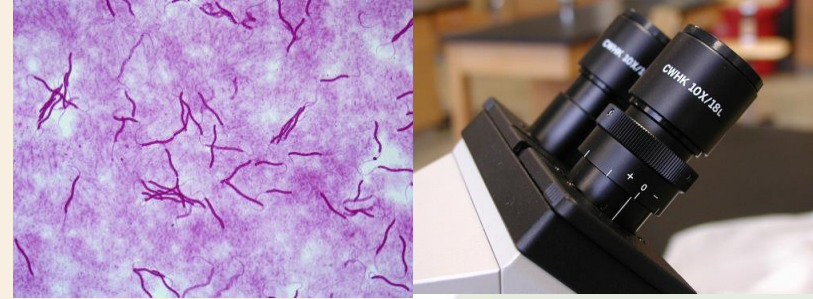


Lyme Borreliosis Complex: Barriers to Successful Treatment Outcome



JOSEPH G. JEMSEK MD, FACP

IM/ID-BC, HIV-AIDS/Lyme Specialist

Jemsek Specialty Clinic

Washington, D.C.

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CHRONIC ILLNESS UNCOVERED:

ACADEMY OF NUTRITIONAL MEDICINE, UK

Disclosure

Dr. Joseph G. Jemsek and the Jemsek Specialty Clinic have no financial relationship or any commercial interests related to the content of this presentation

- ▶ **23 Years background in HIV/ AIDS treatment through 2006**
 - Dr. Jemsek diagnosed the first cases of AIDS in North Carolina in 1983
- ▶ **Fully dedicated treatment of tick-borne illness since 2001**
- ▶ **Destination practice**
 - Patients from every state in America & over a dozen countries
- ▶ **Over 10,000 Lyme Borreliosis Complex (LBC) patients seen by the practice & currently more than 3000 active patients**
- ▶ **30 employees including 5 medical providers, 1 physician-scientist and 2 researchers**



“Chronic, relapsing, or otherwise ‘unexplained’ encephalopathy, arthritic symptoms, and neuropathy generally associated with tick-borne infections, spearheaded by *Borrelia burgdorferi* in combination with co-infecting organisms.”

-Joseph G. Jemsek MD, FACP

“Lyme Borreliosis Complex” is a more appropriate term for persistent Lyme Disease. The definition should be distinct from Acute Borreliosis, aka ‘Lyme disease’

Chronic, Relapsing, and Otherwise “Unexplained”

► I. ENCEPHALOPATHY

- Most common are decline in cognition and executive function, sleep disturbances, personality and mood alterations/disorders

► II. ARTHRITIC & PERIARTICULAR SYMPTOMS

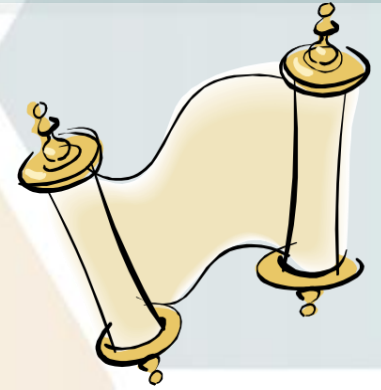
- Periarticular symptoms include inflammatory and non-inflammatory enthesopathies
- Lyme arthritic symptoms are generally migratory and may overlap with several rheumatologic syndromes

