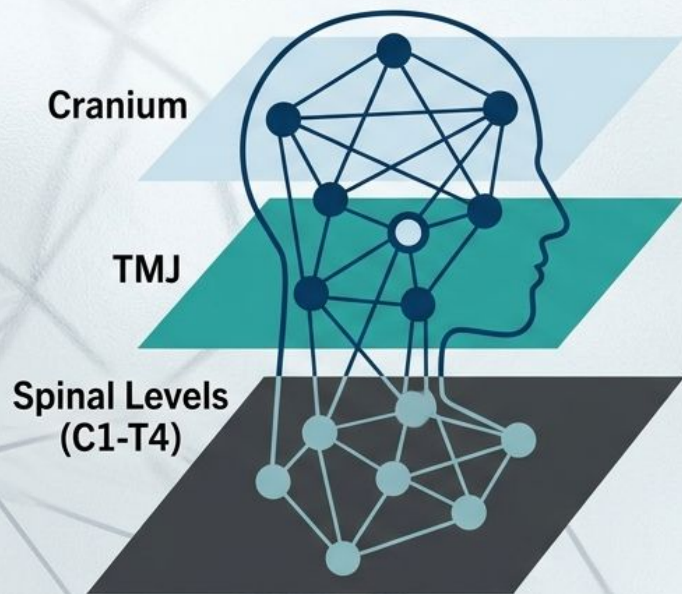
# Craniofacial Complexity: A Neurmyofascial Framework for TMJ, Tinnitus, and Retinal OCT Findings




## Executive Summary & The NMF Model

- 30+ years of clinical practice
- 50,000+ treatments administered
- **Objective:** Shift diagnostic lens to an interconnected regional network

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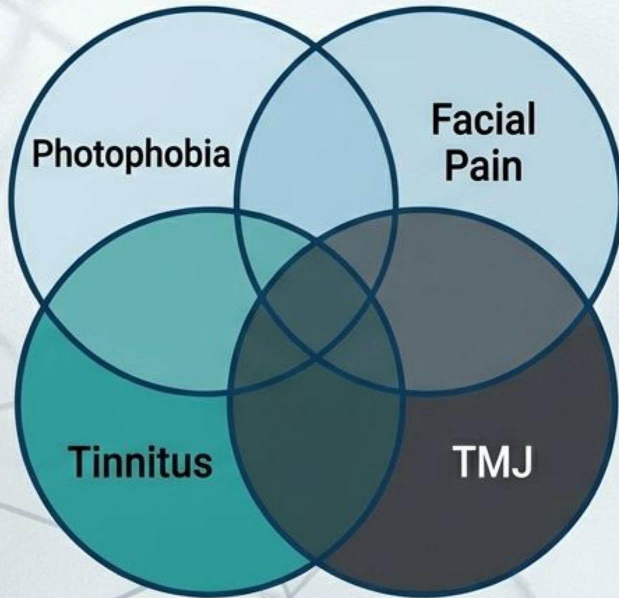
# Slide 2: The Clinical Anomaly (Overlapping Symptoms)

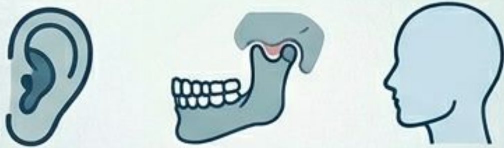



# CLUSTER OF SYMPTOMS

- TMD associated with a 2.62x increased risk of tinnitus
- Patients present with multiple co-occurring conditions.

