

SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO) AND ANXIETY

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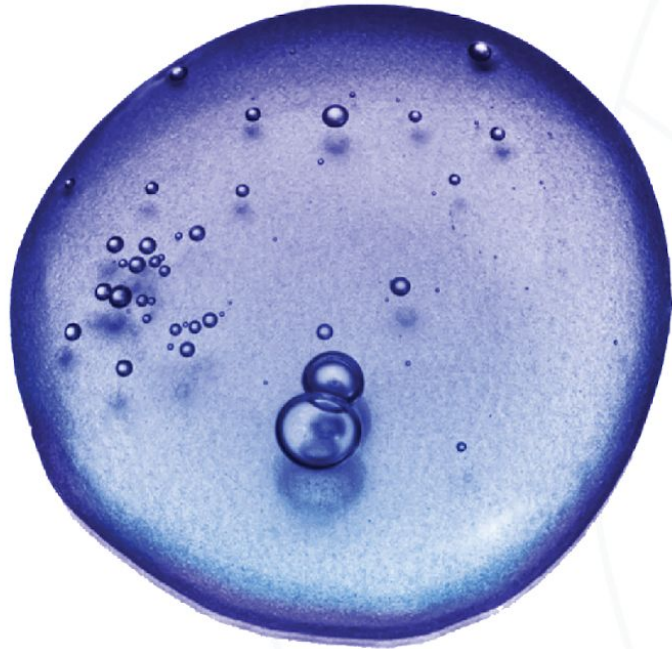


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SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO) PREVALENCE

CAUSATION, SYMPTOMS, SIGNS



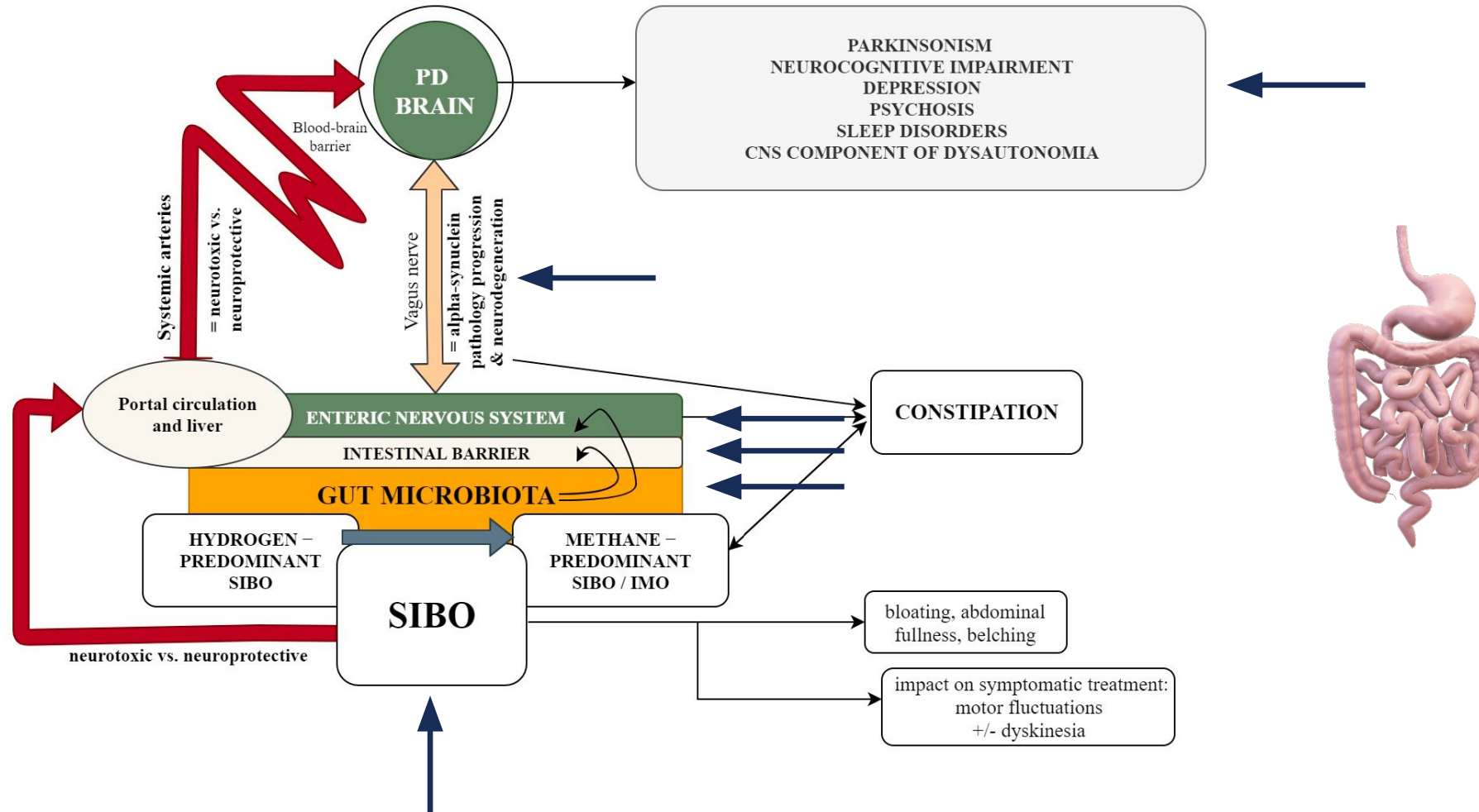
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ORDER OF PATHOLOGY AND PRIORITIES

- Complexity and causation
- Symptomatology
- Breaching the gut barrier
- Breaching multiple barriers
- Motility changes
- Pathway to the brain
- Pathway to brain destruction
- Brain: healthy vs. unhealthy
- Gut – inflammation – stress hormones – brain
- Prolonged neurological inflammation and HPA axis
- Eventual transmitter changes and altered psychological function
- Cortical failure and limbic preservation
- Treatment ideas

