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The relationship between physical exercise and cognition in children with typical development and neurodevelopmental disorders

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Abstract

This research project sought to investigate the relationship between physical exercise and cognition in children with and without a neurodevelopmental condition. To achieve this aim, three approaches were undertaken to explore the exercise and cognition relationship. The first approach sought to understand the efficacy of exercise interventions on cognition in individuals with a neurodevelopmental disorder. The second approach was to understand the effectiveness of an exercise activity when compared to a cognitively-engaging tablet game activity on measures of implicit learning and attention in children with and without a neurodevelopmental condition. The third approach was to investigate if psychophysiological measures could account for the cognitive effect observed after exercising in children with and without a neurodevelopmental condition. Taking the approaches together, this research project focused on investigating the efficacy, effect, and mechanism of the exercise-cognition relationship.

To investigate the efficacy of the exercise interventions, a meta-analytic review was conducted on 22 studies from the neurodevelopmental literature. The main findings from this meta-analysis revealed an overall small-to-medium effect size of exercise on cognition, supporting the efficacy of applying exercise interventions to young individuals with a neurodevelopmental disorder. Similar to the general population, physical exercise has been demonstrated to improve some but not all cognitive functions, with some individuals demonstrating no change in cognitive function after exercising.

In terms of the effects of physical exercise, this project conducted an experimental study comparing a moderate-intensity exercise activity with a tablet game activity for a period of 12 minutes in 35 children aged 6-11 years. Overall, the study found that the effect of exercise was comparable to the tablet activity across the reaction time measures, but not on the accuracy performance of the implicit learning and attention tasks. Overall, exercise

activity led to a better accuracy performance on implicit learning and executive attention compared to the tablet activity, particularly in children with a neurodevelopmental condition.

The last part of this project was an extension of the experimental study whereby psychophysiological measures were investigated based on a proposed detrended fluctuation analysis (DFA). This investigation found that galvanic skin response (GSR), as indexed by its scaling exponent, was related to whether children revealed a change in cognitive function after receiving the exercise activity, particularly on executive attention. Importantly, this relationship was also able to account for children who did not demonstrate a cognitive effect of exercise. This result was not evident in the electroencephalogram (EEG) measures. This investigation concluded that the effect of exercise on executive attention was dependent on the interplay between an individual's arousal system, cognitive task demand, and the novelty of the exercise activity.

Taking the findings together, this project highlights the importance of individual differences to the exercise and cognition relationship. Specifically, this project demonstrated the feasibility of investigating the scaling exponent, via fractal analysis (e.g., DFA), as an index of individual differences. Additionally, fractal analysis is a valuable tool to assist in further understanding the mechanism underlying the exercise-cognition relationship, particularly on the influence of individual differences.

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Dated: 25/08/17

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Introduction

Physical activity has been known to have a broad positive effect on physical and psychosocial health (e.g., Australia Department of Health, 2004; 2014; Kramer & Erickson, 2007; Leavy, Bull, Rosenberg, & Bauman, 2011; Prakash, Voss, Erickson, & Kramer, 2015; World Health Organisation, 2010; 2015). Specifically, the concept of physical exercise leading to a cognitive enhancement in humans is an exciting proposition that has been readily accepted by researchers, media, and the general population, and in certain instances, without much scrutiny (McMorris, Tomporowski, & Audiffren, 2009).

Research on physical exercise in enhancing cognition is not limited to the general population (e.g., McMorris & Hale, 2012; Vazou, Pesce, Lakes, & Smiley-Oyen, 2016). Recently, there has also been an increasing focus on the application of exercise interventions to improve cognitive functions in clinical populations, such as individuals with a cerebrovascular accident (Constans, Pin-barre, Temprado, Decherchi, & Laurin, 2016), Huntington's disease (Cruickshank et al., 2015), schizophrenia (e.g., Firth et al., 2017), Alzheimer's disease (e.g., Morris et al., 2017), autism spectrum disorder (ASD; e.g., Anderson-Hanley, Tureck, & Schneiderman, 2011), attention-deficit/hyperactivity disorder (ADHD; e.g., Grassmann, Alves, Santos-Galduróz, & Galduróz, 2017), overweight (e.g., Crova et al., 2014), and Parkinson's disease (e.g., Caciula, Horvat, Tomporowski, & Nocera, 2016).

Although the facilitating effect of physical exercise on cognition has been generally accepted by researchers (e.g., Chang, Labban, Gapin, & Etnier, 2012; Kramer & Erickson, 2007; Tomporowski, McCullick, Pendleton, & Pesce, 2015; Verburgh, Königs, Scherder, & Oosterlaan, 2014), the exercise and cognition relationship is not well-understood, despite various neurobiological (e.g., Kempermann et al., 2010; Ratey & Loehr, 2011) and cognitive psychological theories (e.g., Audiffren, 2009; Audiffren & André, 2015) being proposed to

explain this relationship. Moreover, some researchers have cautioned about drawing conclusions regarding the effect of physical exercise on cognition, particularly when advocating for its effectiveness as a cognitive intervention (McMorris et al., 2009; Prakash et al., 2015). Indeed, contrary to general belief, physical exercise does not enhance every cognitive function (e.g., Etnier, 2009; Kramer & Erickson, 2007; Tomporowski, Davis, Miller, & Naglieri, 2008), and not every individual will experience a facilitating effect after exercising (e.g., Audiffren, 2009). Regrettably, there has been little research focus on individuals who do not respond to the cognitive effect of physical exercise. Furthermore, the number of individuals who would or would not respond to the cognitive effect of exercise is currently unknown.

Investigations of the influence of individual differences on the exercise and cognition relationship have been limited (Diamond & Ling, 2016; McMorris et al., 2009; Pesce, 2009). Examining individual factors as a moderator of the physical exercise and cognition relationship has been a challenge to researchers. In particular, there are inconsistent findings on what type of individual factors moderate the effect of exercise on cognition (e.g., fitness: Chaddock et al., 2012; Hillman, Kamijo, & Scudder, 2011; Smiley-Oyen, Lowry, Francois, Kohut, & Ekkekakis, 2008). Further, there is the practical consideration of how to take into account the many individual factors that are postulated to affect the exercise and cognition relationship (see Diamond & Ling, 2016). These challenges may have resulted in the exercise-cognition researchers focusing more on the experimental manipulation of physical exercise parameters (e.g., Masley, Roetzheim, & Gualtieri, 2009; Ruscheweyh et al., 2011) over individual differences.

The challenges in the research literature highlighted above are not unique to the general population. Specifically, exercise interventions have been investigated in research with the ASD and ADHD samples, where the aim has been to attempt to improve various

areas of functioning, such as problem behaviours (e.g., Celiberti, Bobo, Kelly, Harris, & Handleman, 1997; Gapin, Labban, & Etnier, 2011), emotional (e.g., Gawrilow, Stadler, Langguth, Naumann, & Boeck, 2016; Hillier, Murphy, & Ferrara, 2011), and social functioning (e.g., Bass, Duchowny, & Llabre, 2009; Kang, Choi, Kang, & Han, 2011). In terms of enhancing cognition through exercise interventions, beneficial effects have been reported by various ASD and ADHD studies (e.g., Anderson-Hanley et al., 2011; Choi, Han, Kang, Jung, & Renshaw, 2015). However, as the application of physical exercise on facilitating cognition in individuals with neurodevelopmental disorders is relatively new, the efficacy of exercise interventions in enhancing various areas of cognitive functions has not been examined. Furthermore, similar to studies with the general population, the number of individuals with ASD or ADHD that would respond to the facilitating cognitive effect of exercise is unknown. Additionally, there is also a need to understand why certain individuals with a neurodevelopmental disorder do not respond to the cognitive effect of exercise.

Although the effect of physical exercise on cognition has been repeatedly demonstrated (e.g., Anderson-Hanley et al., 2011; Chang, Hung, Huang, Hatfield, & Hung, 2014; Pesce, Crova, Cereatti, Casella, & Bellucci, 2009), given that the number of individuals who would respond to the cognitive effect of exercise is currently unknown, the effect of exercise needs to be evaluated, particularly in comparison to an active control group. Recently, video game activity has been linked to improved cognitive processes in children with neurodevelopmental disorders (e.g., Bioulac et al., 2014), and those with a typical development (e.g., Granic, Lobel, & Engels, 2014). As there is some likelihood that the cognitive effect of physical exercise is not significantly different to other cognitively-engaging activities (McMorris et al., 2009), such as video games, it would seem appropriate to consider whether the effect of exercise is better than a video game activity in children with and without a neurodevelopmental condition.

Apart from demonstrating that physical exercise enhances cognitive functions, there is also a need to consider what other non-physical factors, such as individual differences, are involved in the relationship between exercise and cognition (Diamond & Ling, 2016; McMorris et al., 2009; Pesce, 2009). As mentioned earlier, there are significant challenges to the study of individual differences, especially in view of the many factors that could likely moderate the effectiveness of exercise on cognition (e.g., fitness, diagnosis). Nevertheless, the focus on individual differences in the exercise-cognition relationship would aid in further understanding the mechanism underlying this relationship. Ideally, the study of individual factors should provide an account of both those individuals who would demonstrate a cognitive effect after exercising, and those who would be non-responsive to the effect of exercise.

Given that the arousal system has been implicated in the exercise and cognition relationship (e.g., Audiffren, 2009; Audiffren & André, 2015; Chu, Alderman, Wei, & Chang, 2015; Dai, Chang, Huang, & Hung, 2013; Dietrich & Audiffren, 2011; Kamijo, O'Leary, Pontifex, Themanson, & Hillman, 2010), galvanic skin response (GSR) and electroencephalogram (EEG) are possibly useful psychophysiological measures for the study of individual differences. However, instead of investigating how physical exercise leads to a high or low mean value of GSR and EEG measures, this research project adopted a novel method that focused on how these psychophysiological data fluctuate across time (i.e., fractal analysis; Peng, Havlin, Stanley, & Goldberger, 1995). The theoretical rationale for the use of fractal analysis is presented in Chapter 1. Briefly, rather than focusing on what physical exercise should be given to children in order to achieve an optimal cognitive outcome (e.g., Anderson-Hanley et al., 2011; Gallotta et al., 2015; Verret, Guay, Berthiaume, Gardiner, & Béliveau, 2012), the current research project, through the use of fractal analysis, investigated how individuals respond to the effect of exercise. It was postulated that the focus on

individual differences, through fractal analysis, would provide insight into why certain individuals do not respond to the cognitive effect of physical exercise.

The purpose of this research project was threefold. First, this research project sought to determine the efficacy of physical exercise interventions on enhancing cognition in individuals with neurodevelopmental disorders, and to link the research with this clinical population with research reported on the general population. The second aim of this research project was to test the after-effect of an acute physical exercise activity against a cognitively-engaging tablet game activity on measures of implicit learning and attention in children with a typical development and those with a neurodevelopmental condition. Third, this research project also sought to determine if individual differences could account for the children's cognitive performance after performing the acute exercise activity. In addition, a novel method for investigating individual differences is proposed (i.e., fractal analysis). Taking the above aims together, this research project sought to understand the efficacy, effect, and mechanism of the physical exercise and cognition relationship in children with and without a neurodevelopmental condition.

The thesis is divided into six chapters. In Chapter 1, previous research on the effects and mechanisms of physical exercise on cognition is presented. Additionally, the background and rationale for the proposed fractal analysis (i.e., complexity theory) to the investigation of the exercise-cognition relationship is also presented. In Chapter 2, the findings from the meta-analytic review conducted to determine the efficacy of exercise interventions on cognition in individuals with neurodevelopmental disorders is reported. The next three chapters report the experimental and psychophysiological study conducted to investigate the after-effect and mechanism of the acute exercise activity in children with and without a neurodevelopmental condition. Specifically, Chapter 3 provides an overview and details of the methodology used in this project. Chapter 4 presents the results of the experimental study

comparing the after-effects of the acute exercise activity with the tablet game activity on measures of implicit learning and attention. Additionally, Chapter 5 reports the findings of the psychophysiological investigation based on the proposed fractal analysis to account for the exercise-induced cognitive effect observed in Chapter 4. Lastly, a consolidation of the findings reported in this research project is presented and discussed in the context of the exercise-cognition research in Chapter 6.

Chapter 1: Physical Exercise and Cognition

Physical activity has been widely recommended to children both internationally (World Health Organisation, 2010; 2015) and in Australia (Department of Health, 2004; 2014). These guidelines highlight the importance of physical exercise mainly for the prevention of physiological health conditions, such as diseases involving the cardiovascular system, and psychological disorders namely depression and anxiety. However, the benefits of physical exercise on cognition are only mentioned briefly in recent guidelines. Over the years, there has been increased research investigating the relationship between physical exercise and cognition (e.g., Booth et al., 2014; Sibley & Etnier, 2003; Tomporowski, Davis, Miller et al., 2008; Tomporowski, Lambourne, & Okumura, 2011). The general consensus is that moderate-intensity, aerobic-type physical exercise has a positive impact on the development and improvement of cognitive functioning in typical developing children (i.e., executive functioning), although the mechanism whereby exercise affects cognition is currently unclear; with neurophysiological and/or psychosocial factors likely to be involved in the process (e.g., Tomporowski et al., 2011). Additionally, emerging research on children with neurodevelopmental disorders has suggested that physical exercise could be used as an intervention in improving cognitive performance in these populations (e.g., Anderson-Hanley et al., 2011; Gapin & Etnier, 2010).

Autism spectrum disorder (ASD) is a neurodevelopmental disorder that includes disorders previously known as autistic disorder, Asperger's syndrome and pervasive developmental disorder not otherwise specified. ASD is accompanied by various difficulties in areas of social communication, and restricted, repetitive behaviours (American Psychiatric Association, 2013). Previous intervention studies using moderate-intensity, aerobic-type exercise on children with ASD found improvements in academic and work-task performance (Rosenthal-Malek & Mitchell, 1997), classroom involvement time (Nicholson, Kehle, Bray,

& Heest, 2011), attention span (Tan, Cohen, & Pooley, 2013a) and aspects of executive functioning (Anderson-Hanley et al., 2011). These results suggest that the therapeutic use of physical exercise on individuals with ASD is likely to be beneficial. Nonetheless, these studies reported small sample sizes and most of them lacked a control group, which limits generalisation. Furthermore, the efficacy of antecedent exercise on improving aspects of cognitive performance in children with ASD is unknown, indicating a need for further evaluation in this area.

Another type of neurodevelopmental disorder is attention-deficit/hyperactivity disorder (ADHD). It can be classified broadly as either inattention or hyperactivity-impulsivity behaviours, or both (American Psychiatric Association, 2013). ADHD symptoms are pervasive across various life settings and impact on areas, such as academic, social and family functioning. A major associated impairment is the deficit in executive function, such as the ability to inhibit behavioural responses and shift attention (Barkley, 1997; Smith et al., 2013). Studies that engaged individuals with ADHD using exercise interventions have reported progress in impulse control (Smith et al., 2013), speed of processing visual tasks and sustained attention to auditory information (Verret et al., 2012).

A review by Grassmann et al. (2017) examined papers published from 1980 to 2013 on the effects of a single session of exercise intervention on the cognitive functioning of children with ADHD. The authors found three studies that reported improvements in various aspects of executive functions following aerobic exercises of moderate and higher levels of intensity. Recently, Cerrillo-Urbina and colleagues (2015) conducted a meta-analysis on eight randomised controlled studies that investigated the effects of physical exercise on ADHD overall symptomatology in young individuals aged 6-18 years. In relation to cognition, the authors reported moderate and large effect sizes for attentional (i.e., five studies) and global executive functioning (i.e., three studies) measures, respectively. However, the efficacy of

physical exercise on specific types of cognition (e.g., inhibition, set-shifting) in individuals with ADHD is currently unknown.

Although the beneficial effects of physical exercise on children with typical development and those with neurodevelopmental disorders are consistently reported in the exercise-cognition research, its mechanism is unclear (e.g., Anderson-Hanley et al., 2011; Grassmann et al., 2017; Lees & Hopkins, 2013; Piepmeier & Etnier, 2015; Tomporowski et al., 2015). This issue is complicated by potential mediating effects, such as neurophysiological changes in the brain (e.g., brain-derived neurotrophic factor, BDNF) and other psychosocial factors, including self-efficacy (e.g., Ratey & Leohr, 2011; Tomporowski et al., 2011). Most of the previous research on this issue has examined the outcomes of physical exercise but fewer studies have investigated its mechanism. Despite studies that examined changes in neurochemicals (e.g., catecholamine) being informative regarding what happens in the brain or body after exercising (e.g., Ferris, Williams, & Shen, 2007; Wigal, Emmerson, Gehricke, & Galassetti, 2013; Winter et al., 2007), there is currently a lack of understanding regarding the mechanisms by which physical exercise improves cognition.

A recent review by Tomporowski et al. (2015) has provided an overview of the exercise-cognition research. The authors identified that the field is categorised into acute and chronic physical exercise studies, and those that focused either on quantitative and/or qualitative aspects of exercise. Quantitative exercise studies are defined by the authors as those that are based on simple, straightforward and repetitive type of exercises, such as running. These quantitative exercise studies relied on the experimental control of the intensity and duration of physical exercise. Qualitative exercise studies, however, are based upon more complicated movements like the basketball activity. The complex motor coordination is postulated to moderate the degree of cognitive engagement. Therefore, qualitative exercise studies relied on manipulating the components of the exercise activity (e.g., motor

coordination). Hence, quantitative and qualitative physical exercise are assumed to reflect low and high cognitive demands on the individuals, respectively (Tomporowski et al., 2015). Numerous reviews have generally supported the facilitating effects of exercise on cognition in acute and chronic studies, and quantitative and qualitative exercise studies (e.g., Kramer & Erickson, 2007; Lambourne & Tomporowski, 2010; Sibley & Etnier, 2003; Tomporowski, 2003; Tomporowski et al., 2015). However, previous research has consistently indicated that the effect of physical exercise is equivocal, dependent on the type of cognitive tasks or processes. Thus, there is a need to consider the specific effects of physical exercise on various cognitive measures.

Cognitive Effects

The benefits of physical exercise have been associated with a broad array of cognitive functions, including but not limited to, aspects of information processing (e.g., Tomporowski, 2003), memory functions (Pesce et al., 2009), attention (e.g., Janssen, Toussaint, van Mechelen, & Verhagen, 2014), and academic functioning (e.g., Davis & Cooper, 2011; Lees & Hopkins, 2013). Recently, the research literature has narrowed the effects specifically to executive functions (e.g., Audiffren & Andre, 2015; Etnier & Chang, 2009; Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008) as being more sensitive to the effects of physical exercise. Nevertheless, some researchers have also begun to urge further investigation into the connection between exercise and meta-cognition, and how this relationship contributes to children's academic performance (Tomporowski et al., 2015; see Álvarez-Bueno et al., 2017).

The facilitating effect of physical exercise on overall cognition is well-accepted by researchers, however, the findings are mixed when aspects of cognition are considered. Several meta-analyses on typical developing populations have found varying effect sizes of exercise on various executive functions (e.g., acute exercise studies: Chang, Labban et al.,

2012; chronic exercise studies: Verburgh et al., 2014). These meta-analyses reported a consistent effect of exercise particularly on executive function (EF) tasks examining inhibition, with effect sizes ranging between small to medium, but the effects on other EF domains like set-shifting and working memory are less clear. Indeed, relative to control conditions, set-shifting and short-term memory were found to be unaffected by physical exercise in a group of 18 young adults after 40 minutes of moderate-intensity stationary cycling (Coles & Tomporowski, 2008), though aspects of their delayed-recall performance were maintained only in the exercise condition. This finding is consistent with those reported with children by Tomporowski, Davis, Lambourne, Gregoski, and Tkacz (2008), and Craft (1983).

Tomporowski, Davis, Lambourne et al. (2008) administered an acute exercise intervention via walking on a treadmill to a group of 69 overweight children aged 7 and 11 years for 23 minutes. However, the authors could not find a post-intervention facilitating effect on set-shifting compared to an educational video. Similarly, Craft (1983) measured multiple memory tests, including working memory performance in typical developing and hyperactive children during baseline followed by 1, 5 and 10 minutes of stationary cycling, but could not detect any positive effects of physical exercise.

Contrary to the null findings, Chen, Yan, Yin, Pan, and Chang (2014) found that 30 minutes of group running led to improved inhibition, set-shifting and working memory performance in 39 third and fifth grade children compared to a control group (i.e., reading). Likewise, group physical exercise of moderate-vigorous intensity for an hour was also found in another study by Pesce et al. (2009) to enhance both short- and delayed-recall memory performance in 52 older children (i.e., 11-12 years old). Interestingly, in an individual circuit training of comparable exercise intensity and duration, the authors found improvement only on the delayed-recall performance, unlike those found in a group exercise activity (Pesce et

al., 2009). Furthermore, a nine-month longitudinal study that aimed to improve participants' fitness supported the positive effect of mixed exercise activity (i.e., individual/team exercise stations and games) on working memory in children (7-9 years) compared to a waitlist control group (Kamijo et al., 2011). Together, these studies provide evidence that the effect of physical exercise is not uniform across cognitive functions (e.g., Etnier & Chang, 2009; Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008), and is moderated by the differences in the exercise intervention used in studies (e.g., duration, intensity), and other moderators, including health and fitness levels.

Exercise-cognition research has traditionally focused on quantitative aspects of physical exercise. However, some researchers have recently argued that the duration and intensity of the exercise activity may not be the sole factors that are responsible for the enhancement of cognition (e.g., Budde, Voelcker-Rehage, Pietraßyk-Kendziorra, Ribeiro, & Tidow, 2008; Diamond & Ling, 2016; Pesce et al., 2009; Pesce, 2012). These researchers are proponents of the qualitative exercise studies that focused on the enrichment of the exercise activity (Tomporowski et al., 2015). In clarifying the definition of a qualitative exercise, Pesce (2012) stated that motor coordination and cognitive demands are two components of a qualitative exercise that are important in facilitating the exercise-induced cognitive effect. Pesce further proposed that a qualitative exercise activity that encompasses both components should lead to better cognitive performance than would otherwise be obtained via motor coordination or cognitive demands alone.

A key point of qualitative exercises is that the effect of exercise on cognition is dependent on the type of exercise activity (Pesce, 2012; Tomporowski et al., 2015). Specifically, qualitative exercises (i.e., complex motor coordination and high cognitive engagement) are assumed to have a larger effect on cognition than quantitative exercises (i.e., simple, repetitive physical movements and low cognitive engagement). Indeed, this

proposition is supported in a study with 70 primary school-aged children, who were either of average weight or overweight, and separated into a standard exercise program and an enriched exercise program with additional movement and cognitive demands (Crova et al., 2014). The authors reported higher cognitive gains in one aspect of executive functioning (i.e., inhibition but not working memory) in overweight children in the enriched exercise group compared to the standard exercise group. Similarly, Budde et al. (2008) also reported higher attentional performance in 115 adolescents that undertook enriched exercises that emphasised motor coordination compared to a standard exercise activity, though improvements were found in both groups of participants.

Research on qualitative aspects of physical exercise, however, is not without ambiguity as other studies investigating this area in children (Best, 2012; Gallotta et al., 2012), and young adults (O'Leary, Pontifex, Scudder, Brown, & Hillman, 2011), were inconsistent with Pesce's (2012) proposal. These studies contrasted the effects of an enriched exercise activity with challenging motor coordination (i.e., high cognitive demands), with a simple physical exercise (i.e., low cognitive demand), and a non-physical exercise condition (e.g., video game). Collectively, the authors in these studies typically reported that the enriched exercise activity was not better than a simple exercise activity in influencing cognitive performance, though improvements were found in both exercise conditions.

Despite conflicting evidence regarding the superiority of using physical exercises that has a low or high level of motor coordination and cognitive engagement in enhancing cognition, a central issue may be the difference in the "optimal challenge point" that varies among individuals (Pesce et al., 2013). Pesce et al. found that children with movement difficulties perform optimally in an aspect of attention with exercise activity that has a low cognitive demand but not with a high cognitive demanding type of exercise activity. Conversely, children with a typical development performed better with a high cognitive

demanding type of exercise activity compared to an exercise with a low cognitive demand. Pesce et al. suggest that the observed difference between the participants may be attributed to the individuals' respective challenge point, dependent on their age and developmental conditions. The optimal challenge point may partially explain the inconsistencies among studies that have attempted to delineate the level of cognitive and/or motor demands in different physical exercise activities. Furthermore, this study also suggests that individual differences cannot be disregarded when considering the cognitive effect of physical exercise. Indeed, multiple individual factors have been associated with the exercise and cognition relationship, and one of the factors is physical fitness (e.g., Chang, Labban et al., 2012).

Although physical fitness has been identified in previous research as one of the influencing factors that is involved in the effect of exercise on cognition (e.g., Chang, Labban et al., 2012; Diamond & Ling, 2016; Pesce, 2009), its influence is equivocal. Overall, studies have either reported an association between participants' fitness and cognition (e.g., Åberg et al., 2009; Colcombe & Kramer, 2003; Hillman et al., 2011), particularly for individuals that are physically fit (e.g., Chaddock et al., 2012; Hillman et al., 2011; Stroth et al., 2009), or no association between the variables (e.g., Etnier, Nowell, Landers, & Sibley, 2006; Smiley-Oyen et al., 2008). The role of fitness is further complicated by whether the relationship with cognition is based upon cognitive tasks or electrophysiological measures. For example, Kamijo et al. (2010) evaluated the association between fitness level and working memory performance in 72 undergraduate students separated into high- and low-fit groups based on their cardiorespiratory fitness. This study did not find significant differences in working memory performance between both fitness groups, but the EEG findings demonstrated otherwise. In general, the authors found that individuals in the low-fit group were less efficient in the allocation of neural resources in response to cognitive task demands as compared to the high-fit group, particularly in the frontal and central electrode areas.

Despite disparities in the findings pertaining to the influence of fitness levels on the exercise and cognition relationship, Etnier et al. (2006) and Smiley-Oyen et al. (2008) confirmed that correlations between fitness and cognition exist, but differences in fitness levels are unlikely to be the mechanism by which exercise affects cognition. Indeed, these studies reported that fitness accounts for 8-10% of the variance in the relationship. In other words, the current research literature does not support fitness being a mediator but there is some evidence that it can moderate the exercise-cognition relationship. However, fitness as a moderator is not a straightforward matter as it tends to be entangled with physical expertise, as in the case of athletes (Pesce, 2009), and whether the cognitive tasks are measured during or after exercising (Chang, Labban et al., 2012).

With physical fitness being identified as an important moderator in the exercise and cognition relationship (e.g., Chang, Labban et al., 2012; Pesce, 2009), it is not an uncommon practice for researchers to measure participants' fitness level either as a background variable (e.g., Davranche, Brisswalter, & Radel, 2015; Pontifex, Hillman, Fernhall, Thompson, & Valentini, 2009), or as a part of the experimental manipulation (e.g., Chaddock et al., 2012; Kamijo et al., 2010). Nevertheless, there may be a potential confounding issue given that physical exercise is usually involved in the process of evaluating fitness; in some cases, the fitness test is relatively similar to the exercise condition (e.g., Winter et al., 2007). The issue pertaining to the process of evaluating fitness has also been highlighted by some researchers (McMorris et al., 2009; McMorris & Hale, 2012).

Studies that evaluated fitness tend to expose participants to a brief exercise episode till exhaustion (e.g., treadmill or cycling ergometer), while measuring physiological variables, such as oxygen consumption (VO_{2max}), heart rate (HR), and respiratory rate (RR). The potential implication of investigating fitness as a moderator means that the physical exercise intervention may have started prematurely during the evaluation of fitness rather

than at the experimental or observational phase that researchers originally intended. Nevertheless, generally, studies manage to separate the fitness test from the initial exposure to the cognitive task by conducting them on separate days (e.g., Joyce, Graydon, McMorris, & Davranche, 2009; Kamijo et al., 2011), or having cognitive task prior to the fitness test (e.g., Chu et al., 2015; Davranche et al., 2015). However, the confounding issue of measuring fitness appears to be more apparent in cross-sectional studies that were meant to be observational, where some form of exercise was conducted to derive the participants' fitness level (e.g., Castelli, Hillman, Buck, & Erwin, 2007; Davis & Cooper, 2011).

Paradoxically, by evaluating an individual's fitness level even for a brief episode, studies inevitably introduce some form of physical exercise to the exercise-cognition relationship. Further, it is also plausible that in certain instances, exercise may have been given unintendedly to participants in the control group through fitness tests. Although the evaluation of participants' fitness may not directly affect the research outcome, the differentiation between the exercise activity and fitness test becomes difficult to establish. In other words, if fitness tests (i.e., exercise) are conducted on both the experimental group (i.e., exercise activity), and the control group (i.e., non-exercise activity), the true difference between the groups would not be the exercise activity, but rather, the difference lies in the amount of exercise given to the participants. It is noteworthy that the highlighting of the process of evaluating fitness is not meant to discredit previous research, which has undoubtedly made valuable contributions to the understanding of fitness to the exercise and cognition relationship. Rather, this point is raised to highlight one of the existing limitations and challenges of taking into account individual factors.

Research on individual differences in the relationship between physical exercise and cognition is relatively limited (Diamond & Ling, 2016; McMorris et al., 2009; Pesce, 2009). Regardless of whether a quantitative and/or qualitative exercise approach is taken,

researchers investigating this field tend to exhibit the implicit assumption that, if the external quantifiers of the exercise activity are tuned more or less "optimally" (e.g., intensity, duration, motor coordination and cognitive engagement), individuals should be able to demonstrate the cognitive effects of physical exercise. In a seminal paper by Speelman and McGann (2013), the authors cautioned about the limitation of such an assumption in research undermining the importance of individual differences. This point is evident by the fact that exercise-cognition research typically does not report the number of participants that demonstrated cognitive improvements as a result of receiving the exercise activity. As such, despite the overall positive conclusion in the research literature surrounding the cognitive effects of physical exercise (e.g., Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008), the likelihood of whether or not an individual would demonstrate a cognitive benefit as a result of exercise is unknown. Rather, the best conclusion at present is that, on average, participants in a physical exercise group tend to have better performance on some cognitive measures than participants in a control group (e.g., Best, 2012; Chen et al., 2014; Ruscheweyh et al., 2011).

Additionally, McMorris et al. (2009) have also highlighted that the existing research literature does not exclude the likelihood that there may be no difference between the effects of physical exercise and non-exercise activities on cognition. Moreover, research has mostly focused on the search for a universal optimal set of exercise parameters, including intensity (e.g., Ruscheweyh et al., 2011), duration (e.g., Craft, 1983), frequency (e.g., Masley et al., 2009), type (e.g., Pontifex et al., 2009), and more recently, motor coordination and cognitive demands (e.g., Schmidt, Egger, & Conzelmann, 2015); yet, a consistent recommendation regarding the exercise quantifiers does not currently exist, nor can it be established with confidence. Although qualitative exercise researchers do recognise the importance of

individual differences in terms of the optimal challenge point (Pesce et al., 2013), there is yet to be a concrete guideline on how this factor can be measured.

Diamond and Ling (2016) urged researchers to consider incorporating individual factors, including participants' physical and psychological health conditions, sleep quality, emotional and social variables when examining the exercise and cognition relationship. Indeed, there is a need to consider that, regardless of how the exercise activity is applied to individuals in a study, the outcome measures are always influenced by individual differences (Diamond & Ling, 2016). However, efforts to investigate moderating factors, including age, diagnosis, weight, and fitness levels have yielded mixed findings (e.g., Chang, Labban et al., 2012; Chen et al., 2014; Crova et al., 2014; Smiley-Oyen et al., 2008), further restricting the understanding of the exercise and cognition relationship. Furthermore, in view of the number of potential moderators reported in previous research, there is a practical challenge for researchers to consider all these factors in their experiments. Moreover, there is always a potential risk to external validity when too many variables are controlled (Martin, 2008, p. 27). Research on the exercise-cognition relationship indicates a need to shift the focus from external factors (i.e., quantifiers of the physical exercise) to individual differences. In other words, more attention should also be given to how individuals respond to the physical exercise, rather than just what is the "best" physical exercise that improves cognition. To account for the influence of individual differences, there is a need to first explore the mechanism whereby physical exercise could affect cognition.

Mechanism

Multiple reviews exploring the potential mechanism of exercise and cognition have been published. Existing research literature points towards mainly neurobiological pathways that are likely to be responsible for the cognitive effect of physical exercise (e.g., Cotman & Berchtold, 2002; Cotman, Berchtold, & Christie, 2007; McMorris et al., 2009; Piepmeier &

Etnier, 2015; Ratey & Loehr, 2011; Zoladz & Pilc, 2010). Apart from catecholamine and insulin-like growth factor 1 (IGF-1), one of the most commonly cited proteins is the brain-derived neurotrophic factor (BDNF). BDNF is an important protein that is mainly associated with neuroplasticity, learning and memory functions; it regulates, supports, and enhances neuronal activities, particularly in the hippocampus (e.g., Cotman et al., 2007; Ratey & Loehr, 2011). Proponents of the BDNF hypothesis support a neurobiological explanation of physical exercise on cognition in humans (e.g., Cotman & Berchtold, 2002; Cotman et al., 2007). However, evidence in support of this explanation has mostly come from animal studies (e.g., Gómez-Pinilla, Ying, Roy, Molteni, & Edgerton, 2002; Vaynman, Ying, & Gómez-Pinilla, 2004).

Reviews that examined the evidence from human studies do not support a neurobiological mechanism being responsible for the exercise and cognition relationship (Barha, Davis, Falck, Nagamatsu, & Liu-Ambrose, 2017; Kramer & Erickson, 2007; McMorris, 2009; Piepmeier & Etnier, 2015; Zoladz & Pilc, 2010). These reviews have consistently pointed out that more human research is required to confirm the hypothesis and that the existing findings are inconclusive. Indeed, while animal studies consistently reported changes in BDNF as a result of exercise (e.g., Adlard, Perreau, & Cotman, 2005; Berchtold, Chinn, Chou, Kesslak, & Cotman, 2005; Gómez-Pinilla et al., 2002; Vaynman et al., 2004), when examined in humans these changes are less conclusive.

For example, Vaynman et al. (2004) found that experimental rats that are BDNF inhibited lost the ability to demonstrate cognitive improvements and had an attenuation of cognitive performance similar to rats in the control group following exposure to running wheels. In human studies, however, BDNF levels are not significantly related to the exercise and cognition relationship in acute (Gapin, Labban, Bohall, Wooten, & Chang, 2015) and chronic studies (Ruscheweyh et al., 2011). Even in cases where the BDNF levels did increase

or decrease after exercising in human participants, the association with cognitive measures have either not been found (Ferris et al., 2007), or found partially (Winter et al., 2007), or not measured (Currie, Ramsbottom, Ludlow, Nevill, & Gilder, 2009; Vega et al., 2006).

Importantly, the current evidence from human studies does not demonstrate that the BDNF changes induced by physical exercise resulted in enhanced cognition (e.g., Barha et al., 2017). The differences between the BDNF findings from animal and human studies may be partly due to the methods of measuring BDNF levels (e.g., Berchtold et al., 2005; Piepmeier & Etnier, 2015; Vega et al., 2006). Blood samples are typically drawn from peripheral venous sites (e.g., brachial artery) in human participants, in contrast to more invasive procedures (e.g., brain dissection) in animals. Thus, there may be differences in the conclusions pertaining to the BDNF changes due to physical exercise, as the levels from peripheral blood samples may not be the same as those measured from the brain, though BDNF is known to cross the blood-brain barrier (e.g., Pan, Banks, Fasold, Bluth, & Kastin, 1998). Moreover, within the peripheral blood sample, BDNF level also differs depending on whether BDNF is derived from the blood serum or plasma (see Piepmeier & Etnier, 2015).

Notably, BDNF is not the only identified protein that has been postulated to be the mediator of the exercise-cognition relationship. For instance, IGF-1, serotonin and BDNF function closely together and are essential for the survival of neurons, and they are related to the relationship between learning and memory, and exercise (Mattson, Maudsley, & Martin, 2004). Additionally, BDNF levels can also be impacted by oestrogen levels or other neuronal activities, particularly in the medial septal region (Cotman & Berchtold, 2002). Furthermore, cortisol has also been reported to have a negative correlation with BDNF in animals (e.g., Cotman & Berchtold, 2002), though this is in contrast to human participants (Vega et al., 2006).

In general, research at the molecular level mainly focuses on how certain types of protein modulate or affect the production or inhibition of certain chemicals within the brain and body (e.g., Ratey & Loehr, 2011). A key observation is that no single protein functions in isolation (e.g., Cotman et al., 2007; Ratey & Loehr, 2011). Even BDNF is known to interact with other proteins not limited to IGF-1 and vascular endothelial-derived growth factor (VEGF). In addition, BDNF is also related to many other genes within the hippocampus (Cotman & Berchtold, 2002). Consequently, although informative, the greater the number of proteins or neurotransmitters that are involved in the exercise-cognition relationship, the more difficult it is to understand which factors account for the exercise-induced cognitive benefit and which factors are the by-product of physical exercise. As neurobiological theory cannot fully explain the effects of physical exercise on cognition, researchers have begun to seek other psychological explanations (e.g., Audiffren, 2009, Audiffren & André, 2015).

In an attempt to explore a general theoretical framework to account for the exercise-cognition relationship, Audiffren (2009) cited and evaluated a number of existing cognitive and energetic theories proposed by Kahneman, Sanders, Hockey, and Humphreys and Revelle. The review of these theories is beyond the goal of this thesis (see Audiffren, 2009). Nonetheless, the crux of these cognitive-energetic models is the concept of resource competition between external task demands, such as cognitive tasks, and internal resources, including arousal (see Table 1). In general, according to these theories, the mechanism by which physical exercise improves or attenuates cognitive performance is a resource competition, such that if internal resources surpass external demands, an improvement is likely to be observed. Alternatively, a deterioration in cognitive performance occurs when internal resources are constrained by external demands. Additionally, exercise is also incorporated into these models as a means to induce arousal and/or cognitive resources, but

only if the cognitive tasks are not competing or interfering with the exercise (i.e., dual-tasks condition).

Table 1

Summary of Various Cognitive-Energetic Models in Accounting for the Exercise-Cognition Relationship

Note. Reproduced from "Acute Exercise and Psychological Functions: A Cognitive-Energetic Approach", by M. Audiffren, 2009, p. 25. In T. McMorris, P. Tomporowski, & M. Audiffren (Eds.), *Exercise and Cognitive Function*.

Another related cognitive psychological theory has been proposed recently by Audiffren and André (2015). These authors incorporated the work of Baumeister and colleagues on the strength model of self-control in an attempt to provide a theoretical framework to account for the after-effects of acute and chronic physical exercise on executive functions. In particular, Audiffren and André further expanded on the cognitive-energetic models discussed and consolidated in Audiffren's review (2009). Audiffren and André included self-control resources as a limited capacity and the role of positive emotion induced by exercise to the exercise-cognition relationship. Briefly, similar to the cognitive-energetic models, exercise-induced cognitive improvement or attenuation is determined by the cognitive task demand and the availability of cognitive resources (i.e., self-control resources).

Based on this extended model (Audiffren & André, 2015), self-control is conceptualised as a limited resource, such that if self-control is required to perform the physical exercise, the availability of that resource will be further taxed should the after-exercise activity also require more self-control resources (i.e., executive function tasks), leading to an attenuated cognitive performance. Furthermore, cognitive improvements in executive function are due to the availability of the self-control resources that can cope with the demands of the cognitive task. In other words, if the self-control resources are expended to perform the exercise activity, whatever the level of self-control that remains would either lead to a facilitating or detrimental effect on cognition, dependent on the level of cognitive task demand. Hence, the post-exercise cognitive performance is postulated to depend on the availability of the self-control resources. In addition, positive emotion and its variant (e.g., motivation) can also moderate the availability of the self-control resources, which can also impact on the cognitive outcome (Audiffren & André, 2015).

