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Sommario/riassunto	Traumatic injury of the spinal cord affects the entire organism directly and indirectly. Primary injury destroys neurons and severs axons which participate in neural circuits. Secondary injuries and pathologies arise from numerous sources including systemic inflammation, consequential damage of cutaneous, muscular, and visceral tissues, and dysregulation of autonomic, endocrine and sensory-motor functions. Evidence is mounting that spinal cord injury (SCI) affects regions of the nervous system spatially remote from the injury site, as well as peripheral tissues, and alters some basic characteristics of primary afferent cell biology and physiology (cell number, size/frequency, electrophysiology, other). The degree of afferent input and processing above the lesion is generally intact, while that in the peri-lesion area is highly variable, though pathologies emerge in both regions, including a variety of pain syndromes. Primary afferent input to spinal regions below the injury and the processing of this information becomes even more important in the face of complete or partial loss of descending input because such spared sensory

processing can lead to both adaptive and pathological outcomes. This issue hosts review and research articles considering mechanisms of plasticity of primary afferent neurons and sensory processing after SCI, and how such plasticity contributes to sparing and/or recovery of functions, as well as exacerbation of existing and/or emergent pathologies. A critical issue for the majority of the SCI community is chronic above-, peri-, and below-level neuropathic pain, much of which may arise, at least in part, from plasticity of afferent fibers and nociceptive circuitry. For example, autonomic dysreflexia is common hypertensive syndrome that often develops after SCI that is highly reliant on maladaptive nociceptive sensory input and processing below the lesion. Moreover, the loss of descending input leaves the reflexive components of bladder/bowel/sexual function uncoordinated and susceptible to a variety of effects through afferent fiber plasticity. Finally, proper afferent feedback is vital for the effectiveness of activity-dependent rehabilitative therapies, but aberrant nociceptive input may interfere with these approaches since they are often unchecked due to loss of descending modulation.