

CAUSALTOMICS: Causal connectomics subtending oscillatory spread and information flow in the human brain

Main area: HBP01_Human brain intracranial data and their relationship to other aspects of brain organisation

Keywords: brain oscillations, brain synchrony, intracranial encephalography, intracranial direct brain stimulation, oscillatory entrainment, brain connectomics, brain networks, functional connectivity, structural connectivity tensor diffusion imaging, resting stage connectivity, epilepsy

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Abstract

Brain oscillatory activity and interregional synchrony have been shown to subtend network communication between neural assemblies characterizing different aspects of human cognition. These set of coding phenomena allow an efficient activation of extended neural networks and a transfer of information essential for the complex processes subtending high-level cognition. Oscillatory and synchrony phenomena have been extensively explored in animals and humans using correlational approaches. Nonetheless, causal evidence on the physiological features, anatomical constraints and cognitive role played by oscillations and synchrony in the human brain is still lacking. We postulate that the direct manipulation of neural oscillations with rhythmic patterns of brain intracranial stimulation coupled to intracranial electroencephalographic (iEEG) recordings in implanted epilepsy patients has the potential to causally characterize functional and structural interactions, hence extend our understanding on how brain networks operate. Moreover the causal characterization of reliable maps of natural frequencies, oscillatory entrainment and spread and information flow across brain regions has the potential to open novel therapeutic strategies for patients suffering brain diseases subtended by synchrony impairments. In this project we aim to tackle the challenge to model how structural white matter connectivity shapes and constraints information flow induced through rhythmic patterns of activity tested causally by means of electrical stimulation in implanted human epilepsy patients. We will address the following 2 aims: In AIM 1, we will use iEEG recordings to characterize the causal impact of intracranial rhythmic stimulation patterns on ongoing local brain activity and in interconnected brain areas by means of high-resolution intracranial EEG recordings. In AIM 2, we will combine iEEG recordings and diffusion tensor imaging data from each individual patient and using structural connectomics, effective connectivity measures and dynamical network modeling, we will model how the spread of information across brain areas is constrained by white matter connections. Three teams will contribute anonymized patient iEEG data modulated by intracranial stimulation and signal analysis tools for studying the occurrence and distribution of local and network spread of rhythmic activity patterns. Feasibility is supported by recent publications in the field by consortium members (Amengual et al. Sci Reports 2017, Tauste Campo et al. PNAS 2015, Donos et al. Neuroimage 2016). We aim to build a causal connectivity atlas (causal connectome) of the human brain that unambiguously characterizes the electrophysiological features, structural constraints and connection directionality. This compendium will be made available to the scientific and clinical community. It will be used to accelerate the development of large-scale models of human brain connectivity and cognition and as a framework for future modeling brain diseases and in particular oscillopathies such as epilepsy.

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