

Amelioration of the Impact of Physical Fatigue on Cognitive
Performance by Phytochemicals: The Effect of a Blackcurrant
Supplement

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Abstract

Exercise-induced physical fatigue is thought to impair the cognitive functioning, and therefore mental performance, of the brain. Intervention studies have demonstrated that phytochemical supplementation can facilitate improved cognitive and physical performance. However, little is known about phytochemical supplementations' ability to ameliorate physical fatigue effects on cognitive performance upon congestion. To investigate this hypothesis, the present study investigated the effects phytochemical compounds, from a blackcurrant supplement, had in regards to reducing physical fatigue effects on cognitive performance while under mental loads. Seventy-two healthy participants completed >10 mins of a high intensity intermittent cycling task (HIIT) (physical fatigue cohort) or >10 mins watching an emotionally neutral documentary (control cohort). Half of the participants in each condition received a blackcurrant supplement one hour before beginning the experimental session. Baseline cognitive tasks and mood questionnaires were completed before ingestion of a blackcurrant extract, again before post-task measurements were completed, and also immediately following the experimental session. Analysis of the subjective self-reports revealed that HIIT was successful at inducing physical fatigue, however, had no effect on subsequent cognitive performance. Further analyses demonstrated that supplementation with a blackcurrant extract had no influence on cognitive performance. The null results for an effect of physical fatigue on cognitive performance made interpretation of this finding difficult. Overall, effect size calculations indicated that a larger sample size would not have resulted in statistically significant findings. It was concluded that the specific high intensity intermittent exercise used in the present study, did not induce a level of fatigue in participants' that would subsequently impair cognitive performance. Blackcurrant supplement did not demonstrate an ability to enhance cognitive performance following a physically fatiguing task. Possible explanations for these findings are discussed and some potentially useful future studies outlined in the second and third chapters.

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Table of Contents

Abstract.....	2
Acknowledgements.....	3
Table of Contents.....	5
List of Figures.....	9
List of Tables.....	10
Chapter 1:	
Background.....	11
Chapter 2: Phytochemicals and COGNITIVE PERFORMANCE...17	
What are phytochemicals.....	16
Molecular mechanisms underlying phytochemical influence.....	18
Potential public health benefits from phytochemicals.....	20
Phytochemical rich food: benefits to cognitive performance.....	25
Berry fruit phytochemicals and cognitive improvement.....	30
Methodological differences in phytochemical research.....	33
Summary.....	33
Chapter 3: Exercise and COGNITIVE PERFORMANCE.....35	
Theoretical Models of the Interaction in the Exercise-Cognition Relationship.....	41
Methodological differences among previous research.....	42
Exercise induced fatigue & subsequent cognitive performance.....	44
Physiological explanations of the exercise-cognition relationship.....	47
Summary.....	48

The present study.....	49
Hypothesis one.....	50
Hypothesis two.....	52
Chapter 4: Methodology.....	54
Participants.....	54
Group assignment.....	54
Apparatus and Measures.....	55
Cognitive tasks.....	55
Stroop.....	55
Digit Symbol Substitution.....	56
Digit Span Backwards.....	57
Trail Making Task (B).....	58
Choice Reaction Time Task.....	59
Subjective Measures.....	59
Mental and Physical State and Trait Energy and Fatigue Scales.....	60
Ratings of Perceived Exertion.....	60
Baeke Questionnaire and Health Screening.....	61
Heart Rate Measure.....	61
Lactate Measure.....	61
Treatments.....	61
Exercise induced fatigue manipulation.....	61
Control Task.....	62
Phytochemical Manipulation.....	62
Blackcurrant extract.....	62
Design and Analysis.....	62
Procedure.....	63
Familiarisation.....	64

Main Trial.....	65
First stage.....	65
Second stage.....	65
Third stage-Exercise- session.....	66
Fourth stage.....	66
Control Session.....	66
Fifth Stage-Debrief.....	67
Chapter 4: Results.....	68
Fatigue Manipulation.....	68
Stroop- Overall.....	69
Stroop-Congruent.....	69
Stroop-Noncongruent.....	70
Digit Span Backwards.....	70
Digit Symbol Substitution.....	71
Trail Making Task (B).....	73
Choice Reaction Time Task.....	73
Summary.....	74
Chapter 6: Discussion.....	75
Inducing Physical Fatigue.....	77
Hypothesis One: Physical Fatigue Impairs Subsequent Cognitive Performance.....	77
Hypothesis Two: Blackcurrant Supplement Ameliorates the Effects of Fatigue on Cognitive Performance.....	90
Limitations.....	94
Conclusions.....	97
References.....	100
Appendices.....	118
Appendix A: Advertisement Flyer.....	119

Appendix B: Mental & Physical State and Trait Energy and Fatigue	
Scale.....	120
Appendix C: Health Screening Form.....	128
Appendix D: Information Sheet.....	131
Appendix E: Baeke Habitual Physical Activity Questionnaire.....	140
Appendix F: Ratings of Perceived Exertion.....	144
Appendix G: Consent Form.....	145
Appendix H: List of Foods to Avoid.....	147
Appendix I: ANCOVA Between Subjects Tests Tables.....	148

List of Figures

Figure 1: Visual representation of the Yerks & Dodson’s inverted U-hypothesis.....	39
Figure 2: Visual representation of the Stroop task.....	51
Figure 3: Visual representation of the Digit Symbol Substitution.....	52
Figure 4: Visual representation of the Digit Span Backwards.....	53
Figure 5: Visual representation of the Trail Making Test (B).....	54
Figure 5: Visual representation of the Choice Reaction Time Task.....	55
Figure 6: Interaction between condition and intervention for digit span backwards.....	69
Figure 7: Interaction between condition and intervention for Digit Symbol Substitution.....	72

List of Tables

Table 1: Group assignment in accordance to gender and condition.....	51
Table 2: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Stoop (overall).....	69
Table 3: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Stroop (congruent).....	69
Table 4: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Stroop (non congruent).....	70
Table 5: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Digit Span Backwards.....	70
Table 6: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Digit symbol substitution.....	71
Table 7: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Trail Making Task (B).....	73
Table 8: Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Choice Reaction Time Task.....	73

CHAPTER 1 Background

Understanding and influencing the relationship between nutrition and human health is considered a major research objective. A plethora of research has indicated that biologically active food can be beneficial for mental and physical health. The hypothesis that food properties reliably influence an individual's cognitive performance is receiving increasing experimental support (Dye, Lluch, & Blundell, 2000; Maggini & Spitzer, 2013; Tomporowski, 2003; Van-Praag, 2009).

This field of scientific inquiry is still in its infancy. The relationship between nutrition and human functioning is complex, oftentimes revealing unanticipated behavioural consequences; for instance, increased information processing speed was observed just from inhaling the scent of a Rosemary extract (Lindheimer, 2013). Research presently emphasises that some natural foods have multiple benefits, for example, controlling appetite, affecting cognitive performance, or influencing an individual's mood state (Van-Praag, 2009).

Foods that have an impact upon human health, cognitive functioning, and physical performance are defined as functional foods (Rincón-León, 2003). Historically, diet and nutrition have been considered a way of providing the human body with the necessary energy for survival. However, certain functional foods have an ability to provide benefits to human functioning above and beyond what is necessary to merely survive (Rincón-León, 2003). For instance, diets rich in omega-3 fatty acids are recognised for their ability to support higher order mental functioning in humans (Gómez-Pinilla, 2010). These fatty acids are not considered necessary for survival, but can provide additional physiological benefits when consumed. Richardson and Montgomery (2005) tested the hypothesis that omega-3 fatty acids provided additional benefits to higher order cognitive functioning. For instance, children across mainstream schools in the United Kingdom with developmental coordination disorder (DCD) demonstrated improvements in behaviour, reading, and spelling after receiving omega-3 supplements over a 3-month period. This crossover study revealed similar changes in the placebo cohort, which crossed over from the placebo onto the treatment of omega-3 supplement after 3-months. This evidence supports the theory that nutritional supplements are able to exert positive effects on higher order

functioning. However, it is important to exercise caution when drawing conclusions from prior literature. As ability exerted by a specific nutrient providing beneficial effects that can be generalised to global populations are difficult. For example, the above study was performed on children primarily from one ethnic background. Therefore, the claim that functional foods will provide benefits to human functioning still requires further scientific validation.

Although research appears to demonstrate an important impact of functional foods on cognitive performance, a single unifying model of the specific causal actions from functional foods remains elusive. Holistically, associations between different types of functional foods, their specific biological properties, and the mechanisms through which these foods act in a beneficial manner on human health remain poorly understood. Current research hypothesises that functional foods provide benefits through interacting with cellular pathways and modulating neurotransmitters (Sokolov, Pavlova, Klosterhalfen, & Ench, 2013). For instance, in-vivo tests (tests on living organisms) have shown that flavonoids are capable of improving synaptic plasticity and learning (Van-Praag, 2009). In addition, brain-derived neurotropic factor (BDNF), the regulator of synaptic plasticity, also functions as a metabolic modulator in response to signals produced from food consumption.

Research over the years has produced some significant evidence in regards to dietary nutrients and their impact on brain health and cognitive functioning. Since nutrition is an integral aspect of daily living, substances that have the potential to modulate brain health and cognitive performance need to be examined closely. Specifically, evidence has indicated a positive relationship between greater intake of nutrients and cognitive performance. For example, breakfast studies with children have demonstrated improvements in attention span and vigilance tasks, when children were provided with a breakfast. Following an observation that the breakfast cohort outperformed children who did not consume breakfast (Llyod, Peter, Rogers, & Hedderley, 1996).

On the other hand, there are studies that find some dietary patterns are detrimental to health and cognitive functioning. As an example, Gómez-Pinilla (2011) investigated the effects of a diet high in saturated fats on the cognitive functioning of rodents. It was observed that such a diet resulted in a reduced level of molecular substrates that

are necessary for the support of cognitive performance. Such a reduction is associated with increased risk of neurological dysfunction, that is, poorer performance on learning tasks. This study also reported that diets high in sucrose would have similar negative effects on neurological functioning (Gómez-Pinilla, 2011).

The fast-evolving technological world of today represents a complex and challenging environment for humans. As such there is a demand for food sources that will help to increase individuals' performance and productivity. To date, the majority of this research investigating food substances, and their proprietorial beneficial or detrimental effects on brain health and cognitive functioning, has been investigated by scrutinising the major macronutrient groups.

Macronutrients can be divided into three separate food groups; carbohydrates, proteins, and fats. These three foods produce glucose, amino acids, and fatty acids upon consumption (Dye et al., 2000). Previous studies have widely investigated macronutrient relationships on cognitive performance across diverse populations of various ages. As one carbohydrate drink supplement study demonstrated, a significant improvement on vigilance tasks were observed in a military cohort on application of the supplement. (Lieberman, Tharion, Shukitt-Hale, Speckman, & Tulley, 2002). In another study, elderly participants' with poor baseline memory showed that 50g of carbohydrate-rich foods improved verbal declarative memory and performance on a visuomotor task (Kaplan, Greenwood, Wincour, & Wolever, 2000).

The brain's major fuel source is glucose, a derivative of carbohydrate (Hoyland et al., 2008). There is an abundance of studies corroborating that cognitive functioning can be improved following ingestion of glucose or carbohydrate-rich foods (Dye & Blundell, 2002; Kaplan et al., 2000; Lieberman et al., 2002). It should be highlighted, however, that a dose-response relationship appears to exist between glucose and carbohydrate-rich food intake and cognitive performance. Specifically, carbohydrate foods are quantified on a glycaemic index scale; this index refers to the rate of blood-glucose elevation in response to consumption of a carbohydrate (Nabb & Benton, 2006). This, in turn, will have an impact on cognitive functioning. Essentially this can be refined to mean that carbohydrate based macronutrients quantified as having a high glycaemic index will cause a sharp increase in blood glucose levels. Such rapid

increases in blood glucose may impose undue metabolic stress on the brain, thus, creating challenging conditions for cognitive functioning.

The reason behind such a reduced capacity for cognitive functioning with high glycaemic foods is assessed to be associated with insulin level increases during glucose metabolism, causing disturbances to metabolite concentrations. Internally stable metabolic conditions will favour high cognitive functioning, whereas disturbances to internal metabolite concentrations will interfere with cognitive functioning (Fischer et al., 2002; Hoyer, 2000; Nabb & Benton, 2006; Park, 2001). In contrast, carbohydrates quantified with a low glycaemic index, will slowly release glucose into the bloodstream. That in turn, appears to produce more favourable conditions for cognitive functioning. This is considered to occur as a result of a more stable internal environment of metabolic concentrations (Fischer et al., 2002; Kaplan et al., 2000; Nabb & Benton, 2006)

There is also practical importance to macronutrient impacts upon brain health and functioning. Other macronutrient components, nutrients, have the potential to produce similar effects to what has been observed in macronutrient studies. For example, caffeine is widely researched as a popular stimulant additive and is a behaviourally active constituent in many popular foods, e.g. coffee, teas, and colas (Lieberman, 2003). Caffeine appears to exert its effects on the putative inhibitory neurotransmitters, consequently influencing a broad range of cognitive functioning. For instance, GABA, the main inhibitory transmitter, is released during sleep. Individuals suffering from sleep deprivation are likely to experience GABA accumulation in neurons, leading to states of impaired performance. Caffeine increases individual's level of arousal, especially during sleep deprivation, through blocking adenosine, a chemical that causes drowsiness and accumulates during waking periods (Kalat, 2009). Thus, caffeine's most reliable effects tend to occur on vigilance, focus, and related mood states, e.g. fatigue (Lieberman, 2003). To elaborate this dynamics, caffeine doses of 100-600 mg given to participants undergoing physiological stress helps to maintain cognitive performance (Lieberman, 2003). Dietary factors that may influence the performance of individuals involved in cognitively demanding situations, such as for military and police personnel are critically important. In saying this, consideration of the context in which a nutrient is

tested is essential when determining if a particular nutrient is responsible for altering cognitive performance. For instance, tyrosine, an amino acid, possesses factors that mitigate acute stress responses and improves cognitive functioning in individuals experiencing sleep deprivation. However, such cognitive performance enhancements are only observed in acutely stressed individuals; there is no effect on unstressed, rested individuals (Lieberman, 2003). Therefore, when examining the impact of dietary nutrients on cognitive functioning, it is important to take physical states, such as fatigue and stress, into account.

A synergy needs to occur between daily dietary intake and daily physical activity levels in order to achieve an optimal level of physical and cognitive performance. The body must have efficient production of energy and its subsequent availability in order to perform, and recover, at optimal rates. Adenosine triphosphate (ATP) is the body's molecular energy source. ATP is formed from the foods consumed, providing chemical energy to muscles and other body parts when and where it's needed (Kalat, 2009). Primarily, ATP is derived from macronutrients; however, in order for macronutrients to utilise ATP the body requires micronutrients. Micronutrients are dietary components, e.g. vitamins and minerals, which enable the body to utilise macronutrients for all physiological activities (Lukaski, 2004).

While micronutrients are only necessary in minuscule amounts, dietary habits that fail to take into account micronutrient consumption are likely to result in substandard physical ability. Substandard performance only occurs with micronutrient deficiency. Lukaski (2004) implied that there is a dose-response association between micronutrient intake and physical performance specifically, micronutrient intakes will not follow the adage of 'more is better' instead overly saturated levels of micronutrients will lead to enhanced performances. Thus, the following chapter will introduce micronutrient polyphenolic compounds known as phytochemicals. Phytochemicals are family members of the most common class of polyphenolic compounds, referred to collectively as flavonoids. Anthocyanins are one of the largest groups of water-soluble pigments within the plant kingdom (Ghosh & Konishi, 2007). For the purpose of this study, the terminology of phytochemical will be used from here out.

High levels of phytochemicals are found in fruits and vegetables. Previous research has revealed flavonoids produce a multitude of actions that correspondingly enhance mental and physical health in animals and humans (Shukitt-Hale et al., 2005). The following chapter will also introduce and describe health-enhancing actions exerted by flavonoids as observed within current literature. Theoretical underlying mechanisms of actions are discussed. The chapter will conclude with a discussion of evidential reports on individual dietary habits, and how they may affect cognitive functioning. Considerations made regarding methodological differences among studies and their potential role influencing observed effect sizes in literature will also be considered.

CHAPTER 2 Flavonoids and COGNITIVE PERFORMANCE

During the past two decades, polyphenolic research has intensified, resulting from increased awareness, and understanding of the diverse array of protective effects. These effects are elicited through consumption of phytochemical compounds. The evidential data emerging from these studies suggests that phytochemical compounds potentially evoke a multiplicity of health benefiting actions. Such effects are inclusive of antioxidant, antiallergic, antiinflammatory, and anticarcinogenic actions. In addition to these known actions, it is increasingly evident that phytochemicals will also provide some protection against cardiovascular damage (Pojer, Mattivi, Johnson, & Stockley, 2013). Phytochemical compounds possess a unique ability to cross the mammalian blood-brain barrier (Kalt et al., 2008; Papandreou et al., 2009; Shukitt-Hale et al., 2005; Spencer, Vafeiadou, Williams, & Vauzour, 2012). Animal studies have demonstrated that phytochemicals are rapidly absorbed from the stomach, quickly entering the mammalian brain (Pojer et al., 2013).

The emerging evidence emphasizes the importance of dietary factors for control of molecular systems involved in physical, and cognitive performance (Wu, Ying, & Gómez-Pinilla, 2004; van Praag, 2009). Understanding the mechanisms of actions, through which flavonoids exert modulating neuroprotective effects, will help health practitioners manipulate individual dietary habits, in order to promote neuroprotection, leading to a greater evolution of cognitive functioning (Shukitt-Hale et al., 2005; Sokolov et al., 2013).

What are phytochemicals?

It has long been recognised that fruit and vegetables are beneficial for human health. Recent research indicates that the molecular actions, exerting beneficial effects, can be attributed to the specific bioactive polyphenolic compounds within fruit, and vegetables (Hurst & Hurst, 2013). Phytochemical polyphenol molecules are part of a plants defensive mechanism: they are secondary metabolites, generated by the plant to defend against both UV exposure, and infestation – be it insect, bacterial or fungal (Hurst & Hurst, 2013). Additionally, these metabolites can be found in a plant's

pigmentation, used by plants for their vibrant colours that attract insects to increase pollination.

Since the 1950's, research has indicated that phytochemicals have an ability to influence individual human health, and functioning. Thus, accordingly, phytochemical-rich foods have captivated researchers attention. Phytochemicals have demonstrated abilities to produce antioxidant, and antiinflammatory activities within the brain (Shukitt-Hale et al., 2005). Evidence currently supports the hypothesis that phytochemical actions may be responsible for altering age-related deficits, through neuroprotective, and neuromodulatory actions (Shukitt-Hale et al., 2005; Sokolov et al., 2013). In vivo (living organisms) and in vitro (within the glass, cellular) studies, conducted on both human and animal models, indicate that flavonoid benefits on cognition are universal. Occurring in healthy populations as well as in-patient and older populations (Rendeiro et al., 2012).

For a while it was believed that the blood-brain barrier prevented changes instigated by peripheral metabolic events. For instance, meal consumption can affect hormonal balances within the central nervous system (CNS), following an influx of nutrients (Sokolov et al., 2013; Spencer et al., 2012). Research has since indicated that the blood-brain barrier is selectively permeable to a wide range of biologically active substances, such as phytochemicals (Spencer et al., 2012). Identification of phytochemical compounds in brain tissue of rats following a single dose of phytochemical rich extracts, provides direct evidence that these substances can enter the brain immediately after consumption (Papandreou et al., 2009; Rendeiro et al., 2012; Spencer et al., 2012)

Consensus from previous literature is that phytochemicals have an ability to exert a direct positive impact on neuromodulation, and neuroprotection (Nehlig, 2003; Sokolov et al., 2013; Vauzour, Vafeiadou, Rodriguez-Mateos, Rendeiro, & Spencer, 2008). For instance, Andres-Lacueva et al. (2005) identified a correlation between the amount of flavonoids administered, and the significance of neuroenhancing effects in blueberry-fed rats. Specifically, as concentrations of phytochemicals increased in the cortex, memory improvements were also observable. Ross, Winter, and Linseman (2013, pp.279-309) therefore proposed that consumed phytochemicals are rapidly

transported, metabolised, and can be detected within minutes of a single acute dose (Matsumoto et al., 2001; Netzel, Strass, Janssen, Bitsch, & Bitsch, 2001; Passamonti, Vrhovsek, Vanzo, & Mattivi, 2005; Pojer et al., 2013).

The theoretical mechanisms responsible for phytochemical absorption are considered to be similar to the absorption mechanisms utilised in the stomach, and gastrointestinal tract (GI) tract (Youdim, Qaiser, Begley, Rice-Evans, & Abbot, 2004). Once absorbed the polyphenolic cationic structure of phytochemicals stimulate an array of cellular responses. Increased neuronal signalling, exerted by flavonoid concentrations, directly influences behavioural variations (Youdim et al., 2004). In a Morris water maze task, age-related decrements to rodent spatial memory was reversed following blueberry supplementation, over an eight-week period. It was theorised that the mechanism for this enhancement stemmed from the blueberry extract increasing the formation of new neurons throughout different areas in the brain, off-setting the loss that normally accompanies aging (Andres-Lacuava et al., 2005; Youdim et al., 2004). In short, the effects of phytochemicals on behavioural performance are mediated by their ability to provide neuroprotection to vulnerable neurons, enhance cell signalling, increase neurogenesis, and assist blood flow within the brain (Spencer, 2010).

Molecular mechanisms underlying phytochemical influence

There are still gaps in the literature regarding the mechanisms through which water-soluble phytochemical molecules are able to freely cross the blood-brain barrier (BBB). In future, its essential that clarification is attained regarding the correlating sites of action, within the mammalian brain, and how these neurobiological actions induce neuroprotection, following phytochemical compounds cross over through the BBB. Animal model evidence of phytochemical absorption has provided a rudimentary understanding of how flavonoids are able to exert positive effects on health, and performance (Kalt et al., 2008; Passamonti et al., 2005; Pojer et al., 2013). As discussed earlier, animal models have demonstrated rapid absorption of phytochemicals following ingestion. Detection in the blood stream occurs within 6-20 mins, with maximum blood saturation levels reached within 6-60 mins (Pojer et al., 2013). For example, Kalt et al. (2008) fed pigs a diet containing 4% blueberries for

four weeks. The authors' reported that phytochemical compounds had accumulated within the tissues. Specifically, phytochemicals accumulated in the cortex, reaching 0.878 pmol g⁻¹, and 0.664 pmol g⁻¹, furthermore, was also found present in the cerebellum.

Similarly, Passamonti et al. (2005) reported phytochemical concentrations of 192ng/g within the brain, following ten mins of a pure phytochemical extract being introduced into the stomach of rodents. Talavéra et al. (2003) observed phytochemical absorption transpiring in the stomach of rodents following ingestion of a phytochemical-enriched diet for 15 days. This location of phytochemical absorption is of particular interest as it is unusual for nutrients to be directly absorbed through stomach mucosa. A thorough knowledge of the absorption mechanisms from phytochemical rich foods is important for future studies. An illumination of the critical absorption mechanisms will allow these future studies to confidently test phytochemicals actions at the cellular, and molecular level. That in turn, will shed a light on the phytochemical influenced neuroprotective and neuromodulatory effects.