► III. POLYNEUROPATHY / MONONEURITIS MULTIPLEX

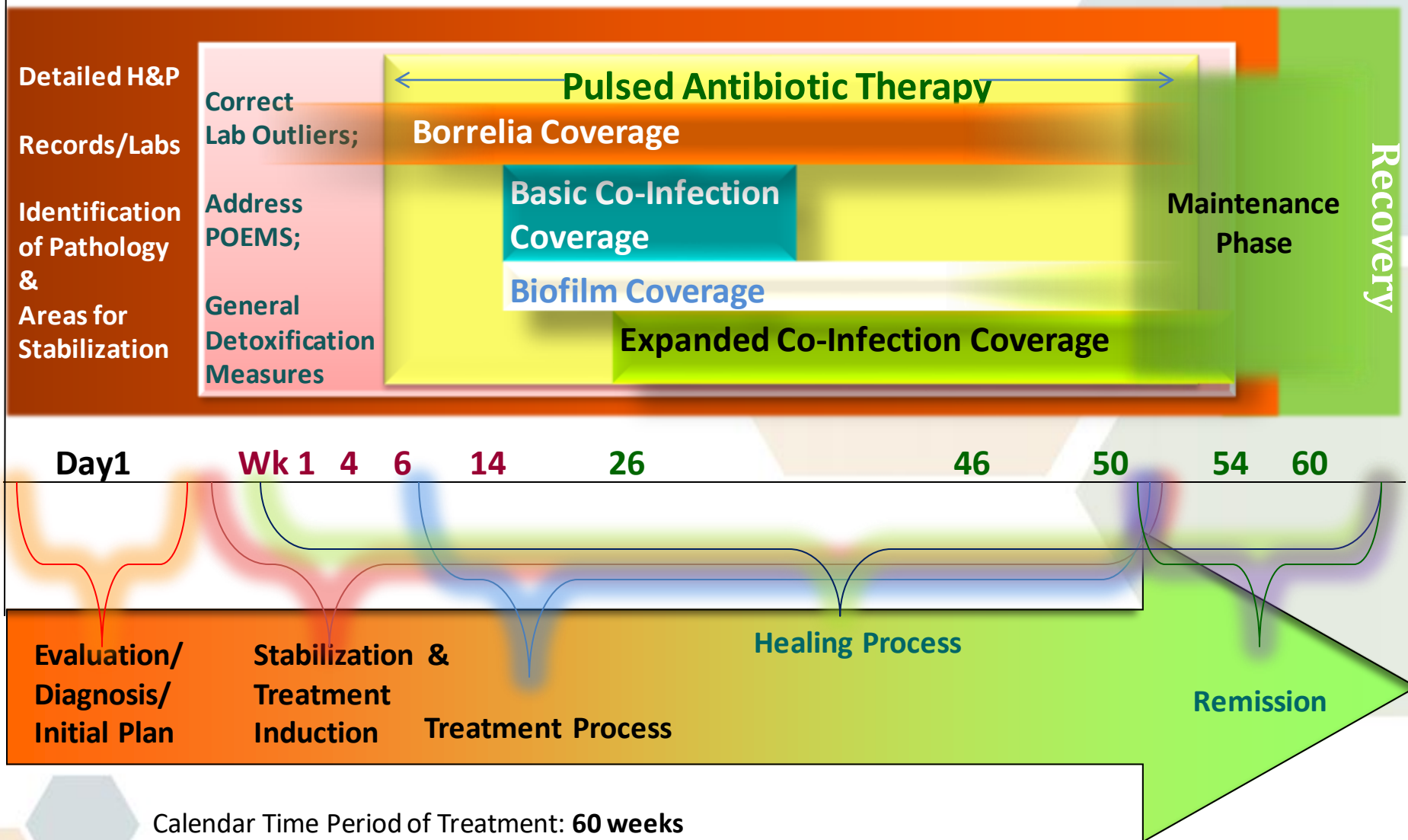
- May include sensory (C-fiber) lesions; cord myelitis; ganglionitis/plexitis and motor neuron disease

► **‘Complex’ reflects:**

- Polymicrobial infection (Multiple co-pathogens, e.g. Bartonella spp., Babesia spp., HGE & HME, etc.)
- Multisystemic disease (Tropism)
- Multi-compartmental neurologic disease (Tropism)
- Immune-evasive and immunosuppressive (Unique survival mechanisms, including Bb capacity for altered life forms, biofilm issues, etc)
- Once an LBC pathogen is “IN” it’s “IN”



