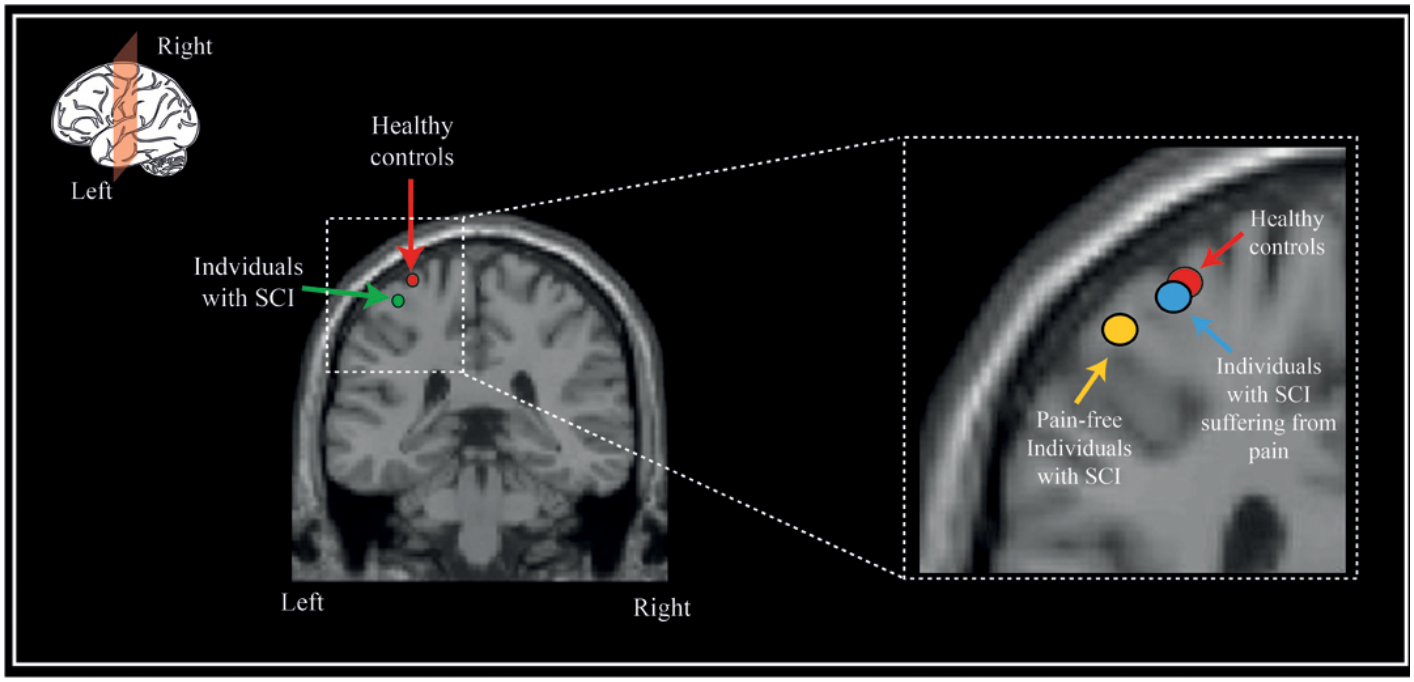
Does neuropathic pain restrict the cortical plasticity after spinal cord injury?

Many studies in animals and humans have shown that the brain reacts with some reorganization (called neuro-adaptive processes) to the amputation of limbs and following a spinal cord injury that leaves the patients with severe motor and sensory deficits. Commonly, neuroimaging methods are used to investigate the reorganization of the brain. As such, functional magnetic resonance imaging (fMRI) was employed in the present study to specifically measure brain activity (i.e., function) by detecting changes associated with blood flow. In fact, the present study yielded cortical reorganization of sensory and motor cortices as a consequence of spinal cord injury. Importantly, alterations in cortical activation were not only related to the sensory deafferentation but also associated with the presence and amount of experienced pain.



As response to spinal cord injury the brain undergoes so-called functional reorganization. In the present fMRI study, healthy controls and individuals with a spinal cord injury performed wrist-extension that activates the primary motor cortex. Importantly, the location of activation during wrist extension is different between healthy controls (red) and individuals with a spinal cord injury (green). Taking a closer look, one can see that the location of activations in individuals with a spinal cord injury suffering from pain (blue) is more similar to the healthy controls compared to the pain-free individuals with SCI (yellow). This might indicate that the functional reorganization constitutes an adaptive process which is hampered by the presence of pain.

Neuropathic pain represents a major secondary complication for people living with spinal cord injury even in those patients that have severe or complete loss of sensory function. Our study provides precious evidence that in distinct brain regions (e.g., primary motor cortex) the reorganization is more emphasized in pain-free individuals with spinal cord injury compared to individuals with a spinal cord injury suffering from neuropathic pain. That is, *preserved* function was accompanied by the presence of neuropathic pain and the magnitude of preservation was correlated with the pain intensity (i.e., the higher the pain, the more preservation of function and structure). Due to the cross-sectional nature of our study the question if pain is casual or consequential to cortical reorganization remains unresolved. In other words, pain might either hamper the cortical reorganization (i.e., pain is the cause) or pain develops because of a lack of cortical reorganization (i.e., pain is the consequence). In this respect, cortical reorganization could be preventing the development of neuropathic pain after SCI – a form of *adaptive* plasticity. As such, reorganization is of protective nature and the failure to reorganize is maladaptive. Interestingly, our findings contrast the widely held belief of the maladaptive plasticity claiming that pain is the trigger for functional reorganization. Briefly, cortical areas encoding for deafferented regions (e.g., legs) shrink and allow for invasion of expanding neighboring areas (e.g., hand). In concert with previous studies, the present study emphasizes a pivotal role of functional adaption of the central nervous system in the initiation and maintenance of neuropathic pain. Nevertheless, the underlying neuronal substrates of cortical reorganization and its role of in the process of pain development remain incompletely understood. Future longitudinal studies are warranted to examine how the development of pain and reorganization of function are allied.

Publication

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