The current issue with the cognitive psychological models is that, though they may aid in partially explaining the effects of physical exercise, these models are rarely cited or

explored in the exercise and cognition studies. Further, the extended strength model of self-control proposed by Audiffren and André (2015) has only been recently added to the exercise-cognition literature. Moreover, researchers tend to adopt neurobiological mechanisms in explaining the facilitating effects of physical exercise (e.g., Ellemberg & St-Louis-Deschênes, 2010; Soga, Shishido, & Nagatomi, 2015). Thus, research on the cognitive psychological models in the exercise-cognition relationship is very limited.

Nevertheless, in terms of the cognitive-energetic models for example, findings from electrophysiological studies do provide some support (e.g., Chu et al., 2015; Kamijo et al., 2010), albeit indirectly. First, physical exercise facilitates attentional resource allocation within the brain (e.g., Chu et al., 2015; Dai et al., 2013). Second, individuals who are physically fit or active tend to be more efficient in the use of neural resources than those who are less fit or active (e.g., Hillman, Belopolsky, Snook, Kramer, & McAuley, 2004; Kamijo et al., 2010). However, Pesce (2009) argues that an increase in resource allocation may not necessitate an improvement in cognitive performance. Indeed, studies either failed (e.g., Hillman et al., 2004; Kamijo et al., 2010) or found some associations (e.g., Dai et al., 2013; Drollette et al., 2014) between resource allocation or efficiency and exercise-induced cognitive enhancement. Hence, there is only partial support for the cognitive-energetic models, suggesting that other factors are involved in the mechanism between physical exercise and cognition.

Similar to the neurobiological models, cognitive psychological models, such as the cognitive-energetic and the strength models of self-control, indicate that there is no single mechanism that can account for every aspect of the exercise and cognition relationship. For instance, the extended strength model of self-control proposed by Audiffren and André (2015) included various cognitive hypotheses, such as the conservation and persistence hypotheses, and also indirectly adopted the dopamine hypothesis from the neurobiological

models to explain the positive emotion induced by physical exercise that led to a cognitive improvement. Indeed, researchers exploring the mechanism by which exercise affects cognition have begun to acknowledge that there is no single pathway responsible for this relationship (Audiffren, 2009; Audiffren & André, 2015; Dietrich & Audiffren, 2011; Lambourne & Tomporowski, 2010; Tomporowski et al., 2011), and that the mechanism differs for acute and chronic exercises, and whether the cognitive tasks are conducted during or after the exercise activity.

Although a detailed discussion of the various neurobiological and/or cognitive psychological models is beyond the scope of this thesis, there is a need to note that other models explaining the mechanism of exercise and cognition exist, such as the reticular-activating hypofrontality (RAH) model (Dietrich & Audiffren. 2011). However, as this research project is on the post-exercise effect on cognition, the RAH model is not elaborated in this chapter since the model is focused specifically on the cognitive effect measured during the acute exercise activity (i.e., dual-task condition).

In summary, the exercise-cognition research reviewed in this chapter can be categorised into those that focused on the cognitive effects of exercise and those that investigated the mechanism of the exercise-cognition relationship. Studies that have focused on effects have sought to investigate the types of cognitive function affected by physical exercise, and which factors modulate the strength of the effects (e.g., Chang, Labban et al., 2012; Pesce et al., 2009). Other studies have attempted to uncover the mechanism by which physical exercise influences cognition (e.g., Gapin et al., 2015; Winter et al., 2007). The studies that have focused on the nature of the effect that physical exercise has on cognition have resulted in four main conclusions.

First, the facilitating effect of physical exercise on cognition is selective, in that not all aspects of cognition improve. Second, the cognitive effect of physical exercise is inconsistent

among individuals and can be moderated by external (e.g., quantitative and qualitative features of exercise) and internal factors (e.g., fitness). Third, there is a likelihood that certain individuals may be resistant to the cognitive effects of physical exercise. These three points suggest that cognitive outcomes are determined not just by physical exercise per se, but also how individuals respond to the exercise activity. At present, one of the most puzzling phenomena in the field of physical exercise and cognition is the finding that some individuals do not show cognitive improvement after exercising (Audiffren, 2009). This issue is an enigma because the existing research supports the positive effect of physical exercise on cognitive functions (e.g., Kramer & Erickson, 2007; Verburgh et al., 2014). Fourth, studies in general tend to focus on the manipulation of physical exercise quantifiers over individual variables. However, it was shown earlier in this chapter that the consideration of individual differences is a complex matter, especially when there are many potential variables (i.e., physiological and psychological factors). Moreover, although some researchers recognise the need to shift the focus of research to individual differences (e.g., Diamond & Ling, 2016; Pesce et al., 2013), practical methods for doing so have yet to be explored.

Similarly, studies that have investigated the mechanism underlying the exercise and cognition relationship also suffer from difficulties in explaining why some individuals do not demonstrate cognitive improvements, despite exhibiting the necessary changes in neurochemicals or neural resources. Nonetheless, both neurobiological and cognitive psychological theories suggest that no single pathway is responsible for the exercise and cognition relationship. Taking the research findings on the effects and mechanisms together, there is a need to take into account individual differences and how these factors affect the exercise and cognition relationship. Specifically, there is a need for a practical method that allows for the measurement of individual factors without compromising external validity. Additionally, the investigation of individual differences should also be able to account for

why individuals' performance on aspects of their cognition improves or declines following exercise activity. Fractal analysis based on the theory of complexity is suggested here as a suitable candidate for investigating individual differences, which may further the understanding of the mechanism underlying the exercise and cognition relationship. The next section of this chapter provides a theoretical background and rationale for the use of fractal analysis in examining the effect of physical exercise on cognition.

Complexity Theory: Background and Rationale of Fractal Analysis

Fractal is a term coined by Mandelbrot in 1977 to describe geometric objects in nature that are of irregular and complex shapes that do not permit accurate descriptions of its physical features. The main characteristic of fractals lies in a scaling feature known as "self-similarity", where the shape of the geometric objects is composed of some unique patterns that exist similarly across many degrees of magnification.

For instance, an example provided by Mandelbrot (1980) is of the shape of coastlines. At a certain scale, the coastline has fragmented shapes, yet when the scale is further magnified regardless of the number of times, the coastline, still retains its irregular though different patterns. Fractals can also be understood in terms of data recorded over multiple time periods with self-similar properties (Pittman-Polletta, Scheer, Butler, Shea, & Hu, 2013), known as scale invariance. An example of scale invariance is shown in Figure 1 (Peng, Hausdorff, & Goldberger, 2000). The figure shows that when parts of the data are extracted from the original data set and repeatedly magnified, they display similar trends to the original data despite differences in scale. In other words, when data are said to be scale invariant, the nonlinear fluctuation pattern within data tends to be visually identical across different scales (Brown & Liebovitch, 2010).

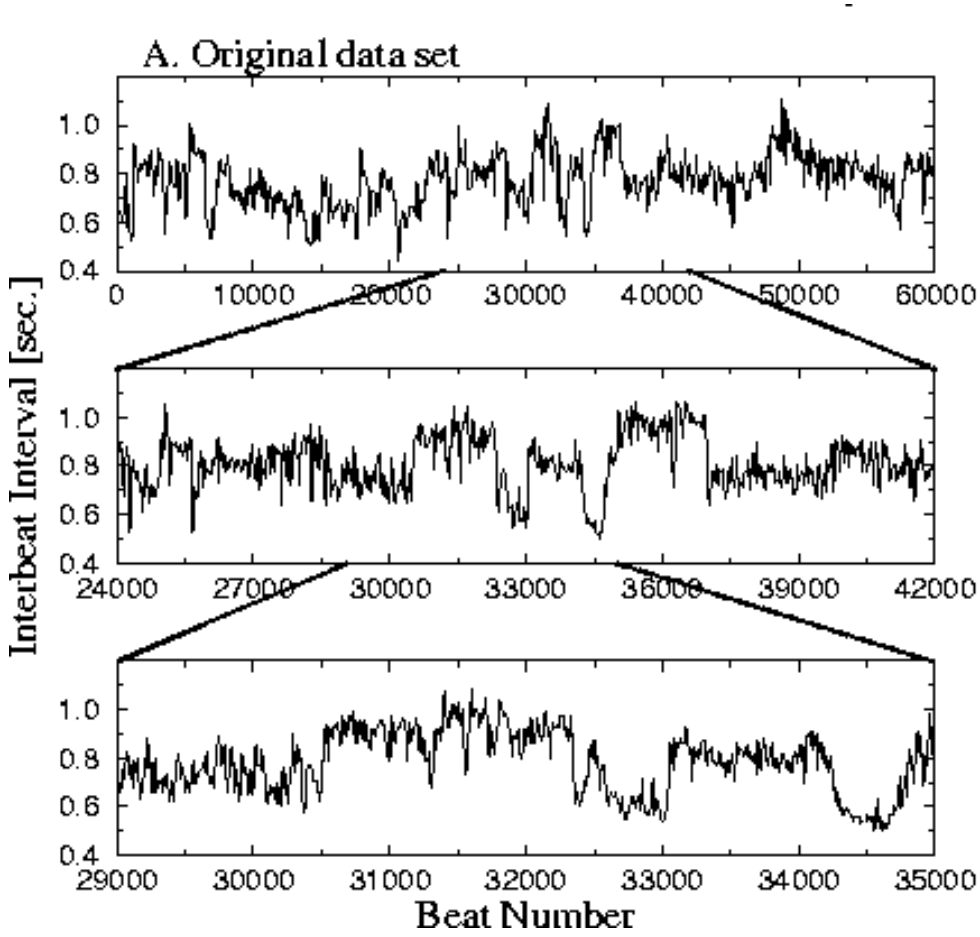

Figure 1. An example from a cardiac interbeat interval recording demonstrating the concept of scale invariance. Adapted from Mapping Real-World Time Series to Self-Similar Processes, by C-K Peng, J. M. Hausdorff, & A. L. Goldberger, 2000, Retrieved January 23, 2016, from https://www.physionet.org/. Copyright 2012 by PhysioNet.

An important implication of complexity theory (i.e., fractal) is its application to the study of human physiological systems. Contrary to the belief that a physiological system always functions in a state of regularity or balance (i.e., homeostasis), studies have demonstrated that many essential human physiological parameters, such as cardiac and respiratory functions, display data that behave non-linearly (e.g., Peng et al., 2000; West, 2006). Furthermore, research on the application of fractal analysis to the study of physiological systems has found significant differences in the way physiological data fluctuates between healthy individuals and those with medical conditions (e.g., Lee et al., 2007; Stam et al., 2005). For example, Figure 2 shows heart rate recordings over 15 minutes for a healthy individual and an individual with sleep apnoea (Goldberger, Moody, & Costa,

2012). Although at first glance, it seems logical that a healthy individual would display a regular heart rate recording, such as those in Figure 2B, this regularity of the heart rate data represents a pathological state.

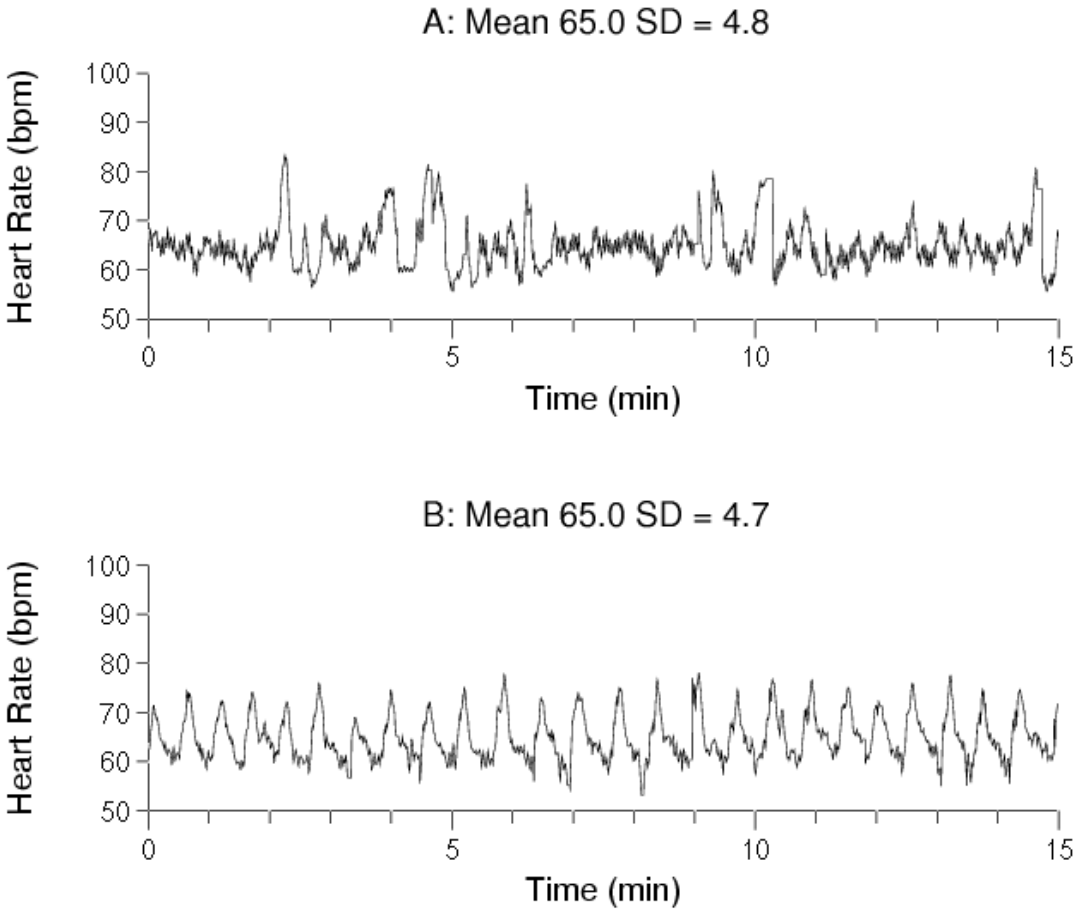

Figure 2. Heart rate measured in beats per minute over a 15 minutes period between A) a healthy individual and B) an individual with obstructive sleep apnoea. Reproduced from Variability vs. complexity, by A. L. Goldberger, G. B. Moody, and M. D. Costa, 2012, Retrieved January 23, 2016, from https://physionet.org/tutorials/cv/. Copyright 2012 by PhysioNet.

Other studies have found similar differences in fluctuation patterns in other physiological parameters among those with conditions like multiple sclerosis (Esteban et al., 2007), bipolar disorder (Indic et al., 2011), schizophrenia (Sandu et al., 2008), autism spectrum disorder (Bhat, Acharya, Adeli, Bairy, & Adeli, 2014), and attention-

deficit/hyperactivity disorder (Navascués, Sebastián, & Valdizán, 2015). The general notion from these clinical studies is that a healthy body system tends to have physiological data that are scale invariant, irregular and non-linear, also referred to as complexity (e.g., Brown & Liebovitch, 2010) or "complex phenomena" (West, 2006).

Complexity is associated with healthy functioning because a system that has complex properties is adaptive and able to fluctuate flexibly in response to external stressors (Goldberger et al., 2012). Additionally, deviations from a healthy state as a result of health conditions affect the way physiological data fluctuate (Goldberger et al., 2012). Similar to the use of the statistical mean, the quantification of complexity in a data set is indicated by its fractal dimension, which is a measure of the level of complexity or how the data fluctuate (Brown & Liebovitch, 2010). Thus, Figure 2A would have a higher fractal dimension indicating complexity (i.e., healthy) as compared to Figure 2B where the fluctuation is more regular and less complex (i.e., sleep apnoea). Therefore, the level of complexity in the data, indicated by its fractal dimension, reflects the functionality of the physiological system (Brown & Liebovitch, 2010; Goldberger et al., 2012).

Briefly, fractal analysis is a way of conceptualising trends in non-linear data, especially in cases where measures of central tendency (e.g., the mean) cannot provide a good representation of the observed data (Brown & Liebovitch, 2010). For instance, Figure 2A and 2B have the same mean and similar standard deviations, but relying only on these statistical measures would naturally lead to the conclusion that there is no significant difference between both sets of recordings. This example highlights the limitation of relying solely on the mean (e.g., Speelman & McGann, 2013; West, 2006), leading to the masking of other potentially important information within the data (i.e., fluctuation pattern). Given that fluctuations within data are the product of many interactions among the components that make up the system (Brown & Liebovitch, 2010), examining fractal dimension in addition to

standard parametric analysis provides further information about the underlying processes of the physiological system.

Fractality and Neurodevelopmental Disorders

Recent studies have investigated fractality in physiological parameters as a potential diagnostic indicator in distinguishing healthy individuals from individuals with ASD and ADHD (e.g., Bhat et al., 2014; Bosl, Tierney, Tager-Flusberg, & Nelson, 2011; Ghassemi, Moradi, Tehrani-Doost, & Abootalebi, 2012; Lai et al., 2010; Li et al., 2007; Navascués et al., 2015). Bosl et al. (2011) examined the level of complexity of brain development based on nonlinear analysis of EEG signals. This analysis showed that there are significant differences between healthy infants and those that are at risk of developing ASD (i.e., having siblings diagnosed with ASD). Specifically, the authors reported that the brain development between at-risk and healthy control groups have a similar trajectory in terms of the changes in the level of complexity from 6 to 18 months old. However, at-risk infants have generally less complex EEG signals particularly in the left frontal region compared to healthy participants, especially between the ages of 9 to 12 months. In addition, another study also found lower complexity only in brain areas associated with ASD (e.g., inferior frontal gyrus) in 30 ASD male adults compared with 33 healthy male participants (Lai et al., 2010).

Similarly, the results from studies with ADHD children also reported lower levels of complexity in comparison to healthy participants in the left (Li et al., 2007) and right prefrontal regions (Navascués et al., 2015), which are areas that are commonly reported to be associated with the disorder (Halperin & Schulz, 2006). Together, the findings of both ASD and ADHD studies show that complexity is lower in individuals with neurodevelopmental disorders than those with typical development, especially in brain areas that are typically implicated in the respective disorders. These studies indicate the value of examining fractality in physiological data in ASD and ADHD populations.

Fractality and Exercise

Interestingly, the level of complexity of a physiological system can also be influenced by physical exercise. Studies investigating the fractality and exercise relationship have mostly involved cardiac and respiratory systems (e.g., Bardet, Kammoun, & Billat, 2012; BuSha, 2010; West, Griffin, Frederick, & Moon, 2005). Bardet et al. (2012) examined the fractal behaviour of heart rate variability in nine healthy athletes without cardiac conditions during a marathon. The authors found changes in fractality related to fatigue towards the end of the marathon that are similar to individuals with cardiac issues. In another study of the effects of mild and moderate exercise level (i.e., cycling) on fractality, BuSha (2010) reported changes in fractal patterns in respiratory and cardiac parameters. Specifically, exercise reduces fractal behaviour on the respiratory measure but increases fractality on the cardiac parameter. These exercise-fractality studies demonstrate that there are differences in complexity between basal and exercise conditions (Karasik et al., 2002), supporting the notion that exercise affects the fluctuation patterns in physiological measures.

Importantly, the investigation of exercise-induced changes in fractality of physiological systems has provided some insight into its underlying mechanism (e.g., cardiac dynamics). For example, Ivanov, Amaral, Goldberger, and Stanley (1998) established a general model in accounting for the scale invariant, irregular and non-linear fluctuations observed in physiological systems. The model is based on multiple sources of "stochastic feedback", also known as "attractors" (West, 2006) within a physiological system that differentially attract a specific parameter (e.g., cardiac interbeat interval) towards certain values in opposite directions, resulting in erratic fluctuations (Ivanov et al., 1998). These attractors function to limit the range of values within which the specific physiological parameter can vary, and the limiting range changes according to time and external stimulus (Ivanov et al., 1998).

In another study of fractality changes of cardiac dynamics during rest and exercise conditions, Karasik et al. (2002) extended the work of Ivanov et al. (1998) on stochastic feedback of physiological systems, and concluded that the differences in fractality are a result of competition within the autonomic nervous system to accelerate (i.e., sympathetic nervous system) or decelerate (i.e., parasympathetic nervous system) the heart rate. The authors further explained that the data fluctuation as revealed by the fractal analysis is larger at rest when both the opposing nervous systems are operating but becomes smaller because of the singular effect of the sympathetic nervous system that occurs during exercise. Together, these studies have shown the value of investigating fractality in contributing to the understanding of the dynamics of physiological systems, but which is unavailable through standard parametric statistics (Brown & Liebovitch, 2010; West, 2006).

Fractal Analysis: Detrended Fluctuation Analysis

A type of fractal analysis that has been used extensively in the study of many biological systems is detrended fluctuation analysis (DFA; Peng et al., 1995). DFA is particularly useful to the investigation of physiological parameters recorded over a specific time period (i.e., time series) because it can prevent artificial results arising from data that are highly non-linear and irregular (i.e., non-stationary data). The DFA generates a fractal dimension that reflects the level of complexity. This output provides an indication of the functionality of the system under study.

$$y(\mathrm{k}) = \sum_{i=1}^{k} [x_i - \bar{x}] \qquad (1)$$

In Equation 1, y(k) is an integrated time series which is the summation of specific physiological data recorded over a period of time, where every individual value is subtracted by its mean. The integrated time series (i.e., x-axis) is then segmented equally into *n* bins, with each bin of data fitted with its own line of best fit (i.e., method of least squares). Individual *n*th bin is then detrended (see Equation 2) by taking the difference between the

y(k) and the y-coordinate of the fitted line in each bin, y_n(k); this process is calculated for all bins. A log-log graph is then plotted to examine the relationship between each bin size (n) and the mean fluctuation, F(n). The slope of this relationship on a log-log graph is represented by the scaling exponent, α (see Figure 3).

$$F(\mathrm{n}) = \sqrt{\frac{1}{N}\sum_{k=1}^{N}[y(k) - y_n(k)]^2} \qquad (2)$$

As mentioned earlier in the chapter, scale invariance is a property of fractals, and another related property is the existence of a power law distribution (Brown & Liebovitch, 2010). A power law is a type of distribution that is scale invariant and it is observed as a linear line when plotted on log-log axes (see Figure 3). A power law distribution indicates that smaller values are more frequently observed than larger values (Brown & Liebovitch, 2010). In DFA, the scaling exponent α represents the fluctuation pattern of the correlation between the size of the nth bin and the mean fluctuation across time, and ranges between 0 to 2.0 (Stroe-Kunold, Stadnytska, Werner, & Braun, 2009).

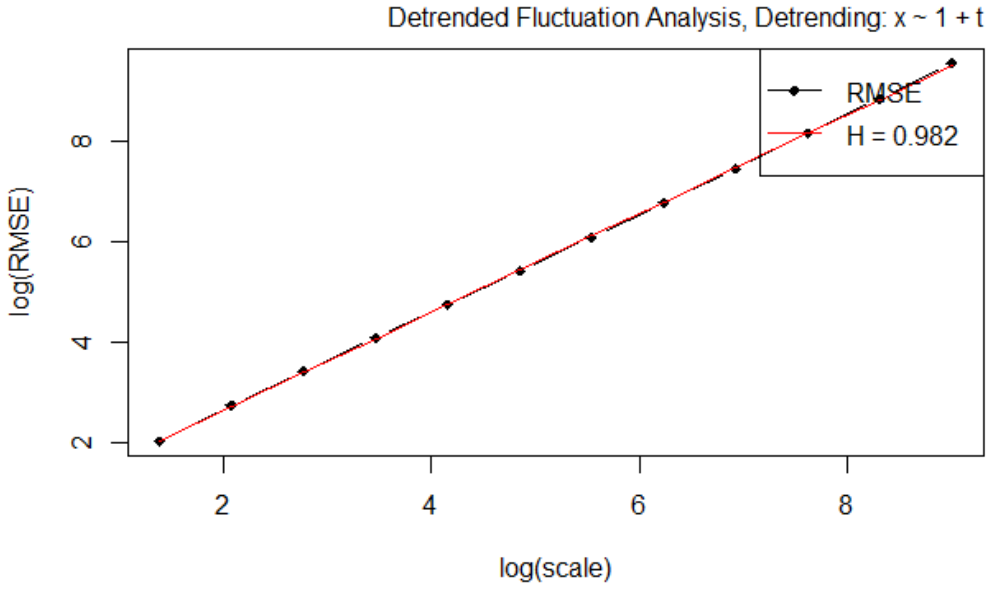

Figure 3. An example of a power law distribution on a log-log plot. *Note*. The scaling exponent on this log-log plot is represented by the letter H. Since the scaling exponent is approximately 1.0, this time series is also an example of a 1/f noise.

There are three types of time series data that can be categorised and described by the scaling exponent α (see Table 2). The scaling exponent α corresponds to the types of data signal that lie between white and brown noise (see Figure 4) (e.g., Gisiger, 2001; Halley & Kunin, 1999; Kantelhardt, 2008; Peng et al., 1995; Stadnitski, 2012). Specifically, a key characteristic of the various types of data signal is the strength of the correlation of the data variables decaying across time. A white noise type of time series fluctuates randomly and does not rely on previous values. Thus, a white noise is an uncorrelated time series which is represented by an α equal to 0.5. In contrast to white noise, the values in a brown noise time series are influenced by its closest previous value in addition to some random variations, leading to a correlation with no decay. A brown noise time series is represented by a scaling exponent of around 1.5. Unlike white and brown noise, 1/f noise is a unique type of time series that features the characteristics of both white and brown noise. Furthermore, 1/f noise follows a power law that decays very slowly and it is scale invariant. The 1/f noise is represented by a scaling exponent of 1.0 (see Figure 3). On the whole, the value of α shows the type of correlations in the data and also indicates the regularity of the time series (see Figure 4). In other words, the lower the value of α (i.e., towards a white noise), the more irregular the fluctuations. Alternatively, the higher the value of α (i.e., towards a brown noise), the more regular the time series.

Table 2

Types of Data Signal and Their Characteristics

Types of data signal	Other names	Correlation	Scaling exponent, α (DFA)
White noise	Random noise	Uncorrelated	0.5
Pink noise	1/f noise; flicker	Long-term correlated	1.0
Brown noise	Brownian noise; random walk	infinite	1.5

Note. Information in this table are obtained from Gisiger (2001) and Stadnitski (2012).

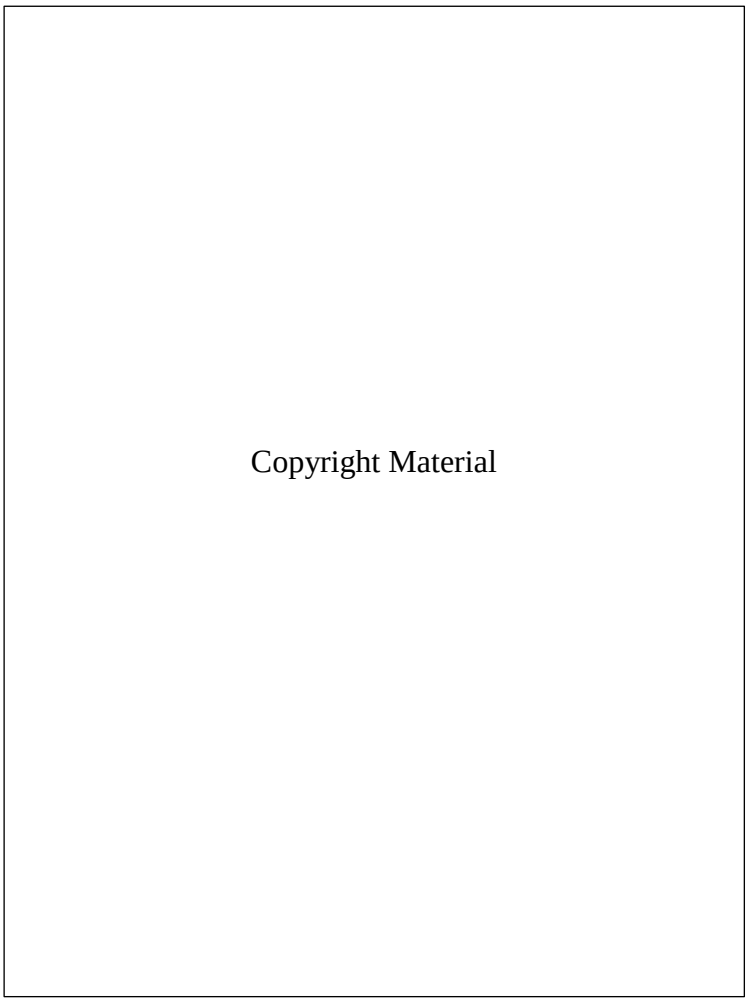

Figure 4. An example of the types of data signal that can be detected with detrended fluctuation analysis. Reprinted from "Scale Invariance in Biology: Coincidence or Footprint of a Universal Mechanism," by T. Gisiger, 2001, *Biological Review, 76,* p. 168. Copyright 2001 by the Cambridge Philosophical Society.

The presence of 1/f noise can be clinically important. A healthy body system tends to have a scaling exponent around 1.0, which corresponds to 1/f noise that carries long-term power law correlations. In contrast, pathological states tend to have data patterns resembling those of white or brown noise (Peng et al., 2000). This breakdown of complexity (i.e., deviations from 1/f noise) being associated with pathological conditions has been supported in various clinical studies (e.g., Esteban et al., 2007; Sandu et al., 2008; Zhang et al., 2013) and represents dysfunction in the ability of the body system to adapt and respond to external stressors (Goldberger, Peng, & Lipsitz, 2002).

Fractality and Cognition

Recent advances in brain and cognition studies have also begun to examine fractality in intracranial EEG (He, Zempel, Snyder, & Raichle, 2010), magnetoencephalography and extracranial EEG (Linkenkaer-Hansen, Nikouline, Palva, & Ilmoniemi, 2001), and brain imaging (Esteban et al., 2007; Mustafa et al., 2012; Sandu et al., 2008). Barnes, Bullmore, and Suckling (2009) administered two versions of a working memory task (i.e., n-back) that differed in terms of their level of task demand and used functional magnetic resonance imaging in 14 healthy adults aged between 21 and 29 years. Barnes et al. reported a transient reduction in the level of fractality during cognitive tasks followed by a gradual return of complexity to baseline level at post-test. Interestingly, greater task demand was associated with a longer rate of recovery compared to the easier task. In another study of brain function and the developmental trajectory of human cognition, fractality was found to be associated with fluid intelligence at a young age and correlated with less cognitive decline as a result of aging (Mustafa et al., 2012).

The research described in this section suggests that fractality is a common property of the brain and cognitive functions (Kello, Beltz, Holden, & Van Orden, 2007; Kello et al., 2010). In particular, 1/f noise has been implicated in normal functioning of the

neurophysiological networks (Wijnants, Cox, Hasselman, Bosman, & Van Orden, 2013) and is consistent with the idea that a breakdown of complexity (i.e., deviations from 1/f noise) is related to abnormal processes (Goldberger et al., 2002), such as Alzheimer's disease (Yang et al., 2013). For example, in a study of 108 geriatric participants with Alzheimer's disease using EEG, deviations from complexity were found to be associated with symptom severity and poorer cognitive performance, particularly in the occipital-parietal regions (Yang et al., 2013).

Application to Physical Exercise and Cognition

Based on the exercise-cognition research reviewed in the earlier sections of this chapter, there is a need to shift the focus of research to incorporate individual differences to the exercise and cognition relationship. However, the current research literature has not provided a method for effectively examining individual factors, particularly in the context of many variables. Given that it is impractical to investigate every known individual variable, fractal analysis may be an efficient way to summarise the functionality of the physiological system. Consequently, examining fractality may also offer some insight into how an individual would respond to the cognitive effect of physical exercise.

The use of psychophysiological measures, as indexed by the scaling exponent, may provide a different perspective on how a change in the physiological system may contribute to the exercise-cognition relationship. As fractal analysis or the theory of complexity is a study of system dynamics, when applied to the exercise-cognition relationship, the analysis of a specific psychophysiological measure (e.g., galvanic skin response) would ipso facto reflect at least some of the individual factors within the physiological system (e.g., arousal system) that regulates the specific psychophysiological parameter. In other words, theoretically, fractal analysis may simplify the large number of individual factors into a single scaling

exponent, which may be further analysed for its ability to account for the exercise and cognition relationship.

Although the representation of individual differences by a scaling exponent may be a simplification of the complexity of the individual factors, the investigation of the scaling exponent would provide an alternative perspective and avenue for further research. First, as discussed earlier in this chapter, there are significant challenges to include the many individual factors when investigating the exercise-cognition relationship. Thus, there is a need for a practical method in synthesising many individual factors and fractal analysis may be a potential candidate.

Second, investigating the scaling exponent may also circumvent the confounding issue of evaluating some of the individual factors, such as fitness levels. The traditional process of evaluating fitness requires individuals to perform some physical exercises before the experimental phase, which may confound the experimental and control conditions (i.e., all participants performed the exercises during the fitness test). Fractal analysis can be used to differentiate between healthy individuals and those with medical conditions based on how certain physiological data fluctuates (e.g., heart rate variability). Thus, fitness levels can likely be derived based on the fractal analysis of physiological data without the need for a fitness test.

Third, as mentioned earlier, the research on the effects and mechanisms of physical exercise on cognition is restricted by the existence of some individuals whose cognition do not seem to be affected by physical exercise. Since fractal analysis can detect the functionality of an individual's physiological system based on the characteristics of the data signal (e.g., white noise), the investigation of psychophysiological measures based on the fractal analysis may account for some of the inconsistences in the magnitude of the exercise-induced cognitive effects reported in previous research.

Summary

The purpose of this chapter was to explore the literature on the effects of physical exercise on cognition, and the possible mechanism responsible for these effects. In addition, this chapter also proposed the investigation of individual differences based on fractal analysis to further understand the relationship between exercise and cognition. The theoretical background and rationale for the proposed fractal analysis to the study of psychophysiological measures was also presented in this chapter.

In summary, the fractal dimension (i.e., scaling exponent) has been widely investigated and has contributed largely to the understanding of human physiological systems. In particular, the establishment of complexity as an indicator of a healthy body system and the breakdown of complexity in relation to disease processes has important clinical implications. Additionally, the existence of fractality in physiological parameters that can be influenced by neurodevelopmental disorders, physical exercise and cognitive tasks further strengthens the value of examining fractality in the exercise and cognition research. On the whole, it is theoretically sound to study the exercise and cognition relationship based on the complexity theory or specifically, fractal analysis. Before proceeding to explore how fractal analysis can contribute to the exercise and cognition relationship, the next chapter addresses the first aim of this project, which is to review the research literature to understand the efficacy of exercise interventions on cognition in the neurodevelopmental population.

Chapter 2: A Meta-Analytic Review of the Efficacy of Physical Exercise Interventions on Cognition in Individuals with Autism Spectrum Disorder and ADHD

The purpose of this chapter is to determine the efficacy of exercise interventions in individuals with neurodevelopmental disorders. Additionally, this chapter also aims to link the research with this clinical population with the research reported on the general population. This chapter was published in the Journal of Autism and Developmental Disorders (Tan, Pooley, & Speelman, 2016).

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To conclude, this chapter has fulfilled the first aim of this research project in understanding the exercise and cognition relationship. Specifically, this chapter has examined the efficacy of exercise interventions in enhancing cognition in young individuals with a neurodevelopmental condition, and has connected the research with this clinical population with research conducted on the typical developing population. Based on the exercise-cognition studies reviewed in Chapter 1 and 2, the number of individuals who would respond to the cognitive effect of exercise remained unknown. Therefore, the effect of exercise on cognition in children with and without a neurodevelopmental condition needs to

be further evaluated. The investigation of the exercise effect on cognition leads to the second and third aims of this project. The second aim of this project is to compare the after-effect of an acute exercise activity against a cognitively engaging tablet game activity on implicit learning and attention in children with and without a neurodevelopmental condition. Finally, the third aim of this project is to conduct psychophysiological investigation based on the proposed fractal analysis introduced in Chapter 1 to determine if individual differences are able to account for the cognitive effect of an acute exercise activity. The following chapter (i.e., Chapter 3) is the beginning of the experimental part of this research project. Chapter 3 provides details of the methodology used in the experimental study and psychophysiological investigations, with the results presented in Chapter 4 and 5, respectively.

Chapter 3: Overview of the Methodology

This chapter describes the experimental and psychophysiological methodologies that were designed to investigate the after-effect of an acute physical exercise in children with and without a neurodevelopmental condition. Overall, this project manipulated both between- and within-subject variables to investigate the after-effects of an acute physical exercise in comparison to a tablet game activity, on measures of implicit learning and attention in children with a neurodevelopmental condition and those with a typical development. This project used two types of measurement, cognitive tasks and psychophysiological measures. The results collected with these measures are presented separately in the next two chapters. The cognitive tasks included an implicit sequence learning task and a modified attention network test. As for the psychophysiological measures, galvanic skin response (GSR) and electroencephalogram (EEG) were measured.

Participants

This study recruited 48 children aged 6-11 years. Participants were recruited from advertisements (Appendix A and B) and information sheets (Appendix C and D) posted around Edith Cowan University campuses, the University's psychology clinic, an online student newsfeed, in a local community printed and online newspaper, and on the website of various organisations and centres that provide services to children with neurodevelopmental disorders in Perth, Western Australia. Participants were assigned either to the typical developmental group or neurodevelopmental group according to the initial ASD and ADHD screening questionnaires. Initial group assignment resulted in 22 children in the typical developmental group and 26 children assigned to the neurodevelopmental group. Out of 26 children in the neurodevelopmental group, 13 children were previously diagnosed by their healthcare provider (e.g., paediatrician) as having ASD (n = 6), ADHD (n = 4), and combined ASD and ADHD (n = 3).

As mentioned in Chapter 2, though ASD and ADHD are diagnosed separately in clinical settings, symptoms that overlap between these two disorders are not uncommon. Indeed, in this study, according to the results of the Autism Spectrum Quotient – child version (AQ-10; Allison, Auyeung, & Baron-Cohen, 2012) and Conners 3rd edition ADHD index form - parents (3AI-P; Conners, 2008), 85% of those children that were previously diagnosed with ASD and/or ADHD also reported a significant number of symptoms overlap between these disorders (i.e., scores above the cut-off point). Thus, children with ASD or ADHD were assigned to the neurodevelopmental group. This group assignment, however, does not suggests that ASD or ADHD is the same disorder. Rather, the assignment of both disorders to the same group is to acknowledge the high comorbidity shared between ASD and ADHD (e.g., Gargaro et al., 2011; Joshi et al., 2014).

Based on the inclusion criteria, children needed to present with no major physical or visual disabilities, no anticipated change in medication regime (if any) over the course of the experiment, and could participate in moderate-intensity physical exercises. Additionally, children also needed to be capable of complying with the research protocol, and would need to demonstrate an IQ equal to or greater than low average (i.e., ≥80), as assessed and categorised by the Woodcock-Johnson III: Brief intellectual ability (Woodcock, McGrew, & Mather, 2001). Out of the 48 children, 1 child was included in the pilot phase to test the program and research sequence, and 4 parents/children withdrew from the study as a result of personal commitments. Furthermore, 3 child participants failed to meet the minimum required IQ level, and 2 children were unable to complete the computer tasks due to behavioural issues. Lastly, the data from 1 participant were excluded due to a third-party interference resulting in data contamination (i.e., the research sequence was disrupted). In total, 37 children were included in the main analyses. Specifically, 17 participants were in the neurodevelopmental group (ASD/ADHD) and 20 participants in the typical developing (TD)

group. A summary of the children's demographic variables including, age, year of study, weight, height, body mass index (BMI), physical activity level in a typical week (i.e., rated from 1-10, least to most active) and medication/supplement is presented in Table 9.

Children in the neurodevelopmental and TD groups did not significantly differ in age, year of study, IQ, and physical activity level (Table 9). However, significant differences were observed for weight, height and BMI between both developmental groups. On average, children in the neurodevelopmental group had larger values in weight, height and BMI, compared to children in the TD group. Additionally, the scores derived from the ASD and ADHD questionnaires (see section on Materials) were significantly higher in children with ASD/ADHD than those with TD, supporting the validity of the group assignment.

