Casual Friday Series

Hormones and Mental Health

A Biogenetix Clinical Presentation

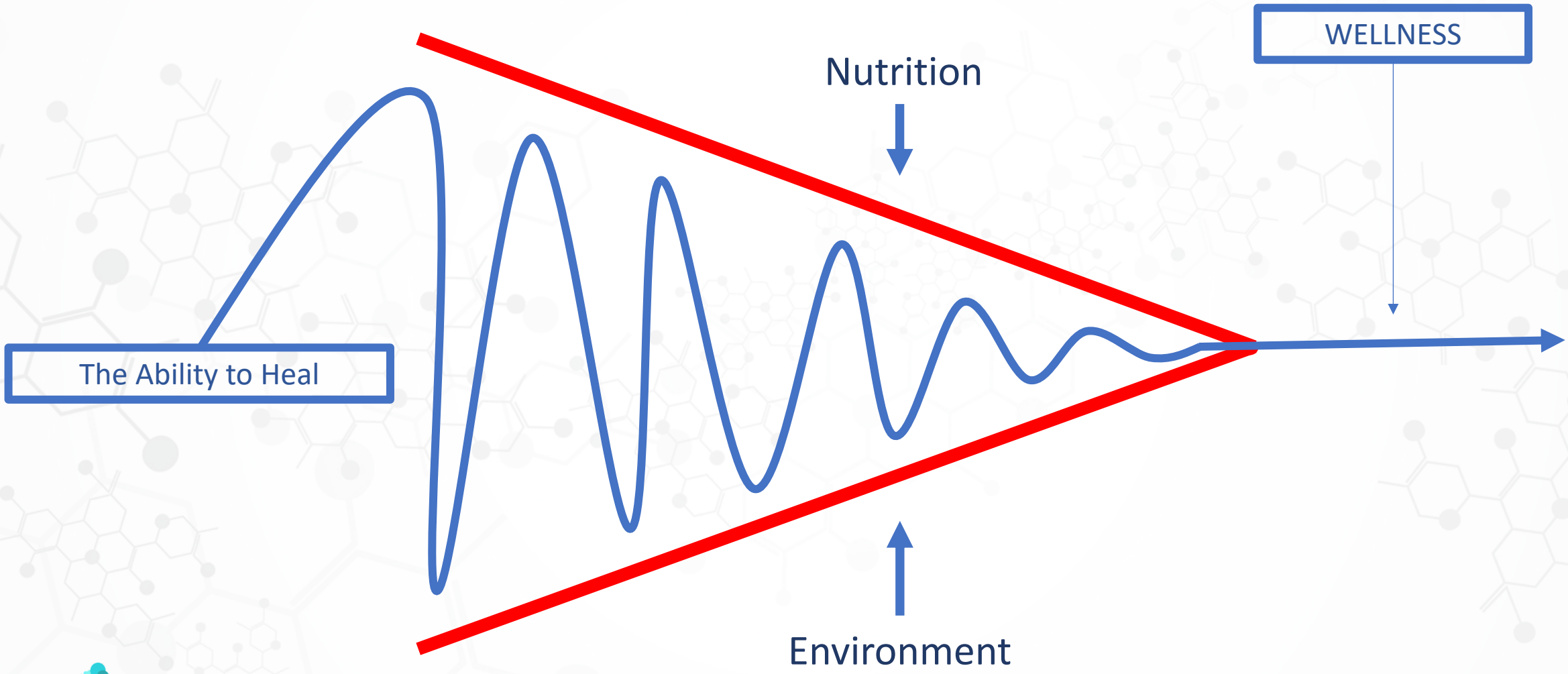
BIOGENETIX.COM

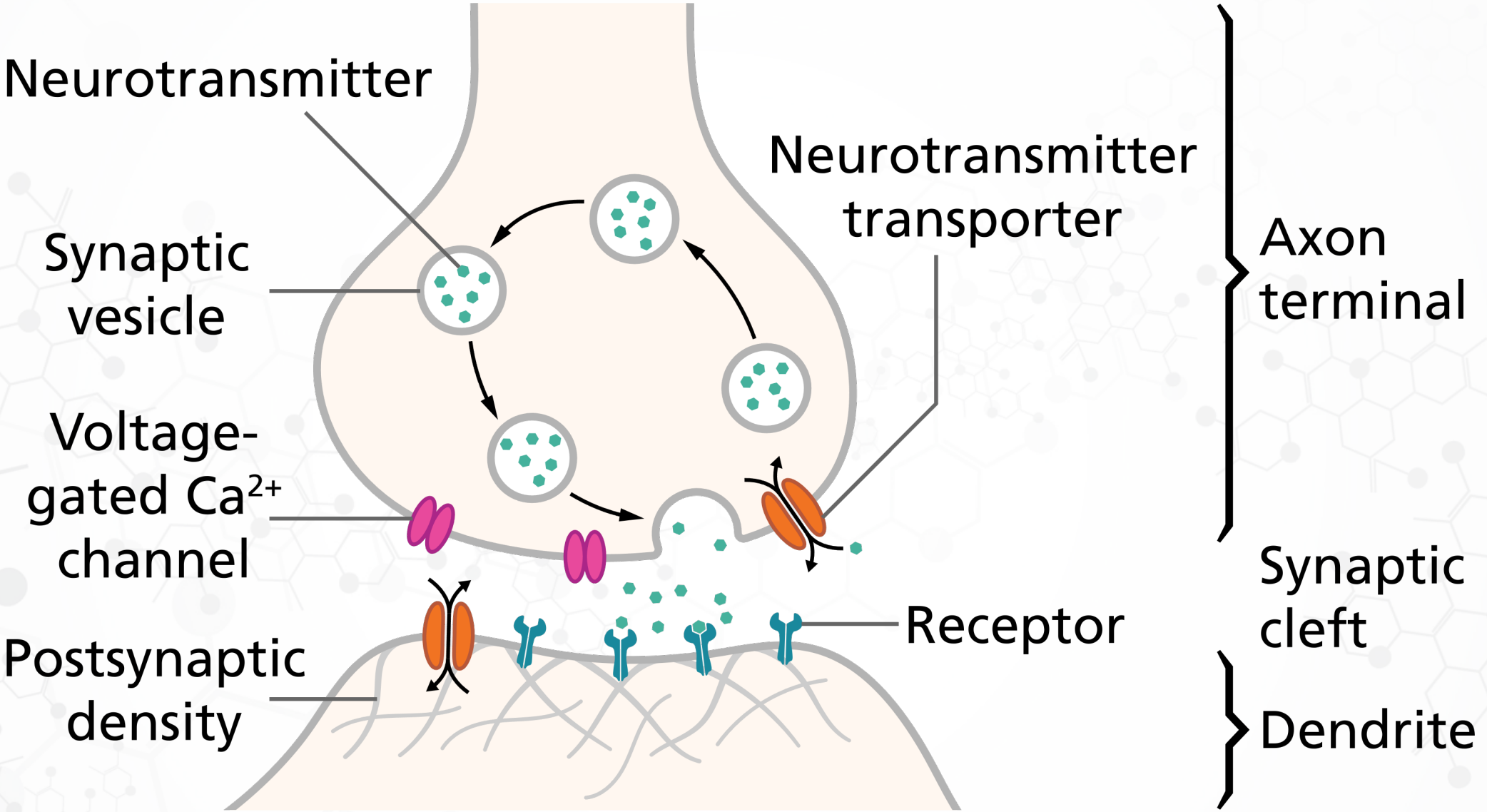
Disclaimer

- *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 IMPROVEMENT

Protocols





https://upload.wikimedia.org/wikipedia/commons/3/30/SynapseSchematic_en.svg

Table 9.1 Some Neurotransmitters and Their Functions

Neurotransmitter	Function	Examples of Malfunctions
Acetylcholine (ACh)	Enables muscle action, learning, and memory.	With Alzheimer's disease, ACh-producing neurons deteriorate.
Dopamine	Influences movement, learning, attention, and emotion.	Oversupply linked to schizophrenia. Undersupply linked to tremors and decreased mobility in Parkinson's disease.
Serotonin	Affects mood, hunger, sleep, and arousal.	Undersupply linked to depression. Some antidepressant drugs raise serotonin levels.
Norepinephrine	Helps control alertness and arousal.	Undersupply can depress mood.
GABA (gamma-aminobutyric acid)	A major inhibitory neurotransmitter.	Undersupply linked to seizures, tremors, and insomnia.
Glutamate	A major excitatory neurotransmitter; involved in memory.	Oversupply can overstimulate the brain, producing migraines or seizures (which is why some people avoid MSG, monosodium glutamate, in food).

https://sites.google.com/a/sduhsd.net/ap-psych-cdoerrer/home/neurotransmitter---functions?overridemobile=true

Review > Climacteric. 2019 Feb;22(1):55-59. doi: 10.1080/13697137.2018.1543265.
Epub 2018 Dec 20.

Optimizing quality of life through sex steroids by their effects on neurotransmitters

Menopause-related symptoms such as hot flushes, night sweats, weight gain, and decreased sexual functioning all have negative impacts on quality of life and affect daily activities such as sleep, work, and leisure activities. During the menopause transition, neurotransmitters, neuropeptides, and neurosteroids undergo important changes as a consequence of the failure of gonadal hormone production, at a time when many central nervous system activities deteriorate. Sex hormones have been implicated in neurite outgrowth, synaptogenesis, dendritic branching, myelination, and other important mechanisms of neural plasticity. Knowledge of interactions

Brain Res. Author manuscript; available in PMC 2019 Aug 15.