SIBO TO THE BRAIN

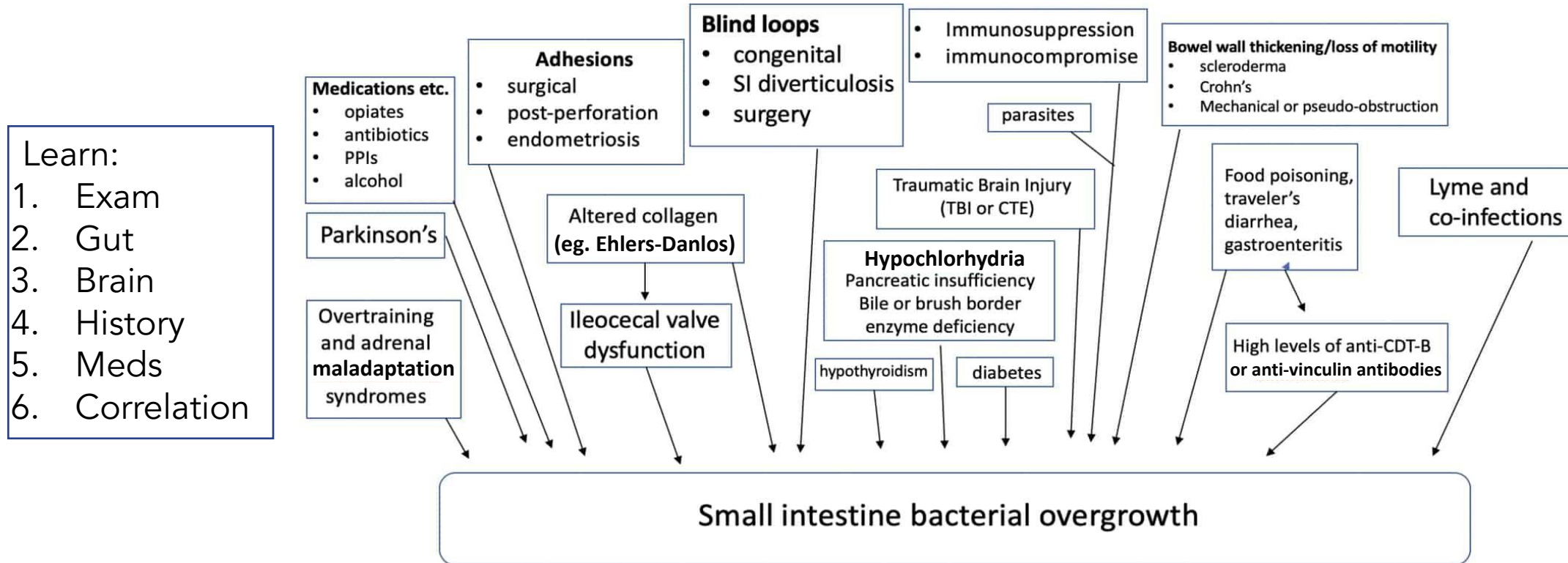
THE BIGGER PICTURE



SIBO TO THE BRAIN

COMPLEXITY OF CAUSATION

Common Causes of SIBO Checklist



Steven Sandberg-Lewis 2014, 2016, 2017

1

SIBO AND SYMPTOMATOLOGY

Hydrogen (H₂) and methane (CH₄) are exclusively produced by microbial metabolism and are exhaled on the breath.

The North American consensus defines a rise in H₂ ≥ 20 parts per million (ppm) from baseline within 90 min of substrate ingestion as positive for the H₂ breath test.

A CH₄ level ≥ ten ppm at any time is defined as positive for the CH₄ breath test.

SIBO is associated with a myriad of causation and symptoms:

Hydrogen

- Mainly diarrhea

Methane

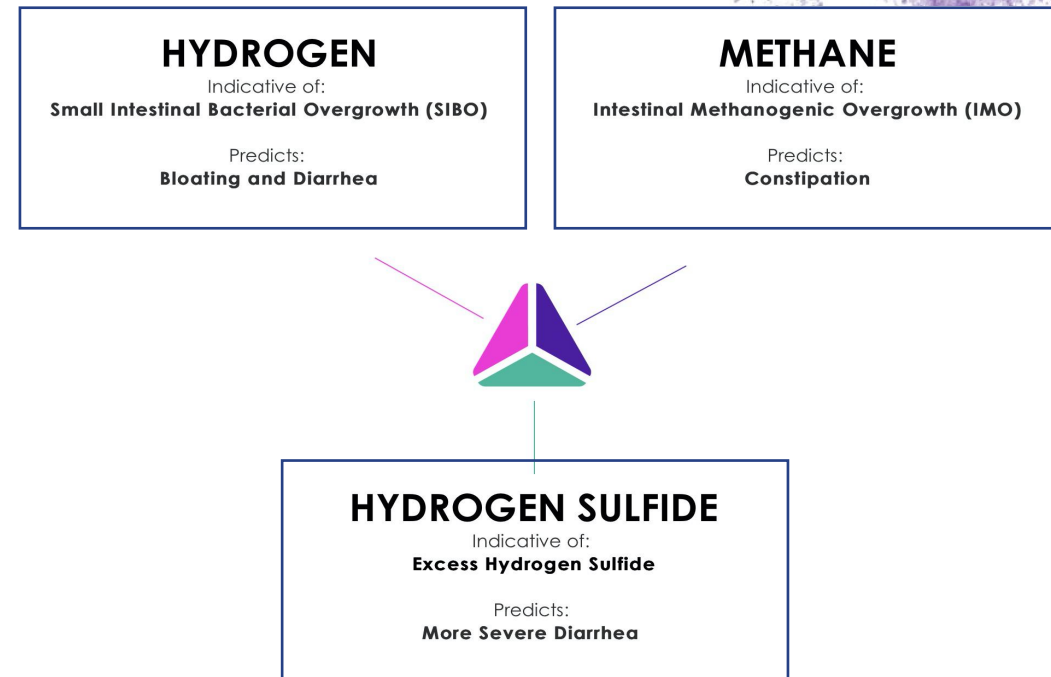
- Mainly constipation

Hydrogen Sulfide

- Diarrhea, body pains, food sensitivities

Common symptoms

- Bloating
- Abdominal pain
- Nausea
- Constipation
- Diarrhea





SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO) PREVALENCE

GOING FROM INTESTINES TO A SYSTEMIC ISSUE

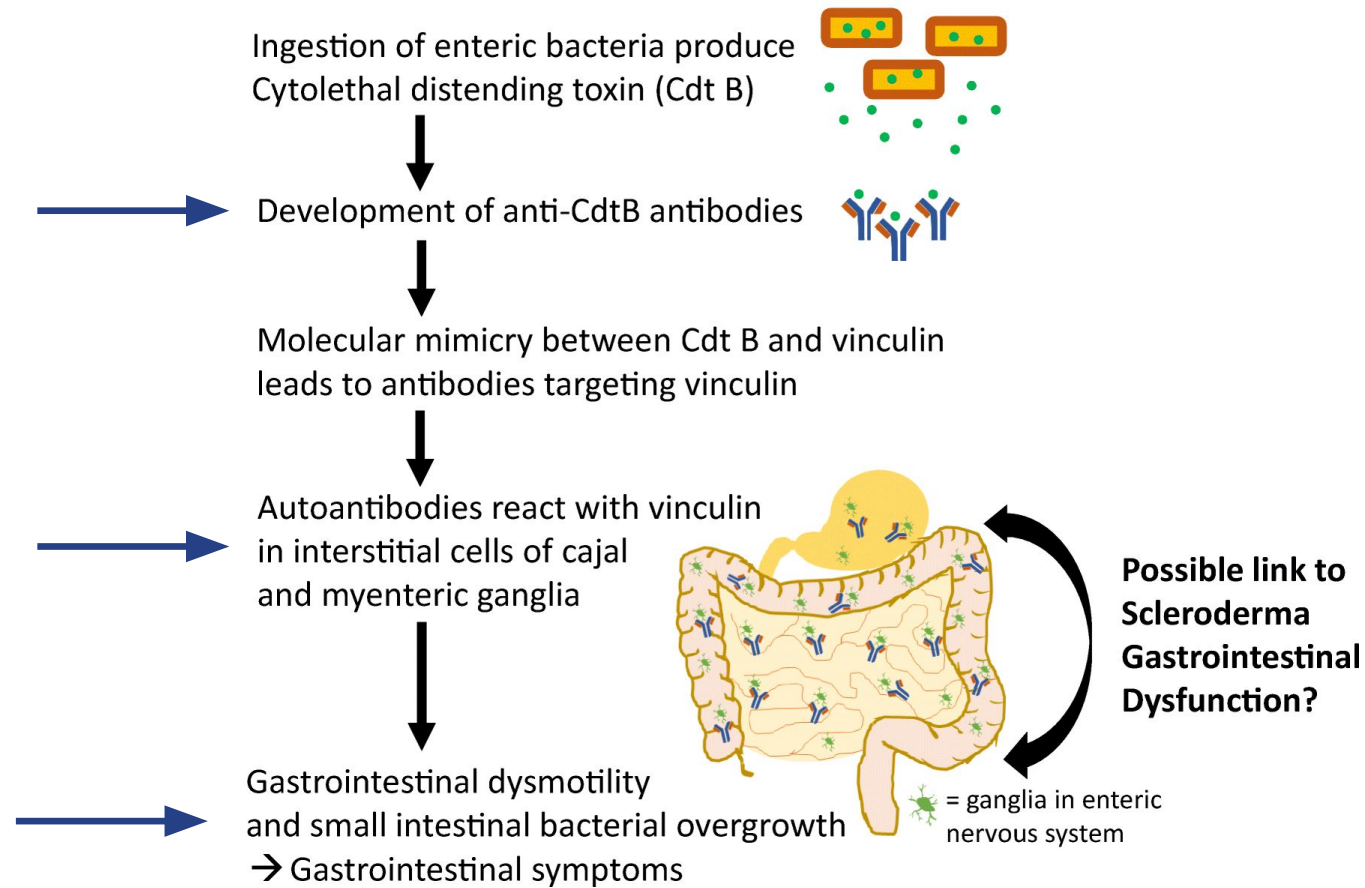