# Slide 3: The Interpretive Shift: Isolated vs. Regional Models

TRADITIONAL ISOLATED ORGAN MODEL

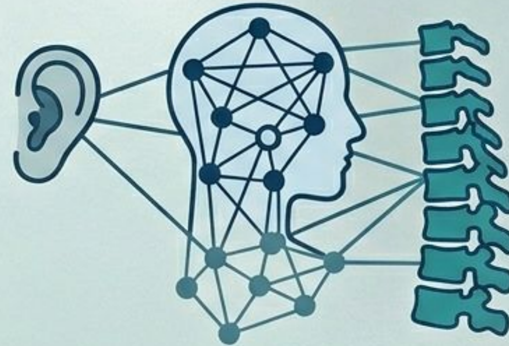


TMJ is primary joint disorder

Tinnitus is primary otologic/acoustic damage

INTERPRETIVE SHIFT

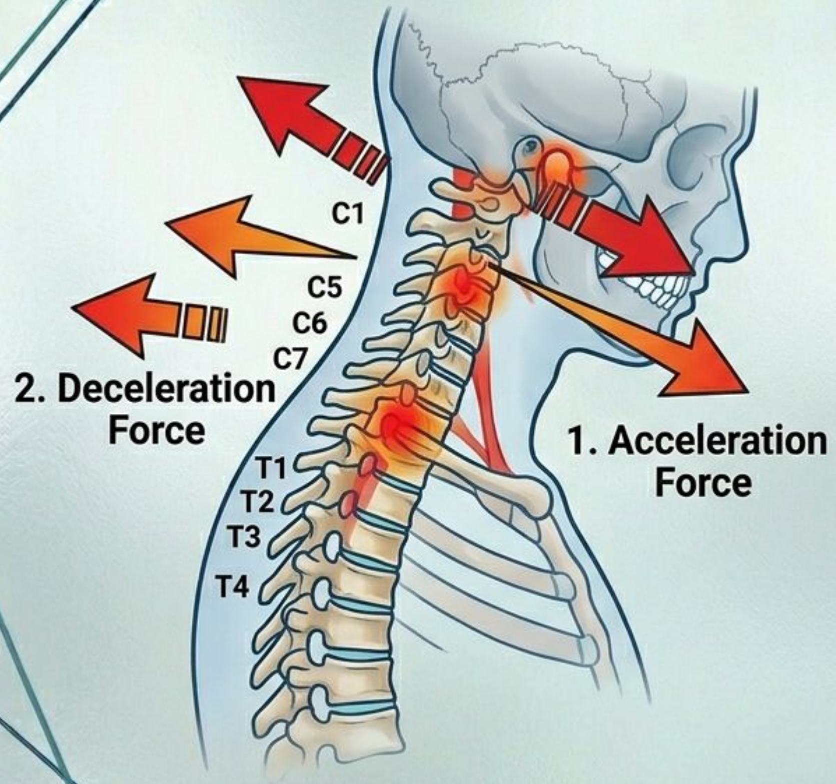
NEUROMYOFASCIAL REGIONAL NETWORK MODEL



**TMJ and Tinnitus are Distal Referred Phenomena**

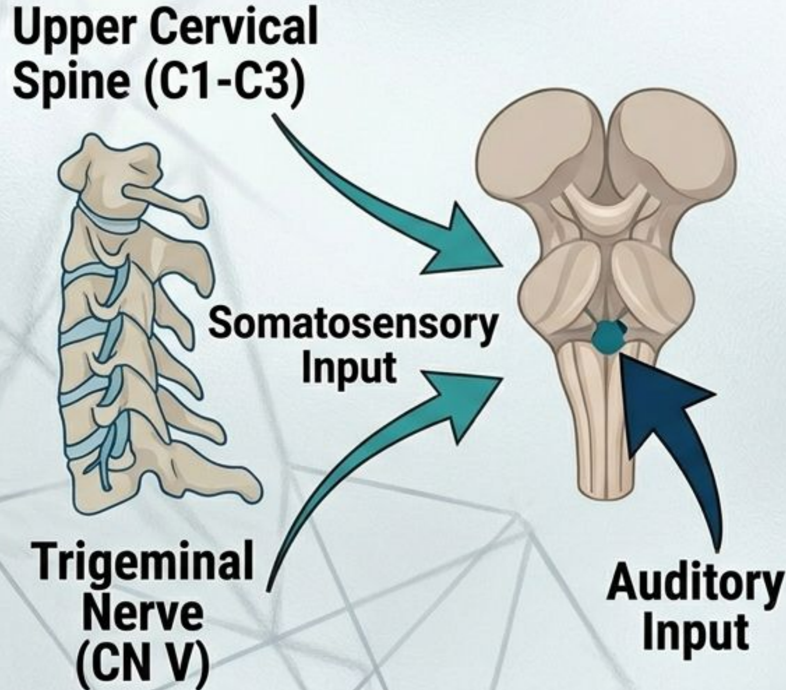
Driven by cumulative trauma and entrapment

# Slide 4: Acceleration-Deceleration & Cumulative Trauma: The Structural Catalyst




- Cumulative Micro-Trauma creates connective tissue change
- Undiagnosed injury at spinal levels (C1-T4)
- Catalyst for the Neuromyofascial Cascade

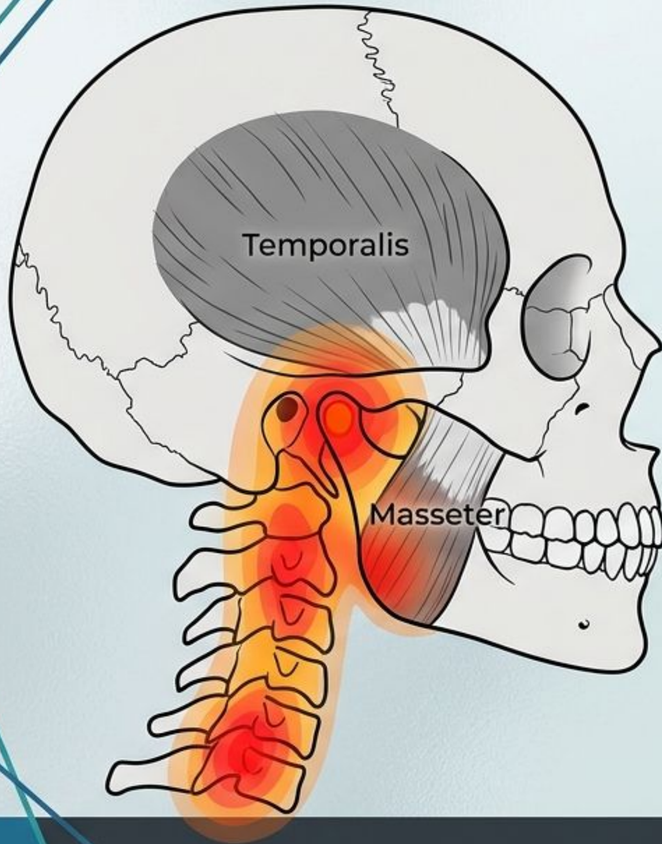
# Slide 5: Mechanism 1: Neural Convergence Schematic