Published in final edited form as:

Brain Res. 2018 Aug 15; 1693(Pt B): 128–133.

doi: 10.1016/j.brainres.2018.03.015

PMCID: PMC6005194

NIHMSID: NIHMS953233

PMID: 29903615

Neurotransmitter modulation by the gut microbiota

The gut microbiota – the trillions of bacteria that reside within the gastrointestinal tract – has been found to not only be an essential component immune and metabolic health, but also seems to influence development and diseases of the enteric and central nervous system, including motility disorders, behavioral disorders, neurodegenerative disease, cerebrovascular accidents, and neuroimmune-mediated disorders. By leveraging animal models, several different pathways of communication have been identified along the "gut-brain-axis" including those driven by the immune system, the vagus nerve, or by modulation of neuroactive compounds by the microbiota. Of the latter, bacteria have been shown to produce and/or consume a wide range of mammalian neurotransmitters, including dopamine, norepinephrine, serotonin, or gamma-aminobutyric acid (GABA). Accumulating evidence in animals suggests that manipulation of these neurotransmitters by bacteria may have an impact in host physiology, and preliminary human studies are showing that microbiota-based interventions can also alter neurotransmitter levels. Nonetheless, substantially more work is required to determine whether microbiota-mediated manipulation of human neurotransmission has any physiological implications, and if so, how it may be leveraged therapeutically.

Brain Res. Author manuscript; available in PMC 2019 Aug 15.

Published in final edited form as:

Brain Res. 2018 Aug 15; 1693(Pt B): 128–133.

