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GABA Signaling: Therapeutic Targets for Neurodegenerative and Neurodevelopmental Disorders

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Message from the Guest Editors

γ-aminobutyric acid (GABA) inhibits adult neurons and depolarizes and excites immature ones due to a developmental shift of the activity of the co-transporters NKCC1 and KCC2, involved in chloride uptake and extrusion, respectively. Indeed, GABA (and glycine) are unique in their capacity to shift the polarity of their actions depending on $[Cl^-]_i$ levels. Interestingly, in a wide range of brain disorders, there is a reversed reduction and enhanced activity of KCC2 and NKCC1, respectively, leading to depolarizing and often excitatory actions of GABA. These alterations are observed not only in developmental disorders but also in neurodegenerative ones, as well as trauma, infarct, and lesions, suggesting that this is a common reaction of networks to insults. These articles, written by experts in theses domains, highlight the therapeutic potential of restoring GABAergic inhibition and a proper E/I balance in key neuronal circuits of the brain.

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Message from the Editor-in-Chief

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