## Dorsal Cochlear Nucleus (DCN)

- Integration of auditory and somatic signals
- Aberrant cervical input causes DCN hyperactivity
- Tinnitus perceived as phantom sound

# Slide 6: TMJ as Referred Masticiatory Dystonia




## THE DYSTONIC LOOP

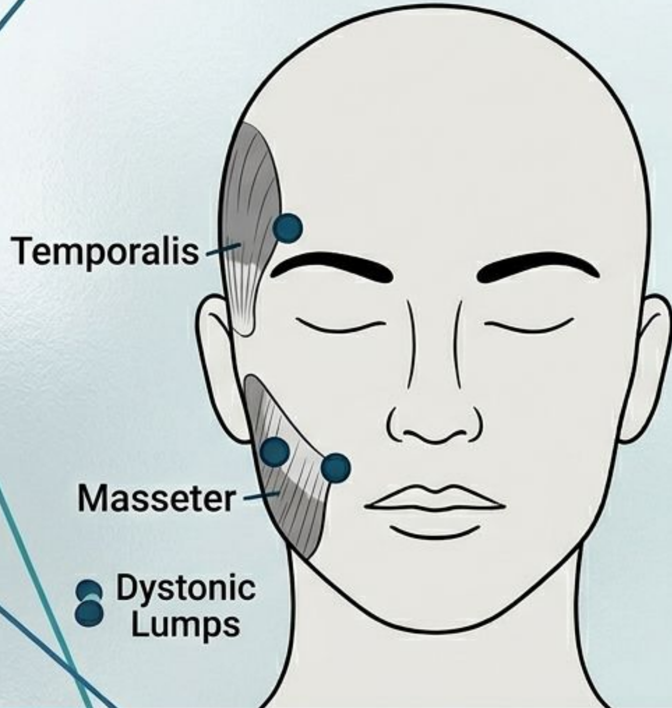
**Spinal drivers** induce chronic spasticity

Jaw dysfunction often a secondary symptom

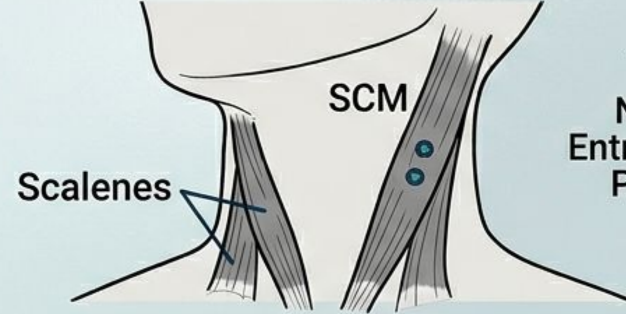
Dystonia creates TMJ subluxation and clicking

