Abstract.

Age-related cognitive decline is associated with measurable changes in macroscopic electrophysiological markers: contraction of intrinsic neural timescales (INT), slowing of the dominant alpha frequency, and reduction of high-frequency power. These shifts are thought to reflect structural degradation, particularly demyelination-induced increases in axonal conduction delays and synaptic loss, but the dynamical mechanisms linking structural changes to these metrics remain poorly understood.

We address this using a computational model of two bidirectionally coupled brain regions, each comprising 800 excitatory and 200 inhibitory spiking Izhikevich neurons with heterogeneous parameters ensuring diverse single-unit dynamics. Internal delays are negligible and the inter-regional axonal delay and coupling strength are systematically varied across the physiologically realistic range for human cortico-cortical connections.

The model reveals that axonal delay shapes the inter-regional synchronization regime, including the phase relationship between populations and the stability of their locking, which in turn modulates coupling effects on INT and dominant frequency. At short delays where populations synchronize in-phase (~7–13ms), coupling promotes INT expansion and frequency decrease. As delays shift the system toward anti-phase synchronization regimes, consistent with age-related demyelination, and coupling decreases due to synaptic loss, the model produces changes qualitatively consistent with the experimental signatures of aging.

By systematically mapping how delay and coupling jointly modulate each metric, the model provides a dynamic landscape where aging trajectories can be traced and metric changes predicted from the underlying synchronization regime, positioning phase dynamics as a mechanistic bridge between white matter integrity and macroscopic temporal markers.