- P** → Pain
- O** → Others: Social Support / Co-morbidities
- E** → Endocrine/Metabolic
- M** → Mood/Psychiatric
- S** → Sleep



Calendar Time Period of Treatment: **60 weeks**

Cumulative Weeks on Antibiotic Combination Therapy: **26 week**

Cumulative Dosing days of Antibiotic Combination Therapy: **102 Days**

Recognize possible presence of

- ▶ Refractory POEMS
- ▶ Biofilm
- ▶ Gut Dysbiosis
- ▶ Polymicrobial Complex
- ▶ Other Infections
- ▶ Methylation Pathways Defects
- ▶ Mitochondrial Defects
- ▶ Paradoxical Drug Reactions
- ▶ Subacute cholecystitis
- ▶ Motor Neuron Predominant Presentation (ALS Equivalence)
- ▶ Peri-Menstrual Volatility
- ▶ Unresolved Comorbidities
- ▶ Heavy Metal Toxicity, GMOs and Other Environmental Neurotoxins

- ▶ Resistance and recurrence are influenced by the formation of different morphological forms of Bb which can exist together in a matrix of different cell forms (cysts, spiral forms, granular, L-forms): all forms are capable of existing in biofilm
- ▶ One unique feature of Borrelia biofilm is the **externalization of its DNA**, which is incorporated into the matrix made by extracellular polymeric substances (EPS), giving the spirochetes a protective coat inside the host
- ▶ Presence of persisters and biofilm aggregate continue to elicit immune response



- ▶ **Bb can co-exist in common biofilms with multiple other pathogens**
- ▶ **Most pathogens capable of producing biofilm – as many as 95% (personal communication with Dr. Alan MacDonald)**
- ▶ **Biofilm enables Bb to survive despite a stressful environment created via actions of the immune system and antibiotics**



- ▶ Frontline drugs like doxycycline and amoxicillin kill replicating spirochetal form of *B. burgdorferi* but have little activity against non-replicating persisters or biofilm-like aggregates or microcolonies of Bb.
- ▶ Difficulty with biofilm disbursement a barrier to successful outcome and increase chances of relapse
- ▶ Intelligent eradication of biofilm in terms of treatment is important in treatment of LBC
- ▶ Novel 'designer' drugs aim at destabilization of the biofilm, opening up new horizons in the treatment and cure of Lyme disease



Human MicroBiome:

The Consortium of the symbiotic bacteria that reside within the body

- ▶ **The GI microbiota play a critical physiologic role and functions akin to a body organ**
- ▶ Healthy gut resists infection
- ▶ Immune surveillance and stimulation
- ▶ Enhancement of GI motility and function
- ▶ Healthy aging correlates with diverse microbiome, while sickness and poor aging is associated with narrowing microdiversity

- ▶ Digestion and Metabolism of plant compounds, xenobiotics and bile acids (for e.g. Demethylation of methylmercury, a bioaccumulative environmental toxicant)
- ▶ Regulation of intestinal bile acids and serotonin metabolism
- ▶ Synthesis of Vitamins (K and B vitamins), short chain fatty acids and polyamines
- ▶ Calcium Absorption /Alteration in Bone Strength and Tissue material properties

- ▶ **Communication between the gut microbiota, immune & CNS impacts our psychopathology through**
- ▶ Vagus nerve (80-90% afferent fibers; only 10-20% efferent)
- ▶ Gut Hormone Signaling
- ▶ Immune Surveillance & Maturation
- ▶ Tryptophan/Serotonin Metabolism
- ▶ Influences molecular pathways of hunger, satiety and energy balance
- ▶ Epigenetic programming - Diet dependent endogenous small-molecule metabolites from the microbiota regulate global histone acetylation/methylation, leading to transcriptional responses