# Slide 7: Clinical Assessment: The Palpation Protocol

## Step 1: Craniofacial Mapping



## Step 2: Lateral Neck Assessment



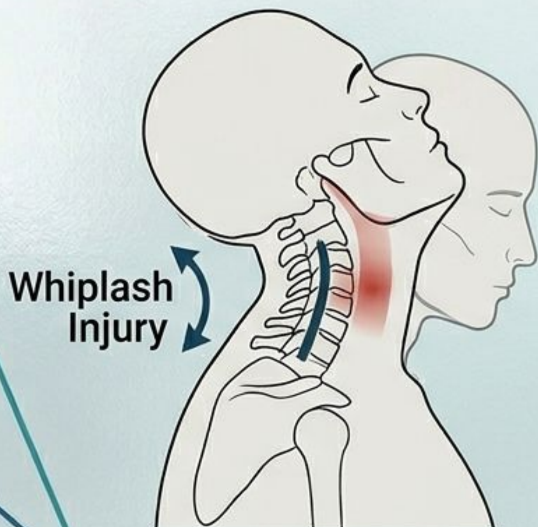

# MAPPING THE NEUROMYOFASCIAL FINGERPRINT

- Identifying referred Extra-Articular pathology

# Slide 8: The Catalyst: Acceleration-Deceleration (Whiplash)

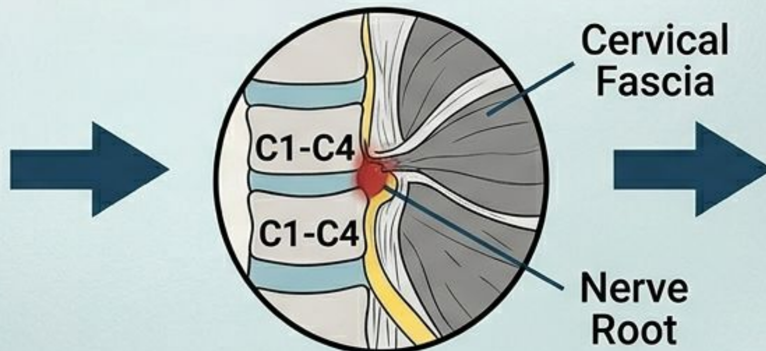
## Neuromyofascial Cascade

### 1. Initial Trauma

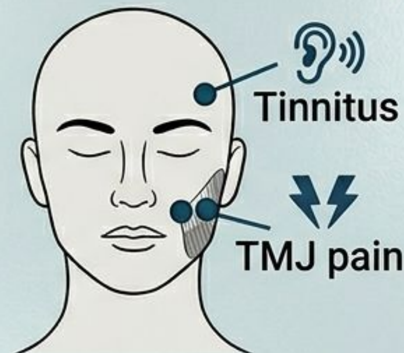



1. Initial Trauma

### 2. Fascial Thickening & Entrapment




### 3. Distal Symptom Manifestation




**SUB-CLINICAL INJURY DRIVES CRANIOFACIAL COMPLEXITY**

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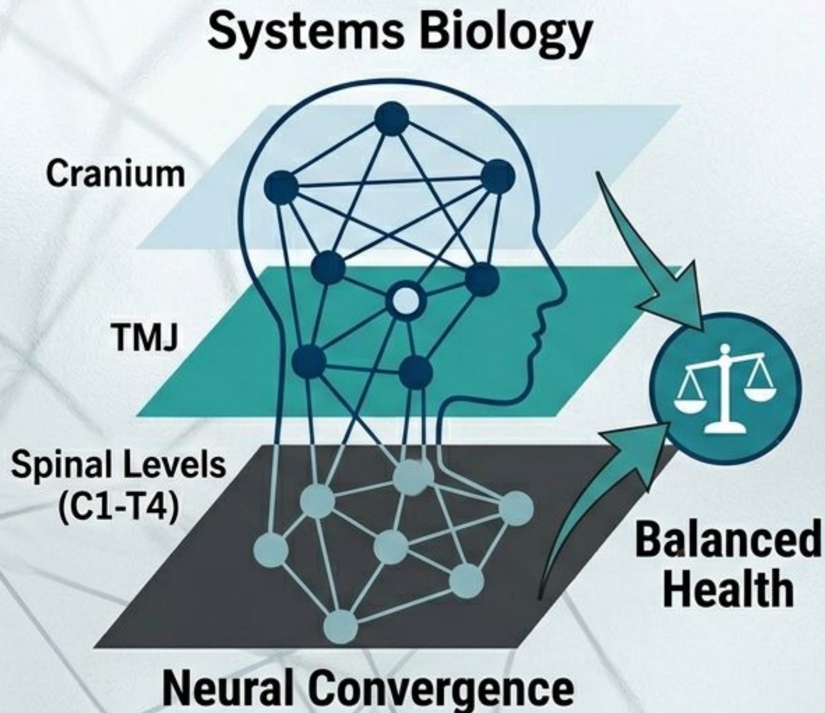
# Slide 9: Peer-Reviewed Support & Evidence Summary

| Study/ Author | Key Finding | Clinical Implication |
|---|---|---|
| Lee et al. (2016) | 2.62x Tinnitus Risk in TMD Patients | Validates TMJ-Tinnitus link |
| Michiels et al. | Cervical PT Reduces Somatosensory Tinnitus | Confirms Cervical Spine as treatment target |
| Kreuzer et al. | Trauma-Associated Tinnitus = Higher Distress | Highlights Whiplash as distinct pathology |

## THE EVIDENCE SEAT

- Data supports the Neuromyofascial model

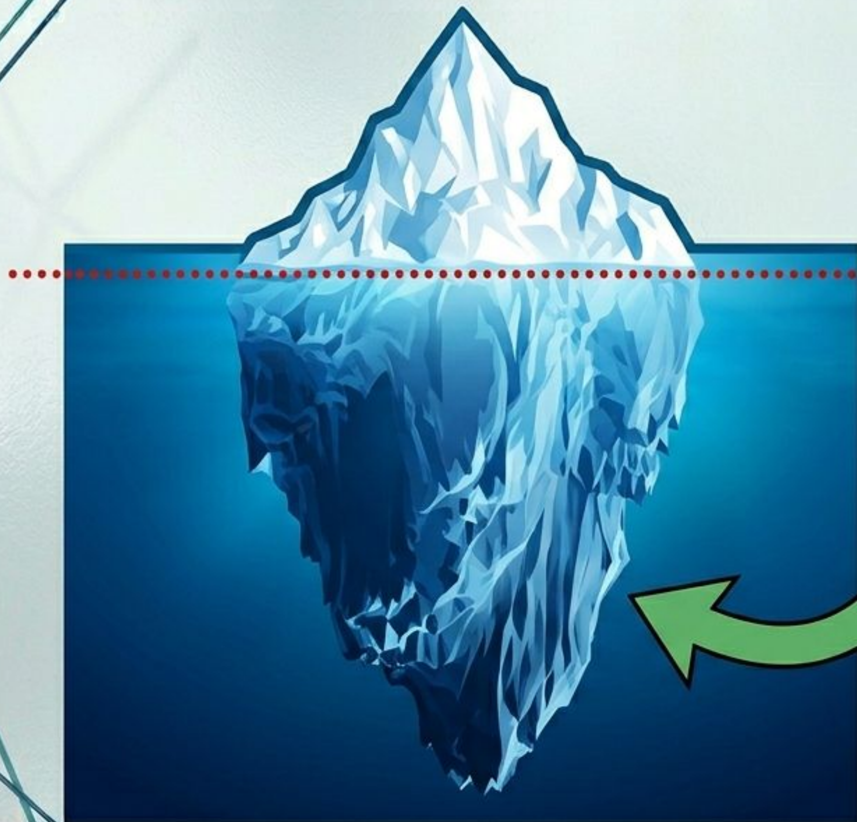
# Slide 10: Conclusion & Interdisciplinary Assessment




