Symposium | Schizophrenia：[Symposium 28] Neurophysiological studies from a perspective of predictive coding in psychiatry

Fri. Sep 26, 2025 9:00 AM - 10:30 AM JST | Fri. Sep 26, 2025 12:00 AM - 1:30 AM UTC Session Room 6 (Conference Room B)

# [Symposium 28] Neurophysiological studies from a perspective of predictive coding in psychiatry

Moderator: Kenji Kirihara (Center for Coproduction of Inclusion, Diversity and Equity, The University of Tokyo)

[SY-28]

## Neurophysiological studies from a perspective of predictive coding in psychiatry

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[SY-28-01]

## Revealing predictive coding impairments in schizophrenia through mismatch negativity: from neurophysiology to clinical implications

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[SY-28-02]

## Predictive coding studies using mismatch negativity in schizophrenia

*Daisuke Koshiyama[1], Reiji Shioda[1], Taiki Kishigami[1], Kenji Kirihara[1], Kiyoto Kasai[1] (1. The University of Tokyo (Japan))

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[SY-28-03]

## Hierarchical Predictive Coding in Autism Spectrum Disorder

*Zenas C Chao[1] (1. The University of Tokyo (Japan))

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[SY-28-04]

## Propagation of prediction signals in the front-temporal network during tone omission

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### [SY-28] Neurophysiological studies from a perspective of predictive coding in psychiatry

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Keywords：predictive coding、electroencephalography、electrocorticography、mismatch negativity

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Recently, a predictive coding theory is thought to provide a useful framework for understanding various symptoms of psychiatric disorders. In the predictive coding theory, the brain generates a model of the world and updates the model by calculating the differences between prediction based on the model and sensory inputs from the world. Alterations in these processes generate a maladaptive model and make a false inference that leads to various symptoms in psychiatric disorders.
In this symposium, 4 speakers will show recent neurophysiological studies that investigate neurobiological mechanisms underlying altered predictive coding in psychiatric disorders. These speakers will show neurophysiological studies of predictive coding using electrocorticography (ECoG) of non-human primates and electroencephalography (EEG) of humans with and without psychiatric disorders. EEG studies of patients with psychiatric disorders are important for clarifying how predictive coding is altered in psychiatric disorders. EEG studies of healthy humans are important for clarifying how predictive coding is presented in human brains. ECoG studies of non-human primates are important for clarifying neurobiological mechanisms underlying predictive coding.
Combining ECoG studies of non-human primates and EEG studies of humans with and without psychiatric disorders will lead to translational studies that are useful for understanding psychiatric disorders and developing better treatment. This symposium will provide the opportunity to show Asian network of neurophysiological studies based on the concept of predictive coding in psychiatry.

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## [Symposium 28] Neurophysiological studies from a perspective of predictive coding in psychiatry

Moderator: Kenji Kirihara (Center for Coproduction of Inclusion, Diversity and Equity, The University of Tokyo)

### [SY-28-01] Revealing predictive coding impairments in schizophrenia through mismatch negativity: from neurophysiology to clinical implications

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Keywords：mismatch negativity、schizophrenia and early psychosis、clinical implication

Mismatch negativity (MMN) is an event-related potential component that reflects the brain's automatic detection of unexpected changes in auditory stimuli. It is widely recognized as a neural index of the predictive coding system, which continuously generates and updates internal models to anticipate sensory input. Within this framework, MMN is thought to be elicited by the detection of a mismatch between predicted and actual auditory input—signaling a prediction error. Consequently, impaired MMN in individuals with schizophrenia has been interpreted as evidence of disrupted predictive coding, contributing to their difficulties in interpreting and adapting to sensory environments. Such MMN impairments have been consistently observed not only in individuals with chronic schizophrenia but also in those at earlier stages of illness, including patients with first-episode psychosis (FEP) and individuals at clinical high risk (CHR) for psychosis. These findings suggest that MMN could serve as a transdiagnostic and stage-sensitive biomarker of neurophysiological dysfunction in psychotic disorders. In this talk, we will review recent electrophysiological research employing MMN paradigms to explore predictive coding deficits in schizophrenia and related conditions. Special emphasis will be placed on how MMN alterations are associated with clinical trajectories, functional outcomes, and symptom dimensions. We will also discuss the potential utility of MMN as a biomarker for early detection and prognosis, and its integration into translational research aiming to guide individualized treatment strategies in psychosis.