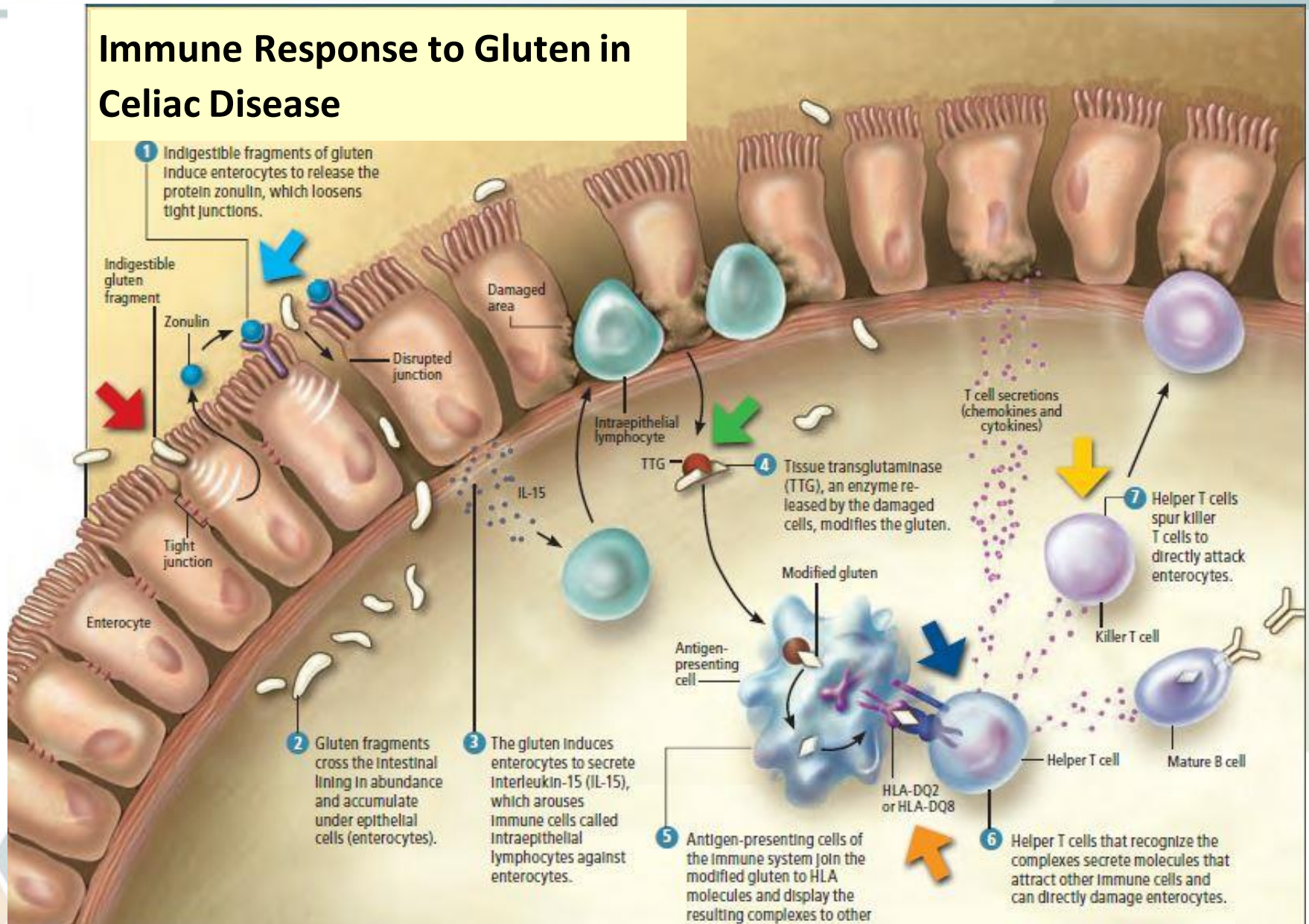
Dysbiosis: Disorder in the normal microbial distribution/quotient in the digestive system resulting to negative health symptoms

