"A Bioengineering Approach to Median Nerve Decompression"

ABSTRACT:

The median nerve is prone to compression neuropathy at the wrist, leading to carpal tunnel syndrome. Carpal tunnel syndrome is currently the most common hand disorder with a prevalence rate greater than 3% in the United States general population. The number of carpal tunnel syndrome cases continues to rise worldwide with the increasing amount of work that demands use of the hands and upper extremities. Symptoms associated with carpal tunnel syndrome include pain, paresthesia, numbness, weakness, and loss of sensory/motor function in the hand. If left untreated, carpal tunnel syndrome can lead to irreversible degeneration of the median nerve, debilitating the hand. Carpal tunnel release surgery is ultimately performed in 25-50% of patients.

The concept of carpal tunnel surgery has existed for a century without fundamental changes, although it is known that the surgical procedure of transecting the transverse carpal ligament disrupts the biomechanical and physiological integrity of the wrist. Alternative, non-surgical treatment options for carpal tunnel syndrome are needed considering the vast number surgical case complications, and recurrence of the syndrome. In our research to understand wrist function and to search for alternative treatments of carpal tunnel syndrome, we discovered novel mechanisms of increasing the cross-sectional area of the carpal tunnel by biomechanically manipulating the carpal tunnel. We have shown that shortening the carpal arch width and/or elongating the transverse carpal ligament are effective in increasing the arch area and reducing carpal tunnel pressure, and therefore potentially decompressing the median nerve and relieving carpal tunnel syndrome symptoms. Another area of our research on carpal tunnel syndrome is to understand the impairment of hand sensorimotor function. At the peripheral level, we investigate the pathokinetematics and pathokinetics of the wrist, hand, and digits, quantifying the subtle or severe deterioration of hand function and dexterity. Although carpal tunnel syndrome has been commonly considered as a peripheral neuropathy, chronic median nerve impairment may cause maladaptation of the central nervous system that exaggerates the deficits in hand function. We investigate the neuroplasticity of the somatosensory and primary motor cortex caused by carpal
tunnel syndrome, and examine the altered cortical representations of the individual digits. We envision that treatment options combining both peripheral and central mechanisms are most effective in restoring the dexterous hand function that is compromised by carpal tunnel syndrome.

**BIO:**
Zong-Ming Li, PhD, is currently the William and Sylvia Rubin Endowed Chair of Orthopedic Research at The University of Arizona, and a Professor of Orthopedic Surgery, Biomedical Engineering, and Bio5 Institute. Dr. Li is also the Vice Chair for Research in Department of Orthopedic Surgery, Director of Robert G. Volz, MD Orthopedic Research Laboratories, Associate Director of University of Arizona Arthritis Center, and Director of the Hand Research Laboratory. Dr. Li has more than 30 years of experience in musculoskeletal research and education with a particular focus on the hand and upper extremity. He has published 121 peer-reviewed articles in 51 journals, given 125 invited lectures worldwide, and presented over 330 podium and poster presentations in professional conferences. Dr. Li has been elected to the College of Fellows of the American Institute for Medical and Biological Engineering (AIMBE) in recognition of his "seminal contributions to hand and wrist biomechanics that impact better outcomes for patients with carpal tunnel syndrome". He has served as Deputy Editor-in-Chief, Associate Editor-in-Chief, Associate Editors and Editorial Board Members for 18 bioengineering and orthopedics related journals.

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**Monday, March 1st, 2021**
12:00-12:50 pm, [https://arizona.zoom.us/j/85468611706](https://arizona.zoom.us/j/85468611706)

**Hosts:** Dr. DK Kang and Dr. Russ Witte
dkkang@arizona.edu and rwitte@arizona.edu

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