Table 9

Means, Standard Deviations and Mann-Whitney U Test Summary for Participant Characteristics in the Two Diagnostic Conditions

	ASD/ADHD (SD)	TD (SD)	*U*	*z*	*P* (2-tailed)
Sample size	17	20			
Male: Female	11: 6	13: 7			
Mean age	8.06 (1.68)	7.70 (1.34)	151.00	-0.59	.56
Mean study year	3.00 (1.66)	2.50 (1.28)	143.00	-0.85	.41
Mean weight	33.77 (9.18)	27.44 (6.46)	93.00	-2.35	.02*
Mean height	1.39 (0.10)	1.32 (0.09)	99.00	-2.15	.03*
BMI	17.30 (3.35)	15.59 (2.46)	105.00	-1.98	.05*
WJ III BIA	100.35 (8.80)	99.85 (12.91)	147.50	-0.69	.50
AQ-10[#]	3.88 (2.62)	1.80 (0.95)	86.50	-2.60	.004*
Conners (T-score)[#]	87.76 (4.15)	51.55 (6.85)	0.00	-5.26	<.001*
Conners (Probability)[#]	89.71 (11.00)	30.40 (15.50)	0.00	-5.22	<.001*
PA level (1-10)[^]	6.88 (1.32)	6.84 (1.57)	161.50	0.00	.51
Methylphenidate (i.e., Concerta)	3	-			
Bronchodilators (e.g., Ventolin)	2	2			
Vitamins/Supplements (e.g., Vitamin B and C, Zinc, Omega 3, Probiotics)	5	5			
Homeopathic Remedy	1	-			

* Statistical significance set at $p = .05$. [#] Exact one-tailed significance.
^ One missing value for the baseline level of physical activity in the healthy group.
BMI – Body mass index, WJ III BIA – Woodcock Johnson III: Brief Intellectual Ability Test, AQ-10 – Autism Spectrum Quotient (Child version, Short Form), Conners – 3rd Edition ADHD Index Form (Parents; 3AI-P), PA – Physical activity.

Materials

The material section was separated into those used for the initial screening and others that were used during the experiment. The initial screening included the ASD and ADHD questionnaires, and the Woodcock-Johnson III. In addition, cognitive tasks and psychophysiological measures were used during the experimental phase.

Screening tools.

Autism Spectrum Quotient – Child version (AQ-10).

Children were evaluated with the Autism Spectrum Quotient – child version (AQ-10; Allison et al., 2012) to screen for symptoms of ASD among those aged 4-11 years (Appendix E). The AQ-10 is a brief parent-rated questionnaire consisting of 10-items, assessing areas of communication, imagination, attention switching, attention to details, and social skills. In terms of its psychometric properties, AQ-10 was reported to have high internal reliability (Cronbach's alpha = .90), and validity. Each item on the AQ-10 was rated on four responses, 'definitely agree' to 'definitely disagree'; a score of 1 was given to items rated as definitely or slightly agree, while items with definitely or slightly disagree were scored as 0. The highest possible overall score for the AQ-10 is 10 and the cut-off of 6 or more indicated the need for further clinical evaluation (Allison et al., 2012). For this study, children who scored 6 or more were assigned to the neurodevelopmental group.

Conners 3rd edition ADHD index form – Parents (3AI-P).

To screen for symptoms consistent with ADHD, children were assessed with the Conners 3rd edition ADHD index form for parents (3AI-P; Conners, 2008). Conners 3AI-P is a 10-item parent-report questionnaire that can differentiate individuals aged 6-18 years with ADHD from the typical population (i.e., good reliability above .84). Each item was rated on a four-point Likert scale from 0-3 (Not true at all, to Very much true) examining behaviours observed in the past month. The total raw score of the questionnaire has a range of 0-20 and

was converted into both a probability and a T-score based on the conversion table provided on the scoring sheet. The probability score ranged from 11-99% and provided an indication of how likely an individual was to be diagnosed with ADHD. The T-score assessed whether the level of symptomatology presented was typical of an ADHD population in respect to age and gender. Overall, the higher the scores obtained, the greater the likelihood of an ADHD diagnosis. For this study, children were assigned to the neurodevelopmental group if both the probability and T-score were elevated (i.e., ≥ 60).

Woodcock-Johnson III: Brief intellectual ability – Australian adaptation.

To determine whether children met the inclusion criteria for IQ level equal to or above low average, children were assessed with the Woodcock-Johnson III: Brief intellectual ability – Australian adaptation (WJ III BIA; Woodcock et al., 2001). The WJ III BIA consisted of verbal comprehension, concept formation, and visual matching tests. The three tests assessed the cognitive domains of comprehension-knowledge, fluid reasoning, and processing speed, respectively (McGrew & Woodcock, 2001). The WJ III BIA also has an excellent median reliability of .95 for children aged 5-19 years old (Mather & Woodcock, 2001). For the purpose of this study, only children with BIA scores equivalent to low average or above (i.e., ≥80), as categorised by the WJ III BIA, were included in the experiment.

Experimental tools.

Cognitive tasks.

Implicit sequence learning task – Probabilistic (ISLT).

To evaluate implicit learning performance, children were assessed with the implicit sequence learning task (ISLT). The ISLT is a modified version of a classical serial reaction time task, and was administered to the participants through a computerised program, gSRT-Soft (Chambaron, Ginhac, & Perruchet, 2008). The ISLT was customised with four horizontal boxes on the computer screen, with each box corresponding to one of the four

keyboard letters, “Z”, “C”, “B”, and “M”. At each trial, a specific cartoon character (i.e., stimulus) from a children’s television program, ‘Adventure Time’, would appear on one of the horizontal boxes (see Figure 6). When a cartoon character was presented, participants were required to respond as quickly as possible by pressing one of the corresponding letters (i.e., “Z”, “C”, “B”, or “M”). For example, the stimulus presented in Figure 6 would require a correct response with “C”.

The sequence of the test block was constructed based on a second-conditional sequence with 85% repeated (i.e., probable trials) and 15% randomised (i.e., improbable trials) trials. For example, a string of repeated sequence (i.e., probable trials) with a 12-unit combination would be Z-B-M-C-B-Z-C-Z-M-B-C-M. The sequence was arranged such that each letter would occur with the same frequency within each test block. Random trials were included in the test block to prevent participants from relying on explicit learning processes to complete the sequence (Shanks, Rowland, & Ranger, 2005). For example, if “M” would typically occur after “Z-B” based on the probable sequence, a random trial would be arranged such that “C” could also follow “Z-B”. Hence, when participants were presented with trials “Z-B”, the next stimulus could either be “M” or “C”. Importantly, the trial sequence was not revealed to the participants. Furthermore, to reduce practice effects and to prevent participants from explicitly remembering the test sequence, two versions of the test block were administered in alternating sequence. Lastly, the response-stimulus interval was set at 250 milliseconds.

The reaction time, measured in milliseconds, and the number of correct and incorrect trials were used to assess the children’s implicit learning task performance. In addition, only correct responses and those above 100 ms were included in the analysis. Overall, the ISLT consisted of a sample block and a test block, with 8 and 100 trials, respectively. In order to ensure that children understood the task, an overall baseline accuracy of above 50% was

required to be included in the main analysis. During the analysis phase, 2 children with a neurodevelopmental condition had a baseline accuracy of below 50%, hence, their performance was excluded only from the main analysis of the ISLT. In total, the data collected on the ISLT from 35 children (15 neurodevelopmental children; 20 typical developing children) were included in the main analysis, with a baseline accuracy ranging between 61-97%.

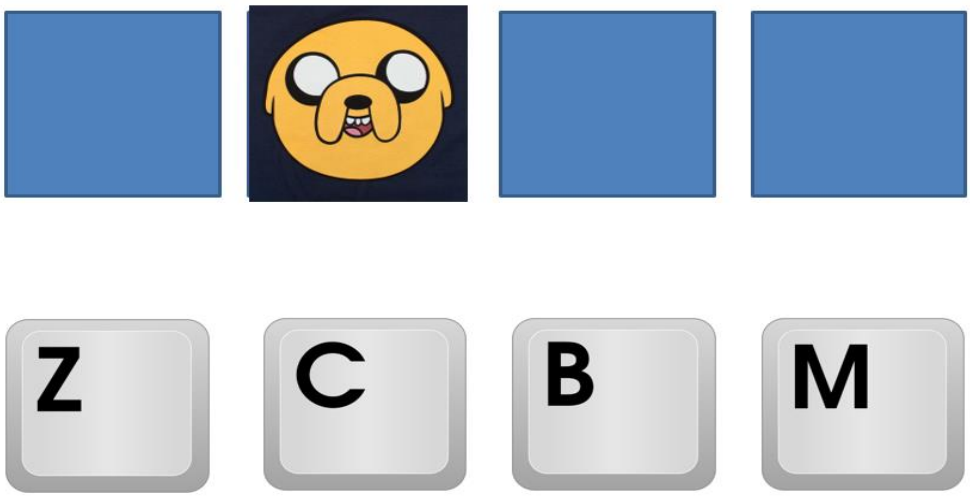


Figure 6. An example of an implicit sequence learning trial. The correct response in this trial is "C".

Modified attention network test (CRSD-ANT).

Children were assessed with a modified version of the attention network test (CRSD-ANT; Docksteader & Scott, 2013). The original ANT was developed by Fan, McCandliss, Sommer, Raz, and Posner (2002), and was based on the theory of attention proposed by Posner and Petersen (1990). The theory suggested that attention consisted of alerting, orienting and conflict networks. These attention networks are labelled as such because of the proposed relation to specific neuroanatomical sites and functions in the brain. For instance, the conflict network is associated with the frontal region and anterior cingulate cortex (Fan, McCandliss, Fossella, Flombaum, & Posner, 2005; Petersen & Posner, 2012), and involves

the neurotransmitter dopamine (Fossella et al., 2002). The alerting network is involved in the maintenance of an acute state of alertness to presented stimuli; the orienting network is involved in directing attention to the relevant sensory information, and the conflict network is responsible for the process of inhibiting irrelevant responses (e.g., Fan & Posner, 2004; Petersen & Posner, 2012).

As the original ANT child version takes approximately 25-30 minutes, a shorter modified version (i.e., 10 minutes), CRSD-ANT was used for this study. The original ANT and CRSD-ANT were found to have high reliability (Weaver, Bédard, & McAuliffe, 2013) and therefore, CRSD-ANT could also be used for investigating the attentional network. However, during the pilot phase of this study, it was observed that the pilot participant was quite restless and reported that the duration of the task was too long. In addition, the maximum duration to respond in each trial appeared to be too fast for the participant (i.e., 1500 ms), as evidenced by the number of responses exceeding the maximum reaction time.

Following consultation with the programming team at CRSD, it was recommended that the number of test blocks be reduced from three to one so as to cater to the needs of the children, particularly for those with ASD and/or ADHD. The recommendation from the CRSD was adopted and the number of test blocks was reduced to a single test block with 64 trials, which could be completed in approximately 3-4 minutes. Furthermore, the maximum response time was extended to 1700 ms, which was consistent with the original ANT-child version (Rueda et al., 2004). Other variables of the CRSD-ANT, such as the initial random fixation period (i.e., 400 to 1200 ms), cue duration (i.e., 100 ms), and interim fixation period after cue prior to stimulus (i.e., 400 ms) were maintained as per the CRSD-ANT program.

Similar to the original ANT, participants were instructed to place their left and right index finger on the left and right arrow buttons on the keyboard while keeping their eyes fixated on a cross symbol in the centre of the computer screen at all times. In each trial, a

group of five cartoon cars appeared horizontally on the screen, participants were then required to indicate on the keyboard the direction of the car in the middle (i.e., facing left or right) as quickly as possible (see Figure 7). The network scores of alerting, orienting and conflict network on the CRSD-ANT were derived using within-task subtractions between cues or flankers (i.e., less informative targets minus more informative targets). In total, there were four cue conditions (i.e., no cue, double, centre and spatial) and two flankers (i.e., incongruent and congruent) (see Figures 7 and 8).

The alerting network score was calculated by subtracting the reaction time in the double cue condition from the reaction time in the no cue condition. The orienting network score was derived from taking the reaction time in the spatial cue condition from the reaction time in the centre cue condition. Finally, the conflict score was the reaction time difference between incongruent and congruent flankers. In general, the lower the network score, the better the efficiency of the particular network. However, the interpretation is much more complicated and requires consideration of the reaction time and accuracy of each cue/flanker condition within the alerting, orienting and conflict network to better understand the findings (Fan & Posner, 2004). Hence, the network scores, mean reaction time in each cue/flanker condition and the percentage of accuracy, and commission errors were analysed. Only correct trials and reaction times between 100 to 1700 ms were included in the analysis.

Based on the research literature, a minimum overall accuracy of 70% was required to be included in the analysis (MacLeod et al., 2010). Out of the 37 participants, the ANT performance of 4 children (i.e., 3 from the neurodevelopmental group and 1 from the typical developmental group) was below the 70% baseline, and hence, the data from these children were excluded only from the CRSD-ANT analysis. Overall, 33 participants (14 neurodevelopmental children; 19 typical developing children) with a baseline accuracy ranging from 71-100% were included in the main analysis of the CRSD-ANT performance.

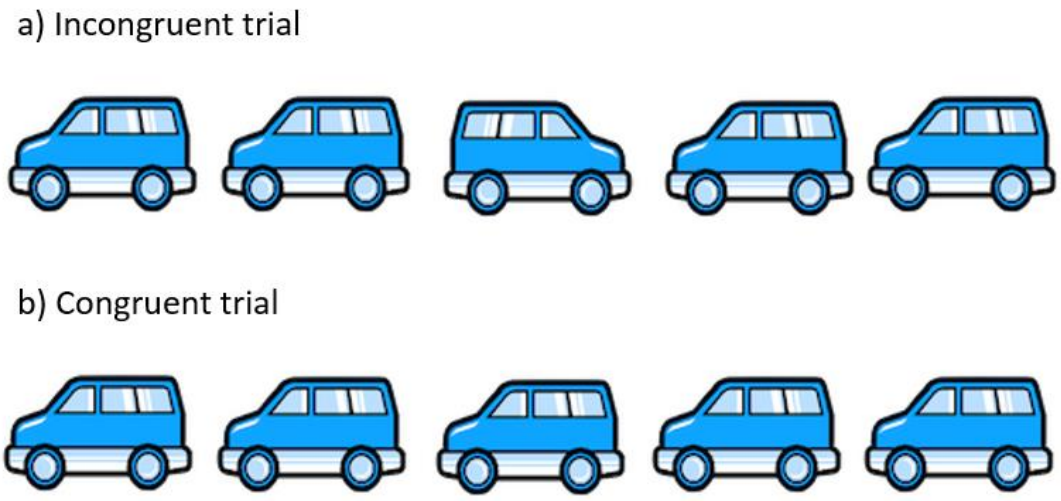


Figure 7. An example of incongruent and congruent trials/flankers in modified attention network test.

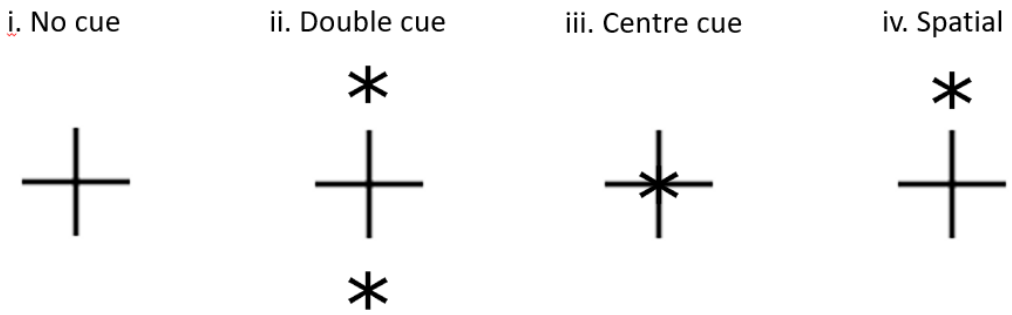


Figure 8. An example of cue conditions in the modified attention network test. Note: For spatial cue condition, the asterisk can be either above or below the cross.

Psychophysiological tools.

SenseWear armband – Pro 3.

SenseWear Pro 3 armband provided multiple physiological measurements, such as physical activity intensity level (i.e., light, moderate, vigorous and very vigorous), number of steps taken, distance travelled, galvanic skin response (GSR), skin temperature and heat flux level (BodyMedia Inc.). SenseWear Pro 3 armband and its predecessors have been found to be somewhat comparable to other validated physiological measurement instruments (see validation studies, e.g., Andreacci, Dixon, Dube, & McConnell, 2007; Dwyer, Alison,

McKeough, Elkins, & Bye, 2009; Johannsen et al., 2010). The current version of the device and its predecessors have been used in studies investigating areas including cognitive load (Haapalainen, Kim, Forlizzi, & Dey, 2010), sleep (e.g., Sharif & BaHammam, 2013); daily physical activity and energy expenditure, in non-clinical (e.g., Johannsen et al., 2010) and clinical populations (e.g., rheumatoid arthritis, see Almeida, Wasko, Jeong, Moore, & Piva, 2011; cystic fibrosis, see Dwyer et al., 2009). In addition, the SenseWear armband has also been used in children aged 3-6 years (Vorwerg, Petroff, Kiess, & Blüher, 2013), 7-10 years (Andreacci et al., 2007), and 8-11 years (Bäcklund, Sundelin, & Larsson, 2010).

In the current study, the SenseWear Pro 3 armband was attached to the dominant arm of the children for the entire duration of each experimental session (see Procedure). The armband provided an estimation of the physical intensity of the exercise and tablet activity. Sampling rate was set at 32 samples per second for the galvanic skin response. The physiological data provided by the armband was analysed with the SenseWear Professional software version 8.0 (BodyMedia Inc.).

Emotiv EPOC+.

Children's EEG data were measured with the Emotiv EPOC+ wireless headset via the TestBench software program (Emotiv Inc., 2013). The Emotiv EPOC+ records from 14 channels with 2 additional reference electrodes on the left and right mastoid area (Figure 9). Past studies using the Emotiv headset have been published in research areas, such as working memory load (Wang, Gwizdka, & Chaovalitwongse, 2015), emotions (Sourina & Liu, 2011), music intervention and depression (Ramirez, Palencia-Lefler, Giraldo, & Vamvakousis, 2015), consumer behaviour (Khushaba et al., 2013), and linguistic and perceptual processes (Louwerse & Hutchinson, 2012). In current study, EEG data were recorded only during baseline and post-intervention cognitive tasks (see Procedure). Additionally, EEG data were sampled at 128 samples per second.

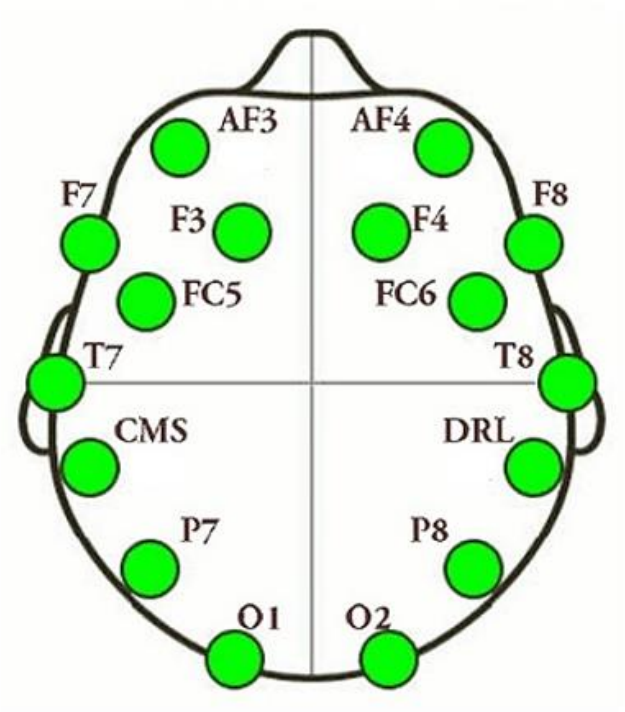


Figure 9. A top view of a head model showing the electrode sites of the Emotiv headset. Note: Common mode sense (CMS) and driven right leg (DRL) are reference electrodes located on the left and right mastoid area, respectively.

Procedure

This study was approved by the Human Research Ethics Committee at Edith Cowan University. Informed consent was obtained from the parents/guardians prior to the screening and cognitive assessment (Appendix F and G). Furthermore, verbal assent was also obtained from the children at various stages of the research. An overview of the research protocol is shown in Figure 10. The research protocol included an assessment on the first visit and another four sessions of either physical exercise or tablet activity. It should be noted that the four separate sessions of the activity sequence were counterbalanced such that children who were randomly assigned to begin the exercise activity received two sessions of the exercise activity, before switching to another two sessions of the tablet game activity. This counterbalancing of the activity sequence is also applied to children who started with the tablet activity (i.e., two sessions of the tablet activity, followed by two sessions of the

exercise activity). The sessions were about a week apart and parents were advised to ensure that their children were not given strenuous physical activities prior to each session.

At the initial assessment, parents were asked to fill in the ASD and ADHD screening questionnaires while waiting for their child to complete the cognitive assessment. The administration and scoring of the Woodcock-Johnson and screening questionnaires were conducted by the researcher. Other demographic information, such as the child's age, year of study, weight, height, physical activity level in a typical week, and current medication or supplement were also obtained from the parents/guardians. Based on the screening questionnaires (i.e., AQ-10 and 3AI-P), children were sorted into either the neurodevelopmental group or the typical developmental group. Children that were not previously diagnosed with ASD or ADHD, but obtained elevated scores on the screening questionnaires were also assigned to the neurodevelopmental group. In addition, these parents were given referrals to seek further clinical evaluation. After the initial assessment, children in both diagnostic groups were randomly assigned to either a physical exercise or tablet activity group using a selection procedure derived from values generated with the 'RAND' function in Microsoft Excel.

In the experimental phase, the SenseWear armband and the Emotiv headset were attached to the child. An initial five minutes of resting psychophysiological measures (i.e., GSR and EEG) were then recorded prior to the cognitive tasks to allow for the stabilisation of the physiological measures and also the familiarisation of the devices. The armband and headset remained attached to the child during the baseline cognitive tasks. Following baseline psychophysiological and cognitive task measurements, the Emotiv headset was removed but the armband remained on the participants throughout the session. In the intervention phase, children were given either 12 minutes of physical exercise or tablet game activity based on their intervention group assignment. After the intervention, similar to other children studies

(e.g., Best, 2012, Kamijo et al., 2011), children were provided with some water and a small packet of biscuits (i.e., Tiny Teddies) while seated for 10 minutes. Subsequently, children performed the cognitive tasks again while wearing the Emotiv headset and SenseWear armband. The sequence of the cognitive tasks (i.e., ISLT and CRSD-ANT) was counterbalanced to reduce order effects. Lastly, a $20 shopping voucher was provided to each participant for their time in participating in this research.

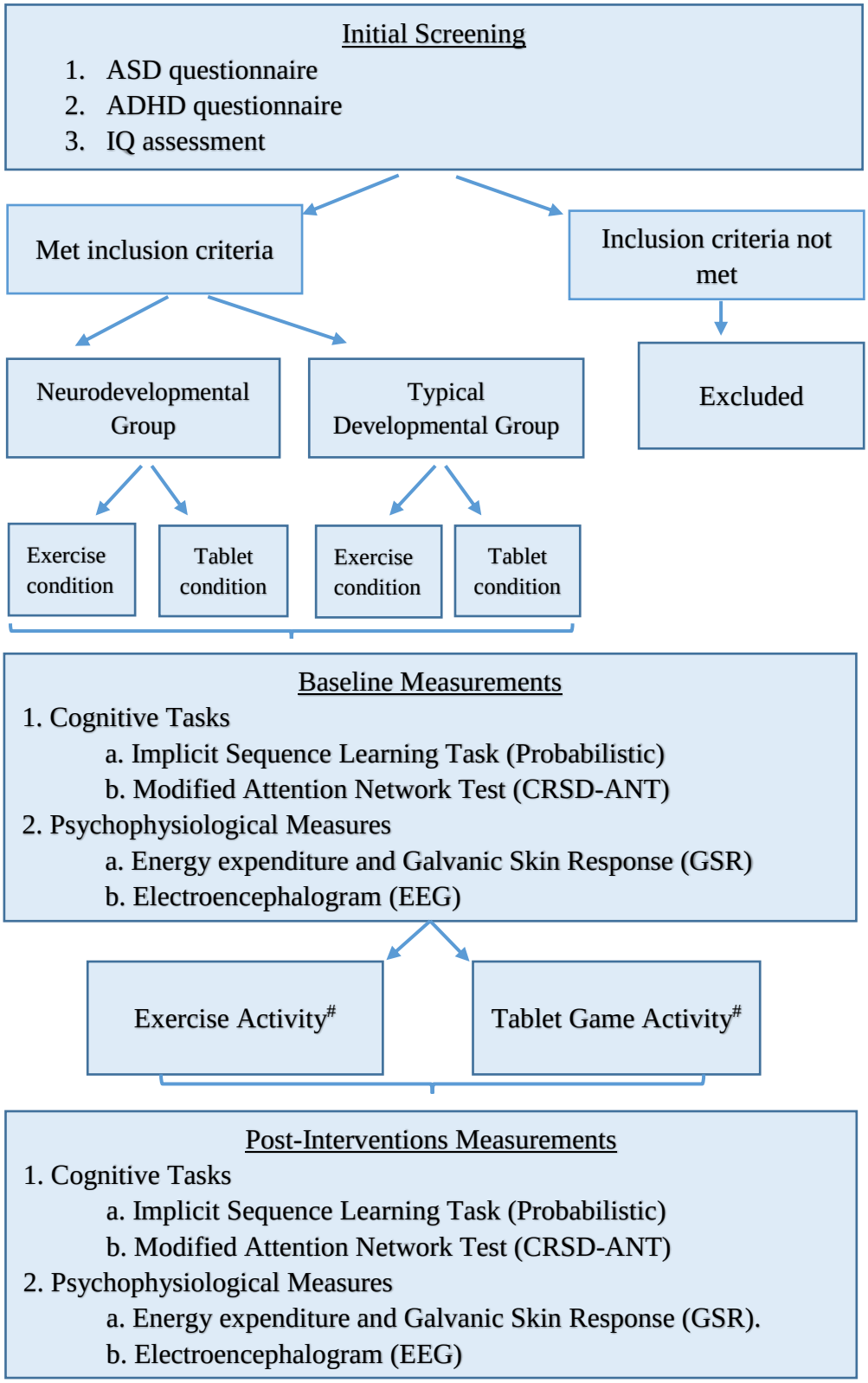


Figure 10. An overview of the research protocol. # - SenseWear armband.

Physical exercise activity (experimental condition)

Children in this condition were guided by the researcher to perform 12 minutes of moderate-intensity physical exercise individually for each session. The physical exercise was customised in a manner similar to that utilised in the study by Budde et al. (2008), which entailed a set of six coordinative movements using a basketball, with each type lasting for two minutes (see Figure 11). First, the child was instructed to bounce the basketball with his/her dominant hand while walking for two minutes, followed by the non-dominant hand for another two minutes (Figure 11a). Second, the researcher and the child stood about three metres apart and the basketball was passed to-and-fro between both individuals while in a stationary position (Figure 11b). Third, the child was asked to dribble the ball with his/her dominant hand while jogging (Figure 11c). Fourth, while walking, the child was instructed to bounce the ball alternating between both hands (Figure 11d). Lastly, both the researcher and the child stood about three metres apart. In this final activity, the child threw the ball to the researcher, then sprinted towards the researcher's position (at the same time, the researcher also sprinted to swap his position with the child), and catch the ball thrown by the researcher (Figure 11e). The process of throwing the ball, sprinting to switch position, and receiving the ball continued for up to two minutes.

Physiological data from the SenseWear armband were used to determine the exercise intensity. The physiological data for participants in the exercise condition included the total number of footsteps taken (M = 1, 491 steps, SD = 181.05) and total distance travelled (M = 2.19 km, SD = 0.25). According to the average metabolic equivalent of task (MET), participants in the exercise condition demonstrated an activity level representing a moderate-intensity physical activity (M = 4.0 MET, SD = 0.66).

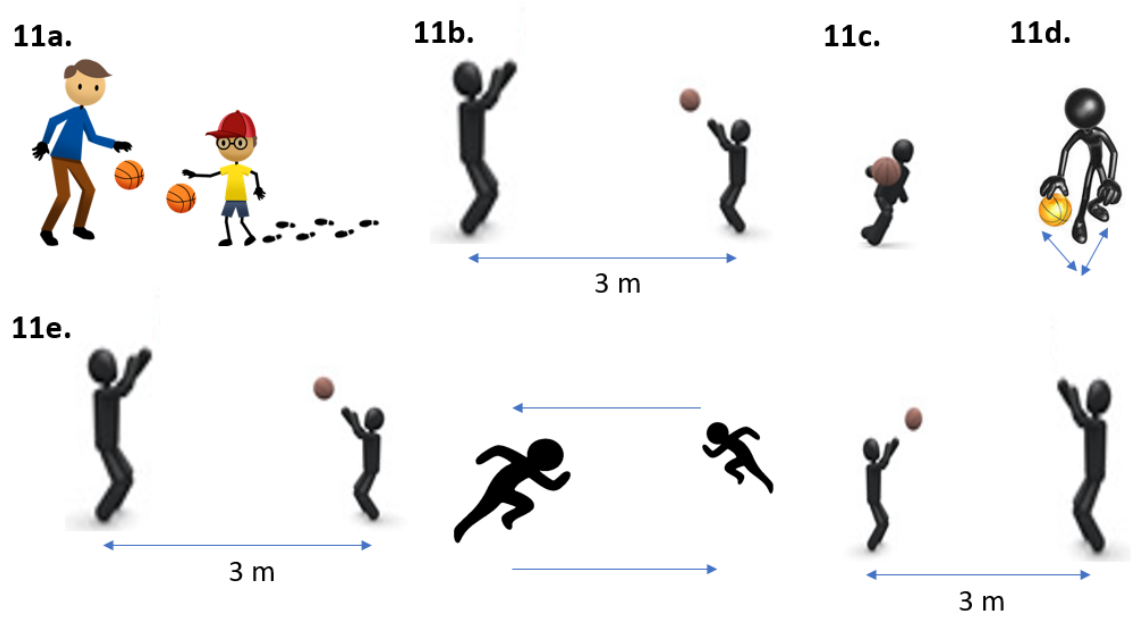


Figure 11. A pictorial form of the physical exercise activity.

Tablet game activity (control condition)

Children in the tablet activity condition were given a Samsung digital tablet to play individually on an Android game application titled, 'Call of Honey', for 12 minutes per session (see Figure 12). The application is a type of brick-breaker game that requires the player to swipe his/her finger on the screen to bounce off a ball. The goal of the game is to prevent the ball(s) from falling out of the given zone on the screen and to clear all the bricks. The difficulty of the game increases gradually as the player progresses through the stages. This game was chosen because it requires constant monitoring and attention on the ball to play the game. Furthermore, the game contains additional items that appear periodically, and the player is required to decide whether to obtain those items that could result in a positive outcome (e.g., a fireball to clear the bricks faster) or avoid those items that could result in a negative outcome (e.g., a partial blackout on the screen). Lastly, children reported that they had not previously seen or played this game.

Similar to the exercise activity, children in this condition wore the SenseWear armband throughout the tablet activity. The armband recorded the total number of footsteps taken (M = 90 steps, SD = 138.74) and total distance travelled (M = 0.05 km, SD = 0.16).

According to the average metabolic equivalent of task (MET), participants in the tablet activity condition demonstrated an activity level representing a light-intensity physical activity (M = 2.4 MET, SD = 0.43).

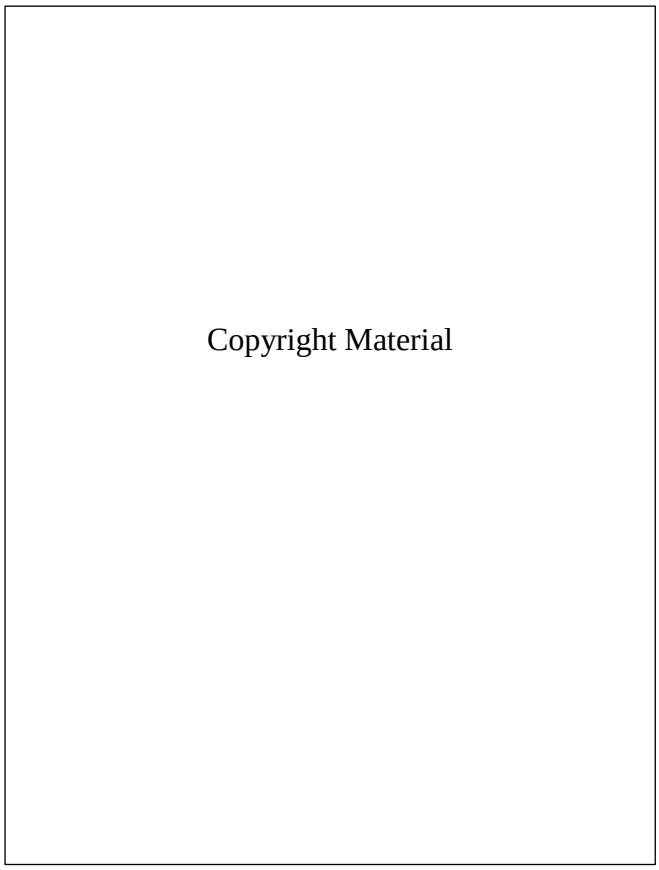


Figure 12. A screenshot of the tablet game activity.

Overview of the Statistical Analysis

Statistical analyses were conducted in two parts: cognitive performance (Chapter 4) and psychophysiological measures (Chapter 5). The alpha value across all analyses was set at .05. Although the original intention of the study was to analyse participants' cognitive performance across four counterbalanced intervention sessions to exclude the possibility of order and practice effects, preliminary analyses revealed ceiling effects on sessions 3 and 4. The ceiling effects complicated the interpretation of the results and therefore, only data collected before the change in intervention type (i.e., sessions 1 and 2) were included in the main analyses.

Summary

This chapter has provided an overview and details of the methodology adopted for the experimental phase of this project. Overall, this project contained an experimental study separated into two parts, cognitive tasks that examined the after-effects of acute exercise and the psychophysiological investigations that focused on the mechanism of acute exercise. The experimental study based on the results of the cognitive tasks included an implicit sequence learning task and a modified attention network test, of which the findings are reported in the next chapter (Chapter 4). In terms of the psychophysiological investigations, GSR and EEG data were measured and analysed based on the proposed detrended fluctuation analysis, and the results are presented in Chapter 5.

Chapter 4: Examining the Effects of Physical Exercise on Measures of the Implicit Learning and Attentional Network Tasks

The main purpose of this chapter is to test the after-effects of an acute physical exercise in comparison with a tablet game activity on measures of implicit learning and attention network. Specifically, the research questions are whether an acute physical exercise improves cognition better than a tablet activity, and whether the exercise effect is different in children with neurodevelopmental conditions compared to those with a typical development.

Statistical Analysis: Cognitive Measures

Cognitive performance was analysed with IBM SPSS 24. Multiple separate mixed-design analyses of variance (ANOVAs) were conducted on data collected with the implicit learning (ISLT) and attention network (CRSD-ANT) measures. Significant interactions were followed up with simple effects analysis to examine the interactions. Initial assumption testing on ISLT and CRSD-ANT data revealed significant violations of normality and homogeneity of variance assumptions. As winsorized means are relatively unaffected by extreme values (Wilcox, 2012, p 30-31), outliers were winsorized using Tukey hinges (i.e., 1.5*interquartile range) prior to the analysis. It is noteworthy that the analysis results based on the winsorized mean did not significantly differ from the arithmetic mean, however, the winsorized mean was selected because of improved normality and homogeneity of variance, as well as negating the need to remove outliers. The details of the assumptions testing and corrections are presented in Appendix H.

Results

Implicit Sequence Learning Task (ISLT)

Two mixed design ANOVAs were conducted on data collected from 35 participants (15 neurodevelopmental children; 20 typical developing children). One ANOVA examined reaction time, and the other analysed error scores on the ISLT. In the reaction time analysis,

the effects of two between-subject variables of intervention (exercise and tablet activity) and diagnostic group (neurodevelopmental and typical developmental), and three within-subject variables of session (1 and 2), time (pre- and post-intervention trial), and probability type (probable and improbable trial) were tested. In the error rates analysis, the effects of two between-subject variables of intervention and diagnostic groups, and two within-subject variables of session and time were tested.

Mean reaction time.

According to the ANOVA, there was a main effect of probability type on the reaction time of the ISLT, $F(1, 31) = 18.99$, $p < .001$, $r = .61$, with performance being faster on the probable trials ($M = 691.85$ms, $SD = 145.81$, 95% CI = 641.61, 742.09) than on the improbable trials ($M = 712.22$ms, $SD = 151.26$, 95% CI = 660.10, 764.33). This main effect of probability suggested the presence of sequence learning (Shanks et al., 2005), where participants learned some aspects of the sequence presented on the ISLT and thus were faster on probable trials than improbable trials. A summary of the reaction time performance across the first to the fourth administration of the ISLT is shown in Figure 13 (i.e., each administration consisted of 100 trials). In addition, the effect of probability was dependent on the interaction between session, intervention and pre/post-intervention trial, $F(1, 31) = 7.07$, $p = .01$, $r = .43$.

As can be seen in Figure 14, regardless of the type of intervention, reaction time in session 1 and 2 was faster in post-intervention trials than in pre-intervention trials, though the pre-to-post intervention improvement in reaction time was larger in session 1 than in session 2. Overall, the improvement in reaction time following an intervention was more evident in session 1 than session 2. This general difference in reaction time between sessions was supported by simple effects analyses of session within all levels of intervention, time and probability type (see Table 10). Lastly, there were no significant effects of the type of

intervention, $F(1, 31) = 0.46$, $p = .50$, $r = .12$, diagnosis, $F(1, 31) = 0.06$, $p = .80$, $r = .04$, or their interaction, $F(1, 31) = 2.18$, $p = .15$, $r = .26$.

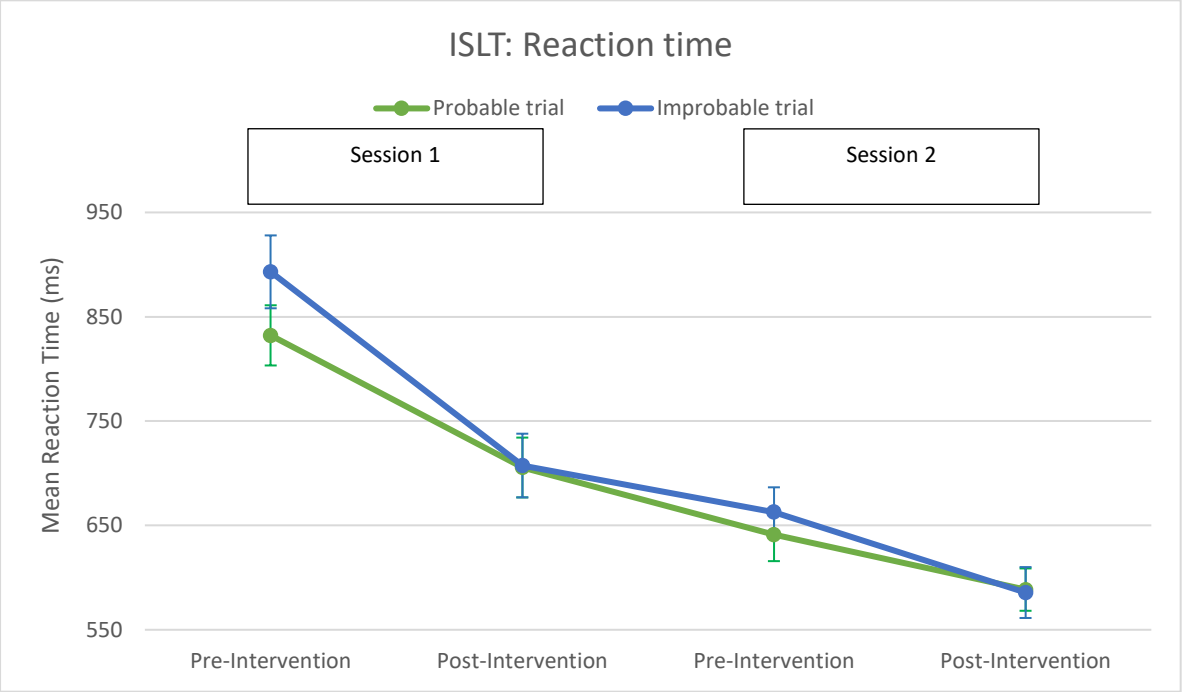


Figure 13. An overview of the mean reaction time across trials (ISLT). The error bars presented above are in standard errors.

