**The Muscle Health Research Centre at York University presents the**

**11th Annual Muscle Health Awareness Day (MHAD11)**

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**Last Name: Chen First Name: Chris**

**University: York Supervisor: Dr. David Hood**

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**Abstract Title (**all CAPS**):** PARKIN MEDIATED MITOPHAGY IN SKELETAL MUSCLE WITH ENDURANCE TRAINING

**Authors (Bold): Chris C.W. Chen and David A. Hood**

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**Abstract text (no figures, no Bold):** Skeletal muscle is a highly adaptive tissue that is responsive to environmental cues. With exercise training, intracellular signaling pathways are activated that promote the synthesis of mitochondria, a process termed biogenesis. The increased number of mitochondria allows for efficient substrate utilization and greater muscle endurance. However, little is known regarding mitochondrial turnover with training. Mitophagy is a process involved in the specific elimination of dysfunctional mitochondria. It is unclear if mitophagy undergoes adaptive responses in muscle with training. Parkin is a ubiquitin ligase involved in neuronal mitophagy but its role in muscle remains inconclusive. To investigate Parkin’s role in muscle with exercise training, we subjected 3-month-old wild-type (WT) and Parkin knock out (KO) mice to a 6-week voluntary wheel running training paradigm. Overall, WT mice ran 28% longer than KO mice in average total running distance. Endurance training elicited 1.5- and 1.4-fold increases in whole muscle mitochondrial content in WT and KO mice, respectively. Furthermore, subunits I and IV of cytochrome c oxidase and mitochondrial transcription factor A were augmented on intermyofibrillar mitochondria (IMF) isolated from hindlimb muscles of trained WT animals. This was supported by a 1.7-fold increase in State 3 (active) IMF respiration in trained WT mice. Following 6 weeks of training, both Parkin KO and WT mice exhibited 2-fold increases in running performance during an exhaustive bout of exercise, compared to their untrained counterparts. Interestingly, basal mitophagy flux and mitochondrial localization of LC3II were augmented by 1.5-fold in muscle of trained WT mice. However, this increase was abolished in KO animals. However, during exercise-induced mitophagy, an attenuation of autophagy proteins localized on mitochondria occurs, which is partly Parkin-mediated. With training, exercise-induced mitophagic signaling is reduced, likely due to enhanced mitochondrial biogenesis, leading to improved endurance. Supported by NSERC.