The objective of this proposal is to investigate whether enhanced activity of a selective population of hindlimb motor neurons (MNs), using a targeted gene transfer approach, improves the recovery of motor function following spinal cord transection (SCT). We aim to solve a problem, which accompanies the majority of experimental therapies implemented following SCT, and stems from unspecific and uncontrolled stimulation of the circuitry of preserved neurons below the injury site. Paradigms of activation of the whole network lead to moderate improvement of motor functions, however they do not restore the functional equilibrium between different groups of MNs and muscles. Application of sensory or pharmacological activation to the whole network does not take into account differential demands of functionally different MN groups for stimulation compensating the deficits of innervation and supply of neurotrophic factors. We and others showed that as opposed to inputs to MNs innervating *flexor* muscles operating at the ankle joint, inputs to MNs innervating *extensor* muscles at that joint are severely impoverished after SCT. **These observations led us to hypothesize that the proposed gene constructs targeted to a selective population of MNs with innervation deficits should lead to beneficial changes in local synaptic plasticity, and subsequent recovery of equilibrium in innervation and signaling between MNs controlling the antagonistic muscles acting at the ankle joint.** If so, we expect that the resulting physiological effects and restoration of motor functions will be superior to those achieved with general network activation. The expected results will help to optimize current therapies following SCI by (i) the use of a reduced invasiveness approach for gene therapy and (ii) further applications of validated novel methods of neuron activation. Identification of synaptic and receptor changes evoked by spatially and temporally controlled activation of MNs will increase our understanding of basic mechanisms of regulation of MN excitability in vivo.