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## [Symposium 28] Neurophysiological studies from a perspective of predictive coding in psychiatry

Moderator: Kenji Kirihara (Center for Coproduction of Inclusion, Diversity and Equity, The University of Tokyo)

### [SY-28-02] Predictive coding studies using mismatch negativity in schizophrenia

*Daisuke Koshiyama[1], Reiji Shioda[1], Taiki Kishigami[1], Kenji Kirihara[1], Kiyoto Kasai[1] (1. The University of Tokyo (Japan))

Keywords：predictive coding、mismatch negativity、schizophrenia

The predictive coding hypothesis postulates that the brain creates a model based on bottom-up sensory input from the environment, uses that model to predict the next sensory input in a top-down system, and then updates that model by calculating the prediction error between the actual sensory input and the prediction. Recent studies suggest altered predictive coding in patients with schizophrenia. Mismatch negativity (MMN) is thought to be a useful biological indicator that reflect prediction error. Auditory MMN has been repeatedly reported to be reduced in amplitude in patients with schizophrenia and is a biological index of electroencephalography (EEG) reflecting glutamatergic neuronal dysfunction, a leading pathological hypothesis for schizophrenia. We found that MMN amplitude is already reduced before the onset of schizophrenia and is associated with overall levels of social adjustment. We also found that MMN is hierarchically related to social adjustment level via negative symptoms and cognitive dysfunction. In order to investigate whether reduced MMN amplitude reflect altered predictive coding or altered adaptation, we deconstructed MMN into the adaptation component and the deviance detection component. We found that the deviance detection component, but not adaptation component was impaired in patients with schizophrenia. The results indicated that auditory MMN impairment in patients with schizophrenia reflects altered predictive coding in schizophrenia. We also estimated the sources of MMN reduction in patients with schizophrenia using EEG, identified the sources in the frontal and temporal cortices, and provided spatial information of neural networks underlying MMN. These studies bridge animal and clinical studies and greatly contribute to establish MMN as a biological index reflecting predictive coding to understand the pathophysiology and develop novel therapeutics of schizophrenia.

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## [Symposium 28] Neurophysiological studies from a perspective of predictive coding in psychiatry

Moderator: Kenji Kirihara (Center for Coproduction of Inclusion, Diversity and Equity, The University of Tokyo)

### [SY-28-03] Hierarchical Predictive Coding in Autism Spectrum Disorder

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Keywords：Autism spectrum disorder、Predictive coding、Hierarchy、Marmoset model、Human patient

Autism spectrum disorder (ASD) is characterized by atypical sensory experiences, which are often linked to irregularities in predictive coding. Predictive coding theory proposes that the brain constructs hierarchical sensory models through reciprocal interactions of predictions and prediction errors. While irregularities in predictive coding have been proposed to underlie sensory hypersensitivity and cognitive inflexibility in ASD, it remains unclear how these disturbances manifest across different functional hierarchies in the brain. To address this question, we examined a marmoset model of ASD induced by prenatal valproic acid (VPA) treatment. High-density electrocorticography (ECoG) was recorded during an auditory task that engaged two layers of temporal prediction. We then applied a quantitative modeling approach to evaluate the integrity of predictive coding across distinct hierarchies. Our results demonstrate persistent patterns of sensory hypersensitivity and unstable predictions across two cortical hierarchies in VPA-treated animals, accompanied by specific spatio-spectro-temporal neural signatures. Importantly, although imprecise predictions occurred regularly, they manifested in diverse ways, with some neural populations underestimating and others overestimating sensory regularities. This heterogeneity was further reflected in human ASD patients performing a comparable two-level prediction behavioral task. These findings suggest that ASD is not marked by a single deficit in predictive coding, but by diverse and hierarchy-dependent irregularities that may contribute to the wide variability of symptoms observed in patients. For clinicians, this work highlights the possibility of developing multi-level neural biomarkers of predictive coding that could be applied across species. Such biomarkers may help identify subgroups within ASD, link neural irregularities to individual differences in sensory and cognitive symptoms, and eventually guide more targeted interventions.

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### [SY-28-04] Propagation of prediction signals in the front-temporal network during tone omission

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Keywords：prediction、cerebral cortex、macaque monkey

Proactive sensory processing based on prediction is a fundamental ability for humans to respond appropriately to external events. Our brain forms predictions based on sensory context, and produces prediction error signals from comparisons with the actual sensory events, suggesting a mutually active exchange of information between prefrontal areas that generate predictions, and sensory areas that represent sensory information. To elucidate the neural network involved in prediction and prediction error, we investigated functional coupling between prefrontal and temporal cortices from 256 electrocorticogram (ECoG) electrodes implanted in two macaque monkeys using an omission paradigm, which examined responses to omission events embedded in repetitive tone stimuli. Monkeys were presented with tone stimuli under two conditions: one where the timing of omission could be predicted, and one where it could not. We found differences in ERP before omission onset mainly at frontal pole, DLPFC and peri-arcuate area electrodes, whereas differences after omission onset were mainly observed at VLPFC and peri-arcuate area electrodes. θ and α-band phase synchrony between STG and each prefrontal cortices increased for predicted tone omission compared to unpredicted tone omission before omission onset, whereas α and β-band phase synchrony increased after omission onset. Phase directionality analysis suggest that information involved in omission prediction may be propagated between front-temporal cortices, with bottom-up signals conveyed through θ-band and top-down signals through α and β-bands. Considering their time course, θ-band phase synchrony may be involved in generating prediction itself, and β-band in generating prediction error, and α-band in both during tone omission.