Table 2. Physiologic Drivers of Network Instability in Migraine and Tinnitus.

Physiologic Driver	Neural Network-Level Effect	Implication for Tinnitus Stability
Sleep disruption	Reduces inhibitory control and increases cortical excitability.	Heightens vulnerability to perceptual fluctuation and tinnitus exacerbation.
Hormonal fluctuation	Alters thalamocortical regulation and neurotransmitter balance.	Promotes oscillatory instability, leading to increased tinnitus perception.
Systemic inflammation	Enhances neuronal responsiveness through neuroinflammatory processes.	Amplifies internally generated auditory activity, contributing to tinnitus persistence.
Sensory overload (e.g., loud sounds)	Exceeds adaptive filtering capacity, leading to sustained salience (attention) network activation.	Prevents perceptual down-regulation, maintaining tinnitus salience.
Metabolic stress (Physical or psychological)	Disrupts energy-dependent inhibitory processes, impairing homeostatic regulation.	Favors recurrent signal amplification, exacerbating tinnitus symptoms.
Excitation–inhibition imbalance	Biases toward excitatory signaling, destabilizing auditory network regulation.	Reinforces dysrhythmic auditory processing, contributing to tinnitus instability (fluctuation).
Impaired habituation	Persistent response to repetitive input due to failure in adaptive filtering mechanisms.	Prevents perceptual down-regulation, maintaining tinnitus salience.
Reduced inhibitory filtering	Weakens thalamic filtering mechanisms, allowing greater cortical access for internally generated signals.	Limits adaptive suppression of the tinnitus percept, leading to increased awareness and distress.