Available Spinal Cord Injury Research Projects for TAMU MSRPP Medical Students

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#spinalcordinjury #pain #opioids #depression #osteoporosis

My laboratory, located at the Medical Research and Education Building in Bryan, is focused on recovery of function after spinal cord injury (SCI). Approximately 300,000 people are living with SCI in the United States alone, with 17-18,000 new injuries occurring each year. The consequences of SCI depend on the level of injury, but include paralysis, sensory deficits, loss of bowel and bladder function, loss of sexual function, bone loss, and pain. People living with SCI also have a higher incidence of depression, significantly decreasing their quality of life. While many research labs are focused on recovery of motor function, my laboratory focuses on relatively overlooked consequences of SCI- pain, depression and bone loss. In addition to their immediate clinical relevance, each of these research projects provide diverse opportunities for students to develop their surgical, behavioral, histological, molecular and analytical skills. A synopsis of each area of my lab's research is provided below:

Pain management after SCI. Immediately after a SCI, patients are given opioids to treat the pain associated with damage to the spinal cord, spinal nerves, spinal vertebrae and accompanying peripheral injuries. Unfortunately, however, my lab discovered that *early opioid administration decreases the prognosis for recovery* after SCI, reducing recovery of locomotor function, increasing neuron loss and increasing chronic pain in a rodent SCI model. We have recently extended these studies to the clinical population, and have found that higher amounts of opioids, given in the first 24 hrs after SCI, are also associated with increased chronic pain (at 1 year post injury) in humans. But opioids are indispensable. They are among the most effective analgesics for the management of acute pain. We must identify the molecular changes that lead to opioids adverse effects, and block them while retaining the analgesic efficacy of the medications. This research is supported by the Department of Defense.

Depression and cognitive impairment after SCI. The incidence of depression is 3X greater after SCI, relative to the able-bodied population. This may not be surprising, given the overwhelming life changes induced by injury, but my lab discovered that *30% of rats also develop depression after SCI--* in the absence of psychosocial stressors experienced by the clinical population. This finding suggests that *molecular changes, induced by SCI, potentiate the development of depression after injury*. To date, our research has focused on the role of inflammation in the development of depression, and we are beginning to explore the effects of changes in the gut microbiome. We have also recently extended these studies to look at the effects of SCI on cognitive function. Both depression and SCI have been associated with an increased risk of developing Alzheimer's disease, but this has not been investigated in an animal model. This research is supported by Mission Connect, a project of the TIRR foundation.

Bone loss after SCI. Approximately 80% of individuals living with SCI are diagnosed with osteopenia or osteoporosis. Bone loss begins immediately after injury, continuing for at least the next 2 years. Studies vary, but it is estimated that there is a 30-40% decrease in bone mineral density in the legs following SCI. Because of this *people with SCI are 104 times more likely, than the general population, to have a fracture by the age of 50, with post fracture complications occurring in 54% of patients*. Complications include, but are not limited to, respiratory and urinary tract infections, venous thromboembolic events, fracture non-union or mal-union, and depression. Fractures post injury significantly compromise rehabilitation, as well as increasing morbidity, mortality and healthcare costs. Unfortunately, current treatments for osteopenia and osteoporosis are not effective in the SCI population. Our research aims to derive the etiology of the profound decrements in bone microarchitecture following SCI, and to generate new effective therapeutic strategies to address an unmet need: to mitigate bone loss in both the acute and chronic phases of injury. This research is supported by the Craig H. Neilsen Foundation.