These neurobiological actions, from phytochemical consumption, are speculated to occur via three processes, modulating signal transduction, an increase in peripheral, and cerebral vascular blood flow, and further, scavenging neurotoxic species within the brain. Phytochemicals will interact with cellular signalling pathways, modulating signal transduction (Goyarzu et al., 2004). This modulation of the signalling transduction pathway will increase protein synthesis in order to maintain long-term potentiation (LTP) (Sokolov et al., 2013). LTP is the process that facilitates formation of long-term memories (Sokolov et al., 2013).

Further, phytochemical compounds may impact cognition through inducing increased peripheral, and cerebral vascular blood flow, that may lead to an induction of angiogenesis of nerve cell growth (Zafra-Stone et al., 2007). Evidential support for increased cerebral blood flow was shown in human participants up to one to two hours post phytochemical rich food consumption (Spencer, 2010; Zafra-Stone et al., 2007). As an example, an acute intervention with a cocoa drink, containing 400-900 mg of phytochemicals, resulted in an acute increase in blood flow within the brain. Additionally, while completing a task-switching test, blood oxygen levels in

participants, that consumed an acute dose of 450 mg of cocoa, resulted in an increase in blood oxygen levels during response, in comparison to the control group (Sokolov et al., 2013).

Moreover, phytochemical molecules will directly react, and scavenge neurotoxic species, and pro inflammatory species, that are produced through normal and abnormal aging (Spencer, 2010). Williams et al. (2008) reported that aged rats demonstrated improvements in spatial working memory following a phytochemical-rich diet. The authors' attributed these changes to activation of cAMP-response-element binding protein (CREB), and brain derived neurotrophic factor (BDNF), through enhanced extracellular signalling in the hippocampus. Williams et al. (2008) proposed that phytochemicals would promote the release of BDNF, responsible for neurogenesis, and synaptic growth within the CNS. Particular importance is the possibility that phytochemical interaction with BDNF, may provide positive health promoting possibilities, especially for individuals suffering from, or susceptible of acquiring cognitive degeneration.

Potential Public Health Benefits from Phytochemicals.

Numerous studies have alluded to the multiplicity of benefits flavonoids exert on human functioning (MacReady et al., 2010; Shukitt-Hale et al., 2005; Youdim et al., 2004). For an individual, increased dietary habits, which include high levels of phytochemical rich foods, may result in improved protection against a number of age related neurological, and metabolic diseases. Prior literature has clearly established that these water-soluble pigments possess a unique ability to cross the BBB. Which in turn, provides a unique opportunity for the treatment of neurological disorders, as the BBB selectively limits absorption into the central nervous system, which in turn, hinders treatment for neurological disorders. Therefore, possible naturally accessible treatments that potentially retard the development of neurological diseases, thus, improving cognitive functioning, and evolution are valuable. Not only at an individual level but also for the global population (Gómez-Pinilla, 2008).

Phytochemical compounds possess antioxidant, antidiabetic, antiobesity, and anticarcinogenic properties, in addition to cardiovascular and neuroprotective

properties (Cooke, Steward, Andreas, Gescher, & Marczylo, 2005; He & Gisutsi, 2010; Mazza, 2007; Pojer et al., 2013; Prior & Wu, 2006; Thomaset et al., 2009; Tsuda, 2012; Wang & Stoner, 2008). Antioxidants are paramount as a neuroprotective barrier against oxidative stress, and subsequent damage to the brain (Floyd & Hensley, 2002). In spite of this, the brain is not highly enriched in antioxidant protective defences. Therefore, an emphasis on dietary intake of antioxidant-rich foods, will strongly influencing the incidence, and onset of oxidative stress, as well as neurodegenerative disorders (Floyd & Hensley, 2002; Spencer, 2010).

Oxidative stress can be defined as an imbalance of the cellular antioxidant system, leading to over production of reactive oxygen species (ROS) (Shukitt-Hale et al., 2005). Oxidative stress and subsequent ROS damage is linked to cell death, and glial cell activation associated with normal aging as well as neurodegenerative disease development (Papandreou et al., 2009). Indeed, oxidative stress is caused by ROS, which is a by-product of normal aerobic metabolism, occurring at all times. However, accumulation of ROS within the brain, through a decreased capacity to neutralize free radicals, will result in oxidative damage to the brain.

Literature has demonstrated that even at low concentrations, phytochemicals in the body can induce neuroprotective actions. The effect phytochemicals have within the individual brain is pivotal for enhanced neuroprotection. For instance, animal model research reveals promising benefits for Parkinson's disease. Briefly, Parkinson's is caused through reduced dopamine neurons, resulting from oxidative damage accumulation. However, boysenberry and blackcurrant phytochemicals were observed to protect dopamine neurons from oxidative induced damage (Ghosh & Konishi, 2007; Gómez-Pinilla, 2008; Kim et al., 2010). Indicative, that the antioxidant properties, within phytochemical compounds, will exert neuroprotective effects (Youdim et al., 2004).

In 2001, Tedesco et al.'s *in vitro* study of H₂O₂-treated human erythrocytes, as a representative of an oxidative model, discovered that red wine, recognised as abundantly rich in phytochemicals, significantly lowered ROS production within human red blood cells. A later study on rodents found that 3.2 mg of phytochemical extract decreased DNA damage, to both the hippocampal, and cortical tissues. Studies

such as these supports the possibility that phytochemical compounds provide a protective effect from oxidative stress, neurotoxins, and neuroinflammation induced neuronal injury caused from a build-up of ROS (Barros et al., 2006; Spencer et al., 2011).

Eating is basic human routine; there is abundant evidence that the foods consumed will have a carry-on effect for individual energy metabolism, as well as neuronal communication. The human brain will consume an immense amount of energy in relation to the rest of the body. As it has already been demonstrated, transfer of energy from foods to neurons is important for the control of brain functions (Gómez-Pinilla, 2008). Environmental factors are a strong predictor of obesity and type 2 diabetes developments. Obesity and diabetes are metabolic diseases caused by insulin resistance (Tsuda, Horio, Uchida, Aoki, & Osawa, 2003). However, *in vitro* and *in vivo* (animal model studies) studies indicate that phytochemical derived antioxidants may actually ameliorate insulin resistance and fatty tissue accumulation within metabolic disorders (Tsuda et al., 2003).

Diabetes is compounded by the development of a metabolic disorder that will cause a severe reduction in adiponectin molecule development. These molecules are necessary for glucose regulation and fatty acid breakdown (Pojer et al., 2013). Recently, phytochemical dietary constituents were recognised as possessing a mechanisms that can help to regulate blood glucose levels, and induce insulin production through pancreatic B cells. These phytochemical compounds will directly interact with adiponectin molecules, reducing dysfunctions within the development of adiponectin molecules, helping to reduce the environmental factors of development of diabetes (Pojer et al., 2013).

Furthermore, studies have demonstrated that phytochemicals help to stimulate insulin secretion within pancreatic B cells (Prior & Wu, 2006). In rodent clinical trials Tsuda, Horio, & Osawa (2002) fed purified blueberry extract for a period of eight weeks. They reported that supplementation resulted in lower body fat weight gain when compared against controls. Western diets are recognised as containing high levels of fat. That subsequently, has been linked with the development of hyperglycemia, and hyperinsulinemia (Tsuda et al., 2003). However, *in vivo* modelled studies have shown

that hyperglycemia, and hyperinsulinemia can be reduced. Specifically, a rodent study revealed that dietary intake of purple corn compounds over 12 weeks may help to ameliorate high fat induced insulin resistance (Prior & Wu, 2006). Thus phytochemical compounds possess an ability to protect pancreatic *B* cells and insulin secretion - beneficial to human health.

Although presently there has been no extensive *in vivo* or clinical studies validating these proposed antidiabetic, and antiobesity observations (Ghosh & Konishi, 2007), other trials have demonstrated that different compositions of phytochemical extract will respond differently to body fat and insulin resistance. For instance, whole blueberries and black raspberries phytochemical extracts did not result in decreased levels of body fat or overall weight. Instead, the blueberries contributed to increased obesity levels, whereas black raspberries neither prevented, nor contributed to body fat accumulation in rodents (Prior et al., 2010).

Such discrepancies within prior literature have been linked to the different experimental conditions applied. For example, the percentage of fat administered in a high fat diet study will change the exchange of energy – from foods to neurons – in these high fat diets. Future studies should aim to establish a consensus regarding prescribed percentage of fat within clinical trials high fat diets. Additionally, the dosage of the particular phytochemical rich extract will also cause divergent results. Research that can establish the most effective dosage of phytochemical extract for antiobesity and antidiabetic effects, would improve future techniques to combat these degenerative effects. Treatments that improve insulin resistance will help to prevent type 2 diabetes and suppress obesity development (Ghosh & Konishi, 2007; Gómez-Pinilla, 2008; Pojer et al., 2013; Vendemiale, Grattagliano, & Altomare, 1999). This area of research provides a promising avenue for the treatment of metabolic disorders, especially for a world where obesity has become an epidemic worldwide phenomenon.

Contemporary drug therapies used in the treatment of inflammation, are also associated with the development and advancement of cancers (Coussens & Werb, 2002). Currently, observational evidence has reportedly hinted towards a possibility that phytochemical compounds can exert pharmacological effects. Specifically,

phytochemicals have been observed as a cancer chemo-preventive agent in animal and human models (Cooke et al., 2005; Thomasset, Teller et al., 2009; Thomasset et al., 2009; Wang & Stoner, 2009). Preclinical evidence suggests that phytochemical extracts can delay or revoke carcinogenesis in rodents. Evidence of phytochemical efficacy for carcinogenesis cells is most convincing for gastrointestinal cells. For example, *Apc^{min}* mice carry a gene mutation that causes the development of adenoma tumours in the intestinal tract. A concentrated dose of blueberry, approximately 3.0 mg/ day, moderately reduced the production of small intestinal adenomas (Cooke et al., 2005).

In human malignant tissues, phytochemicals can exert growth-inhibitory effects on cancer cells. For example, phytochemical extracts of grapes, bilberries or chokeberries, at doses of 25-75 mg/ml, inhibited the growth of human malignant colon cancer cells. It was noted, however, there was no effect on non-malignant colon cells (Cooke et al., 2005). It can be inferred, therefore, that phytochemical compounds are able to survive biophase conditions for an acceptable period of time, playing a key role in anticarcinogenic mechanisms that is consistent with chemoprevention. Hence, phytochemical chemo-preventive activity, observed in pre-clinical trials, strongly advocates for further study with these compounds (Thomasset et al., 2009).

Unfortunately, overall, human epidemiological work has produced inconsistent results regarding phytochemicals and their anti-carcinogenic effects; more research is required in order to provide compelling evidence for such anti-carcinogenic effects on humans.

In summary, although the current literature tentatively demonstrates a beneficial relationship between phytochemical consumption and a multitude of health-promoting actions, further empirical work is required. Specifically, it is vitally important to clarify how the mechanisms of action work when phytochemical compounds exert their neuroprotective, antioxidant, and antiinflammatory processes. Phytochemical rich foods have a potential as a pharmacologic treatment for cancer cells, and preventative effects for the development of metabolic and neurodegenerative diseases. The claim that intake of phytochemical compounds, heighten neurological signalling, and communication appears to be true, as previous literature have alluded that phytochemical enriched foods enhance cognitive

functioning in patient populations. This has sparked increased interest in investigating the role phytochemical consumption has on cognitive functioning within healthy populations. A summative discussion of the literature in this area to date is provided in the following section.

Phytochemical rich food: benefits to cognitive performance

Research surrounding dietary constituents rich in phytochemical compounds reveal positive influences from both acute and chronic consumption of phytochemical rich foods (Desideri et al., 2012; Lamport et al., 2012; Kesse-Guyog et al., 2012; Nurk et al., 2008; Lettenneur, Proust-Lima, Dartigues, & Barberger-Gateau, 2007). As detailed earlier, the majority of prior evidence has been focused on subsequent second order effects following either acute or chronic phytochemical rich ingestion in elderly and patient populations.

A longitudinal study by Lettenneur et al. (2007) assessed phytochemical intake and cognitive decline over a ten-year period. The authors' revealed high levels of phytochemical intake was associated with a better evolution of cognitive functioning. This report was made in comparison to those who reported dietary habits containing low intakes of phytochemicals. For instance, a comparative longitudinal study, involving over 2000 elderly persons, revealed that habitual intakes of phytochemical-rich foods such as tea, chocolate, and wine correlated with higher cognitive functioning. Specifically, participants' who reported consuming all three phytochemical-rich food items had the highest mean test scores in a variety of cognitive tasks. Furthermore, it was observed those participants' had a lower prevalence of poor cognitive performance in comparison to participants' that reported no chocolate, tea, or wine consumption.

Because of the protective actions put forth from red wine consumption, often referred to as the 'French paradox', in reference to the low incidence of cardiovascular disease observed in the French population despite the standard French dietary patterns being high in fat consumption (Cooke et al., 2008; Nurk et al., 2008). It has been reported that drinking wine in moderation may potentially lower the risk for developing Alzheimer's or dementia (Nurk et al., 2008). Its noteworthy that literature has

reported that moderate intake of wine provides greater protection effects against cognitive impairment than non-drinkers. These protective actions are attributed to wine's anti-inflammatory, antioxidant and a reduction in cardiovascular risk.

Epidemiological studies have primarily reported positive effects from phytochemical intake on cognitive functioning (Desideri et al., 2012; Lambourne & Tomporowski, 2010). However, in spite of these epidemiological reports, the principal interpretation of a beneficial association between flavonoid intake and cognition should be cautiously approached. Continued research is required in order to understand the specific biological actions induced by flavonoids on specific cognitive domains for young and healthy populations (see Chang, Labban, Gapin, & Etnier, 2012; Etnier & Chang, 2009; Tomporowski, 2003; McMorris & Hale, 2012, for reviews). Presently, the existing evidence, reporting favourable associations between phytochemical compounds and cognitive functioning, are for a specific source of phytochemical compounds, or a precise mixture of phytochemical compounds (Kesse-Guyot et al., 2011).

For instance, acute studies investigating phytochemicals found in cocoa has revealed enhanced performances on cognitive measures. For instance, Scholey et al. (2009) revealed benefits to working memory and attention following cocoa consumption. Furthermore, Field, Williams, & Butler (2011) also demonstrated improvements on cognitive measures. Specifically, improvements on a spatial working memory task were demonstrated 120 mins after consumption of 773 mg of dark chocolate phytochemicals. This improvement was observed in comparison to the control group that consumed white chocolate. Thus, cocoa, or dark chocolate consumption appears to generate maximum benefits to cognitive functioning. In particular when participants' mean intake of cocoa phytochemicals is concentrated at 10 g (Nurk et al., 2008).

Further reports of cognitive benefits from cocoa phytochemical consumption has been reported, for instance improvements were observed in participants' working memory and attention, 90-150 mins after consumption of a beverage containing between 520 mg and 994 mg of cocoa flavonols. In comparison to controls' whom were administered only 46 mg of cocoa flavonols (Scholey et al., 2009). Also, Camfield et

al. (2011) administered two different types of cocoa beverages and then examined their effects on participants' brain activity via an EEG. The authors' observed that "average amplitude and phases of evoked potentials within brain sites significantly differed between the groups during memory encoding and memory retrieval" (Camfield et al., 2012, p.953). Thus, the authors' concluded that the consumption of cocoa phytochemicals was associated with enhanced spatial working memory (Camfield et al., 2012).

In contrast to the evidence reported above, an intervention study on younger adults consumption of a chocolate drink over a five-day period revealed no enhancements on cognitive functioning. In sum, the evidence obtained from cocoa phytochemical research extrapolates that acute doses of cocoa phytochemicals might have beneficial effects on working memory and attention. The conflicting evidence across the studies by Francis, Head, Morris, & Macdonald (2006), Camfield et al. (2012), Nurk et al. (2008), and Scholey et al. (2009) could be attributed to the sources of cocoa phytochemicals that were used within these studies. Not all brands of chocolate or cocoa beverages will contain the same levels of cocoa phytochemicals, leading to differing results among the literature in terms of benefits, or lack thereof, to cognitive functioning.

Research surrounding cognitive enhancement abilities from natural food sources is primarily directed towards efforts to find and develop cognitive enhancing drugs in order to treat age related cognitive decline. Further, owing to the fast evolving technological world of today, requiring individual's to maintain optimal cognitive performance levels 24 hours, 7 days a week, researchers are also directing energies towards identifying a super nutrient that will provide enhanced cognitive ability during physically demanding and aversive circumstances. For instance, its widely accepted that improved performance on cognitive tasks frequently occur following caffeine intake (Lieberman et al., 1987). Caffeine is a widely consumed source of phytochemicals, and long been associated with positive effects on cognition and psychomotor functioning. Especially, for example, when people are functioning under a deficit such as fatigue (Hogervorst, Riedel, Jeukendrup, & Jolles, 2008). Observations of enhanced cognitive performance are commonly described in terms of increased vigilance, arousal, psychomotor speed and alertness (Riedel et al., 1995). In

addition, studies have also associated tea with neuroprotective effects (Ng, Feng, Niti, Kua & Yap, 2008). As tealeaves are not only comprised from tea phytochemicals but, also contain components of caffeine phytochemicals (Ng et al., 2008).

However, while evidence suggests that an acute caffeinated beverage will increase cerebral consumption of glucose, the major energy source for the brain, the majority neuroprotective effects from caffeine on cognitive performance are likely to be demonstrated after long-term consumption, at least 2-6 weeks of free access to caffeine (Rosso, Mossey, & Lippa, 2008). Notably, caffeine studies have predicted that performance enhancements are likely to occur within the cognitive domains predominantly associated with age related deficits (Rosso et al., 2008). Furthermore, evidence has revealed that caffeine phytochemicals will exert cognitive benefits in a dose-dependent manner. Thus, a low concentration of caffeine will not consistently produce enhanced cognitive functioning, whereas a high dose will reliably act as a positive neuromodulator of cognitive functioning (Adan & Serra-Grabulosa, 2010). Presently, the evidence suggests that regular consumption of tea and/or coffee will provide neuroprotective effects, lowering the risks of cognitive impairment and decline (Ng et al., 2008). Further studies are needed to confirm this association and investigate the mechanisms through which caffeine and tea phytochemicals affect cognitive decline (Russo et al., 2008).

In summary, longitudinal studies investigating populations of older men and woman reveal greater cognitive evolution with habitual intake of phytochemical-rich foods (Devore, Kang, Breteler, & Grodstein, 2012; Kesse-Guyot et al., 2011; Nurk et al., 2008). While, studies investigating the effects of acute doses of phytochemical compounds involving healthy, young populations' reveal inconsistent patterns of their predicted beneficial effect, (Henrickson & Matte, 2010; Francis, Head, Morris, & Macdonald, 2006), systematic reviews of the literature have observed that berry fruit phytochemical compounds provide evidence of improved cognitive functioning (Lamport et al., 2012). The results of these studies will be discussed in the following section.

Berry fruit phytochemical and cognitive improvement

The initial studies involving berry fruit phytochemicals was to investigate their potential to act as a therapeutic agent against oxidative stress (Joseph, Shukitt-Hale, & Fisher, 2005; Willis, Shukitt-Hale, & Hale, 2009). It's been predicted that berry fruit, high in antioxidant properties, would enhance the body's natural defensive system against metabolic-induced injuries (Willis et al., 2009). However, reports have observed that berry fruit phytochemicals possess additional health benefiting actions. The majority of these demonstrated biological actions exerted from phytochemicals have primarily been evident within animal model studies (Joesph, Shukitt-Hale, & Lau 2007, Shukitt-Hale, Carey, Jenkins, Rabin, & Joseph, 2007; Spencer, Vafeiadou, Williams, & Vauzour, 2011).

Cognitive functioning in animal model studies are primarily assessed through performance on the Morris Water Maze task. Animals that are exposed to inflammatory and oxidative stressors will manifest behaviours, in animals and humans, which closely resemble behavioural and cognitive impairments seen with aging. Joesph et al. (2003) investigated the role of blueberry supplements on APP/PSI transgenic mice. This breed of mice possesses a genetically mutated gene that causes the production of B amyloid to be increased in the brain, leading to the development of Alzheimer's disease. Blueberry supplements were began at four months old and continued till the mice were 12 months old. At this point they were tested on the Y-maze, revealing performances similar to non-transgenic mice. Thus the authors' demonstrated that blueberry supplemented mice significantly outperformed the control group which were not supplemented. Such enhanced performance was attributed to the higher concentrations of hippocampal protein kinase C observed in these blueberry-fed mice. This protein is important for development and conversion of short-term memories to long-term memory in the brain.

In a later study, Shukitt-Hale et al. (2008) administered kainic acid to young rodents in order to induce an inflammatory response that causes neurodegeneration. The damaging effect from this treatment was observed from rodents' decreased performance on all cognitive measures. However, rodents that were fed a diet of 2%

blueberry, two months prior to kainic acid administration to the hippocampal region, demonstrated significantly reduced levels of impairment on a spatial memory task, in comparison to controls. Such findings support the hypothesis that berry fruit supplementation may act as a neuroprotective agent against age-related neurodegenerative diseases.

An important question that needs answering however is whether the benefits of a berry diet evident in rodent studies can be extended to humans. Presently, human studies have provided diverging reports regarding the potential beneficial relationship between phytochemical consumption and retardation of neurodegeneration (Joseph et al., 2005; Krikorian et al., 2012). Neuroinflammation is a biological response to vascular insults and can be associated with age-related decrements in cognitive functioning (Joseph et al., 2005; Krikorian, Nash, Shidler, Shukitt-Hale, & Joseph, 2010; Rendeiro et al., 2012). As the brain ages, its capacity to regulate inflammation is progressively diminished. Dietary patterns high in saturated fats, or subsequent deficiencies of fruit and vegetable micronutrient intake may cause an acceleration of this inflammatory process to occur. Therefore, individuals exhibiting a diminished capacity to regulate neuroinflammation is characteristically associated with the onset of neurodegenerative diseases. Thus, research that has demonstrated that berry fruit-supplemented diets have an ability to promote retardation of cognitive deficits that accompany age related neurodegeneration poses great importance for future generations evolution. Provides promising avenues for the treatment of these neurological diseases (Joseph et al., 2005; Krikorian et al., 2010; Krikorian et al., 2012). The current epidemiological studies in this field have indicated that increased berry fruit phytochemical consumption is associated with lowered onset and progression of degenerative neurological risk. Authors, Casadesus et al. (2004), Devore et al. (2012), Lamport et al. (2012), Krikorian et al. (2010), (2012), Wallace & Giusti (2013), attributed the lowered risk following observations of improved cognitive performance in elderly and rodent populations exhibiting mild cognitive impairments.

