

“BDNF and Neurodegeneration: The Rise of the Exercise as a Preventive Care”

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ABSTRACT

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Mini Review

Since early 90's, the protein named brain-derived neurotrophic factor (BDNF) structure, functioning, gene code, location and specificities, as one of the most abundantly expressed growth factors of the Neurotrophins' family, have been massively explored in neuroscience fields [1,2]. With multiple functions in the formation and function of the central nervous system, all of which are lethal, the gene encoding for BDNF (named BDNF) abides highly stable throughout all vertebrates, including human [3]. From an evolution perspective, BDNF is reported as one of the key neurobiological molecule portraying the urge in the homo sapiens brain's grow after their needs for conquering new territories, acquiring memory, and navigation skills that implied in changes in the metabolism profile [4]. Nowadays, as the accumulated scientific knowledge on BDNF has fairly explained the association between brain and metabolism, the concept of neuroplasticity has so crossed boundaries from the molecular through clinical research fields, reaching the everyday conversation of individuals engaged into physical activity and healthy life style [5]. The sense that the brain is built of plastic matter and in so doing it is to a certain point “healable” comes together with an immediate preoccupation of how to protect the brain from malfunctions or else, how to preserve the brain from the losses of neurological diseases progress. Here I will elucidate the importance of BDNF for mechanisms involved in the functional brain's health and introduce the aerobic exercise, more specifically the aerobic energy metabolism, as main player in this scenario.

BDNF and the Brain's Health

Expressed mainly by neural tissue, BDNF participates in orientating cells differentiation into neurons during development processes and also in the adult brain processes as synaptogenesis, dendrite genesis, long-term potentiation and depreciation (memory formation) [6-8]. Corroborant, impairments in brain functions such as in Mild Cognitive Disorder disorders, Parkinson's, Alzheimer's, Levy's Body's and less frequent types of dementia are accompanied by lowered BDNF levels [9]. The very neuronal ability of functional regeneration which is based on the construction and strengthening of connections between neurons is compulsorily supported by BDNF in a vary of ways [10]. The release of BDNF occur after a multi-stage processing that primarily produces a precursor form of the neurotrophin (named pro-BDNF) which is later cleaved into the mature BDNF both before or during the release process. Binding of either the mature BDNF with its high-affinity Tyrosine kinase receptors B or of the released pro-BDNF form with low-affinity to p75 neurotrophin receptors mediate either pro-survival or pro-apoptotic processes, respectively, in different neuronal cell populations, in a highly coordinate dynamics of lethal importance [11].

In blood, the levels of BDNF are believed to be a plausible report of changes in the brain's cognitive performance associated with aging and natural neurodegeneration or neurodegenerative

disorders [12-14]. While reports of studies involving older individuals show a natural decrease in BDNF levels in time; a noticeable reduction in BDNF levels is observed in those individuals with neurodegenerative disorders. Moreover, decreases in BDNF levels appear to be associated with disease progress and cognitive impairment degrees [15]. Although a great effort has been spent into experimental attempts of finding an exogenous manner of manipulating BDNF in the past decades [16,17], the precise dynamics of BDNF release and reuptake differentially orchestrated in different brain regions and cell populations has led the pharmacological research to exhaust the possibilities, before advances to human studies.

The Exercise as a Preventive Care

At a physiological perspective, regulation of BDNF expression under the hub of a main energy metabolism regulator – peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1 α) [18,19] – is thus highly responsive to challenges in aerobic energy demands (see more at De Assis GG, 2018 5). In compliance, studies in healthy populations have revealed that individuals with higher fitness conditions display of higher circulating BDNF levels [20,21]. In the same sense, clinical approaches using the aerobic exercise as intervention show an exercise effect on blood levels of BDNF increase even in aging individuals at the course of cognitive disorders [22]. Likewise, physically active individuals show off better results in executive tasks when compared to sedentary individuals in same social environment [23]. Additionally, the BDNF increases related to exercise have been confirmed to exert a positive impact on the benefits observed in cognition. Furthermore, such BDNF-mediated improvement in cognitive processes can be found in individuals of all ages, when submitted to exercise [24]. Regarding the world's populations are aging as a result of an enhancement in the quality of life which, among other factors, contributes to increases in the prevalence of dementia, the aerobic exercise shall be considered not only as a healthy practice but, and more importantly, as an affordable long-term preventive Health care support, with potential for reducing public expenses.

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