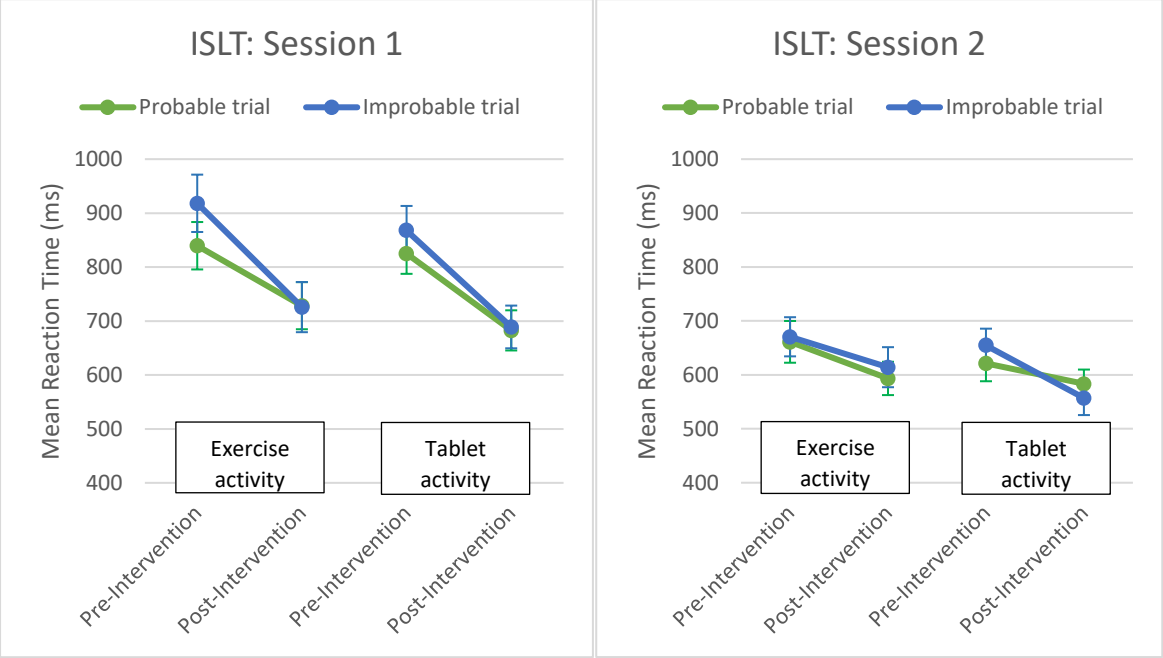


Figure 14. The effect of pre/post-intervention trial, intervention group and probability type as a function of session on the mean reaction time of the ISLT. The error bars presented above are in standard errors.

Table 10
Summary Table for Simple Effects Analysis of Session within Levels of Intervention, Time, and Probability Type on the Reaction Time of the ISLT

Source	*V*	*F*(1, 31)	*p*	*Partial* η^2
Session 1 versus Session 2				
Physical Activity				
Pre-Intervention x Probable	0.72	77.93	<.001*	.72
Post-Intervention x Probable	0.48	28.94	<.001*	.48
Pre-Intervention x Improbable	0.66	59.34	<.001*	.66
Post-Intervention x Improbable	0.25	10.33	.003*	.25
Tablet Activity				
Pre-Intervention x Probable	0.82	140.21	<.001*	.82
Post-Intervention x Probable	0.41	21.48	<.001*	.41
Pre-Intervention x Improbable	0.66	60.66	<.001*	.66
Post-Intervention x Improbable	0.39	19.91	<.001*	.39

* $p = .05$. Pillai's trace.

Mean error rate.

In examining errors on the ISLT, there was a significant difference between children in the neurodevelopmental group and those in the typical developmental group, $F(1, 31) = 4.67$, $p = .04$, $r = .36$. Children with a neurodevelopmental condition produced more errors on the ISLT ($M = 20.05\%$, $SD = 9.83$, 95% CI = 14.87, 25.24) than typically developing children ($M = 12.95\%$, $SD = 9.34$, 95% CI = 8.70, 17.20). However, there was no significant interaction between diagnostic group and intervention, $F(1, 31) = 0.34$, $p = .56$, $r = .10$. Nevertheless, there was a significant interaction effect of intervention and time, $F(1, 31) = 5.55$, $p = .03$, $r = .39$, such that the difference in the number of errors made before and after an intervention was dependent on whether children were given the physical exercise or the tablet activity. In the exercise activity group, the error rate was similar in pre-intervention ($M = 15.38\%$, $SD = 10.00$, 95% CI = 10.29, 20.47) and post-intervention trials ($M = 15.54\%$, $SD = 11.44$, 95% CI = 9.71, 21.37). Conversely, in the tablet activity group, the error rate was higher after participants received the tablet activity ($M = 20.62\%$, $SD = 10.64$, 95% CI =

15.65, 25.59) compared to before (*M* = 14.46%, *SD* = 9.29, 95% CI = 10.13, 18.80). The difference in pre- and post-intervention errors in the tablet activity group was significant based on a simple effects analysis of time within intervention group (Pillai's trace), V = 0.31, $F(1, 31) = 13.94$, $p = .001$, *partial* $\eta^2 = .31$.

Modified Attention Network Test (CRSD-ANT)

To investigate the effects of the interventions on the attention network test, three mixed ANOVAs were conducted on data from 33 participants (14 neurodevelopmental children; 19 typical developing children) on the alerting, orienting and conflict networks. Each ANOVA analysed one of the dependent variables: 1) attention network scores, 2) mean reaction time, and 3) mean error rates. For the attention network scores and error rates, separate 2 x 2 x 2 x 2 mixed ANOVAs were conducted, with two between-subject variables of intervention (exercise activity and tablet activity) and diagnosis (neurodevelopmental and typical developing group), as well as two within-subject variables of session (1 and 2) and time (pre- and post-intervention trial). In the reaction time ANOVA, an additional within-subject variable of cue/flanker type with two levels was included (i.e., 2 x 2 x 2 x 2 x 2 mixed ANOVA).

Attention network scores.

No significant main or interaction effect of intervention was found across the three network scores (see Table 11). Nonetheless, there was a significant effect of diagnosis on the conflict network, $F(1, 29) = 6.05$, $p = .02$, $r = 42$. Descriptive statistics show that children with a neurodevelopmental condition had higher conflict network scores (*M* = 122.09ms, *SD* = 40.65, 95% CI = 99.86, 144.33) than typical developing children (*M* = 87.45ms, *SD* = 39.07, 95% CI = 69.13, 105.77), suggesting a poorer efficiency in resolving conflict stimuli. As the network scores were based on differences between either the type of cue or flanker (see Chapter 3), detailed analysis of individual network reaction time and accuracy was

recommended for accurate interpretation of the ANT results (Fan & Posner, 2004). The following analyses examined the reaction time and error scores based on the type of cue or flanker used in alerting, orienting and conflict network.

Table 11
Summary Table for Mixed Analysis of Variance of the Between-Subject Effects on the Attention Network Scores

Source	*SS*	*MS*	*F*(1, 29)	*p*	*r*
Alerting Network					
Intervention	54.55	54.55	0.03	.86	.03
Diagnosis	55.64	55.64	0.03	.85	.03
Intervention x Diagnosis	1122.60	1122.60	0.69	.41	.15
Error	47059.45	1622.74			
Orienting Network					
Intervention	11.02	11.02	0.01	.94	.02
Diagnosis	5092.21	5092.21	2.97	.10	.30
Intervention x Diagnosis	400.22	400.22	0.23	.63	.09
Error	49690.36	1713.46			
Conflict Network					
Intervention	2583.15	2583.15	1.70	.20	.24
Diagnosis	9192.76	9192.76	6.05	.02*	.42
Intervention x Diagnosis	106.26	106.26	0.10	.79	.06
Error	44078.30	1519.94			

* p = .05. N = 33

Alerting network (no cue and double cue).

A main effect of cue was found to be significant on the alerting network, $F(1, 29) = 44.44$, $p = <.001$, $r = .78$. The mean reaction time for the type of cue conditions revealed that children were faster in the double cue condition (M = 864.29ms, SD = 94.54, 95% CI = 830.60, 897.98) than in the no cue condition (M = 912.83ms, SD = 97.52, 95% CI = 878.08, 947.58). However, the effect of the double cue on reaction time did not significantly differ between children with or without a neurodevelopmental condition, and whether they were given the exercise or tablet activity, $F(1, 29) = 1.45$, $p = .24$, $r = .22$. Similarly, when the

error rates were considered, the performance on the type of cues for children in both diagnostic groups did not significantly differ for those who received the exercise or tablet activity, $F(1, 29) = 2.14$, $p = .15$, $r = .26$.

Orienting network (centre and spatial cue).

In terms of orienting network, a main effect of cue condition was found to be significant, $F(1, 29) = 45.25$, $p < .001$, $r = .78$. Overall, children performed faster with more informative spatial cues ($M = 831.41$ms, $SD = 108.31$, 95% CI = 792.81, 870.01) compared to the less informative centre cues ($M = 882.51$ms, $SD = 100.22$, 95% CI = 846.80, 918.22). However, no significant interactions were found for the type of cues between children who received the exercise or tablet activity, and whether children were in the neurodevelopmental or typical developmental groups, on the reaction time, $F(1, 29) = 0.29$, $p = .59$, $r = .10$, and error measures, $F(1, 29) = 0.05$, $p = .83$, $r = .04$.

Conflict network (incongruent and congruent flanker).

With regards to the conflict network, the effect of flanker type was found to be significant, $F(1. 29) = 204.55$, $p < .001$, $r = .94$. In general, children performed faster on trials with congruent flankers ($M = 820.35$ms, $SD = 98.61$, 95% CI = 785.21, 855.49) than incongruent flankers ($M = 925.67$ms, $SD = 99.42$, 95% CI = 890.24, 961.10). However, the size of the effect of flanker type was significantly dependent on whether or not children had a neurodevelopmental condition, $F(1, 29) = 4.53$, $p = .04$, $r = .37$. According to Figure 15, although children in both diagnostic groups were faster on trials with congruent flankers compared to incongruent flankers, children in the neurodevelopmental group were more affected by the congruent flankers than children in the typical developmental group (as indicated by a larger reaction time difference between incongruent and congruent flankers). The effect of flanker type on children with a neurodevelopmental condition was consistent with the finding of the conflict network score reported earlier that children in this group had

greater difficulty processing trials with incongruent flankers. However, no influence of the type of intervention, diagnostic group, and the flanker type interaction on the reaction time of the conflict network was found, $F(1, 29) = 0.00$, $p = .98$, $r = .00$.

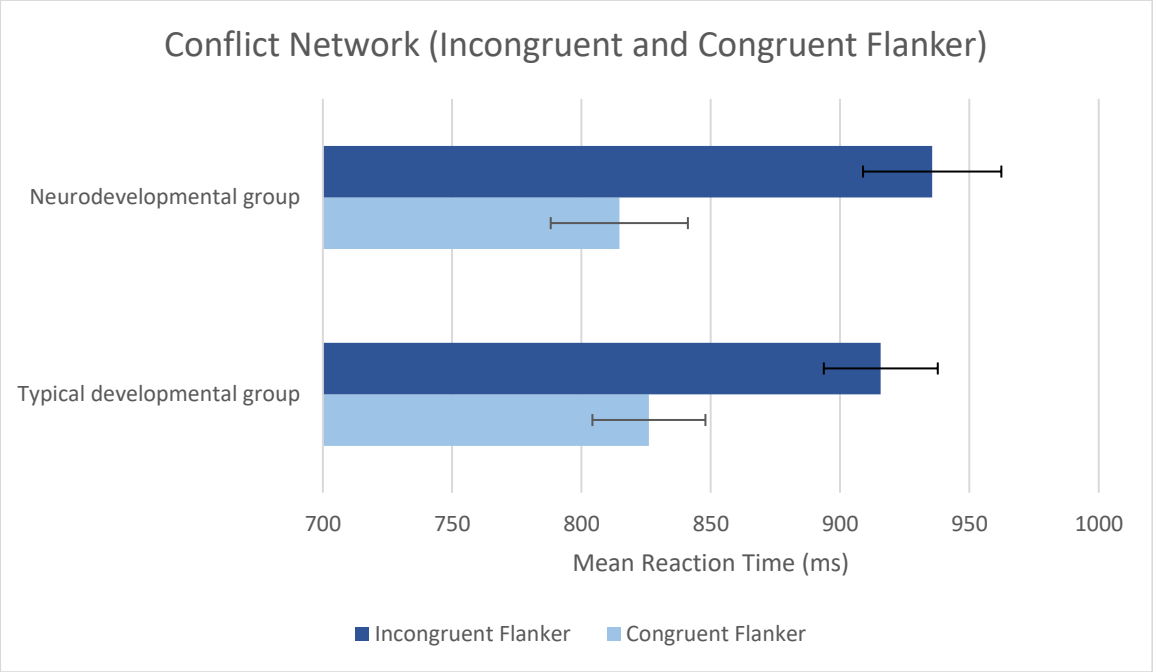


Figure 15. Mean reaction time performance on the conflict network between incongruent and congruent flankers for neurodevelopmental and typical developmental group. The error bars presented above are in standard errors.

In terms of the error rates on the conflict network, there was a significant interaction between the effects of the type of flanker, diagnosis, intervention group and pre/post-intervention trial (i.e., time), $F(1, 29) = 5.53$, $p = .03$, $r = .40$. As depicted in Figure 16, regardless of the diagnostic status, children in the exercise activity group tended to produce fewer errors on congruent trials than incongruent trials. In addition, the error rates for both congruent and incongruent flanker trials remained similar before and after physical exercise for this group of children. In the tablet activity group, however, although lower error rates were also generally found on congruent trials than incongruent trials, post-tablet activity appeared to have different effects for children dependent on whether or not they had a neurodevelopmental condition. Specifically, following tablet activity, children in the typical developmental group made fewer errors on incongruent trials ($M = 8.24\%$, $SD = 11.12$, 95%

CI = 3.03, 13.46) compared to baseline (*M* = 13.70%, *SD* = 11.51, 95% CI = 7.69, 19.72). Conversely, children in the neurodevelopmental group produced more errors on incongruent trials after tablet activity (*M* = 19.17%, *SD* = 9.54, 95% CI = 13.95, 24.38) relative to baseline (*M* = 13.89%, *SD* = 11.00, 95% CI = 7.88, 19.90). Indeed, using Pillai's trace, a simple effects analysis of pre/post-intervention trial within levels of diagnosis, flanker type and intervention, revealed significant differences in pre and post-intervention error rates on incongruent flanker trials following the tablet activity, in children with a neurodevelopmental condition, V = 0.25, $F(1, 29) = 9.60$, $p = .004$, *partial* $\eta^2 = .25$, and children with a typical development, V = 0.26, $F(1, 29) = 10.28$, $p = .003$, *partial* $\eta^2 = .26$.

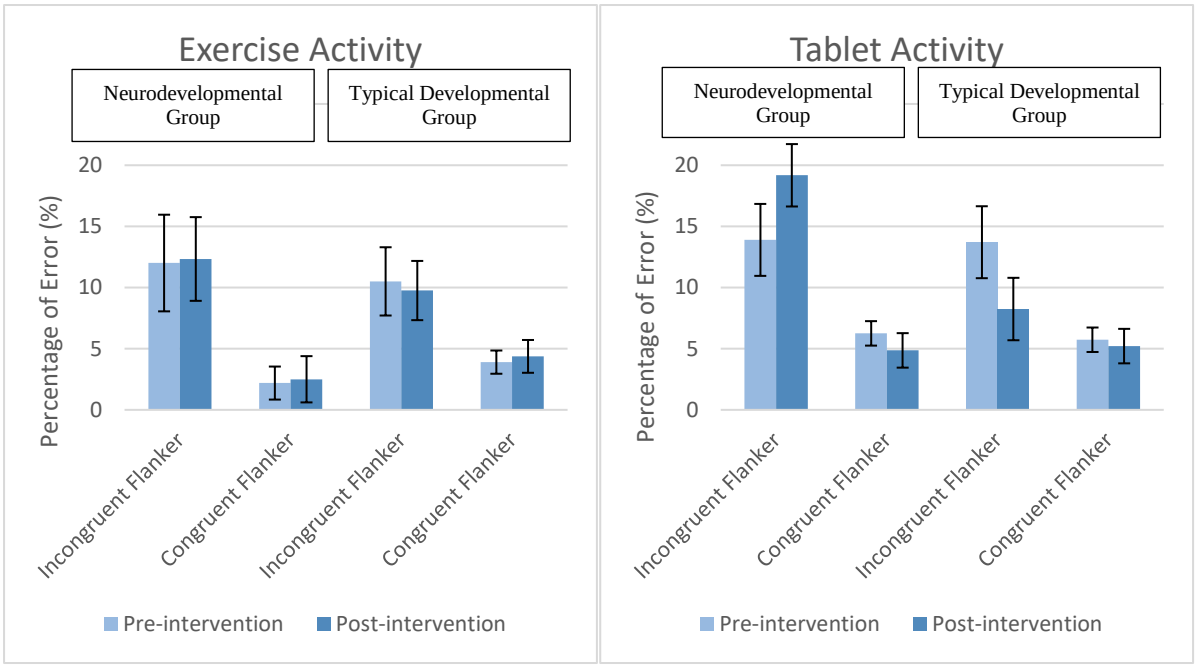


Figure 16. Error rates on the conflict network as a result of flanker type, pre/post-intervention trial, diagnostic group and intervention. The error bars presented above are in standard errors.

Discussion

The purpose of this chapter was to evaluate the effects of physical exercise in comparison to a tablet activity intervention on implicit sequence learning and attentional network in children 6-11 years with and without neurodevelopmental conditions. The overarching research questions are whether an acute exercise activity is more, equal or less

effective in enhancing aspects of cognition than a tablet activity, and whether the effect of an acute physical exercise is different between children with and without neurodevelopmental conditions.

Cognitive Effects of Physical Exercise on Implicit Learning

Reaction time differences between the probability types were evident in the first 100 trials (see Figure 13). This result was unexpected, as previous research with implicit learning tasks reported this occurring after at least 200 trials (e.g., Shanks, Channon, Wilkinson, & Curran, 2006). Although reaction times on probable trials were generally faster than on improbable trials, indicating the presence of sequence learning, the reaction time difference between probability type was narrowed following the exercise or tablet activity. Albeit speculative, the narrowing of reaction time differences may indicate a facilitation effect of exercise or tablet activity in enabling faster processing of both probable and improbable trials. One of the few studies that investigated the effect of physical exercise on implicit motor learning suggests that exercise activity may enhance the rate of implicit learning relative to a resting condition (Mang, Snow, Campbell, Ross, & Boyd, 2014), such that fewer trials are required to learn the implicit sequence. Thus, physical exercise may have reduced the number of trials needed for participants to demonstrate implicit learning.

Whether a child performed in the exercise or tablet condition did not result in any differences in reaction time on the ISLT, suggesting that physical exercise may not be more effective in enhancing implicit learning than a tablet activity. Nevertheless, in terms of error rates, children who performed the tablet activity made more errors on the implicit learning task than children who performed the exercise activity. Regarding the exercise activity, however, antecedent exercise did not appear to affect accuracy on the implicit task, which is consistent with another study that reported no change in accuracy (i.e., statistical learning task) following 15 and 30 minutes of stationary cycling (Stevens, Arciuli, & Anderson,

2016). Lastly, regardless of exercise or tablet activity, children with neurodevelopmental conditions produced higher error rates than children with a typical development.

Considering these results as a whole, it would appear that the effect of physical exercise did not particularly differ in comparison to a tablet activity on reaction time performance of the implicit learning task, in that both interventions improved reaction time compared to baseline. Nonetheless, tablet activity led to higher error rates, but this was not the case with physical exercise where no significant change in accuracy was observed.

Cognitive Effects of Physical Exercise on Attention Network

This study did not find significant differences between the effects of exercise or tablet activity on the alerting and orienting network. Regarding conflict network, on average, children with neurodevelopmental conditions had poorer efficiency in resolving conflict stimuli relative to children with a typical development. The difficulty in conflict network in children with neurodevelopmental conditions is consistent with previous research (e.g., Fan et al., 2012; Johnson et al., 2008; Mullane, Corkum, Klein, McLaughlin, & Lawrence, 2011). When the type of interventions is considered, the effect of physical exercise on error rates in the conflict network is similar in children with and without neurodevelopmental conditions. Conversely, post-tablet activity significantly increased errors on incongruent flanker trials for children with neurodevelopmental conditions but reduced errors for children with a typical development.

In other words, following tablet activity, children with neurodevelopmental conditions have greater difficulty resolving conflict relative to baseline and also in comparison to children with a typical development. It is noteworthy that the tablet activity used in this study contained components that could be considered a form of “cognitive training” where participants are required to fulfil game objectives (see Chapter 3) while actively avoiding random digital items that can positively or negatively affect the game, yet improvements in

conflict resolution are only observed for children with a typical development. This result indicates that the tablet activity may have a negative cognitive effect for children with neurodevelopmental conditions.

Research in the area of video games and cognition in children with neurodevelopmental conditions is limited (see reviews, Durkin, 2010; Durkin, Boyle, Hunter, & Conti-Ramsden, 2015). However, past correlational studies found negative effects of spending time on video games for this group of children (Chan & Rabinowitz, 2006; Mazurek & Engelhardt, 2013). In particular, these studies indicated that young individuals with ADHD or ASD that spent more than an hour a day on video games were more likely to exhibit symptoms of inattention, hyperactivity, and disrupted academic and social functioning. Contrary to the negative findings in the current study, previous research in children with ADHD has reported some areas of cognition being temporarily enhanced when engaged in 14-15 minutes of video games (Bioulac et al., 2014; Shaw, Grayson, & Lewis, 2005), though such performances are not reflected in formal neuropsychological tasks. Despite inconsistencies in the research regarding the effects of video games on children with neurodevelopmental conditions, the current findings suggest that the tablet activity have a negative effect on this group of children, especially on their ability to resolve conflict information. Consequently, such difficulties in conflict resolution may further compound the existing problems in educational and psychosocial functioning for children with neurodevelopmental conditions (Posner & Rothbart, 2005).

In comparison to the tablet activity, the effects of the exercise activity across the alerting and orienting networks appear to be similar, but with respect to resolving competing information, the exercise activity resulted in better performance than the tablet activity, but only for children with neurodevelopmental conditions. Indeed, the positive effect of physical exercise on inhibition in children has been reported repeatedly in ADHD studies (e.g., Gapin

et al., 2015; Gawrilow et al., 2016; Pan et al., 2015) and less so in ASD research (i.e., Anderson-Hanley et al., 2011). Nevertheless, it is noteworthy that, in this study, physical exercise is not found to improve the conflict network. Rather, the performance on the conflict network is preserved after the exercise activity. Conversely, the tablet activity resulted in reduced accuracy on the conflict network. Hence, physical exercise is considered more beneficial than the tablet activity for children with neurodevelopmental conditions.

Physical Exercise and Cognition Relationship

Overall, the results of this study suggest that the acute exercise activity is relatively more effective in improving cognition than the tablet activity. In terms of implicit learning, children with or without a neurodevelopmental condition typically performed better after the exercise activity than the tablet activity. However, for the conflict network, performance following the exercise activity was only better than following the tablet activity for children with a neurodevelopmental condition. These differences in the effects between exercise and tablet activity were mainly due to the negative effects of the tablet activity on accuracy (i.e., increased error). In other words, the exercise activity mainly served to maintain cognitive performance.

The results of this experiment might appear to invalidate the positive effects of physical exercise on cognition. There are reasons, however, why such a conclusion may not be appropriate.

First, the current physical exercise intervention consisted of a series of movements and visual-motor coordination (see Chapter 3), and differs from other simple physical exercises, such as running or cycling. Thus, the complexity of the exercise activity employed in the current study can be regarded as a mixture of both motor coordination and cognitive engagement (Budde et al., 2008; Pesce, 2012). Second, to explore the mechanism of why and how physical exercise affects cognition, the tablet activity was designed to be an active

control condition because it involves the high cognitive engagement component that is also a feature of physical exercise (Best, 2010; 2012). Thus, there is a possibility that the cognitive engagement component shared between the exercise and the tablet activity, may have resulted in some of the non-significant differences in the cognitive outcomes between both interventions.

Third, previous studies that have reported positive effects of physical exercise on cognition have reported comparisons with either a waitlist control group (e.g., Alesi, Bianco, Luppina, Palma, & Pepi, 2016; Tan et al., 2013a; Ziereis & Jansen, 2015) or a sedentary control group (e.g., Berse et al., 2015; Chuang et al., 2015; Gawrilow et al., 2016). Hence, the effect of physical exercise on cognition would likely be more apparent when compared to a waitlist or sedentary control group than to a cognitively engaging tablet game activity (Best, 2010). Indeed, a recent meta-analytic review on chronic exercise studies by Vazou et al. (2016) found a larger effect size of an enriched exercise activity when the comparison was based on a waitlist or sedentary control group/condition. However, there was a trivial effect of an enriched exercise activity when the comparison was based on an active control group (e.g., simple exercises). The findings by Vazou et al. suggest that the type of control group moderates the magnitude of the effect of exercise activity on cognition.

Fourth, it has been suggested in the exercise-cognition literature that physical exercise may not be a unique intervention in improving aspects of cognition, and there remains a possibility that other non-exercise activities may also be as effective (McMorris et al., 2009). This proposition is partially supported by the current findings, in that the tablet activity is somewhat comparable to physical exercise in enhancing cognition, especially on reaction time measures. Specifically, children who were engaged in the tablet activity were generally able to match the reaction time performance of those who were engaged in the exercise activity, but not on the error measures. An exception to the negative effect of tablet activity

on error rates lies in the conflict network data, where children with a typical development produced significantly fewer errors following the tablet activity than children with neurodevelopmental conditions. The effect of the tablet activity suggests that diagnostic status was influential. In particular, children with neurodevelopmental conditions were more likely to make conflict network errors after tablet activity than after exercise activity, but this negative effect was not found in typical developing children. This differential effect on the conflict network further supports the argument that tablet or video games, though attractive to children with neurodevelopmental conditions, may not be a suitable activity for this group of children (Chan & Rabinowitz, 2006; Mazurek & Engelhardt, 2013).

Lastly, as physical exercise was not found to have negative effects on implicit learning or attention network in children with and without neurodevelopmental conditions, it can be concluded that the effect of physical exercise on cognition is generally larger than that which follows the tablet activity. Furthermore, the finding from this study supports the proposal by Pesce (2012) that an exercise activity that encompasses both motor coordination and cognitive engagement should lead to better performance than would otherwise be obtained via either components. Previous research has also demonstrated the superiority of physical exercise with both motor coordination and cognitive engagement over simple exercise activity (e.g., Anderson-Hanley et al., 2012; Budde et al., 2008) or video games (Best, 2012). On the whole, the interaction between both the components of cognitive engagement and motor coordination is crucial to the exercise-cognition relationship (Pesce, 2012).

Limitations/Future Studies

An important limitation that needs to be highlighted is the small and unequal sample sizes in this study, particularly for children with a neurodevelopmental condition that was assigned to the exercise activity group (n = 5), and the tablet activity group (n = 10). For

children with a typical development, 11 were assigned to the exercise activity group and 9 children were in the tablet activity group. Regrettably, the aim of the original design of the study was to counterbalance the exercise and tablet activity, such that every participant would go through both activities via four separate sessions, to minimise the influence of individual variability. However, despite all participants having gone through both the exercise and tablet activity, ceiling effects were observed in session 3 and 4 during the analysis phase. Moreover, the observed ceiling effects occurred despite the use of alternating versions of the implicit learning task, and also a randomised attention network test. The ceiling effects complicated the interpretation of the results, such that it was unclear if it was the exercise and/or the tablet activity that resulted in the ceiling effects. Additionally, the ceiling effects may also be due to the opportunity to practice on the cognitive tasks across multiple sessions. As such, data from sessions 3 and 4 (i.e., before a change in the activity) were excluded to allow for a clearer interpretation of the results, however this resulted in the unequal group assignment.

Although efforts were made to achieve an equal, yet randomised group assignment, the unequal sample size as a result of the above post-hoc decision was unexpected. In addition, the minimum accuracy criterion (e.g., 70%) in the baseline cognitive measure further impacted on group assignment, particularly to children with neurodevelopmental conditions. Furthermore, despite various recruitment efforts in the community (see Chapter 3) for a period of eight months, the number of children in this study remained limited. Hence, the results of this study would need to be validated in future research with a larger sample size and to also consider the possibility of a ceiling effect.

Another limitation of this study is the validation of the ASD and ADHD diagnoses. Although the participants' behavioural symptoms were assessed based on the parent-rated autism (i.e., AQ-10), and ADHD (i.e., Conners 3AI-P) questionnaires, a detailed confirmation of the diagnosis (i.e., structured interview with school teachers and parents) was

not conducted. Nevertheless, the AQ-10 (Allison et al., 2012) and Conners 3AI-P (Conners, 2008) were known to have sound psychometric properties. Moreover, children were only assigned to the neurodevelopmental group if the ASD or ADHD symptoms were rated above the cut-off points recommended by the respective developers. Even though a high rated score on the autism and ADHD questionnaires may not confirm that a participant has an ASD or ADHD diagnosis, the elevated scores do suggest that the child has some existing behavioural symptoms that exceeded what was typically observed in their peers.

Additionally, there is a need to also take into account the duration of the resting condition. Although the inclusion of a resting period after an exercise activity is not uncommon in children studies, the resting duration in this study is relatively longer than those reported in other studies (e.g., 2 minutes; see Best, 2012). Thus, this extended resting period (i.e., 10 minutes) may have also influenced the findings. Nevertheless, the resting duration included the time needed for the child to cool down, have some water and a snack, and to reattach the EEG device back on the child.

As mentioned earlier, the inclusion of a waitlist or sedentary control group would most likely demonstrate a greater or clearer effect of physical exercise on cognition. However, the purpose of the study was not to determine if physical exercise has an effect on aspects of cognition, because the exercise-cognition literature has repeatedly demonstrated the existence of such effects. Rather, the purpose of the study was to investigate whether physical exercise is any better than a non-exercise activity that requires a high-level of cognitive engagement (i.e., tablet game activity). Importantly, this study also investigated the influence of individual differences that underlies the relationship between physical exercise and cognition, and this is considered in the next chapter. Thus, the use of an active control group was designed to eliminate the high cognitive engagement component that might be responsible for previous reports of the cognitive effects observed following physical exercise.

Indeed, the finding in this study that the exercise activity produced better performance than the tablet activity on accuracy in implicit learning and conflict network further strengthens the notion that both motor coordination and cognitive engagement are implicated in the physical exercise and cognition relationship.

Although it is common in the research literature to include some form of recognition test after an implicit learning task to examine the level of explicit learning processes that may have influenced task performance (e.g., Chambaron et al., 2008), this was not included in this study partly to limit the duration of testing, given that the duration per session was about an hour. Another reason concerned the difficulty in separating implicit and explicit learning processes. Previous researchers have acknowledged the challenges of measuring pure implicit learning and suggest it is unlikely to be measured without the involvement of explicit cognition (e.g., Shanks et al., 2005; Wilkinson & Shanks, 2004). Moreover, research on the effects of physical exercise on implicit learning performance is limited, with the few studies that have examined this relationship focusing more on motor skills acquisition rather than implicit cognition (Roig, Skriver, Lundbye-Jensen, Kiens, & Nielsen, 2012; Statton, Encarnacion, Celnik, & Bastian, 2015). Nevertheless, the finding that physical exercise has no detrimental effect on implicit learning performance suggests that this physical exercise and implicit learning relationship may be further evaluated in future research by investigating its effect on longer trials with more blocks and a recognition test.

Conclusion

This study fulfilled the second aim of this research project that was to compare the after-effects of an acute physical exercise and tablet activity on measures of implicit learning and attention network in typical developing children and those with neurodevelopmental conditions. Overall, children who engaged in the exercise activity performed better than those who engaged in the tablet activity, particularly with respect to the accuracy of their

performance on the implicit learning task. Furthermore, specific to children with neurodevelopmental conditions, the exercise activity did not affect accuracy pertaining to the conflict network, whereas the tablet activity produced more errors. The results support the notion that the interaction between the components of motor coordination and cognitive engagement is likely to be central to the relationship between physical exercise and cognition.

The third aim of this project is presented in the next chapter (i.e., Chapter 5). The third aim is to evaluate the psychophysiological data based on the proposed fractal analysis to account for the influence of individual differences on the cognitive effects of an acute exercise activity observed in this chapter.

Chapter 5: A Psychophysiological Investigation of the Galvanic Skin Response and Electroencephalogram in Accounting for the Exercise-Cognition Relationship

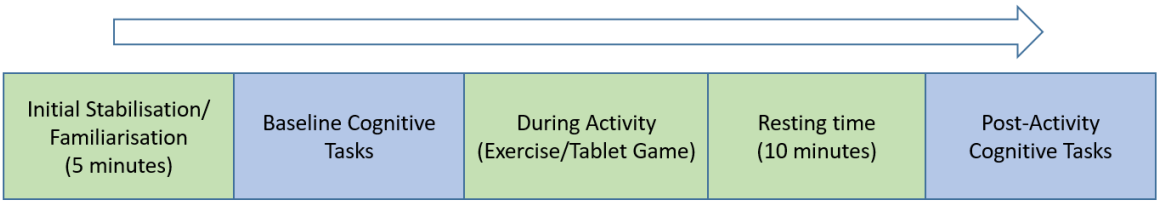
The purpose of this chapter is to explore whether the galvanic skin response (GSR) and electroencephalogram (EEG) measures, as indexed by their scaling exponents, could account for the cognitive performance reported in the previous chapter. Specifically, this chapter aims to test whether variations in the psychophysiological measures could account for how a child responds to the cognitive effect of an acute physical exercise activity. Based on the complexity theory introduced in Chapter 1, a healthy physiological system tends to exhibit a scaling exponent of around 1.0, or 1/f noise, whereas a pathological state would display a scaling exponent different to this value (e.g., Goldberger et al., 2002; Peng et al., 2000). Based on this theory, this project hypothesised that children who had cognitive improvements after performing the exercise or tablet activity would have a scaling exponent around DFA $\alpha \approx 1.0$, or 1/f noise, compared to those who did not improve (i.e., deviation from DFA $\alpha = 1.0$). Similarly, it was also hypothesised that children with a neurodevelopmental condition would have a scaling exponent different to DFA $\alpha \approx 1.0$, in comparison to children with a typical development.

Statistical Analysis: Psychophysiological Measures

Psychophysiological data were analysed with IBM SPSS 24 and R program 3.3.1 (R Core Team, 2016) with fractal statistical package version 2.0-1 (Constantine & Percival, 2016). A conceptual representation of EEG and GSR data segments used for statistical analyses is shown in Figures 17. According to this figure, an initial five minutes of EEG and GSR data were collected at the beginning of every session to allow for the stabilisation of the recordings and for the child to familiarise with the psychophysiological devices. Hence, the initial five minutes of physiological data were not included in the main analysis. Further, EEG data were segmented into those that were recorded at baseline and those measured

following the exercise or tablet activity (see Figure 17A). Conversely, GSR data were segmented into three parts, including those measured at baseline, during and following the exercise or tablet activity (see Figure 17B). Prior to the main analysis, EEG data were pre-processed to remove data artifacts (e.g., movements) using EEGLAB version 13.5.4b (Delorme & Makeig, 2004) and Neurophysiological Biomarker Toolbox (NBT) version 0.5.5-public (Poil, Simpraga, & Linkenkaer-Hansen, 2016), which both programs run on Matlab version R2013a (The MathWorks Inc., 2013).

A. EEG data segments (2) included for data analysis.



B. GSR data segments (3) included for data analysis.

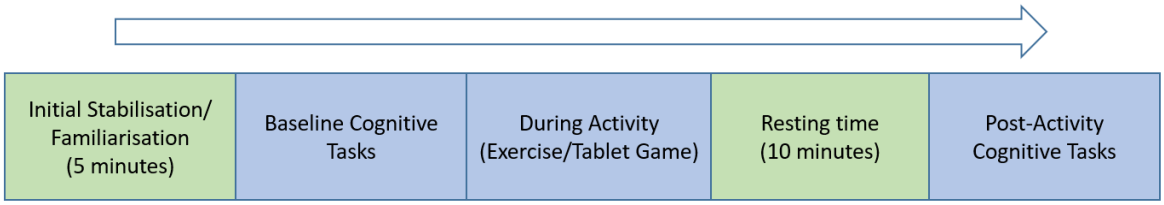


Figure 17. A linear conceptual representation of the research protocol illustrating the data segments used for data analysis of A) EEG data, and B) GSR data. *Note*. Segments highlighted in blue were included in the data analysis, and those highlighted in green were excluded. EEG data were not measured during the exercise activity due to high level of movements that would contaminate the EEG recording.

EEG data pre-processing.

The pre-processing method was adopted from the EEGLAB and NBT tutorial materials and guidelines written by Chaumon, Bishop, and Busch (2015), Delorme and Makeig (2012), Onton (2010), and Poil, Jansen et al. (2016). The pre-processing and analysis of EEG data was only conducted for data recorded during the baseline and post-activity cognitive tasks (see Figure 17A). Overall, there were six steps involved in the process of artefact rejection. First, EEG data were high-pass filtered at 1 Hz. Second, data were screened

visually for noisy or large movement artefacts, which were rejected. Third, independent component analysis was conducted using the runica algorithm (i.e., 'extended 1' option). Fourth, independent component activities across time were visually checked for component activities that were non-independent (i.e., deflections occurring across multiple components at the same time), which were removed. Fifth, data were re-analysed with independent component analysis (i.e., runica algorithm) to improve decomposition. Sixth, component activities were plotted onto the scalp maps (see example, Figure 18).

Based on the component properties and its location on the scalp map, components containing artefacts (e.g., muscle artefact) were removed, while retaining those with brain activities (e.g., alpha wave, 8-12 Hz). In the example shown in Figure 18, the component activities were isolated at T8 and the power spectrum demonstrated high frequencies above 20 Hz. Furthermore, noisy activities were detected across time series (not shown). All these properties are known characteristics of a muscle artefact (Chaumon et al., 2015). Additionally, the computation of component statistics for this component demonstrated that its distribution was non-gaussian, further indicating that this component was likely to be artefactual (Poil, Jansen et al., 2016). Hence, such a component with a muscle artefact was removed during the pre-processing stage prior to the main analysis.