## Outcome Goal: Interdisciplinary Path

- Address spinal drivers in parallel with conventional treatments
- Enhance clinical outcomes for complex cases
- Cervical & thoracic spine must be included in diagnostic process
- NMF Science LLC - 30+ Years, 50,000+ Treatments

# Slide 11: The Iceberg Diagnostic Path



1. **VISIBLE SYMPTOMS**
   (The Tip)

TMJ Pain Tinnitus

2. **THE DRIVER** (The Base)

Cervical-Thoracic
Spinal Column (C1-T4)
Sub-clinical Whiplash

**RESOLVING COMPLEXITY REQUIRES TREATING THE ENTIRE STRUCTURE**

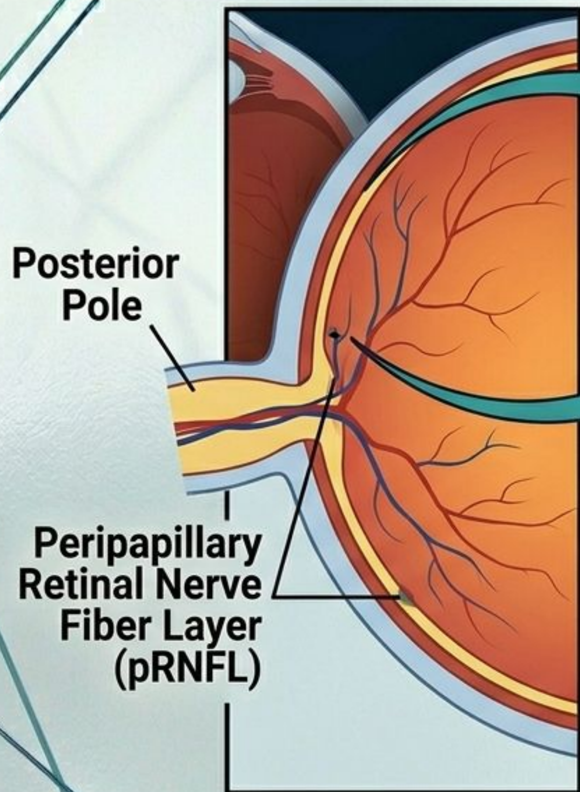
# Slide 12: Peer-Reviewed Support: Tinnitus-TMD Link

| Study | Key Finding | Level of Evidence |
| --- | --- | --- |
| Lee et al. (2016) | 2.62x Increased Tinnitus Risk after TMD | High |

## Lee et al. (2016) Meta-Analysis

- Data validates the TMJ-Tinnitus correlation
- NMF framework focuses on spinal pathology as original driver
- Reframes TMD as referred manifestation of cumulative trauma

# Slide 13: The Retinal OCT Biomarker (pRNFL)



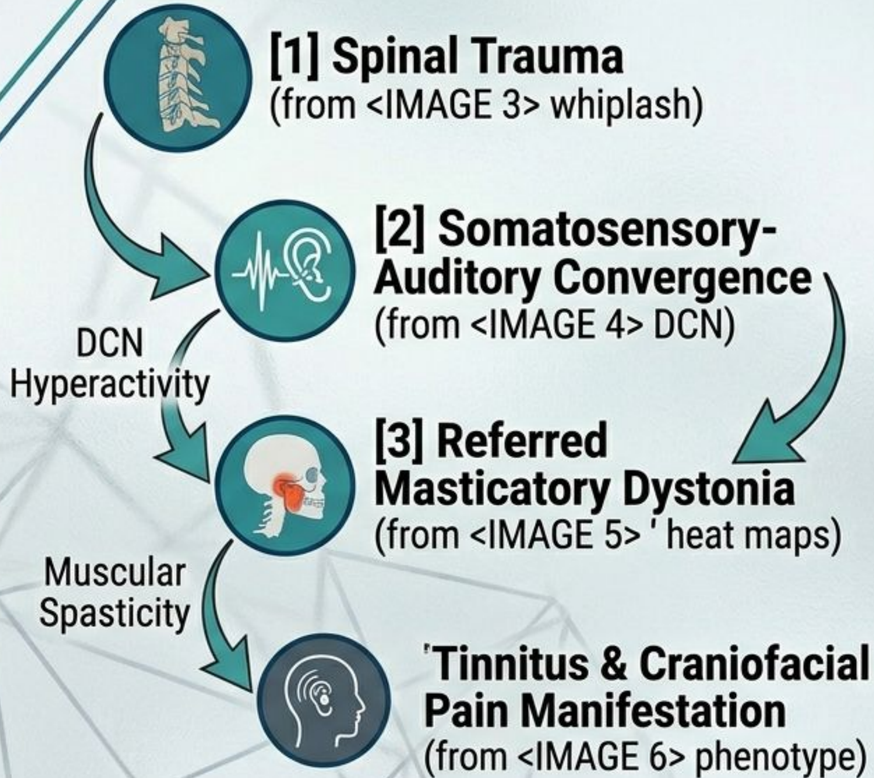
Healthy Control

Generalized Spinal Pathology (from IMAGE-3 drivers)

## Emerging Objective Metric

- pRNFL thinning (generalized) observed in NMF patients
- May indicate indirect neuroaxonal stress/tethering
- Potential screening tool for hidden spinal drivers