For example, older adults with mild cognitive impairment were provided with a Concord grape juice supplement for 16 weeks. With improvements to recognition memory being observed. Specifically, participants' in the Concord grape juice cohort

demonstrated better discrimination ability; correctly selecting previously learned material over interfering (not previously learned) material (Krikorian et al., 2012). In an earlier study with Concord juice and older adults that exhibited memory declines, observed significant improvements to verbal learning, and a non-significant improvement to spatial recall (Kirkorian et al., 2010). These improvements were achieved following 12-week supplementation with Concord juice. Dosage was determined by body weight and ranged between 6-9 ml/kg. In sum, the current evidence suggests that phytochemicals will provide health-benefiting effects that may in turn provide an alternative therapeutic agent upon age related impairments to memory and cognition in humans. Despite the positive evidence, a cautionary note must be made; both of the above studies had a small number of participants. Thus, generalisations to the wider population should not be made from such small sample sized studies. Future research needs to utilise larger sample pools where it's possible in order to gain conclusive evidence. However, while prior literature is only able to detect small effects in this area. Such possibilities promote the importance of fruit juice and berry supplements in daily dietary intake.

In spite of the promising foundational research relating to an association between berry fruit flavonoids and improved cognitive performance, caution is required. This is evidenced by studies such as that of Lamport et al. (2012) whose meta-review reported that 16 published studies revealed a significant positive association between flavonoid consumption and cognitive functioning, but contrastingly, 12 published studies reported a negative association. Methodological differences among the studies may have attributed to the conflicting results.

Methodological differences in phytochemical research

The conflicting reports between studies are common in research relating to nutrient intake and cognition. Part of the problem is that while cognitive tests are a prominent way to evaluate cognitive performance, very few human studies in this area of research have used the same set(s) of cognitive test(s) even when employing similar study regimes (MacReady et al., 2010). Furthermore, checks are rarely made to see if, for example, two different measures of working memory correlate with one another. Literature reviews counted the use of 80 different cognitive tasks, employed across 28

different studies (Lamport et al., 2008). It is highly unlikely that all 80 tests have the same level of sensitivity to their predicted cognitive domain. In future, research should consider developing a widely agreed upon bank of cognitive tests that can be used in all phytochemical extract studies. Only then can it be assured that the type of test chosen is not confounding the outcome of a study. Development of such a test bank would provide research in this area with more powerful and valid results (Hoyland, Lawton, & Dye, 2008). Hoyland et al. (2008) found that flavonoid intervention studies produce the strongest improvements with cognitive tasks that impose high levels of cognitive demand. Thus, the lack of consensus among previous studies may actually result from a lack of sensitivity from a cognitive task to detect an effect from the intervention extract, not a true absence of an effect (MacReady et al., 2010).

Summary

The benefits achieved through nutritional supplements, as cited in human health and performance research, has resulted in an increased awareness on the importance of dietary nutrients and their subsequent effects on individual health and performance. In particular, this chapter discussed the observed effects of phytochemical supplementation. Which has been linked to enhancing health and performance in animals and humans. For instance, promoting anticarcinogenic, antioxidant, and antiinflammatory actions. As well as resulting in improved cognitive functioning. While this area of research continues to grow, a dominant focus of future studies should be directed towards clarifying the underlying mechanisms of action of flavonoids. The identification of the brain processes and structures involved should lend to a better understanding of the cognitive changes that occur. Moreover, the majority of literature has focused on potential enhancements in populations with initial stages of neurodegenerative diseases, for example, mild cognitive impairment. It is important to examine the role phytochemicals have on healthy populations in acute and chronic supplementation models.

CHAPTER 3 Exercise and COGNITIVE PERFORMANCE

Physical exercise and cognitive performance has long been of interest to researchers. The pioneering studies in this area began in 1978 by Spirduso & Clifford, which compared athletes and sedentary individuals on simple, choice, and movement time tasks. Performance on these tasks was substantially better for athletes than sedentary adults (Colcombe & Kramer, 2003). Later, cross sectional evidence (see Etnier et al., 1997; Griffin et al., 2011 for a review) observed that older adults who participated in lifelong cardiovascular exercise, exhibited greater brain preservation than age-matched counterparts (Colcombe et al., 2006). Hence, research regarding brain preservation through daily exercise regimes has important implications. Presently, with modern medicine, adults are surviving to advanced ages. For instance, the projected cost of caring for older adults is staggering. This cost will rise with the number of individuals who require specialised care for neurological disorders. Consequently, mechanisms that can be employed to offset, or reverse age related structural decline within the human brain is increasingly important.

While evidence in this area is resolute in the premise that through regular exercise activities benefits to cognitive performance are achievable, sporting environments, that tested athletes and non-athletes cognitive abilities observed that cognitive benefit potential demonstrates quadratic trends (Chang & Etnier, 2009). Specifically, the majority of the literature reviewed concluded that moderate intensity exercise for 30 to 60 mins are likely to have a positive effect on cognitive performance (Chang et al., 2012; Lambourne & Tomporowski, 2010; McMorris & Hale, 2012; Tomporowski, 2003; Smith et al., 2010). Upon review, its conceivable that this observed quadratic trend is due to an association between exercise behaviour and energy metabolism (Vaynman & Gómez-Pinilla, 2006). Indicating that regular arousal of the motor system will help to facilitate molecular systems that are important for learning, memory, and synaptic plasticity (Vaynman & Gómez-Pinilla, 2006). Although, technological advancement in today's world has led to industrialised society's exhibiting sedentary lifestyles. But, this technological advancement has not reduced the fast moving pace of life, nor has it lead to an evened out work-life balance. More than ever individuals are expected to be able to meet ever increasing and demanding

productivity outputs. Whereby, regular exercise behaviours are impracticable to manage.

The impact of these demanding environments, coupled with lower physical activity levels worldwide, has had little empirical attention. It must be said that this is an important area of research, if only due to evidence to date suggestive that strenuous activity will cause biochemical changes in the body, causing interference in cerebral cortex functioning (Hebb, 1955). Thus, a primary focus of research into the exercise-cognition relationship should be directed towards understanding the extent of cognitive deficits that is caused by aversive circumstances. Further this research should be extended to investigate potential solutions that forestall any induced deficits. One approach to accomplishing this task is through nutrition and its relationship with exercise-cognition relationship (Chang et al., 2012; Ekkekakis, 2009; Etiner & Chang, 2009).

There has been an abundance of research investigating the effect of physical exercise on cognitive functioning (see Chang et al., 2012; Etnier et al., 1997; Lambourne & Tomporowski, 2010; McMorris & Hale, 2012; Tomporowski, 2003; Tomporowski & Ellis, 1986, for reviews). Once again, the majority of these reviews lack clarity regarding the exercise-cognition relationship. With many of the inconsistent results reviewed arising from methodological differences in the exercising regime applied and the various types of cognitive assessments employed (Hopkins, Davis, Vantighem, Whalen, & Bucci, 2012). The exercise-cognition interaction is considered to improve and peak, at particular optimal levels of arousal, specific for each individual's fitness level before returning rapidly to performance levels similar to or below baseline levels. Nonetheless, its repeatedly observed that certain domains of cognition are more sensitive than others to physiological arousal, for instance reaction time tasks.

Owing to the wide methodological approaches in this field of research, meta-analytic reviews have classed these types of studies into one of four categories. Firstly, studies that find a beneficial relationship, secondly, studies that find a detrimental relationship, thirdly, those that find both beneficial and detrimental relationships, and finally, studies that reveal no relationship (Tomporowski & Ellis, 1986). Therefore,

comparison across studies can be difficult, thus, the effects of increased physiological arousal from exercise upon cognitive performance remain unclear (Bisswaller, Collardeou, & Réne, 2002).

Nonetheless, these contradictory findings have provided some important insights into the exercise-cognition relationship. For instance, it may be said that such diverging relationships exist due to differing physiological demands applied; further, cardiovascular demands may differently affect various cognitive domains. Additionally, musculoskeletal demands impose different effects to cognitive domains, thus, differing musculoskeletal demands, imposed by different exercise modalities, could have a range of effects on cognitive functioning (Pontifex et al., 2009). For instance, aerobic exercise while reportedly producing evidence of a positive, negative, or no relationship between exercise and cognitive ability, is regularly recognised as producing facilitative effects on cognitive performance (Birsswaller et al., 2002; Lambourne & Tomporowski, 2010; Pontifex, Hillman, & Polich, 2009; McMorris et al., 2011; Tomporowski, 2003). Such a positive relationship is inconclusive regarding resistance exercise regimes and subsequent effects on cognitive functioning. Resistance exercise modalities employ a muscle straining exercise, but do not contain a high cardiovascular output.

For example, Pontifex et al. (2009) investigated the differences between aerobic and resistance exercise programmes on working memory performance. The working memory task was performed immediately following exercise and again 30 mins after exercise cessation. While reaction time (RT) was observed to be shorter after completing the acute bout of aerobic exercise, similar effects were non-existent for the resistance exercise group, RT relative to baseline and control scores did not differ (Pontifex et al., 2009). This example serves to illustrate that different exercise modalities may have quite different effects on cognitive performance. Therefore, when comparing research findings across studies, the type of exercise modality utilised in a study must be taken into consideration.

Exercise modality is not the only major methodological difference within exercise-cognition studies. Aerobic exercise studies have also utilised differing exercise regimes to induce physiological arousal. For instance, Gutin and Di Gennaro (1968a;

b) employed two different exercise regimes; the earlier study employed a step up task to examine the effects of exercise on cognitive performance, while the latter used an exhaustive treadmill run. In contrast, Tomporowski et al. (2005) conducted two studies investigating the effects of short-term after-effects of aerobic exercise on cognitive function. In the first study, participants cycled at a steady rate for 40 mins. While the second study applied a graded maximal cycling exercise, for up to 120 mins or until subjects reached volitional exhaustion. Likewise, Griffen et al. (2009) used a graded cycling exercise procedure to investigate the effects of an acute bout and chronic, five weeks, of an intensive exercise program on cognitive functioning. The arbitrary use of the various exercise regimes' employed produces a complex picture of the exercise-cognition relationship. Future research needs to develop a comprehensive understanding of the relationship between particular exercise regimes' and subsequent cognitive changes. What is clearly apparent in this field of literature is that there is a fine line between exercise regimes producing a facilitating, debilitating, or no effect on cognitive performance (Arent, 2003). For instance, Gutin (1973) demonstrated that exercise bouts for 45 sec to 2 mins, that raised HR levels to 90-120 bpm, produced a positive effect on cognitive performance. However, exercise lasting 6 mins, increasing HR to 150 bpm, resulted in performance levels similar to baseline.

One possible reason for the complexity surrounding the exercise-cognition relationship is from cognitive task selection among studies. Therefore, understanding how exercises protocols and cognitive task selection interacts, leading to inducing either a positive, negative, or curvilinear outcome remains a key challenge within this area of research (MacReady et al., 2010; Pontifex et al., 2009). The characteristic of exercise-cognition interactions producing negative, positive, or curvilinear outcomes indicates that aerobic exercise will not facilitate all aspects of cognitive functioning. Literature reviews have demonstrated that cognitive task selection will moderate the outcomes achieved (Chang et al., 2012; Lambourne & Tomporowski, 2010). For instance, Chang et al. (2012) meta-analysis reported negative effects for the digit span backwards task, a measure of verbal working memory. However, the authors' reported a positive effect in studies examining choice reaction time measures.

Clarification regarding the exercise-cognition relationship may be achieved if the cognitive tasks administered were tasks that imposed variable demands across

different cognitive domains (Ponifex et al., 2009). Previously, it has been stated that future research would benefit from cognitive task selection based on a strong rationale, for instance, using the exact tests as used in similar studies, or test selection based on strong theoretical rationales (Brisswalter et al., 2002, Hogervorst et al., 1996; Lambourne & Tomporowski, 2010). For instance, investigative research has suggested larger benefits would be observable for tasks that require greater amounts of executive control, comparative to cognitive tasks that have smaller executive control requirements. Thus, utilising single cognitive tasks that measure multiple areas of executive control may provide greater relationship clarification.

There are a vast number of cognitive domains encompassing, reasoning, memory, language, and attention to name a few. In order to fully elucidate the effects of exercise on cognition, a wide range of tasks is required to fully assess cognitive ability (MacReady et al., 2010). However, expectation of a one-to-one correspondence between two cognitive tasks and the cognitive domain claimed to be measured is unwise (MacReady et al., 2010). Different tests, that measure the same cognitive attribute, are likely to produce different results. Thus, the inconclusive evidence in the exercise-cognition literature may be a result of a lack of correspondence between the different cognitive tasks selected and their level of sensitivity to a particular cognitive domain (MacReady et al., 2010).

Assembled together, the diverse exercise regimes employed, the plethora of cognitive tasks administered hinders any conclusive comparison being made. In addition, exercise-cognition studies have conducted exercise tasks at differing levels of intensity. Prior investigation into the mechanisms of effect has identified exercise intensity as an observable moderator within acute (one off) exercise studies (Chang et al., 2012). Primarily, exercise-cognition studies have employed an exercising task to one of the following three intensity levels: maximal intensity, submaximal intensity, and exercise in conjunction with hydration (Chang et al., 2012).

Exercise intensity is characterised as the imposed changes to the central nervous system coded by the speed of neuronal firing (Dietrich & Sparling, 2004). Submaximal intensity exercise tasks are considered to induce arousal of $\sim 60\%$ $\dot{V}O_2$ max, but do not increase blood lactate levels above the threshold e.g. blood lactate

level (above ~ 10 nmol/L). Maximal intensity exercise induces arousal to $\sim 80\%$ Vo_2 max, and causes elevated blood lactate levels (Draper, McMorris, & Parker, 2010). At present, there is no uniformed method of measuring exercise intensity induced effects (Tompsonowski & Ellis, 1986).

Currently, in order to achieve a particular exercise intensity during or following, an exercise regime researchers' are likely to measure participants' maximum heart rate (HR) or maximum oxygen uptake (Vo_2 max). Overall, the consensus is that physical activities performed within moderate intensity levels will produce positive effects on cognitive functioning. However, exercise tasks that are performed within maximal intensity levels will cause impairments to cognitive functioning (Dietrich & Sparling, 2004; Chang & Etnier, 2009; Lambourne & Tomporowski, 2010). In an attempt to explain the underlying mechanisms responsible for differing exercise intensity effects, two prominent models have been applied and are discussed next.

Theoretical Models of the Interaction in the Exercise-Cognition Relationship

The first model that endeavoured to link the underlying mechanisms of action on cognition following an acute exercise task is the Yerkes and Dodson's inverted-U theory (Yerkes & Dodson, 1908). This theory proposed that variations in arousal from different exercise intensities would cause an equivalent variation in attentional demands (Yerkes & Dodson, 1908). Therefore, exercise performed at moderate exercise intensity levels would induce significantly better performance effects on cognition. In comparison to any performance effects observed following completion of an exercise task applied at a high or low level of intensity (Lyons, Al-Nakeeb, & Nevill, 2006). Essentially, when graphed, the quality of performance effects on a cognitive task resembles an inverted-U shaped function of exercise intensity (see *Figure 1*) (Lyons et al., 2006; Yerkes & Dodson, 1908).

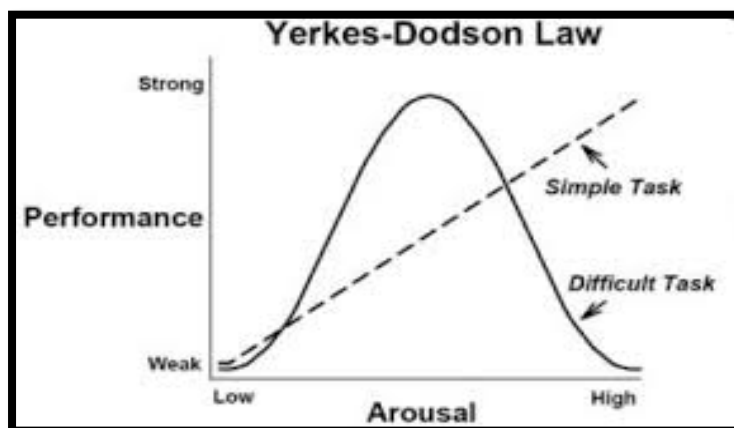


Figure 1: Yerks & Dodson's inverted-U hypothesis

Source: <http://www.intructional1.calstatela.edu/dfrankl/curr/kin380/pdf/Anxiety-Arousal-Ch4-6spp.pdf>

Later, Easterbrook (1959) developed the second prominent theoretical model, referred to as the cue utilisation theory. Easterbrook's (1959) cue utilisation theory was a development on the Yerkes-Dodson inverted-U hypothesis. Easterbrook (1959) developed his cue utilisation theory in an attempt to theorise why variations to exercise induced arousal would cause divergent performance effects. This theory proposed individuals concurrently attend to and respond to environmental cues within a given situation. Conjecturing that low aroused states would allow an individual to focus on, and respond to, both relevant and irrelevant cues. In turn revealing observable poor performance effects on cognitive measures (Lyons et al., 2006). However, as arousal level increased to moderate levels (submaximal), individual's attention would narrow focusing on task-relevant cues only. Consequently, facilitating optimal performance effects to be observed (Lyons et al., 2006). Yet, if an individual's arousal state increased past moderate arousal levels attention would begin to narrow. Increasing the risk that an individual would miss task relevant cues, revealing performance similar to performance measured in baseline scores (Easterbrook, 1959; Lyons et al., 2006).

These two exercise-cognition models are two of the most prominent, and frequently used rationales, underlying prior literature theoretical explanations of the divergent performance effects, which are observed from the different exercise intensities that have been applied. Overwhelmingly, the methodologies that are applied in exercise-cognition studies appear to be a critical aspect on predicted performance effects and

must be considered when analysing evidential data on the exercise-cognition relationship (Labelle et al., 2014).

Methodological differences among previous research

The methodological protocols that have been used to measure the exercise-cognition relationship have differed widely across prior studies'. Some studies' will employ a dual task design concurrently applying an exercise task whilst simultaneously testing cognition performance. The literature reviews of dual task studies' has demonstrated that this protocol is not moderated by exercise intensity, instead these protocols are moderated by the timing of the cognitive measurement,, significantly influencing performance effects (Chang et al., 2012). For instance, negligible effects were observed in the first 10 mins of exercise, but after exercising for 11 to 20 mins, negative effects were observed on cognitive performance (Chang et al., 2012). However, participants that exercised for 20 mins before cognitive task administration demonstrated a positive effect (Chang et al., 2012; Lambourne & Tomporowski, 2010).

Meanwhile, other studies' have employed a single task protocol, whereby cognitive performance was measured following an exercise task. Prior research demonstrates that these protocols reveal significant effects, but moderated by exercise intensity (Chang et al., 2012). Specifically, significant negative effects were observed in studies' that applied cognitive testing directly after and up to 10 mins following exercise cessation. Though, positive effects were observed when cognitive task(s) were administered following a delay of 11-20 mins. Any delays that is longer than 20 min, following exercise cessation and cognitive testing, have only demonstrated small positive effects (Chang et al., 2012).

While a discussion on the differing attentional demands that is required from participants' completing these different modalities is not warranted for this thesis. It's worth mentioning that attentional demands during dual task protocols compared to single task protocols may markedly differ. Consequently, comparison and interpretation of previously reported evidential data remains difficult (Lambourne & Tomporowski, 2010). In order to develop the database of knowledge on the exercise-

cognition relationship, clarity regarding the different exercise modalities and their effect on modifying cognitive performance effects needs to be achieved.

Moreover, there is poor understanding regarding the different attentional demands that is required from different exercise modalities on cognitive performance, and how these demands may contribute to the conflicting evidence in exercise-cognition studies. For example, two of the most commonly used exercise modalities are running, or cycling protocols. Both protocols induce different attentional demand effects for successful completion of the exercise task. During exercise the central nervous system is bombarded by stimuli from various cardiac, skeletal, and muscular muscles (Gutin, 2013). Thus, interference from muscular muscles, e.g. from a cycling task, may cause cognitive interference that differs from interference stemming from cardiac and respiratory muscles. Therefore, the variable attentional demands provoked by different exercise modalities is likely to contribute towards divergent evidential data (Brisswalter et al., 2002; Coles & Tomporowski, 2008; Chang et al., 2012; Kramer & Erickson 2007).

In summary, the overall evidence from meta-analytic studies indicates that acute bouts of exercise may produce beneficial or detrimental effects on cognitive performance during dual task or single task protocols (Chang et al., 2012; Lambourne & Tomporowski, 2010, Tomporowski, 2003). The characteristic of the exercise-cognition interaction revealed, e.g. positive, negative, or curvilinear, will be dependent on a number of moderating factors. Generally, prior evidence indicates that cognitive benefits are larger for protocols that apply a moderate intensity exercise protocol. Specifically, exercise tasks that increase an individual's physical arousal to an optimal, moderate level, will benefit cognitive performance. But, physiological arousal beyond an individual's optimal arousal level will result in cognitive deficits, as proposed by the two prominent theoretical models, the inverted-U hypothesis and the cue utilisation theory (Easterbrook, 1959; Yerkes & Dodson, 1908). These two models have been the founding theoretical basis for the underlying mechanisms of action of the exercise-cognition relationship. Further clarification of the conditions whereby, particular exercise protocols, cognitive tasks, and methodological applications will, or will not derive cognitive performance effects remains a key challenge for future research. In an attempt to clarify underlying causal factors is the

proposed hypothesis that central fatigue underlie the observed cognitive decline associated with maximal intensity exercise, and thus, will be discussed below (Féry, Ferry, Vom Hofe, & Rieu, 1997).

Exercise-induced fatigue & subsequent cognitive performance

Given the contradictory evidence regarding the exercise-cognition relationship, a case could be made for claiming that exercise intensity is not what produces the conflicting results. Rather, it may be that the exercise-induced fatigued states are the contributing factor in cognitive performance deficits in response to exercise-induced arousal (Alves et al., 2014). The current study considered that exercise-induced fatigue would be a contributing factor in inducing cognitive impairments. Thus, some discussion of the evidence regarding the exercise-induced fatigue effects on cognitive performance is warranted.

Human factor research has long been interested in the potential debilitating effects of exercise-induced fatigue on operational performance (Lambourne & Tomporowski, 2010). In order to expand this awareness further, its important to understand the implication exercise, exercise-induced fatigue, and overall performance effects has within many diverse environmental situations. Hence, identification of fatigue-induced changes to cognitive performance would help ascertain guidelines ensuring facilitation of cognitive functioning, in relation to exercise habits, instead of cognitive impairments (Labelle et al., 2014). Unfortunately, there is not an agreed upon definition of fatigue, instead it appears to be a multidimensional concept. One description, referred to fatigue as reduction in an individual's ability to generate muscle force or power, arising from prolonged periods of motor activity (Gandevia, 2001). Alternatively, Takuwa (1971) referred to fatigue as a combined output of mental activity and physiological functions.

Physical fatigue and its subsequent effect on cognitive performance have important practical implications, but presently conclusive evidence of these effects remains elusive. Generally, fatigue is considered to be the body's security system, indicating dangerous levels of resource depletion and in need of rest. Meanwhile, it's recognised that experiencing such high levels of fatigue will result in diminished capacity.