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SIBO TO THE BRAIN

SLOWING MOTILITY

Proposed Model for Pathogenesis of Irritable Bowel Syndrome



SIBO TO THE BRAIN

BREACHING THE BARRIERS



SIBO TO THE BRAIN

BREACHING THE WALLS

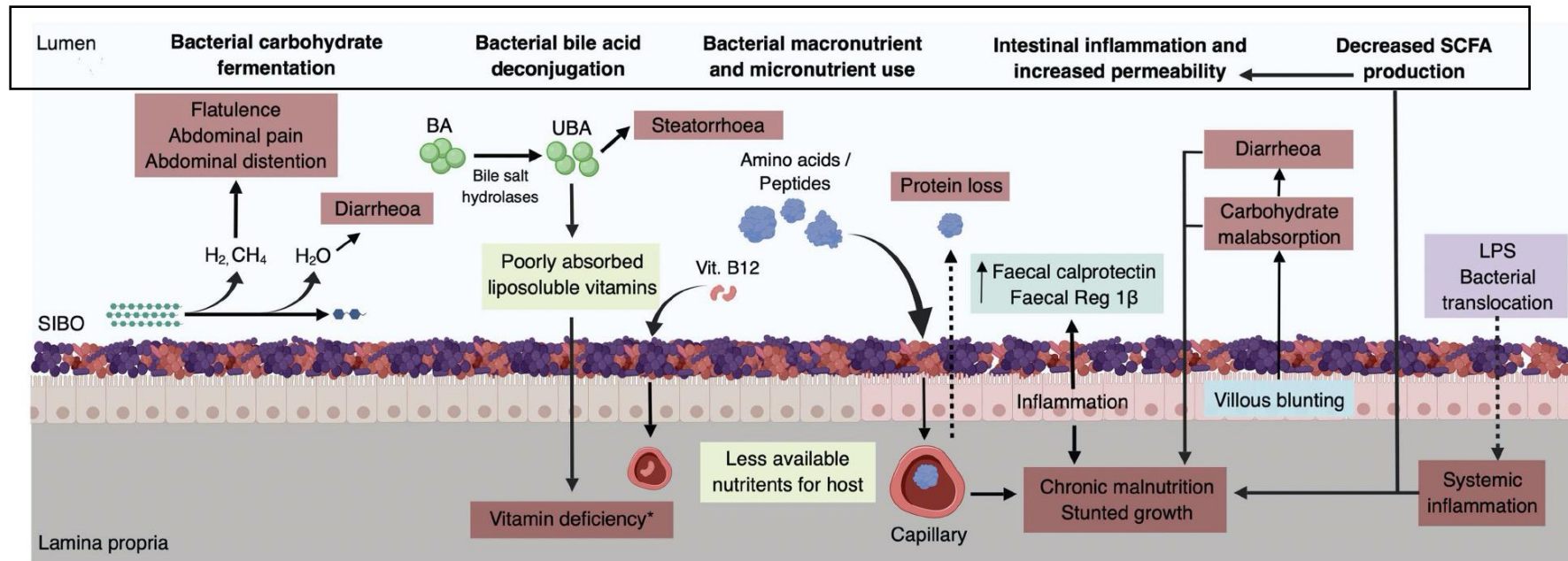
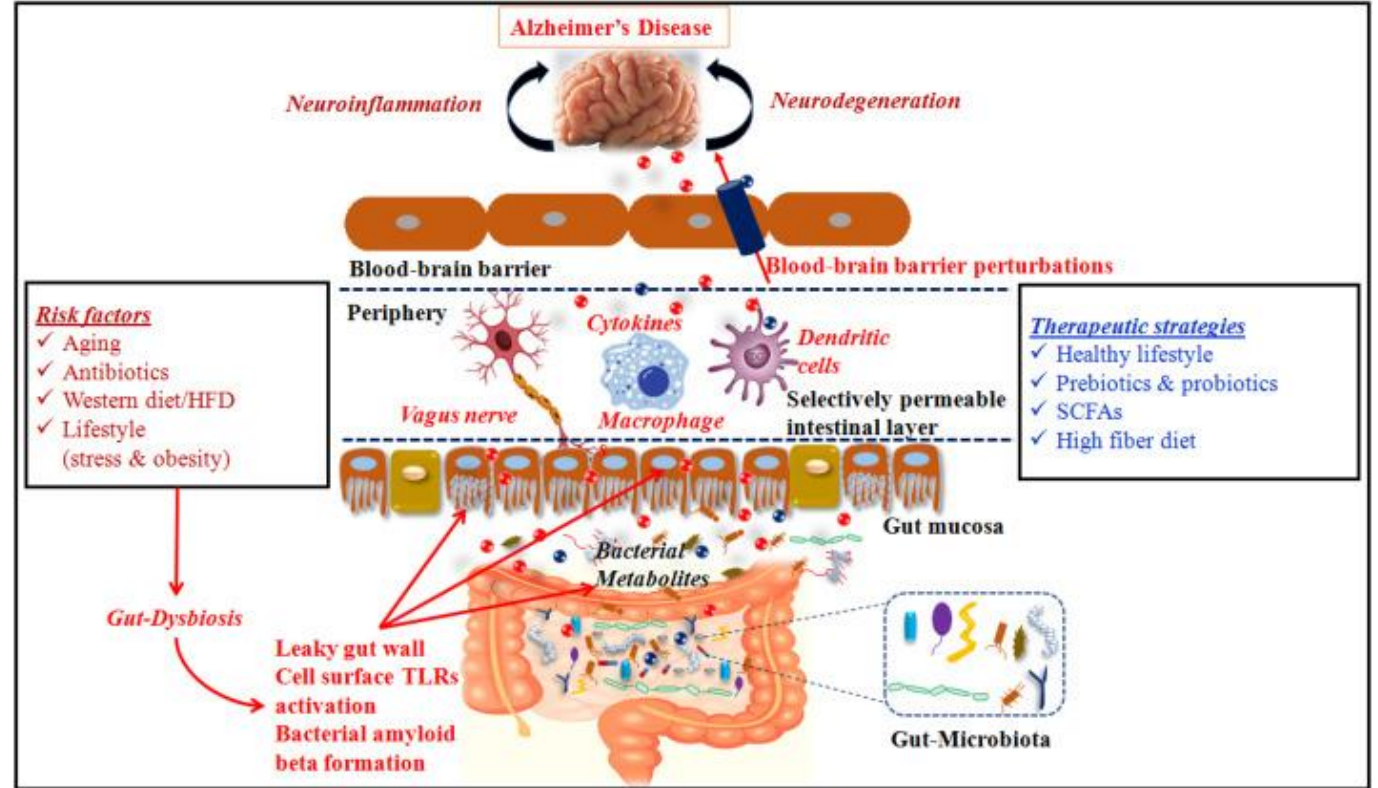
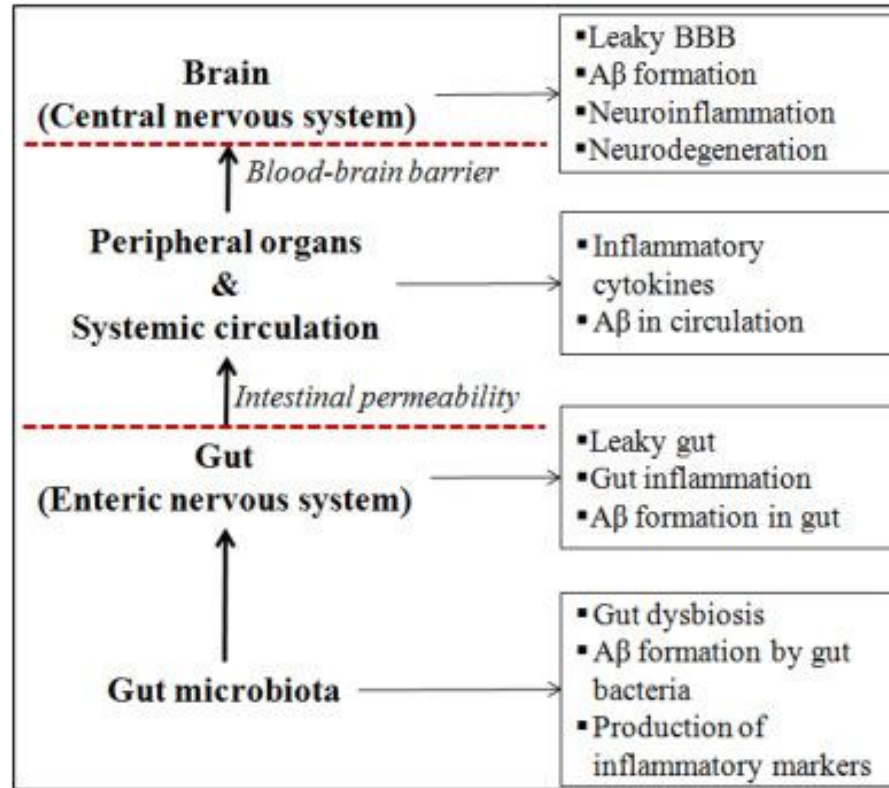


