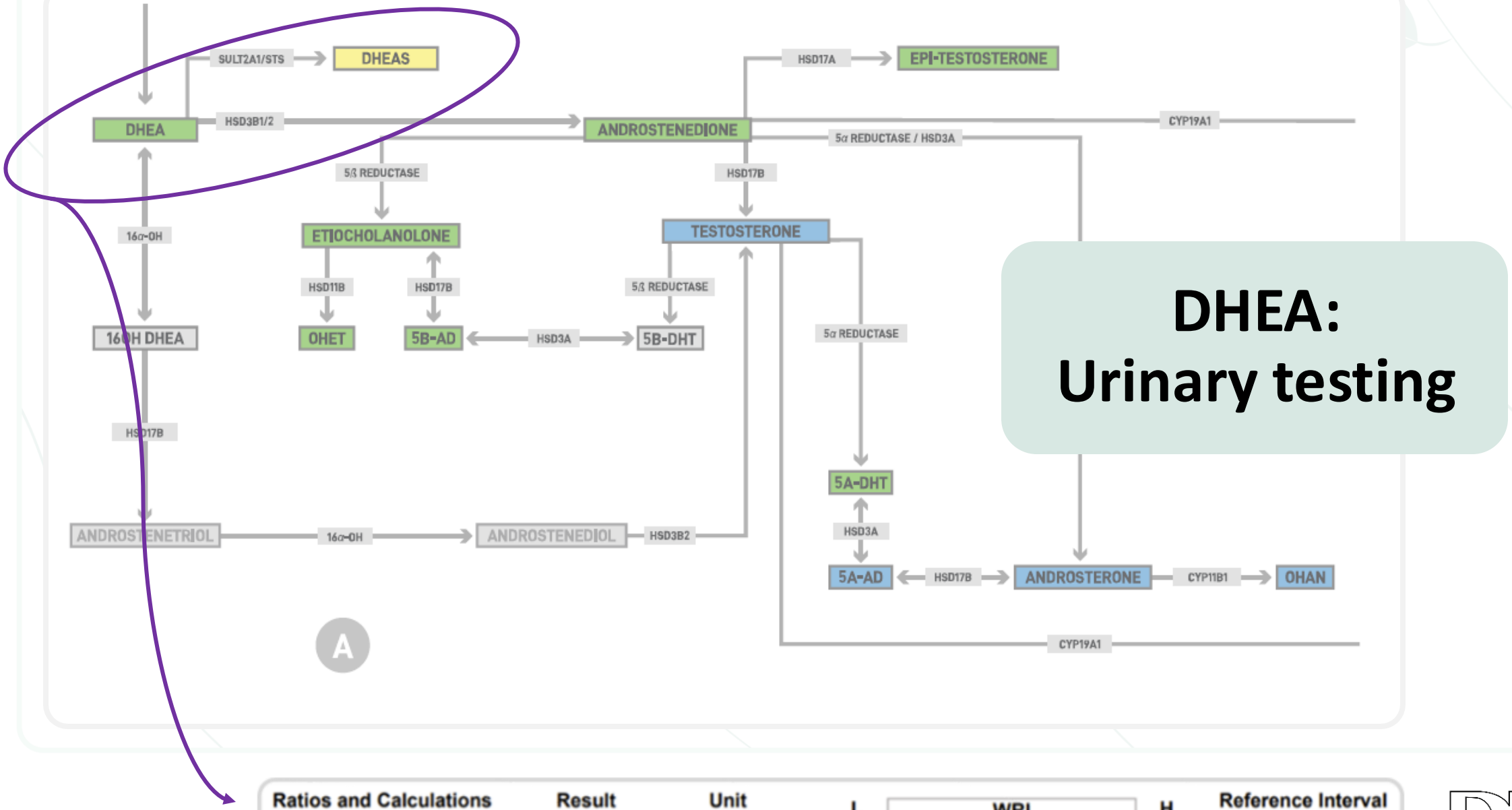

DHEA: Urinary testing

Ratios and Calculations	Result	Unit	L	WRI	H	Reference Interval
DHEA+DHEAS‡	620	ng/mg Creat/Day				50 – 2000

DHEA supplementation does NOT increase cortisol levels!

- Nor will it strengthen the area of the adrenal glands that makes cortisol (zona fasciculata) or exert negative feedback on the HPA axis.

DHEA Dosing

- A male with Addison's disease requires 50-70 mg
- Physiologic (not replacement) doses will likely not exceed:
  - Males – 15 to 50 mg/day
  - Females – 2 to 25mg/day
- Starting dose: 10 mg (males) and 5 mg (females).
- Route of administration: oral, SL, topical*
  - *When DHEA is combined with other hormones in a cream it may interfere with their absorption, especially progesterone.

Hunt PJ, et al . Improvement in mood and fatigue after dehydroepiandrosterone replacement in Addison's disease in a randomized, double blind trial. *J Clin Endocrinol Metab*. 2000; 85: 4650-6.

DHEA supplementation is <u>not</u> for children!

- Children's adrenal glands will not produce DHEA as their zona reticularis in the cortex is at rest
- **Never give a child DHEA –it can promote early puberty and cause the epiphyseal plates to fuse** …which can lead to reduced height.

HPA axis Summary

- Understand that you are not measuring “adrenals” but the HPA axis, both stress-induced alterations and the patient’s circadian rhythms.
- Cortisol Awakening Response (CAR) can be used as a provocation test to elucidate a patient’s reaction to stressful stimuli as well as anticipatory and perceived stress
  - Timing is crucial for interpretation
- Urinary metabolite testing may give us more insight into how the body utilizes cortisol including how metabolic processes affect cortisol
- DHEA is an important marker of adrenal health and is independent of feedback mechanisms seen with cortisol- supplementing DHEA will not improve cortisol

