Impairments in working memory (WM) are a core cognitive deficit in schizophrenia. Recent models have emphasized the potential role of neural synchrony as a pathophysiological mechanism underlying impaired WM performance in the disorder, suggesting that impaired WM may be related to deficits in coordinating the precise timing of activity across neuronal populations.

Traditionally researchers have explained patients’ impairments with reference to the prefrontal cortex and its role in memory maintenance and retrieval. However, recent studies (including my own) have increasingly emphasized the importance of stimulus encoding and early sensory processing. In the first part of the talk, I will summarize evidence from my EEG (ERPs and synchronised oscillatory activity) and fMRI studies establishing the neurophysiological basis of WM encoding deficits in patients with schizophrenia.

In the second part of my talk, I will present two very recent studies. First, I will present work aiming to further identify the contribution of early visual activity to WM in healthy participants. Specifically, I will present a study investigating the contributions of the magno- and parvocellular visual pathways to successful WM performance. Finally, I will present a study, examining the neurochemical basis of oscillatory activity using concurrent EEG and MR-spectroscopy measures of glutamate concentration.

In summary, I emphasized the importance of understanding early perceptual and encoding processes for both successful and impaired WM. Furthermore, these findings show that the combination of different neuroimaging techniques provide a window into the underlying neurochemical substrates of neuronal network activity, which has important implications for understanding the basis of WM deficits in schizophrenia.