

Corrigendum: Recovery of neuronal and network excitability after spinal cord injury and implications for spasticity

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A corrigendum on

Recovery of neuronal and network excitability after spinal cord injury and implications for spasticity

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In **Figure 10** of D'Amico et al. (2014) the KCC2 co-transporter was incorrectly drawn as a co-exchanger. This has been

corrected in this version of the figure to show that both chloride and potassium are pumped out of the motoneuron.

Conflict of Interest Statement: The author declares that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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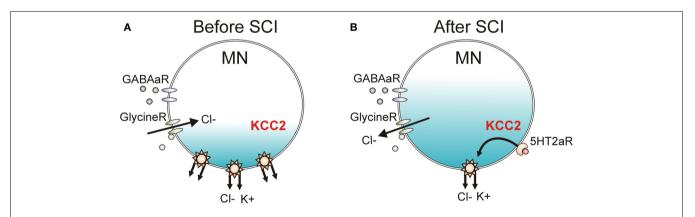


FIGURE 10 | KCC2 cotransporter and chloride equilibrium before and after SCI. (A) A potassium chloride cotransporter (KCC2) transports both chloride (Cl⁻) and potassium (K⁺) out of the motoneuron (MN) to maintain Cl⁻ equilibrium potential below resting membrane potential, allowing Cl⁻ influx and MN hyperpolarization during activation of GABA and Glycine receptors (R). **(B)** Downregulation of KCC2 expression in motoneurons after SCI increases intracellular Cl⁻ concentration, depolarizing Cl⁻ equilibrium potential to above rest. This produces efflux of Cl⁻ and depolarization of MN during activation of GABA and Glycine receptors increases cell membrane expression of KCC2 after SCI to restore endogenous inhibition.