doi: 10.1016/j.brainres.2018.03.015

PMCID: PMC6005194

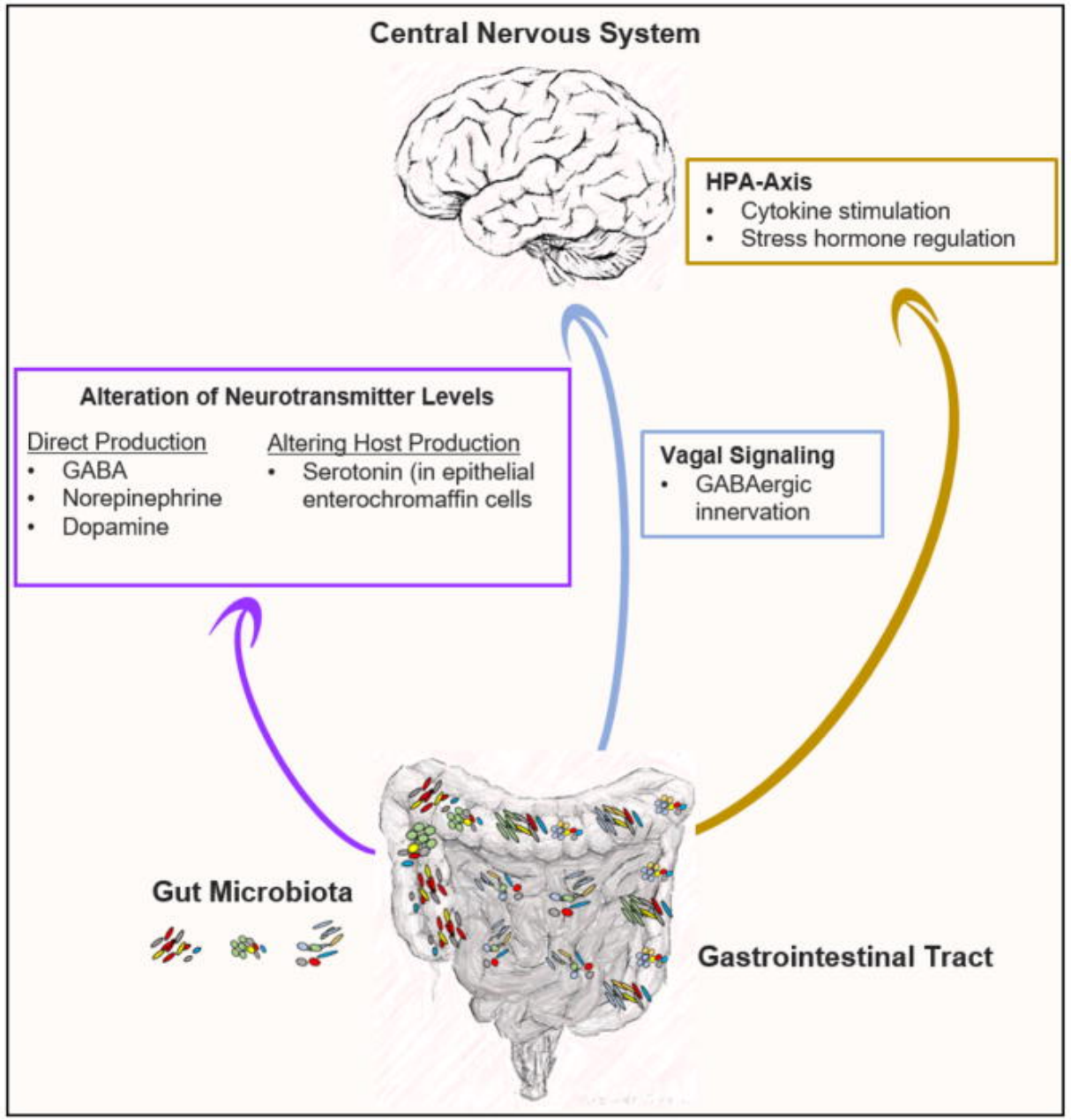
NIHMSID: NIHMS953233

PMID: 29903615

Neurotransmitter modulation by the gut microbiota

Philip Strandwitz[a]

An attractive and simple exploratory technique to determine whether the microbiota may be involved in a disease is to eliminate bacteria from an animal (either through treatment with a combination of broad-spectrum antibiotics, or use of germ free lines/facilities), and determine if end points in a model of interest change. Using this approach, a seminal 2004 study found that germ free mice exhibited an increased response to induced stress via the restraint model, and that this behavioral alteration could be restored by recolonizing these animals with a complete microbiota (via stool transplant) or by monocolonization with *Bifidobacterium infantis* (but not *Escherichia coli*) (Sudo et al., 2004). Since then, bacteria-depleted



https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6005194/table/T1/?report=objectonly

Brain Res. Author manuscript; available in PMC 2019 Aug 15.

Published in final edited form as:

Brain Res. 2018 Aug 15; 1693(Pt B): 128–133.