- ▶ Antibiotic-induced microbiota alterations
- ▶ Lyme Disease
- ▶ Medications (e.g. disruption of mucosal barrier through NSAIDS-induced prostaglandin deficiency)
- ▶ Increased Incidence of Caesarean Deliveries
- ▶ Lack of Breastfeeding
- ▶ Infections/Co-infections
- ▶ Stress
- ▶ Environmental influences - GMOs and Heavy Metal Toxicity, Other Environmental Neurotoxins
- ▶ Host Genetics

- ▶ Borrelia may infect mucosal , muscular or serosal surface of the bowel wall
- ▶ Involved in Biofilm formation in mucosal membrane, blood, brain & tissues
- ▶ Cytotoxin release from biofilm alter Brush Border Membrane Permeability (resulting to 'leaky gut')
- ▶ Increased antigenic mimicry- new allergies and autoimmune dysfunction.
- ▶ Upregulation of lymphocytic subtypes ⇒ stronger IgE response, eosinophilic accumulation and heightened allergic sensitivity.
- ▶ Mild & diffused Vasculities in GI vasculature
- ▶ Biliary Stasis & Other secretory problems & Poor nutrient absorption

Treating Dybiosis prevents overgrowth of pathogenic strains during antibiotic Tx, disrupts spirochetal biofilm, ensures adequate nutritional support and helps restore effective immune response.

Immune Response to Gluten in Celiac Disease



EUBIOSIS

- ▶ **Symbiotic coexistence of Host and Microflora**
- ▶ Protection of the intestinal mucosa against invading microorganisms
- ▶ **Contributes to immune system maturation and proper stimulation**
- ▶ Antagonistic effect on pathogenic bacteria and fungal overgrowth
- ▶ **Nutrient Digestion**
- ▶ Vitamin and protein synthesis
- ▶ **Better tolerance to antibiotic treatment**

DYSBIOSIS

- ▶ Harmful coexistence of Host & Microflora
- ▶ **Damage to the intestinal epithelium-
>Gut wall thickening and reduced nutrient resorption**
- ▶ Weakening of the Immune System
- ▶ **Unprocessed Antigen and Allergen exposure: Increases food sensitivity and non-specific immune reactions.**
- ▶ Increased gas production (H_2S , NH_3 , CH_4 , CO_2)
- ▶ **Acceleration of cell turnover – increased energy need**
- ▶ Vitamin deficiencies

- ▶ **Identify Aggravating Factors**
- ▶ **Remove/control offending agents including yeast overgrowth**
- ▶ **Reinoculate the gut with beneficial microbes**
 - Probiotics/Kefir with beneficial Strains e.g. *L. rhamnosus*
- ▶ **Repair mucosal lining**
 - Glutamine/SCFA
- ▶ **Histamine Management Considerations**
- ▶ **Non-Hepatic Hyperammonemia Management Considerations**
- ▶ **Gluten Avoidance**
- ▶ **Increase Fiber Content/Prebiotics**

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- ▶ **Multiple failed previous therapies**
- ▶ **Recrudescent Babesiosis**
- ▶ **Difficulty with Biofilm disbursement**
- ▶ **Poor co-morbidity management**
- ▶ **Poor Social Support Structure**
- ▶ **High and persistent Biomarkers of Oxidative Stress**
- ▶ **Motor Neuron Predominant Presentation (ALS Equivalence)**
- ▶ **High Heavy metal levels**
- ▶ **Multiple Methylation Pathways Defects of high clinical significance**
- ▶ **Poor tolerance to nutritional supplements**
- ▶ **Pattern of Paradoxical Drug Reactions**