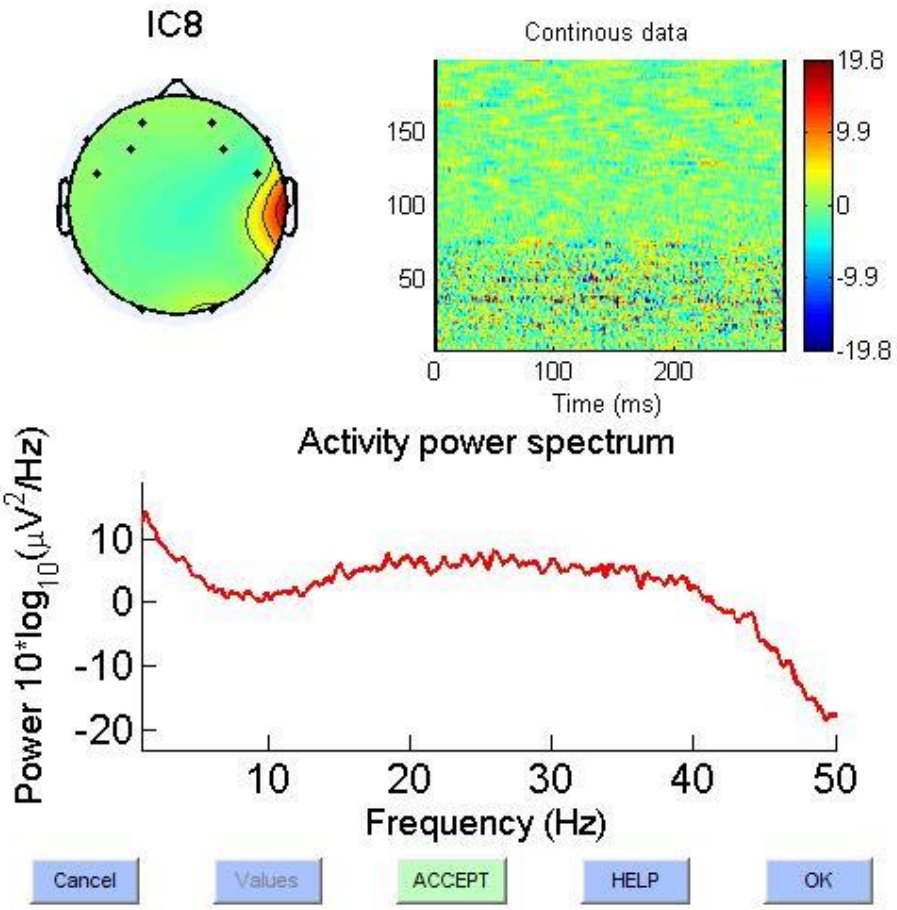
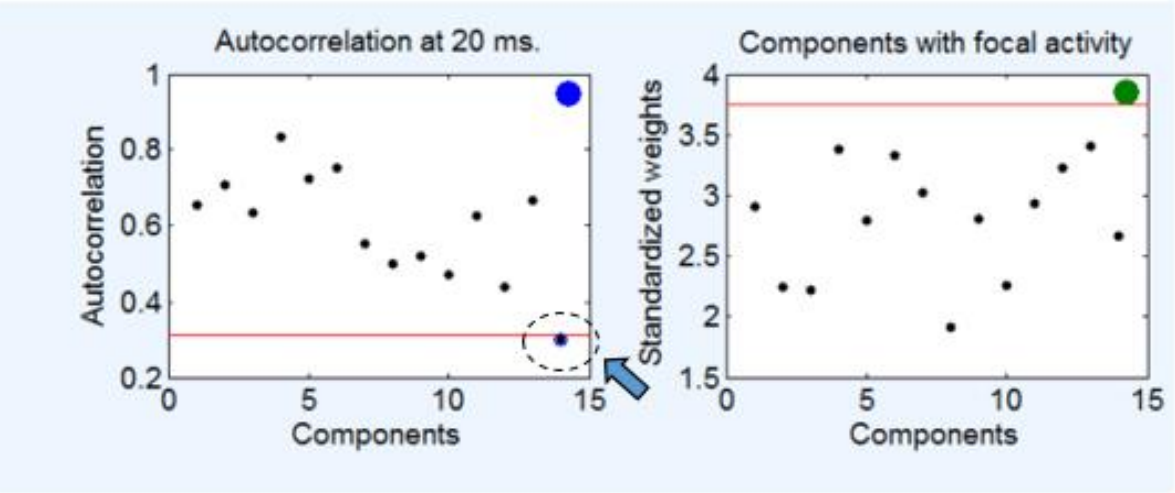


Figure 18. An example of a component artefact (i.e., muscle) located at T8.

Apart from manually examining the component activation, topographic plot, power spectrum, and component statistics, a semi-automatic rejection method was also adopted to aid in the identification of artefactual components. This additional protocol included the use of an EEGLAB plugin, known as the Semi-Automated Selection of Independent Components of the electroencephalogram for Artefact correction (SASICA; Chaumon et al., 2015). A detailed description of SASICA is provided in the guideline paper written by Chaumon et al. (2015). Briefly, SASICA provides additional information about whether a component is likely to be an artefact by providing the user with a scatterplot of all components and a bar graph of each component (see Figure 19). The scatterplot and bar graph come with a threshold line where any component that crosses this line would indicate that the particular component is likely to be an artefact. Specifically, two types of artefact detection based on

the SASICA were used in this project to detect the muscle (LoAC) and bad channel (FocCh) artefacts.

A) Scatterplot of 14 EEG components



B) Bar graph of one EEG component surpassing the artefactual threshold

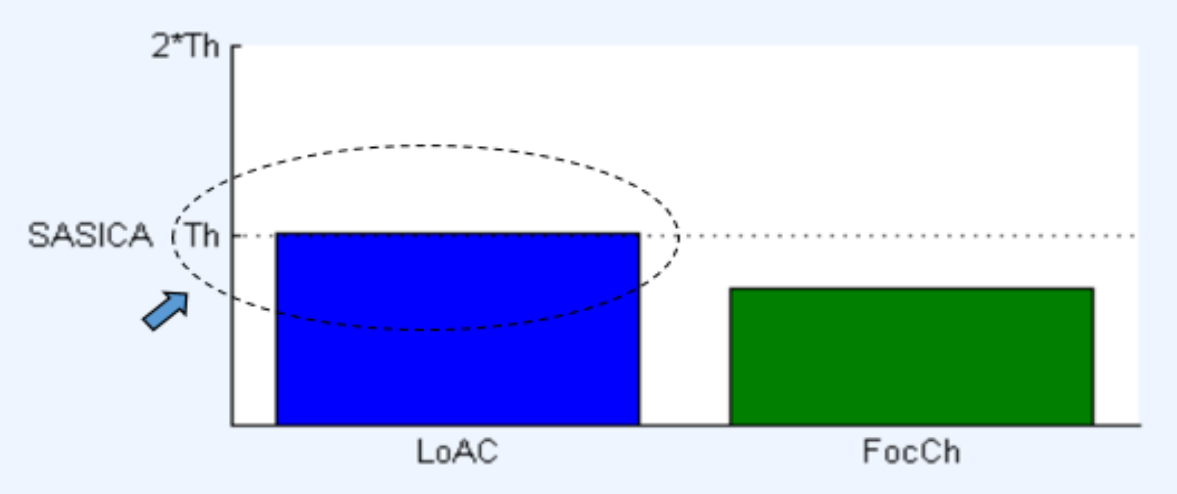


Figure 19. An example of a SASICA output displayed in A) a scatterplot, and B) a bar graph. *Note.* One component was detected to have surpassed the threshold line (i.e., see arrows), indicating a possibility of a muscle artefact.

It is necessary to highlight that the use of an automated rejection plugin like SASICA is not a guaranteed solution in detecting artefactual components in EEG recordings (Chaumon et al., 2015). Similar to other automated rejection plugins, neither the reliance of SASICA nor the use of manual rejection methods (e.g., power spectrum, topographic plot) on its own is adequate in detecting EEG artefacts. This point is due to the nature of error

inherent in both rejection protocols (i.e., misclassification of artefactual components, see Chaumon et al., 2015). Hence, it has been recommended that the best practice is to adopt both protocols (i.e., semi-automated) to minimise the risk of errors in artefact detection (Chaumon et al., 2015). Indeed, during the EEG pre-processing phase in this project, inconsistencies between both rejection protocols were found. For instance, a component was flagged by SASICA to be a muscle artefact, but careful inspection of the component activation, power spectrum, topographic plots, and component statistics, indicated that the component was likely to be a mixture of both neural activity and artefacts, and such a component should not be removed (Chaumon et al., 2015).

Data preparation for detrended fluctuation analysis (DFA).

EEG - Hilbert transform.

Following the pre-processing of the EEG data to remove artifacts (e.g., eye blinks), the "cleaned" EEG signals were subjected to a Hilbert transform to extract theta (4-8 Hz), alpha (8-13 Hz), and beta (13-30 Hz) amplitude envelopes via the NBT program (Hardstone et al., 2012; Poil, Jansen et al., 2016). The advantages of the Hilbert transform over Fourier or Wavelet methods are described elsewhere (e.g., Singh & Goyat, 2016). Briefly, in contrast with the Fourier or Wavelet functions, the Hilbert transform can be applied to data that are non-stationary and non-linear, which are known properties of many human physiological systems including EEG (e.g., Goldberger et al., 2012; Peng et al., 2000; Singh & Goyat, 2016; West, 2006).

After the Hilbert transform was applied, the extracted alpha, beta and theta frequency bands across the 14 EEG channels were trimmed to equal time length of 3 minutes (i.e., 23,040 samples/channel) based on the lowest time length available. Additionally, DFA conducted via the R program (i.e., fractal package) was then applied to each of the EEG channels to derive the scaling properties of alpha, beta and theta frequency bands. The

syntaxes for the amplitude envelope extraction via NBT and the DFA conducted through R are presented in Appendix I and J, respectively.

Galvanic skin response (GSR).

The segmented GSR data of baseline, during activity, and post-activity (see Figure 17B) were trimmed to an equal time length of 5.28 minutes (i.e., 10, 145 samples/segment) based on the lowest time length available. These three segmented GSR data were then subjected to the DFA conducted via the R program (i.e., fractal package). As the scaling property of the GSR was found in previous research to be a Brownian signal (Wijnants et al., 2013), a 'bridge' detrended fluctuation analysis was conducted on all three segments of the GSR data, as this function has been reported to capture the Brownian signal better than a non-bridged DFA (Stroe-Kunold et al., 2009). The bridge DFA was also conducted via the R program with the fractal package. An example of the bridge syntax is also provided in Appendix K.

Surrogate test (random shuffling) - DFA.

To check the validity of the scaling exponent output provided by the DFA, a surrogate data set based on 10 randomly selected participants' GSR and EEG data (i.e., 5 children with typical development and 5 children with neurodevelopmental conditions) was generated (Goldberger et al., 2000; Peng et al., 2000). The surrogate data was generated by randomly shuffling the participants' baseline GSR and EEG data (i.e., alpha, beta and theta). Both the surrogate and the original data were then subjected to the DFA. Although both types of data had equal statistical properties (e.g., means and standard deviations), if the scaling exponent of the original data is dependent on how the data fluctuate across time (Goldberger et al., 2000; Hardstone et al., 2012), the scaling exponent should differ from that of the surrogate data, given that the order of the data would have been disrupted by the random shuffling. This test showed that the average scaling exponents for the original data of the GSR was $\alpha \approx 1.5$

(Brownian noise), and $\alpha \approx 1.0$ (1/f noise) for the EEG data across alpha, beta and theta frequency bands. Consistent with the expectation, the average scaling exponent for the shuffled data of both the GSR and EEG was $\alpha \approx 0.5$, or white noise. The outcome of this surrogate test supports the validity of the DFA by indicating that the fractal behaviour of the original GSR and EEG data were based on how the data fluctuate across time (Goldberger et al., 2000; Hardstone et al., 2012). Furthermore, the outcome of this test was consistent with what was known about the various types of time series data, including EEG data being 1/f noise (e.g., Ferri, Rundo, Bruni, Terzano, & Stam, 2005; Lee, Kim, Kim, Suk Park, & Kim, 2004), GSR as a Brownian noise (Wijnants et al., 2013), and a random and uncorrelated data, as in the case of the surrogate data, being white noise (Kantelhardt, 2008; Peng et al., 1995).

Results

Galvanic Skin Response (GSR)

A 2 (session) x 2 (intervention group) x 2 (diagnosis) x 3 (time) mixed ANOVA was conducted on the scaling exponent of the GSR measure. The within-subjects variable of time included scaling exponents measured at baseline, during and after an intervention. The between-subjects variable of intervention consisted of the physical exercise and tablet activity groups. The other between-subjects variable of diagnostic status included children with a typical development and those with a neurodevelopmental condition. Tests of normality (Shapiro-Wilk) and homogeneity of variance (Levene's test) assumptions were met for the mixed ANOVA. Mauchly's tests indicated violations of the assumption of sphericity for within-subject effects of time, and session and time interaction. Hence, Huynh-Feldt estimates of sphericity were applied to correct the degrees of freedom in the tests of these effects.

Detrended fluctuation analysis – GSR.

Based on the ANOVA, there was a significant difference in the scaling exponents of the GSR measured during baseline, intervention, and after an intervention, $F(1.79, 55.35) = 7.76$, $p = .002$, *partial* $\eta^2 = .20$. There was also a significant interaction between time and the type of intervention group, $F(1.79, 55.35) = 11.30$, $p = <.001$, *partial* $\eta^2 = .27$. As shown in Figure 20, only children in the exercise activity group demonstrated a change in scaling exponents during and after an exercise intervention. In the exercise activity group, a slight reduction in the scaling value was observed during exercise intervention relative to baseline, followed by an increase after exercise. This trend, however, was not observed for children in the tablet activity group, where the scaling values remained constant before, during, and after the intervention. A simple effects analysis of the interaction between intervention group and time revealed a significant difference between the exercise activity and tablet activity groups in the scaling exponents, but only at post-intervention, $F(1, 31) = 16.19$, $p = <.001$, $r = .59$, (see Table 12). In particular, after the intervention, children in the exercise activity group obtained a larger scaling exponent ($M = 1.6$, $SD = 0.20$, 95% CI = 1.5, 1.7), in comparison to children in the tablet activity group ($M = 1.4$, $SD = 0.17$, 95% CI = 1.3, 1.4). The interaction effect of time and intervention, however, was not influenced by whether a child had a neurodevelopmental condition, $F(1.79, 55.35) = 0.05$, $p = .94$, *partial* $\eta^2 = .001$.

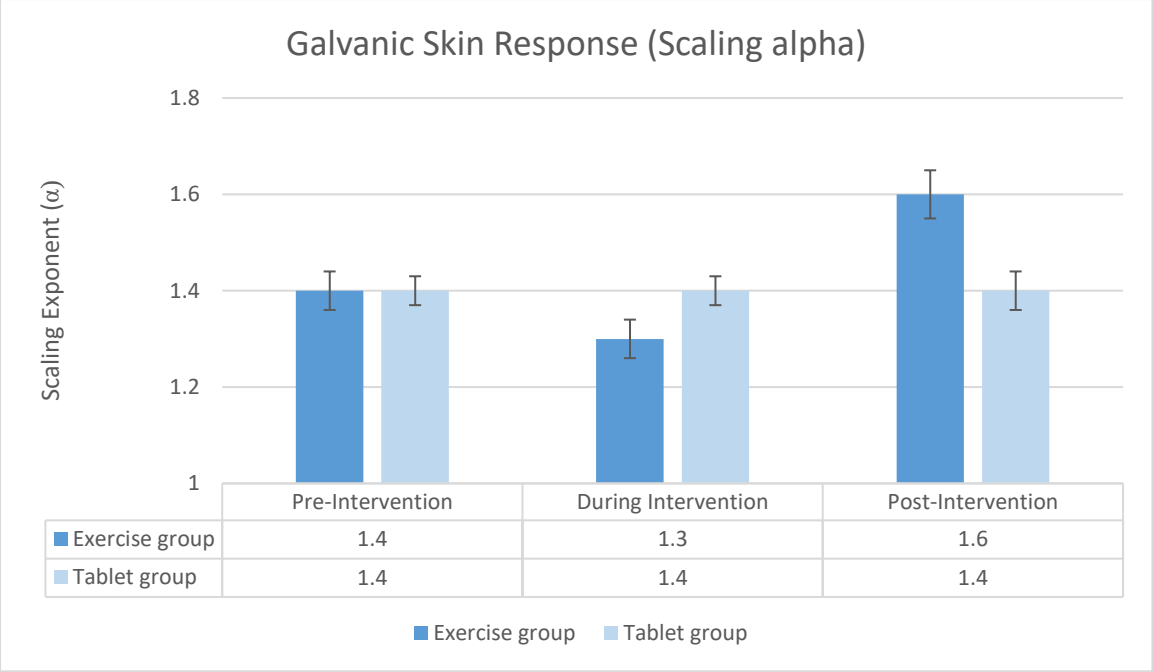


Figure 20. The effect of intervention group on the scaling exponents of the galvanic skin response. The error bars presented above are in standard errors.

Table 12
Summary Table for Simple Effects Analysis of Intervention Group within Time (GSR Scaling Exponent)

Source	*SS*	*MS*	*F*(1, 31)	*p*	*r*
Pre-Intervention					
Exercise versus Tablet group	0.00	0.00	0.11	.74	.06
Error	0.58	0.02			
During Intervention					
Exercise versus Tablet group	0.01	0.01	0.26	.62	.09
Error	0.64	0.02			
Post-Intervention					
Exercise versus Tablet group	0.51	0.51	16.19	<.001*	.59
Error	0.97	0.03			

* $p = .05$.

Although differences in the scaling exponents were found between children who exercised and those who performed the tablet activity, no cognitive measures were included in the above ANOVA. Thus, such differences in the GSR scaling exponent may not be related to the cognitive performance presented in Chapter 4. Indeed, post-intervention

differences between exercise and tablet activity were also found for the absolute GSR levels, measured in µS, $F(1.55, 48.16) = 29.97$, $p < .001$, *partial* $\eta^2 = .49$ (see Figure 21). Hence, to demonstrate that the differences in the GSR scaling exponent between children who performed the exercise and those who engaged in the tablet activity were related to the cognitive performance reported in Chapter 4 (i.e., significant effects of intervention only), multiple ANCOVAs were conducted specifically on the accuracy performance of the implicit sequence learning task (ISLT) and modified attention network test (CRSD-ANT) - incongruent flanker trials.

Since the aim of this chapter was to determine whether there were differences in psychophysiological measures between those children who responded to the cognitive effect of exercise and those who did not exhibit an exercise effect on cognition, children's accuracy performance was also sorted according to whether a child, following an exercise or tablet activity, demonstrated a cognitive progress or decline (i.e., accuracy change).

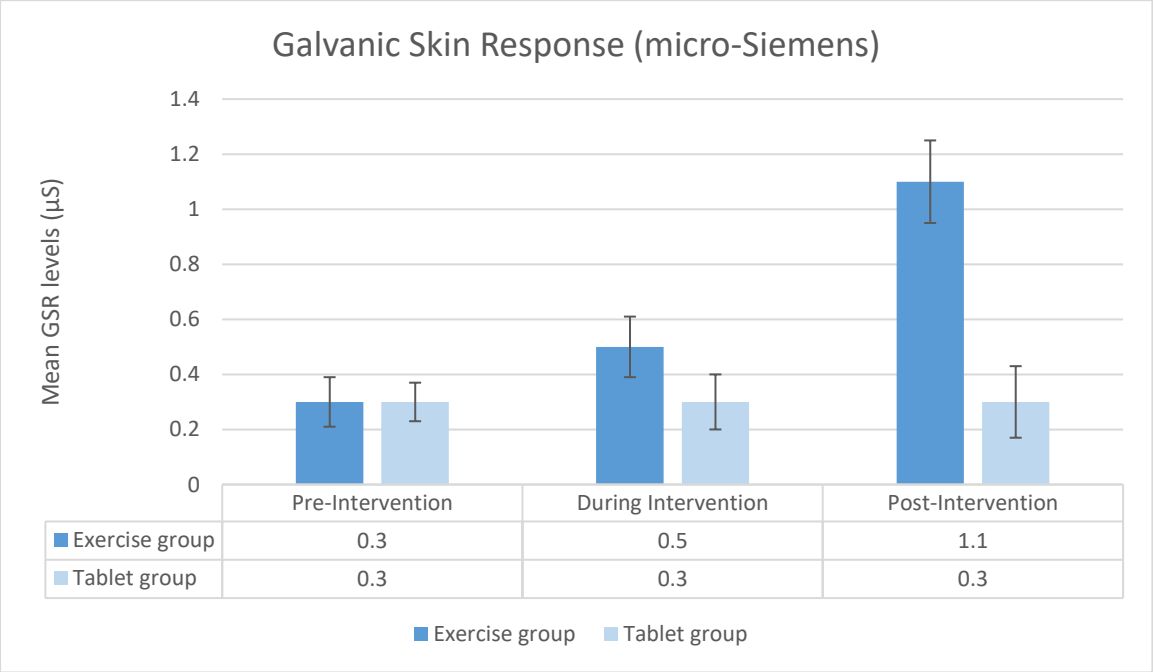


Figure 21. The effect of intervention group on the galvanic skin response (micro-Siemens). The error bars presented above are in standard errors.

GSR-DFA and cognitive tasks.

Galvanic skin response (GSR) level is known to differ largely among individuals (Nourbakhsh, Wang, Chen, & Calvo, 2012), and can be influenced by physical activity due to increased sweat production (Critchley, 2002; Novak et al., 2010). Indeed, according to Figure 21, the absolute GSR level increased during exercise and peaked following the exercise activity. This linear increment in absolute GSR levels was not observed in the tablet activity group. Thus, the increased GSR levels in the exercise group indicated a physiological response to the exercise activity due to increased sweat production. Furthermore, though the average GSR levels were equal in the exercise and tablet activity group at baseline, an inspection of the individual values indicated a large variation in the basal GSR levels ranged between 0.05 μS to 2.27μS. On the whole, regardless of the cognitive performance, basal GSR levels (μS) were not only different among individuals, exercise also elevated the absolute GSR levels due to increased sweat production.

Although the GSR scaling exponent was an index of how data fluctuate across time (Hardstone et al., 2012) and differed from the absolute GSR level (i.e., mean statistic), the influence of sweat production due to physical activity on the data fluctuation cannot be ruled out. Moreover, according to Figures 20 and 21, paralleled increment was observed in both the scaling exponent and the absolute GSR level measured following exercise. Therefore, to ensure that the elevated scaling exponent after the exercise activity was not confounded by increased sweat production during exercise and individual GSR differences at baseline, the pre-intervention and during intervention GSR scaling exponents need to be controlled as covariates. Prior to the analyses, GSR scaling exponents of pre-intervention and during intervention were assessed to determine whether these two variables were appropriate covariates for ANCOVAs. Initial t-tests conducted showed that the pre-intervention and during intervention scaling exponents were not significantly different across diagnosis,

intervention, and whether individuals showed improvement on the cognitive tasks. These results indicated that the pre-intervention and during intervention scaling exponents measured in session 1 and 2 were appropriate to be included as covariates in the analyses. All the assumptions for ANCOVA were met. Four 2 (intervention) x 2 (diagnosis) x 2 (accuracy change) ANCOVAs were conducted for the implicit learning and attention network tests performance (i.e., accuracy change) in session 1 and 2. The covariates of pre-intervention and during intervention GSR scaling exponents from session 1 and 2 were used for the respective ANCOVA (i.e., covariates measured in session 1 used for session 1 ANCOVAs, covariates measured in session 2 used for session 2 ANCOVAs).

GSR-DFA: Implicit Sequence learning task (accuracy change).

Based on the ANCOVA, in session 1, the covariate, pre-intervention scaling exponent, was significantly related to the post-intervention scaling exponent, $F(1, 25) = 9.90$, $p = .004$, *partial* $\eta^2 = .28$. This result was not observed for the other covariate (i.e., during intervention scaling exponent), $F(1, 25) = 1.09$, $p = .31$, *partial* $\eta^2 = .04$.There was a significant effect of intervention group, $F(1, 25) = 17.64$, $p < .001$, *partial* $\eta^2 = .41$, and the change in accuracy performance, $F(1, 25) = 5.61$, $p = .03$, *partial* $\eta^2 = .18$, on the post-intervention scaling exponent, when the scaling exponents of pre-intervention and during intervention were controlled. Further, there was also a significant interaction between intervention and accuracy change, $F(1, 25) = 6.37$, $p = .02$, *partial* $\eta^2 = .20$. In the physical exercise group, following the exercise intervention, participants that exhibited an increased error rate on the implicit sequence learning task (ISLT), had a larger scaling exponent ($M_{\text{adjusted}} = 1.9$, $SD = 0.20$, 95% CI = 1.7, 2.0) relative to children who had a reduced or static error rate ($M_{\text{adjusted}} = 1.5$, $SD = 0.21$, 95% CI = 1.4, 1.6). Conversely, children who performed the tablet activity showed no difference in the scaling exponents regardless of whether they made more errors ($M_{\text{adjusted}} = 1.4$, $SD = 0.20$, 95% CI = 1.3, 1.5) or less/static error rate

($M_{adjusted}$ = 1.4, *SD* = 0.20, 95% CI = 1.2, 1.5) on the ISLT. These results were supported by the simple effects analyses of the change in accuracy performance within levels of intervention group (see Table 13). The interaction effect of intervention and accuracy change, however, did not differ in children with or without a neurodevelopmental condition, $F(1, 25) = 3.89$, $p = .06$, *partial* $\eta^2 = .14$.

Table 13
Summary Table for Simple Effects Analysis of Accuracy Change (Session 1) Within Levels of Intervention Group (ISLT)

Source	*SS*	*MS*	*F*(1, 25)	*p*	Partial η^2
Exercise Activity					
Increased versus Reduced Error	0.31	0.31	9.57	.01*	.28
Error	0.80	0.03			
Tablet Activity					
Increased versus Reduced Error	0.00	0.00	0.00	.98	.00
Error	0.80	0.03			

* $p = .05$.

In session 2, the covariate, pre-intervention scaling exponent, was found to be significantly related to the post-intervention scaling exponent, $F(1 , 25) = 5.32$, $p = .03$, *partial* $\eta^2 = .18$. This result was not observed for the other covariate (i.e., during intervention scaling exponent), $F(1, 25) = 0.55$, $p = .46$, *partial* $\eta^2 = .02$. There was also a significant effect of intervention group on the post-intervention exponent, when the covariates were included, $F(1\ 25) = 5.53$, $p = .03$, *partial* $\eta^2 = .18$. Although the scaling exponent was larger for children who received the physical exercise ($M_{adjusted}$ = 1.6, *SD* = 0.24, 95% CI = 1.4, 1.7) compared to those who received the tablet activity ($M_{adjusted}$ = 1.3, *SD* = 0.31, 95% CI = 1.2, 1.5), there were no significant effects related to the accuracy change on the ISLT. This result indicates that there were no significant differences in the scaling exponents between children who scored more or less errors on the ISLT in session 2. Lastly, the effect of intervention was not also dependent on whether or not a child had a neurodevelopmental condition, $F(1, 25) = 1.69$, $p = .21$, *partial* $\eta^2 = .06$.

GSR-DFA: Conflict network - incongruent flanker trials (accuracy change).

According to the ANCOVA, in session 1, after controlling for the scaling exponents of pre-intervention, $F(1, 23) = 1.57$, $p = .22$, *partial* $\eta^2 = .06$, and during intervention, $F(1, 23) = 0.47$, $p = .50$, *partial* $\eta^2 = .02$, there was a significant effect of intervention as a function of accuracy change, on the post-intervention scaling exponent of the incongruent flanker trials, $F(1, 23) = 6.19$, $p = .02$, *partial* $\eta^2 = .21$. Specifically, children who made more errors after performing the exercise activity had a lower scaling exponent ($M_{adjusted} = 1.3$, $SD = 0.22$, 95% CI = 1.1, 1.6) compared to those who made fewer or the same errors following the exercise activity ($M_{adjusted} = 1.7$, $SD = 0.20$, 95% CI = 1.6, 1.8). Conversely, following the tablet activity, the scaling exponents did not differ in children who made more errors ($M_{adjusted} = 1.4$, $SD = 0.27$, 95% CI = 1.2, 1.6) or those that had reduced or static error rates ($M_{adjusted} = 1.4$, $SD = 0.21$, 95% CI = 1.2, 1.5) on the incongruent flanker trials. These results were supported by the simple effects analyses of accuracy change within levels of intervention group (see Table 14). Nevertheless, the interaction effect of intervention and accuracy change was not dependent on whether or not a child had a neurodevelopmental condition, $F(1, 23) = 0.01$, $p = .95$, *partial* $\eta^2 = .00$.

Table 14

Summary Table for Simple Effects Analysis of Accuracy Change (Session 1) Within Levels of Intervention Group (CRSD-ANT: Incongruent Flanker Trials)

Source	*SS*	*MS*	$F(1, 23)$	*p*	Partial η^2
Exercise Activity					
Increased versus Reduced Error	0.34	0.34	9.50	.01*	.29
Error	0.81	0.04			
Tablet Activity					
Increased versus Reduced Error	0.01	0.01	0.20	.66	.01
Error	0.81	0.04			

* $p = .05$.

In session 2, the covariate, pre-intervention scaling exponent, was significantly related to the post-intervention scaling exponent, $F(1, 23) = 6.39$, $p = .02$, *partial* $\eta^2 = .22$. This result

was not observed for the other covariate (i.e., during intervention scaling exponent), $F(1, 23) = 0.33$, $p = .57$, *partial* $\eta^2 = .01$. There was also a significant effect of diagnosis, after controlling for the pre-intervention and during intervention scaling exponents, $F(1, 23) = 7.38$, $p = .01$, *partial* $\eta^2 = .24$. Additionally, there was a significant effect of diagnosis as a function of the type of intervention, $F(1, 23) = 4.50$, $p = .05$, *partial* $\eta^2 = .16$. Although the scaling exponent was larger after the exercise activity ($M_{adjusted} = 1.7$, $SD = 0.19$, 95% CI = 1.6, 1.8) compared to the tablet activity ($M_{adjusted} = 1.4$, $SD = 0.21$, 95% CI = 1.3, 1.5), this difference was only observed for children with a typical development. Contrary to typical developing children, children with a neurodevelopmental condition had similar scaling exponents regardless of whether they performed the exercise activity ($M_{adjusted} = 1.3$, $SD = 0.25$, 95% CI = 1.1, 1.6) or the tablet activity ($M_{adjusted} = 1.4$, $SD = 0.21$, 95% CI = 1.2, 1.5). However, the interaction effect of diagnosis and intervention was not significantly related to the accuracy change of the incongruent flanker trials, $F(1, 23) = 1.68$, $p = .21$, *partial* $\eta^2 = .07$.

GSR scaling exponent (*α*) and absolute GSR level (µS).

To ensure that the significant relationships observed between the GSR scaling exponents after physical exercise and accuracy change on the ISLT and CRSD-ANT (i.e., incongruent flanker trials) were not due to high or low absolute GSR levels (µS), the same ANCOVAs conducted for the scaling exponents were repeated on the absolute GSR levels. Controlling for pre-intervention and during intervention absolute GSR levels as covariates, no significant interaction effect of intervention, accuracy change and diagnosis was found on the post-intervention GSR level (see Table 15). This finding indicates that the significant relationship observed between the post-exercise GSR scaling exponent and accuracy change on the ISLT and incongruent flanker trials was not related to the absolute GSR levels. In other words, a high or low mean GSR level was not equivalent to the value of a scaling

exponent. Hence, there is support that the GSR scaling exponent differed from the absolute GSR level. The difference between the results of the GSR scaling exponent and the absolute GSR level also demonstrated the advantage of fractal analysis in providing additional information (i.e., data fluctuation) that is not provided through standard mean statistics (Brown & Liebovitch, 2010; West, 2006).

Table 15
Analysis of Covariance of Post-Intervention Mean GSR level (μS) as a Function of Accuracy Change, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR levels as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Implicit Sequence Learning Task (N = 35)						
Session 1						
Intervention x Accuracy change	1	0.18	0.18	1.18	.29	.05
Intervention x Accuracy change x Diagnosis	1	0.00	0.00	0.01	.92	.00
Error	25	3.85	0.15			
Session 2						
Intervention x Accuracy change	1	0.01	0.01	0.06	.81	.00
Intervention x Accuracy change x Diagnosis	1	0.30	0.30	1.37	.25	.05
Error	25	5.55	0.22			
CRSD-ANT: Incongruent Flanker (N = 33)						
Session 1						
Intervention x Accuracy change	1	0.42	0.42	2.98	.10	.12
Intervention x Accuracy change x Diagnosis	1	0.13	0.13	0.92	.35	.04
Error	23	3.21	0.14			
Session 2						
Intervention x Accuracy change	1	0.45	0.45	2.27	.15	.09
Intervention x Accuracy change x Diagnosis	1	0.03	0.03	0.15	.70	.01
Error	23	4.57	0.20			

* $p = .05$.

To further support the significant association between GSR post-exercise scaling exponents and accuracy change (i.e., ISLT and CRSD-ANT incongruent flanker), multiple ANCOVAs, with pre-intervention and during intervention scaling exponents controlled as covariates, were conducted on other ISLT and CRSD-ANT variables (e.g., reaction time

measures). There was no significant relationship between intervention and performance change for the other cognitive variables (see Appendix L, Table 23-29). Interestingly, significant interaction effects of intervention and performance change (i.e., incongruent flanker trials – reaction time) on the post-exercise GSR scaling exponent was found in session 1, $F(1, 24) = 5.52$, $p = .03$, *partial* $\eta^2 = .19$, and session 2, $F(1, 23) = 5.72$, $p = .03$, *partial* $\eta^2 = .20$. In other words, following the exercise activity, the scaling exponent was significantly different between children who improved in their reaction time (RT) and those that did not improve on this measure. This result was unexpected, given that there was no significant main or interaction effect of intervention found on this RT measure (see Chapter 4).

Although further exploration on the association between the RT change of the incongruent flanker trials and the GSR scaling exponent would be interesting, this approach is inconsistent with the aim of this chapter, which is to investigate the GSR scaling exponents to account for the significant effects (i.e., intervention) reported in Chapter 4. Therefore, the RT change for the incongruent flanker trials was not explored here, since the exercise intervention was not found to have a significant effect on this RT measure (see Chapter 4). In Chapter 4, the differences in intervention were only found on the accuracy measures of the ISLT and incongruent flanker trials. Hence, on the whole, the significant relationship between post-exercise GSR scaling exponents and accuracy change on the ISLT and incongruent flanker trials were mostly consistent with the findings reported in Chapter 4.

EEG Frequency Bands

To test if there are differences in the scaling exponents derived from the EEG data, a 2 (session) x 2 (time: Pre- and post-intervention trial) x 2 (intervention) x 2 (diagnosis) x 14 (EEG channels) mixed ANOVA was conducted separately for the alpha, beta and theta frequency bands. Tests of normality (Shapiro-Wilk) and homogeneity of variance (Levene's

test) assumptions were met for the mixed ANOVAs. Mauchly's tests, however, indicated multiple violations of the assumption of sphericity for alpha, beta, and theta frequency bands. Hence, Greenhouse-Geisser estimates of sphericity were applied to correct the degrees of freedom for the ANOVAs.

Detrended fluctuation analysis – EEG.

Few significant main or interaction effects of diagnosis and intervention group were found across the alpha, beta and theta frequency bands (see Table 16). The few that were observed were in the beta and theta frequencies. In the beta frequency band, a significant main effect of time was found, $F(1, 22) = 4.50$, $p = .05$, $r = .41$. Additionally, in the theta frequency band, the scaling exponents were also found to be significantly different among channels, $F(3.63, 79.94) = 3.40$, $p = .02$, *partial* $\eta^2 = .13$, and diagnostic group, $F(1, 22) = 4.35$, $p = .05$, $r = .41$. However, when the means and standard errors of these significant variables were examined, the mean differences were very small. For example, although the scaling exponent in the theta frequency band was found to be significantly different between children with a neurodevelopmental condition and those with a typical development, the means of both groups was about $\alpha = 1.0$, with a trivial difference of 0.002 and a standard error of 0.001. Such small differences were also found for the main effect of time (beta frequency band), and channel (theta frequency band).

A further 2 (time) x 2 (intervention) x 2 (diagnosis) x 2 (accuracy change) x 14 (EEG channels) mixed ANOVA was conducted on alpha, beta and theta frequency bands to determine whether the EEG scaling exponent was significantly different for children who had reduced errors and those who made more errors on the ISLT and incongruent flanker trials. However, there was no significant relationship between the EEG scaling exponent and error rates (see Appendix L, Table 30).

Table 16
Summary Table for Mixed Analysis of Variance of the Between-Subject Effects on the Scaling Exponents of EEG Frequency Bands

Source	ANOVA
Alpha	
Intervention	$F(1, 22) = 0.05$, $p = .83$, $r = .05$
Diagnosis	$F(1, 22) = 2.98$, $p = .10$, $r = .35$
Intervention x Diagnosis	$F(1, 22) = 0.05$, $p = .83$, $r = .05$
Beta	
Intervention	$F(1, 22) = 0.21$, $p = .65$, $r = .10$
Diagnosis	$F(1, 22) = 2.65$, $p = .12$, $r = .33$
Intervention x Diagnosis	$F(1, 22) = 1.86$, $p = .19$, $r = .28$
Theta	
Intervention	$F(1, 22) = 0.54$, $p = .47$, $r = .15$
Diagnosis*	$F(1, 22) = 4.35$, $p = .05$, $r = .41$
Intervention x Diagnosis	$F(1, 22) = 0.59$, $p = .45$, $r = .16$

* $p = .05$. N = 26. *Note*. The other ANOVA values such as sum of squares, mean square, and errors are not reported as these values are smaller than 0.00.

Discussion

The purpose of this chapter was to investigate whether individual differences, measured in terms of GSR and EEG and indexed by their scaling exponents, could account for how children respond to the cognitive effects of an acute physical exercise activity. Specifically, this chapter investigated whether the scaling exponents of GSR and EEG data could be related to the accuracy performance following physical exercise. This study found that the scaling exponent of the GSR measure, but not the EEG measure, is related to the cognitive progress or decline in accuracy on tests of implicit learning and executive attention. The hypothesis that children who demonstrated cognitive improvement would have a scaling exponent of DFA $\alpha = 1.0$, or 1/f noise, compared to those who did not improve (i.e., deviation from DFA $\alpha = 1.0$) was not supported. Furthermore, the hypothesis that children with a neurodevelopmental condition would have a scaling exponent different to DFA $\alpha = 1.0$, compared to those with typical development, was only partially supported.

Electroencephalogram (EEG) Findings

This study did not find any significant differences in the scaling exponent as a result of intervention, diagnosis, and accuracy change on the alpha, beta and theta frequency bands. The scaling exponent across the EEG frequency bands found in this study are in general, $\alpha \approx 1.0$, or 1/f noise. The scaling exponents (EEG) are, however, consistent with previous research. Studies have typically found that brain waves in conscious humans, measured by the EEG, have a scaling alpha close to 1.0 (e.g., Ferri et al., 2005; Lee et al., 2004), and can fluctuate increasingly above 1.0 during various sleep stages and return to baseline upon awakening. In addition, EEG studies do report changes in scaling exponents in very short epochs (i.e., 30 seconds), for example, from Brownian to 1/f noise (see Ferri et al., 2005). Further DFA was conducted on shorter time periods of 2 minutes, 1 minute, and 30 seconds for some of the participants (see Appendix L, Table 31 - 36), but the scaling exponents did not deviate appreciably from those obtained in the 3-minute periods. Therefore, there is a high likelihood that the non-significant findings in this study may be due to the rest period (i.e., 10 minutes) given to the participants after physical exercise prior to the EEG measurement, resulting in the scaling exponent returning swiftly to baseline (see Chang, Labban et al., 2012, regarding the duration of the acute exercise effect).

Galvanic Skin Response (GSR) Findings

In Chapter 4, the effects of physical exercise and tablet activity significantly differed on accuracy measures of the ISLT and CRSD-ANT incongruent flanker trials. Similarly, the scaling exponent (GSR) after an intervention (physical exercise) was significantly related to the performance on these accuracy measures. In particular, after receiving the physical exercise, children with a higher scaling exponent made more errors on the ISLT, compared to those who had a lower scaling exponent. Contrary to the results of the ISLT, among children who received the exercise activity, a lower scaling exponent was found to be related to more

errors made on the incongruent flanker trials than a higher scaling exponent. Tablet activity, however, was not related to the accuracy change of the ISLT and incongruent flanker trials. The difference in the directionality of the scaling exponent in relation to physical exercise and cognition, could be better interpreted in the context of what scaling exponents mean.