Nevertheless, it is not always feasible for an individual to stop and rest when experiencing fatigue e.g. defence personnel, and emergency services. Still, high levels of fatigue will not result in a complete and sudden drop in performance.

Characteristically, optimal performance phases will be interrupted with increasing frequency from lapses in task engagement (Nozaki et al., 2009; van der Linden, Frese, & Sonnentag, 2003). Physical fatigue is complex and likely to be the outcome from changes in both the peripheral nervous system, and the central nervous system consequentially leading to detrimental performance changes (Meeusen, Watson, Hasegawa, Roeland, & Piacentini, 2007). Primarily, studies measure fatigued states through subjective self reports and/or an individual's reduced ability to produce muscle force (Zijdewind, van Duinen, Zielman, & Lorist, 2006).

Thus, perceptions of physical fatigue are predominantly experienced from muscle changes, specifically as they begin to weaken. Once muscle fatigue is established, individuals may be able to generate some muscle power for a period of time.

However, prolonged effort to activate these motor neurons will increase an individual's awareness of perceived fatigue (Lorist, Kernell, Meijman, & Zijdewind, 2002). An increased perception of fatigue is further associated with a diminished willingness to exert effort. Especially when demands exceed the upper limit of what people are willing to put out (Macrona, Staiano, & Manning, 2009). In conjunction with perceptions of diminished capacity, physiologically the body will force alterations to resources. Specifically, as processing in the brain is hierarchal, and competitive during exercise bouts. Cardiac output to the brain will be downgraded, due to blood flow being required in other structures, resulting from increased activation from exercise (Wu, Ying, & Gomez-Pinilla, 2004).

For instance, exercising at maximal intensity reduces cerebral blood delivery volume by 25% per heartbeat compared to the resting state (Wu et al., 2004). Additionally, the brain works on a fixed amount of metabolic resources (Ide & Secher, 2000). Therefore, a high level of exercise arousal above resting states will place severe strains on the brain. Thus, decreased blood flow to the brain is likely to be an underlying reason behind cognitive performance deficits observed immediately following strenuous exercise. However, there is a lack of empirical work on direct effects of physical fatigue on the brain, reducing cognitive performance

(Tompsonowski & Ellis, 1986; McMorris, Collard, Corbett, Dicks, & Swain, 2008; Etiner et al., 1997). Furthermore, neuronal and cognitive processes require an energy supply in order to maintain neuronal excitability and synaptic function. Furthermore, brain tissues are metabolically active, requiring a constant supply of glucose in order to meet energy needs (Wu et al., 2004). Evidence emerging suggests that imbalances in energy levels may cause a detrimental impact on neurochemical and neurophysiological activity, causing cognitive performance deficits (Wu et al., 2004; Vaynman et al., 2006).

In sum, exercise-induced physical fatigue may make it difficult for individuals to focus on task-relevant stimuli, as a result of a variety of changes occurring within the central nervous system, and any experienced physiological fluctuations, such as HR, body temperature and blood pressure. More specific physiological explanations for the physical exercise-cognition relationship have been proposed by researchers, and are discussed below.

Physiological Explanations of Exercise-Cognition Relationship

A decrease in cognitive performance following or during exercise is often linked to the negative effects of physiological fatigue on mental processes (Gutin & Di Gennaro, 1968). The physiological effects from exercise are transient, revealing both positive and negative effects on cognitive performance. Currently prevalent theories have attempted to explain the relationship between exercise-induced fatigue and cognitive performance. The multidimensional allocation of resources theory (Kahneman, 1973) produces one hypothesis relevant to the *a priori* expectations of the present study. This hypothesis states that, high intensity exercise regimens will induce physical fatigue, which will subsequently impair cognitive performance. Evidence from physiological studies has established that neural activation will occur across a large number of neural structures (Dietrich, 2006). Such evidence has demonstrated that exercise at high intensity levels, will result in the brain receiving four times less blood volume per heartbeat compared to resting states (Dietrich, 2006). The brain functions under a finite amount of resources, therefore it is feasible that individual brain capacities, to maintain neural activation of motor neurons will

occur at the expense of other neural structures responsible for cognition, owing to the finite number of resources available (Dietrich, 2006).

Previously, literature has grossly underestimated the amount of brain tissue that is activated just simply by moving (Dietrich, 2006). For instance, a study investigating brain activation in response to exercise, found that when rats were run for 30 mins on a treadmill at 85% maximum O₂ uptake, significant increase of local cerebral glucose utilisation (LCGU) was observed (Vissing, Anderson, & Diemer, 1996). Increased LCGU was observed in all brain structures expect for a few, which included the prefrontal and frontal cortex. Illuminating that physical exercise will require a large number of neural structure activation across the brain. While previously researcher's believed that the brain would receive additional metabolic resources during exercise, conclusive evidence of this belief is lacking. As large areas of the brain are devoted to our basic processes, such as autonomic regulation and motor output activation of theses processes during physical activity is compulsory. Thus, because the human brain is unable to maintain activation in all neural structures at a given time in conjunction with its inability to attain additional resources, it has been proposed that a needs-based shift in activation of one structure will occur, at the expense of activation within other structures (Dietrich, 2006).

In addition, it has been proposed that activation of muscular, skeletal and respiratory systems, both during exercise, and physical awareness following exercise, is likely to cause 'neural noise'. Neural noise refers to the bombardment of stimulated neurons in the CNS. Which is purported to cause interference with performance ability when an individual is completing a prescribed cognitive task (Gutin, 1973). Therefore, while exercise may initially facilitate attentional processes through its direct effect on the CNS, several authors have demonstrated that the facilitative effects of exercise on cognitive function are cancelled out by exhaustive exercise (Dempsey, Romer, Rodman, Miller, & Smith 2006; Faria, Parker, & Faria, 2005; Kamijo, Nishihira, Higashiura, & Kuroiwa, 2007).

Summary

Further research is required in order to ascertain under which circumstances physical exercise will result in benefits to cognitive performance and which circumstances will result in cognitive deterioration. To summarise, the current literature on the exercise-cognition relationship points towards a complex relationship. Prior literature has illuminated that cognitive performance effects are likely to be moderated as a result of the wide range of methodologies that has been and can be applied. In future, research should focus on a small battery of specific cognitive test(s) in order to increase reproducibility of results in later studies. Throughout literature in this area there is evidence that is suggestive of an interaction between exercise intensity and cognitive performance will be significantly moderated by individual characteristics, such as, fitness levels. Thus, it is advisable that future studies apply an individualised exercise workload, for instance, using exercise tasks that incrementally increases the exercise workload while simultaneously monitoring levels of intensity measures, e.g. HR, Vo₂ max. If these recommendations are applied then research may be able to produce evidence demonstrative of a definitive answer to the underlying phenomenon of the exercise-cognition relationship. Because, despite decades of research, it is still difficult to determine what effects exercise will generate on cognitive performance. With the growing interest in the influence exercise arousal has on operational performance, further, rigorous investigations are desperately needed.

The Present Study

The previous chapters have demonstrated that exercise-induced fatigue could influence cognitive functioning (Dietrich & Sparling, 2004). In addition, earlier chapters also demonstrated that nutritional supplements, specifically polyphenolic compounds, might influence signalling cascades and connectivity strength in neurons. Providing actions that may lead to enhanced improvements to cognitive functioning. (Desideri et al., 2012; Lampion et al., 2012; Letenneur, Proust-Lima, Gouge, Dartigues, & Barberger-Gateau, 2007; Kess-Guyog et al., 2012; Nurk et al., 2008). Attempting to clarify the effects dietary phytochemical extracts have on cognition requires measurement with an array of cognitive tasks, under a range of different physiological states of arousal. As part of ongoing research into the effects

blackcurrant phytochemicals has on mental and physical performance, the New Zealand Institute of Plant and Food Research (PFR) are conducting the present study. The research investigating whether blackcurrant extracts can ameliorate physical fatigue effects on cognitive performance is apart of an overreaching research project, exploring the effects phytochemical compounds have on physical and mental health.

An earlier PFR-initiated study conducted by Ratlidge (2013) investigated the efficacy of a blackcurrant extract intervention on motor performance, following a mentally fatiguing task. As an extension of Ratlidge (2013) study, the current research was designed to investigate the efficacy of a blackcurrant extract on minimising decrements to cognitive performance caused by exercise-induced fatigue. The purpose of this line of investigation was to discover whether the blackcurrant phytochemical compound could reduce the negative effects of physical fatigue on cognitive performance.

Given the lack of research investigating the interaction between physical fatigue, cognitive performance, and dietary supplementation researchers' are provided an opportunity to generate and test a multitude of hypotheses. As detailed earlier, the existing literature indicates that the relationship between physical fatigue and cognitive performance is complex. While there is a shortage of strong empirical evidence clarifying the relationship between physical fatigue and cognitive performance, there is tentative evidence, suggestive of an association.

The present study appears to be one of the limited few to examine phytochemical influences upon cognitive performance in participants' experiencing a physically fatiguing state. Therefore, the aim of the present study was to provide insight into the effects exhaustive exercise has on cognitive performance and whether a blackcurrant supplement given prior to exercise could influence an individual's post-exercise mental performance.

Hypothesis One

Based on previous evidence, which identified physical fatigue as a potential cause of cognitive performance deficits, it was hypothesised that participants' who are

physically fatigued would demonstrate a decline in cognitive performance on a subsequent battery of cognitive measures, when compared against individuals' who weren't physically fatigued (Tompsonski, 2003; McMorris et al., 2012).

Previously, researchers' have applied exercise tasks for a determined length of time, $N < 15$ min, in order to produce fatigued states in participants'. However, as stated earlier, length of time appears to be a moderator in the performance effects observed within this area of research. Thus, the length of time spent on an exercise task is contended as an inefficient methodology for inducing fatigue in participants (McGlynn, Laughlin, & Bender, 1977). Recently, as a time efficient alternative to induce fatigued states from exercise, researchers' have begun to apply a high intensity intermittent task (HIIT). HIIT has demonstrated that participants' are able to achieve high intensity workloads within a period of fewer than 10 mins. A high intensity exercise task is often performed with repeated bouts of vigorous cycling exertion, thus, causing fatigued states quickly. Furthermore, such exercise tasks enable researchers' to individualise the workload each participant engages in. Thus, the potential moderating variable of individual fitness level can be controlled for.

A common HIIT intervention and the model used in the present study, is the Wingate test. The Wingate requires maximal peddling for 30 secs against a constant force on an ergometer bike. Based on other research, which identified the HIIT exercise as a time efficient alternative to traditional high intensity cardiorespiratory exercise, the present study asked participants' to complete the Wingate HIIT task for up to a maximum of 10 mins or until they reached voluntary exhaustion (Ekkekakis, 2009). The expectation was that physical fatigue would be occur within the 10-min time-on-task period, therefore, it was expected that individual performance in the Wingate test would decline and fatigue would be induced before or at the 10-min limit (Ekkekakis, 2009; Linkis et al., 1995; Kahneman, 1973). The effects physical fatigue has on physiological and attentional resources were expected to carryover to participants' performance on subsequent cognitive tasks. Resulting in decrements to cognitive performance, relative to baseline scores.

Hypothesis Two

The main research question of the present thesis was whether a blackcurrant photochemical extract could ameliorate physical fatigue effects on subsequent cognitive performance. To investigate this, a single acute dose of blackcurrant extract was administered. It was hypothesised that participants' who received the blackcurrant supplement would demonstrate smaller falloffs in cognitive performance following the physically fatiguing task, in relation to participants' who received the placebo.

Animal studies provide evidence that consumption of phytochemical compounds produces a direct interaction with cellular and molecular targets in the brain, notably in regions associated with learning, memory and cognition (Andres-Lacueva et al., 2005; Passamonti et al., 2005; Vauzour et al., 2008). Blackcurrants are a rich source of antioxidants and phytochemicals. Both in vitro and in-vivo animal studies, and to a lesser extent human studies, has suggested that phytochemical compounds provide a neuromodulatory and neuroprotective role that has the potential to decrease age-related cognitive deficits (Shukitt-Hale et al., 2008).

Currently, published studies examining the role blackcurrant supplement has on cognitive performance under physically fatigued states is scarce. However, prior evidential reports in this area have implied that phytochemical compounds may have an ability to reduce cognitive fatigue and improve neuroplasticity (Lindheimer et al., 2013; Miller & Shukitt-Hale, 2010). If blackcurrant extracts reduce physical fatigue effects on cognitive performance, these actions could be attributed to the neuroprotection exerted from blackcurrant phytochemical. Specifically, if blackcurrant phytochemicals increase the number of synapses in the brain thereby improving neuron connectivity during aversive circumstances, e.g. fatigue, participants would demonstrate relatively stable performance, or improved performance following exhaustive exercise. In comparison with the controls', whose accuracy would be expected to decline.

CHAPTER 4 Methodology

Participants

Seventy-two healthy people, aged between 16- 46 years ($M= 25.51$, $SD = 8.63$) volunteered to participate in the study. Participants' had no known neurological or psychological conditions and motor ability was not impaired in any way. All participants' had normal or corrected to normal vision. Of these participants', 42 were female ($M= 23.48$, $SD= 7.99$), and 30 were male ($M= 28.7$, $SD= 8.86$). Participants were recruited through advertisement flyers placed around Massey University, Plant and Food Research and through word of mouth (see Appendix A). Prior to any testing participants' were provided with an information sheet, and consent form. These were read and signed before any procedures were begun (see Appendix C & D).

Each participant received a \$20 voucher to compensate for his or her time to do the research. All procedures and materials used in the present study were approved from Human and Disability Ethics Committee (HDEC), registration number 14/NTB/109, and Massey University Human Ethics Committee (protocol 13/20).

Group assignment

Participants were randomly assigned to one of four conditions; blackcurrant/ exercise; no black currant/exercise; blackcurrant/ no exercise; no blackcurrant/ no exercise. There were between 8 and 11 participants' assigned to each of the four conditions as shown in Table 1. Randomisation of assignment was handled with a computer random number generator, with the restriction that the ratio of males to females was roughly the same for each group.

Table 1

Group assignment in accordance to gender and condition

	Blackcurrant		No Blackcurrant	
Exercise	Males	8	Males	11
	Females	10	Females	10
	Total	18	Total	21
No Exercise	Males	9	Males	8
	Females	11	Females	11
	Total	20	Total	19

Apparatus and Measures

Cognitive tasks

The primary aim of the present study was to investigate whether blackcurrant supplementation produced ameliorating effects on cognitive performance following a physically fatiguing exercise task. All cognitive tasks selected for the present study was based on their previous use in similar studies, in which they demonstrated sensitivity towards exercise-induced effects on cognitive performance (Audiffren, Tomporowski, & Zagrodnik, 2008; Ferris, Williams, & Shen, 2006; Lambourne & Tomporowski, 2010).

Stroop



Figure 2: The possible stimuli in the Stroop task. Congruent stimuli are those where the word and colour match. The other stimuli are incongruent. See text for further details.

Note: Adapted from [http://www.scienceblogs.com/cognitivedaily/wp-](http://www.scienceblogs.com/cognitivedaily/wp-content/blogs.dir.262/files/2012/04/i-baa00243255a2b7099346b33ae07045d-stroophyp.gif)

[content/blogs.dir.262/files/2012/04/i-baa00243255a2b7099346b33ae07045d-stroophyp.gif](http://www.scienceblogs.com/cognitivedaily/wp-content/blogs.dir.262/files/2012/04/i-baa00243255a2b7099346b33ae07045d-stroophyp.gif)

The Stroop task is a measure of concentration and attention, principally assessing an individual's ability to inhibit habitual responses (Alves et al. 2012; Etnier & Chang, 2009). Participants' performed this inhibition task on a computer. Three words (blue, green, red) were serially presented in random order upon the screen for 1,500ms, or until the participant provided an answer. If the participant failed to respond in the 1500 ms time window the computer registered the missed response as a wrong answer. Participants' were asked to press one of the three coloured keys on the keyboard (blue, green, red) with the correct response corresponding to the ink color of the word presented on the screen (blue, green, red). For instance, if the word "green" appears in red, then the corresponding red key must be pressed for a correct response to be registered. The computer program randomly selected the ink colour, the word presented, and whether this would be given in a congruent or incongruent style. Participants' were asked to use only their dominant hand on the keyboard and only their index, middle and third fingers were to be placed on the corresponding red, green, and blue keys.

Participants' completed a practice set of 20 trials before completing the main block of 72 trials. This was to ensure that participants were comfortable with hand position and key selection within the time window for each trial. If participants' found the task difficult during the 20 practice trial block, another block was provided and continued until the participant felt confident to move onto the main trials. Participants' were instructed to respond as accurately and quickly as possible.

Digit symbol substitution

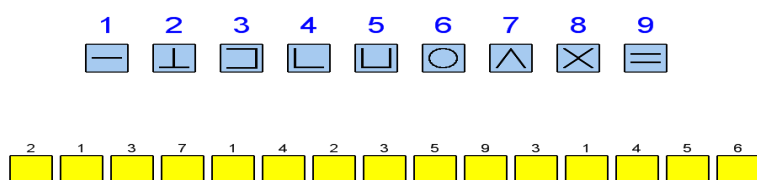


Figure 3: Viewpoint of a computer screen displaying the digit symbol substitution task. Participants' were required to select the corresponding numbered symbol from the top and drag it to the matching numbered empty box underneath.

Source: Loudon, M. (personal communication, May, 14 2015).

The Digit Symbol Substitution Task (DSST) measures individual psychomotor performance ability; in the present study participants' performed this task on a computer. The task had nine symbols that were constantly displayed at the top of the screen, as shown in *Figure 4*. Placed underneath these symbols were 15 empty boxes with the corresponding numbers of the symbols above the boxes. Participants' were required to select the correct symbol to the corresponding numbered empty box below. Participants' were instructed to use their dominant hand on the mouse in order to quickly and accurately input the correct symbol into the box. There were 45 main trials presented in three sets of 15. Participants' completed a practice trial of one set of 15, before moving to the main sets of trials. The computer tracked the speed it took participants' to complete the digit symbol task, so accuracy and processing time were the measures recorded.

Backwards Digit Span Task

Digit-Span presentation	Reported backwards
1. 5 9 0	1. 0 9 5
2. 4 8 6 1	2. 1 6 8 4
3. 7 3 0 9 4	3. 4 9 0 3 7
4. 2 4 9 6 5 8	4. 8 5 6 9 4 2
5. 1 4 6 8 2 4 5	5. 5 4 2 8 6 4 1
6. 3 9 2 1 5 7 6 0	6. 0 6 7 5 1 2 9 3
7. 6 2 5 7 3 9 1 8 4	7. 4 8 1 9 3 7 5 2 6
8. 0 6 3 8 9 4 1 7 2 5	8. 5 2 7 1 4 9 8 3 6 0

Figure 4: Display of the Digit Span Backwards Task. Participants' were required to remember a sequential number pattern displayed on the computer and then re-enter that number sequence in reverse order.

Note: Adapted from

<http://www.socrates.berkeley.edu/~kihlstrm//introductionWeb/images/memory/DigitSpan.JPG>

The reversed Digit Span required participants' to retain a small amount of task-relevant information within their memory, whilst simultaneously juggling the information around mentally in order to complete the task. This cognitive task is considered to measure working memory. The computer displayed a box in the middle of the screen that presented a numerical sequence. Participants' were instructed to enter the displayed sequence into the computer, in the reverse order, to what was

presented, from the last digit presented to the first digit presented. Digits ranged from 0-9 and were randomly presented. The task began with a two-digit sequence, and progressed in length by one digit until a participant either correctly inputted the maximum sequence of digits, $N=10$, or until a participant had made three consecutive input errors. The final score was the number of digits correctly entered.

Trail Making Test B

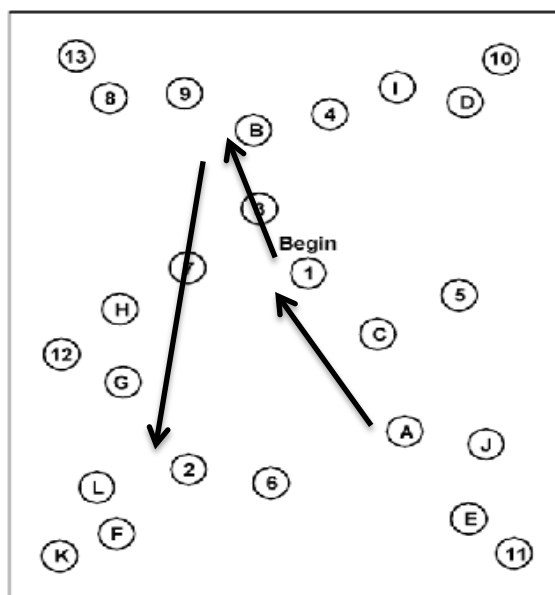


Figure 5: Computer display of the Trail making task (B). Participants' were required to create a trail, starting at the first letter, A, and moving to the first number, 1, following this sequential pattern until successfully reaching number 13.

Source: Adapted from <http://www.nhtsa.gov/people/injury/olddrive/OlderDriversBook/pages/Trail-Making.html>

This test measures visual conceptual and visuomotor tracking; the test can be administered in two parts, A & B. For the purposes of the present study the Trail making test B was used. Participants completed this task on the computer. Test B is constructed as a dual symbol task, whereby, participants' were required to switch between alphabetical letters and numbers in order to form the correct trail. Participants' were required to select the first letter A, and then locate and select the first number 1, carrying on in this manner (A-1, B-2, C-3 etc.), until all the symbols were correctly selected. If participants' selected an incorrect number or letter the computer would not allow its selection and give a beep. The computer recorded both time taken to complete the task and the number of errors made

Choice Reaction Time Task

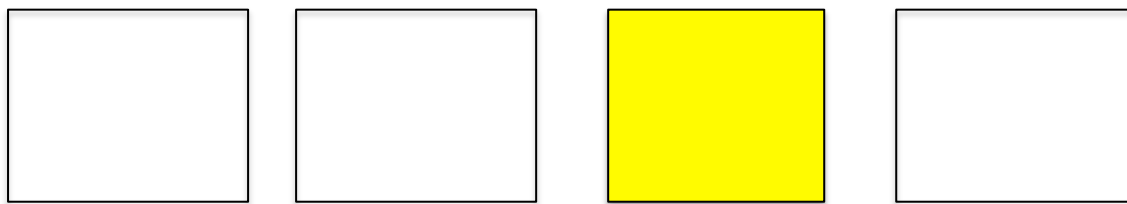


Figure 6: Display of the choice reaction time task. Four boxes were displayed across the computer screen, and participants' were required to press the computer key that matched the highlighted box as quickly as possible. The highlighted box was chosen at random on each trial.

Note: Adapted from personal memory of the choice reaction time task.

Reaction time (RT) tasks are utilised in acute exercise studies and provide a valid indication of individuals' mental processing and decisional speed. This choice reaction time task was completed on the computer. Four boxes were positioned across the middle of the screen, and one box chosen at random was highlighted (yellow) on each trial. Placing their index and middle fingers on the Z and X computer keys with their left hand, and their right hand the index and middle fingers on the N and M keys, participants' were instructed to press the key that corresponded to the highlighted box. RT was recorded to the nearest millisecond.