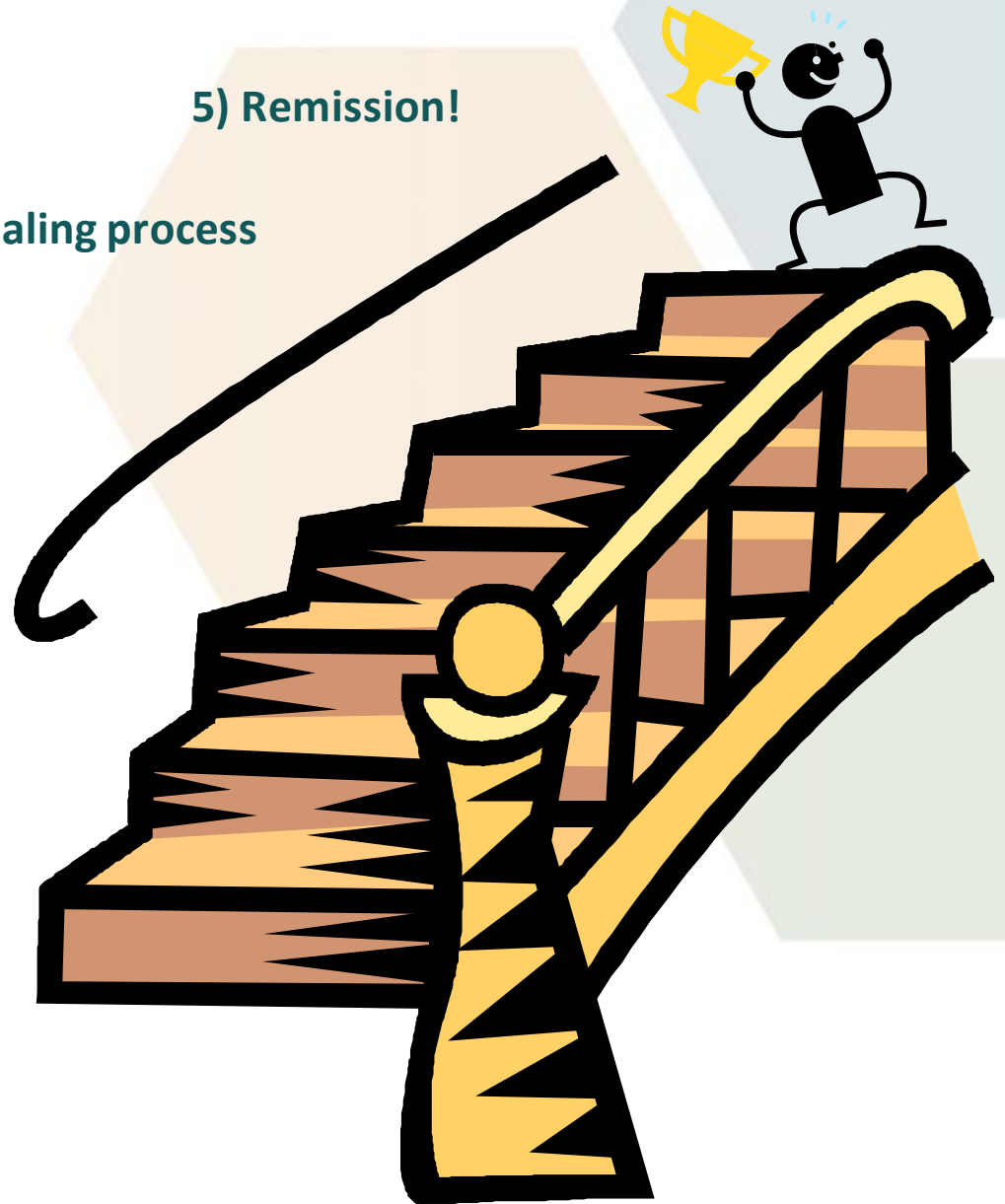
1) Evaluation, interpretation,
and prioritization of major
pathological processes based
on clinical and laboratory
evaluations

2) Stabilization of faulty
central life functions and
reversal of stressors

3) Treatment

4) Healing process

5) Remission!



Thank You for Attending