As indicated in the presentation of complexity theory in Chapter 1, where a scaling exponent (α), as analysed by the detrended fluctuation analysis (DFA), can range from 0 to 2.0, and represents a physiological signal lying between white and brown (Brownian) noise (Kantelhardt, 2008; Peng et al., 1995; Stadnitski, 2012; Stroe-Kunold et al., 2009), respectively. In this study, participants in the physical exercise group had scaling exponents (GSR) ranging from above 1.0 to 2.0, before, during and after receiving the exercise activity. Thus, the scaling exponents for this group of children lies in the range of Brownian noise. The Brownian characteristic of GSR is consistent with what was found by Wijnants et al. (2013), where the scaling exponent of GSR was reported to be in the Brownian range. The Brownian characteristic of GSR is related to its underlying physiology.

Contrary to other physiological systems like the heart, where its regulation (e.g., heart rate) is a combined function between the parasympathetic and sympathetic nervous systems (SNS; Ivanov et al., 1998), galvanic skin response is solely regulated by the SNS (Critchley, 2002). In the field of cardiac dynamics, researchers have concluded that the parasympathetic and sympathetic nervous systems function in opposition to influence the scaling properties of the heart (e.g., Castiglioni et al., 2011; Ivanov et al., 1998; Karasik et al., 2002). Specifically, the parasympathetic nervous system (PNS) behaves in the range of a white noise, as opposed to the Brownian noise exhibited by the SNS (Castiglioni et al., 2011). As the competing function of parasympathetic and sympathetic systems work in tandem, as in the case of heart rate dynamics in healthy individuals, the scaling exponent tends to be around 1.0, or 1/f noise (e.g., Heffernan et al., 2008; Schmitt & Ivanov, 2007), though variability in the scaling

exponent exists even among seemingly healthy individuals (Heffernan et al., 2008). Overall, previous research supports the idea that a healthy physiological system tends to have a scaling exponent around 1.0, whereas an abnormality or dysfunction within the system has a scaling exponent deviated towards white or Brownian noise (e.g., Esteban et al., 2007; Heffernan et al., 2008; Peng et al., 2000; Sandu et al., 2008).

A time series data that behave as a 1/f noise has characteristics of both white and Brownian noise (Gisiger, 2001). On one hand, a white noise is categorised by data fluctuations that are unpredictable, erratic and irregular (Kloos & Van Orden, 2010). On the other hand, Brownian noise refers to data that fluctuate in a manner that is highly predictable, stable and regular (Kloos & Van Orden, 2010). In terms of cardiac dynamics, 1/f noise is observed due to the simultaneous input from the parasympathetic (i.e., white noise) and sympathetic (i.e., Brownian noise) nervous systems (e.g., Castiglioni et al., 2011; Heffernan et al., 2008; Schmitt & Ivanov, 2007). The concurrent physiological contributions from both nervous systems are typically observed in a healthy individual. Conversely, when inputs from the PNS and SNS are not synchronised, such that one of the nervous system dominates, the data fluctuation behaviour will deviate from the 1/f noise to either a white noise (i.e., PNS input > SNS input) or that of Brownian noise (i.e., SNS input > PNS input). Such deviations from 1/f noise are typically observed in individuals with a medical condition, such as cardiac issues or abnormalities. An individual with a cardiac condition had an unequal PNS and SNS contributions resulting in changes to the way a cardiac parameter (e.g., heart rate variability) fluctuates (i.e., Brownian noise), which reflects a decreased capacity to respond to external stressors (Goldberger et al., 2002; Heffernan et al., 2008; Platisa & Gal, 2008).

It is not appropriate, however, to interpret a deviation of scaling exponents from 1/f noise as representing a pathological state when the GSR measure is considered. As the GSR is predominantly driven by the SNS (Critchley, 2002), the scaling exponent should behave as

Brownian noise (see Castiglioni et al., 2011, on the influence of the SNS; Wijnants et al., 2013). Indeed, Wijnants et al. (2013) reported that GSR was found to have data fluctuations that behave as Brownian noise in a group of typical developing young adults. Similarly, in the current study, the GSR scaling exponents were in the Brownian range, and did not differ in children with a typical development and those with a neurodevelopmental condition. Since the GSR scaling exponent could only behave as Brownian noise in typical developing children and children with a neurodevelopmental condition, the interpretation of Brownian noise as representing a pathological state cannot be substantiated in the measurement of GSR.

The Brownian behaviour of the GSR measure may be theoretically functional for the arousal system responsible for the GSR signal that is particularly responsive to intrinsic and extrinsic stimuli (e.g., emotions, fear-provoking stimulus, and cognitive task demand) (Critchley, 2002). Coincidentally, the highly predictable, stable and regular characteristics of the Brownian noise (Kloos & Van Orden, 2010) are consistent with the GSR that is sensitive to intrinsic and extrinsic stimuli (Critchley, 2002). Thus, since the GSR scaling exponent could only vary within the Brownian range (i.e., $\alpha \approx$ above 1.0 to 2.0), the range of the GSR scaling exponent that is allow to vary may be conceptualised as an indicator of the level of the responsiveness of the underlying arousal system. In other words, the higher the GSR scaling exponent (i.e., towards $\alpha \approx 2.0$), the more responsive the arousal system is to intrinsic and extrinsic stimuli. Conversely, the lower the GSR scaling exponent (i.e., towards $\alpha \approx 1.0$), the less responsive of the system that generated the GSR to arousing stimuli.

Overall, the results of this study indicate that physical exercise generally resulted in a higher GSR scaling exponent compared to the tablet activity. However, the relationship between the post-exercise GSR scaling exponent and the accuracy performance of the ISLT and incongruent flanker trials, is not unidirectional. Specifically, following physical exercise, children who made more errors on the ISLT had a larger scaling exponent relative to those

who did not make more errors. The opposite direction, however, was found for the incongruent flanker trials. Among children who exercised, those who had a lower scaling exponent made more errors on the incongruent flanker trials than those who had a higher scaling exponent. The bi-directionality of the relationship between post-exercise GSR scaling exponents and accuracy performance could be explained via an interplay between an individual's arousal system (i.e., sensitivity) and cognitive task demands.

The influence of individual differences and task demands are not new to the physical exercise and cognition literature (see Pesce, 2009). Studies in this area generally support the notion that physical exercise tends to have a facilitating effect particularly on cognitive tasks that are demanding, like executive function tasks (e.g., Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008), compared to simpler tasks. Furthermore, individual factors, such as fitness and health conditions are known to moderate the cognitive effect of physical exercise (e.g., Chang, Labban et al., 2012; Crova et al., 2014), though findings are mixed. Several researchers have acknowledged the interplay between individual differences and cognitive task demands in the physical exercise and cognition relationship (e.g., Chang, Labban et al., 2012; Pesce, 2009). However, this interplay has been known to be particularly difficult to disentangle or comprehend (e.g., Etnier, 2009; Pesce, 2009).

The cognitive tasks used in this study differed in the level of cognitive demands, such that a higher cognitive demand is needed for the CRSD-ANT incongruent flanker trials (see Chang, Pesce, Chiang, Kuo, & Fong, 2015), contrary to the ISLT (i.e., lower cognitive demand). The incongruent flanker trials measure executive attention which is the ability to resolve conflicting information (Fan & Posner, 2004). Hence, a highly responsive arousal system, indexed by a high GSR scaling exponent, would be theoretically useful to detect incongruent flankers. This point was supported in this study, such that following the exercise activity, children with a lower scaling exponent (i.e., less sensitive arousal system),

performed poorer on the incongruent flanker trials than children who had a higher scaling exponent (i.e., high sensitive arousal system).

The ISLT is a simple reaction time task meant to tap implicit learning processes (Chambaron et al., 2008; Shanks et al., 2005). This study showed that children with a higher GSR scaling exponent after exercising made more errors on a simpler, less cognitive demanding task like the ISLT. This result suggests that a highly responsive arousal system may not be suited to a task with few cognitive demands. For example, an overly-sensitive arousal system may be distracted by every aspect of the simple task, which may affect performance.

The results of this study may help explain inconsistencies in the cognitive effect of physical exercise that have been reported in previous research. First, it has been found in this study that physical exercise generally increased the GSR scaling exponent or sensitivity of the arousal system, and was related to better performance on the CRSD-ANT incongruent flanker trials but not on the ISLT. This finding is consistent with research that reports the facilitating effect of physical exercise is selective towards those tasks that are cognitively demanding (i.e., executive function tasks) (e.g., Etnier, 2009; Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008). Second, the effect of physical exercise is also dependent on individual differences (Diamond & Ling, 2016; Pesce, 2009). Indeed, this study found that children who performed the physical exercise may not necessarily showed improved cognitive performance, even in the cognitive demanding task (i.e., incongruent flanker trials). Further, this point is also supported by the finding that some children's GSR scaling exponent or sensitivity of the arousal system, did not increase following the exercise activity (i.e., low scaling exponent).

Third, the physical exercise and cognition relationship was dependent on the interplay between individual variability and cognitive task demands (Pesce, 2009). This study found a

significant relationship between an individual's arousal system (i.e., as indicated by GSR scaling exponents) and cognitive performance following physical exercise. Specifically, although a high scaling exponent was related to better performance on the incongruent flanker trials (CRSD-ANT), it was also negatively related to the performance on the implicit learning task (ISLT). Conversely, a lower scaling exponent was related to better performance on the ISLT but poorer performance on the incongruent flanker trials. These results suggest that a compatibility between individual differences, in terms of the sensitivity of the arousal system, and task demands may be necessary to observe a facilitation effect of exercise on cognitive performance. Thus, non-significant findings of the effects of physical exercise on some aspects of cognition reported by previous research (e.g., Craft, 1983; Tomporowski, Davis, Lambourne et al., 2008) may be partly due to an incompatibility between individual variability and cognitive task demands (Pesce, 2009).

It is important to highlight that the sensitivity of an arousal system, measured by the GSR and indexed by the scaling exponent, does not reflect the absolute GSR level. As reported earlier in the results section, a high or low GSR level, measured in μS, is not equivalent to the value of the scaling exponent. Furthermore, it has been shown in this study, that the absolute GSR level is not related to cognitive performance (i.e., accuracy change), unlike the GSR scaling exponents. Thus, regardless of an individual's absolute GSR level, the changes in the sensitivity of the arousal system as a response to physical exercise are related to the accuracy performance observed on the implicit learning and attention network tasks. Specifically, whether or not an individual is likely to have a cognitive improvement after physical exercise is related to changes in his/her arousal system (i.e., sensitivity), and the nature of the cognitive task (i.e., high versus low demand).

Interestingly, the ability of the interplay between an individual's arousal system and the demands of a cognitive task to account for the physical exercise and cognition

relationship is limited to the first time the participant undertakes the exercise. When the same physical exercise sequence is performed by the participants in the subsequent session, the significant interaction between individual variability in the arousal system and task demand on the cognitive performance ceased to exist. Nevertheless, some participants still improved or maintained their cognitive performance after receiving the physical exercise for the second time (see Tables 17 and 18 at the end of this chapter). Therefore, it is not that the cognitive effect of physical exercise does not occur in repeated sessions. Rather, the effect of physical exercise on cognition no longer relies on the interplay between an individual's arousal system and task demands, when the same exercise sequence is repeated.

Since the facilitation effect of exercise on cognition no longer depends on the sensitivity of the arousal system and cognitive task demand upon repeated exercise with the same sequence, there is a possibility that other factors may have influenced the exercise-cognition effect. Coincidentally, the effect of diagnosis, however, only occurred in the subsequent session of the incongruent flanker trials. The findings revealed that children with a typical development had a significant increase in the scaling exponent after performing the physical exercise. Conversely, for children with a neurodevelopmental condition, no changes in the scaling exponent was found after performing the exercise activity.

Alternatively, as the human physiological system is particularly adaptive to stress, especially from repeated physical exercise (Marosi & Mattson, 2014; van Praag, Fleshner, Schwartz, & Mattson, 2014), physiological changes may have also exerted an influence on the effect of exercise on cognition upon repeated exercise activity.

On the whole, then, the influence of an individual's arousal system and cognitive task demand interplay seems to be applicable only to novel exercise activity. Such a finding suggests that varying the sequence of the exercise intervention each time it is given to the children (e.g., see Chapter 3, starting the basketball activity from step six to one, or step one,

three, and five etc.) may optimise the cognitive effect of physical exercise (e.g., see Pesce, Croce et al., 2016). Hence, there is a possibility that in future research, the exercise-cognition effect could be predicted in subsequent sessions through the experimental manipulation of the interactions between the cognitive task demand, exercise sequence, and an individual's arousal system.

Limitations/Future Studies

Although this is the first study, to the author's knowledge, that demonstrated that the scaling exponent of the GSR measure could account for a child's cognitive performance following physical exercise, several limitations must be taken into consideration. First, even though there is a practical difficulty in controlling for the number of participants that would or would not show improvements in the cognitive tasks a priori, the unequal sample sizes, particularly for those who made task errors in the exercise activity group (e.g., 4 out of 15 children on the CRSD-ANT in session 1; see Tables 17 and 18 at the end of this chapter), must be noted. Nevertheless, the significant relationship between the post-exercise GSR scaling exponent and cognitive performance specific to the ISLT and CRSD-ANT (i.e., incongruent accuracy trials) was consistent with the findings reported in the previous chapter.

Additionally, the interplay found in this study between individual variability and task constraints on the physical exercise and cognition relationship was also consistent with previous research. Furthermore, the finding that the absolute GSR level (μS) was not related to cognitive performance, unlike the GSR scaling exponent, further strengthens the validity of the results. Specifically, the scaling exponent of the GSR measure is a feasible index of the arousal system that is related to the cognitive effect of physical exercise. Nevertheless, in view of the small sample size, the findings from this study should be considered as an exploratory study. Thus, future studies with a large sample size could validate whether the

scaling exponent derived from the GSR is related to the physical exercise and cognition relationship.

Secondly, detrended fluctuation analysis (DFA) is not the only fractal analysis available in the research literature, and other methods including spectral analysis (Goldberger et al., 2000; Wijnants, Cox, Hasselman, Bosman, & Van Orden, 2012), approximate entropy (Ho et al., 1997), wavelet-based multifractal analysis (Ihlen & Vereijken, 2010) and others (see Stroe-Kunold et al., 2009) are also used for analysing the fractal dimension of physiological data (i.e., time-series). Although DFA has been used extensively in previous physiological studies (e.g., Castiglioni et al., 2011; Lee et al., 2007; Stam et al., 2005; Wijnants et al., 2012), future research could consider validating the scaling properties of GSR reported in this project by using other fractal analysis methods, such as spectral analysis.

Recently, neurocognitive researchers studying fractal behaviours and human cognition (e.g., Ihlen & Vereijken, 2010; Zorick & Mandelkern, 2013) have begun to shift the analysis of time series data from the use of a monofractal analysis (e.g., DFA, spectral analysis) to the multifractal analysis method (e.g., wavelet-based multifractal analysis). The main difference between monofractal and multifractal analysis lies in whether the physiological data is characterised by a single or multiple scaling exponents, respectively (Stanley et al., 1999). Hence, multifractal analysis would provide more complex information about the physiological data than a monofractal analysis, by revealing more in-depth underlying processes (see Ihlen & Vereijken, 2010; Stanley et al., 1999; Zorick & Mandelkern, 2013). As the current project is an exploratory study, multifractal analysis was not adopted. However, this study found a significant relationship between the scaling exponent and whether or not a child demonstrated a cognitive effect of exercise. Thus, future investigations on the fractal behaviours (i.e., mono- or multifractal) of physiological measures, and its contribution to the exercise-cognition relationship is warranted.

Lastly, as there is a high possibility that the non-significant findings pertaining to the scaling exponent of the EEG data could be a result of the delay in recording the data, future studies may need to consider shortening or removing the resting period after exercise. Additionally, due to technological limitations, the lack of significant findings in EEG data may also be due to the lack of channels in the Emotiv device surrounding the central scalp region that may be important for the physical exercise and cognition relationship (i.e., sensory-motor).

Conclusion

This chapter concluded the third aim of this research project that is to investigate the psychophysiological measures to account for the cognitive effect of an acute exercise activity. To conclude, the interaction between individual variability and task demands is not new to the physical exercise and cognition literature. However, the method of exploring the scaling exponent of GSR, as an index of individual differences, in accounting for the effects of physical exercise is novel. Specifically, this study has demonstrated the feasibility of investigating the scaling exponents of the arousal system, measured via the GSR. Further, the GSR scaling exponent has the potential to account for the influence of individual differences and task demands on the exercise and cognition relationship. In the final chapter, the implication of the findings from this research project are discussed further in the context of the physical exercise and cognition literature.

Table 17

Unadjusted and Adjusted Means, and Standard Deviations for Group Conditions as a Function of Accuracy Change (ISLT), with Pre-Intervention and During Intervention Scaling Exponents as Covariates

	Accuracy Change					
	Increased Error			Reduced/Static Error		
Group Conditions	Number of Children	*M*	*SD*	Number of Children	*M*	*SD*
Session 1						
Unadjusted means						
Exercise activity	4 (25%)	1.8	0.05	12 (75%)	1.6	0.26
Tablet activity	11 (58%)	1.3	0.16	8 (42%)	1.4	0.18
Adjusted means						
Exercise activity	4 (25%)	1.9	0.20	12 (75%)	1.5	0.21
Tablet activity	11 (58%)	1.4	0.19	8 (42%)	1.4	0.20
Session 2						
Unadjusted means						
Exercise activity	8 (50%)	1.6	0.27	8 (50%)	1.6	0.21
Tablet activity	15 (79%)	1.4	0.25	4 (21%)	1.3	0.15
Adjusted means						
Exercise activity	8 (50%)	1.6	0.25	8 (50%)	1.5	0.23
Tablet activity	15 (79%)	1.4	0.23	4 (21%)	1.3	0.26

Note: $N = 35$. Exercise activity group ($n = 16$), Tablet activity group ($n = 19$). The above presented means are GSR scaling exponents.

Table 18

Unadjusted and Adjusted Means, and Standard Deviations for Group Conditions as a Function of Accuracy Change (CRSD-ANT: Incongruent Flanker Trials), with Pre-Intervention and During Intervention Scaling Exponents as Covariates

	Accuracy Change					
	Increased Error			Reduced/Static Error		
Group Conditions	Number of Children	*M*	*SD*	Number of Children	*M*	*SD*
Session 1						
Unadjusted means						
Exercise activity	4 (27%)	1.4	0.29	11 (73%)	1.7	0.18
Tablet activity	6 (33%)	1.4	0.22	12 (67%)	1.3	0.13
Adjusted means						
Exercise activity	4 (27%)	1.3	0.22	11 (73%)	1.7	0.20
Tablet activity	6 (33%)	1.4	0.27	12 (67%)	1.4	0.21
Session 2						
Unadjusted means						
Exercise activity	8 (53%)	1.7	0.23	7 (47%)	1.5	0.23
Tablet activity	10 (56%)	1.3	0.26	8 (44%)	1.4	0.21
Adjusted means						
Exercise activity	8 (53%)	1.7	0.20	7 (47%)	1.4	0.29
Tablet activity	10 (56%)	1.4	0.22	8 (44%)	1.4	0.20

Note: $N = 33$. Exercise activity group ($n = 15$), Tablet activity group ($n = 18$). The above presented means are GSR scaling exponents.

Chapter 6: Consolidation

This research project sought to investigate the relationship between physical exercise and cognition. To understand this relationship, three approaches were taken to investigate the effects of physical exercise on cognition, and the possible mechanism underlying this effect. First, a meta-analysis was conducted to determine the efficacy of exercise interventions on cognition in individuals with a neurodevelopmental disorder. The goal of this meta-analytic review was to determine if physical exercise is effective in facilitating cognitive improvements in individuals with autism spectrum disorders (ASD) and/or with an attention-deficit/hyperactivity disorder (ADHD). Additionally, this review also sought to link the exercise-cognition research conducted in the neurodevelopmental population with those reported in the general population.

Second, an experimental study was conducted to compare the after-effect of an acute physical exercise activity against a tablet game activity on measures of implicit learning and attention. This experiment was designed to determine if an exercise activity with components of motor coordination and cognitive engagement, would be comparable to a cognitively-engaging tablet activity in their effects on cognition. Furthermore, the effects of the exercise or tablet activity was compared between children with and without a neurodevelopmental condition, to investigate the influence of diagnostic status.

Third, to investigate the mechanism that might be responsible for the after-effects of an acute physical exercise activity on cognitive performance, GSR and EEG measures were analysed with detrended fluctuation analysis. The goal of this study was to determine if GSR and EEG, as indexed by their scaling exponents, could account for the children's cognitive performance following the exercise activity. Together, the three approaches of this research project were aimed at furthering the understanding of the exercise and cognition relationship, particularly in children with and without a neurodevelopmental condition. This chapter

provides summaries of the main findings of this research project, and discusses the findings within the context of previous research.

Efficacy of Exercise: Summary of the Meta-Analytic Review

The meta-analytic review reported in Chapter 2 evaluated 22 experimental studies from the neurodevelopmental research to determine the efficacy of physical exercise interventions on cognitive performance. Based on the meta-analytic findings, exercise on cognition was found to have a small-to-medium sized effect in young individuals aged 3–25 years, with ASD and/or ADHD. The findings also supported the efficacy of exercise interventions on cognition in individuals with a neurodevelopmental disorder. Furthermore, the findings were consistent with those reported in the general population that the magnitude of the cognitive effects of exercise is moderated by the type of cognitive tasks, and that some individuals may not demonstrate cognitive improvement with exercise.

Effects of Exercise: Summary of the Experimental Study

The experimental study reported in Chapter 4 contrasted the after-effect of an acute exercise activity with a tablet game activity on measures of implicit learning and attention. This study involved children aged 6-11 years, of which 15 children had a neurodevelopmental condition and 20 children had a typical development. The study found that the effect of exercise was, in general, comparable to the tablet activity on reaction time measures but not on the accuracy of the implicit learning and attention network tasks (i.e., conflict network). Specifically, regardless of diagnostic status, children typically made more errors on the implicit sequence learning task after receiving the tablet activity compared to those that received the exercise activity.

Additionally, following the tablet activity, children with a neurodevelopmental condition performed poorer particularly on the incongruent flanker trials relative to baseline performance. This trend, however, was not observed in children with a typical development,

where fewer errors were made following the tablet activity compared to baseline performance. Contrary to the tablet activity, following the exercise, both those children with a neurodevelopmental condition and those with a typical development were able to maintain their accuracy performance on the implicit learning and attention network tasks. In summary, exercise activity was generally better than a tablet activity in enhancing cognition, especially in children with a neurodevelopmental condition.

Mechanisms Underlying the Effect of Exercise: Summary of the Psychophysiological Investigation

In Chapter 5, a psychophysiological investigation examined the GSR and EEG measures to complement the findings of the experimental study reported in Chapter 4. The investigation found that GSR, but not EEG, was related to cognitive performance on the implicit learning and attention network tasks. Consistent with the findings from the experimental study, GSR indexed by its scaling exponent was related to performance on the accuracy measures of the implicit learning and conflict network tasks (i.e., incongruent flanker trials). This study found that whether a child improves or maintain performance on the cognitive tasks was related to the changes in his/her arousal system that occurred in response to physical exercise. These changes were indicated by the scaling exponent of the GSR, which is theorised to be an index of the level of sensitivity of the arousal system, such that the higher the scaling exponent, the higher the sensitivity.

The relationship between physical exercise and cognition is indeed complex. The findings from this study suggests that the cognitive effect of an acute exercise activity is dependent on the interplay between an individual's arousal system, cognitive task demand, and the novelty of the exercise activity. The results demonstrated that, overall, the scaling exponent of the GSR, was significantly elevated following physical exercise relative to the tablet activity. However, an elevated scaling exponent was only related to better accuracy

performance on the more demanding incongruent flanker trials but not on the simpler, implicit learning task. Conversely, a lower scaling exponent was related to better accuracy performance on the implicit learning task than a higher scaling exponent. This interplay between the scaling exponent and task demand was also limited to when participants first exercised. In summary, this study suggests that the facilitating effect of acute exercise on cognition is a result of the interaction between an individual's arousal system, cognitive task demand and the novelty of the exercise activity.

General Discussion

Based on the overall findings, two common themes emerged consistently across the various approaches undertaken by this research project: individual and task variability. The findings of the meta-analysis, experimental study and psychophysiological investigation in this research project indicate that the relationship between physical exercise and cognition is moderated by individual differences and cognitive task demands. Recent reviews and experimental studies have highlighted the influence of individual and task variability on the exercise and cognition relationship (e.g., Diamond & Ling, 2016; McMorris et al., 2009; Morris et al., 2017; Pesce, 2009). However, there has been little research focus on the influence of individual differences to the relationship between exercise and cognition (McMorris et al., 2009, p 314).

The research literature on physical exercise and cognition has evolved from a focus on the quantitative aspects of physical exercise to a focus on qualitative exercises. As introduced in Chapter 1, quantitative and qualitative exercises differ in the movement complexity of the exercise activity and the level of cognitive engagement that results from exercising (Pesce 2012; Tomporowski et al., 2015). Quantitative types of exercise are based on simple physical movements contrary to qualitative types of exercise that contain complex motor coordination. Further, quantitative exercises result in a low cognitive engagement

compared to the high cognitive engagement derived from performing qualitative exercises. Recently, a new movement has emerged from the proponents of qualitative physical exercise, which proposes an ecological approach towards a holistic exercise activity (Pesce, Croce et al., 2016; Pesce, Masci et al., 2016; Pesce, Leone, Motta, Marchetti, & Tomporowski, 2016), by focusing on improving executive functions and motor skills, concurrently.

In an extensive review, Pesce, Croce et al. (2016) incorporated developmental and learning theories from the field of motor skills acquisition and neurocognitive science research to provide a theoretical framework for the effects of chronic physical exercise on executive functions. The ecological approach focuses on the variability in the components of physical exercise that are required to facilitate cognition improvements, particularly with executive functions. In the proposed framework, Pesce, Croce et al. (2016) highlighted the need to vary the components of exercise to prevent habituation of cognitive engagement, to maintain a process of challenging executive functions involved during physical exercise. Consequently, cognitive improvements are hypothesised in areas of executive functions (e.g., inhibition) that are challenged during the exercise activity (Best, 2010; Diamond & Ling, 2016; Moreau & Conway, 2013; Pesce, 2012; Pesce, Croce et al., 2016; Tomporowski, Horvat, & McCullick, 2010).

Similar to the quantitative and qualitative physical exercise approach, the ecological approach also focuses on the search for a set of optimal exercise parameters that can best improve cognition. In addressing the influence of individual differences, researchers have focused their efforts on locating an ideal exercise intervention that is specifically titrated to suit various clinical populations, such as individuals with Alzheimer's disease (e.g., Morris et al., 2017), schizophrenia (e.g., Firth et al., 2017), Parkinson's disease (e.g., Caciula et al., 2016) and overweight children (e.g., Gallotta et al., 2015). Although such efforts are indicated and important, there are three issues that require consideration.

Quantitative Exercise versus Qualitative Exercise

The first issue is the mixed research findings regarding the superiority of quantitative exercise versus qualitative exercise in enhancing cognitive functions. Previous research has demonstrated that different exercise parameters have varying effects on cognition (e.g., Chang, Labban et al., 2012; McMorris & Hale, 2012; Moreau, Morrison, & Conway, 2015), though these differences may also be attributed to the type of comparison groups or conditions (Best, 2010; Vazou et al., 2016). Large effect sizes are observed when physical exercises are compared with sedentary or waitlist control groups/conditions (Vazou et al., 2016). However, the cognitive effect of quantitative versus qualitative types of exercise are unclear. Previous studies either reported larger effects of exercises with qualitative components over quantitative exercises (Gallotta et al., 2015; Moreau et al., 2015), or quantitative exercises over exercises with qualitative components (Best, 2012; O'Leary et al., 2011), or no difference between both type of exercises (e.g., Vazou et al., 2016; Van den Berg et al., 2016).

Moreau et al. (2015) evaluated a working memory task performance in 67 participants aged 18-52 years. The participants were separated into three groups that either performed a simple aerobic exercise, an enriched exercise with complex motor coordination, or cognitive training. Following eight weeks of intervention, Moreau et al. reported the largest improvement on working memory for participants in the enriched exercise group, followed by those who received the cognitive training, and then the simple aerobic exercise group. Similarly, Gallotta et al. (2015) concluded that the accuracy measures of an attention task greatly improved in a qualitative exercise group relative to a quantitative exercise group in 157 primary school children. However, baseline group differences in cognitive performance between children who performed the quantitative exercise and those that were engaged in the qualitative exercise are an important consideration for Gallotta's study. Furthermore, based

on the reported means, the quantitative exercise group (M = 5.36%, SD = 5.06) had similar, if not better, performance on the error measure than the qualitative exercise group (M = 5.89%, SD = 3.19), after performing the respective exercise interventions. Hence, despite the amount of change in measures of attention reported by Gallotta et al. indicating a larger improvement for children in the qualitative exercise group compared to the quantitative exercise group, the differences may be due to better performance at baseline for participants in the quantitative exercise group (i.e., ceiling effect).

Contrary to Moreau et al.'s (2015) and Gallotta et al.'s (2015) findings, O'Leary et al. (2011) investigated the cognitive effects of a 20-minute simple treadmill activity compared with a challenging exergame (i.e., aerobic exercise and video game), video game activity, and a resting condition in a group of 36 young adults aged 18-25 years. The authors found significant improvements on executive control only in the simple treadmill condition. Consistent with this finding, Best (2012) reported greater improvement on executive control in 33 children ranging from 6 to 10 years old, after receiving the simple exergame condition (i.e., aerobic exercise only), compared to the challenging exergame (i.e., aerobic exercise and video game), and control conditions. Although these exercise studies (Best, 2012; O'Leary et al., 2011) were delivered via different modalities (i.e., jogging on a treadmill or an exergame), they suggest that the quantitative aspects of exercise are responsible for improving cognition, particularly with regard to inhibition.

In the current experimental study, children aged 6-11 years performed a 12-minute moderate-intensity exercise via a series of coordinative movements with a basketball. In terms of the reaction time measures of the implicit learning and attention tasks, the performance of children in the exercise group was comparable to children that received a 12-minute tablet game activity. Nevertheless, the performance on the accuracy measures was generally better in children who performed the exercise activity than children in the tablet

activity group. As this study did not include a quantitative physical exercise as a comparison, it is difficult to conclude which type of exercise (i.e., quantitative versus qualitative) is better than the other in enhancing cognition. However, the results from this study suggest that the cognitive effect of a qualitative physical exercise is relatively larger compared to a cognitively-engaging tablet game activity, particularly on the accuracy measures.

The influence of the exercise characteristics on cognition is further complicated by a recent meta-analytic review showing that there are no significant differences between quantitative and qualitative exercises on cognitive performance (Vazou et al., 2016). Indeed, in some studies, cognitive improvements were found, regardless of the magnitude, in both quantitative and qualitative exercises (e.g., Budde et al., 2008; Gallotta et al., 2015). However, there may be a difficulty in investigating the effects of pure quantitative or qualitative types of exercise on cognition, as components including motor coordination and cognitive engagement are likely to overlap in both types of exercise (Vazou et al., 2016). Furthermore, the inconsistent findings between both types of exercise may be due to factors beyond the exercise activity, and one possibility is the optimal challenge point that varies among individuals (Guadagnoli & Lee, 2004; Pesce et al., 2013).

Non-Responders to the Cognitive Effect of Exercise

The second issue lies with the existence of some individuals who are non-responsive to the exercise-induced cognitive effect, which may be explained by the optimal challenge point (Guadagnoli & Lee, 2004; Pesce et al., 2013). The optimal challenge point was originally conceptualised as a theoretical framework to understand the relationship between practice conditions and motor learning (see Guadagnoli & Lee, 2004). According to Guadagnoli and Lee, the optimal challenge point is a conceptual point when a task difficulty matches an individual's skill level, such that motor learning is most optimal for that individual. Pesce et al. (2013) extended the concept of the optimal challenge point to the

exercise-cognition relationship, where a maximum cognitive benefit is assumed when an ideal exercise matches an individual's motor skill level. Importantly, the optimal challenge point is depended on an individual's motor development and age. As such, the optimal challenge point differs among individuals.

Indeed, the findings from this research project together with previous research (e.g., Audiffren, 2009; Kramer & Erickson, 2007), have shown that not every individual exhibit improved cognition following physical exercise. The current experimental study found that 24% of the children with a typical development and 30% of those with a neurodevelopmental condition did not exhibit a facilitating effect of exercise on cognition. Additionally, the meta-analytic findings from this research project also reported that 24-41% of individuals with a neurodevelopmental condition were estimated to be non-responsive to the cognitive effect of exercise. Therefore, individuals who do not demonstrate cognitive improvements with exercise exist, and cannot be ignored. Although exercise is beneficial for cognitive health, there is also a need to focus on why some individuals do not demonstrate a cognitive benefit following exercise. On the whole, there is evidence that the cognitive effect of exercise differs among individuals.

Since exercise interventions are typically standardised within an experiment, the causal factor that determines whether participants show an improvement or reduction in cognitive performance cannot be solely attributed to the effect of physical exercise. Rather, the main factor that influences whether an individual would experience an exercise-induced cognitive benefit is individual differences (Pesce, 2009; Pesce, Masci et al., 2016). Indeed, the meta-analytic findings from this research project corroborated the findings reported by Pesce, Masci et al. (2016) that both quantitative and qualitative types of exercise account only for a small amount of variance in the exercise-cognition relationship. Hence, the experimental manipulation of exercise parameters, including duration, intensity, cognitive engagement and

motor coordination cannot account for whether individuals would exhibit an exercise-induced cognitive effect. Although the optimal challenge point may explain why some individuals do not demonstrate cognitive improvements following exercise, there is no clear indication on how this factor can be measured.

Measuring the Optimal Challenge Point

The third issue concerns the lack of a practical method for investigating individual differences, or specifically, the optimal challenge point (Pesce et al., 2013). The quantitative and qualitative exercise-cognition research, and recently, the ecological approach, are important to the understanding of the exercise and cognition relationship. Although these approaches acknowledge the influence of individual differences, a practical method on how the optimal challenge point could be measured in the exercise-cognition relationship has not been proposed. The measurement of the optimal challenge point is important, especially to account for individuals who do not respond to the cognitive effect of exercise. However, exercise-cognition researchers tend to focus on the quantitative or qualitative aspects of physical exercise over individual differences (e.g., Gallota et al., 2015; Masley et al., 2009; Ruscheweyh et al., 2011; Schmidt et al., 2015). Furthermore, it is plausible that individuals who do not respond to the effect of exercise may remain non-responsive, regardless of the exercise parameters. Therefore, the search for the ideal exercise intervention may be an endeavour that benefits only those who would respond to the effect of exercise. Moreover, the current exercise-cognition literature does not provide an indication of the likelihood of whether or not an individual would demonstrate a cognitive effect after exercising. Hence, over-focussing on locating the ideal exercise intervention may restrict understanding of both the effects and mechanism underlying the exercise-cognition relationship.

Scaling Exponent as an Index of the Optimal Challenge Point

To further understand the mechanism underlying the exercise-cognition relationship, this research project focused on both those children that demonstrated an exercise-induced cognitive improvement, and those who did not exhibit a cognitive effect after exercising. This research project investigated the scaling properties of GSR and EEG measures through a detrended fluctuation analysis. Consistent with the optimal challenge point that is postulated to moderate the cognitive effect of exercise among individuals (Pesce et al., 2013), this research project suggests that the GSR scaling exponent could be an index of the optimal challenge point. The GSR scaling exponent (i.e., arousal system) was found to be related to children's accuracy performance on tasks measuring implicit learning and executive attention. The main findings suggest that whether a child improves in their cognition is dependent on how the child's arousal system changes in response to exercise. Children whose arousal system increased in sensitivity following exercise tended to improve or maintain their performance on the challenging incongruent flanker trials. Conversely, children whose arousal system remained relatively unresponsive to the exercise activity had an attenuation of their performance on the incongruent flanker trials.

Interestingly, this research project also found that those children whose arousal systems increased in sensitivity following exercise made more errors on the simple implicit learning task. This finding suggests that an arousal system with enhanced sensitivity following exercise may not necessarily benefit every cognitive task. This finding is also consistent with previous research, in that not every cognitive function is improved with physical exercise (e.g., Etnier, 2009; Kramer & Erickson, 2007; Tomporowski, Davis, Miller et al., 2008). Indeed, accumulating evidence suggests that the effect of physical exercise on cognition is more likely to benefit executive functions (e.g., Audiffren & Andre, 2015; Etnier, 2009; Kramer & Erickson, 2007; McMorris et al., 2009; Pesce, Croce et al., 2016;

Tomporowski, Davis, Miller et al., 2008), rather than global cognitive processes, though improvements in areas other than executive functions have been reported (e.g., Chang, Labban et al., 2012).

The interplay between an individual's arousal system and cognitive task demand is also affected by the novelty of the exercise activity (Klusmann et al., 2010; Moreau & Conway, 2013). This interplay ceased to hold when children repeated in performing the same exercise activity. Neurophysiological research suggests that the brain recruits executive function processes that peak during the initial stages of learning a novel task (Gentili, Bradberry, Oh, Hatfield, & Contreras-Vidal, 2011; Gentili, Shewokis, Ayaz, & Contreras-Vidal, 2013; see also, Pendleton, Sakalik, Moore, & Tomporowski, 2016, regarding mental engagement and heart-rate variability). With repeated practice, the brain gradually recruits fewer cognitive resources, suggesting a neurophysiological adaptation that occurs when individuals become skilful at a task. Consistent with the neurophysiological research, the ecological approach proposes that the exercise parameters need to vary each time the exercise is performed by the individuals to preserve the level of cognitive engagement (Pesce, Croce et al. 2016). The variability in the exercise intervention is postulated to maintain the involvement of various executive function processes and prevent neurophysiological adaptation, which results in post-exercise cognitive enhancement. As the participants in this project performed the same exercise parameters (i.e., the same sequence, movements, and the degree of challenge) in the second session, the level of cognitive engagement is theorised to be reduced according to the ecological approach (Pesce, Croce et al., 2016), leading to a neurophysiological adaptation (Gentili et al., 2011; 2013; van Praag et al., 2014). Indeed, when the exercise activity was repeated in the second session, the sensitivity of the arousal system and task demands no longer accounted for whether the children's cognitive performance improved or declined. Hence, variability of the exercise activity is also an

important factor in the exercise-cognition relationship (Pesce, Croce et al., 2016; Tomporowski et al., 2010).

The interaction between an individual's arousal system, cognitive task demand and the variability of the exercise activity has been observed in this project to be the likely factors underlying whether exercise enhances cognitive performance. These conditions are similar to the exercise-cognition pathways discussed in Best's (2010) review. Best conducted a review exploring the relationship between aerobic exercise and executive function development in children. Best stated that aerobic exercises can be considered as a form of cognitive training dependent on the movement complexity and the context in which the exercises are performed. According to the review, Best (2010) highlighted that there are at least three basic pathways by which exercise could affect cognition (i.e., executive function). The first pathway refers to the cognitive demands embedded within the context of the exercise activity, such as group sports or games. The context in which these activities are conducted requires cognitive effort and involves multiple executive function processes. For example, a team sports context involves strategy, planning, monitoring behaviours of self and other players, and a prompt reaction to situational changes during sports play, to fulfil the goal of the sports activity (e.g., winning). Thus, cognitive effort is required to perform in a cognitively challenging context (i.e., contextual interference; see Tomporowski et al., 2010). The second pathway refers to the cognitive demands needed to perform complex coordinative movements (Best, 2010). Pesce (2012) summarised the first two pathways from Best's review as cognitive engagement and motor coordinative components found in qualitative types of exercise. These two pathways may not be mutually exclusive as they both require cognitive effort that can be "activated" through qualitative exercises (Best, 2010; Pesce, 2012). The third pathway that exercise could influence executive function refers to the physiological changes (e.g., BDNF) that occur due to exercise (Best, 2010).

