Working Summary: Dan's Theory of Obesity as a Transmissible Microbial Disorder

Overview:
This document outlines a comprehensive theory of obesity rooted in the presence and behavior of a transmissible microbiome pathogen. The model departs from conventional paradigms that frame obesity solely in terms of caloric imbalance, psychological addiction, or metabolic dysregulation. Instead, it posits a layered causal framework—termed "the triad"—that explains not only initial weight gain but also long-term difficulty in weight loss and the high relapse rate after dieting.

The Triad Theory of Obesity

1. Pathogenic Microbiota (Root Cause)
A transmissible microbial or fungal agent, likely acquired through close interpersonal contact (e.g., cohabitation, romantic partners), colonizes the lower gastrointestinal tract. This pathogen:

* Survives at low levels in many people without overt symptoms.
* Modulates appetite to increase caloric intake beyond homeostatic needs.
* Thrives on surplus dietary carbohydrates, particularly processed starch and sugar.
* May exhibit biofilm or spore-forming behavior, making eradication extremely difficult.

2. Metabolic Consequences (Insulin Resistance Loop)
Sustained overfeeding—driven by the pathogen—increases insulin secretion and eventually causes insulin resistance:

* Appetite becomes dysregulated as leptin and insulin signaling break down.
* Blood sugar volatility increases cravings for high-glycemic foods.
* Fat storage becomes easier and fat burning harder, reinforcing overfeeding.

3. Behavioral Addiction (Psychological Layer)
Over time, food use becomes conditioned:

* Emotional, habitual, and identity-based eating emerges.
* Food transitions from nutrition to ritual, reward, and relief.
* Even after the pathogen is suppressed and insulin resistance reduced, psychological hunger remains.

4. Reinstatement & Relapse (Post-Diet Risk Layer)
After successful weight loss and containment of the pathogen:

* Reintroduction of addictive foods (especially high-starch, high-sugar) can reawaken the pathogen.
* Behavioral addiction resurfaces through familiar rituals and justifications (e.g., "I bought it, so why not eat it?").
* Appetite quickly escalates, and obesity begins to return.

Case Narrative: Dan's Experience

* Obesity began in marriage, coinciding with stress and high-carb feeding patterns.
* Appetite surged chronically for decades, unaffected by willpower.
* A short-term suppression of appetite occurred following a painful dental procedure, unintentionally mimicking a starvation protocol.
* A period of sustained calorie and carb restriction led to major weight loss (\~80 lbs).
* Relapse began subtly: family visiting brought favorite snacks into the home, triggering the addiction-behavior pattern.
* Full relapse followed after a brief return to indulgent food behaviors.

Reframing the Problem:

* Obesity is not a moral failing.
* It is not strictly behavioral.
* It is not only hormonal.
* It is an infectious, behavioral, and neurochemical cascade that begins in the gut and spreads through routine human contact and food environments.

Next Step:
Develop a structured, repeatable clinical treatment regimen based on the triad model that addresses:

1. Suppression of the pathogen (terrain modification)
2. Resetting of metabolic feedback loops
3. Reprogramming of food-related behavior and identity
4. Strategies to defend against relapse and reinfection

This working theory is unvalidated until weight loss is sustained under the current kratom-based appetite suppression experiment. Further data will inform whether this model holds under rigorous application.