activPAL accelerometer for 7 days. Percentage sedentary, standing and stepping time of waking time were calculated. Participants had on average 6.4 valid days (≥14h of monitoring) and 90% wore the device >4 days. Men spent significantly more time sedentary than women (63.3±10.3% versus 57.0±10.1%); standing and stepping time were significantly higher in women (30.1±7.9%; 12.9±4.4%) than in men (24.7±7.4%; 12.1±4.6%). Sedentary time significantly increased with increasing age while standing time decreased; no clear age-gradient in stepping time was found.

MULTI-SENSOR MEASUREMENT OF PHYSICAL BEHAVIOR IN THE FINNISH RETIREMENT AND AGING STUDY

Sari Stenholm, *University of Turku, Turku, Finland*

Retirement is a life transition that is accompanied by changes in time use and removal of work-related exposures. The Finnish Retirement and Aging study (FIREA) was established in 2013 to examine changes in 24-hour physical behavior, lifestyle factors, health and functioning by following aging workers annually from full-time work to retirement. FIREA activity substudy includes 1200 participants (mean age 63.2 years, 85% women) and their physical behavior has been measured annually with multiple accelerometers (wrist ActiGraph, thigh Axivity and hip SenseDoc including GPS). The mean number of measurement days is seven at each measurement wave with a valid wake wear time 16.0 hours before retirement and 15.6 hours after retirement. Currently, information is available from 3-5 measurement waves. Transition to retirement induced changes in 24-h physical behavior towards increased sleep and less physical activity among women, especially in those retiring from manual occupations.

SESSION 7095 (SYMPOSIUM)

NUTRITIONAL MEDIATORS OF CELLULAR DECLINE AND MITOCHONDRIAL DYSFUNCTION IN OLDER ADULTS

Chair: Roger Fielding

Aging is the primary risk factor for progressive loss of function, onset of disease, and increased vulnerability to negative health-related outcomes. These clinical manifestations arise from a decline in mitochondrial and metabolic processes considered the hallmarks of aging. Collectively, these changes can be defined as age associated cellular decline (AACD) and are often associated with signs and symptoms such as fatigue, reduced strength and low physical activity. This symposium will explore mechanisms, clinical signs, and emerging nutritional interventions for AACD. Dr. Feige's presentation will serve as an introduction by highlighting mechanisms underlying functional declines in skeletal muscle with aging. He will discuss the Multi-Ethnic Molecular determinants of Sarcopenia (MEMOSA) study, which found impaired mitochondrial bioenergetic capacity in skeletal muscle of older adults with sarcopenia compared to age-matched controls, and identified mitochondrial function as a key target for intervention. Dr. Guralnik will discuss the connection between cellular changes and clinical manifestations of AACD. He will report on an expert consensus study group which developed an initial framework to identify self-reported symptoms and observable signs of AACD in adults over50 years. Lastly, Dr. Singh will discuss the evidence for nutritional interventions to address sources of AACD, focusing on those targeting mitochondrial dysfunction. Recent research on dietary interventions with urolithin A (an activator of mitophagy) and nicotinamide riboside (an NAD+ booster) will be reviewed. Overall, this symposium will highlight key mechanisms and clinical signs of AACD, and the potential for novel nutrition interventions to support cellular function and healthy aging.

EARLY DETECTION OF AGE-ASSOCIATED CELLULAR DECLINE: REPORT OF AN EXPERT CONSENSUS

Jack Guralnik,[1] Matteo Cesari,[2] Ariel Beresniak,[3] Leocadio Rodriguez-Manas,[4] and Antonio Cherubini,[5]
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Cellular processes often decline with age and cells lose their ability to function optimally, which may lead to organ-specific dysfunction and the development of systemic age-related diseases. The cellular hallmarks of aging are associated with clinical signs and symptoms and can be termed Age Associated Cellular Decline. An expert consensus study group was convened to provide an initial framework for the development of a tool for adults over 50 years old, which identifies self-reported symptoms and observable signs likely to be early and/or surrogate markers of age associated cellular decline. A total of 16 potential early signs and symptoms of age associated cellular decline were identified and need to be validated in further research.

NUTRITIONAL STRATEGIES TO COUNTERACT MITOCHONDRIAL DYSFUNCTION AND NAD+ DEFICIENCY IN HUMAN SARCOPENIA

Jerome Feige, *Nestlé Research, Lausanne, Switzerland*

The causes of impaired skeletal muscle mass and strength during aging are well-studied in healthy populations. Less is known on pathological age-related muscle wasting and weakness termed sarcopenia, which directly impacts physical autonomy and survival. We compared genome-wide transcriptional changes of sarcopenia versus age-matched controls in muscle biopsies from 119 older men of different ethnicity. Individuals with sarcopenia demonstrate a prominent transcriptional signature of mitochondrial bioenergetic dysfunction in skeletal muscle, with low PGC-1α/ERRα signalling, and downregulation of oxidative phosphorylation and mitochondrial proteostasis genes. These changes translate functionally into fewer mitochondria, reduced bioenergetic activity, and NAD+ deficiency in sarcopenic muscle. Our results point to mitochondrial homeostasis as a key mediator of pathological muscle aging. Novel nutritional solutions enhancing muscle strength and performance by enhancing mitochondrial function are being tested clinically and will be reviewed. These include activating mitophagy with Urolithin A or restoring NAD+ levels via tryptophane/kynurenine or with nicotinamide riboside.