Neurotransmitters

COMPREHENSIVE
NT PROFILE

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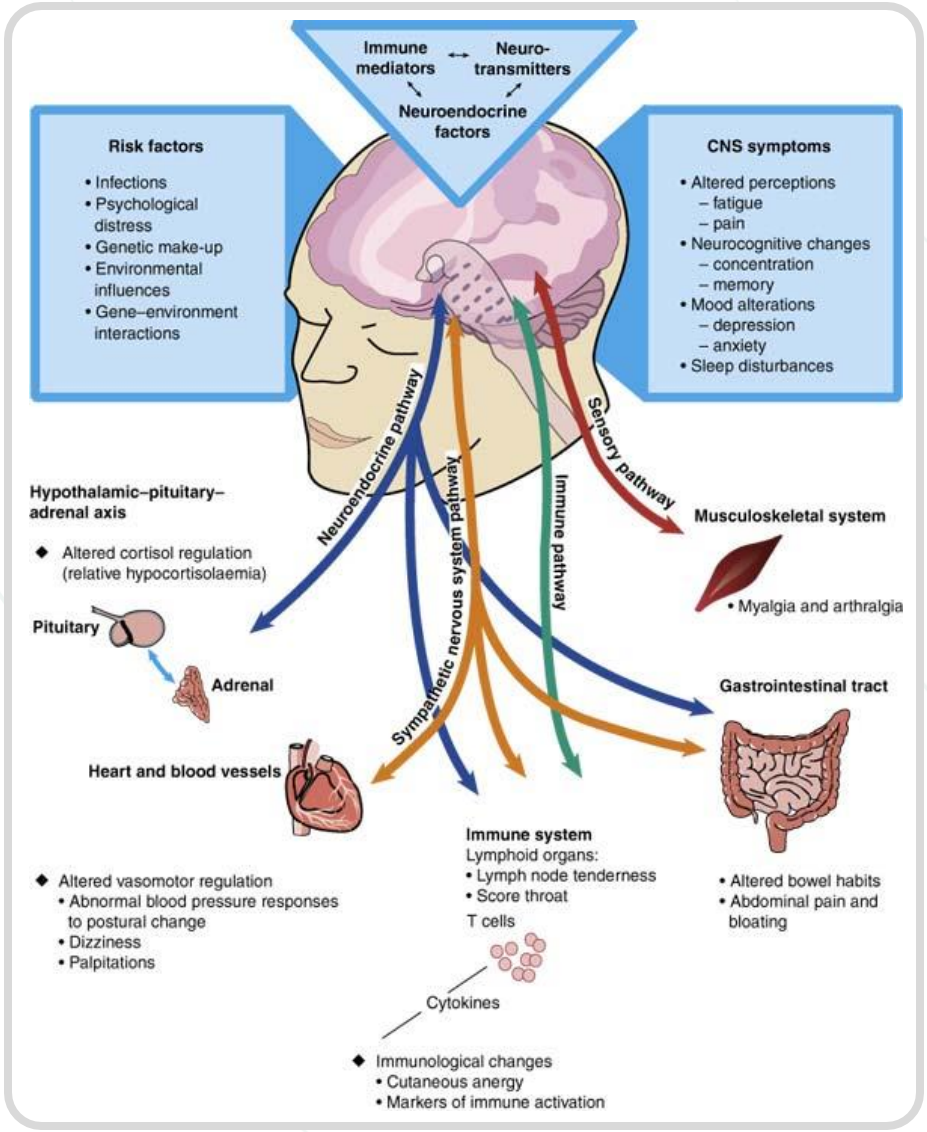
Receptors for neurotransmitters exist in many systems **throughout the body, not just in the CNS**



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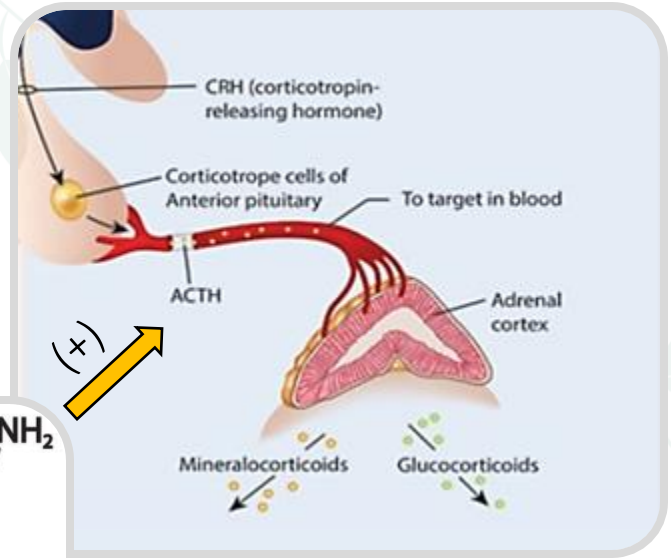
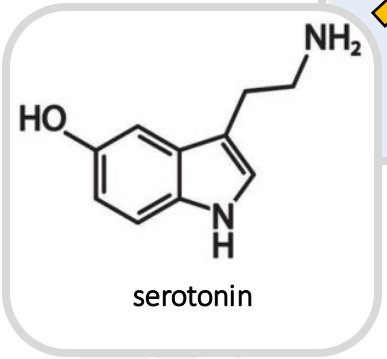
Serotonin and the HPA axis

- Serotonin is involved in healthy HPA axis function as it facilitates the release of ACTH by the pituitary, which then promotes release of cortisol and DHEA.
- Low serotonin, therefore, can contribute to depleted cortisol.

