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MITOCHONDRIAL DNA VARIATION IMPACTS CELLULAR AND PHYSICAL FUNCTION IN OLDER ADULTS

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Acquired mitochondrial DNA (mtDNA) mutations accumulate with age at a higher rate than nuclear DNA and can lead to mitochondrial dysfunction. The condition when mitochondria have a mixture of both normal and mutated mtDNA is called "heteroplasmy." Because mtDNA exists in up to ~1,000 copies per cell, each cell may contain a mixture of normal and mutated mtDNA. When a threshold of mutant mtDNA is reached, mitochondrial dysfunction and pathogenic conditions can occur. Accumulation of somatic mutations in mtDNA occurs in multiple organs and tissues with increasing age. Acquired mtDNA variation explains variability in physiological function and growing body of evidence supports a role of accumulated mtDNA mutations in age-related disorders. Our previous work has shown that mtDNA heteroplasmy was associated with reduced walking speed, grip strength, cognitive function, and increased mortality in older adults (70–79 years) (1,2). In this presentation we will show new muscle mtDNA heteroplasmy data collected from >700 men and women age 70-89 years from the Study of Muscle, Mobility and Aging (SOMMA) cohort (3). We will highlight the role of muscle mtDNA heteroplasmy in muscle mitochondrial oxidative capacity, muscle mass, strength, VO2peak, and walking speed in older adults.

Keywords: *heteroplasmy, mitochondria, mtDNA, muscle, human cohort.*