



Exercise for Cardiovascular Integrity and Plasticity During Ageing

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Editorial

Cardiovascular dysfunction together with various types of heart problems is associated with the ageing process since both impaired cardiac function and the affected individual's quality-of-life with the eventual entrapment of the life-expectancy of elderly ageing citizens. Systematic literature reviews and meta-analyses have revealed that the potential of socio-cultural determinants of exercise and diets for health manifestations are identifiable [1]. The impact of optimal exercise upon a mouse model of non-ischemic dilated cardiomyopathy was evidenced by an improved outcome in non-ischemic dilated heart failure [2]. In a rat model of ageing, low-intensity aerobic exercise along with limb blood-flow restriction may ameliorate ageing-induced muscle atrophy and nicotinic acetylcholine receptors at the neuromuscular junction [3]. It was observed that the restriction of blood-flow concomitant with a mild exercise regime produced potentially ameliorative effects in the protection and augmentation of muscle mass and nicotinic acetylcholine receptors clustering at the neuromuscular junction among older rats. Among older women, strength exercise training schedules were beneficial, both for a sustained lifespan and against mortality, if maintained at moderate levels of exercise duration, independent of the type or level of aerobic exercise. Among middle-aged or older individuals who presented lower aortic stiffness, without hypertension, habitual bouts of aerobic exercise offered straightforward benefits, as estimated through use of carotid-femoral pulse wave velocity [4-6]. Nevertheless, the extent of aortic stiffness was observed to be resilient against the influence of clinically relevant augmentation of responses to habitual aerobic exercise when placed in combination with the hypertension displayed by middle-aged and older adults [7], thereby promoting a form of physiological resilience. In this regard, in observations of older men both the participation in strenuous exercise and the gradual increments of walking-speed that these subjects performed were associated with reductions in all-cause mortality and also in specific mortality, such as cardiovascular disease and heart problems [8]. In a cohort of race-ethnically diversified older female participants (mean age=78.9 years), after adjustment was made for age, ear age, time, race-ethnicity, and other potential confounding variables, physical exercise/activity parameters, as assessed through accelerometry were associated positively with mean levels of high-density lipoproteins, triglycerides, glucose, C-reactive protein, body mass index, waist girth, and the Reynolds Risk Score [9]. The exercise induced enhancement of the transcription factor, nuclear factor- κ B-related factor-2, that regulates several anti-inflammatory genes to counteract oxidative damage in several organs, including heart and brain has been observed also [10].

The age-dependent decline in the function of cardiovascular tissues initiates eventually the retardations in cerebral blood-flow regulation and maintenance, which in turn, leads to a sequential cascade that culminates in the impairment of neuronal micro-environmental homeostasis. Concurrently, the tremendous high level of metabolic activity of the brain and CNS combined with limited capabilities intracellular energy storage places critical demands upon the logistics of cerebral blood-flow to maintain adequate functioning neuronal metabolism. In both Alzheimer's disease and normal-aged elderly ageing, cerebral hyperperfusion, increased cerebral blood-flow plasticity and

dysregulation of blood pressure control during orthostatic have been found to be harbingers of exaggerated, age-related deterioration in both cerebral and cardiovascular function with the highest levels of all-cause mortality. Hazard risk occurring among those individuals presenting limitations of both physical mobility and cognition as opposed to mobility only, cognition only, or no limitation. Although there remains available a great variety of exercise programs, e.g. in the case of cardiovascular limitations, remarkably analogous benefits and parameters may be expressed, independent of program details, such as the high-intensity interval aerobic training that was given to a group of elderly Japanese men (aged 60-69 years) produced equivalent levels of feasibility, exercise tolerance and perceived exertion as a traditional moderate-intensity continuous aerobic training. In a population of elderly Turkish citizens (n=2976) displaying coronary heart disease, it was observed, as a matter of concern, that over 75% were sedentary and 63% were either overweight or obese. It has been shown that brachial shear rate patterns, that affect the endothelium as well as the development and progression of atherosclerosis, and brachial artery intima-media thickness bear relationships to advancing age. Among one hundred and twenty middle-aged and older individuals, who were separated into exercise and control groups, who were submitted to a 12-week interval of aerobic exercise, that the exercise schedule augmented the antegrade shear rate and decreased the retrograde shear rate and brachial artery intima-media thickness. At the same time, the alterations observed in the and low-density lipoprotein cholesterol, concurrent with an increase in aerobic capacity, which changes among the control subjects. This pattern of results entails that heart-rate targeted aerobic exercise ameliorates aortic stiffness and improvement of metabolic and fitness parameters. Finally, in elderly male patients presenting essential hypertension, both concurrent exercise, these are programs that attempt to concomitantly develop resistance-to-fatigue (through endurance-based exercise) and increased muscle mass (through resistance-based exercise), and aerobic exercise induced post-exercise hypertension although this effect was

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longer lasting for the aerobic exercise.

There is an ever-increasing necessity to derive lifestyle strategies and related neurobiological mechanisms/tactics for the reduction of ageing-related motor, mood-decay and cognitive impairments as well as for the advancement of plasticity among the aged. Thus, it has been observed that aerobic exercise could reduce age-related decline in cognition and brain functioning. A lifelong adherence to the minimum recommended physical exercise bears a strong relationship with markers of cognitive function and biomarkers of performance as well as neuronal integrity as the ageing process continues [11]. In the laboratory animal model of Brown-Norway/Fischer 344/F1 hybrid rats, expressions of locomotor activity start to deteriorate already during middle age (age level: 12-18 months), accompanied by reduced expression of the glial-derived neurotrophic factor family receptor, GFR-1, which is reduced in substantia nigra [12]. Moderate levels of exercise that were initiated at 18 months of age, implemented the nigral GFR-1 and tyrosine hydroxylase expression no later than 2 months afterwards. Among the aged rats, the replenishment of ageing-related loss of GFR-1 in the substantia nigra enhanced tyrosine hydroxylase levels in the substantia nigra alone as well as locomotor activity. Moderate levels of exercise regimens were then initiated among a sedentary group of male Brown-Norway/Fischer 344/F1 hybrid rats as a longitudinal effort to evaluate whether or not exercise could ameliorate the ageing-related motor deterioration which initiated at two different age-levels during the later stages of the lifespan (i.e., 18 or 24 months of age). It was observed that the motor deterioration was reversed among the 18-month, but not 24-month-old rats.

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