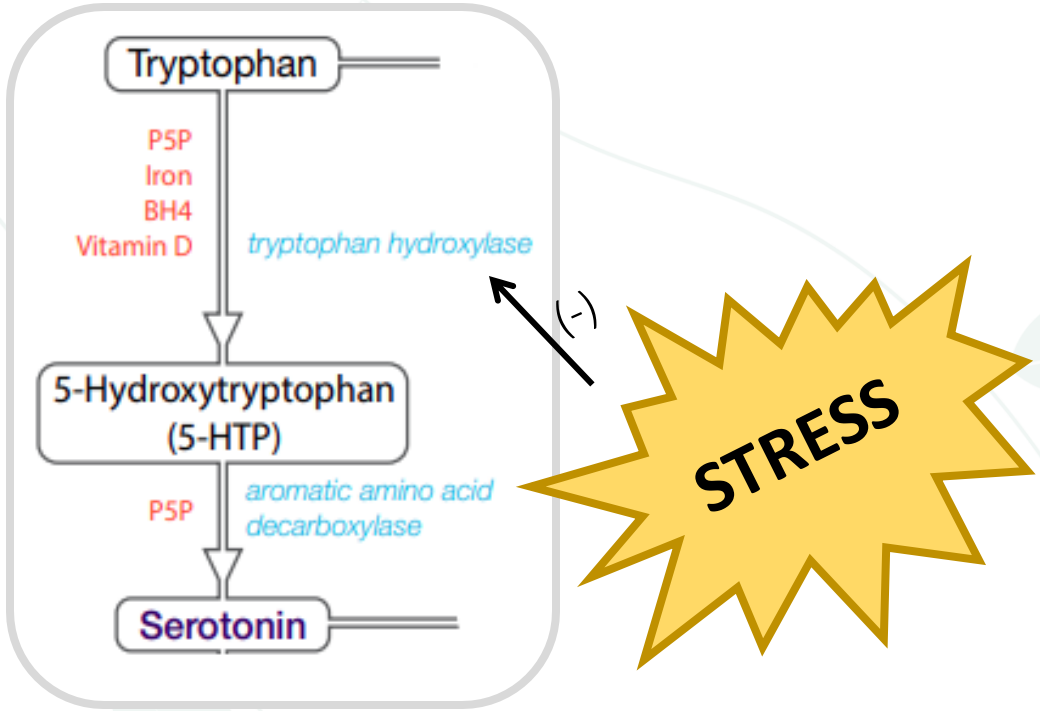




serotonin

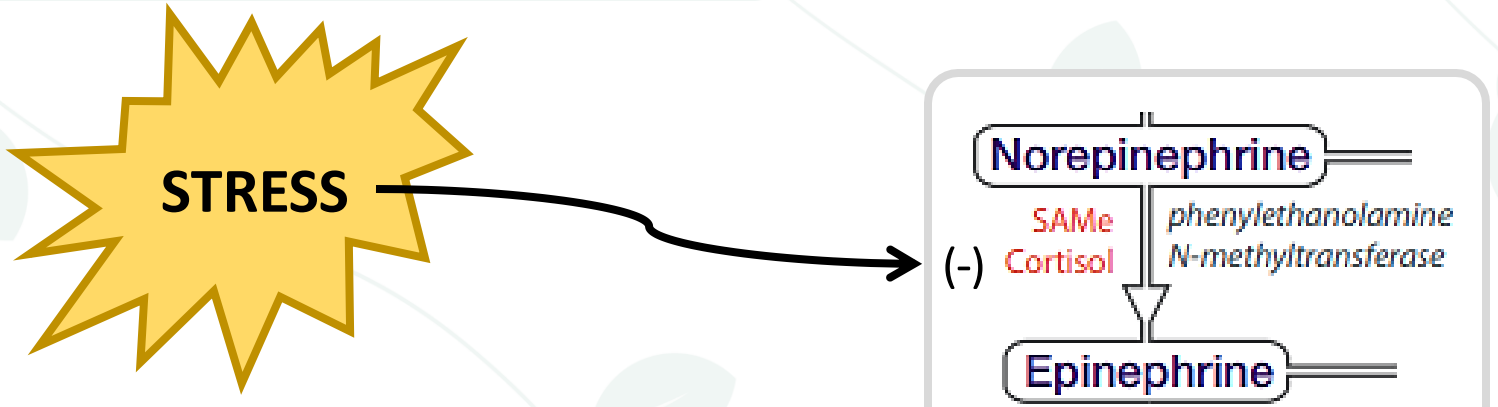
Serotonin and the HPA axis

- Stress inhibits the conversion of tryptophan to 5-HTP, which is the rate limiting step in the production of serotonin.



Heilser LK, et al. Serotonin activates the hypothalamic-pituitary-adrenal axis via serotonin 2 C receptor stimulation. J Neurosci. 2007;27:6956-64. 3. Lieberman, Michael PhD, Marks, Allan MD. Basic Medical Biochemistry: A Clinical Approach. Philadelphia: Wolters Kluwer. 2013.

Chronic Stress



Norepinephrine	22	µg/g	22 – 50
Epinephrine	0.6	µg/g	1.6 – 8.3
Norepinephrine / Epinephrine ratio	36.6		< 13

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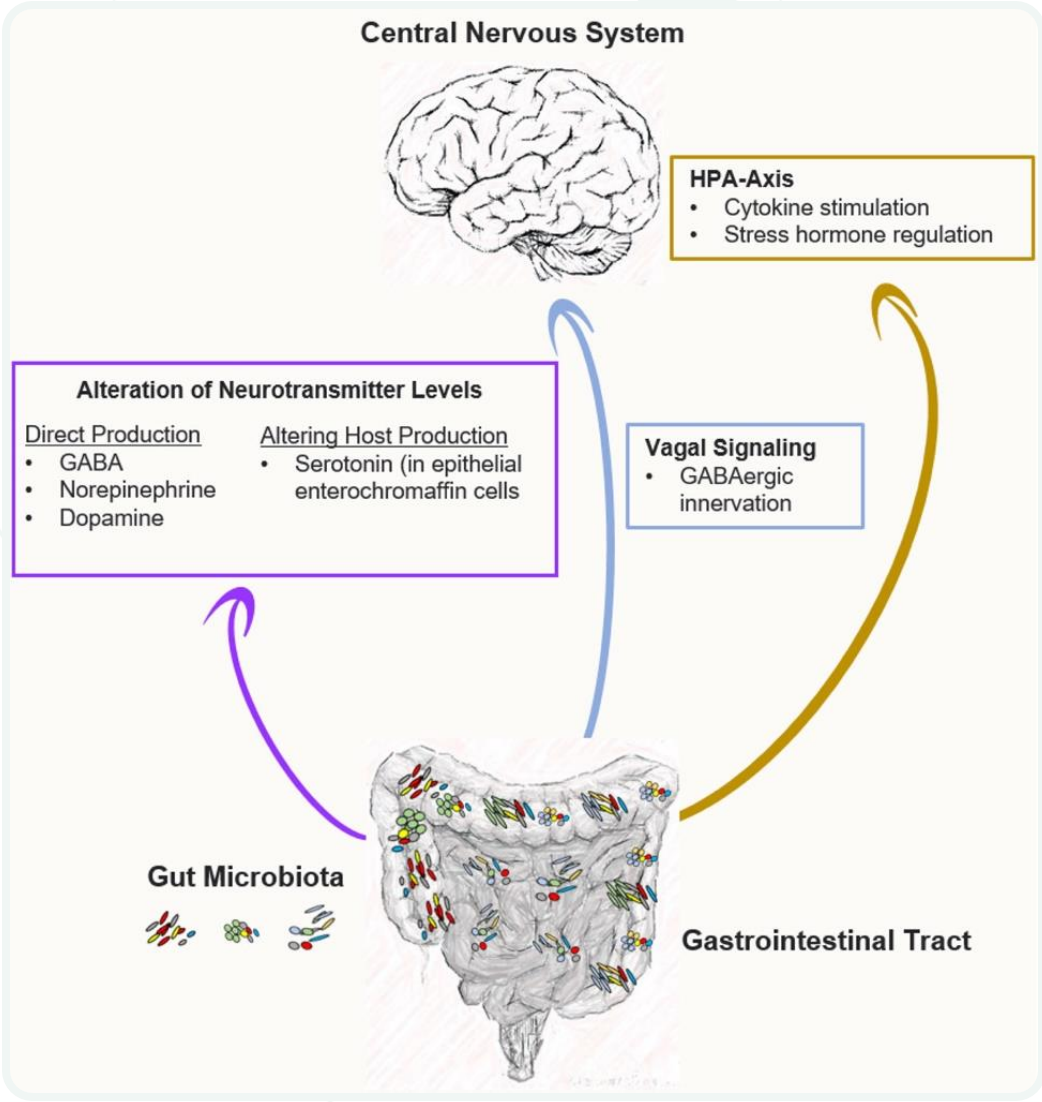
Stress and Gastrointestinal Health

