

Cognitive Health and Brain Plasticity due to Physical Exercise during Ageing

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Introduction

Several aspects concerning the loss of vascular and metabolic integrity that are associated with the ageing process cause wide-ranging impairments in regional, biomarker and functional domains. For example, the progressive white matter injury arising from a prevailing vascular pathophysiology incorporates both normal ageing and neurodegenerative features of dementia, as well as microvascular brain injury, which originates from a condition of chronic recurrent white matter ischemia that induces oxidative stress and an elevation in the number of total oligodendrocyte lineage cells. Subsequently, perturbances in vascular-induced cognitive deficit and dementia are expressed in the selectively-impaired vasodilator functions of white matter-penetrating arterioles in microvascular brain injury. These perturbances occurs in association with aberrant differentiation of oligodendrocyte progenitor cells as the result of white matter injury; these notions imply simply that myelination maturational processes are associated with perturbations in the maturation of myelinating oligodendrocyte cells [1]. Clinically-relevant improvements in insulin resistance/glucose homeostasis within different populations of ageing adults were observed to be related to the augmentation of cognitive functioning resulting from programs of exercise/activity [2]. Amongst a group of older, elderly adults (mean age=72.6 years), expressions of adherence to compliance for exercise were in favour of social dancing exercise, especially regarding its attractiveness for promoting cognitive and social functioning and several indicators of health and well-being [3]. The presence of depression-related problems among the aged has been well-established with cognitive deficits among the more serious health burdens: in this regard, exercise training, bestowing pro-resilience and anti-depressant effects, produces beneficial effects on stress-related mental disorders, indicative of clinical potential. For example, acute physical exercise produces marked increments in the individual alpha peak frequency of subjects offering a cortical parameter associated with neural information processing speed [4]. Further, the effects of exercise upon mood states remains an invariably favourable process, independent of the period of exercise duration or the recovery-from-exercise period [5]. Taken together the consensus of burgeoning evidence is that physical exercise may improve, but most certainly delay, the negative influences of ageing and Alzheimer's disease [6].

Physical exercise compliance is promoted increasingly as the major onslaught against the implacableness of age-related declines in physical and cognitive health among older and elder older adults, despite a certain sparsity for reliable indications for the relative efficacies of aerobic, endurance as opposed to resistance, heavy regimes. As a barometer condition for cognitive health, it has been demonstrated repeatedly that the exercise process induces numerous positive effects upon brain functioning, not least through angiogenetic mobilisation of nutrient logistics, as demonstrated by enhanced executive cognitive and working memory functioning, possibly arising from the neurophysiological adaptations through increments in physical fitness independent of any specific type of exercise regime. Thus, among the aged, physical exercise of most different variations enhances cognitive performances, promote brain plasticity, through release of brain-derived neurotrophic factor (BDNF), and enhance brain health [7-9],

and both human and animal laboratory studies attest to the singular role of BDNF in mediating the effects of exercise upon cognition [10]. Nevertheless, from the perspective of the evidence, discussed above, with the circling type of exercise, there is available a multitude of indications are that high-intensity interval training is a potent stimulus to promote cardiovascular fitness that relates markedly to cognitive performance. In a comparison of sixty-seven older participants (aged 55-75 years) assigned to 16-weeks of either: (i) resistance training, (ii) high-intensity aerobic interval training, (iii) moderate continuous aerobic training and (iv) a control condition, that were tested on the Stroop task for executive functioning, and with the Timed-Up-and-Go (TUG) procedures and 'submaximal' Bruce treadmill tests for physical functioning, it was observed that the combination of resistance training and high-intensity aerobic interval training provoked superior performance on the enhancement of older individuals' executive cognitive function than the moderate continuous aerobic training with the high-intensity aerobic interval training proving the most beneficial for improvement in information processing speed, providing the greatest gains regarding physical function [11].

In this regard, it has been shown repeatedly that simultaneous exercise-training interventions, especially those applying multimodal exercise programs together with auxiliary tasks that regulated through sensory cues, have promoted cognitive health remarkably well in both healthy older adults and clinically-affected, neurodegenerative patients, e.g. bearing Alzheimer's disease [12]. In an animal model of Alzheimer's disease, physical exercise, through treadmill-running (five sessions per week over four weeks), enhanced protein levels of AMPK-activated protein kinase activity and induced an up-regulation of the peroxisome proliferator-activated receptor gamma co-activator 1 alpha, fibronectin in type III domain-containing 5, and the brain-derived neurotrophic pathway thereby instigating and mediating the beneficial effects of exercise on amyloid β -induced learning and memory impairments [13]. Post-operative cognitive decline may be afflictive among more than ten percent of surgical patients and may induce markedly higher expressions of risk factors associated with advanced age, perioperative infections, and specific metabolic conditions that include obesity and insulin resistance. The rat model of metabolic syndrome, i.e., the "low capacity runner" rats differ 10-fold in their aerobic exercise capacity from the "high capacity runner" rats, the former of which, post-operatively, demonstrate an exaggerated cognitive decline both at three days and at three months of testing; this effect was prevented by pre-operative exercise training [14]. Over several instances of

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Received: January 16, 2018; Accepted: February 07, 2018; Published: February 15, 2018

Citation: Archer T (2018) Cognitive Health and Brain Plasticity due to Physical Exercise during Ageing. Clin Exp Psychol 4: 183. doi: [10.4172/2471-2701.1000183](https://doi.org/10.4172/2471-2701.1000183)

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cognition and learning, the mediatory effects of BDNF are persistent in the establishment of cellular plasticity [15,16] and are reliably achieved through various forms of physical exertion [17]. Suitably, the characteristics, impact, and scalability of these exercise programs have been documented [18]. Among the diversity of executive functions, controlled behavioral inhibition benefits more selectively from physical exercise than the other executive functions [19]. The epigenetic relationships arising from exercise predispositions gain ever-greater prevalence and frequency, even in the context of cognitive expressions. In the performance of this specific type of behavioral inhibition expression, it appears that the presence of BDNFVal66Val and Val66Met polymorphisms interacted variably with physical exercise parameters on controlled behavioral inhibition performance since Val carriers without exercise exhibited poorer executive functions performance at age 75 years than their comparison groups with higher exercise levels [20]. In a further study of inhibitory behavior performance, it was shown that the BDNFVal66Met polymorphism interacted with exercise such that inactive Val-homozygous participants, 114 healthy elderly volunteers, with a mean age of 71.53 years, demonstrated a lower inhibition performance than active Val homozygotes and inactive Met carriers [21]. Loss of telomeric proteins, oxidative stress, and hypoxia, contribute to shortening of telomere length generally and cardiac telomere length particularly and disruption of heart function whereas antioxidants, calorie restriction, and exercise can prevent both cardiac telomere attrition and the progression of chronic heart disease [22].

Conclusion

The inevitable, sacrosanct aspect of the progression of ageing, whether examined at cellular, tissue, organ, epigenetic or functional level, must be endured, and even endorsed, if individuals are to harvest the profits from self-developed opportunities for optimal health manifestations, rather than the neuro generative scenario that generally accompanies ageing. There is an upsurge of findings from various domains posit that the engagement and commitment to healthy lifestyle behaviors, such as physical exercise program, cognitive and social participation, stress reduction, and resilience training, may both prevent and protect against illnesses and disease states that accumulate ageing and concomitantly promote psychological and somatic health.

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