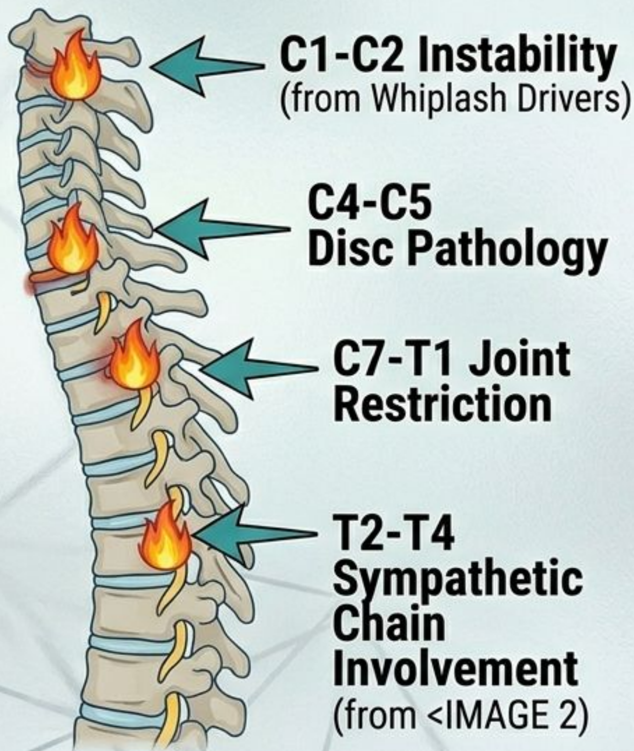
# Slide 14: Integrating the 4 Mechanisms (The NMF Cascade)




## System-Wide Neuromyofascial Cascade

- Cervical trauma initiates somatosensory dysregulation
- Aberrant signals drive DCN hyperactivity (tinnitus percepetion)
- Dystonia alters TMJ mechanics and retinal nerve tension

# Slide 15: Assessing Cervical-Thoracic Pathology

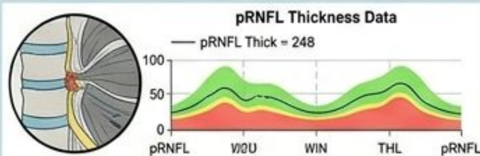


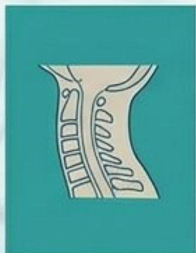

## Diagnostic Focus: The Structural Core

- Evaluate for intersegmental instability and restriction
- Assess sympathetic chain involvement (potential retinal impact)
- Correlate spinal findings with distal craniofacial symptoms

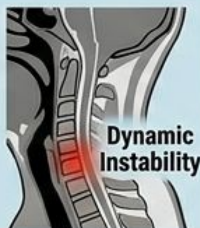
# Slide 16: Interdisciplinary Diagnostics: OCT + MRI + Palpation

## 1. OCT (Retinal Biomarker)



## 2. Upright MRI (Structural Drivers)

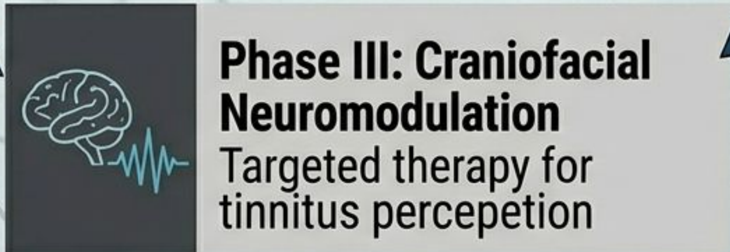


## 3. NMF Palpation (Tissue Quality)

# Unified Diagnostic Profile

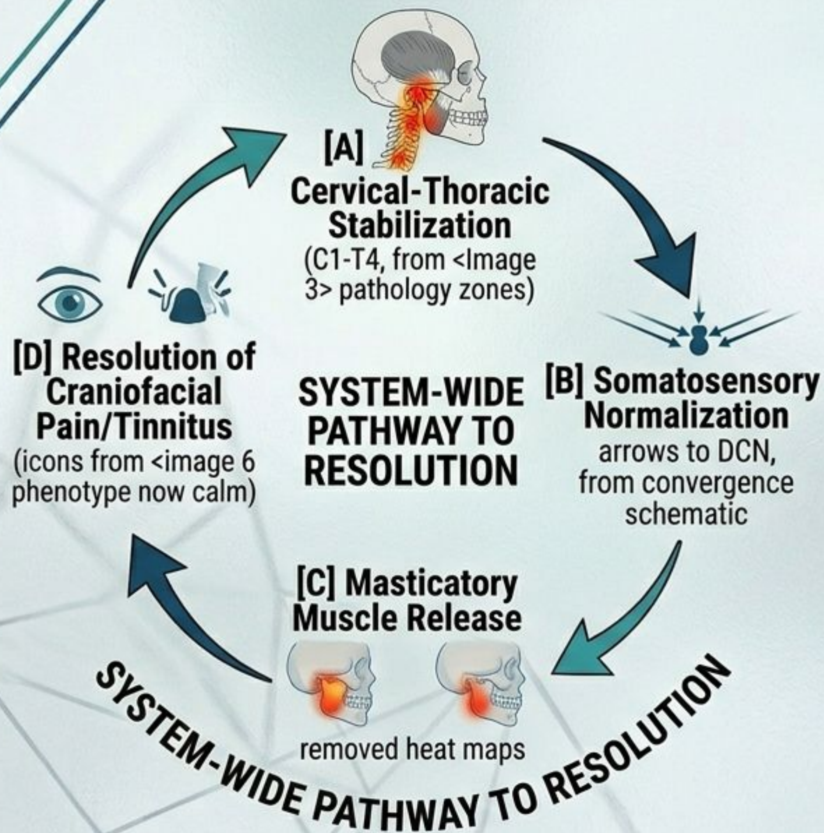
- Integrate objective neurological metrics (OCT)
- Correlate with structural spinal drivers (Dynamic MRI)
- Validate with localized neuromyofacial findings (Palpation)