“The Second Brain”



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Brain Res. Author manuscript; available in PMC 2019 Aug 15 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6005194/

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Stress = intestinal permeability

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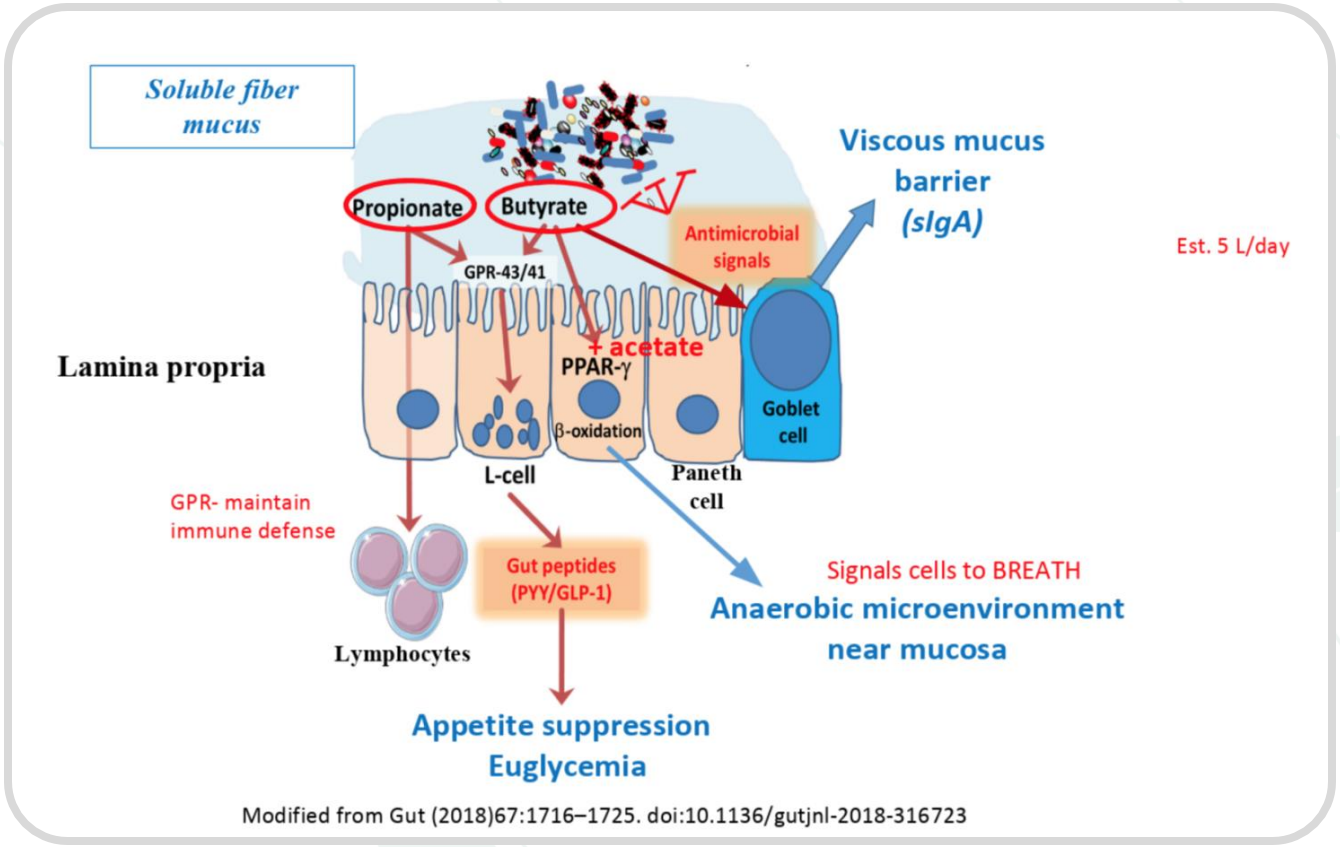
Stress & Intestinal Permeability