doi: 10.1016/j.brainres.2018.03.015

PMCID: PMC6005194

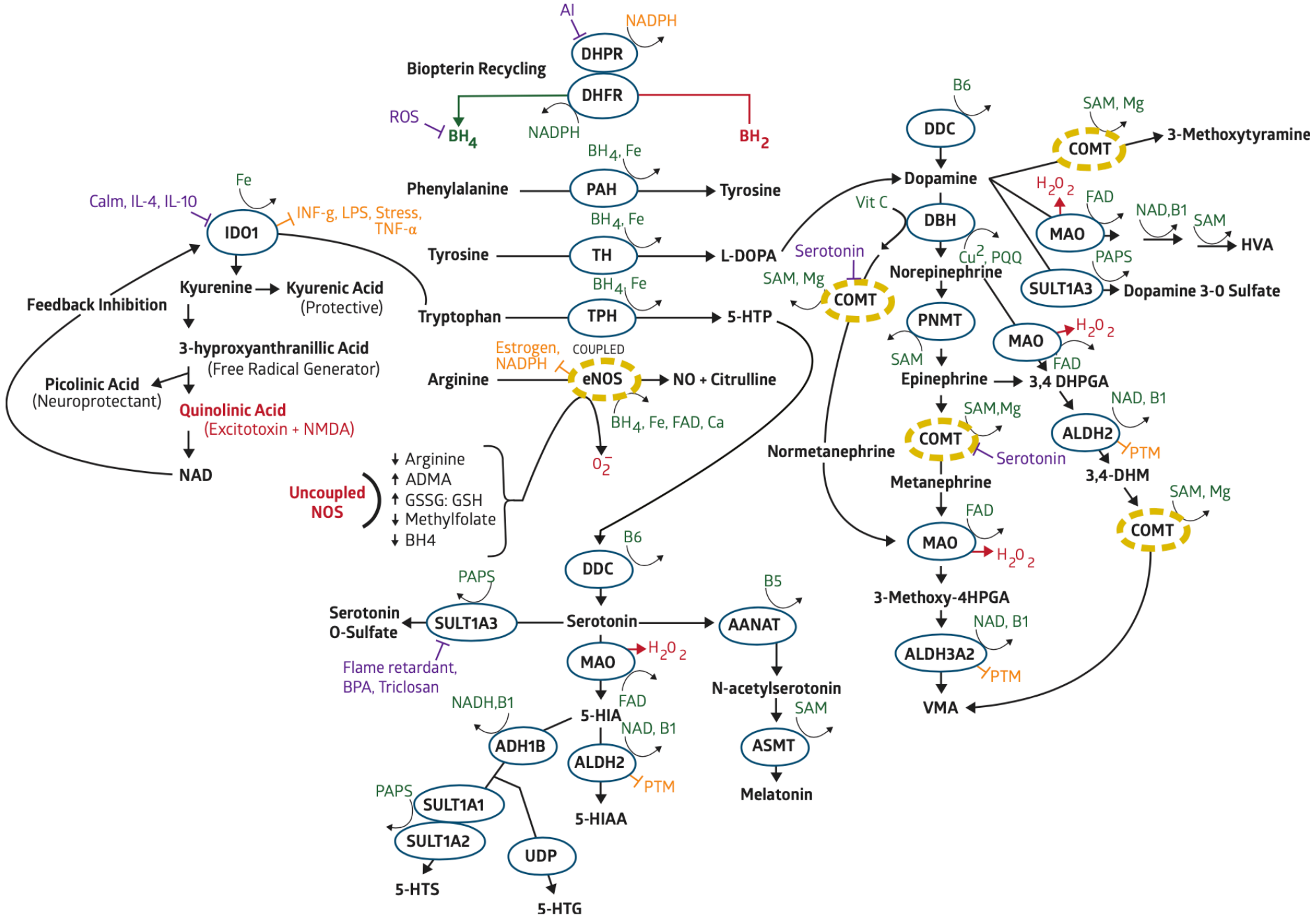
NIHMSID: NIHMS953233

PMID: 29903615

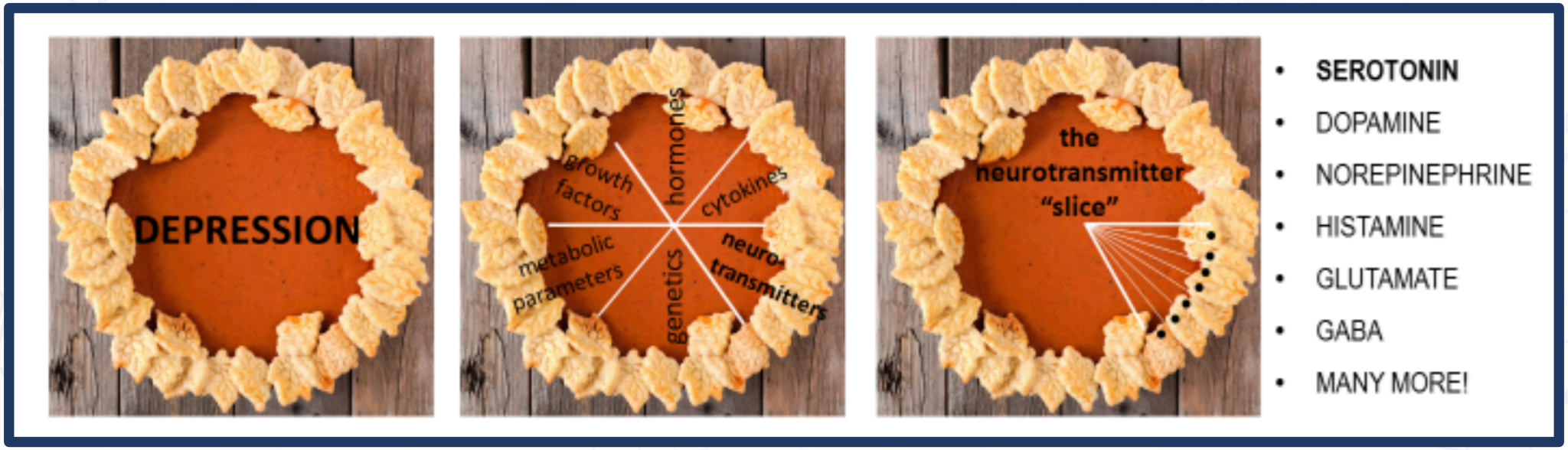
Neurotransmitter modulation by the gut microbiota

Philip Strandwitz[a]

Given the abundance of serotonin in the GI tract, it is perhaps not surprising that an expanding list of literature is linking the microbiota to host levels of serotonin. In germ free animals, there is a significant reduction of serotonin in the blood and colon of mice compared to controls (Wikoff et al., 2009), a feature which can be restored via recolonization with a microbiota or with a consortium of spore-forming species. Notably, while several strains of bacteria are reported to produce serotonin (Table 1), such capabilities have not been identified in the gut microbiota. Instead the alteration of host serotonin levels appears to mediated via secretion of small molecules (like short chain fatty acids or secondary bile acids) that signal ECs to produce serotonin via expression of tryptophan hydroxylase (Yano et al., 2015). There is also evidence that