FIGURE 3 | Mechanisms through which SIBO affects the host. The dotted arrows indicate increased intestinal permeability. * Includes vitamin A, D, E, and vitamin B 12. Vitamin K is synthesised by the gut microbiota, and thus its deficiency in this context is very unlikely. BA, bile acids; UBA, unconjugated bile acids; LPS, liposaccharides. Created with BioRender.com.

SIBO TO THE BRAIN

BREACHING MULTIPLE WALLS





SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO) PREVALENCE

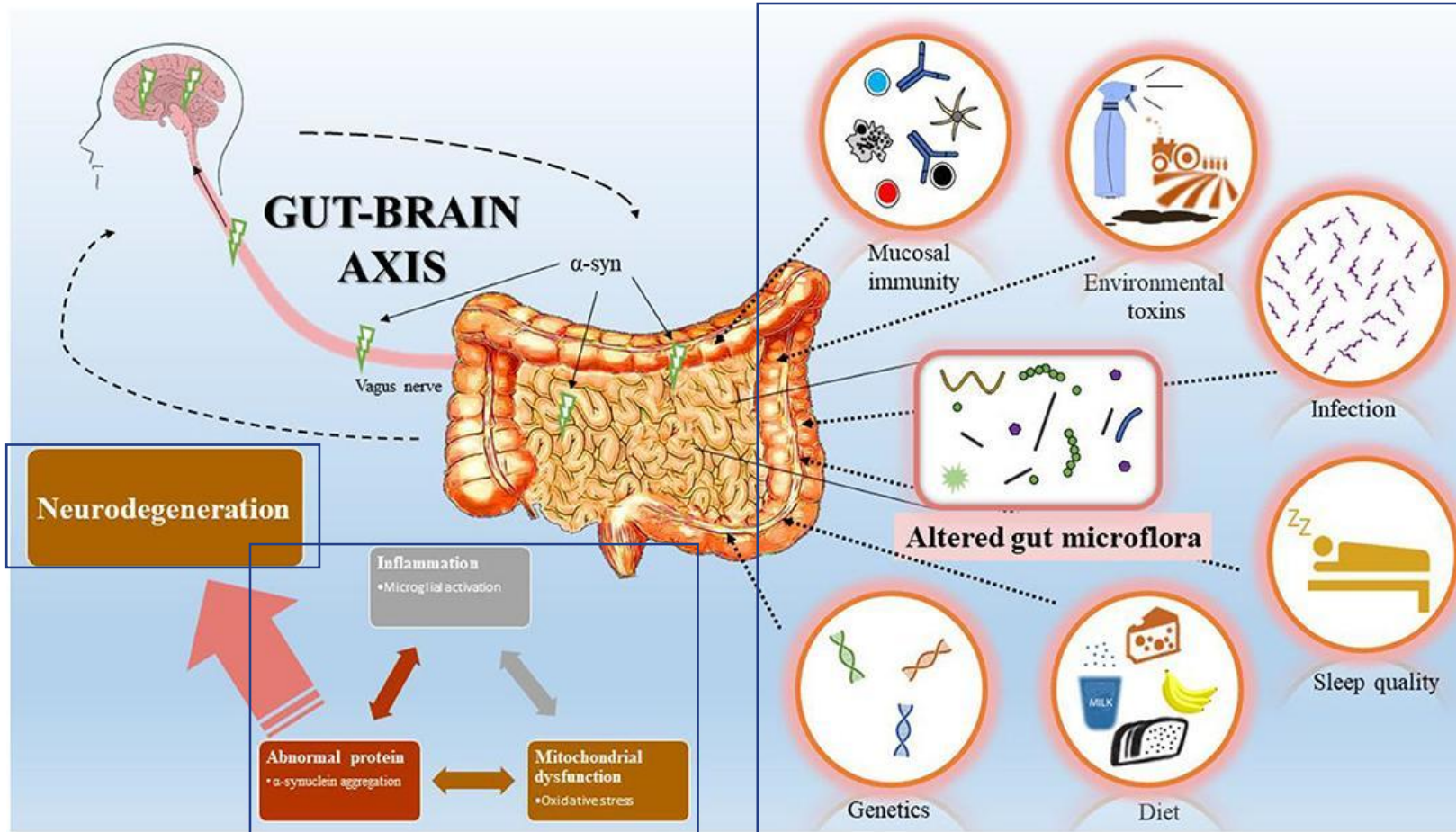
GETTING TO THE BRAIN



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SIBO TO THE BRAIN

IT WILL EVENTUALLY GET THERE



SIBO TO THE BRAIN

THE PATH OF BRAIN DESTRUCTION

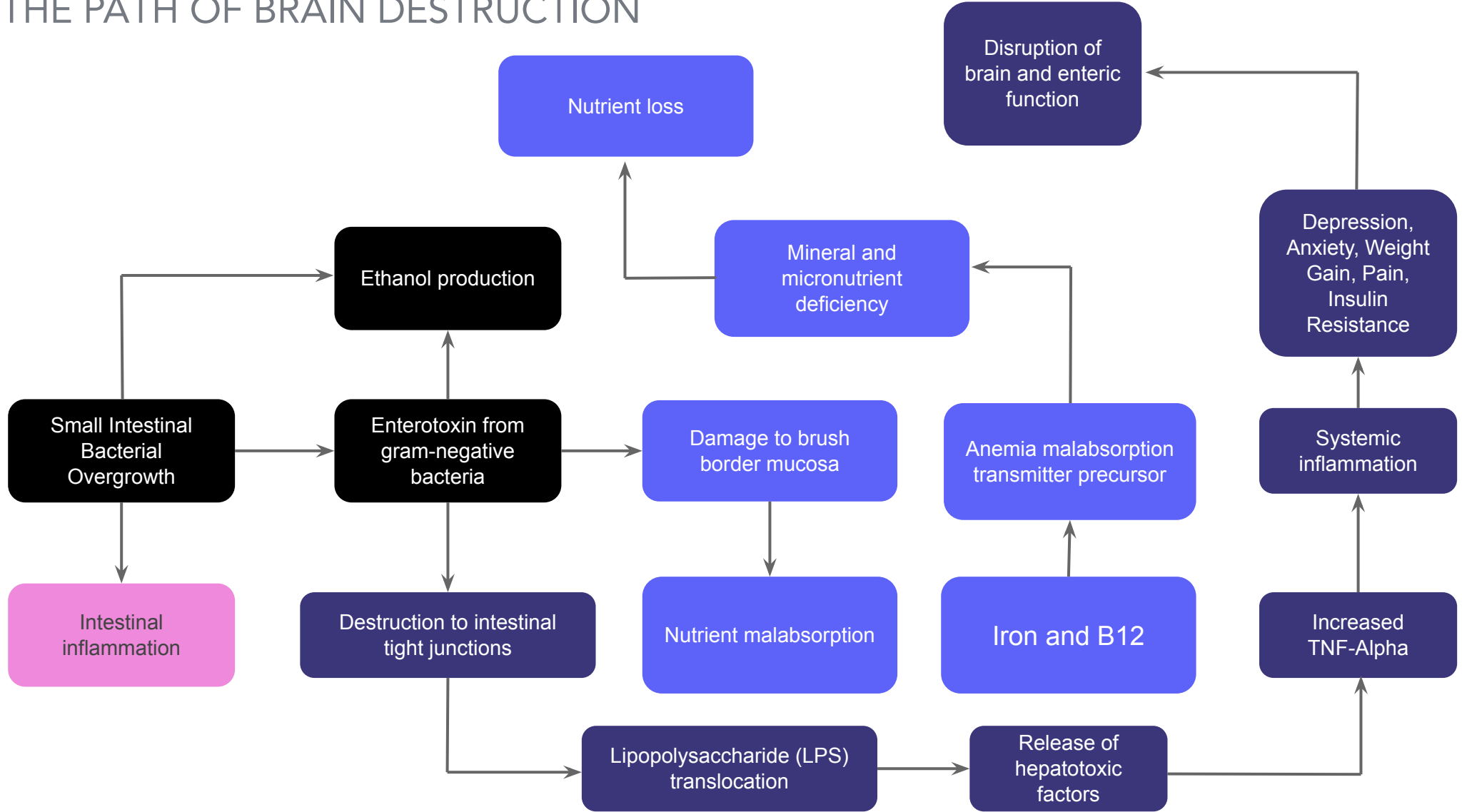
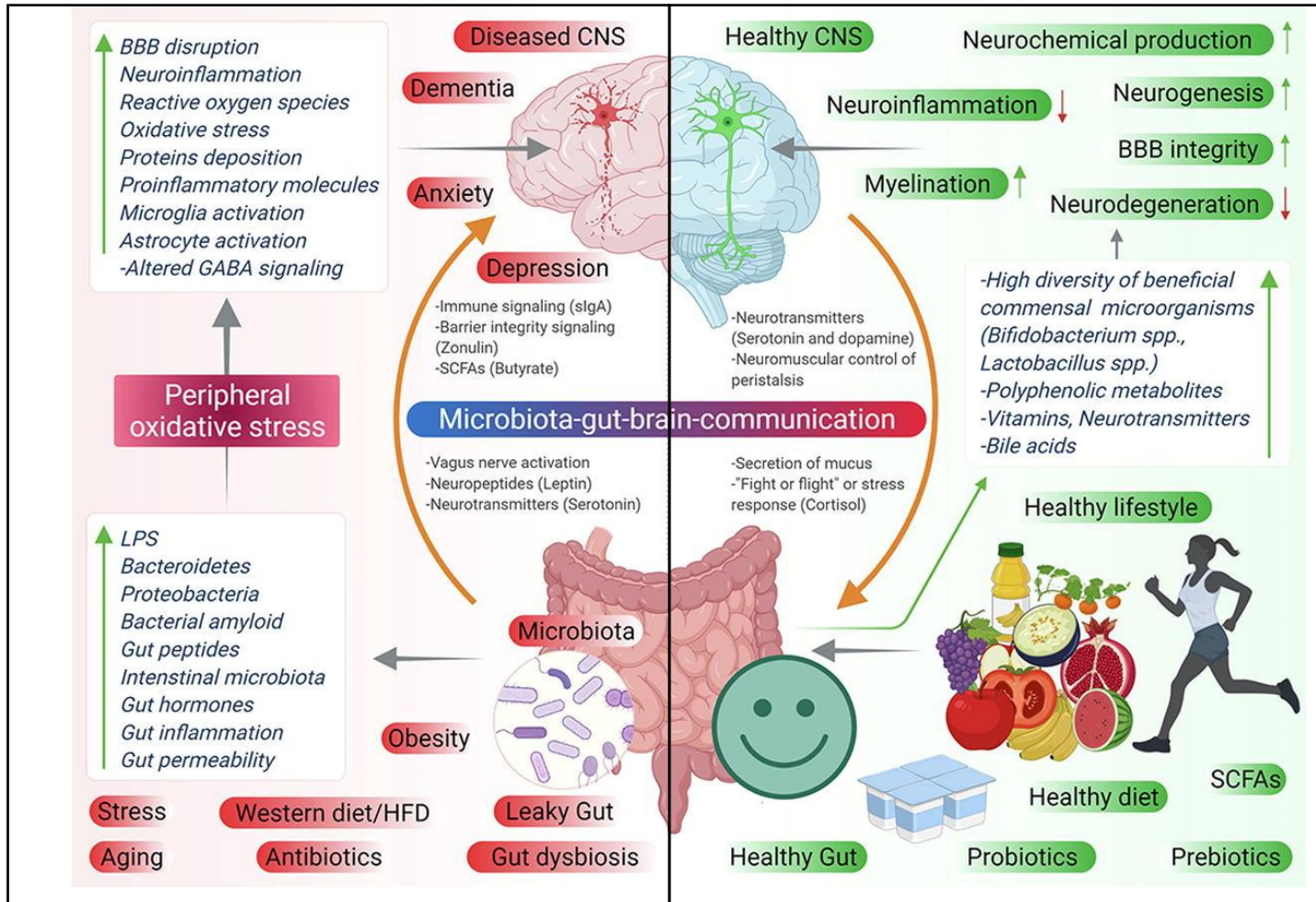


