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Exercise Alleviates Autism Spectrum Disorder Deficits

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Introduction

Amongst other behavioral deficits, children afflicted with Autism Spectrum Disorders (ASDs) present an array of motor skill impairments [1-3]. These deficits include problems the planning and performance of goal-directed behaviors [4,5]. Simermeyer and Ketcham (2015) have studied aspects of motor planning ability in ASD-diagnosed children (aged 5-13 years) through application of a method consisting of fine and gross motor tasks and postural components through altering sensory input [6]. They observed that ASD children expressed greater variability in hand selection during the "dial-turning task" and a tendency to plan movements that were not in accordance with 'end-state comfort'. These children displayed a reduced ability to imitate movements correctly, presented lower scores for both the drawing and stickler tasks, and required longer time to 'bead the bracelet' than the comparison control group. The notion of end-state comfort refers to planning movements that allow individuals to attain comfort at task completion despite an initial phase of no-comfort/discomfort [7]. In children presenting normal development, a near completion of end-state performance is reached by 10 years-of-age [8]. Thus, the impairments by ASD children described by Simermeyer and Ketcham (2015) imply a serious disadvantage. The purpose of this treatise is to examine the notion of physical exercise as intervention to facilitate a positive developmental trajectory, as has been observed both under normal conditions and those associated with developmental disturbance, e.g. ADHD [9-14]. Motor skills difficulties, such as balance, posture and gait and movement speed, are present in children diagnosed with ASD [15,16]. Some of the behaviors associated with ASD stereotypical, repetitive and counterproductive. Episodes of physical exercise and/or activity were found to reduce the stereotypies and increase positive behaviors, such as time spent on tasks [17,18]. Other deficits in ASD involve alterations in the neurophysiological response to stress, including impairments in heart rate adaptation to challenges set by attentional demands and social encounters [19-21]. In circumstances of physical exertion or stress that occur during exercise it has been observed repeatedly that individuals (children) presenting ASD generally displayed lowered physiologic, i.e., heart rate, adaptations [22-24]. Pace and Bricout (2015) have shown that in comparison with a group of healthy children, a group of ASD children (aged 10 +1.45 years) evidenced lowered heart rate: at pre-test, during physical evaluation and at maximal exertion [25]. The ASD children displayed also a higher number of falls on the balance test, lower force on the handgrip test, lower levels of performance on the plate tapping test, vertical and broad jump tests, the Euro fit sit-up test and the test of reactive speed. They required too a greater length of time to achieve the motor educational course. They concluded that the ASD children in the sample may be characterized by motor impairments, lower daily skills abilities, and deficits in cardiac adaptation to physical exertion. Nevertheless, the consensus of the findings implied that physical activity programs initiated early ought to be maintained into adulthood thereby ensuring against cardiovascular risks associated with a sedentary lifestyle. Perinatal administration of the anti-epileptic drug, valproic acid, has been applied as a neurobehavioral model of autism in rodents [26]. It induces symptoms of autism that involve social and cognitive deficits and repetitive behaviors, suppresses the number of BrdU-positive (5-bromo-2'-deoxyuridine-positive) cells, linked to reelin, in the hippocampus induced autistic-like behavior in male rats through administration of valproic acid (400 mg/kg) on postnatal day 14 [27-29]. They were assigned to either exercise or sedentary groups from postnatal day 28 onwards for four weeks. Treadmill exercise was maintained five times/week during a 30 min session each day. The treadmill exercise load was: 1st five-min period at a speed of 2 meters/min, 2nd five-min period 5 meters/min, and 8 meters/min over the final 20 min. In tests of social behavior, the heightened aggressive behavior of valproate-injected rats was reduced by the treadmill exercise regime concurrent with improved cognitive performance in an eight-arm, radial arm maze. Postnatal valproic acid reduced reelin, an extracellular matrix glycoprotein that regulates neuronal migration and positioning during brain development, in the hippocampus, whereas, the treadmill running intervention increased reelin expression in the valproate-treated rats. The utility of animal models of ASD needs to be exploited more completely since rodents adapt rapidly to treadmill type running exercise interventions thereby facilitating the examination of a multiple of symptom phenotypes and biomarkers of disorder [30]. Despite the promise of exercise intervention for the alleviation of ASD symptoms, several conditions require fulfillment for effective improvements to be obtained; these include:

(i) The notion of individual participation and compliance in physical exercise programmes.

(ii) The pervading presence of issues concerning balance impairments which require taylor-made exercise forms, and

(iii) The reality of auditory hypersensitivity which complicates the choice of exercise regimes.

A paucity of intervention studies have concentrated upon the notion of 'individual participation'. Adair et al., (2015) have shown that individually-tailored, educational and mentoring programmes enhanced participation outcomes, particularly with regard to exercise regimes wherein in cases where participation was only a secondary outcome, little or no effect was registered [31]. Balance deficits are present in ASD and are exacerbated by alterations of stance yet these deficits were unrelated to symptom severity when age-of-subject was taken into account, the complications associated with interventions are under study [32,33]. Finally, in ASD children, auditory modality hypersensitivity presents an important feature of disability. The "listening project protocol" offers a new intervention, a form of 'neural exercise' that applies acoustic stimulation to recruit the neural

regulation of the middle ear muscles [34]. Listening project protocol was hypothesized to reduce auditory hypersensitivities by increasing the neural tone to the middle ear muscles to functionally dampen competing sounds in frequencies lower than human speech. Their experimental trials demonstrated that listening project protocol, when contrasted to control conditions, selectively reduced auditory hypersensitivities. These findings are consistent with the polyvagal theory, which emphasizes the role of the middle ear muscles in social communication.

Despite the established genetic and neuroimmune connections, environmental factors, such as diet and gastrointestinal complications, are being taken into account increasingly [35-38]. The notion of "plural autisms" affecting the expressions of developmental trajectories focuses attention on influences of diet. For example, it appears that differing responses to the use of a gluten- and casein-free diet, defined as best- and non-response, has combined with some progress on determining the underlying genetic and biological correlates potentially related to such dietary elements [39]. Currais et al. have shown that the dietary glycemic index induces a marked impact upon ASD the phenotype [40]. In BTBR mice, a model of ASD, they found that the diet modulated plasma metabolites, neuroinflammation, and brain markers of neurogenesis to mimic the human condition. Puig-Alcaraz, et al. measured homocysteine, glutathione, methionine, and 3-nitrotyrosine in the urine of ASD children. They observed the increase in homocysteine was directly related to the severity of the communication skills deficits but the deficits in socialization skills or the preponderance of repetitive/restricted behaviors [41]. In an examination of several elements of dietary supplements in 56% of children presenting ASD, Stewart et al. found deficiencies in vitamin D and calcium; supplementation caused excess vitamin A, folate, and zinc, as well as vitamin C and copper (2–3 years), and manganese and copper (4–8 years) [42]. Thus, the dangers of dietary supplement ought to be observed.

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