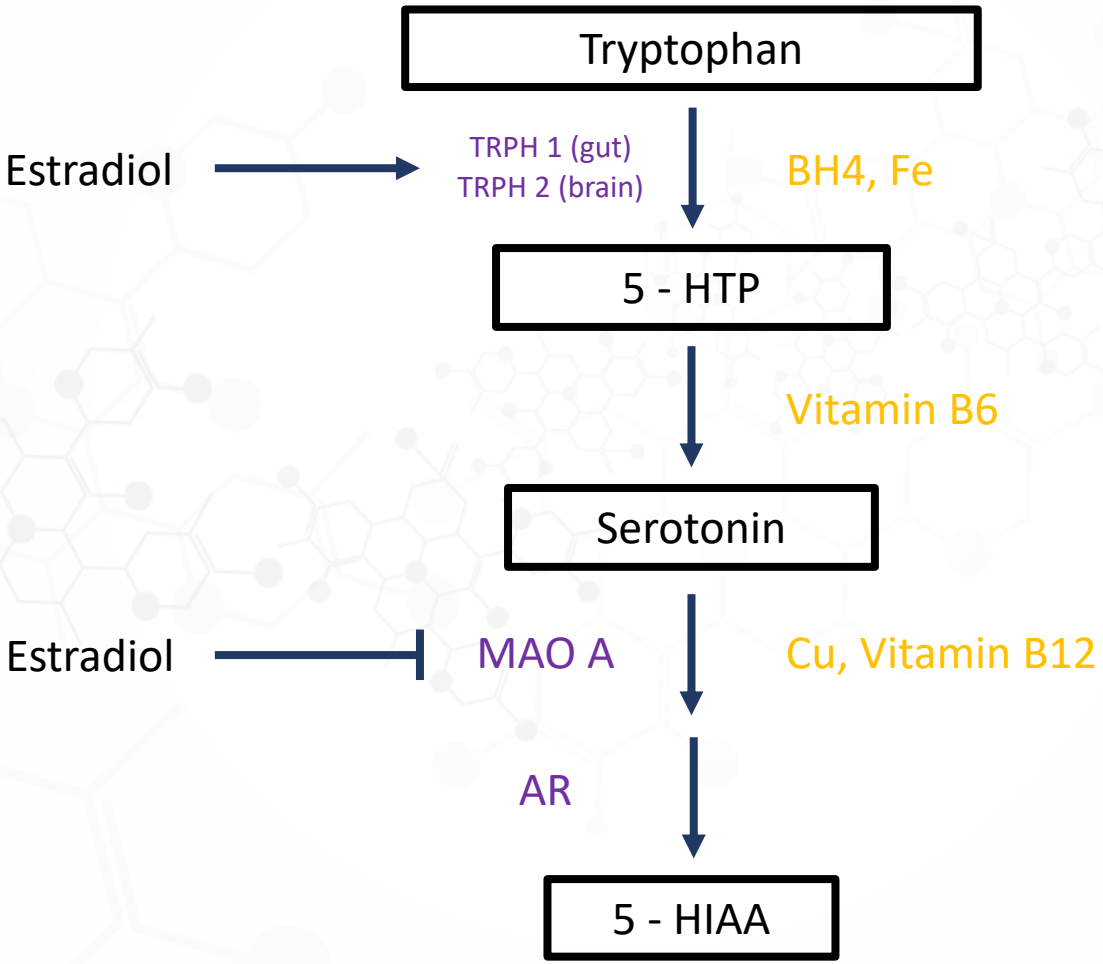

Biopterin Recycling
AI
NADPH
DHPR
DHFR
ROS
BH4
NADPH
BH2
Phenylalanine
BH4, Fe
PAH
Tyrosine
Tyrosine
BH4, Fe
TH
L-DOPA
Tryptophan
BH4, Fe
TPH
5-HTP
Arginine
Estrogen, NADPH
COUPLED
eNOS
NO + Citrulline
BH4, Fe, FAD, Ca
O2-
Uncoupled NOS
↓ Arginine
↑ ADMA
↑ GSSG: GSH
↓ Methylfolate
↓ BH4
Calm, IL-4, IL-10
Fe
IDO1
INF-g, LPS, Stress, TNF-α
Feedback Inhibition
Kyurenine
Kyurenic Acid
(Protective)
3-hyproxyanthranillic Acid
(Free Radical Generator)
Picolinic Acid
(Neuroprotectant)
Quinolinic Acid
(Excitotoxin + NMDA)
NAD
B6
DDC
Dopamine
Vit C
DBH
Cu2, PQQ
Norepinephrine
Serotonin
SAM, Mg
COMT
PNMT
SAM
Epinephrine
Normetanephrine
SAM,Mg
COMT
Serotonin
Metanephrine
FAD
MAO
H2O2
3-Methoxy-4HPGA
NAD, B1
ALDH3A2
PTM
VMA
SAM, Mg
COMT
3-Methoxytyramine
H2O2
FAD
MAO
NAD,B1
SAM
HVA
PAPS
SULT1A3
Dopamine 3-0 Sulfate
MAO
H2O2
FAD
3,4 DHPGA
NAD, B1
ALDH2
PTM
3,4-DHM
SAM, Mg
COMT
B6
DDC
PAPS
SULT1A3
Serotonin O-Sulfate
Flame retardant, BPA, Triclosan
Serotonin
B5
AANAT
N-acetylserotonin
SAM
ASMT
Melatonin
MAO
H2O2
FAD
5-HIA
NADH,B1
ADH1B
NAD, B1
ALDH2
PTM
5-HIAA
PAPS
SULT1A1
SULT1A2
5-HTS
UDP
5-HTG