# Slide 17: Multi-Modal Treatment Approach




## Sequential System-Wide Therapy

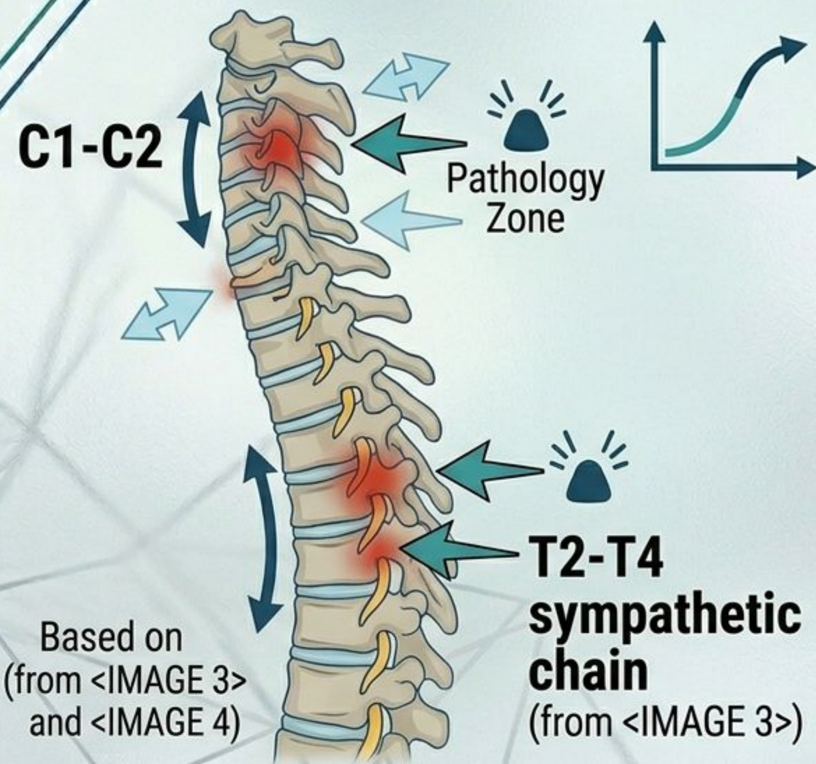
- Address primary spinal structural drivers first
- Release restricted restricted neuromyofascial tissues
- Target persistent central nervous symptoms (DCN hyperactivity)




# Treating the Structural Drivers

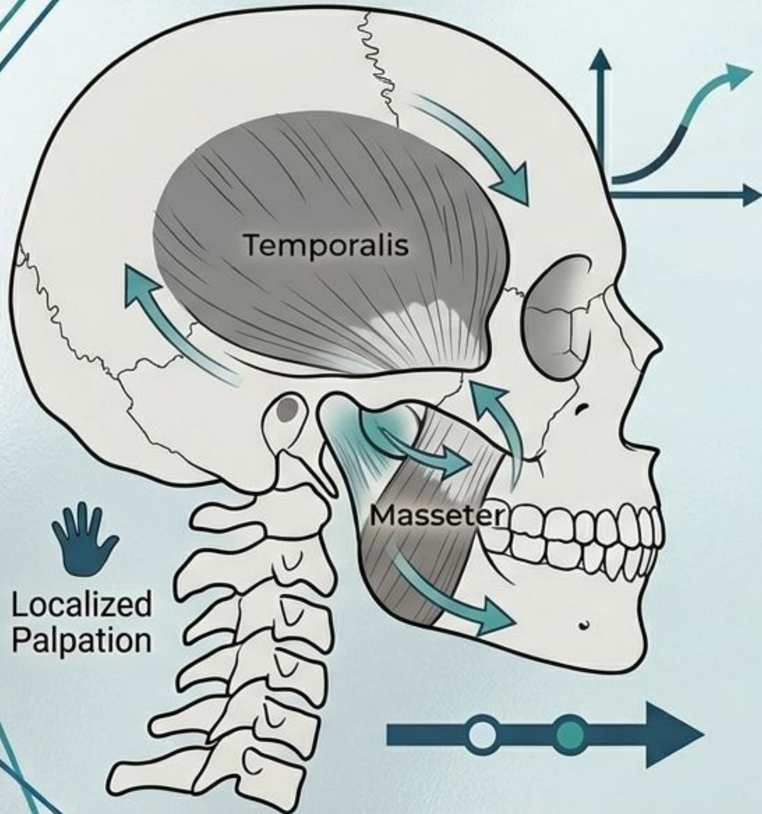
- Addressing the structural core resolves distal complex symptoms
- Sequential approach is required
- Targeting tissues without addressing underlying pathology is **ineffective**