Subjective Measures

In conjunction with the cognitive tasks participants' were required to complete a mood and motivational questionnaire. This questionnaire assessed participants' mental and physical fatigue states prior to and following treatment manipulation. It must be noted that while the data from the energy and fatigue scale, ratings of perceived exertion, the Baeke, HR, and lactate levels were collected for the overall research programme these will not be reported in this thesis. The latter concentrates on cognitive changes in the presence of exhausting exercise and a blackcurrant supplement.

Mental and Physical State and Trait Energy and Fatigue Scales

The items used in this scale were developed by Patrick O'Connor (2006), and provide a concise measure of acute and chronic feelings of mental and physical energy and fatigue (O'Connor, 2006). Reports of how an individual is feeling at a given moment are considered to be the most accurate measures of physical states. The questionnaire covered eight dimensions; physical energy state, physical fatigue state, mental energy

state, mental fatigue state, trait physical energy, trait physical fatigue, trait mental energy and trait mental fatigue. These dimensions are arranged into three separate parts. Firstly, the questionnaire examines the demographic background of participants', but this was excluded from the present study as the Baecke Questionnaire and Health Screening form for demographical background checks was used instead. Secondly, the scale assessed participants' current feelings of energy and fatigue. Participants' were instructed to mark a horizontal line on a Likert-type scale that indicated their current feelings of mental energy and mental fatigue, and physical energy and physical fatigue. This scale ranged from "I feel I have no energy" to "strongest feelings of energy felt". Thirdly, this scale required participants' to rate how often they perceived themselves to experience six different states while completing mental and physical activities, e.g. energetic; fatigued; vigorous; exhausted; full of pep; worn out. A rating for each item was recorded on a 5-point Likert-type scale ranging from 0-4, where 0 represented 'never' and four represented 'always'. This questionnaire was administered as soon as participants arrived at the lab, and following the exercise/control intervention.

Ratings of Perceived Exertion

Borg's (1990) ratings of perceived exertion (RPE) is a subjective measure, used to monitor aerobic exercise intensity, thus, indicating a participants' level of physical strain on a scale of six to 20. These can be used to represent heart rates ranging from 60-200bpm. Borg created this scale so that it increased linearly with exercise intensity upon a cycle ergometer, with oxygen and heart rate increasing linearly with work. Participants' were asked to verbally rate their perceived feelings of exertion prior to commencing the exercise intervention and immediately following completion of the exercise intervention.

Baecke Questionnaire and Health Screening

This questionnaire was used to evaluate participants' physical activity levels within the past 12 months. The questionnaire items covered three dimensions of habitual physical activity occupational physical activity, physical exercise in leisure, and leisure and locomotion activities. The total 16 questions added together comprised of the total score for habitual physical activity.

Heart Rate Measure

Upon entering the laboratory, participants' were fitted with a heart rate (HR) monitor strap and watch. Participants' HR's were monitored throughout the trial. Those in the exercise condition had their HR recorded following every 30 s cycling bouts completed, each participant completed at least three all out cycling bouts. Those assigned to the control condition had HR's recorded before resting for 10 mins and once after <10 mins rest.

Lactate Measure

Lactate was measured from bloods collected from participants' ear lobe. The test strip collected an exact amount of blood that was then placed into the Lactate Pro - Lactate Analyser that measured the amount of lactate within the drop of blood. Blood lactate was measured several times during the trial, before supplementation, before the fatiguing exercise or control task, immediately following fatiguing exercise or rest, and following the second administration of cognitive testing.

Treatments

Exercise-induced fatigue manipulation

Participants' completed a high intensity interval task (HIIT), used to elicit physical fatigue. The task consisted of a series of 30 s bursts of high intensity cycling on the Wingate cycle. Participants' were required to cycle at full capacity for 30 s and then rest for one min, with this sequence continuing until participants' reached volitional exhaustion. The weighted resistance participants' were asked to cycle against was calculated from a percentage of their overall body weight, that is, 2%, 3%, or 4% body weight. HR was monitored from a HR monitor strapped to the participant's chest throughout the entire two hours of the experiment. Participants' donated blood, from an ear prick, twice during the fatiguing exercise task, once prior to beginning the workout, and once more immediately following exercise cessation. The blood sample was used to provide estimates of lactate level. Participants' were exercising for a length of time no shorter than three mins, but no longer than 10 min.

Control Task

The control task consisted of participants' watching 10 mins of a David Attenborough Planet Earth documentary (Fothergill, 2006). This series was chosen based on the theme being considered emotionally neutral by the researchers.

Phytochemical Manipulation

Blackcurrant extract

Participants' randomly assigned to the intervention treatment were administered a single dose of blackcurrant extract in capsule form. These capsules were made up by PFR on the day of the main trial. The supplement was given 1 hour prior to the exercise/rest condition beginning. This length of time, between supplement ingestion, and commencement of the testing phase was based on the fact that previous literature demonstrated that maximum levels of blackcurrant phytochemicals occurred around the 45 mins, to one hour, post ingestion (Nielsen, Dragsted, Ravn-Haren, Freese, & Rasmussen, 2003).

Design and Analysis

The design for the present study was a 2 (Time: T1, T2) x 2 (Intervention: Blackcurrant, No Blackcurrant) x 2 (Condition: Exercise, No Exercise) factorial analysis of covariance (ANCOVA). The covariate was the scores on the various cognitive tests obtained before the intervention (Time 1). Pallant (2010) notes that this approach is useful with small sample sizes, and where the expected effect sizes are small to modest.

A planned approach was applied to the analysis; the specific research objectives were predetermined prior to the study's commencement. Thus, the data was analysed with cognizance of research objectives (research questions). This approach is expected to improve the statistical power (SP) of the study (Pallant, 2010).

Potential carryover effects were not of concern for the present study. As baseline measures were completed approximately 1.2 hours before the supplement intervention was ingested, and 2 hours prior to the Condition treatment. Therefore, any differences

within cognitive scores that occurred at Time 2 could be attributed to ongoing effects of the blackcurrant supplement, which has at least a 10-hour wash out effect.

The following statistical analyses were conducted using a statistical package SPSS for Macintosh, version 20.0 (IBM Corp, 2012). Standard statistical outcomes were reported alongside the F values and associated statistics. The principal measures reported were effect sizes and significance levels; calculated in accordance with the guidelines suggested by Cohen (1988); $d = .20$ or $\eta_p^2 = .01$ is a small effect; $d = .50$ or $\eta_p^2 = .06$ is a medium effect; and $d = .80$ or $\eta_p^2 = .14$ is a large effect. Effect size approximation was achieved using partial eta squared (η_p^2), calculated within SPSS. The family-wise significance level was set at .05. ANCOVA tables can be found in Appendix G.

Procedure

Participants' completed two sessions for the current study, one familiarisation session, and the second the main trial.

Familiarisation Session

The familiarisation session was conducted prior to the main trial for all participants' taking approximately 45 mins to complete. Practice effects are known to influence a number of commonly used measures of cognitive function. Thus, to ensure practice effects were minimised in the present study, a familiarisation session was required. This involved participants' being exposed to an example of the exercise task. Participants' were required to undergo a short cycle on the Wingate ergometer whereby researchers would be able to gather information on correct seat height for example; knee angle needed to be straight on the downward pedal stroke. This session also provided researchers the opportunity to establish participants' individual fatiguing load, as calculated from their bodyweight. Instructions to remain seated throughout the exercising phase and maintain a minimum 100RPM during the 30 s bursts was given before cycling commenced. Each participant received a verbal 5 s countdown before each 30 s bout, allowing participants' to prepare for peddling all out. A counterbalanced lever arm was used to control the onset of the task; pushing

the micro switch for the counterbalanced arm immediately dropped the weighted resistance onto the flying wheel. The Monark anaerobic test software monitored participants' output from the moment the counterbalanced arm was dropped till completion of the task. The task typically took a maximum of 10 mins to complete.

During this familiarisation stage, weighted resistance loads were increased incrementally 0.5% after each 30 s burst, continuing until researchers observed that the increased resistance load forced participants' output to depreciate. At this point, it was usually a struggle for participants' to maintain a rate above 100 rpm throughout the entire 30 s burst. The fatiguing resistance load (the highest load an individual could maintain at 100 rpm for 30 s) was recorded and the exercise task terminated.

Participants' were then required to complete a single training session on all five cognitive tasks. It was hoped that this familiarisation with the cognitive tasks would minimise the effects of practice, which otherwise might confound the changes in performance between baseline and post treatment in the main trial.

Participants' were then asked if they would like to continue with the study. If they were agreeable a list of foods to avoid 48 hours prior to the main trial day was provided (see Appendix F for these forms).

Main Trial

Two days prior to coming to the laboratory to complete the main trial, participants' were contacted and asked to make sure to avoid the foods from the list provided at familiarisation.

First stage of the main trial

Each main trial session ran for 2.5 hours. As participants' arrived at the laboratory they were fitted with a HR monitor and watch that was worn throughout the entirety of the experiment. They then completed the mental and physical state and trait energy and fatigue measure (O'Connor, 2006). Following this, participants' completed the baseline measurement of cognitive performance, after which blood lactate samples were obtained. Participants' were then required to complete the first HIT cycling bout on the Wingate Cycle, after which another blood sample was taken.

Second stage of the main trial

Participants' were then required to rest for 30 mins, to ensure any physiological arousal that had occurred from the HIT cycling bout returned to baseline levels. After 30 mins participants' were administered the blackcurrant or placebo intervention, and another blood sample was taken. Participants' then rested for an hour following ingestion of the intervention. After this rest period, a further blood sample was taken. At this stage participants' were informed of what condition they had been randomly assigned to (exercise vs. no exercise).

Third stage of main trial - Exercise session

Participants' completed the high intensity, repeated 30 s Wingate cycling test, with 1 min rest intervals between each 30 s bout. Participants' cycled as hard and as fast as they could against their individual weighted resistance load. The Monark anaerobic test software program monitored participants' power output, based on their pedal turnover. They were encouraged to continue peddling to maximum capacity during each 30 s bout until volitional exhaustion occurred in conjunction with the computer software registering a decline in performance power output.

Fourth stage of main trial

After participants' reached this exhausted state, Borg's (1990) RPE scale was administered and another blood sample obtained. Participants' then completed the post-exercise mental and physical state and trait energy and fatigue scale. Before moving on to have their final blood sample was taken.

Control session

Participants' who were not in the exercise condition were also required to have blood samples taken, analysed as part of the overall aims of the study. This was achieved at the 1hour mark 30-mins after the initial high intensity intermittent cycling task was completed. Afterwards, participants' spent 10 mins watching an emotionally neutral documentary movie about planet earth (Attenborough et al., 2005). The time frame of 10 mins was selected, as this was the maximum length of time that a participant could

spend completing the HIIT Wingate task. It was expected that all participants' would fatigue before this 10 min mark was reached. Another blood sample was then obtained from participants', and a verbal account of their rating of perceived exertion (Borg, 1990) was recorded. Participants' then completed the post-exercise mental and physical state and trait energy and fatigue scale. Afterwards, participants' completed the cognitive tasks and a final blood sample was taken.

Fifth stage of the main trial – debrief

At the conclusion of the trial, participants' were debriefed and any questions they may have were answered. They were thanked for taking part in the study and provided with a \$20 shopping voucher and a chocolate fish.

CHAPTER 5 Results

Preliminary ANOVAs indicated that there were no differences due to Time of Day (a.m. or p.m.) or to Gender differences. Therefore, the results were combined across these two variables for further analysis.

Further, it was considered that the mental and physical state and trait energy and fatigue scales and the perceived exertion scale were considered as possible covariates, but scores did not correlate sufficiently with the dependent variables to warrant their inclusion.

A series of individual ANCOVAs were conducted for each of the five cognitive tasks. It was expected that the physical fatiguing task would impair cognitive performance for those participants' randomly assigned to the fatiguing condition. It was also predicted that those participants' who received the blackcurrant extract would demonstrate improvements to cognitive performance across all cognitive tasks. In comparison to those participants' who received the placebo. For both research questions the independent variables were Condition (exercise vs. no exercise) and Intervention (blackcurrant vs. placebo). The dependent variables were the scores achieved on each cognitive test at Time 2. Scores obtained at Time 1 (prior to treatment) were entered as a covariate adjusting for variation in the scores prior to treatment. Note that all tables of means (M) and standard errors (SE) provide the adjusted values.

All ANCOVA assumptions (linearity, normality, homogeneity of regression slopes, and equality of error variances) were met unless otherwise stated. For ease of reading, both hypotheses are dealt together for each separate cognitive task. Dependent measures for the cognitive tests were either accuracy or processing speed. Error rates for processing speed and RT were too few to analyse.

Stroop

Separate analyses were conducted for congruent, incongruent and the overall Stroop scores. It was expected in particular that exercise would negatively affect accuracy for

the incongruent stimuli and that the blackcurrant supplement would reduce the impact of exercise.

Stroop Overall

Table 2

Adjusted means, standard errors (in parentheses) and 95% confidence intervals for Stroop (overall) cognitive task performance as a function of Time 2, Intervention and Condition.

NBc/NEx	98% CI		NBc/Ex	98% CI		BC/NEx	98% CI		BC/Ex	98% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
55.9(1.1)	55.7	58.2	55.6(1.1)	52.9	57.9	55.3(1.1)	52.9	57.7	54.2(1.0)	52.0	56.4

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: Stroop overall = accuracy.

Table 2 provides the adjusted means, standard errors, and 95% confidence interval (CIs) for all 4 groups. By examining the Exercise and No Exercise groups that did not take the blackcurrant extract, it can be seen that exercise had a negligible effect on the total Stroop accuracy score, $F < 1$. Neither did the blackcurrant supplement produce any effect, $F < 1$. Therefore, neither hypothesis is supported by these results.

Stroop Congruent

Table 3

Adjusted means and standard errors (in parentheses) and 95% Confidence intervals for Stroop Congruent stimuli, as a function of Time 2, Intervention, and Condition

NBc/NEx	95% CI		NBc/Ex	95% CI		Bc/NEx	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
56.6(1.4)	51.0	56.2	52.8(1.3)	50.3	55.5	54.2(1.4)	51.5	56.9	54.0(1.2)	51.6	56.5

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: Stroop congruent = accuracy.

Table 3 provides the adjusted means, standard errors, and 95% confidence interval (CIs) for all 4 groups. By examining the Exercise and No Exercise groups not taking the supplement it can be seen that exercise had a slight effect on total Stroop congruent accuracy scores, however this effect did not produce a significant main effect for Exercise, $F(1,72) = .116, p = .734, \eta_p^2 = .002$. Blackcurrant had a negligible

effect, revealing no significant interaction effects $F < 1$. Thus, support cannot be provided by to either of the two hypotheses being tested.

Stroop Non-congruent

Table 4

Adjusted means and standard errors (in parentheses), and 95% Confidence interval's for Stroop Non-congruent stimuli as a function of Time 2, Intervention, and Condition

NBc/NEx	95% CI		NBC/Ex	95% CI		Bc/NEx	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
56.6(1.4)	53.9	59.3	55.5(1.3)	52.7	58.1	56.8(1.4)	53.9	59.6	55.5(1.3)	52.9	58.0

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: Stroop non-congruent = accuracy.

Table 4 provides the adjusted means, standard errors, and 95% CIs for all 4 groups. Examination of accuracy scores for the Exercise and No Exercising groups not taking the supplement it can be seen that exercise had a negligible effect for accuracy on Stroop Non-congruent stimuli $F < 1$. Examination of the blackcurrant effects also produced no effects on accuracy $F < 1$. Therefore, neither hypothesis is supported by these results.

Digit Span Backwards

Table 5

Adjusted means and standard errors (in parentheses), and 95% CI's for Digit span backwards, as a function of Time 2, Intervention and Condition

NBc/NEx	95% CI		NBC/Ex	95% CI		Bc/NEx	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
6.4(.3)	5.8	6.9	6.2(.3)	5.6	6.8	6.3(.3)	5.7	6.9	6.5(.3)	5.9	7.0

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: digit span backwards = highest level achieved (1-10).

Table 5 provides the adjusted means, standard errors, and 95% CI's for all 4 groups. Examination of the highest level achieved scores for the Exercise and No Exercise groups not taking the supplement it can be seen that Exercise has a negligible effect on score level achieved at Time 2, $F(1, 72) = .000$, $p = .986$, $\eta_p^2 = .000$. Further, non-significant interaction effects for blackcurrant was revealed, $F(1, 72) = .220$, $p = .640$, $\eta_p^2 = .003$. The estimated marginal means plot displays this interaction as dis-

ordinal (see *Figure 7*). Implying that one of the factors had one kind of effect in one condition, yet a different kind of effect in the other condition. So support cannot be provided to either of the two hypotheses being tested.

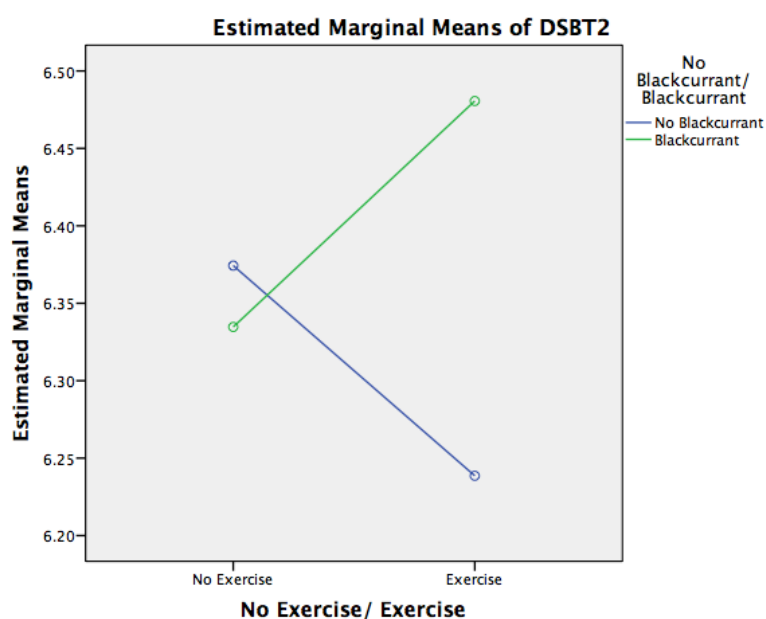


Figure 7. Interaction between condition and intervention for overall score achieved at Time 2.

Source: IBM Corp. (2012). IBM SPSS Statistics for Macintosh (Version 21.0). Armonk, NY: IBM Corp

Digit symbol substitution

Table 6

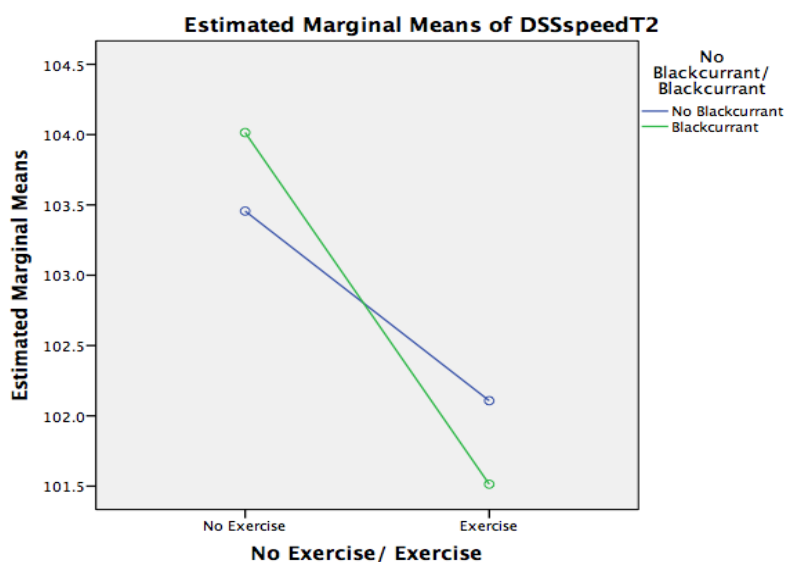
Adjusted means and standard errors (in parentheses), and 95% Confidence Interval's for Digit symbol substitution as a function of Time 2, Intervention, and Condition

NBc/NEx	95% CI		NBc/Ex	95% CI		Bc/NEx	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
103.4(1.8)	99.8	107.0	102.1(1.8)	98.4	105.7	104.0(1.9)	100.1	107.8	101.5(1.7)	98.0	104.9

Note: NBc = no blackcurrant; NEx = no exercise; Bc = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: digit symbol substitution = Reaction speed.

Table 6 provides the adjusted means, standard errors, and the CI's for all 4 groups. Interestingly, examination of mean performance across all conditions, demonstrated a decline in reaction speed at Time 2 (see Table 6). However the main effect for

reaction speed for the digit symbol substitution did not reach significance for Condition (exercise vs. no exercise), suggesting that exercise induced physical fatigue failed to negatively influence participants' cognitive performance on this task, $F(1, 72) = 1.12, p = .293, \eta_p^2 = .016$. It is noteworthy to mention that the effect size of, $\eta_p^2 = .016$, indicated that a larger sample size may result in significant main effects being observed. Reaction speed on this task showed no significant interaction effects for performance at Time 2 as a resulting from the treatment interventions, (blackcurrant vs. placebo & exercise vs. no exercise), $F < 1$. Once again, the estimated means plot revealed a dis-ordinal interaction between Condition and Intervention (see *Figure 8*). Consequently, neither hypothesis is supported by these results.



Covariates appearing in the model are evaluated at the following values: DSSspeedT1 = 106.79

Figure 8. Interaction between condition and interaction for reaction speed at Time 2.

Source: IBM Corp. (2012). IBM SPSS Statistics for Macintosh (Version 21.0). Armonk, NY: IBM Corp

Trail making task (B)

Table 7

Adjusted means and standard errors (in parentheses) and confidence intervals for Trail making task (B), as a function of Time 2, Condition, and Intervention

NBC/NEx	95% CI		NBC/Ex	95% CI		Bc/NEx	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
44.3(2.3)	39.7	48.7	44.4(2.3)	39.9	48.9	44.2(2.4)	39.3	49.0	50.3	46.0	54.6

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: Trail making task (B) = Reaction speed.

Examination of the adjusted mean performance, standard errors, and 95% CI's for all 4 groups revealed a decreased reaction speed at Time 2 across all groups (see Table 7). The main effect for condition did not reach statistical significance, $F(1, 72) = .1.9$, $p = .17$, $\eta_p^2 = .028$. It should be noted examination of the effect sizes for the main effect for Condition, and the interaction effect for Condition x Intervention, were considerably large, $\eta_p^2 = .28$, $\eta_p^2 = .025$, respectively. It's possible that a larger sample size would produce statistically significant results. Finally, there was no significant interaction effects observed, $F < 1$. Therefore, support cannot be provided to either of the two hypotheses being tested.

Choice Reaction Time Task

Table 8

Adjusted means and standard errors (in parentheses), and 95% CI for complex reaction time task as a function of Time 2, Intervention, and Condition.