Chart creation: Dr. Brandon Brock 2023

SIBO TO THE BRAIN

HEALTHY AND UNHEALTHY



SIBO TO THE BRAIN

INFLAMMATION AND STRESS

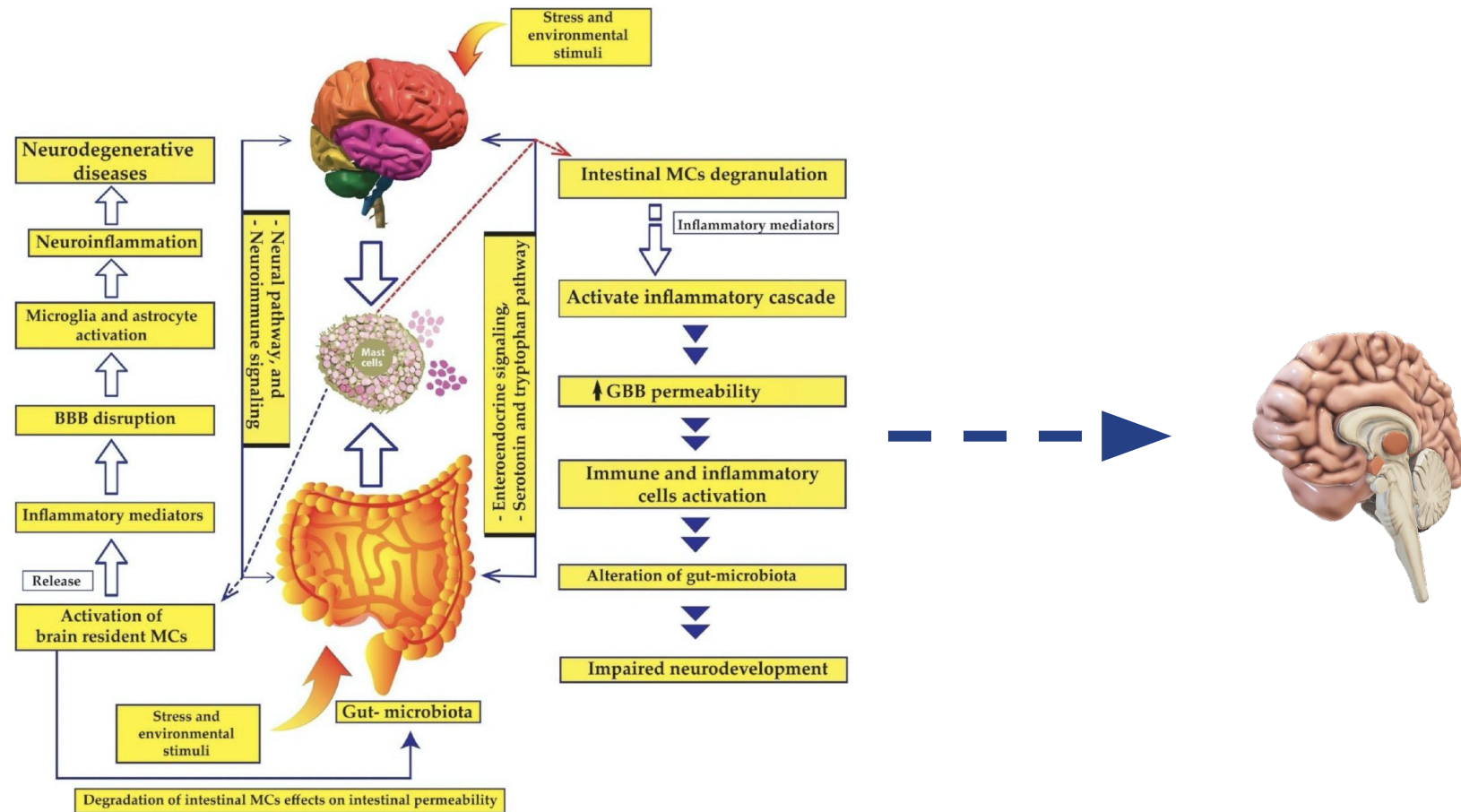
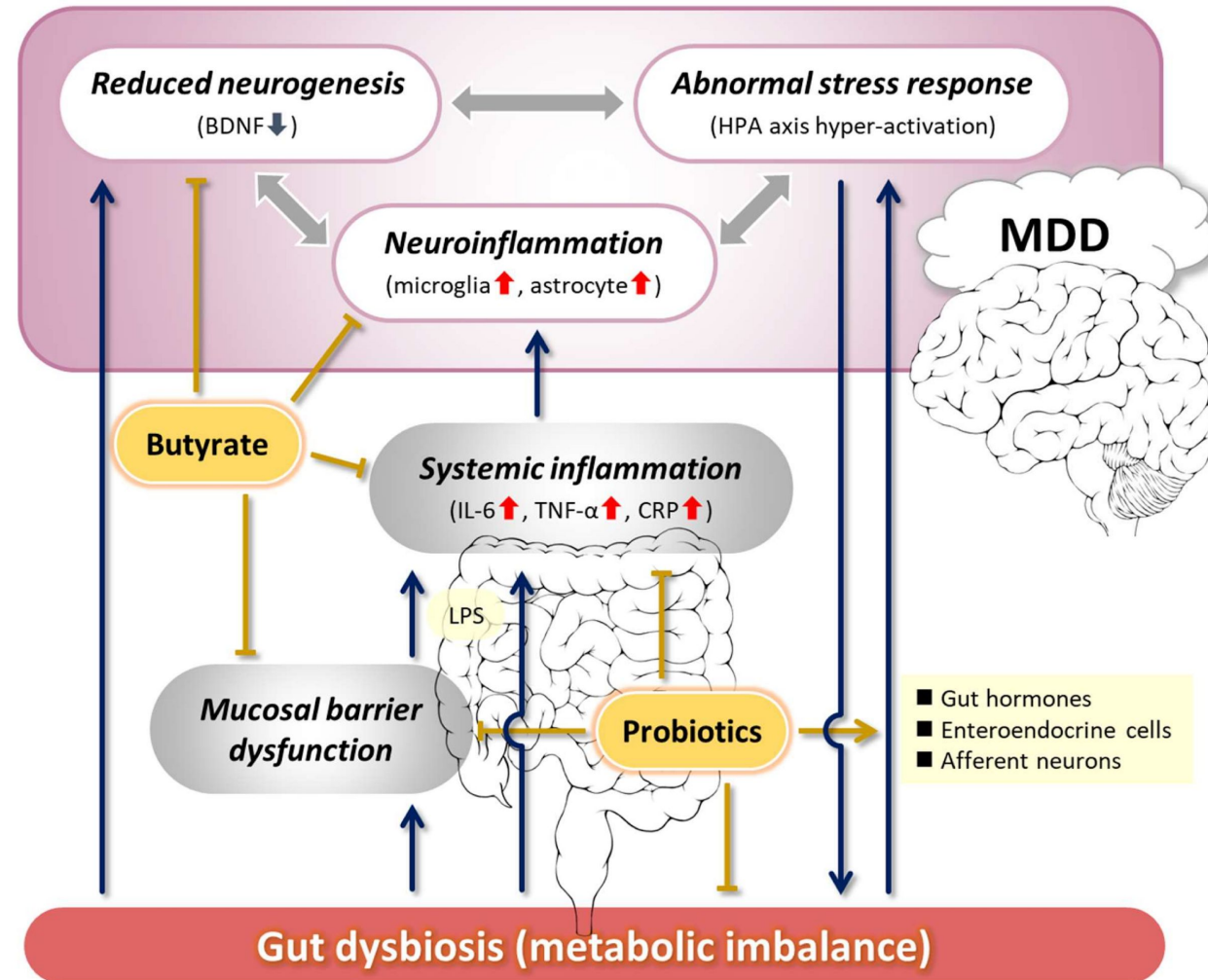