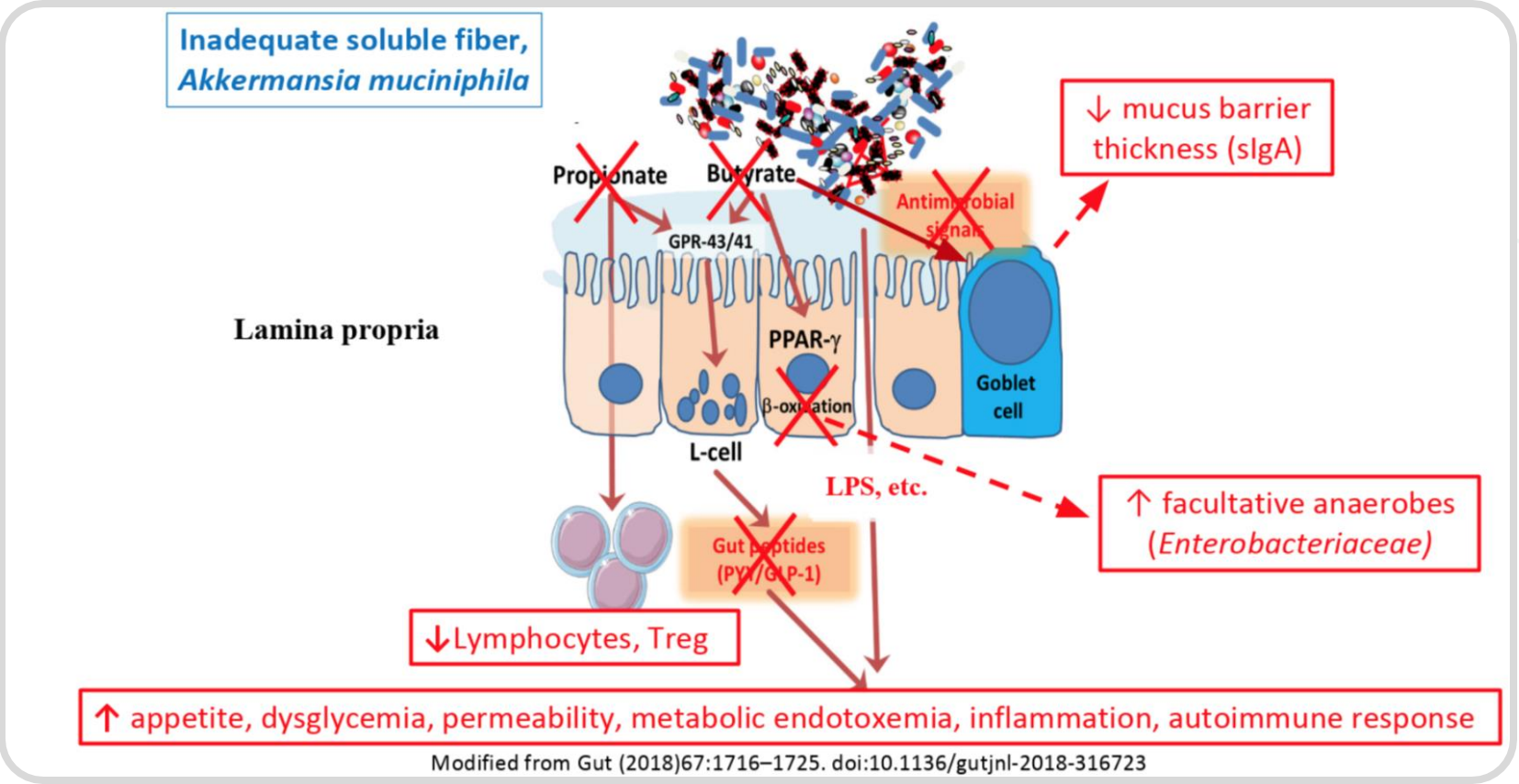
- Norwegian army undergoing training in the Arctic
- Stress & anxiety affect intestinal microbiota composition (e.g., decrease diversity, increase abundance of proinflammatory taxa, and decrease abundance of beneficial taxa) and increase intestinal impermeability. Leads to inflammation and GI distress
- Intestinal microbiota responses were characterized by increased α-diversity and changes in the relative abundance of >50% of identified genera, including increased abundance of less dominant taxa at the expense of more dominant taxa such as *Bacteroides*

https://doi.org/10.1152/ajpgi.00066.2017

Healthy microbial host crosstalk



Dysbiosis induced breakdown



Modified from Gut (2018)67:1716–1725. doi:10.1136/gutjnl-2018-316723

Intestinal permeability

- Lifestyle - alcohol, drugs *(prednisone, **NSAIDs, PPIs**)*
- Gut-derived protein fragments (gliadin) and endotoxins
- Epithelial cell damage- environmental toxins *(e.g. glyphosate)*
- Cancer treatments/radiation
- Associated with common additives, including sugar, metal oxide nanoparticles (e.g. titanium dioxide, a white colorant in yogurt etc.), surfactants and sodium chloride
- Intestinal permeability can induce immune and inflammatory responses that exacerbate intestinal barrier damage and further increase IP
- Increased intestinal permeability has been correlated with aging and disease, including type 2 diabetes, Crohn's disease, celiac disease, multiple sclerosis and irritable bowel syndrome.

- BMC Gastroenterol (2014)14:189 Scientific Rep(2014)4:5551 DOI: 10.1038/srep05551 Clin Gastroenterol Hepatol(2012)10:1096-1100 World J Gastroenterol(2014)20:17107-14
- Curr Nutr Food Sci (2013)9:99–107 Gastroenterol (2015)148 doi: 10.1016/S0016-5085(15)30192X

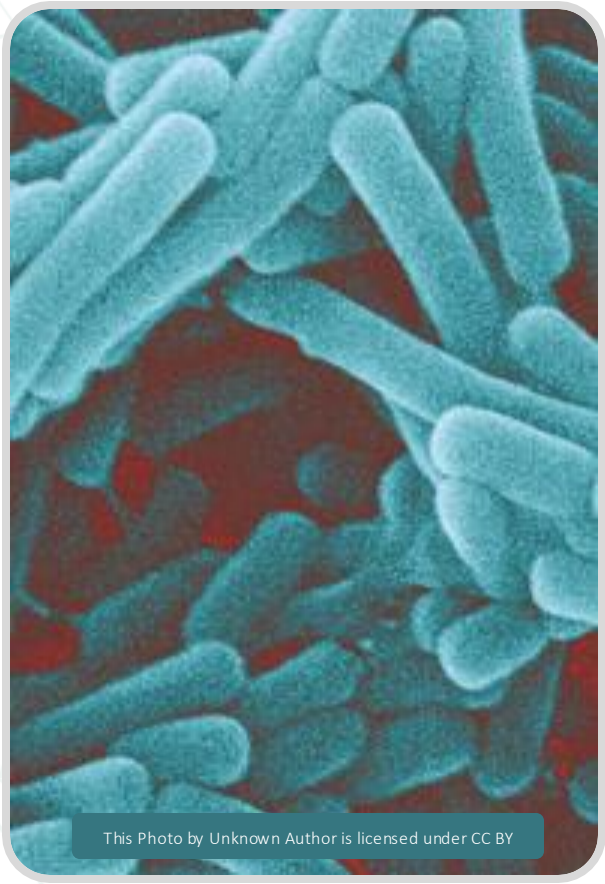
Restoration of the gastrointestinal mucosal barrier

- Dietary changes, GF
- Treatment of dysbiosis
- Digestive supports and anti-inflammatory therapies
- Quercetin, vitamin D, retinol, curcumin, gamma-linolenic acid, omega-3 fatty acids (EPA, DHA), and aloe vera
- Zinc, beta-carotene, pantothenic acid, and L-glutamine may provide some support for rejuvenation of the GI mucosa

- BMC Gastroenterol(2014)14:189 (www.biomedcentral.com/1471-230X/14/189)
- Br J Nutr(2003)89:597-606 Frontiers Immunol(2015) doi: 10.3389/frimmu.2015.00612n

Stress, Bifidobacterium & Lactobacillus

- Stress caused by maternal separation of rhesus monkeys changed their microbiome via decreased *Bifidobacterium* and *Lactobacillus*
- Rat pups separated from their mother exhibited decreased *Lactobacillus*
- *Lactobacillus* remained decreased into adulthood
- Intestinal dysbiosis and subsequent chronic low-grade inflammation follow



- Alper E., & Ceylan M., (2015) The Gut-Brain Axis: The Missing Link in Depression. Clin Psychopharmacol Neurosci. doi: 10.9758/cpn.2015.13.3.239
- Campos-Rodriguez, R., et all. Stress modulates intestinal secretory immunoglobulin A. Frnters Intgrtv Nurosci. 02 December 2013.