# Slide 19: Phase I: Cervical-Thoracic Stabilization




## Phase I Goal: Structural Core Integrity

- Address primary spinal structural drivers first (from whiplash drivers)
- Evaluate for intersegmental instability and joint (from <IMAGE 3)
- Reduce sympathetic chain involvement and neuroaxonal stress




# Phase II Goal: Decompress & Release Restricted Tissue

- Target localized masticatory muscle spasticity and “lumpy” bands (from <IMAGE 3> heat maps)
- Integrate deep palpation to release distal entrapment points
- Correct aberrant somatosensory input to the Dorsal Cochlear Nucleus (DCN hyperactivity)

# Slide 21: Phase III: Craniofacial Neuromodulation

Acoustic Neuromodulation

Original aberrant somatosensory signals (from <Image 3>, <IMAGE 2) now calm

Dorsal Cochlear Nucleus (DCN)

## Phase III Goal: Consolidate Central Normalization

- Address persistent Dorsal Cochlear Nucleus (DCN hyperactivity) following structural normalization
- Utilize non-invasive neuromodulation (e.g., targeted acoustic therapy)
- Stabilize the normalized central auditory/somatosensory pathways

# Slide 22: Clinical Protocol: Phase I-III Summary

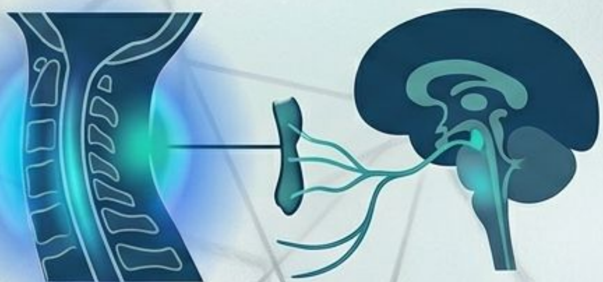
**Phase I: Spinal Stabilization**
(C1-T4
from <IMAGE 3>
and <IMAGE 4)

**Phase II: NMF Release**
(Craniofacial/Neck,
from <IMAGE 5>
and < IMAGE 6)

**Phase III: Neuromodulation**
(Acoustic/DCN
from <IMAGE 7>
and < IMAGE 2)

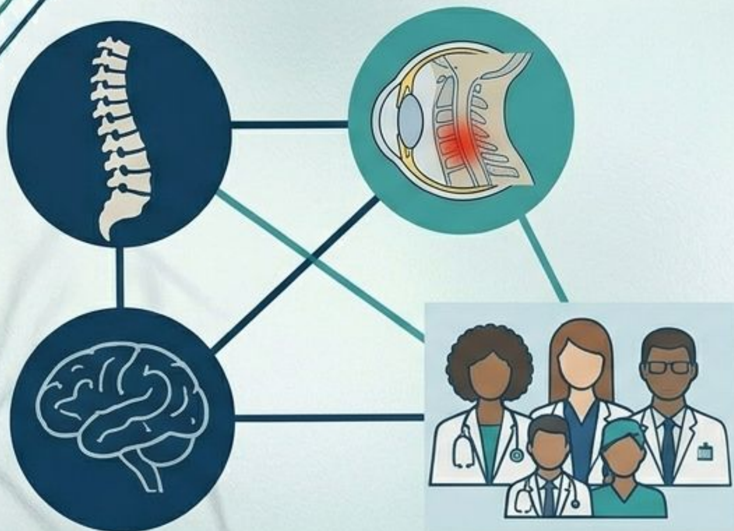
**Outcome: Resolved Phenotype**
(from <IMAGE 8>
and <IMAGE 9>
now calm)

**SYSTEM-WIDE PATHWAY TO RESOLUTION**



- Address the **structural core** (the driver) first
- Release **restricted neuromyofascial tissues** (the manifestation)
- Target **persistent central hyperactivity** (DCN)

# Slide 23: Call to Action: Interdisciplinary Collaboration



## Unified Path to Resolution

- Complexity requires interdisciplinary approach approach approach
- Integrate subjective patient symptoms (Tinnitus, TMJ) with objective metrics (OCT, MRI)
- Contact NMF Science LLC for referral and collaboration information

Unified Path to Resolution

# Slide 24: Key References & Supporting Literature

## SCIENTIFIC FOUNDATION

- **Lee et al. (2016):** LEVEL I: Meta-Analysis
  - Study: "Association between TMD and tinnitus."
  - Quantifies 2.62x increased tinnitus risk in TMD patients. Validates the TMJ-Tinnitus correlation.
- **Michiels et al. (2018):** LEVEL II: Systematic Review
  - "Cervical physical therapy for somatosensory tinnitus."
  - Finding: Multi-modal physical therapy significantly improves somatosensory tinnitus symptoms.
- **Kreuzer et al. (2014):** LEVEL III: Controlled Trial
  - "Trauma-associated tinnitus and clinical distress."
  - Finding: Post-whiplash and trauma-related tinnitus yield higher clinical distress scores.
- **Kaya et al. (2021):** Level IV: Emerging Evidence
  - "Ocular findings in patients with chronic symptoms."
  - Finding: Documents pRNFL thinning in complex craniofacial pain phenotypes.
- Data supports the system-wide Neuromyofascial cascade model.