Autoimmune "Three-Legged Stool" Assessment

Client Name: _____ Date: ___

Practitioner Goal: Use this tool to identify which "legs" of the autoimmune stool are currently active and determine the client's position on the Spectrum of Loss of Tolerance.

Section 1: The Three-Legged Stool (Risk Factors)

Check all that apply to identify the primary drivers of immune dysfunction.

Leg 1: Genetic Susceptibility (The Blueprint)

- [ ] Family history of autoimmunity (Thyroid, RA, Celiac, Lupus, MS, etc.)
- [ ] Known HLA-type SNPs (if genomic testing has been completed)
- [ ] History of "mystery" symptoms in childhood or adolescence

Leg 2: Environmental Triggers (The Exposome)

- [ ] **Stealth Pathogens:** History of Mono/EBV, Lyme, or frequent viral infections.
- [ ] **Toxic Metals:** Presence of dental amalgams, high seafood intake, or industrial exposure.
- [ ] **Chemicals:** Frequent use of plastics (BPA), scented products (Phthalates), or pesticides.
- [ ] **Dietary Antigens:** High intake of gluten, A1 dairy, soy, or processed "fast" foods.

Leg 3: Intestinal Permeability (The Gateway)

- [ ] Chronic bloating, gas, or "leaky gut" symptoms.
- [ ] History of frequent antibiotic, NSAID (Advil/Motrin), or PPI use.
- [ ] High-stress lifestyle (Cortisol resistance).
- [ ] Known food sensitivities or "reacting to everything."

Section 2: Th17 Dominance & Lifestyle Drivers

Th17 cells drive tissue destruction. Assess these factors to gauge "fire" intensity.

Factor	Status/Frequency	Notes
Salt Intake	High / Med / Low	High sodium induces Th17 cells.
Vitamin D Level	<30 / 30-50 / >60	Goal: 60-80 ng/mL for immunomodulation.
Stress Levels	High / Med / Low	Chronic stress allows IL-17 to run unchecked.
Gut Health	Poor / Fair / Good	Segmented Filamentous Bacteria (SFB) drive Th17.

Section 3: Identifying the Autoimmune Spectrum

Where is the client currently positioned? (Circle One)

1. **Silent Autoimmunity:** Positive antibodies found on labs, but NO symptoms or tissue damage.
2. **Autoimmune Reactivity:** Positive antibodies AND symptoms (brain fog, joint pain, fatigue), but no "official" diagnosis.
3. **Autoimmune Disease:** Significant tissue destruction and clinical diagnosis (e.g., Hashimoto's, RA).

Section 4: Practitioner Observations & Reflection

Primary Mechanism Suspected: ( ) Molecular Mimicry ( ) Bystander Activation ( ) Epitope Spreading

Clinical Priority (Which leg do we address first?):

Observations:

Next Steps:

- [ ] **Reveal:** Order/Review antibody panels (TPO, ANA, etc.) and Zonulin levels.
- [ ] **Remove:** Eliminate "Big Four" triggers identified in Section 1.
- [ ] **Repair:** Initiate gut barrier healing protocol (The "Safety" on the gun).
- [ ] **Modulate:** Optimize Vitamin D and manage Th17 drivers (Stress/Salt).