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Stress Eating

- Using food as a substance, rather than sustenance
- Emotional eating means that your emotions — not your body — dictate when and how much you eat
- High-carbohydrate, high-calorie, ultra-processed foods with low nutritional value
- Opioid response to gluteomorphins (gluten) & caseomorphins (milk)
- Sugar is more addictive than cocaine (dopamine influence, microbiome abundance)
- 19% of US adults report engaging in these behaviors weekly or more; may begin as age 8

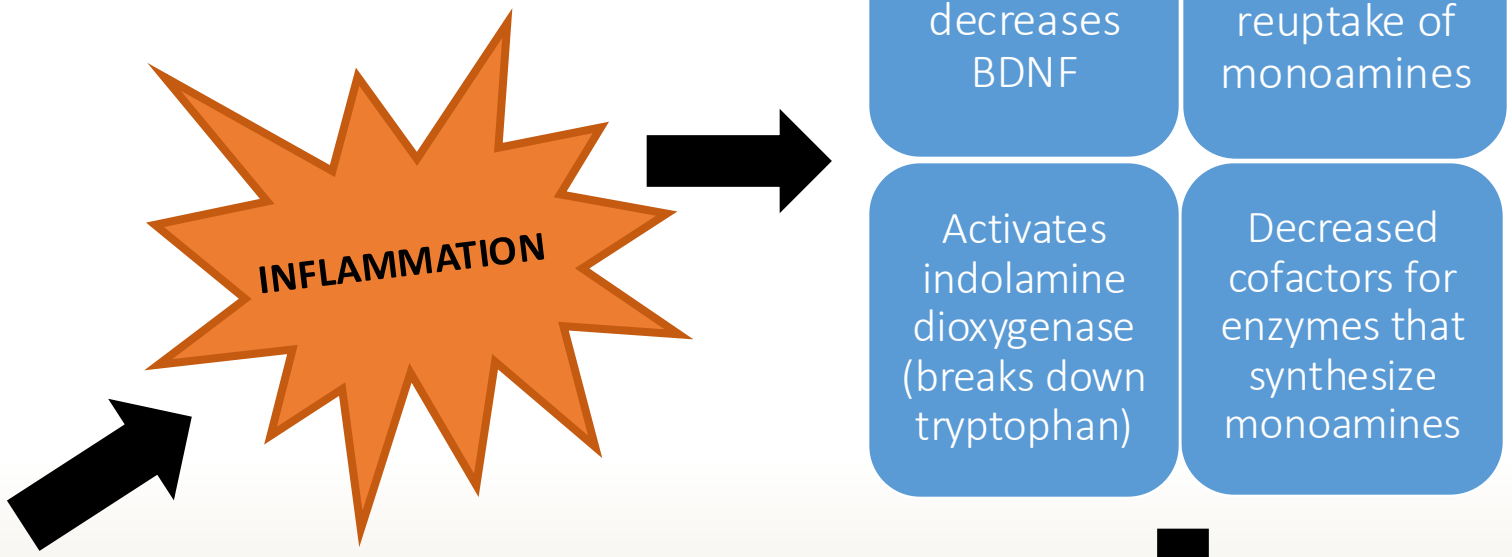
https://www.apa.org/news/press/releases/stress/2013/eating
https://www.sciencedirect.com/science/article/abs/pii/S0195666317309625?via%3Dihub

Inflammation

- A low-fiber, high-protein, and high-fat diet has been reported to increase intestinal inflammation
- Dysbiosis is associated with inflammation, increased metabolic and immune disorders
- An impaired intestinal barrier can increase the translocation of the Gram-negative bacteria cell membrane component LPS into circulation, which results in metabolic endotoxemia and low-grade inflammation

Table 1. Factors associated with increased inflammation

Obesity
Sedentary lifestyle
Disordered sleep
Childhood maltreatment
Emotional and physical trauma
Medical illnesses (eg, cardiovascular disease, diabetes, cancer, autoimmune and inflammatory disorders)
Bacterial or viral infection (including exposure to a high pathogen load [eg, unsanitary living conditions, poor dentition, poor hygiene])
Medical treatments (eg, surgery, radiation, chemotherapy)
Antidepressant treatment resistance



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Miller AH, Five Things to Know About Inflammation and Depression. **Psychiatric Times**, Vol 35 No 4, Volume 35, Issue 4. 30 April 2018.

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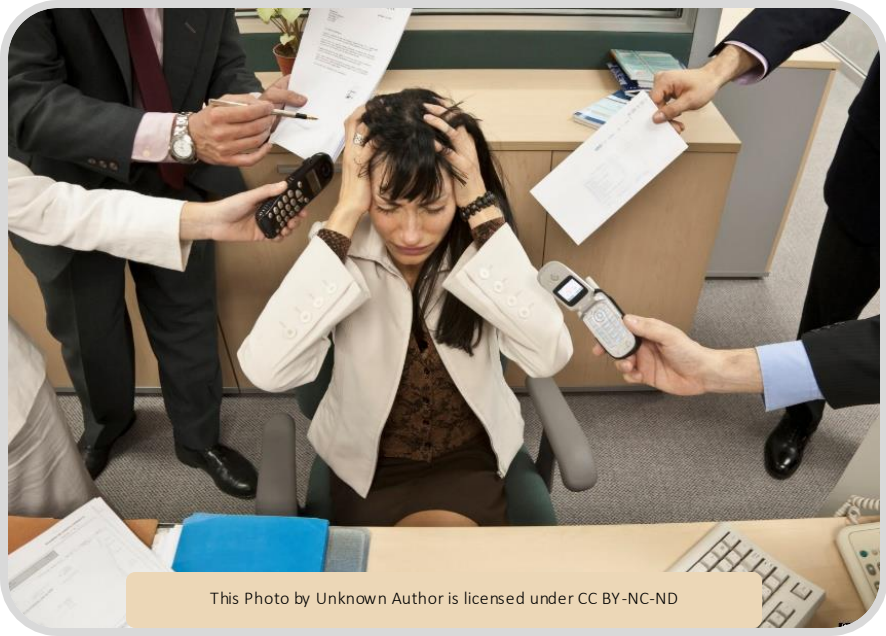
Stressed Gut Case

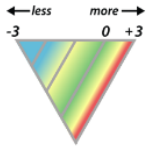


- 57 yo f
- Typical daily intake:
  - B: Greek yogurt with granola
  - Snacks: Almonds, chocolate, apples and peanut butter
  - L: Grilled Chicken Salad
  - D: Baked salmon, veggies and rice
  - Occasionally binges late night on “healthy food” in the pantry
- Rates job stress 9/10
  - “I'm constantly under a microscope, juggling major decisions, endless meetings…it's exhausting, and sometimes it feels like there's no room to breathe. I can barely keep up, let alone step back and reflect.”