Although researchers can experimentally manipulate the exercise parameters to achieve the cognitive engagement and motor coordination pathways of the exercise-cognition relationship (Best, 2010; Pesce, 2012; Tomporowski et al., 2010), there is an inherent difficulty in accounting for the physiological pathway (i.e., individual differences). Qualitative exercise research tends to focus on manipulating the exercise parameters of cognitive engagement and motor coordination (e.g., Budde et al., 2008; Gallotta et al., 2012), but the physiological changes tend to be unaccounted. In other words, the physiological pathway proposal is mostly a research assumption that exercise should lead to the underlying physiological changes (e.g., BDNF). Although previous research supports exercise-induced physiological changes in animals (e.g., Adlard et al., 2005), and some research also demonstrated this in humans (e.g., Winter et al., 2007), there is yet to be conclusive evidence that these physiological changes are the mechanism by which exercise enhances cognition in humans (e.g., Barha et al., 2017). As discussed in Chapter 1, there are significant challenges to account for individual differences. Specifically, the issue of controlling for the influence of the many individual factors (e.g., fitness, BDNF) that could affect the exercise-cognition relationship pose a practical challenge for researchers.

The current research project proposed that the investigation of the GSR scaling exponent could be an index of the optimal challenge point. An important finding of this research project is that the effect of exercise on cognition is dependent on an individual's arousal system. Overall, the findings indicate that changes in the GSR scaling exponent in response to exercise were observed in those children who demonstrated a facilitating effect of exercise on cognition. Alternatively, children whose GSR scaling exponent remained unchanged following exercise failed to demonstrate a cognitive effect. In other words, if an individual's arousal system is responsive to exercise, that person is likely to exhibit improvements in cognition following exercise. The importance of an individual's arousal

system to the exercise-cognition relationship supports the argument that the effect of exercise on cognition is dependent on individual differences (Pesce, 2009; Pesce, Masci et al., 2016). Further, the findings of the GSR scaling exponent also suggest that the cognitive effects of exercise with components of cognitive engagement and motor coordination are dependent on the exercise-induced physiological changes. Hence, physiological changes due to exercise, such as the GSR scaling exponent, should be measured and accounted for in exercise-cognition research.

Consistent with the three exercise-cognition pathways reported in Best's (2010) review, the current research project demonstrated that exercise improves or maintain cognitive performance through specific conditions with respect to cognitive task demand, novelty of the exercise activity, and an individual's arousal system. Nevertheless, there are two reasons why these conditions are unlikely to be the only mechanism that exercise influences cognition. First, the connection between an individual's arousal system and cognitive task demand was not found to influence cognition when children repeated the exercise activity. However, repeated exercise activity did result in some children exhibiting a cognitive improvement (i.e., 49%), though this number was of a smaller percentage compared to when children first performed the exercise activity (i.e., 74%). Second, some children that performed the tablet game activity also had cognitive improvements without demonstrating a connection with an individual's arousal system, cognitive task demand, or the novelty of the tablet activity. Hence, there is likely to be more than one mechanism in which exercise enhances cognition (Best, 2010).

Previous research has concluded that no single mechanism (e.g., neurobiological or cognitive psychological theories), or factors (e.g., diagnosis, fitness) are responsible for the cognitive effect induced by physical exercise (e.g., Audiffren, 2009; Audiffren & André, 2015; Best, 2010; Davis & Lambourne, 2009; Diamond & Ling, 2016; Dietrich & Audiffren,

2011; Lambourne & Tomporowski, 2010; McMorris et al., 2009; Tomporowski et al., 2011). Interestingly, a recent study in rodents found that both the aerobic and resistance exercises resulted in similar enhancement in learning and spatial memory, but via different neurobiological pathways (e.g., BDNF and IGF-1; see Cassilhas et al., 2012). On the whole, exercise-cognition research suggests that the effect of exercise on cognition is not a straightforward matter, such that there is more than one mechanism that exercise influences cognition.

The heterogeneity of the mechanism in which physical exercise affects cognition is likely to have contributed to the difficulty of locating an optimal set of exercise parameters that best influence cognition. Further, the lack of a suitable measure of the optimal challenge point may have also added to the challenge of finding the ideal physical exercise that enhances cognition. Nevertheless, the non-linear mechanism by which exercise influences cognition may reflect the plasticity of the brain in adapting to various conditions (Gentili et al., 2011; 2013; van Praag et al., 2014). Thus, there may be a possibility that individuals who are non-responders to the cognitive effect of exercise may benefit via a different mechanism other than those reported in the exercise-cognition research (e.g., cognitive engagement and motor coordination). For instance, the current research project found that children whose arousal systems were non-responsive to the qualitative exercise activity (i.e., cognitive engagement and motor coordination) did not exhibit a facilitating cognitive effect. If there were more than one mechanism that exercise influences cognition, it may be possible that the non-responsive arousal system pathway could be bypassed to improve cognition. In other words, there may be other mechanisms by which exercise could improve cognition in individuals who are non-responsive to the physiological effect of exercise (e.g., via the arousal system). However, such a research question could only be addressed in future

research that focuses specifically on individuals who are non-responders to the exercise-cognition effect.

In summary, this research project found that the relationship between physical exercise and cognition is dependent on the connection between individual differences (i.e., arousal system), cognitive task demand and the variability of the exercise activity. Hence, apart from investigating the exercise parameters (e.g., Barha et al., 2017; Caciula et al., 2016; Vazou et al., 2016), there is also a need to shift the focus of research to individual factors (Diamond & Ling, 2016; Pesce, 2009). Investigating the scaling properties of psychophysiological measures may be a suitable index of the optimal challenge point that is different among various individuals.

Testing the Conclusions Based on the Mean

To ensure that the conclusions reported in this project are not influenced by limitations of the mean (see Speelman & McGann, 2013), two statements about the relationship between GSR scaling exponents and cognitive performance following exercise are compared against the individual values (see Table 19 and 20 at the end of this chapter):

1. Attention network test - incongruent flanker trials (accuracy): The lower the GSR scaling exponent, the poorer the performance on this measure. Alternatively, the higher the GSR scaling exponent, the better the performance on this measure.
2. Implicit sequence learning task (accuracy): The higher the GSR scaling exponent, the poorer the performance on this measure. Alternatively, the lower the GSR scaling exponent, the better the performance on this measure.

Statement 1: Attention network test - incongruent flanker trials (accuracy).

The accuracy means of the attention network test - incongruent flanker trials indicated that the lower the GSR scaling exponent, the poorer the performance. Three out of four participants that made more errors had a GSR scaling exponent ranging between 1.2 to 1.4,

with the exception of a participant in this group who had a GSR scaling exponent of 1.8. The statement that the lower the GSR scaling exponent, the poorer the accuracy performance on the incongruent flanker trials can likely be somewhat supported, as 75% of the children that had a scaling exponent equal to or below 1.4 made more errors on this measure.

Alternatively, the accuracy means of the incongruent flanker trials indicated that the higher the GSR scaling exponent, the better the performance. When the individual scores are considered, 10 out of 11 participants that made fewer errors, or maintained their error rate had a GSR scaling exponent ranging from 1.5 to 2.0. The exception from this group is that one participant that made fewer errors had a GSR scaling exponent of 1.4, which is the same value as one of the participants from the more errors group. The conclusion that the higher the GSR scaling exponent, the better the accuracy on the incongruent flanker trials can likely be supported, as 91% of the children who made fewer errors, or maintained their error rate had a GSR scaling exponent of equal to or above 1.5.

Statement 2: Implicit sequence learning task (accuracy).

The accuracy means of the implicit learning task indicated that participants who had a higher GSR scaling exponent performed poorer on the implicit learning task compared to those with a lower GSR scaling exponent. Four out of four participants that made errors had a high GSR scaling exponent ranging from 1.7 to 1.8. However, 5 out of 12 participants who made fewer errors, or maintained their error rate also had a GSR scaling exponent ranging from 1.7 to 2.0. Hence, the conclusion that the higher the GSR scaling exponent, the poorer the performance on the implicit learning task needs to be taken with caution. Although all participants who made errors on the implicit learning task had a GSR scaling exponent between 1.7 to 1.8, 42% of the participants who made fewer errors, also had a GSR scaling exponent in that range (i.e., ≥ 1.7). Therefore, the appropriate conclusion is that all children who made more errors on the implicit learning task after exercise, had a GSR scaling

exponent above 1.7, but not all children who had a GSR scaling exponent above this value made more errors on this task.

Alternatively, based on the accuracy means, the lower the GSR scaling exponent, the better the performance on the implicit learning task than a higher GSR scaling exponent. When individual scores are considered, 7 out of 12 children (58%) in the group that made fewer errors had a GSR scaling exponent lying between 1.2 to 1.6. In addition, no individuals from the group that made more errors had a GSR scaling exponent within this range. Hence, there may be some support for the statement that the lower the GSR scaling exponent (i.e., ≤ 1.6), the likelihood of better performance on the implicit learning task. However, this conclusion cannot be confidently established.

This section demonstrates that the mean may not always be an accurate representation of individual performance (Speelman & McGann, 2013). When conclusions are made solely based on the mean, there is an inherent risk of drawing a conclusion that is non-representative of the individual participants. Though it is not always possible that every participant's performance will be consistent with the mean (i.e., influence of confounding variables not within the experimenter's control), the comparison of individual scores with the mean provides another perspective on the findings. Furthermore, such an approach may also resolve some of the inconsistencies in the previous research findings, given that all, if not most of the research based their conclusions solely on the mean.

Overall, the clearest finding in this research project is that, following the exercise activity, a GSR scaling exponent that is equal to or larger than $\alpha = 1.5$ is related to better accuracy performance on the conflict network task (i.e., incongruent flanker trials) than a GSR scaling exponent lower than this value. Also, a GSR scaling exponent that is equal to or lower than $\alpha = 1.4$ is related to poorer performance on the conflict network task compared to a GSR scaling exponent that is greater than this value. The relationship between the GSR

scaling exponent and the accuracy performance on the implicit learning task seems unclear. Hence, the findings regarding the GSR scaling exponent or the sensitivity of the arousal system and the accuracy on the implicit learning task should be treated with caution. Nevertheless, on the whole, the consistency between the individual GSR scaling exponent and the findings based on the mean, strengthens the conclusion that the performance of the children following physical exercise on executive attention is dependent on the arousal system, task demand, and the novelty of the exercise intervention.

Clinical Implications

One of the goals of this research project was to understand the influence of diagnosis on the exercise and cognition relationship. In particular, the results from the meta-analysis and experimental study found that physical exercise is effective in enhancing aspects of cognitive performance in children with a neurodevelopmental condition. The meta-analytic review of 22 studies reported a significant small-to-medium effect size of exercise interventions on cognition, supporting its application to children and young individuals with an ASD and/or ADHD diagnosis. In terms of the experimental study, physical exercise is better than a tablet game activity in enhancing or maintaining cognition, particularly on the accuracy measures of the implicit learning and conflict network task. These findings provide support for the application of physical exercise activity in facilitating aspects of cognition to children with a neurodevelopmental condition.

Interestingly, the effect of exercise does not seem to differ between children with a neurodevelopmental condition and those with a typical development. The overall effect size reported by the meta-analysis from this research project is similar to those reported in the typical developing population (Verburgh et al., 2014) and other children populations with/without learning or physical disabilities (Fedewa & Ahn, 2011; Sibley & Etnier, 2003). Furthermore, the experimental study in this research project did not indicate that the effect of

exercise differed for children with or without a neurodevelopmental condition. Even though on average, children with a neurodevelopmental condition were less efficient in resolving conflicting stimuli, and made more errors on the implicit learning task relative to children with a typical development, these differences were not dependent on the effect of the exercise or tablet activity.

Consistent with the results of the meta-analysis, the experimental study found that the overall percentage of children who demonstrated an exercise-induced cognitive improvement in the first session, was similar in both those children in the typical developmental group and those that were in the neurodevelopmental group. Specifically, for children with a neurodevelopmental condition, 70% on average, exhibited the cognitive effect of exercise. Similarly, 76% of children with a typical development also exhibited the cognitive effect of exercise. Diagnosis did have an effect, but only following the tablet activity, particularly with performance on the conflict network. The results showed that, after performing the tablet activity, children with a neurodevelopmental condition made more errors on the incongruent flanker trials relative to baseline performance. Conversely, children with a typical development made fewer errors on the incongruent flanker trials following the tablet activity. Thus, unlike children with a typical development, the effect of tablet activity in children with a neurodevelopmental condition may be negative (Chan & Rabinowitz, 2006; Mazurek & Engelhardt, 2013). Specifically, tablet activity was found in the current research project to reduce the efficiency in processing conflict information in children with a neurodevelopmental condition.

On the whole, the findings from this research project and previous research suggest that the cognitive facilitating effect of exercise is unlikely to differ between children with a typical development and those with a neurodevelopmental condition. Importantly, the results of this research project support the efficacy of applying physical exercise interventions in

enhancing aspects of cognition in children with a neurodevelopmental condition. Conversely, the tablet game activity may not be a suitable activity to enhance cognition in children with a neurodevelopmental condition.

General Limitations and Future Directions

The specific limitations of the various approaches undertaken by this research project have been discussed in the respective chapters. This section highlights the general limitations of this research project and some questions that could be addressed in future research. First, as the investigation of the GSR scaling exponent is novel to the exercise-cognition research, the psychophysiological findings need to be considered as an exploratory study. Specifically, the main finding that a GSR scaling exponent equivalent to or larger than $\alpha = 1.5$, was related to better accuracy performance on the executive attention task relative to a scaling exponent lower than this value, needs to be validated in future studies. Importantly, this research project could not confirm whether any child who exhibits a cognitive effect of exercise on the executive attention task would have a GSR scaling exponent that is equal to or above $\alpha = 1.5$. Rather, the findings suggest a positive relationship between an individual's arousal system and executive attention, such that the higher the scaling exponent (i.e., higher sensitivity of the arousal system), the better the executive attention.

Second, although this project found differences in the GSR scaling exponent between children who demonstrated a facilitating effect of exercise and those who did not exhibit a cognitive improvement with exercise, this project could not account for why such differences in scaling exponents occurred. In other words, there is a need for future investigations into why certain children's GSR scaling exponents, or arousal system, failed to respond to the effect of exercise. Research that focuses on individuals who do not respond to exercise will lead to possible interventions that improve the likelihood of these individuals exhibiting the exercise-induced cognitive effect. Further, research with individuals who are non-responders

to exercise will also advance understanding of the mechanism by which physical exercise improves cognition.

Third, as the experimental study only included one task that measured executive attention (i.e., CRSD-ANT: Conflict network test), this project could not exclude the task impurity issue that exists in most executive function tasks (see Chapter 2; e.g., Suchy, 2009). Thus, to ensure that a particular executive function is indeed implicated in the exercise-cognition relationship, future studies would need to consider including multiple tasks to measure a single executive function (e.g., Ziereis & Jansen, 2015). Further, future research could also investigate whether an increased GSR scaling exponent (i.e., increased sensitivity of the arousal system) is associated with enhanced performance on other executive functions (e.g., set-shifting, planning and working memory).

Fourth, the findings from this project may also be influenced by other factors, such as self-efficacy (e.g., Tomporowski et al., 2011). Further, the unequal researcher-child interaction that is more prevalent in the exercise group compared to the tablet activity group may have also affected the findings. According to the contextual interference effect (see Tomporowski et al., 2010), the researcher-child interaction in the exercise group would be cognitively demanding as the child had to learn new motor skills with the basketball (e.g., bouncing the ball with the non-dominant hand, and a series of running and passing/receiving the ball). As presented in Chapter 3, the exercise sequence required the child to observe and imitate the exercise activity demonstrated by the researcher. Additionally, the child needed to self-monitor and adjust his/her movements to perform the challenging exercise activity (e.g., bouncing the ball alternating between both hands while walking). Conversely, the tablet game activity was an individual activity. Therefore, the tablet activity, though cognitively engaging, may not be as cognitively demanding compared to the exercise activity. Hence, the

unequal cognitive demands between the exercise and tablet activity may have also affected the findings.

Lastly, there are some other factors that could be addressed in future research. For example, future studies should try to report the number of individuals that had cognitive improvements with exercise. This information will assist exercise-cognition researchers in predicting the likelihood of an individual who would benefit from physical exercise. Furthermore, the practice of reporting the actual numbers of participants that improved in cognition will also allow researchers to evaluate the effectiveness of their exercise interventions. Additionally, future studies may consider the search for an early physiological marker (e.g., GSR) that can predict the likelihood of an individual responding to the cognitive effect of exercise prior to the exercise intervention. The early physiological marker will assist researchers in identifying individuals who are non-responsive to the effect of exercise so that these individuals can be specifically targeted in research. Moreover, the early physiological marker will also assist in understanding the pre-requisite factors for humans to experience the cognitive effect of exercise, or more generally, physical activity.

Conclusion

This research project investigated the physical exercise and cognition relationship in typical developing children and children with neurodevelopmental conditions. The current research project focused on determining the efficacy, effect, and mechanism underlying the exercise-cognition relationship. In addition, fractal analysis (i.e., scaling exponent) was proposed as a viable analytical tool to investigate the influence of individual differences to the relationship between exercise and cognition. Specifically, the scaling exponent of psychophysiological measures (e.g., GSR) could be an index of an individual's optimal challenge point.

According to the fractal analysis, this research project revealed that the cognitive effect of exercise is dependent on an individual's arousal system that changes in response to exercise. Overall, the findings from this research project indicate that there is a need to shift the focus of research from the over-emphasis of exercise parameters to the influence of individual differences. Further, similar to previous research, this research project found that some children appeared to be resistant to the cognitive effect of exercise. Thus, there is also a need for future studies to acknowledge the existence of individuals who are non-responsive to the exercise-induced cognitive effect and to direct the focus of research to this group of individuals. Individuals who are non-responders to the exercise-cognition effect are an important, yet often neglected population in the exercise-cognition research. Hence, future research on individuals who are non-responsive to the exercise-cognitive effect would further advance the understanding of the mechanism underlying physical exercise and cognition.

Table 19

Participants' Post-Exercise GSR Scaling Exponents and Performance on the Incongruent Flanker Trials (N = 15)

Cognitive Performance (More Errors)			
Count	Participant (Gender)	Diagnostic status	Scaling exponent
1	A (Male)	Neurodevelopment	1.2
2	B (Female)	Typical development	1.3
3	C (Male)	Typical development	1.4
4	D (Female)	Typical development	1.8*
Cognitive Performance (Less/Maintained Errors)			
Count	Participant (Gender)	Diagnostic status	Scaling exponent
1	E (Male)	Typical development	1.4*
2	F (Female)	Neurodevelopment	1.5
3	G (Male)	Typical development	1.6
4	H (Male)	Typical development	1.7
5	I (Male)	Typical development	1.7
6	J (Male)	Neurodevelopment	1.8
7	K (Male)	Neurodevelopment	1.8
8	L (Male)	Typical development	1.8
9	M (Male)	Neurodevelopment	1.8
10	N (Male)	Typical development	1.9
11	O (Male)	Typical development	2.0

* Refers to values that are inconsistent with the direction of the mean.

Table 20

Participants' Post-Exercise GSR Scaling Exponents and Performance on the Implicit Sequence Learning Task (N = 16)

Cognitive Performance (More Errors)			
Count	Participant (Gender)	Diagnostic status	Scaling exponent
1	I (Male)	Typical development	1.7
2	M (Male)	Neurodevelopment	1.8
3	J (Male)	Neurodevelopment	1.8
4	D (Female)	Typical development	1.8
Cognitive Performance (Less/Maintained Errors)			
Count	Participant (Gender)	Diagnostic status	Scaling exponent
1	A (Male)	Neurodevelopment	1.2
2	B (Female)	Typical development	1.3
3	C (Male)	Typical development	1.4
4	E (Male)	Typical development	1.4
5	F (Female)	Neurodevelopment	1.5
6	P (Female)	Typical development	1.5
7	G (Male)	Typical development	1.6
8	H (Male)	Typical development	1.7*
9	K (Male)	Neurodevelopment	1.8*
10	L (Male)	Typical development	1.8*
11	N (Male)	Typical development	1.9*
12	O (Male)	Typical development	2.0*

Note. The alphabets attached to each participant in the above table are the same as Table 19.
* Refers to values that are inconsistent with the direction of the mean.

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Appendix A

Research Recruitment Poster for Children with a Neurodevelopmental Condition

Research Project
School of Psychology and Social Science

AUSTRALIA ECU EDITH COWAN UNIVERSITY

An investigation of the relationship between physical activity and cognitive performance

To participate:

1) 6-11 years old
2) *High-functioning* Autism, Aspergers, or Pervasive Developmental Disorder Not Otherwise Specified; or ADHD
3) No major movement or vision difficulties
4) Able to participate in physical activities
5) No anticipated changes in medications during participation

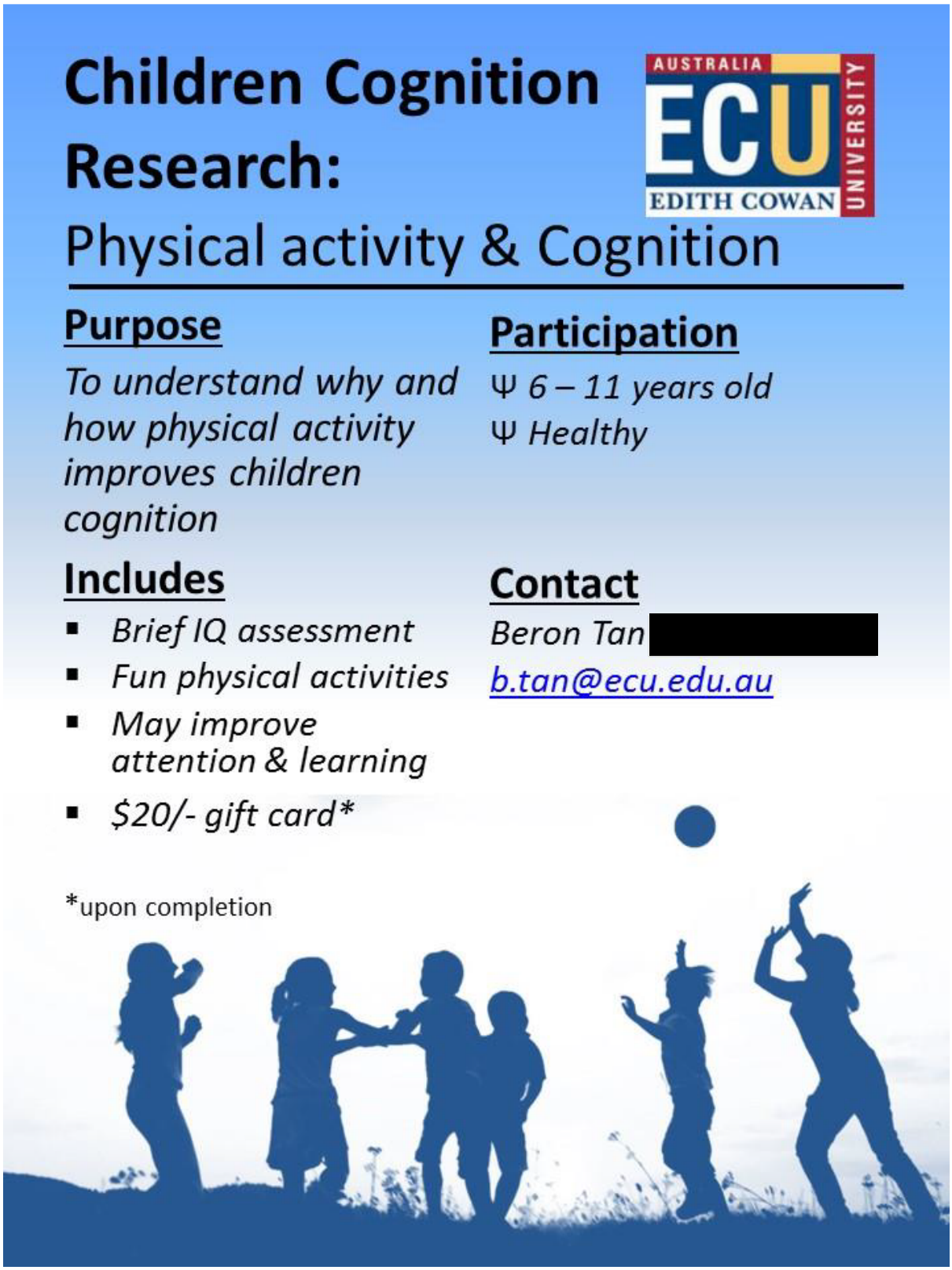
Benefits?

- ✓ May improve attention and aspects of learning
- ✓ Fun physical activities and games
- ✓ Help to advance the understanding of how physical activity works on children's mental performance

Contact the researcher:
Beron Tan
[redacted]
b.tan@ecu.edu.au

Appendix B

Research Recruitment Poster for Children with a Typical Development



Appendix C

Parental Information Letter for Children with a Neurodevelopmental Condition

Dear Parent/Guardian,

My name is Beron Tan and I am currently undertaking a Doctoral Degree in Psychology at Edith Cowan University. My research topic is the investigation of the effects of physical activity on mental performance in children with typical development, autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). Previous studies have found improvements in aspects of learning and academic performance in typical developing children following physical activity. However, the reason why such improvements are found remains unclear. As a result, it is not known how physical activity can be applied successfully to improve learning in the children population. In particular, children with childhood disorders such as autism have significant difficulties with learning, and physical activity may be able to enhance their abilities to learn. In order to establish such findings, the research requires support from children with developmental disorders so as to understand why and how physical activities can improve learning performance in the children population. Specifically, this study seeks children aged 6-11 years who are formally diagnosed with high-functioning autism or ADHD (i.e., IQ above low average); able to participate in mild to moderate-intensity physical activities (e.g., jogging), with no major movement/visual difficulties; and no anticipated changes to their prescribed medications in the coming months.

This study will include two stages, 1) psychological assessment and 2) participation in physical activities and learning tasks. The initial assessment includes a parent/guardian interview or questionnaire to confirm the child's diagnosis of ASD or ADHD, and a brief assessment of the child's intellectual functioning. The second stage will involve four separate sessions of physical activities, where the child will be guided to perform some coordinated movements with a basketball (e.g., passing) and he/she will be evaluated on the level of attention and learning performance using computerised tasks. In addition, measures such as skin response, movement and temperature will be recorded to further understand what happens to the body during physical activities.

Participation in this research will be over a period of 5 to 6 weeks (1 session/week); lasting not more than an hour and a half each. The location of the research will be at ECU Joondalup Campus. Although there may be risks such as falls during physical activities, the researcher who is trained in first-aid will be on-site at all times during the activity and will ensure that such risks are kept to the minimum. The outcome of this study will be provided to

you after the completion of the research project. In addition, a gift voucher worth $20 will be given as a token of appreciation for your time and effort in supporting this research. Participation is voluntary and any identifiable information will be kept confidential. If you have any enquiries or are interested in participating in this research, please contact me at [redacted] or via email at b.tan@ecu.edu.au. You may also contact my supervisors Associate Professor Julie Ann Pooley at 6304 5591 or j.pooley@ecu.edu.au or Professor Craig Speelman at 6304 5724 or c.speelman@ecu.edu.au. Alternatively, if you have any concerns about the research project and wish to speak to an independent person, you may contact:

Research Ethics Officer
Edith Cowan University
270 Joondalup Drive
JOONDALUP WA 6027
Phone: (08) 6304 2170
Email: research.ethics@ecu.edu.au

Thank you for your consideration to contribute to this study and I look forward to hearing from you soon.

Warm Regards
Beron Tan

Appendix D

Parental Information Letter for Children with a Typical Development

Dear Parent/Guardian,

My name is Beron Tan and I am currently undertaking a Doctoral Degree in Psychology at Edith Cowan University. My research topic is on the investigation of the effects of physical activity on mental performance in children with typical development, autism spectrum disorder (ASD) and attention-deficit/hyperactivity disorder (ADHD). Previous studies have found improvements in aspects of learning and academic performance in typical developing children following physical activity. However, the reason why such improvements are found remains unclear. As a result, it is not known how physical activity can be applied successfully to improve learning in the children population. In particular, children with childhood disorders such as autism and ADHD have significant difficulties with learning, and physical activity may be able to enhance their abilities to learn. In order to establish such findings, apart from recruiting child participants with childhood disorders, the research also requires support from children with typical development so as to understand why and how physical activities can improve learning performance in the children population. Specifically, this study seeks child participants aged 6-11 years, not previously diagnosed with developmental disorders; do not have major movement/visual difficulties and are able to participate in mild to moderate-intensity physical activities (e.g., jogging); and do not have anticipated changes in prescribed medications (if any) in the coming months.

This study will include two stages, 1) psychological assessment and 2) participation in physical activities and learning tasks. Potential child participants will be assessed by the researcher who is a registered psychologist, to screen for risks of childhood disorders. During the assessment, the parent/guardian will be required to fill in questionnaires about their child. Following the clearance of their diagnostic status, the child participants will be evaluated on their level of intellectual functioning. The purpose of the assessment is to ensure that the child participants are not having undiagnosed childhood problems. If in the event that the child is suspected of having risks of childhood problems, the parent/guardian will be provided with the information to seek further clinical evaluation, and it may not be appropriate for the child to participate further in the research. Child participants will only proceed to participate in the second stage of the research if they are cleared from the assessment.

The second stage will involve four separate sessions of physical activities, where the child will be guided to perform some coordinated movements with a basketball (e.g., passing)

and he/she will be evaluated on their level of attention and learning performance using computerised tasks. In addition, measures such as skin response, movement and temperature will be recorded to further understand what happens to the body during physical activities.

Participation in this research will be over a period of 5 to 6 weeks (1 session/week); lasting not more than an hour and a half each. The location of the research will be at ECU Joondalup Campus. Although there may be risks such as falls during physical activities, the researcher who is trained in first-aid will be on-site at all times during the activity and will ensure that such risks are kept to the minimum. The outcome of this study will be provided to you after the completion of the research project. In addition, a gift voucher worth $20 will be given as a token of appreciation for your time and effort in supporting this research. Participation is voluntary and any identifiable information will be kept confidential. If you have any enquiries or are interested in participating in this research, please contact me at [redacted] or via email at b.tan@ecu.edu.au. You may also contact my supervisors Associate Professor Julie Ann Pooley at 6304 5591 or j.pooley@ecu.edu.au or Professor Craig Speelman at 6304 5724 or c.speelman@ecu.edu.au. Alternatively, if you have any concerns about the research project and wish to speak to an independent person, you may contact:

Research Ethics Officer
Edith Cowan University
270 Joondalup Drive
JOONDALUP WA 6027
Phone: (08) 6304 2170
Email: research.ethics@ecu.edu.au

Thank you for your consideration to contribute to this study and I look forward to hearing from you soon.

Warm Regards
Beron Tan

Appendix E

Autism Spectrum Quotient (AQ-10)

NHS National Institute for Health Research

AQ-10 (Child Version)

Autism Spectrum Quotient (AQ)

A quick referral guide for parents to complete about a child aged 4-11 years with suspected autism who does not have a learning disability.

Please tick one option per question only:		**Definitely Agree**	**Slightly Agree**	**Slightly Disagree**	**Definitely Disagree**
1	S/he often notices small sounds when others do not				
2	S/he usually concentrates more on the whole picture, rather than the small details				
3	In a social group, s/he can easily keep track of several different people's conversations				
4	S/he finds it easy to go back and forth between different activities				
5	S/he doesn't know how to keep a conversation going with his/her peers				
6	S/he is good at social chit-chat				
7	When s/he is read a story, s/he finds it difficult to work out the character's intentions or feelings				
8	When s/he was in preschool, s/he used to enjoy playing games involving pretending with other children				
9	S/he finds it easy to work out what someone is thinking or feeling just by looking at their face				
10	S/he finds it hard to make new friends				

SCORING: Only 1 point can be scored for each question. *Score 1 point for Definitely or Slightly Agree on each of items 1, 5, 7 and 10. Score 1 point for Definitely or Slightly Disagree on each of items 2, 3, 4, 6, 8 and 9.* If the individual scores **more than 6 out of 10**, consider referring them for a specialist diagnostic assessment.

USE: This is the child version of the test recommended in the NICE clinical guideline CG142. www.nice.org.uk/CG142

Key reference: Allison C, Auyeung B, and Baron-Cohen S, (2012) *Journal of the American Academy of Child and Adolescent Psychiatry* 51(2):202-12.

UNIVERSITY OF CAMBRIDGE

autism research centre arc

Appendix F

Informed Consent for Parents/Guardians

Project title: The relationship between physical exercise and cognition in children with typical development and neurodevelopmental disorders

I have read and understood:

- The outline and nature of the research study via the information letter provided to me.
- I have the opportunity and right to clarify any doubts to my satisfaction about the study through the contact of the researcher provided in the information letter.
- I am required to complete a questionnaire and/or an interview during the period of the research study.
- My personal identification/information will be kept confidential and will not be disclosed without my consent.
- The data collected for the purpose of this research study may be used in future research purposes provided that my name and any identifying information are removed.
- I have the right to withdraw at any point of the study without incurring any penalty and no explanation is required.

I have read and fully understood all of the above information provided to me and I agree to participate in this research study at my own will.

_____ _____

Participant's name/signature/date Witness's name/signature/date

Appendix G

Informed Consent on Behalf of the Child Participant

Project title: The relationship between physical exercise and cognition in children with typical development and neurodevelopmental disorders

I have read and understood:

- The outline and nature of the research study via the information letter provided to me.
- I have the opportunity and the right to clarify any doubts to my satisfaction about the study through the contact of the researcher provided in the information letter.
- My child is required to participate in a series of physical activities (i.e., basketball) and non-physical activities (e.g., video games) for a period of 5-6 weeks.
- To the best of my knowledge, my child, as of current, does not have any known medical issues that may prevent him/her from participating in physical activities.
- If in the event that I am aware that my child is not suitable to participate in this research due to medical reasons, I will inform the researcher soonest possible.
- My child's personal identification/information will be kept confidential and will not be disclosed without my consent.
- The data collected for the purpose of this research study may be used in future research purposes provided that my child's name and any identifying information are removed.
- My child and/or I (on his/her behalf) have the right to withdraw from participating in this study at any point of the research without incurring any penalty and no explanation is required.
- I understand that there may be risks involved for my child in participating in this research study and I trust that the researcher(s) will ensure that the risks are kept to the minimal.

I have read and fully understood all of the above information provided to me and I agree to allow my child to participate in this research study at my own will.

_____ _____

Parent/guardian's name/signature/date Witness's name/signature/date

*delete where appropriate

Appendix H: Assumption Testing and Corrections

Prior to the main analyses, data were screened for sphericity, normality, and homogeneity of variance assumptions. The sphericity assumption was assumed in all analyses as there were only two levels in each independent variable used in Chapter 4. Regarding the implicit learning task (i.e., ISLT), variances were found to be equal for all reaction time measures across children in the neurodevelopmental group and those in the typical developing group. In terms of normality, several deviations were detected on the mean reaction time measure for children in the typical developing group including in session 1, post-intervention improbable trials, $W(20) = 0.89$, $p = .03$; in session 2, pre-intervention improbable trial, $W(20) = 0.85$, $p = .01$, and post-intervention improbable trials, $W(20) = 0.84$, $p = .004$, as well as pre-intervention probable trials, $W(20) = 0.87$, $p = .01$, and post-intervention probable trials, $W(20) = 0.83$, $p = .002$. In addition, combined positive skewness ranged from 1.16 to 1.53 ($SE = 0.51$), and positive kurtosis between 0.93 to 2.21 ($SE = 0.99$).

As for error rates of the ISLT, the homogeneity of variance test revealed unequal variance between diagnostic groups on the pre-intervention trials in session 2, $F(1, 33) = 6.63$, $p = .02$. Additionally, non-normality was found for children in the typical developing group on pre-intervention trial, $W(20) = 0.86$, $p = .01$, and post-intervention trials $W(20) = 0.84$, $p = .003$, in session 1; and also on post-intervention trial in session 2, $W(20) = 0.78$, $p = <.001$. Skewness and kurtosis values for children in the typical developmental group ranged between 1.11 and 2.15 ($SE = 0.51$), and 0.28 and 6.44 ($SE = 0.99$), respectively. Non-normality was also detected on children in the neurodevelopmental group only on post-intervention trials in session 2, $W(15) = 0.87$, $p = .03$, with skewness of 1.41 ($SE = 0.58$) and kurtosis of 1.92 ($SE = 1.12$).

Tests of homogeneity of variance and normality were also conducted on the modified attention network test (CRSD-ANT): network scores, reaction time, and error rates on the

alerting, orienting and conflict network measures. In terms of the network scores, the homogeneity of variance assumption was met for alerting and orienting network, except for conflict network, where unequal variance was found on post-intervention conflict scores in session 2, $F(1, 31) = 5.08$, $p = .03$. For alerting and orienting network scores, a test of normality was not significant for children in the typical developing group. However, deviations from normality were found for children in the neurodevelopmental group on pre-intervention alerting network scores in session 2, $W(14) = 0.86$, $p = .03$, skewness = -1.13 ($SE = 0.60$), and kurtosis = 2.05 ($SE = 1.15$); in the orienting network, pre-intervention network scores in session 1, $W(14) = 0.83$, $p = .01$, skewness = -1.88 ($SE = 0.60$), and kurtosis = 4.81 ($SE = 1.15$), and post-intervention network score in session 2, $W(14) = 0.83$, $p = .01$, skewness = -1.43 ($SE = 0.60$), and kurtosis = 4.86 ($SE = 1.15$). In the conflict network, non-normality was only identified in session 1 of post-intervention network scores for children in the typical developing group, $W(19) = 0.82$, $p = .002$, skewness = 2.04 ($SE = 0.52$), and kurtosis = 6.85 ($SE = 1.01$).

Regarding reaction time measures, a homogeneity of variance test showed equal variances across the alerting, orienting and conflict network. In the neurodevelopmental group, data distribution was non-normal for post-intervention double cue trials (i.e., alerting network) in session 2, $W(14) = 0.87$, $p = .04$, skewness = 1.57 ($SE = 0.60$), and kurtosis = ($SE = 1.15$), and post-intervention spatial cue trials (i.e., orienting network) in session 2, $W(14) = 0.85$, $p = .02$, skewness = 1.44 ($SE = 0.60$), and kurtosis = 1.71 ($SE = 1.15$). In the typical developing group, non-normality was identified on pre-intervention no cue trials (i.e., alerting network) in session 2, $W(19) = 0.90$, $p = .05$, skewness = 0.34 ($SE = 0.52$), and kurtosis = 2.77 ($SE = 1.01$), and pre-intervention congruent flankers (i.e., conflict network) in session 1, $W(14) = 0.89$, $p = .03$, skewness = 0.03 ($SE = 0.52$), and kurtosis = -1.71 ($SE = 1.01$).

In terms of error rates, the homogeneity of variance assumption was met only for the orienting network. For the alerting network, variances were unequal in session 2 for pre-intervention errors on double cue trials, $F(1, 31) = 4.90$, $p = .03$, and post-intervention error on no cue trials, $F(1, 31) = 5.97$, $p = .02$. Additionally, unequal variances were also detected on the incongruent flankers of the conflict network in session 2 for pre-intervention errors, $F(1, 31) = 4.15$, $p = .05$, and post-intervention errors, $F(1, 31) = 4.67$, $p = .04$.

Pertaining to normality, deviations were present across the alerting, orienting and conflict network (see Table 21 and 22). In the neurodevelopmental group, combined skewness ranged from 0.36 to 2.41 (*SE* = 0.60) and kurtosis between -1.46 and 6.48 (*SE* = 1.15) for the alerting network; skewness of -0.16 to 1.92 (*SE* = 0.60), and kurtosis of -1.93 to 4.23 (*SE* = 1.15) for the orienting network; for the conflict network, values ranged between 0.82 and 3.21(*SE* = 0.60), and -0.78 and 11.08 (*SE* = 1.15) for skewness and kurtosis, respectively. In the typical developing group, combined skewness ranged from 0.29 to 2.83 (*SE* = 0.52), and kurtosis of -1.14 to 9.88 (*SE* = 1.01) for alerting network; skewness of 0.29 to 2.35 (*SE* = 0.52) and kurtosis of -1.14 to 6.49 (*SE* = 1.01) for orienting network; lastly, for the conflict network, skewness between 0.60 and 2.13 (*SE* = 0.52), and kurtosis of -1.10 to 6.32 (*SE* = 1.01).