NBC/NEx	95% CI		NBC/Ex	95% CI		Bc/NBC	95% CI		Bc/Ex	95% CI	
	Lower	Upper		Lower	Upper		Lower	Upper		Lower	Upper
435.0(5.8)	423.5	446.6	432.3(5.8)	420.7	444.0	447.0(6.1)	434.8	459.3	439.2(5.5)	428.1	450.3

Note: NBC = no blackcurrant; NEx = no exercise; BC = blackcurrant; Ex = exercise, CI = confidence interval. Units of measurement: Complex reaction time task = Reaction speed.

Table 8 provides the adjusted means, standard errors, and 95% CI's for all 4 groups. By examining the Exercise and No Exercise groups reaction time did not reach a statistically significant main effect for condition, $F(1, 72) = .821$, $p = .368$,

$\eta_p^2 = .012$. Nor was there a statistically significant interaction effect between Condition (exercise vs. no exercise) and Intervention (blackcurrant vs. placebo) after adjustment for the covariate (complex reaction time), $F < 1$. The main effect for Intervention also failed to reach statistical significance, $F < 1$. Consequently, neither hypothesis is supported by these results.

Summary

Performance across the battery of cognitive tasks appeared to remain stable with only slight increases or decreases evident in mean performance (see Table 1-8) none of these resulted in statistically significant effects. Thus, it can be summarised from these results that support cannot be provided for either of the two hypotheses being tested. Suggesting that physical fatigue did not induce effects to participants' cognitive performance. Nor, did the blackcurrant extract account for any performance increases at Time 2.

CHAPTER 6 Discussion

The present study aimed to establish whether physical fatigue would debilitate cognitive performance on a battery of executive functioning tasks. In addition, the present study aimed to examine if ingestion of a phytochemical compound, extracted from blackcurrant, would produce ameliorating effects on debilitated cognitive performance. The evidence gathered did not provide support for either of the two hypotheses being tested.

In order to investigate the aims of the present study, two questions were proposed. Firstly, does physical fatigue impair cognitive performance, and secondly, would a blackcurrant extract ameliorate the effects physical fatigue had on cognitive performance. However, before these questions can be discussed, it's necessary to ensure that the high intensity intermittent task (HIIT), completed on the Wingate ergometer, successfully induced physical fatigue. Discussion of this will be considered below.

Previously, Gibala & McGee (2008) demonstrated that HIIT was a successful time efficient alternative for inducing physical fatigue. In comparison to traditional tasks, comprised long duration and high intensity exercise. As expected, and in support of previous evidential claims (Gibala & McGee, 2008), the HIIT used in the present study was sufficient at inducing physical fatigue. Generation of physical fatigue was demonstrated through participants' self reported increased perceptions of physical fatigue at Time 2. By way of measurement, on the physical state, trait energy, and fatigue self-report scale (O'Connor, 2006). The previous research, investigating the role of fatigue upon cognitive performance, has produced multifaceted effects. Specifically, these studies have demonstrated that positive, negative and curvilinear effects can occur. Further, these multifaceted effects appear to be dependent upon the applied methodologies in this field.

The present study predicted that cognitive performance would decline after completing the HIIT regime. When compared against the control cohort, the exercising cohort would demonstrate debilitated cognitive performance at Time 2.

Whereas, the control cohort would not demonstrate any such debilitated performance. However, there was no significant difference in cognitive performance across the battery of cognitive tasks at Time 2 between the two cohorts. This evidence is suggestive that an acute generation of physical fatigue, produced from a HIIT regime, did not elicit a level of fatigue within the experimental group, which correspondingly caused an observable negative change in cognitive performance.

In spite of the null findings, regarding acute generation of physical fatigue on cognitive performance, the second hypothesis remained. Would a phytochemical compound, obtained from blackcurrant, provide influential effects on cognitive performance, for those physically fatigued? It's widely accepted that caffeine phytochemicals demonstrate ameliorating effects towards impaired cognitive performance, caused through physical fatigue states (Hogervorst et al., 2008). While receiving less attention, prior literature has demonstrated that berry fruit phytochemicals also provide ameliorating effects to cognition. Specifically, these compounds have demonstrated an ability to produce neuromodulatory actions on neural signalling. Such that, at least in part, berry fruit phytochemicals will modify interference to cellular communication interference, caused through stress responses, e.g. fatigue (Willis, Shukitt-Hale, & Joseph, 2008). Therefore, it was expected that the stress response elicited from the HIIT regime would interfere with cellular communication, resulting in debilitated cognitive performance at Time 2. It was expected that the exercising cohort whom ingested the phytochemical extract would demonstrate minimal cognitive performance decrements. Specifically, the phytochemical compounds in blackcurrant would provide ameliorating effects to any cellular interference. However, contrary to expectation, examination of the present data failed to provide support for the second hypothesis.

Inducing Physical Fatigue

There was a significant main effect for Fatigue x Condition for the HIT task. Specifically, participants' perception of fatigue was significantly increased at Time 2,

for the exercising cohort, compared with the control participants' (no exercise). Perceptions or a feeling of fatigue is a multifaceted psychological construct, indicating feelings that are experienced on both mental and physical states (Acitelli, 2012). As feelings are subjective, a self-report measure of perceived feelings of fatigue, at the two time points, was a suitable measure for the present study (O'Connor, 2006). Therefore, the present study provides additional evidence to Gibala & McGee (2008) report, that HIIT is sufficiently successful at inducing physical fatigue within participants'.

Hypothesis 1: Physical Fatigue Impairs Subsequent Cognitive Performance

The objective of investigating the first hypothesis was to establish whether exercise induced fatigue could negatively impact subsequent cognitive performance; just as cognitive fatigue can have an impact on physical performance (Bray, Graham, Ginis, & Hicks, 2012; Mehta & Agnew, 2012). It was expected that physical fatigue would have an impact on cognitive performance. Causing debilitated cognitive performance at Time 2, relative to baseline scores, following completion of the HIIT.

The predicted decline in cognitive performance was thought to occur due to a mechanism of a reduction in levels of energy that an individual would experience, following completion of the HIIT. Thus, the present study predicted that physical fatigue would infer with synaptic functioning, and communication, within the cognitive domains measured, creating deteriorations in cognitive performance. This presumption was based on the theory that the brain is competitive. As such, sustained activation of motor neurons, during exhaustive exercise, would come at a price. The price of sustained motor neuron activation, would directly impact a participants' ability to sustain neural activity involved in completing the battery of cognitive tasks administered (Dietrich & Sparling, 2004). Therefore, it was conceivable to expect that declined cognitive performance observed in the exercising condition, would be as a direct result of experienced physical fatigue.

The battery of cognitive tasks selected to measure participants' cognitive ability, following the exercising, or control task, all measured executive functioning.

Executive functioning is defined as, processes that control higher-level actions (Perner & Lang, 1999). Precisely, cognitive processes responsible for planning, inhibition, and coordination. Involving domains that are necessary for controlling action sequences in order to maintain a specified goal, without inference from distracting alternatives (MacReady et al., 2010). The mechanism of action, for the predicted debilitation to performance, was based on a theoretical cornerstone of cognitive psychology (Dietrich, 2006). Specifically, the brain has a limited amount of resources that can be utilised for cognitive functioning, when the body is concurrently utilising resources for physiological arousal (Dietrich, 2006).

Therefore, within modern brain research, the brain is conceptualised as hierarchical during periods of physical exertion. Therefore, deleterious effects to cognitive performance will occur within domains responsible for executive functioning, before deteriorations will begin to effect lower cognitive functioning domains. Theorised to occur as a direct result of the necessary resources being allocated directly towards the prioritised structures for physical activity, e.g. motor neurons and cardiovascular systems. Moreover, it has been recognised that increased fatigue states are associated with a diminished willingness to exert effort (Macrona et al., 2009), thus may result in systematic declines in cognitive performance (Tomprowski, 2003).

As it was, the current expectation, of a decline in cognitive performance, was inconsistent with the results. In general, the exercising cohort did not show any significant deterioration to cognitive performance between the two time points, or further, when compared with the control group. Any deteriorations that may have occurred was not large enough to produce significant effects overall ($\eta_p^2 = >.05$). The implications of these findings, and their relation with the methodological differences between previous research, and the present study will be discussed below.

Anecdotal evidence, of debilitating effects to cognition, as a result of acute physical fatigue, has been overwhelming (Lambourne & Tomporowski, 2010). However, previous attempts to confirm this anecdotal evidence, surrounding the exercise-cognition relationship, have been inconsistent in demonstrating the anecdotal debilitating effects (Lambourne & Tomporowski, 2010). This is not to say that such a

debilitating relationship does not exist. Rather, the potential physical fatigue has, to cause cognitive deficits, is hampered. For instance, previous research has applied a scattergun approach to investigate the exercise-cognition relationship. As such, this unsystematic methodological approach may, at least in part, contribute to the different outcomes that have been obtained. It's important that future research in this field recognises that the different methodologies applied are a critical aspect to take into account, when assessing the relationship between acute exercise and cognition. Presently, such differing methodological approaches, reduce compatibility, and further make interpretation difficult. Therefore, it was important, in the present study, to take into account the methodological differences, when drawing conclusions of the current results, with the results from previous studies.

To date, the methodological differences, attributed to the observed conflicting evidence, include the various cognitive measures applied. The fixed intensity of the exercise protocol, for example low, medium, or high intensity levels have been administered previously. Furthermore, duration of the physical exercise task has varied considerably. In addition, the exercise regime has varied between the prior studies, ranging from a running, cycling, or a resistance task. Moreover, the timeframe of cognitive measurement has differed, with previous studies either conducting cognitive testing during exercise, or following the exercising task. Further, the population's selected to assess the relationship between acute exercise and cognition has varied, predominantly using patient, or athlete based populations (Grego et al., 2005). Therefore, while the current evidence fails to support the first hypothesis, this null finding is not out of context in this area. As the present study's results can be considered consistent with some previous literature, (Audiffren et al., 2008; Tomporowski, 2003) while, inconsistent with others (Cian et al., 2000; Grego et al., 2004; Tomporowski et al., 2007).

The lack of Time x Condition interactions in the present study is inconsistent with previous reports, whereby cognitive performance did demonstrate an effect of physical fatigue (Cian et al., 2000; Grego et al., 2004; Tomporowski et al., 2007). The number of methodological differences among prior studies, and the present may contribute to the contradictory findings. Firstly, the type of cognitive tasks used to measure cognitive performance differed among the previous, and present studies. The

present study selected the battery of cognitive tasks applied, based on prior observations, of sensitivity to physical exertion effects (McMorris & Hale, 2012). However, while previous meta-analytic evidence demonstrated that these selected tasks were sensitive to physiological changes, resulting in influencing cognitive performance. The five, presently applied, cognitive tasks demonstrated no such sensitivity to an acute session of high intensity cycling.

While cognitive tests are a prominent way to evaluate cognitive performance, few human studies, in this field of physical exercise effects on cognition, have used the same cognitive tests. With the majority of previous studies adopting unsystematic approaches in regards to the selection, and application, of the cognitive measures (MacReady et al., 2010). For instance, cognitive tasks will encompass a range of cognitive domains, e.g. attention, reaction speed, working memory, inhibition control, to name a few. Some studies' exploring the effects of exercise on a singular cognitive domain will require only a single task for cognitive measurement. Whilst other studies' that aims to measure multiple cognitive domains, will require a battery of cognitive tasks.

Additionally, all cognitive measures are designed and validated in order to substantiate the cognitive domains their purported to measure. However, checks are rarely made to see if two different measures of a particular cognitive domain correlate with one another (MacReady et al., 2010). Furthermore as the majority of cognitive measures have no physical anchor, it's unclear how to equate these various cognitive tasks (Spencer et al., 2011). Its possible that a particular cognitive task, designed to measure performance within a primary cognitive domain, may also tap into or influence a secondary cognitive domain (Blundell, Burley, Cotton, & Lawton, 2000, MacReady et al., 2010). Therefore, its difficult to determine if the subsequent effects induced from physical fatigue, are specifically associated with the changes that occur within the cognitive domain, for each participant tested. Consequently, this still remains to be a limiting factor within exercise-cognition literature. Further limiting interpretation of the present results, in comparison to previous evidence, is the fact that cognitive measures are not assessed regarding their sensitivity to measuring performance, while an individual is experiencing acute physical fatigue. Thus, its plausible, that the observed lack of correspondence between previous studies reported

findings, may be a result of different cognitive tasks lacking sensitivity for different physiological states, e.g. physiological fatigue.

To elaborate further, Tomporowski and colleagues (2005) demonstrated that cycling for two hours, causing physiological fatigue, produced a facilitatory effect to participants' performance on a Stroop task. Similarly, Ferris et al. (2007) observed improved performance on the Stroop colour word task, following a graded cycling task, and an endurance cycle on an ergometer. Contrastingly, Griffen et al. (2009) reported that a graded cycling task, performed until participants' reached volitional exhaustion, did not provide demonstration of changes to performance on the Stroop word-color task. A graded cycling exercise task is similar to the HIIT employed in the current study.

Furthermore, cognitive performance on tasks such as digit span backwards (Shaw, 1956), and digit symbol association (Burgess & Hokanson, 1964), failed to show any debilitated task performance, following the intensive cycling task in the present study. This result is consistent with an earlier study by Gutin & DiGennaro (1968a), who concurrently measured cognitive performance, while participants completed an exhausting treadmill run. The authors reported no significant effect on mental arithmetic task performance. In contrast to this report, a later study, by the same authors (1968b), did reveal impaired performance on a simple arithmetic task. However the later study did employ a different exercise task. Participants' completed either a 1 min or a 5 min step-up task. However, this observed significant difference, was only evident in the unfit cohort, which completed a 1 min bout of step-ups. No difference to performance was observed for the control or trained athletes. Therefore, it may be speculated that physical fitness may have also accounted for the lack of evidential findings in cognitive performance following an acute bout of exhaustive exercise. Physiological mechanisms are transient, and impacted by exercise intensity, cognitive assessment timing and physiological fitness. In future, understanding these transient physiological mechanisms will allow for a better understanding of the influential relationship physiological arousal has on cognitive functioning (Chang et al., 2012). Therefore, the present study observed that an acute bout of exhaustive exercise did not appear to impair cognitive domains responsible for working memory,

response inhibition, and selective attention in the current study. Despite such deleterious effects been demonstrated in previous studies.

Secondly, as demonstrated earlier, physical fitness level represents another moderating factor, that could have, at least in part, contributed to the negative outcomes that were presently observed. Leading authors in this field have proposed an inverted-U shape, in regards to the exercise-cognition relationship (Yerkes-Dodson, 1908). With superior performance being observed under submaximal arousal, whereas negative performance is observed during minimal or maximal arousal. Accordingly, direct measurement of the brain's electrical activity, event related brain potential's (ERPs), during periods of physical activity, corroborated the inverted U-shaped curve (Budde et al., 2012).

ERP amplitudes represent basic aspects of cognitive processing; lower ERP values represent a reduction to cognitive processing in the CNS, while higher values represent enhanced cognitive processing. Consequently, these ERP amplitudes will attenuate the body's devotion of attentional resources extended in order to complete a cognitive measure administered. Such ERP fluctuations were observed in Kamijo et al. (2004) that reported higher ERP amplitudes after moderate exercise, and lower ERP amplitudes following high intensity exercise.

However, behavioural studies have not always demonstrated supporting evidence for the inverted-U relationship. With discrepant observations of cognitive performance not always being negatively effected by a session of high intensity exercise (McMorris & Graydon, 1996). A potential explanation for such discrepancies' among studies, despite evidence demonstrating reduced ERP amplitude during maximal exercise (Kamijo et al., 2004), may be attributed to the individual characteristics of the participants tested, such as their level of physical fitness. It has been recognised that previous studies rarely take into account physical fitness levels, and sports participation of their participants, when analysing an acute exercise session on cognition (Budde et al., 2012).

Thus, individual characteristics, such as physical fitness, can be identified as playing a fundamental role, within the effects observed, from an acute exercise bout, on

cognitive performance. This potential explanation for the current results, arose from the theoretical perspective, that an increase in exercise intensity, would cause an increase in arousal (Brisswalter et al., 1997). However, the achieved level of increased arousal would differ between the diverse fitness levels within a population. Specifically, physical activity, or physical training, would create metabolic changes within the frontal cortical regions of the brain, that could have, at least in part, contribute to adaptive arousal levels in participants. For instance, regular physical training would only moderately increase arousal levels within fit subjects, at high intensity exercise. However, unfit subjects performing exercise at high intensities, would result in maximal arousal increase being achieved.

For instance, an earlier study, performed by Gutin & DiGennaro, (1968b), demonstrated that trained athletes are more capable at maintaining cognitive performance skills, during exhaustive exercise, in comparison to their unfit counterparts. For example, the study applied a step up exercising task. Individuals with low levels of physical fitness completed 1 min on the step up, while individuals with reported high levels of physical fitness were assigned to complete 5 min on the step up task. However, while the study reported no overall significant effects, of physical exercise on cognition. The low fitness cohort was, somewhat, more affected by physical exertion, in comparison to the control and high fitness level cohorts.

Prior literature has alluded that a reduction of attentional resources following high intensity exercise is responsible for negative cognitive performance effects (Kamijo et al., 2004). However, the exercise paradigms, used previously, have been criticised as being insufficient to induce high intensity outputs in participants, which subsequently have a negative influence on cognition, while taking into account individual fitness levels. Thus, owing to the fact that steady state exercise protocols do not fully account for individual fitness levels, or prior experience within particular sporting activities, e.g. a frequent runner, or cyclist. By applying an intermittent maximal intensity task, for a set amount of time ($N = <10$ min), it was hoped that the exercise intervention would provide similar physiological fatigue outcomes, regardless of individual physical fitness levels within the population tested. Since its introduction the Wingate anaerobic, high intensity intermittent test (Ayalon, Inbar, & Bar-Or, 1974), has been used extensively as a measure of high intensity interval training. Primarily, it is

considered a suitable time efficient alternative, for measuring maximal insensitive exercise. Moreover, the weighted resistance, on the Wingate anaerobic test, allows researchers to assign individual resistance weight levels. As based on a participant's individual body weight (Lunn, Zenoni, Carndall, Dress, & Berglund, 2015), thereby, providing a means of control for individual fitness levels, ensuring each participant, in the experimental condition, will reach a state of physiological fatigue.

Therefore given that intermittent exercising protocols are considered highly intensive, it was expected that following an acute session of a HIIT regime, subsequent performance scores across the battery of cognitive tasks applied would be decline (Alves et al., 2014). While, the present high intensity intermittent protocol was successful at inducing a level of physical fatigue, as evident in the increased physical fatigue scores, it was not sufficient to impose significant differences to post-task cognitive performance.

Previously, experimental interpolation and transcranial magnetic stimulators have demonstrated that a high intensity intermittent protocols will induce central fatigue (Amann & Dempsey., 2008; Gandevia. 2001) However, the current study demonstrated that while participants' experienced exercise induced fatigue, the imposed central motor drive fatigue was not adequate to produce carry over fatigue sensations, into another domain, e.g. the frontal cortex. Therefore it is possible that the present study's lack of overall difference in cognitive performance between the conditions, may be attributed to the absence of carry over effects from one domain to another (Ratlidge, 2014). Suggesting that even when individual's experience strain within a physiological domain e.g. physical, it is possible to maintain performance within cognitive domains.

Thirdly, the exercise protocols, used to induce a specific state of physiological arousal, have differed between previous studies, and the present. Such discrepancies among the applied exercising tasks may account for the contradictory findings demonstrated across the literature, regarding exercise induced physical fatigue, and its subsequent effects observed on cognitive performance. Primarily, the exercise protocols that are selected predominately include, running on a treadmill, or cycling on an ergometer. Furthermore, but to a lesser extent, resistance exercise protocols

have been applied within prior literature. While both running and cycling protocols are similar in the regard that these activities will utilise the large muscle groups. These tasks will still cause differing muscle recruitment and energy expenditure required for completion of the task (Lambourne & Tomporowski, 2010). Therefore, cycling will require less metabolic energy in comparison to running, as the body's centre of mass will be reduced in the cycling. Whereas, maintaining the vertical position for running, will demand more muscle expenditure, thus, may result in larger amounts of metabolic energy consumption (Lambourne & Tomporowski, 2010).

Therefore, it is difficult to determine if the present study's failure to produce similarly reported debilitated effects, of physical fatigue on cognitive performance, is representative of a boundary condition of physical fatigue induced from intermittent high intensity exercise. Or otherwise mirrors the idiosyncrasies of the different exercise protocols applied across the previous literature (MacReady et al., 2010). Furthermore, perhaps the present study applied a task that induced the wrong type of physical fatigue, in order to generate deleterious effects to cognitive performance. For instance, the population tested in the present study, comprised of primarily, students, workers, or young adult athletes. Thus, it may be that volunteers physiological system was accustomed to short term fatiguing situations.

Furthermore, neuroimaging studies reveal that neural activation, associated with task processing, quickly returns to baseline levels following cessation of an exercise task (Lyons et al., 2006). Therefore, in light of the present findings, it's essential to consider the possibility of recovery speed, for the exercising participants, as an explanation of the lack of support evident for the first hypothesis. Exercise-cognition theoretical models provide a framework that tentatively accounts for how exercise can induce both, positive, and deleterious effects. The majority of these theories are collated from an amalgamation of cognitive psychology and energetic methodologies. Thus, they are somewhat limited, in the sense that, they are not specific to the exercise-cognition relationship. Accordingly, any deleterious cognitive performance effects demonstrated by physically fatigued participants' may be as a result of competition. Specifically, an internal competition between motor neuron activation, and their subsequent resource needs, over prefrontal cortex neural activation, and their subsequent resource needs (Labelle et al., 2014).

For instance, the brain has a limited amount of metabolic resources, e.g. glucose, and oxygen. High levels of exercise will strain individual's metabolic resource stores, causing a reduction in ability to exert effort upon task relevant cues. Recent near infrared spectroscopy studies generally support the proposed loss of metabolic resources, as caused by intense exercise. Through revealing a decrease in oxygenation within the prefrontal lobes of the brain (Ekkekakis. 2009). However, it has been stated that replenishment of resources is quick, especially for physically fit individuals. For instance Brisswalter et al. (1997) demonstrated that well trained athletes are better fortified in maintaining cognitive skills during fatigue, than less fit counterparts. Further, Zervas et al. (1991) also observed an interaction between previous physical activity participation, and acute exercise effects on cognitive performance. In this longitudinal twin study, on young individuals, significant differences were observed between the groups that completed a physical training program for 6-months, and those that did not complete the 6-month training program. Specifically, the exercising cohort revealed better performance at post-task cognitive measure, than the control cohort following a high intensity treadmill run (N= 20 min). As such the failure of the current study to support our proposal of decreased cognitive functioning could be attributed to a quick recovery of resources following exercise. In future, it would be beneficial to measure cognitive functioning following long-term physical fatigue, e.g. marathon runners, or truck drivers level of concentration over long haul multiple trips.