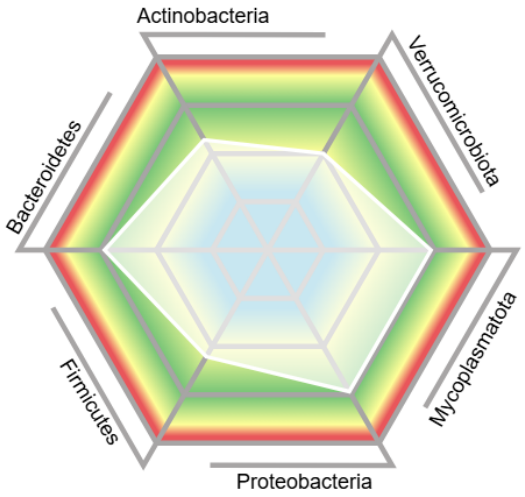
Microbiome Abundance and Diversity Summary

The abundance and diversity of gastrointestinal bacteria provide an indication of gastrointestinal health, and gut microbial imbalances can contribute to dysbiosis and other chronic disease states. The GI360™ Microbiome Profile is a gut microbiota DNA analysis tool that identifies and characterizes more than 45 targeted analytes across six Phyla using PCR and compares the patient results to a characterized normobiotic reference population. The web chart illustrates the degree to which an individual's microbiome profile deviates from normobiosis.

LEGEND



The web image shows the relative diversity and balance among bacteria belonging to the six primary Phyla. The white shaded area represents the patient's results compared to a normobiotic reference population. The center of the web represents less abundance while the outer edges represent more than normobiotic.



Dysbiosis and Diversity Index

These indexes are calculated from the results of the Microbiome Profile, with scores ranging from 1 to 5, and do not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

The Dysbiosis Index the (DI) is calculated strictly from the results of the Microbiome Profile, with scores from 1 to 5. A DI score above 2 indicates dysbiosis; a microbiota profile that differs from the defined normobiotic reference population. The higher the DI above 2, the more the sample deviates from the normobiotic profile. The dysbiosis test and DI does not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

A diversity score of 3 indicates an expected amount of diversity, with 4 & 5 indicating an increased distribution of bacteria based on the number of different species and their abundance in the sample, calculated based on Shannon's diversity index. Scores of 1 or 2 indicate less diversity than the defined normobiotic reference population.

Dysbiosis Index

Diversity Score

GI 360

57 yo F; stress 9/10

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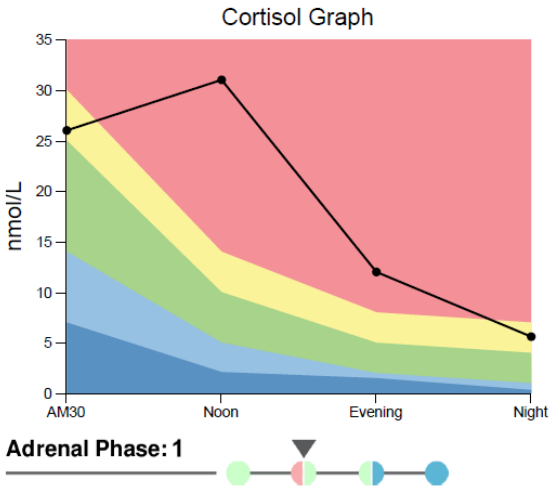
57yof, stress 9/10

Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol AM30	26	nmol/L		◆		14.0 – 25.0	7.0 – 30.0
Cortisol Noon	31	nmol/L			↑	5.0 – 10.0	2.1 – 14.0
Cortisol Evening	12	nmol/L			↑	2.0 – 5.0	1.5 – 8.0
Cortisol Night	5.6	nmol/L		◆		1.0 – 4.0	0.33 – 7.0
DHEA*	47	pg/mL	↓				106 – 300