Based on the results of the assumption tests, a large proportion of the data across the ISLT and CRSD-ANT variables significantly violated the assumptions of normality and homogeneity of variance needed for mixed ANOVA. Thus, corrections were made prior to running the main analyses using winsorized means. The process of winsorizing was similar to that used in a study by Kim, Park, Song, Koo and An (2011). First, an acceptable range of values were established using the upper and lower quartile to derive the interquartile range based on Tukey hinges. Second, the interquartile range was multiplied by 1.5. Third, the product of the multiplication was then subtracted from the lower quartile to derive the

minimum limit acceptable for a data value. Similarly, to obtain the maximum acceptable limit for a data value, instead of subtraction, the product of the multiplication was added to the upper quartile. Therefore, the minimum and maximum values formed a data window, such that any data that fell beyond either end of the limit would be considered an outlier. Lastly, identified outliers were replaced with either the minimum or maximum acceptable value depending on whether they exceeded the lower or upper end of the data window (i.e., a lower end outlier would be replaced with the minimum acceptable value and an upper end outlier replaced with the maximum acceptable value).

After winsorizing outliers, significant improvements in normality and homogeneity of variance were generally observed across all dependent variables of the ISLT and CRSD-ANT. However, the data distribution of error rates, though improved, were still identified as non-normal. On closer inspection of the histograms and boxplots, the non-normality of the error rates was due to the majority of the participants obtaining zero or very low error rates leading to positive skewness and kurtosis. Furthermore, given that the ISLT and CRSD-ANT required participants to obtain a minimum level of accuracy above 50% and 70%, respectively, in order to be included in the analysis, the shape of the distribution was not unexpected. Nevertheless, overall, the extent of deviations from normality after winsorizing was better than the uncorrected data.

Table 21

Summary Table for Test of Normality (Shapiro-Wilk) on the Error Rates of the Modified Attention Network Test (CRSD-ANT) in the Neurodevelopmental Group

Source	*df*	*W*	*p*
Alerting network			
Session 1			
No cue/Pre-intervention	14	0.82	.01*
No cue/Post-intervention	14	0.88	.05*
Double cue/Pre-intervention	14	0.94	.40
Double cue/Post-intervention	14	0.85	.02*
Session 2			
No cue/Pre-intervention	14	0.90	.13
No cue/Post-intervention	14	0.78	.003*
Double cue/Pre-intervention	14	0.81	.01*
Double cue/Post-intervention	14	0.68	<.001*
Orienting network			
Session 1			
Centre cue/Pre-intervention	14	0.86	.03*
Centre cue/Post-intervention	14	0.75	.001*
Spatial cue/Pre-intervention	14	0.84	.02*
Spatial cue/Post-intervention	14	0.76	.002*
Session 2			
Centre cue/Pre-intervention	14	0.76	.002*
Centre cue/Post-intervention	14	0.83	.01*
Spatial cue/Pre-intervention	14	0.81	.01*
Spatial cue/Post-intervention	14	0.84	.02
Conflict network			
Session 1			
Incongruent flanker/Pre-intervention	14	0.91	.13
Incongruent flanker/Post-intervention	14	0.86	.03*
Congruent flanker/Pre-intervention	14	0.63	<.001*
Congruent flanker/Post-intervention	14	0.82	.01*
Session 2			
Incongruent flanker/Pre-intervention	14	0.86	.03*
Incongruent flanker/Post-intervention	14	0.92	.22
Congruent flanker/Pre-intervention	14	0.76	.002*
Congruent flanker/Post-intervention	14	0.55	<.001*

* $p = .05$.

Table 22

Summary Table for Test of Normality (Shapiro-Wilk) on the Error Rates of the Modified Attention Network Test (CRSD-ANT) in the Typical Developing Group

Source	*df*	*W*	*p*
Alerting network			
Session 1			
No cue/Pre-intervention	19	0.84	.01*
No cue/Post-intervention	19	0.79	.001*
Double cue/Pre-intervention	19	0.92	.10
Double cue/Post-intervention	19	0.75	<.001*
Session 2			
No cue/Pre-intervention	19	0.64	<.001*
No cue/Post-intervention	19	0.81	.001*
Double cue/Pre-intervention	19	0.86	.01*
Double cue/Post-intervention	19	0.91	.07
Orienting network			
Session 1			
Centre cue/Pre-intervention	19	0.85	.01*
Centre cue/Post-intervention	19	0.75	<.001*
Spatial cue/Pre-intervention	19	0.90	.051
Spatial cue/Post-intervention	19	0.60	<.001*
Session 2			
Centre cue/Pre-intervention	19	0.77	<.001*
Centre cue/Post-intervention	19	0.87	.02*
Spatial cue/Pre-intervention	19	0.64	<.001*
Spatial cue/Post-intervention	19	0.84	.01*
Conflict network			
Session 1			
Incongruent flanker/Pre-intervention	19	0.88	.02*
Incongruent flanker/Post-intervention	19	0.76	<.001*
Congruent flanker/Pre-intervention	19	0.84	.01*
Congruent flanker/Post-intervention	19	0.79	.001*
Session 2			
Incongruent flanker/Pre-intervention	19	0.90	.04*
Incongruent flanker/Post-intervention	19	0.83	.003*
Congruent flanker/Pre-intervention	19	0.66	<.001*
Congruent flanker/Post-intervention	19	0.85	.01*

* $p = .05$.

Appendix I

Program Syntax (EEG - Amplitude Envelope)

Neurophysiological Biomarker Toolbox (NBT) 0.5.5-public

```
# Theta 4-8Hz Signal
[AmplitudeEnvelope,AmplitudeEnvelopeInfo] =nbt_GetAmplitudeEnvelope (Signal,
SignalInfo, 4, 8, 4/8);
# Alpha 8-13Hz Signal
[AmplitudeEnvelope,AmplitudeEnvelopeInfo] =nbt_GetAmplitudeEnvelope (Signal,
SignalInfo, 8, 13, 2/8);
# Beta 13-30Hz Signal
[AmplitudeEnvelope,AmplitudeEnvelopeInfo] =nbt_GetAmplitudeEnvelope (Signal,
SignalInfo, 13, 30, 2/8);
```

Appendix J

Program Syntax (EEG – Detrended Fluctuation Analysis)

R program 3.3.1 (fractal package 2.0-1)

```
# clear workspace

rm(list=ls())

# load fractal package

require(fractal)

# read EEG data

EEG <- read.csv("C:\\User\\location_of_the_file\\file name.csv”, header = F)

# labelling EEG channels

names(EEG) <- c("AF3", "F7", "F3", "FC5", "T7", "P7", "O1", "O2", "P8", "T8", "FC6",

"F4", "F8", "AF4")

# Detrended fluctuation analysis AF3

DFA.AF3 <- DFA(EEG$AF3, detrend="poly1", sum.order=1)

# print results for AF3

print(DFA.AF3)

# plot results for AF3

eda.plot(DFA.AF3)

# Detrended fluctuation analysis F7

DFA.F7 <- DFA(EEG$F7, detrend="poly1", sum.order=1)

# print results for F7

print(DFA.F7)

# plot results for F7

eda.plot(DFA.F7)
```

```
# Detrended fluctuation analysis F3

DFA.F3 <- DFA(EEG$F3, detrend="poly1", sum.order=1)

# print results for F3

print(DFA.F3)

# plot results for F3

eda.plot(DFA.F3)

# Detrended fluctuation analysis FC5

DFA.FC5 <- DFA(EEG$FC5, detrend="poly1", sum.order=1)

# print results for FC5

print(DFA.FC5)

# plot results for FC5

eda.plot(DFA.FC5)

# Detrended fluctuation analysis T7

DFA.T7 <- DFA(EEG$T7, detrend="poly1", sum.order=1)

# print results for T7

print(DFA.T7)

# plot results for T7

eda.plot(DFA.T7)

# Detrended fluctuation analysis P7

DFA.P7 <- DFA(EEG$P7, detrend="poly1", sum.order=1)

# print results for P7

print(DFA.P7)

# plot results for P7

eda.plot(DFA.P7)
```

```
# Detrended fluctuation analysis O1
DFA.O1 <- DFA(EEG$O1, detrend="poly1", sum.order=1)
# print results for O1
print(DFA.O1)
# plot results for O1
eda.plot(DFA.O1)
# Detrended fluctuation analysis O2
DFA.O2 <- DFA(EEG$O2, detrend="poly1", sum.order=1)
# print results for O2
print(DFA.O2)
# plot results for O2
eda.plot(DFA.O2)
# Detrended fluctuation analysis P8
DFA.P8 <- DFA(EEG$P8, detrend="poly1", sum.order=1)
# print results for P8
print(DFA.P8)
# plot results for P8
eda.plot(DFA.P8)
# Detrended fluctuation analysis T8
DFA.T8 <- DFA(EEG$T8, detrend="poly1", sum.order=1)
# print results for T8
print(DFA.T8)
# plot results for T8
eda.plot(DFA.T8)
```

```
# Detrended fluctuation analysis FC6

DFA.FC6 <- DFA(EEG$FC6, detrend="poly1", sum.order=1)

# print results for FC6

print(DFA.FC6)

# plot results for FC6

eda.plot(DFA.FC6)

# Detrended fluctuation analysis F4

DFA.F4 <- DFA(EEG$F4, detrend="poly1", sum.order=1)

# print results for F4

print(DFA.F4)

# plot results F4

eda.plot(DFA.F4)

# Detrended fluctuation analysis F8

DFA.F8 <- DFA(EEG$F8, detrend="poly1", sum.order=1)

# print results for F8

print(DFA.F8)

# plot results for F8

eda.plot(DFA.F8)

# Detrended fluctuation analysis AF4

DFA.AF4 <- DFA(EEG$AF4, detrend="poly1", sum.order=1)

# print results for AF4

print(DFA.AF4)

# plot results for AF4

eda.plot(DFA.AF4)
```

Appendix K

Program Syntax (GSR – "Bridge" Detrended Fluctuation Analysis)

R program 3.3.1 (fractal package 2.0-1)

```
# clear workspace

rm(list=ls())

# load fractal package

require(fractal)

# read GSR data

gsr <- read.csv(("C:\\User\\location_of_the_file\\file name.csv", header = F)

# labelling GSR data segments of baseline, during, post-activity

names(gsr) <- c("base", "during", "post")

# Central tendency

summary(gsr)

# Detrended fluctuation analysis – baseline GSR

DFA.base <- DFA(gsr$base, detrend="bridge", sum.order=1)

# print results for baseline GSR

print(DFA.base)

# plot results for baseline GSR

eda.plot(DFA.base)

# Detrended fluctuation analysis – during activity GSR

DFA.during <- DFA(gsr$during, detrend="bridge", sum.order=1)

# print results for during activity GSR

print(DFA.during)

# plot results for during activity GSR

eda.plot(DFA.during)
```

```
# Detrended fluctuation analysis – post-activity GSR

DFA.post <- DFA(gsr$post, detrend="bridge", sum.order=1)

# print results for post-activity GSR

print(DFA.post)

# plot results for post-activity GSR

eda.plot(DFA.post)
```

Appendix L: Supplementary ANOVA Tables for Chapter 5

Scaling Exponents (GSR) with other ISLT and ANT variables

Table 23

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Implicit Sequence Learning Task Performance, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Session 1						
Probable trials (RT)						
Intervention x Performance	-	-	-	-	-	-
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	28	0.99	0.04			
Improbable trials (RT)						
Intervention x Performance	-	-	-	-	-	-
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	29	1.20	0.04			
Session 2						
Probable trials (RT)						
Intervention x Performance	1	0.04	0.04	0.82	.37	.03
Intervention x Performance x Diagnosis	1	0.08	0.08	1.82	.19	.07
Error	25	1.13	0.05			
Improbable trials (RT)						
Intervention x Performance	1	0.03	0.03	0.69	.41	.03
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	26	1.21	0.05			

* $p = .05$. N = 35. *Note*. Some ANOVA values are not available as all or majority of the children improved in their reaction time (RT).

Table 24

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Alerting Network Reaction Time Performance, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Alerting Network: Session 1						
No Cue (RT)						
Intervention x Performance	1	0.00	0.00	0.00	.99	.00
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	25	1.17	0.05			
Double Cue (RT)						
Intervention x Performance	-	-	-	-	-	-
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	25	1.07	0.04			
Alerting Network: Session 2						
No Cue (RT)						
Intervention x Performance	1	0.02	0.02	0.35	.56	.02
Intervention x Performance x Diagnosis	1	0.00	0.00	0.01	.91	.00
Error	23	1.20	0.05			
Double Cue (RT)						
Intervention x Performance	1	0.01	0.01	0.27	.61	.01
Intervention x Performance x Diagnosis	1	0.11	0.11	2.76	.11	.11
Error	23	0.89	0.04			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 25

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Alerting Network Accuracy, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Alerting Network: Session 1						
No Cue (Accuracy)						
Intervention x Performance	1	0.02	0.02	0.47	.50	.02
Intervention x Performance x Diagnosis	1	0.01	0.01	0.29	.59	.01
Error	23	1.01	0.04			
Double Cue (Accuracy)						
Intervention x Performance	-	-	-	-	-	-
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	25	1.04	0.04			
Alerting Network: Session 2						
No Cue (Accuracy)						
Intervention x Performance	1	0.01	0.01	0.37	.55	.02
Intervention x Performance x Diagnosis	1	0.04	0.04	0.99	.33	.04
Error	23	0.85	0.04			
Double Cue (Accuracy)						
Intervention x Performance	1	0.02	0.02	0.38	.54	.02
Intervention x Performance x Diagnosis	1	0.07	0.07	1.44	.24	.06
Error	23	1.05	0.05			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 26

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Orienting Network Reaction Time Performance, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Orienting Network: Session 1						
Centre Cue (RT)						
Intervention x Performance	1	0.00	0.00	0.04	.85	.00
Intervention x Performance x Diagnosis	1	0.03	0.03	0.53	.47	.02
Error	23	1.10	0.05			
Spatial Cue (RT)						
Intervention x Performance	-	-	-	-	-	-
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	25	1.16	0.05			
Orienting Network: Session 2						
Centre Cue (RT)						
Intervention x Performance	1	0.07	0.07	1.50	.23	.06
Intervention x Performance x Diagnosis	1	0.00	0.00	0.05	.82	.00
Error	23	1.06	0.05			
Spatial Cue (RT)						
Intervention x Performance	1	0.02	0.02	0.34	.57	.01
Intervention x Performance x Diagnosis	1	0.00	0.00	0.01	.92	.00
Error	23	1.11	0.05			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 27

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Orienting Network Accuracy, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Orienting Network: Session 1						
Centre Cue Accuracy						
Intervention x Performance	1	0.00	0.00	0.05	.82	.002
Intervention x Performance x Diagnosis	1	0.13	0.13	2.85	.11	.11
Error	23	1.03	0.05			
Spatial Cue Accuracy						
Intervention x Performance	1	0.08	0.08	1.76	.20	.07
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	24	1.07	0.05			
Orienting Network: Session 2						
Centre Cue Accuracy						
Intervention x Performance	1	0.00	0.00	0.01	.94	.00
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	24	1.09	0.05			
Spatial Cue Accuracy						
Intervention x Performance	1	0.06	0.06	1.22	.28	.05
Intervention x Performance x Diagnosis	1	0.00	0.00	0.02	.89	.001
Error	23	1.15	0.05			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 28

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Conflict Network Reaction Time Performance, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Conflict Network: Session 1						
Congruent Flanker (RT)						
Intervention x Performance	1	0.02	0.02	0.42	.53	.02
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	24	1.14	0.05			
Incongruent Flanker (RT)						
Intervention x Performance	1	0.21	0.21	5.52	.03*	.19
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	24	0.91	0.04			
Conflict Network: Session 2						
Congruent Flanker (RT)						
Intervention x Performance	1	0.00	0.00	0.02	.88	.00
Intervention x Performance x Diagnosis	1	0.11	0.11	2.86	.10	.11
Error	23	0.90	0.04			
Incongruent Flanker (RT)						
Intervention x Performance	1	0.23	0.23	5.72	.03*	.20
Intervention x Performance x Diagnosis	1	0.10	0.10	2.38	.14	.09
Error	23	0.92	0.05			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 29

Analysis of Covariance of Post-Intervention GSR Scaling Exponent as a Function of Conflict Network Accuracy, Intervention and Diagnosis, With Pre-Intervention and During Intervention GSR Scaling Exponents as Covariates

Source	*df*	*SS*	*MS*	*F*	*p*	Partial η^2
Conflict Network: Session 1						
Congruent Flanker Accuracy						
Intervention x Performance	1	0.01	0.01	0.21	.65	.01
Intervention x Performance x Diagnosis	-	-	-	-	-	-
Error	24	1.15	0.05			
Incongruent Flanker Accuracy						
Intervention x Performance	1	0.22	0.22	6.19	.02*	.21
Intervention x Performance x Diagnosis	1	0.00	0.00	0.01	.95	.00
Error	23	0.81	0.04			
Conflict Network: Session 2						
Congruent Flanker Accuracy						
Intervention x Performance	1	0.02	0.02	0.41	.53	.02
Intervention x Performance x Diagnosis	1	0.04	0.04	0.87	.36	.04
Error	23	1.10	0.05			
Incongruent Flanker Accuracy						
Intervention x Performance	1	0.15	0.15	3.83	.06	.14
Intervention x Performance x Diagnosis	1	0.07	0.07	1.68	.21	.07
Error	23	0.88	0.04			

* $p = .05$. N = 33. Some ANOVA values are not available as all or majority of the children improved in their cognitive performance.

Table 30

Mixed Analysis of Variance of the Scaling Exponents of EEG Frequency Bands as a Function of Accuracy Change, Intervention, and Diagnosis.

Conflict Network – Incongruent Flanker Trials

	ANOVA	
Source	Session 1	Session 2
Theta		
Intervention x Performance	$F(1, 20) = 0.57$, $p = .46$, $r = .17$	$F(1, 20) = 0.79$, $p = .38$, $r = .19$
Intervention x Performance x Diagnosis	$F(1, 20) = 0.15$, $p = .70$, $r = .09$	$F(1, 20) = 0.10$, $p = .75$, $r = .07$
Alpha		
Intervention x Performance	$F(1, 20) = 0.28$, $p = .60$, $r = .12$	$F(1, 20) = 1.29$, $p = .27$, $r = .25$
Intervention x Performance x Diagnosis	$F(1, 20) = 0.94$, $p = .34$, $r = .21$	$F(1, 20) = 0.29$, $p = .60$, $r = .12$
Beta		
Intervention x Performance	$F(1, 20) = 0.39$, $p = .54$, $r = .14$	$F(1, 20) = 2.11$, $p = .16$, $r = .31$
Intervention x Performance x Diagnosis	$F(1, 20) = 0.02$, $p = .90$, $r = .03$	$F(1, 20) = 0.56$, $p = .46$, $r = .17$

Implicit Sequence Learning Task

	ANOVA	
Source	Session 1	Session 2
Theta		
Intervention x Performance	$F(1, 21) = 0.01$, $p = .94$, $r = .02$	$F(1, 20) = 0.21$, $p = .66$, $r = .10$
Intervention x Performance x Diagnosis	-	$F(1, 20) = 0.13$, $p = .73$, $r = .08$
Alpha		
Intervention x Performance	$F(1, 21) = 3.29$, $p = .08$, $r = .37$	$F(1, 20) = 0.66$, $p = .43$, $r = .18$
Intervention x Performance x Diagnosis	-	$F(1, 20) = 0.01$, $p = .92$, $r = .02$
Beta		
Intervention x Performance	$F(1, 21) = 0.09$, $p = .77$, $r = .07$	$F(1, 20) = 0.25$, $p = .62$, $r = .11$
Intervention x Performance x Diagnosis	-	$F(1, 20) = 0.00$, $p = .97$, $r = .00$

* $p = .05$. N = 28. *Note*. The other ANOVA values such as sum of squares, mean square, and errors are not reported as these values are smaller than 0.00.

Table 31

Detrended Fluctuation Analysis of EEG Alpha Frequency Band of Various Time Length for Children with a Neurodevelopmental Condition

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: ASD/ADHD - Increased Error	3 mins/ 23040	Pre	0.9813	0.9828	0.9833	0.9830	0.9825	0.9825	0.9819	0.9810	0.9809	0.9807	0.9811	0.9811	0.9812	0.9811
		Post	0.9841	0.9830	0.9850	0.9842	0.9808	0.9786	0.9830	0.9805	0.9808	0.9816	0.9827	0.9835	0.9822	0.9828
	2 mins/ 15360	Pre	0.9818	0.9822	0.9818	0.9823	0.9825	0.9821	0.9830	0.9785	0.9814	0.9810	0.9814	0.9815	0.9813	0.9808
		Post	0.9828	0.9818	0.9834	0.9818	0.9810	0.9818	0.9845	0.9812	0.9809	0.9808	0.9808	0.9821	0.9807	0.9815
	1 min/ 7680	Pre	0.9786	0.9775	0.9790	0.9784	0.9803	0.9803	0.9808	0.9777	0.9789	0.9782	0.9773	0.9786	0.9777	0.9806
		Post	0.9830	0.9827	0.9831	0.9830	0.9806	0.9782	0.9843	0.9803	0.9806	0.9817	0.9808	0.9789	0.9816	0.9782
	30 secs/ 3840	Pre	0.9835	0.9820	0.9843	0.9823	0.9852	0.9827	0.9833	0.9807	0.9822	0.9826	0.9825	0.9810	0.9821	0.9828
		Post	0.9813	0.9823	0.9821	0.9820	0.9822	0.9788	0.9802	0.9775	0.9793	0.9821	0.9797	0.9805	0.9813	0.9802
2: ASD/ADHD - Reduced error	3 mins/ 23040	Pre	0.9814	0.9835	0.9834	0.9823	0.9825	0.9820	0.9815	0.9836	0.9833	0.9844	0.9841	0.9832	0.9820	0.9814
		Post	0.9864	0.9856	0.9845	0.9855	0.9851	0.9859	0.9873	0.9863	0.9873	0.9865	0.9872	0.9866	0.9869	0.9866
	2 mins/ 15360	Pre	0.9814	0.9835	0.9837	0.9839	0.9823	0.9808	0.9803	0.9827	0.9822	0.9832	0.9829	0.9829	0.9804	0.9804
		Post	0.9862	0.9854	0.9841	0.9854	0.9847	0.9864	0.9867	0.9858	0.9865	0.9867	0.9877	0.9866	0.9866	0.9865
	1 min/ 7680	Pre	0.9810	0.9812	0.9811	0.9814	0.9829	0.9814	0.9829	0.9824	0.9790	0.9803	0.9830	0.9831	0.9820	0.9807
		Post	0.9786	0.9780	0.9755	0.9772	0.9793	0.9799	0.9819	0.9820	0.9834	0.9824	0.9804	0.9792	0.9788	0.9779
	30 secs/ 3840	Pre	0.9815	0.9855	0.9799	0.9870	0.9840	0.9765	0.9799	0.9844	0.9810	0.9831	0.9848	0.9848	0.9816	0.9795
		Post	0.9815	0.9799	0.9789	0.9808	0.9796	0.9768	0.9787	0.9759	0.9789	0.9805	0.9808	0.9815	0.9813	0.9812

Table 32

Detrended Fluctuation Analysis of EEG Alpha Frequency Band of Various Time Length for Children with a Typical Development

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: TD - Increased Error	3 mins/ 23040	Pre	0.9763	0.9809	0.9769	0.9768	0.9806	0.9717	0.9780	0.9790	0.9751	0.9765	0.9831	0.9770	0.9771	0.9772
		Post	0.9805	0.9844	0.9804	0.9814	0.9813	0.9759	0.9762	0.9796	0.9787	0.9814	0.9820	0.9814	0.9831	0.9812
	2 mins/ 15360	Pre	0.9794	0.9835	0.9803	0.9818	0.9845	0.9789	0.9826	0.9829	0.9804	0.9803	0.9823	0.9788	0.9797	0.9795
		Post	0.9769	0.9786	0.9770	0.9772	0.9802	0.9756	0.9766	0.9794	0.9779	0.9794	0.9781	0.9775	0.9791	0.9776
	1 min/ 7680	Pre	0.9783	0.9807	0.9797	0.9793	0.9837	0.9802	0.9821	0.9816	0.9802	0.9802	0.9796	0.9783	0.9793	0.9791
		Post	0.9794	0.9816	0.9796	0.9798	0.9803	0.9797	0.9803	0.9777	0.9756	0.9799	0.9789	0.9795	0.9804	0.9798
	30 secs/ 3840	Pre	0.9744	0.9775	0.9761	0.9763	0.9797	0.9809	0.9820	0.9805	0.9761	0.9749	0.9764	0.9753	0.9745	0.9748
		Post	0.9811	0.9832	0.9818	0.9802	0.9794	0.9705	0.9712	0.9730	0.9652	0.9782	0.9790	0.9818	0.9807	0.9812
2: TD - Reduced error	3 mins/ 23040	Pre	0.9803	0.9825	0.9804	0.9811	0.9801	0.9812	0.9796	0.9794	0.9801	0.9828	0.9832	0.9682	0.9776	0.9809
		Post	0.9825	0.9818	0.9831	0.9703	0.9838	0.9818	0.9784	0.9797	0.9815	0.9842	0.9837	0.9837	0.9833	0.9829
	2 mins/ 15360	Pre	0.9805	0.9824	0.9801	0.9807	0.9781	0.9790	0.9751	0.9769	0.9795	0.9823	0.9820	0.9623	0.9766	0.9808
		Post	0.9797	0.9794	0.9799	0.9639	0.9824	0.9819	0.9755	0.9748	0.9783	0.9823	0.9824	0.9812	0.9825	0.9820
	1 min/ 7680	Pre	0.9813	0.9816	0.9819	0.9777	0.9817	0.9779	0.9769	0.9814	0.9799	0.9842	0.9834	0.9617	0.9737	0.9814
		Post	0.9803	0.9820	0.9813	0.9533	0.9820	0.9807	0.9763	0.9773	0.9767	0.9810	0.9814	0.9808	0.9815	0.9822
	30 secs/ 3840	Pre	0.9836	0.9825	0.9823	0.9803	0.9802	0.9779	0.9800	0.9785	0.9795	0.9869	0.9814	0.9488	0.9743	0.9847
		Post	0.9823	0.9808	0.9827	0.9605	0.9815	0.9839	0.9777	0.9782	0.9789	0.9834	0.9855	0.9843	0.9847	0.9860

Table 33

Detrended Fluctuation Analysis of EEG Beta Frequency Band of Various Time Length for Children with a Neurodevelopmental Condition

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: ASD/ADHD - Increased Error	3 mins/ 23040	Pre	0.9857	0.9856	0.9849	0.9855	0.9852	0.9832	0.9847	0.9851	0.9850	0.9852	0.9854	0.9843	0.9853	0.9851
		Post	0.9848	0.9838	0.9851	0.9830	0.9831	0.9776	0.9847	0.9823	0.9836	0.9843	0.9841	0.9846	0.9840	0.9847
	2 mins/ 15360	Pre	0.9855	0.9838	0.9832	0.9831	0.9835	0.9806	0.9823	0.9836	0.9843	0.9848	0.9842	0.9830	0.9840	0.9847
		Post	0.9849	0.9835	0.9853	0.9811	0.9823	0.9772	0.9840	0.9815	0.9825	0.9838	0.9836	0.9837	0.9835	0.9841
	1 min/ 7680	Pre	0.9835	0.9838	0.9820	0.9802	0.9823	0.9781	0.9813	0.9842	0.9820	0.9835	0.9831	0.9816	0.9833	0.9845
		Post	0.9857	0.9818	0.9852	0.9807	0.9793	0.9740	0.9836	0.9798	0.9803	0.9809	0.9809	0.9820	0.9805	0.9829
	30 secs/ 3840	Pre	0.9867	0.9846	0.9824	0.9800	0.9837	0.9779	0.9831	0.9827	0.9809	0.9841	0.9847	0.9811	0.9843	0.9847
		Post	0.9866	0.9811	0.9866	0.9778	0.9763	0.9720	0.9813	0.9811	0.9840	0.9807	0.9812	0.9853	0.9800	0.9865
2: ASD/ADHD - Reduced error	3 mins/ 23040	Pre	0.9838	0.9851	0.9831	0.9843	0.9849	0.9843	0.9832	0.9836	0.9834	0.9854	0.9846	0.9827	0.9834	0.9829
		Post	0.9850	0.9845	0.9855	0.9847	0.9815	0.9843	0.9847	0.9842	0.9856	0.9852	0.9853	0.9854	0.9852	0.9855
	2 mins/ 15360	Pre	0.9823	0.9828	0.9827	0.9827	0.9821	0.9818	0.9816	0.9812	0.9807	0.9823	0.9825	0.9806	0.9811	0.9808
		Post	0.9837	0.9836	0.9836	0.9835	0.9809	0.9827	0.9840	0.9827	0.9838	0.9836	0.9846	0.9844	0.9842	0.9845
	1 min/ 7680	Pre	0.9815	0.9820	0.9803	0.9806	0.9817	0.9774	0.9800	0.9804	0.9787	0.9812	0.9822	0.9794	0.9807	0.9788
		Post	0.9820	0.9809	0.9818	0.9813	0.9765	0.9809	0.9835	0.9800	0.9820	0.9829	0.9830	0.9841	0.9824	0.9830
	30 secs/ 3840	Pre	0.9783	0.9806	0.9767	0.9812	0.9783	0.9752	0.9767	0.9742	0.9729	0.9799	0.9801	0.9755	0.9772	0.9760
		Post	0.9804	0.9805	0.9793	0.9808	0.9730	0.9824	0.9817	0.9770	0.9767	0.9810	0.9847	0.9839	0.9833	0.9827

Table 34

Detrended Fluctuation Analysis of EEG Beta Frequency Band of Various Time Length for Children with a Typical Development

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: TD - Increased Error	3 mins/ 23040	Pre	0.9836	0.9836	0.9841	0.9828	0.9850	0.9840	0.9838	0.9853	0.9843	0.9848	0.9825	0.9829	0.9844	0.9840
		Post	0.9838	0.9875	0.9832	0.9844	0.9849	0.9823	0.9821	0.9855	0.9842	0.9841	0.9809	0.9838	0.9865	0.9847
	2 mins/ 15360	Pre	0.9841	0.9814	0.9841	0.9844	0.9828	0.9829	0.9838	0.9850	0.9830	0.9847	0.9784	0.9822	0.9841	0.9838
		Post	0.9826	0.9848	0.9827	0.9827	0.9850	0.9819	0.9813	0.9844	0.9838	0.9835	0.9786	0.9830	0.9845	0.9834
	1 min/ 7680	Pre	0.9829	0.9822	0.9823	0.9835	0.9838	0.9825	0.9811	0.9844	0.9832	0.9841	0.9758	0.9834	0.9838	0.9836
		Post	0.9821	0.9858	0.9821	0.9838	0.9852	0.9819	0.9810	0.9847	0.9821	0.9836	0.9780	0.9820	0.9835	0.9826
	30 secs/ 3840	Pre	0.9824	0.9815	0.9819	0.9822	0.9821	0.9826	0.9833	0.9861	0.9838	0.9840	0.9716	0.9834	0.9829	0.9828
		Post	0.9805	0.9853	0.9816	0.9811	0.9858	0.9810	0.9804	0.9831	0.9785	0.9825	0.9773	0.9791	0.9817	0.9811
2: TD - Reduced error	3 mins/ 23040	Pre	0.9820	0.9837	0.9810	0.9833	0.9840	0.9820	0.9832	0.9819	0.9815	0.9862	0.9834	0.9800	0.9817	0.9833
		Post	0.9829	0.9839	0.9827	0.9803	0.9845	0.9840	0.9831	0.9834	0.9817	0.9842	0.9832	0.9833	0.9833	0.9828
	2 mins/ 15360	Pre	0.9812	0.9831	0.9799	0.9816	0.9833	0.9823	0.9812	0.9814	0.9822	0.9863	0.9838	0.9777	0.9792	0.9829
		Post	0.9803	0.9823	0.9796	0.9775	0.9835	0.9828	0.9820	0.9801	0.9787	0.9832	0.9817	0.9811	0.9818	0.9804
	1 min/ 7680	Pre	0.9819	0.9824	0.9812	0.9798	0.9829	0.9820	0.9798	0.9816	0.9827	0.9859	0.9826	0.9779	0.9769	0.9833
		Post	0.9810	0.9821	0.9813	0.9777	0.9822	0.9786	0.9810	0.9821	0.9756	0.9832	0.9821	0.9816	0.9827	0.9808
	30 secs/ 3840	Pre	0.9791	0.9783	0.9778	0.9757	0.9832	0.9804	0.9812	0.9842	0.9859	0.9849	0.9801	0.9760	0.9734	0.9809
		Post	0.9825	0.9815	0.9823	0.9773	0.9829	0.9799	0.9859	0.9810	0.9788	0.9856	0.9834	0.9825	0.9836	0.9806

Table 35

Detrended Fluctuation Analysis of EEG Theta Frequency Band of Various Time Length for Children with a Neurodevelopmental Condition

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: ASD/ADHD - Increased Error	3 mins/ 23040	Pre	0.9836	0.9844	0.9839	0.9834	0.9829	0.9821	0.9835	0.9849	0.9836	0.9842	0.9836	0.9838	0.9845	0.9816
		Post	0.9839	0.9857	0.9836	0.9841	0.9862	0.9801	0.9817	0.9811	0.9826	0.9848	0.9851	0.9838	0.9859	0.9838
	2 mins/ 15360	Pre	0.9848	0.9848	0.9849	0.9835	0.9835	0.9820	0.9848	0.9843	0.9841	0.9839	0.9828	0.9826	0.9841	0.9807
		Post	0.9835	0.9843	0.9826	0.9830	0.9860	0.9809	0.9830	0.9802	0.9816	0.9845	0.9851	0.9843	0.9859	0.9837
	1 min/ 7680	Pre	0.9853	0.9838	0.9839	0.9853	0.9846	0.9845	0.9845	0.9888	0.9840	0.9823	0.9820	0.9846	0.9835	0.9849
		Post	0.9829	0.9826	0.9830	0.9839	0.9847	0.9789	0.9821	0.9765	0.9808	0.9830	0.9839	0.9868	0.9844	0.9855
	30 secs/ 3840	Pre	0.9829	0.9788	0.9843	0.9798	0.9804	0.9808	0.9815	0.9900	0.9860	0.9802	0.9746	0.9825	0.9778	0.9808
		Post	0.9798	0.9834	0.9784	0.9839	0.9845	0.9823	0.9818	0.9826	0.9788	0.9813	0.9798	0.9820	0.9822	0.9844
2: ASD/ADHD - Reduced error	3 mins/ 23040	Pre	0.9742	0.9802	0.9799	0.9768	0.9816	0.9830	0.9802	0.9815	0.9832	0.9829	0.9802	0.9791	0.9751	0.9752
		Post	0.9814	0.9826	0.9802	0.9821	0.9808	0.9790	0.9824	0.9833	0.9846	0.9829	0.9807	0.9813	0.9816	0.9816
	2 mins/ 15360	Pre	0.9736	0.9809	0.9815	0.9828	0.9791	0.9839	0.9819	0.9809	0.9820	0.9824	0.9803	0.9800	0.9740	0.9745
		Post	0.9830	0.9843	0.9816	0.9829	0.9811	0.9798	0.9832	0.9847	0.9857	0.9840	0.9831	0.9838	0.9831	0.9837
	1 min/ 7680	Pre	0.9813	0.9831	0.9835	0.9859	0.9805	0.9862	0.9877	0.9831	0.9823	0.9809	0.9829	0.9835	0.9798	0.9808
		Post	0.9818	0.9826	0.9780	0.9818	0.9801	0.9828	0.9843	0.9860	0.9856	0.9830	0.9797	0.9812	0.9809	0.9804
	30 secs/ 3840	Pre	0.9718	0.9751	0.9741	0.9818	0.9779	0.9841	0.9821	0.9762	0.9772	0.9743	0.9772	0.9773	0.9744	0.9743
		Post	0.9827	0.9799	0.9865	0.9804	0.9799	0.9846	0.9807	0.9874	0.9825	0.9847	0.9814	0.9832	0.9817	0.9823

Table 36

Detrended Fluctuation Analysis of EEG Theta Frequency Band of Various Time Length for Children with a Typical Development

Participant	Duration/ Sample length	Time	AF3	F7	F3	FC5	T7	P7	O1	O2	P8	T8	FC6	F4	F8	AF4
1: TD - Increased Error	3 mins/ 23040	Pre	0.9811	0.9797	0.9815	0.9794	0.9782	0.9803	0.9817	0.9826	0.9823	0.9814	0.9822	0.9805	0.9815	0.9813
		Post	0.9836	0.9873	0.9841	0.9825	0.9849	0.9808	0.9804	0.9809	0.9776	0.9820	0.9828	0.9832	0.9854	0.9842
	2 mins/ 15360	Pre	0.9856	0.9803	0.9865	0.9835	0.9818	0.9836	0.9817	0.9837	0.9859	0.9853	0.9822	0.9843	0.9857	0.9856
		Post	0.9827	0.9845	0.9827	0.9823	0.9828	0.9811	0.9812	0.9826	0.9775	0.9818	0.9803	0.9826	0.9845	0.9833
	1 min/ 7680	Pre	0.9830	0.9798	0.9844	0.9780	0.9817	0.9853	0.9820	0.9844	0.9859	0.9845	0.9800	0.9813	0.9841	0.9833
		Post	0.9817	0.9839	0.9814	0.9817	0.9829	0.9820	0.9840	0.9819	0.9704	0.9803	0.9779	0.9812	0.9825	0.9817
	30 secs/ 3840	Pre	0.9789	0.9782	0.9802	0.9771	0.9813	0.9830	0.9836	0.9832	0.9803	0.9807	0.9808	0.9783	0.9806	0.9793
		Post	0.9842	0.9895	0.9822	0.9852	0.9858	0.9786	0.9797	0.9819	0.9505	0.9831	0.9789	0.9828	0.9851	0.9845
2: TD - Reduced error	3 mins/ 23040	Pre	0.9837	0.9815	0.9828	0.9775	0.9812	0.9852	0.9864	0.9838	0.9842	0.9837	0.9847	0.9562	0.9647	0.9845
		Post	0.9836	0.9835	0.9827	0.9639	0.9838	0.9816	0.9805	0.9792	0.9790	0.9828	0.9823	0.9837	0.9817	0.9826
	2 mins/ 15360	Pre	0.9832	0.9801	0.9823	0.9765	0.9794	0.9872	0.9836	0.9826	0.9835	0.9824	0.9824	0.9442	0.9656	0.9838
		Post	0.9817	0.9829	0.9809	0.9577	0.9854	0.9829	0.9778	0.9773	0.9779	0.9825	0.9820	0.9828	0.9828	0.9825
	1 min/ 7680	Pre	0.9876	0.9809	0.9879	0.9786	0.9832	0.9863	0.9889	0.9854	0.9833	0.9856	0.9847	0.9517	0.9701	0.9864
		Post	0.9816	0.9820	0.9813	0.9463	0.9869	0.9812	0.9721	0.9772	0.9765	0.9838	0.9842	0.9825	0.9869	0.9831
	30 secs/ 3840	Pre	0.9844	0.9768	0.9838	0.9804	0.9801	0.9867	0.9869	0.9890	0.9892	0.9899	0.9897	0.9286	0.9762	0.9861
		Post	0.9835	0.9852	0.9844	0.9600	0.9860	0.9817	0.9744	0.9761	0.9834	0.9887	0.9887	0.9870	0.9896	0.9900