Fig. 1. Diagrammatic representation of the mast cell and gut-microbiota showing the proposed bidirectional interaction. MCs and gut-microbiota influences neural, neuroimmune, enteroendocrine, serotonin and tryptophan signaling pathways, which plays a significant role in neuroinflammatory mechanisms, neuro-immune responses, and neurobiological functions. Under stress and environmental stimuli may activate MCs and alter gut-microbiota composition lead to release of neuromediators, inflammatory cytokines, chemokines and histamine. These mediators may induce increased permeability of the intestinal gut microbiota and disrupt BBB, and Revised figures Click here to access/download;Figure;Revised figure.pdf activated microglia, astrocytes cells enhancing neuroinflammation result in neurodegenerative diseases. (MCs: Mast cells, GBB: Gut-blood barrier, BBB: Blood-brain barrier).

SIBO TO THE BRAIN

NEUROGENESIS AND DEGENERATION





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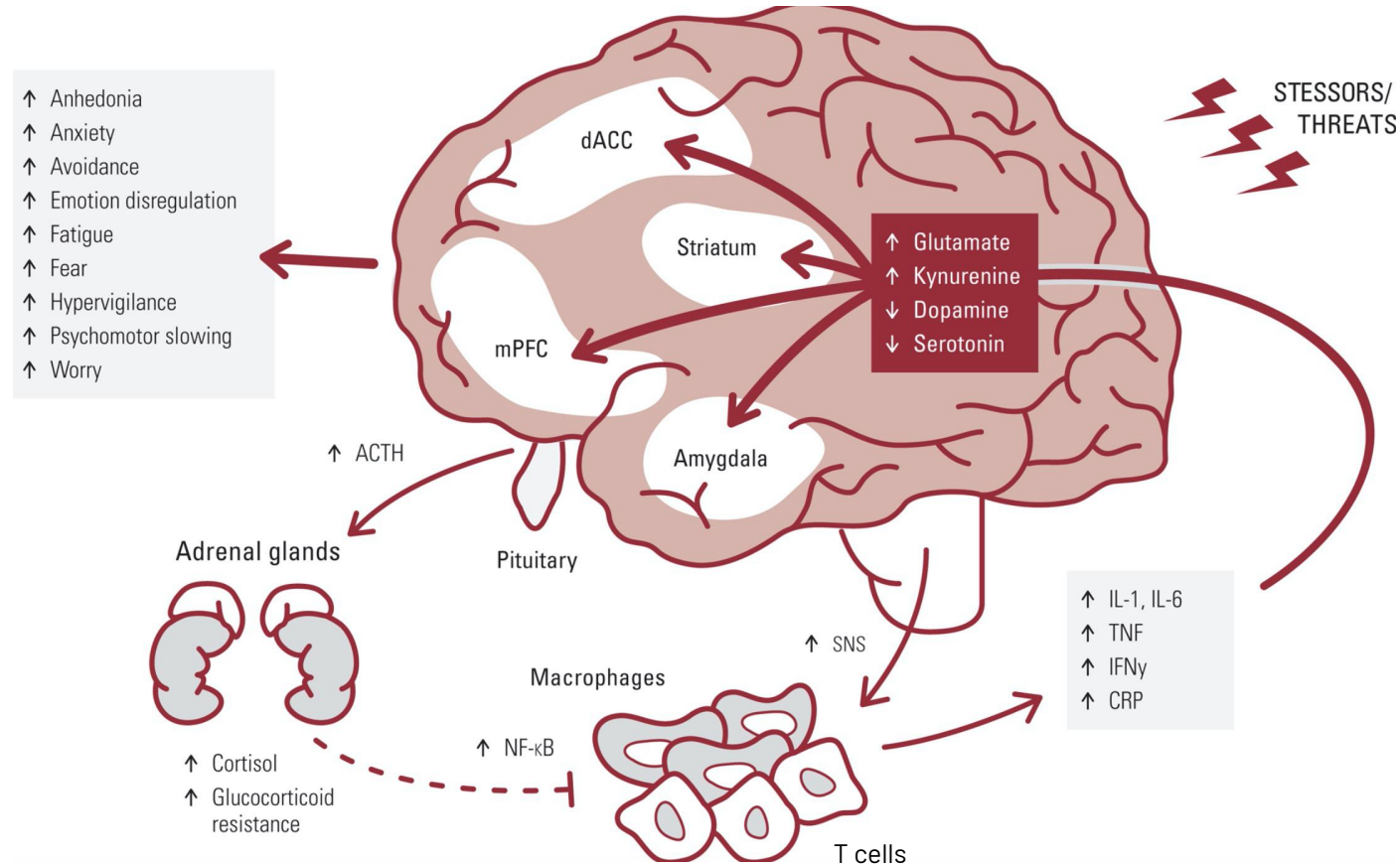
CORTICAL AND CHANGES IN THE CNS