The fourth major methodological difference among previous studies, and the present, is the timing of cognitive testing. Specifically, cognitive measures have been applied, in either a dual-task protocol, concurrently completing the exercise task and testing cognitive performance. Or following the exercising task. Previously, Lambourne & Tomporowski (2010) reported that larger effect sizes have been demonstrated in studies that apply post-exercise testing, in comparison to the effect sizes observed in dual-task protocols. In spite of this, dual-task protocols are extensively measured in prior literature. Investigation is necessary to determine whether exercise induced fatigue will result in debilitation, or facilitation, to cognitive performance. Further, it necessary to establish a time point whereby physical fatigue will generate either facilitation, or debilitation to cognitive performance. Unfortunately, there is no singular unifying pattern of facilitative or debilitative effects of physical exercise

within particular time frames of exercise cessation and length of time on the exercising task. However, there is tentative consensus in prior literature that post-exercise methodologies reveal facilitation to performance when measured at least 20 mins after exercise cessation. But, dual-task methodologies revealed negative effects on cognition within the first 20 mins of exercise (Lambourne & Tomporowski, 2010). Further understanding, of the mechanisms involved in the exercise-cognition relationship, will ensure future research reaches a clarified consensus of the influential relationship physiological arousal may impose on cognitive functioning (Chang et al., 2012).

Another possible explanation for the lack of observed effects may be that participants “expectancy ideas” played a role. Specifically, positive impacts from regular exercise on cognition, and physical health, are a well-accepted phenomenon. Thus, participants’ may have compensated effort, in order to produce what they believed to be the expected result. Furthermore, Kahneman (1973) postulated that task attractiveness would, to some degree, account for cognitive performance, following an exercise protocol. Specifically, if an individual considered a task as challenging, he/she would exert more attentional resources towards task completion. Thus, causing no exercise effects to be observed on cognitive performance. It has been suggested that, exercise protocols performed at maximal intensity levels, causing a state physiological fatigue, theoretically, would produce neural noise, and metabolic stress, that would cause interference with neuronal communication (Dietrich & Sparling, 2004). Neural noise refers to the large cascading release of hormones, and synaptic firing, in response to intense physical activity. Suggesting that such increased neural noise effects would play a role in debilitating cognitive performance (Cooper, 1973). However, given the highly challenging nature of the cognitive tasks in the present study, it is possible that despite distractibility from internal cues, the cognitive tasks were challenging enough for participants to exert additional attentional resources for task performance. Explaining the lack of exercise effects observed on subsequent cognitive performance (Bisswaller et al., 2002; Kahneman, 1973).

Future studies should aim towards verifying how neuropsychological tasks that measure different cognitive domains are affected by acute sessions of high intensity exercise within different populations with varying physical activity levels. As

individuals with some degree of physical fitness, may have superior adaption abilities in order to manage effects physically fatigue generates. Its possible the present study's results may be confounded by the number of active participants involved, despite attempts to control for differing individual fitness ability. The current study recruited volunteers through posting advertising flyers within Massey University, and Plant and Food Research. Additionally participants' volunteered after hearing about the study through word of mouth, from other participants'.

The recruitment flyer consisted of picture of an individual cycling, and a detailed list of participation expectations. Such as, 30 second bouts of high intensity cycling, cognitive brain game tasks, small donations of blood, and consumption of a gelatine capsule containing a fruit extract (see Appendix D). Such an advertisement may have encouraged individuals' of particularly active lifestyles to volunteer, above and beyond non-active volunteers. That may in turn, have contributed towards discouraging volunteers that were aware of their lower level of fitness. Therefore, the present study's null results provide a further basis for the argument, that an interaction relationship exists between past physical activities experience, and acute exercise effects on cognitive performance. Thus, the number of active participants' in the present study may have outnumbered any non-active participants, potentially masking the effects physical fatigue has on cognitive performance.

Moving forward in this area of study, researchers must recognise that the relationship between exercise, and cognition, will demonstrate negative, positive, or curvilinear effects (Chang et al., 2012). Further, the clearest conception that has emerged from assessing the exercise-cognition relationship is that the relationship is complex (Gutin, 1973). The surrounding contradictory evidence in this field is considered to owe from multiple moderating factors, as discussed above. Thus, the number of methodological differences among previous studies, and the present study, make it difficult to determine whether the current lack of physical fatigue effects on cognitive performance are specific to the particular cognitive measures selected, the intensity and exercise regime selected, the participant population (Chang et al., 2012; Tomporowski, 2003). Despite the methodological differences, the previous evidence reported is still valuable with interpretation of the presently attained results.

In summary, the results of the current study failed to support the hypothesis that physically fatiguing exercise task would cause deteriorations to subsequent cognitive performance. The possible explanations for the lack of effect can be summarised as thus; the HIIT task was not sufficient to induce physiological fatigue sufficient to carryover to cognitive functioning domains. The cognitive tasks were challenging, and thus, participants' states of fatigue weren't enough curb additional attentional resources being directed towards task processing. Recovery time between cessation of exercise, and commencement of cognitive tasks was enough to replenish depleted resources. Further, the population sample was predominantly physically active individuals, which have better coping strategies with fatigued states than sedentary counterparts. Finally despite large effect sizes reported in an earlier meta-analysis (McMorris & Hale, 2012), the cognitive measures employed were not sensitive to register fatiguing exercise effects.

Hypothesis 2: Blackcurrant Supplement Ameliorates the Effects of Fatigue on Cognitive Performance

The main research question of the present study was whether an acute dose of blackcurrant supplement could reduce the effects physical fatigue on cognitive performance. Previous research produced a mixed, but somewhat significant body of research regarding the effects of a dietary intervention on cognitive performance. Overall the general consensus from dietary studies reveals that phytochemicals play a role in both cognitive and physical performance (Chung et al., 2012). For instance, prior literature revealed that phytochemicals have an ability to directly exert, positive influences through neuromodulation and neuroprotection in the brain (Kirkorian, Shilder et al., 2010; Miller & Shukitt-Hale, 2012; Papandreou et al., 2009; Shukitt-Hale et al., 2005; Sokolov et al., 2013). Primarily, dietary intervention studies investigated the effects phytochemicals have on physical performance (McLeay et al., 2012; Malaguti et al., 2013) or separately on cognitive performance (Hoyland et al., 2008; Kirkorian et al., 2010; Kirkorian, Shidler et al., 2010). Thus, the present study may be one of the first studies to investigate a phytochemical extract's ability to provide ameliorating effects from an acute generation of physical fatigue, whilst simultaneously investigating the extracts ability to influence cognitive performance. Therefore, based on consensus drawn from previous literary evidence, it was

anticipated that the present study would reveal ameliorated performance to cognition following acute generation of physical fatigue, in comparison to those that received the placebo before being physically fatigued.

It was anticipated that an acute dose of blackcurrant would act in one of two ways: firstly, through prevention of physical fatigue in those assigned to the exercising condition, and secondly, the blackcurrant would act as a barrier to any exercise induced effects on cognitive performance. However, while the exercising task revealed a successful generation of acute physiological fatigue, cognitive performance did not result in any observable declined performance, as a result of a physically fatigued state. Therefore, the modality of anticipated fatigue effects was not supported. The performance means differed only slightly between the experimental conditions and the intervention groups. These miniscule differences were not large enough to cause any significant effects for Cognitive performance x Condition x Intervention. Suggestive, that the blackcurrant extract did not reduce fatigue effects for participants' to any greater degree than participants' who received the placebo. Furthermore, the blackcurrant extract did not influence cognitive performance within either intervention groups, both revealing similar time related changes in cognitive performance from baseline to post-task measurement. Given these lack of effects relating to physical fatigue debilitating cognitive performance at Time 2, it is difficult to draw generalised conclusions regarding the effects of an acute dose of blackcurrant extract. It may be that had participants' cognitive performance been impaired, following the prescribed HIIT the effects from the blackcurrant extract would have been greater than what was observed.

In spite of the failure to support the second hypothesis, there are a number of possible explanations as to why the blackcurrant extract had no significant effects in the present study. For instance, just as sensitivity issues were important in regards the lack of support for the first hypothesis, sensitivity issues are also relevant to mention here. Particularly, these sensitivity issues once again refer to the cognitive tasks administered within previous studies, in relation to the present study. Not surprisingly there has been little correspondence between prior studies. Cognition functioning is vast, and thus, cognitive tasks mirror the vastness of cognitive domains. Therefore, in order for characterisation of dietary supplementation effects on human cognition,

researchers have needed to utilise a wide range of tasks in order to establish a comprehensive picture of cognitive ability, following ingestion of a dietary supplementation (Hoyland et al., 2008).

Previous research has revealed that phytochemicals are capable to crossing the BBB and accumulating directly within a range of regions in the brain (Nehlig, 2003). The direct association between phytochemicals and the brain is considered to be the manner through which phytochemicals will influence physical and cognitive performance (Papandreou et al., 2009; Spencer et al., 2009). However, at present the direct mechanisms of causal actions of phytochemicals influence upon cognitive and physical performance remains unclear.

In light of this, it is possible that the blackcurrant extract used in the present study did not exploit these particular cognitive tasks domain function. Therefore, the lack of observable effects at present may be as a result of a lack of sensitivity for these cognitive tasks applied, and not due to a true absence of effect. Consequently, the present findings emphasise an important need for future studies to isolate the cognitive tasks that are sensitive to phytochemical manipulations. Furthermore, an assumption of a one to one correspondence between the different cognitive measures and the cognitive domain being assessed will continue to generate contradictory reports of conflicting evidence. Thus, a manner in which to equate different tests of a particular cognitive domain needs to be established (Hoyland et al., 2008; MacReady et al., 2010; Waters & Caplan, 2003).

The dose of the blackcurrant supplement used here may also play a contributing role to the currant null findings. It's well known that foods demonstrate differing time course effects on cognitive performance, emphasising the need for dose response curves to be considered (Hoyland et al., 2008). Previous research has demonstrated significant effects from phytochemical consumption and performance, in both physical and cognitive domains. However, predominantly these studies are constructed as longitudinal and chronic supplementation of phytochemical compounds. The majority of which will supply subjects with phytochemical supplements from 7 days to 4 months (Papandreou et al., 2009; Katz et al., 2011; Miller & Shukitt-Hale, 2012; Shukitt-Hale et al., 2005). However, with limited

studies investigating the immediate actions of phytochemicals on cognitive and physical performance, gaps still remain within the literature (Sokolov et al., 2013). Shukitt-Hale et al. (2006) emphasised the importance of the dose-response curve with differing food sources of phytochemicals. For instance, consumption of a 10% concentrated grape juice successfully reduced cognitive performance decrements, however, in order to observe an effect for physical performance, participants' needed to consume a 50% grape juice supplemented diet. Additionally, Ratlidge (2014) revealed that a dose of 3.2mg/kg of body weight was not sufficient to produce and effect of motor performance changes resulting from cognitive fatigue. Thus, while published data reveal that phytochemical compounds, applied in either whole extract, or pure form clearly result in antioxidant activity in vitro (Ghosh & Konishi, 2007). The dose-response curve status quo currently indicates a gap in the literature. Representative of an area in need of clarification, in order to determine a consensus regarding the role phytochemical compounds play in overall cognitive performance evolution.

Furthermore, it is noteworthy to mention that sex differences in the actions of long term and acute ingestion of phytochemical remain largely unknown (Sokolov et al., 2013). These undetermined sex difference effects may have been a contributing factor in the present lack of significant findings. Previously, it has been reported that sex related variability e.g. female physiology and distinct menstrual cycle phases, could be a leading cause for observed lack of significant effects (Sokolov et al., 2013). While sex differences within the population tested in the present study was not overly disproportionate, there was overall more females. Thus, sex differences within the population tested may, in part, account for the lack of support achieved for the second hypothesis. In future it is important that research investigates any potential sex related differences, as menstrual cycles and menopausal phases may add to the already contradictory field of data.

In summary, the present study failed to support the hypothesis that blackcurrant supplement would ameliorate physical fatigue effects on subsequent measures of cognitive performance. Therefore, overall it was concluded that the blackcurrant extract provided did not generate any effects on cognitive performance within either of the two conditions (exercise vs. no exercise). Interpretation of the observed results

presented difficulties as the present study demonstrated no observable effects of physical fatigue on cognitive performance. Nonetheless, potential explanations for the lack of significant findings were warranted. These can be summed, as thus, firstly, the cognitive tasks employed may not have been sensitive to identify effects of the blackcurrant extract; specifically the cognitive domain influenced by the blackcurrant extract was not the particular domain that was involved for the processing of the applied cognitive task. Secondly, the dose of the extract was not sensitive enough to produce observable effects, and thirdly, sex related differences not yet determined, may have accounted for the lack of significant effects.

Limitations

The present study appears to be one of the first to investigate the potential ameliorating effects a phytochemical extract may have on cognitive performance while under a physiologically fatigued state. Therefore, it can be said that this study's methodology reflected an exploratory method in order to determine if an acute generation of physical fatigue would cause decrements to cognitive performance. Furthermore, whether an acute ingestion of a blackcurrant extract would provide ameliorating effects to cognitive functioning in aversive conditions, specifically, physical fatigue. Thus, given its explorative nature, a discussion of the limitations surrounding the present study that may have affected the findings and generalizability of the findings are warranted.

The small sample size used in this study appears to be a major contributing factor in the results achieved. A third of the studies that have been reviewed in this field cite sample size limitations in producing adequate statistical power (MacReady et al., 2010). For the majority of the analyses conducted to assess the effects, minimal to no effects were observed upon cognitive performance, any differences weren't of a sufficient size to produce statistical significance. A larger sample size would have likely lead to some significant interactions between Condition and Time for two of the cognitive tasks. Specifically, the Digit Span Backwards and the Digit Symbol Substitution. However, the marginal means plot, revealed these interactions as disordinal. Indicating that one of the factors had one kind of effect in one condition, and

a different kind of effect in the other condition. Thus, while there is evidence to suggest that physical fatigue is capable of impairing performance on some cognitive domains, clarity regarding the factors interacting remained elusive. Had physical fatigue effects been demonstrated to a greater extent, then it is feasible that any blackcurrant supplement induced effects would have been observed.

Self-report measures reliability and validity is a long recognised issue in psychological and behavioural studies (Westerterp, 2009). Thus, a potential limitation to the present study was the fact that a self-report measure of physical activity was used in order to assess physical activity levels within individuals. While such measures can be adequately applied as a manner of ranking activity within individuals, Westerterp (2009) demonstrated that such activity ranking instruments, possess low reliability and validity of physiological fitness compared to other physiological based measures. Despite this limitation, these measures are easily accessible, and quickly applied, without the need for any additional medical training. Therefore, because the present study used a self-report measure of physical activity levels, instead of a physiological measure, with an additional bias potential from a prevalently high number of physical active participants. It could be speculated that participants' had higher levels of physical activity and greater experience in motor coordination than what was reported, and thus, were more capable of maintaining cognitive skills during fatigue than their less fit counterparts. Therefore, despite volunteers being randomly assigned to the exercising or resting Conditions, it may be that this factor resulted in a lack of power for the study to detect negative effects, thus masking the results. Future studies should combine physiological measures of physical fitness when appropriate and feasible to do so, instead of selecting easily accessible self reported questionnaires.

The difficulty in equating cognitive measures sensitivity to assessment of various cognitive domains and their ability to measure cognitive change within aversive circumstances, such as physical fatigue makes comparability within the literature elusive, owing primarily to the range of cognitive measures that have been used previously. Against which the present study sought to account for this limitation by selecting cognitive measures that have been used previously, and reportedly demonstrated effects regarding physiological arousal effects on cognitive

performance (McMorris et al., 2005). It may be that the present failure to detect an effect of cognitive deficit from physical fatigue, or performance facilitation from the blackcurrant extract, represents a lack of sensitivity of the test, and not a true absence of effect (MacReady et al., 2010). This limitation could be overcome in future by developing a better understanding of the conditions under which a particular physiological state, and phytochemical extract does or does not derive positive, negative, or curvilinear effects to cognitive performance.

Participants' were required to abstain from a range of food items, that either contained high levels of phytochemicals, or caffeine, for 2 days prior to testing. However, there was no way to ensure that participants complied with this prerequisite. It is likely that some participants' may have slipped in this regard. Completing the laboratory exercise having consumed restricted food items, failing to disclose the information to researchers' may have masked the results. As was previously demonstrated, the foods' that are consumed by an individual will have carry over effects in the transfer of energy from foods to neurons (Gómez-Pinilla, 2008). Thus, any foods consumed that are recognised for their ability to affect physical and cognitive performance before measurement, will persistently continue to bias the evidence reported if adequate control measures are not included into the research methodologies. While the present study completed verbal compliance checks before completion of the trial, it is advisable that future research attempt stricture protocols regarding compliance of restricted food consumption.

Conclusion

The present study investigated the influence a physically fatigued state had on decreasing participants' ability to complete a battery of cognitive tasks. Previous anecdotal and clinical studies have identified that high intensity exercise, completed till exhaustion can impair executive cognitive functioning (Alves et al., 2014; Gutin & DiGennaro, 1968; Lambourne & Tomporowski, 2010; Meeusen et al., 2007). As well, nutritional research on phytochemical compounds suggests that phytochemicals may improve cell signalling that in turn will enhance neuroprotection from increased antioxidant properties, leading to improved cognitive ability (Dye et al., 2000;

Hoyland et al., 2008; MacReady et al., 2010; Panandreu et al., 2009; Spencer, 2010). The present study appears to be one of the first to integrate the two and investigate any interaction between phytochemicals and fatigue related declines on cognition. The fast-evolving technological world of today generates environmental conditions that are complex and challenging. Thus, given these physically demanding environments we are living in, and the potential effects arising from fatigue, research that examines aversive circumstances such as fatigue is directly applicable.

With the current available evidence it is difficult to determine whether physical arousal, to the point of fatigue, coupled with phytochemical extract effects cognitive performance to any significant effect. The results of the present study are inconsistent with previous findings, with physical fatigue having demonstrating no significant effects on subsequent cognitive performance. This observation made the interpretation of potential influences from the blackcurrant extract difficult; however it appeared that the supplement had little, to no ameliorating effects on cognitive performance. Despite contradictory findings between previous research and the present study, important issues' regarding future research was identified and needs addressing when moving forward with research of this nature. The results indicate that methodology is a critical aspect to take into account when assessing the outcomes of such research. The vast range of cognitive domains and physical states required to be measured, in order to characterise the effects of phytochemical compounds on cognitive and physical performance, has resulted in researchers applying a scattergun approach across studies. Cognitive tasks that are not sensitive to cognitive change in response to physiological exhaustion or phytochemical extract influences, may mask any observable effects achieved. In addition, physical exercising tasks, not suitable for inducing a suitable state of physiological arousal may also result in ambiguous evidence reporting.

Currently research is spread thin across the various cognitive and physical domains, causing a lack of correspondence among studies. Limitations of the study included the use of a small sample population, sensitivity issues' regarding cognitive tasks applied and the breadth of these cognitive measures used, physical activity self report measures, inadequate control measures of restricted food intake before measurement, and physical fitness differences within the population tested. Finally, if time had

permitted it would have been interesting to increase the sample size, including a larger variety of physical fitness ability. If by increasing the sample size and addressing the reported limitations mentioned, future research may be able to clarify if a blackcurrant extract successfully ameliorates the effects of physiological fatigue on cognitive performance. This may prove influential in understanding the influential relationship between nutrition, physical activities, and overall performance abilities. If such findings were confirmed in populations, it could prove influential, not just for cognitive enhancing purposes, but for patient populations, and individuals' involved in physically demanding situations that requires a high level of cognitive output. This may result in improvements in performances within the workplace, maintaining attention in learning environments, and reduce the number of accidents caused by fatigue.

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Appendices

Appendix A: Advertisement Flyer

VOLUNTEERS WANTED

Plant and Food Research are conducting a study to evaluate the effect of fruit supplement on exercise and cognitive performance

Participants will be required to take part in:

- (a) Computerised “Brain game” tasks
- (b) Short (30 sec) High Intensity cycle tests
- (c) Consumption of a single gelatine capsule containing fruit supplement

The study will take a few hours over 2 days (max 1 hr on first day).

Small donations of blood (ear pricks) will be taken at set periods

Participants will be reimbursed for their time

**For more information please contact
Unita Harold on [REDACTED], or email
Unita.Harold@[REDACTED].co.nz**

Appendix B: Mental and Physical State and Trait Energy and Fatigue Scales

Part I - How you feel right now.

Directions. This part of the questionnaire asks about your current feelings of energy and fatigue. We are interested in how you feel right now, even if it is different than how you usually feel. Therefore, it is important that you focus on how you feel right now at this moment in responding to each item. There are no right or wrong answers. Please be as honest and accurate as possible in your responses. Make a vertical line through each horizontal line below to indicate the intensity of your current feelings. If you have a complete absence of the feeling described then place a vertical mark at the left edge of the horizontal line. If your feelings are the strongest intensity that you have ever experienced then place a vertical mark at the right edge of the horizontal line. If your feelings are between these two extremes, then use the distance from the left edge to represent the intensity of your feelings.

Example:

I feel I have no energy

Strongest feelings of
energy ever felt

How do you feel right now with regard to your capacity to perform your typical *PHYSICAL ACTIVITIES*....

Subject Number

1. I feel I have no energy
energy ever felt

Strongest feelings of

107

2. I feel no fatigue

Strongest feelings of fatigue ever felt

3. I feel I have no vigor

Strongest feelings of
vigor ever felt

4. I feel no exhaustion

Strongest feelings of
exhaustion ever felt

5. I feel I have no pep

Strongest feelings of
pep ever felt

6. I have no feelings of being worn out

Strongest feelings of
being worn out ever
felt

**How do you feel right now with regard to your capacity to perform
your typical *MENTAL ACTIVITIES*....**

7. I feel I have no energy

Strongest feelings of
energy ever felt

8. I feel no fatigue

Strongest feelings of
fatigue ever felt

9. I feel I have no vigor

Strongest feelings of
vigor ever felt

10. I feel no exhaustion ever felt

Strongest feelings of
exhaustion

11. I feel I have no pep ever felt

Strongest feelings of
pep

12. I have no feelings of being worn out

Strongest feelings of
being worn out ever
felt

Part II - How you usually feel.

Directions. This part of the questionnaire asks about how you usually feel. Therefore, it is important that you focus on how you usually feel in responding to each item. There are no right or wrong answers. Please be as honest and accurate as possible in your responses. Circle the response that best represents how you usually feel.

With regard to your capacity to perform **PHYSICAL ACTIVITIES** how often do you usually feel....

13. ENERGETIC

never

a little bit of the time

sometimes

most of the time

always

14. FATIGUED

never

a little bit of the time

sometimes

most of the time

always

15. VIGOROUS

never

a little bit of the time

sometimes

most of the time

always

16. EXHAUSTED

never

a little bit of the time

sometimes

most of the time

always

17. FULL OF PEP

never

a little bit of the time

sometimes

most of the time

always

18. WORN OUT

never

a little bit of the time

sometimes

most of the time

always

With regard to your capacity to perform *MENTAL ACTIVITIES* how often do you usually feel.....

