

J. Aguilar. PhD.

Experimental Neurophysiology Group.

Research Unit of the National Hospital for Paraplegics. SESCAM.

Toledo. Spain.

<https://orcid.org/0000-0002-8070-3923>

Characterizing neural activity in the somatosensory cortex to re-define cortical reorganization after SCI.

A cortical reorganization has been described in the long-term after spinal cord injury that consists in a functional invasion of neural activity from the intact cortex to the deafferented cortex. It has been proposed that axonal rewiring of cortico-cortical connection occurs in order to compensate the lack of sensory inputs in the deafferented cortex. However, there is not agreement in the field about what are the neural mechanisms that drive the cortical reorganization after a spinal lesion. Our main objective is to determine the main physiological features in neuronal population of the somatosensory cortex that explain the basis of cortical reorganization after a spinal cord injury. We have studied the cortical activity from the immediate moments after spinal cord injury to the long-term using an animal model of complete thoracic section. For that purpose simultaneous extracellular and intracellular recordings were obtained from intact somatosensory cortex (forelimb coordinates) and deafferented cortex (hindlimb coordinates) in order to study the neural dynamics in each cortical locations. We analyzed the spontaneous activity and the evoked responses to peripheral stimulation at different time windows after spinal lesion. Our results demonstrate that a spinal cord injury immediately triggers a state of slow-wave activity in the somatosensory cortex, which indicates a reduced neural excitability (Aguilar et al J. Neurosci 2010). We found that cortical evoked responses were increased due to a state-dependency, in addition, synaptic inputs magnitude were increased in the intact cortex and deafferented cortex in a state-independent manner (Humanes-Valera et al PLoS One 2013). In the long-term, our results show a temporal heterogeneous neural plasticity that take place in Layer V of somatosensory cortex in animals with chronic spinal cord injury. We found that synaptic properties of pyramidal neurons are different depending on the time from lesion, and that intrinsic excitability of cortical network is increased (Humanes-Valera et al Cereb Cortex 2017). Our most recent results have been obtained using cranial implanted screws for EEG recordings in animals with chronic SCI. We found that evoked responses obtained from EEG extracranial recordings and evoked responses obtained from intracranial (located at Layer V) recordings in chronic animals showed different tendencies related to increased or decreased response magnitudes depending on the recording location. Taking into account that EEG extracranial is composed by simultaneous signals from all six layers of somatosensory cortex, we consider that each different layer in the cortex could suffer a different process of neural plasticity after SCI. Therefore, neural plasticity at cortical level after SCI is a heterogeneous process that depends on local neural network of different cortical layers. In summary our results provide a new perspective of neural dynamics involved in the somatosensory cortex reorganization after a spinal cord injury.