INTRODUCTION
Cerebral palsy (CP) is a condition where brain lesions are thought to result in involuntary contraction of skeletal muscles leading to the development of spasticity in these muscles. Spastic skeletal muscles are characterised by increased passive stiffness, shortened fibers and decreased serial sarcomere numbers, leading to a decrease in movement control and a loss of mobility in CP patients [1]. Skeletal muscles are highly adaptable where protein turnover and decreases in sarcomere number occur within 10 hours of active shortening of the muscle [1,2]. In order to artificially mimic the involuntary contractions found in CP patients, electrical stimulation can be used to induce continuous muscle contraction. In previous studies, a ~25% sarcomere loss was observed in New Zealand White Rabbits Triceps Surae muscles following ten hours of electrical stimulation [2]. This serial sarcomere loss is fully recovered within two days, providing an experimental model of sarcomerogenesis. However, the ten hour stimulation protocol is hard on the rabbits, causing weakness and loss of appetite. Therefore, the purpose of this experiment was to quantify sarcomere loss following only five hours of muscle activation. We hypothesized that serial sarcomere loss is the same after five and ten hours of low level electrical stimulation.

METHODS
A nerve stimulating electrode was surgically implanted on the tibial nerve of the experimental leg of New Zealand White rabbits (n=3) while the contralateral tibial nerve was transected in order to prevent any possibility of a cross training effect. Five hours of stimulation at 20 Hz and three times the α motor nerve threshold was applied to the nerve innervating the medial gastrocnemius (MG), plantaris (PLT), and soleus muscles (SOL). The extensor digitorum longus (EDL) muscle served as a non-stimulated control. After stimulation, animals were sacrificed and the hind limbs were placed in a 10% formalin solution with the knee and ankle joint at ~90°. The target muscles were separated into four to six regions. After nitric acid digestion, individual fascicles were isolated from each region and placed on slides for sarcomere length measurement by laser diffraction and fascicle length measurement using a specialized camera and software.

RESULTS
A 11 ± 13 % loss of sarcomeres in the experimental leg was observed in the MG, a 27 ± 1 % in PLT, 44 ± 4 % in SOL and 1 ± 5 % in EDL.

DISCUSSION AND CONCLUSIONS
PLT and SOL lost a similar percentage ( %) of serial sarcomeres in five hours of electrical stimulation as was found in previous works in ten or twelve hours, thereby confirming our hypothesis. However, sarcomere loss was substantially smaller in the MG for reasons that we do not understand at this point. If this result is confirmed once we have a solid number of independent observations, then we can use PLT and SOL, but not MG for studies of sarcomere loss and addition in the rabbit without causing the distress caused by ten hours of anesthesia and electrical muscle stimulation

REFERENCES