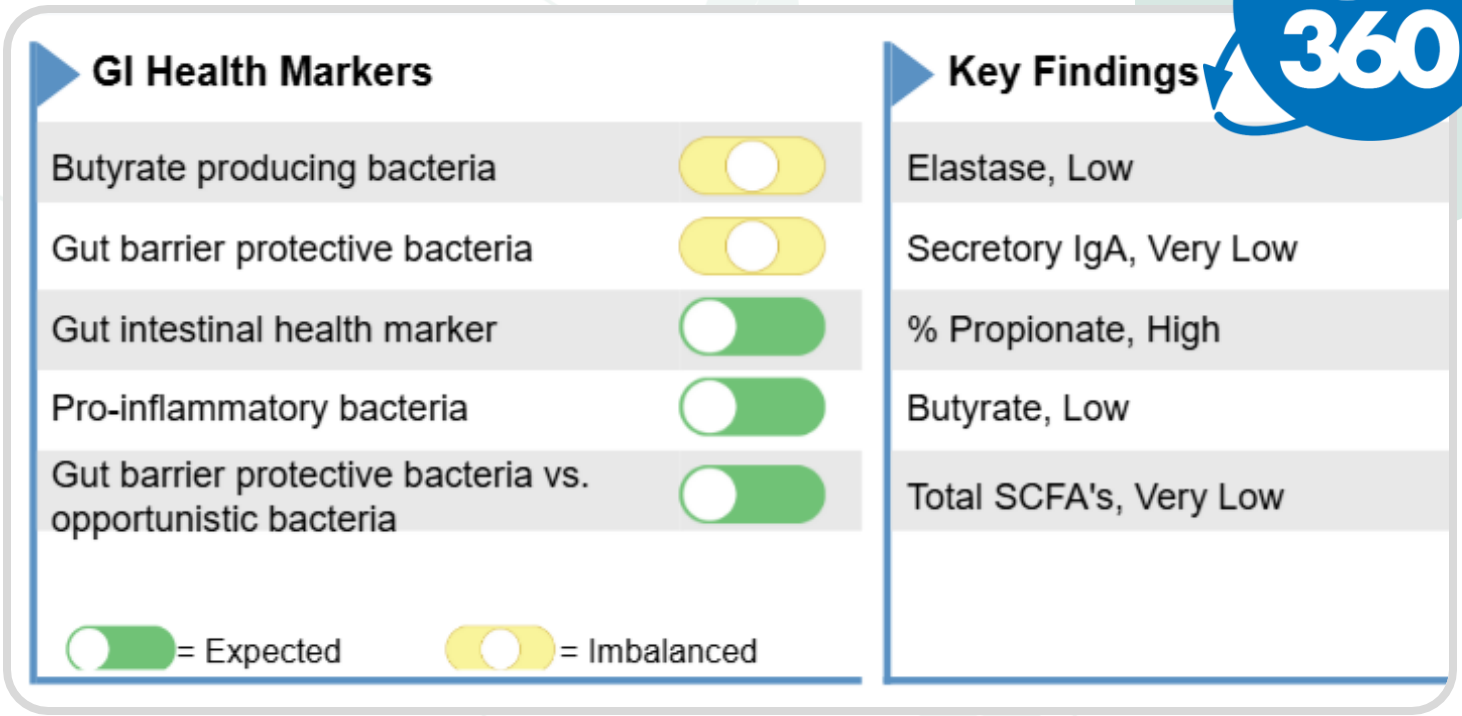
Hormone Comments

- The elevated cortisol level(s) and diurnal pattern are consistent with hypothalamic pituitary axis (HPA) dysregulation (Phase 1), although cortisol or glucocorticoid derivative supplementation cannot be excluded. Query use of steroidal inhalers or topical creams.
- DHEA levels typically decline with age and the level measured here is below the reference range. Note: Supplementation with DHEA may increase testosterone and/or estradiol levels.

Wellness Wednesday

DOCTOR'S DATA INC.

57yof, stress 9/10



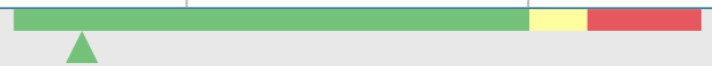
GI Health Markers	
Butyrate producing bacteria	
Gut barrier protective bacteria	
Gut intestinal health marker	
Pro-inflammatory bacteria	
Gut barrier protective bacteria vs. opportunistic bacteria	

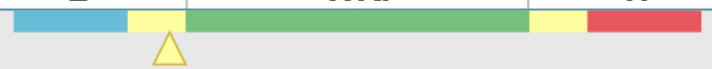
= Expected = Imbalanced

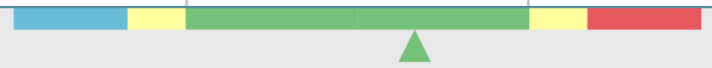
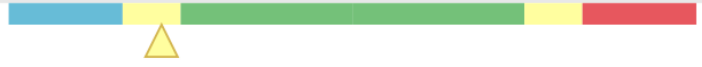
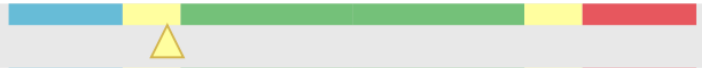
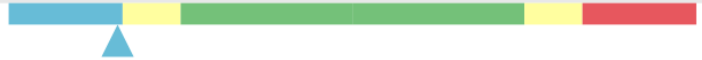
Key Findings
Elastase, Low
Secretory IgA, Very Low
% Propionate, High
Butyrate, Low
Total SCFA's, Very Low

Actinobacteria	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Actinobacteria	0		0
Actinomycetales	0		0
Bifidobacterium family	-1		0

Firmicutes	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Faecalibacterium prausnitzii	-2		0
Lachnospiraceae	0		0
Ligilactobacillus ruminis & *Pediococcus acidilactici*	-1		0
Lactobacillus family	-1		0
Phascolarctobacterium spp.	0		0
Ruminococcus albus & R. bromii	0		0
Mediterraneibacter gnavus	-1		0
Streptococcus agalactiae & *Agathobacter rectalis*	0		0

Inflammation	Result	Unit	L WRI H	Reference Interval
Lactoferrin	1.4	µg/mL		< 7.3
Lysozyme*	102	ng/mL		≤ 500
Calprotectin	<10	µg/g		≤ 50

Immunology	Result	Unit	L WRI H	Reference Interval
Secretory IgA*	27.2	mg/dL		30 – 275

Short Chain Fatty Acids	Result	Unit	L WRI H	Reference Interval
% Acetate‡	64			50 – 72
% Propionate‡	16			11 – 25
% Butyrate‡	19			11 – 32
% Valerate‡	0.7			0.8 – 5.0
Butyrate‡	0.73	mg/mL		0.8 – 4.0
Total SCFA's‡	3.8	mg/mL		5.0 – 16.0

57 yo F

Zonulin Family Protein; stool

	RESULT / UNIT	REFERENCE INTERVAL	LOW MOD HIGH
Zonulin Family Protein*	**90.5 ng/mL**	< 80.0	

Action Items

- Low *Lactobacillus:* Consider *Lactobacillus* probiotics and/or polyphenols
- Low Bifidobacterium: Consider Bifidobacterium probiotics and/or polyphenols and soluble fiber (8-15 grams QD)
- Low butyrate producing bacteria: soluble fiber
- Low IgA: Ω-3 fatty acids, olive oil, zinc, vitamins A and D_3
- Low elastase: Digestive enzymes and/or eating hygiene
- **Mucosal barrier support:** quercetin, vitamin D, retinol, curcumin, gamma-linolenic acid, omega-3 fatty acids (EPA, DHA), and aloe vera, glutamine
- **Stress management**
  - Exercise
  - Mindfulness & Meditation
  - Social Support

Key Takeaways:
The HPA Axis, Stress, and the Gut Microbiota

- The HPA axis regulates more than stress—it anticipates daily energy needs and modulates metabolism and immune readiness.
- It adjusts metabolic systems to align with diurnal rhythms and expected physical activity.
- During emergencies, it overrides normal functions to prioritize survival and immediate energy availability.
- The gut microbiota and the HPA axis are deeply interconnected via neurotransmitters and immune markers.

Key Takeaways:
The HPA Axis, Stress, and the Gut Microbiota

- Stress alters gut function through:
  - Increased intestinal permeability
  - Shifts in SCFAs and microbial composition
- Stress-related behaviors, like stress eating, can further disrupt the gut microbiota, creating a feedback loop.
- Dietary patterns significantly influence this gut-brain communication.
- Pairing cortisol testing with gut microbiome analysis can provide a fuller picture of physiology that may be acting as a chronic stressor.
- Addressing the gut microbiota may reduce stress-related adverse outcomes.
- True stress resilience comes from supporting both the brain *and* the gut.

QUESTIONS?