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SIBO TO THE BRAIN

STRESS AND TRANSMITTERS



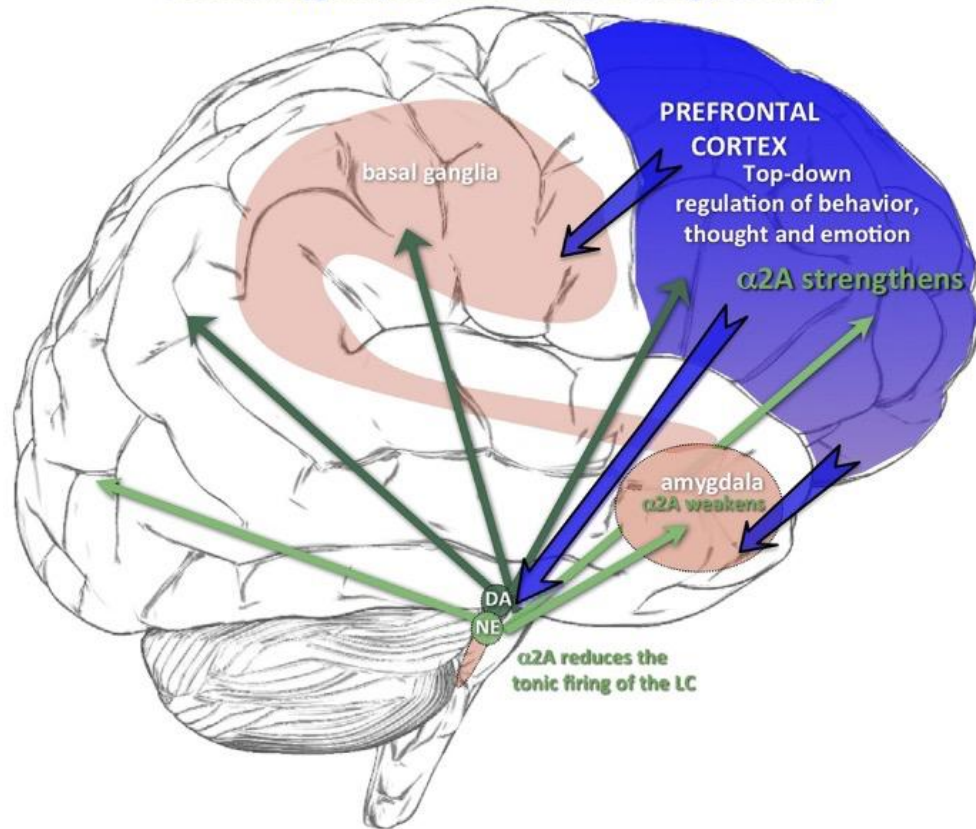
- Exposure to chronic stressors and threats drives adrenocorticotrophic hormone (ACTH), cortisol release, and increased sympathetic nervous system activity (SNS).
- SNS activation of NF-κB activity in immune cells increases expression of pro-inflammatory cytokines (e.g., IL-1, IL-6, TNF, IFN- γ) and CRP.
- Glucocorticoid resistance develops wherein cortisol does not as effectively inhibit NF-κB activity, thus creating a pro-inflammatory allostatic state that can contribute to psychiatric symptoms via cytokine actions on glutamate, kynurenine, dopamine and serotonin systems in brain regions underlying emotion regulation and affect, including the striatum, dorsal anterior cingulate (dACC), medial prefrontal cortex (mPFC) and amygdala.

SIBO TO THE BRAIN

EVENTUAL INFLAMMATORY CHANGES

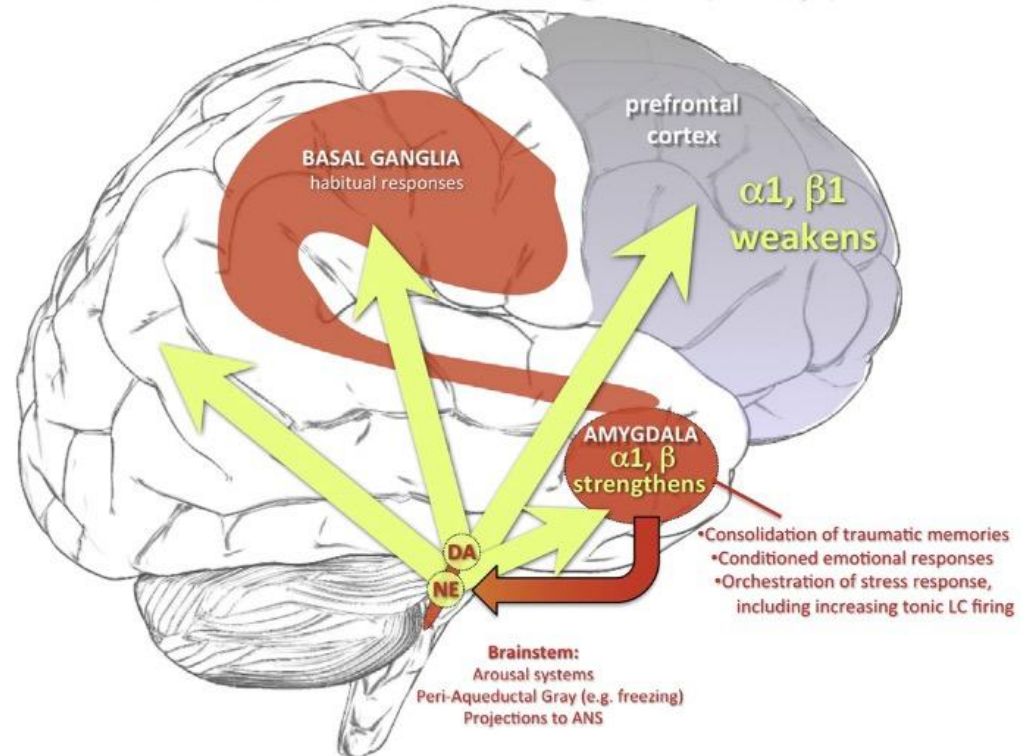
ALERT, SAFE, INTERESTED

Moderate levels of catecholamine release strengthen dlPFC, weaken amygdala, and reduce tonic LC firing (NE: $\alpha 2A$)



STRESS

High levels of catecholamine release weaken dlPFC, strengthen amygdala and striatum, and increase the tonic firing of the LC (NE: $\alpha 1, \beta 1$)





SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO) PREVALENCE

LOOKING AT TREATMENT



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TREATMENT CONSIDERATIONS FROM GUT TO BRAIN

- Look at the causation first and remove if you can – i.e., alcoholism
- Dietary and lifestyle considerations
- Eradicate causation (Medication / herbal)
- Removal of toxins
- Break down food: Enzymes – Bile – HCL – SCFA
- Restore the lining and reduce inflammation
- Support appropriate prebiotics and probiotics
- Motility support
- Barrier support
- Transmitter support
- BDNF support
- Vagal support
- Visceral massage
- Brain-based exercises
- Understand the presence of antibodies in the gut and brain to determine long-term care
- Look for other environmental perpetuators



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