Depression Pie

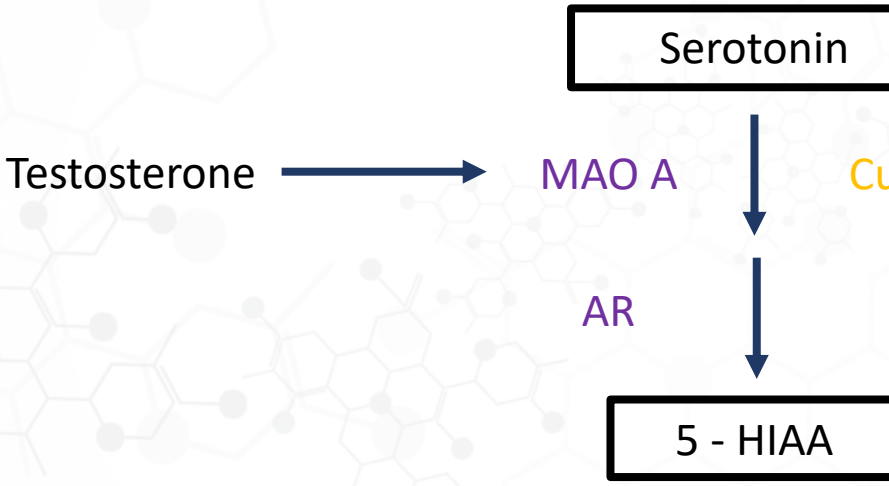


https://www.zrtlab.com/blog/archive/impact-hormones-serotonin-depression/

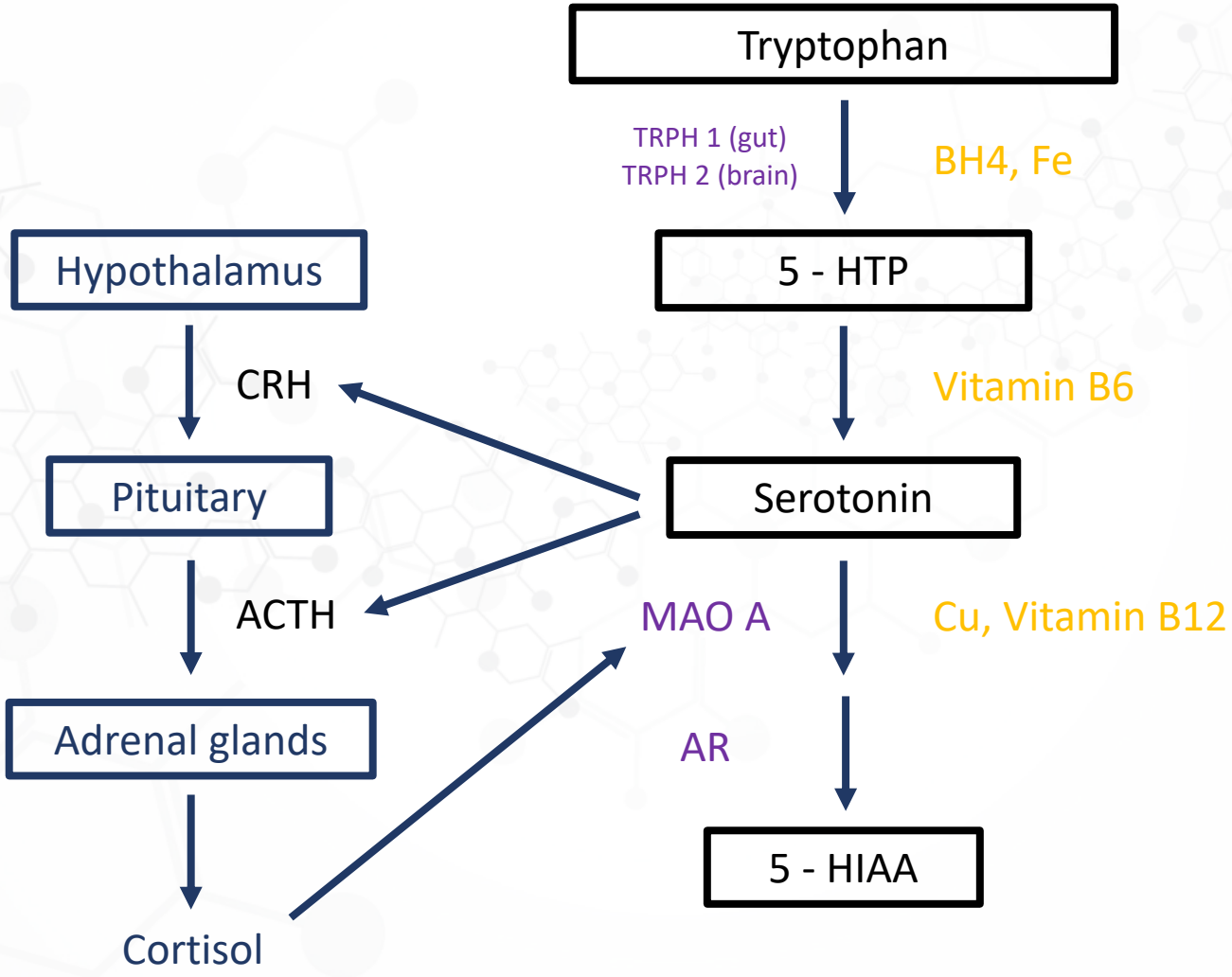
E2 – Serotonin Connection



Testosterone – Serotonin Connection



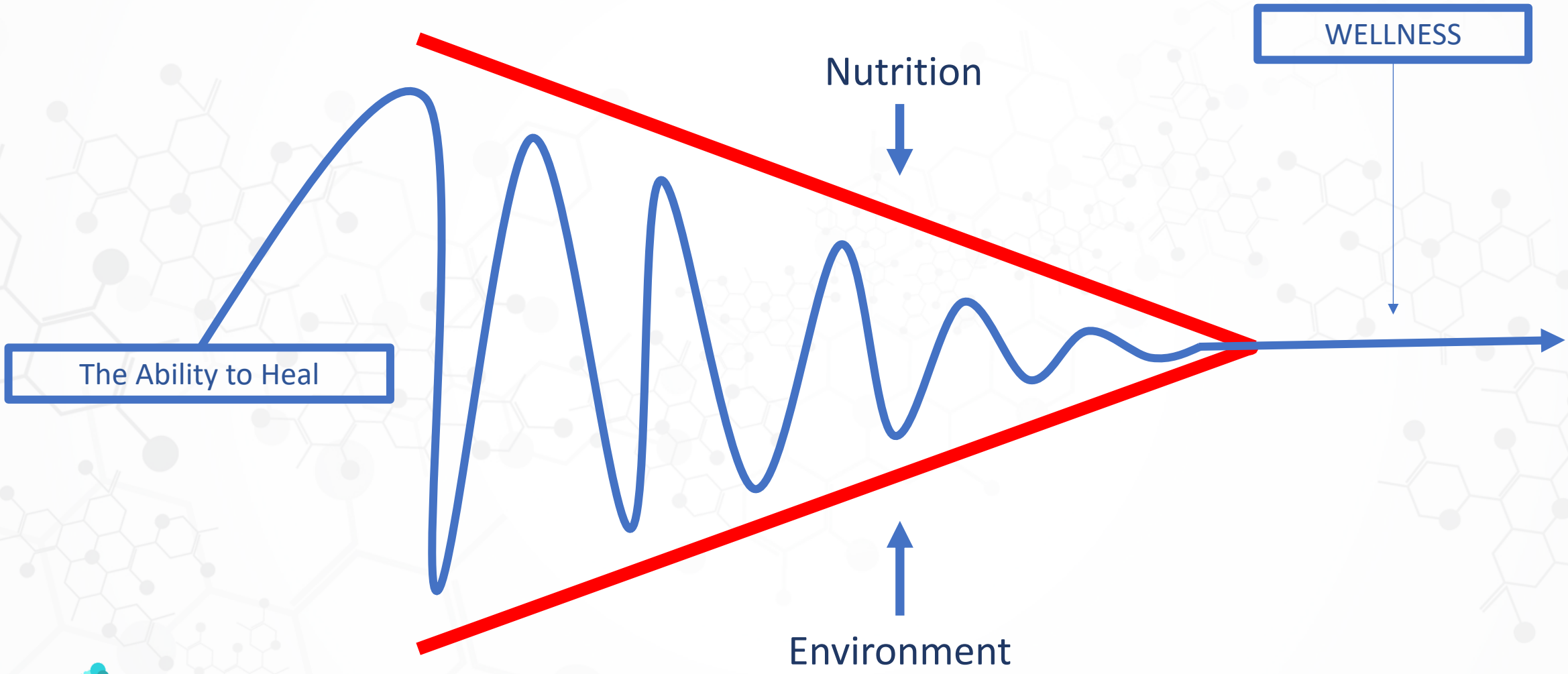
HPA axis – Serotonin Connection



Summary

- Gut Flora Health (neurotransmitter creation and modulation)
- Metabolism (building blocks and cofactors for ATP)
- Antioxidant Maintenance (cleaning up ROS)
- Inflammation Management (cytokines)
- HPA Axis adaptation to perceived stressors
- Genetic variants
- Give me a pill?

Protocols



How to Assess

- Organic acid testing
- Blood chemistry
- Dutch testing
- Stool testing
- Strategene

Or just take a pill? 

Biogenetix: 833-525-0001

zeb@biogenetix.com

kim@biogenetix.com