# GABAergic transmission and inhibitory postsynaptic potentials: implications for neurological disorders.

**Ricky Wang***

Department of Genome Engineering Centre, University of Guangzhou, Guangzhou, China

## Abstract

**The gamma-amino butyric acid (GABA) system, as one of the major inhibitory neurotransmitter systems in the central nervous system (CNS), plays a critical role in regulating neural activity and maintaining the balance between excitation and inhibition. GABAergic transmission, mediated by GABA receptors, leads to inhibitory postsynaptic potentials (IPSPs) that contribute to shaping neural circuits and network dynamics. Dysfunction of GABAergic transmission and alterations in IPSPs has been implicated in various neurological disorders, providing important insights into the pathophysiology and potential therapeutic targets for these conditions.**

**Keywords**: GABA receptors, Central nervous system, GABAergic transmission, Synchronous neuronal firing, Schizophrenia.

## Introduction

One of the key roles of GABAergic transmission and IPSPs is to regulate neuronal excitability. GABAergic interneurons, which are local inhibitory neurons that release GABA onto nearby neurons, provide feedback inhibition and control the firing patterns of excitatory neurons. IPSPs generated by GABAergic transmission hyperpolarize the postsynaptic neuron, reducing its likelihood of firing an action potential. In neurological disorders such as epilepsy, a condition characterized by excessive and synchronous neuronal firing, dysfunction of GABAergic transmission and alterations in IPSPs can disrupt the delicate balance between excitation and inhibition, leading to increased neuronal excitability and seizure activity [1].

GABAergic transmission and IPSPs also play a crucial role in the development and plasticity of neural circuits. During critical periods of brain development, GABAergic signalling regulates the refinement of synaptic connections and promotes the maturation of neural circuits. Alterations in GABAergic transmission during development have been implicated in neurodevelopmental disorders such as autism spectrum disorder (ASD) and schizophrenia. For example, disruptions in the balance between excitatory and inhibitory synaptic inputs onto pyramidal neurons in the prefrontal cortex, mediated by GABAergic transmission and IPSPs, have been implicated in the pathophysiology of ASD and schizophrenia, leading to altered circuit development and synaptic plasticity. Furthermore, GABAergic transmission and IPSPs have been implicated in the regulation of mood and emotion and their dysfunction has been associated with mood disorders such as anxiety and depression [2]. GABAergic interneurons in regions such as the amygdala and the prefrontal cortex play a crucial role in regulating emotional processing and fear responses. Altered GABAergic transmission and IPSPs in these regions have been implicated in the pathophysiology of anxiety and depression, leading to aberrant emotional processing and mood deregulations [3].

Dysfunction of GABAergic transmission and IPSPs has also been implicated in movement disorders such as Parkinson's disease. GABAergic interneurons in the basal ganglia, a group of nuclei involved in motor control, play a role in regulating the activity of dopaminergic neurons. Alterations in GABAergic transmission in the basal ganglia have been implicated in the pathophysiology of Parkinson's disease, leading to aberrant motor control and motor symptoms [4].

Emerging evidence also suggests that GABAergic transmission and IPSPs may play a role in neurodegenerative diseases such as Alzheimer's disease. GABAergic interneurons in the hippocampus, a region critical for learning and memory, are vulnerable to degeneration in Alzheimer's disease, leading to disrupted GABAergic transmission and alterations in IPSPs. These alterations may contribute to the synaptic dysfunction and cognitive deficits observed in Alzheimer's disease [5].

## Conclusion

Understanding the implications of GABAergic transmission and IPSPs in neurological disorders opens up potential therapeutic avenues. Modulation of GABAergic transmission and IPSPs has been explored as a potential therapeutic strategy for various neurological disorders.

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***Correspondence to:** Ricky Wang, Department of Genome Engineering Centre, University of Guangzhou, Guangzhou, China, E-mail: lisajud@medicine.wisc.edu

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