19. ENERGETIC

never

a little bit of the time

sometimes

most of the time

always

20. FATIGUED

never

a little bit of the time

sometimes

most of the time

always

21. VIGOROUS

never

a little bit of the time

sometimes

most of the time

always

22. EXHAUSTED

never

a little bit of the time

sometimes

most of the time

always

23. FULL OF PEP

never

a little bit of the time

sometimes

most of the time

always

24. WORN OUT

never

a little bit of the time

sometimes

most of the time

always

Appendix C: Health Screening Form

HEALTH SCREENING FORM (Version 1, 05/10/2013)

Personal details

Name: _____

Age: _____

Contact details _____ : _____

Emergency contact

Name: _____

Contact details _____ : _____

Family doctor

Name: _____

Contact details _____ : _____

Health history

Have you or anyone in your family ever experienced any of the following (tick for yes)?

- High blood pressure
- Low blood pressure
- Heart problems
- Stroke
- Breathing problems
- Cancer or tumours
- Asthma
- Diabetes
- Epilepsy
- Arthritis
- Kidney/bladder disorders
- Stomach disorders

- 🍏 Hernia
- 🍏 Allergies/asthma
- 🍏 Blood disorder/diseases e.g. hepatitis
- 🍏 Chronic conditions e.g. lupus, arthritis
- 🍏 Other (please identify) _____

If yes to any of the above, please explain:

Are you currently pregnant (or planning)?

Are you currently taking any medication and/or dietary supplements (what/why)?

Do you any have food sensitivities/allergies (if yes, please give details)?

Do you smoke (how many/day)?

Do you drink (how much, frequency)?

Exercise history

Do you currently exercise or participate in any sport? _____

Details (frequency, duration, type, intensity):

Have you ever experienced any injuries or on-going pain (where, when, how bad)?

Working computers.

Do you suffer with headaches, migraines? If so when and how bad?

Do you suffer with any visual disturbances (i.e. flashing lights) when using working with computers? If so when and how bad?

Do have problems distinguishing between different colours (i.e. colour blindness)? If so which colours do you particularly have problems with?

Is there any other information, not discussed, that you feel is relevant?

I (print name) _____ have given true and complete information to the best of my knowledge.

Signature: _____ Date: _____

Researcher: _____ Date: _____

Appendix D: Information Sheet

Information Sheet (*Version 1, 21/07/2014*)

Title of Project:	Effect of blackcurrant consumption and physical fatigue on cognitive performance
Locality:	Plant and Food Research, Food Industry Science Centre, Fitzherbert Science Centre, Batchelar Road Palmerston North 4474 Phone: 06 953 7700
Lead Investigator	Dr Suzanne Hurst
Trial co-ordinators	Dr Dominic Lomiwes (contact person) and Kirsty Lyall
Co-investigators	Assoc. Prof. John Podd, Unita Harold (MSc student), Prof. Roger Hurst

Introduction

You are invited to take part in a health research study that looks at how acute physical fatigue and / or taking a blackcurrant extract affect mental processing.

This participation information sheet will help you decide if you would like to take part. It tells you why we are doing the study, what you need to do, and what the benefits and possible risks to you might be.

We will go through the form (**there are 8 pages**) with you and answer any questions you may have, this will take about 10-15 mins. You may also like to take away the form to talk through the study with family, whānau, friends, or your general medical practitioner.

Please make sure you have read all the pages. If you require an interpreter, this can be arranged.

You have a period of 2 weeks to make a decision on whether you wish to take part (participate).

Taking part in this study is entirely your choice. If you agree to take part, you may withdraw from the study at any stage, without giving a reason.

If you agree to take part in this study, you will be asked to sign the consent form at the back of this document. You will also be given a copy of both the participant information sheet and the consent form to keep.

Free parking is available at the facility.

Background and purpose of this research

Moderate exercise, such as a 30 min walk, has been shown to maintain mental activity (i.e. cognitive performance). However, physical exercise causing fatigue and oxidative stress appears to have the opposite effect and actually impairs mental activity.

Regular consumption of fruit, especially berryfruit, has been shown to boost cognitive performance and maintain mental health. Furthermore, since eating blackcurrants also reduces exercise-induced oxidative stress, it is possible that eating blackcurrants before doing a fatiguing -type exercise will prevent a decrease in cognitive performance.

In this study, we look at the relationship between exercise-induced physical fatigue and consumption of a blackcurrant supplement on the ability to carry out mental tasks.

How will participants be recruited?

As a participant (individuals who agree to take part), you will be recruited through flyers posted at Plant and Food Research and Massey University, Palmerston North, or at specific sites throughout Palmerston North: local gyms, AgResearch and Fonterra.

Interested individuals will be contacted by the trial coordinator (Dominic Lomiwes) to make sure they meet study criteria.

If you are a healthy individual, (any gender) aged between 19 and 50 years old, you may take part in the study.

You will not be considered for this study if you have blood-borne diseases (such as hepatitis), clinically diagnosed high/low blood pressure, are pregnant or planning to get pregnant, have had a recent bacterial or viral illness, or take medication for blood disorders, e.g. anti-clotting medication.

You will not be able to take part if you have a health conditions that affect your ability to perform the exercise task required for this study (this includes problems with your heart or breathing during exercise, joint or back pain, a hernia or injury/recovery from injury).

Furthermore, since this study involves doing computer “brain games” you will not be able to take part if you have any of the following health concerns: colour blindness, migraines or epilepsy brought on by rapid moving computer images.

In addition, if you have intolerance, sensitivity or allergy to berryfruits or berry fruit products you will be unable to take part in stage 3 of this study.

You will be asked to complete a health questionnaire. The completed questionnaire is passed onto an independent health advisor who will assess your suitability to take part in this study. None of the investigators in this study will see your health questionnaire and if requested, it will be returned to you at the end of the study.

You may also like to seek advice from your general medical practitioner or primary care individual if you have any concerns about taking part in this study; however, this will be at your own expense.

What happens in this research?

This study aims to look at the relationship between physical fatigue and consumption of a blackcurrant extract on mental health. You will initially be asked to attend a familiarization session, where you will have your fitness assessed, do a series bicycle exercises that cause physical fatigue and be shown how to complete a computer “brain game” task. This should take about 45 mins.

In the main trial, you will be asked to initially fill out some forms and questionnaires that ask you about how you feel, followed by a computer “brain games” task and a cycle exercise. You will then be asked take a gelatin capsule containing a fruit extract and asked relax for about 1 hr, after which you may be asked to either (i) relax for a further 10 mins or (ii) do a series of cycle exercises until you are fatigued (i.e. can’t do any more), after which you will be asked to fill in some more forms and questionnaires, repeat the computer “brain games” task and perform another cycle exercise. This main trial should take about 2½ hrs.

In addition, you will be asked to donate a small amount of blood (from an ear prick) at specific times throughout the familiarization session and main trial. Details of this and the physical and brain exercises you will be asked are described below.

Specific details of the study, and what is expected of you.

Familiarisation session (~ 45 mins).

On the morning of this familiarization session, you will be asked to come to Plant and Food Research in your gym wear. The trial coordinators (Dominic Lomiwes & Kirsty Lyall) will meet you, answer any of your concerns about the trial, and take you to the exercise gym. You will be introduced to the co-investigator (Unita Harold) who will guide you through a series of computerised brain games and allow you time to familiarise yourself with this computer task.

The trial co-ordinators will then ask you to fill out a form that will assess how much exercise you currently do and then you will be weighed. They will then show you how to use the Monark stationary bike and ask you to try it. If you feel comfortable, you will then be asked to do a short cycle. This will allow you and the trial co-

ordinators to assess whether you are capable of taking part in this study. If you still would like to proceed, the trial coordinators will put a heart monitor around your chest and ask you to do a series of short cycle exercises (~ 4), which will involve you cycling as fast as you can for 30 seconds, with a 1 minute rest in between. After each cycle exercise, you will be asked how you feel and whether you would like to continue. You will also be asked to donate a small amount of blood (taken from an ear prick) before and after each 30-second cycle exercise (4 times). At the end of this exercise session the heart monitor will be removed, and since you will probably be feeling very tired, you will be given something to drink and eat and allowed time to recover.

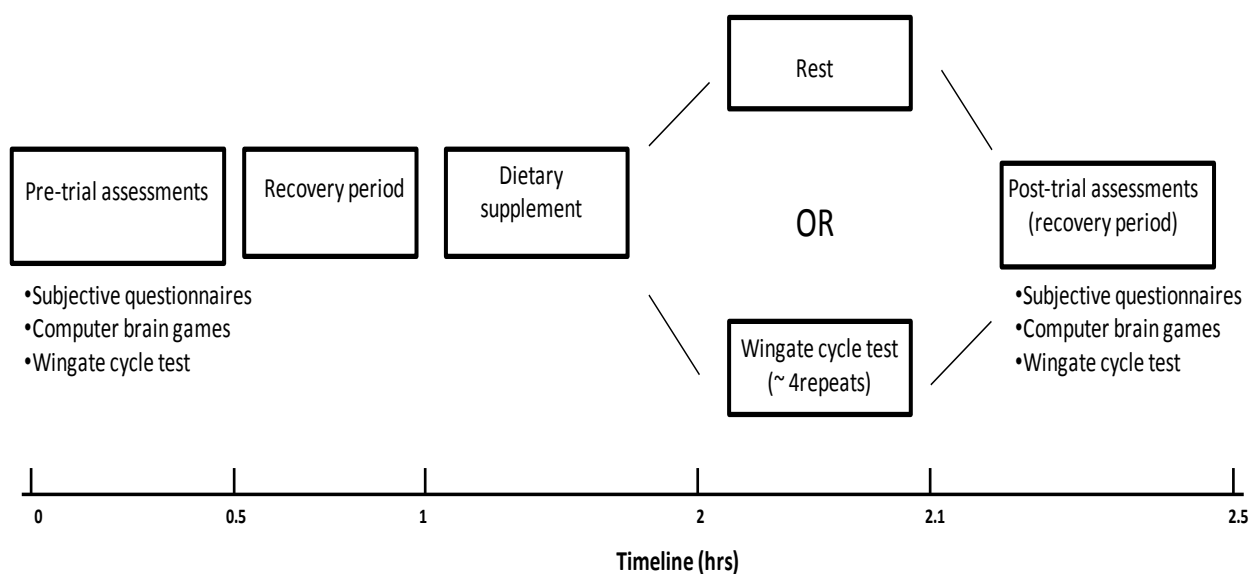
After you have recovered and before leaving, the trial coordinators will ask you how you feel and answer any questions you may have. The trial coordinators will also provide you with a list of dietary supplements and foods to avoid eating 2 days before the start of the main trial and discuss any concerns you may have about that. You will also be advised to discuss with your health practitioner about not taking any dietary supplements you regularly take. In addition, you will be asked not to do any strenuous exercise 2 days before the start of the trial.

Main trial (~ 2½ hours).

Pre-trial. The trial coordinator will be in contact with you 2 days before the start of the main trial to ask you if you would like to take part in the study and to stop eating certain foods on the list provided to you in the familiarization session. You will also be asked not to do any strenuous exercise. If you have any concerns with this or have lost the food list, the trial coordinators will provide another one for you. They will also arrange with you a time to do the main trial and ask you to eat breakfast on the day of the trial at least 1 hour before.

Day of the trial. You will be asked to come to Plant and Food Research in your gym wear and the trial coordinators will meet you, take you to the exercise gym and ask if you would like to carry on with the trial and ask whether you have had breakfast and at what time. They will also ask you if you have eaten any foods or dietary supplements on the list provided, or taken part in any strenuous exercise. If you are happy to do the trial, then the trial coordinators and co-investigators will guide you through a series of tasks.

The diagram below shows what you will be expected to do and the time it will take at each stage of the trial.



The trial coordinators will be present throughout the whole trial and will answer any concerns you may have. Before you start you will be fitted with a heart monitor, which you will be asked to wear throughout the duration of the trial. You will also be asked to donate a small amount of blood (~ 0.5 mL) from ear pricks 6 times throughout the course of the trial (a total of ~ 5mL).

At the start of the trial you will then be asked by a co-investigator (Unita Harold) to fill in some forms that ask how about how you are feeling. She will then ask you to complete a computer “brain games” task similar to the one you did in the familiarisation session. After this, the trial coordinators will ask you to donate a blood sample (ear prick) and then ask you to do a 30 second Wingate cycle test (similar to the one you did in the familiarization session) as fast as you can. You will be coached by the trial co-ordinator to complete this test. Immediately after this you will be asked to donate another blood sample (ear prick) and then asked to relax for about 30 minutes. During this period you will be taken to a separate area where you can watch a video or just relax (if you are a PFR employee, you may return to work but must not do any strenuous exercise). During this rest period you will only be allowed to drink water.

After this time, you will be asked to donate another blood sample (ear prick) and then given a small gelatine capsule to take with water. You will then be asked to rest for a further 1 hour; during this period you will be able to leave but be advised not to do any exercise and only drink water. However, if you feel you need something to eat, the trial coordinators will provide you with food. After 1 hour you will be asked to donate another blood sample (ear prick) and asked to either relax for a further 10 minutes or go to the gym where the trial coordinators will ask you to carry out an exercise involving repeated 30 second Wingate cycling tests with 1 minute resting intervals between each cycling bout. The trial co-ordinators will coach you through this and you will be asked how you feel after you finish each 30 second cycle and asked to continue until you can no longer do it or the trial coordinator tells you to stop. Since this exercise is designed to cause physical fatigue, the trial coordinators will monitor you

throughout the exercise and offer you a drink and/or glucose sweet at the end of the exercise.

After this 10-minute period (rest or exercising to fatigue) you will be asked to donate a blood again (ear prick) and fill in forms asking about how you feel. You will then be asked to immediately do a computer “brain games” task similar to the one you did at the beginning of the trial. After completing this, you will be asked to donate a final blood sample (ear prick) and to repeat the same 30 second Wingate cycle test you were asked to do at the beginning of the trial.

Upon completion, the heart monitor will be taken off and you will be offered something drink and eat and asked to relax for about 10 minutes, depending upon how you feel. Once you have recovered, you will be free to leave.

Reassurances.

On first reading, this participation information sheet may appear complicated and too much to remember. To reassure you, the trial co-ordinators will promise to do the following:

- Provide you with the necessary information for you to decide whether you would like to take part in this study.
- Keep you informed about what is expected from you 2-days before the start of the main trial (i.e. dietary omission and exercise restrictions).
- Make sure you are informed and comfortable with each step of the trial
- Monitor your wellbeing at each step of the study.
- Coach you through each of the bicycle exercise session
- Provide drink and food and an area for you to relax when required.

What are the benefits of this study?

Personal benefit. This study will provide you with an insight into how exercise, especially physical fatigue, and appropriate nutrition impacts upon your mental health and your ability to do mental tasks that are important for everyday activities such as recalling items on a list.

Scientific benefit. This study will advance the scientific knowledge underpinning the relationship between physical fatigue and nutritional intervention on cognitive performance and mental health. Findings from this study will fulfil the requirements for an MSc thesis submission (Unita Harold, Massey University) and be published in scientific journals, and presented at seminars at Massey University, Plant and Food Research, and national/international scientific conferences.

What are the discomforts and risks of this study?

Changes in Diet. You may feel unsure about what foods, drinks and supplements you are able to eat before doing this study. Moreover, you may feel uneasy about stopping taking dietary supplements or taking a fruit supplement. There is also small chance that you may experience digestive problems or a skin rash after taking the supplement.

Exercise requirements. You may feel uneasy about performing a series of cycle exercises designed to induced fatigue.

Blood sampling. You may feel uneasy about giving blood. There may be minor physical discomfort and you may experience some ear stinging after donating blood. You may also feel light-headed and dizzy after donating blood, especially after exercise to fatigue. This is not caused by blood loss from the amount of blood you will be asked to give in this study, but due to the possibility that you may feel queasy from the appearance of blood. You may also have cultural concerns about donating blood.

How will these discomforts and risks be alleviated?

Changes in Diet. As a participant, you will be reassured and advised throughout the entire study and closely monitored by the trial co-ordinator who will assist you on what foods will be OK to have during the trial period.

The blackcurrant extract you will be asked to eat will be within a gelatine capsule. The amount of fruit we are asking to take within this trial lies within the recommended daily amount advised by New Zealand dieticians. It should have no adverse effect on your health. However, if you do experience digestive and skin problems relating to taking the blackcurrant supplement then you are asked to let the trial coordinator know and consult with your general medical practitioner (GP). Consultation with your GP or other health practitioner will be at your own expense.

Exercise requirements. Before you agree to take part in the trial you will be asked to fill out a form that assess how often you exercise and taken to the gym, where you will be shown the Monark stationary bike and asked to try it out. The trial coordinators will be present to coach you through the cycling exercises throughout the familiarisation session and the main trial. If at any time during these exercises you feel uncomfortable then you do not have to continue. First Aiders will be on hand in the unlikely case of injury.

Blood sampling. To ease any concerns you may have about giving blood, the trial coordinators who hold a NZ MedLab certificate and First Aiders will be the only persons taking your blood. The blood sample we are asking you to donate is small and only involves an ear prick. Nevertheless, if you feel light headed after having blood taken you will be asked to lie down with feet slightly elevated, which will minimize the feeling of light headedness/dizziness after blood giving. This only happens with a small percentage of the population. You will also be given something to eat and drink. If you have cultural concerns about giving blood, you should talk to your whānau support before taking part in this study. We can assure you that the blood donated in this study will not be given to anyone else and although it cannot be returned to you for health and safety reasons, we will destroy it by incineration

What treatment is immediately available should an injury occur?

Trial coordinator is a First Aider and will be on hand in the gym if you hurt yourself during the exercise sessions. There are also a number of trained First Aiders based at Plant and Food Research who will be on hand to provide immediate medical treatment, if required. In the unlikelihood that you require medical attention, we will

arrange transport to the nearest hospital, medical centre or health practitioner of your request.

What are the costs involved in this research?

There is no cost to you taking part in this study, except your time.

How will my privacy be protected?

No material, which could personally identify you, will be used in any reports or on the labelling of samples. All personal records, physiological data, biological samples and cognitive performance information will be kept in a secure area at Plant and Food Research or School of Psychology, Massey University, respectively, with access limited to the principal investigator and the co-investigators named on the first page of this document.

The information gathered is confidential and will only be used for this project. Data will be stored for a maximum of 10 years and will be the responsibility of the principal investigator, after which it will be destroyed.

The blood collected in the study will not be passed on to anybody else and will be destroyed by incineration.

The health questionnaire will be assessed independently and none of the researchers in this study will have access to your personal information. At the end of the trial, the health forms will be destroyed by incineration or, if requested, returned to you.

Will I receive feedback on the results of this research?

At the end of the study, you will be invited to a seminar at Plant and Food Research, Palmerston North in which the main findings of the study will be presented.

Unfortunately, since your blood will be used in bioassay analysis, we will be unable to provide you with specific information about your personal samples. However, the principal investigator will gladly give you an overview of the main findings of the study and answer any questions you may have about this research.

Please note that a significant delay may occur between data collection and the publication of any results, which is normal.

What support can I access to better understand this research?

You may have a friend, family or whānau support to help you understand the risks and/or benefits of this study and any other explanation you may require.

Since this study requires you to take part in exercise where you are asked to donate blood, we strongly advise that you discuss this with your whānau. Your blood will ONLY be used in this study and afterwards will be destroyed by incineration.

Blood or immune cells will not be stored and will not be passed on to any other research groups.

If requested, an interpreter will be provided.

What do I do if I have concerns about this research?

If you have any queries or concerns regarding your rights as a participant in this study, you may wish to contact an independent Health and Disability Advocate at 0800 555 050 or at advocacy@hdc.org.nz.

For general information we advise participants read the ERMA document, which outlines the development of the ethics framework in New Zealand. This can be found on the ERMA Webpages. <http://www.ermanz.govt.nz/about/eap.html>

For Maori issues we advise participants consult the ERMA Webpages. <http://www.ermanz.govt.nz/resources/publications/policy/no/consultmaorihuman.html>

Employees of Plant and Food Research can also contact the General Manager for Human Resources, Craig Jensen (09 815 4200 or cjensen@hortresearch.co.nz).

Who do I contact for further information?

Dr Suzanne Hurst (Principal investigator)
Plant and Food Research Ltd.,
Palmerston North, Private Bag 11600
Palmerston North 4442
Tel. 06 355 6231
Email. Suzanne.Hurst@plantandfood.co.nz

Dr Dominic Lomiwes (Trial Coordinator)
Plant and Food Research Ltd.,
Palmerston North, Private Bag 11600
Palmerston North 4442
Tel. 06 953 6224
Email. Dominic.Lomiwes@plantandfood.co.nz

Appendix E: Baeke Habitual Physical Activity Questionnaire

The Questionnaire for Measurement of a Person's Habitual Physical Activity

Overview:

*Baeke *et al.* (1982) developed a questionnaire for evaluating a person's physical activity and separating it into three distinct dimensions. The authors were from the Netherlands.

*Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr* 1982; 36:936-42.

Indices for physical activity:

- (1) work activity
- (2) sports activity
- (3) leisure activity

Work Index

Question	Response	Points
What is your main occupation?	Low activity	1
	Moderate activity	3
	High activity	5
At work I sit	Never	1
	Seldom	2
	Sometimes	3
	Often	4
	Always	5
At work I stand	Never	1
	Seldom	2
	Sometimes	3
	Often	4
	Always	5
At work I walk	Never	1
	Seldom	2
	Sometimes	3

	Often	4
	Always	5

Question	Response	Points
At work I lift heavy loads	Never	1
	Seldom	2
	Sometimes	3
	Often	4
	Always	5
After work I am tired	Very often	5
	Often	4
	Sometimes	3
	Seldom	2
	Never	1
At work I sweat	Very often	5
	Often	4
	Sometimes	3
	Seldom	2
	Never	1
In comparison with others of my own age I think my work is physically	Much heavier	5
	Heavier	4
	As heavy	3
	Lighter	2
	Much lighter	1

Where: • The work activity is according to the Netherlands Nutrition Council with (1) low activity including clerical work, driving, shopkeeping, teaching, studying, housework, medical practice and occupations requiring a university education; (2) middle activity including factory work, plumbing, carpentry and farming; (3) high activity includes dock work, construction work and professional sport.

Work index = ((6 – (points for sitting)) + SUM (points for the other 7 parameters)) / 8

Sport Index

Question	Response	Points
Do you play sports?	Yes then calculate sport score	(see below)
	• sport score ≥ 12	5
	• sport score 8 to <12	4
	• sport score 4 to <8	3
	• sport score 0.01 to <4	2
	• sport score = 0	1
	No	1
In comparison with others of my own age I think my physical activity during leisure time is	Much more	5
	More	4
	The same	3
	Less	2
	Much less	1
During leisure time I sweat	Very often	5
	Often	4
	Sometimes	3
	Seldom	2
	Never	1
During leisure time I play sport	Never	1
	Seldom	2
	Sometimes	3
	Often	4
	Very often	5

Data on Most Frequently Played Sport	Finding	Value
What sport do you play most frequently	Low intensity	0.76
	Medium intensity	1.26
	High intensity	1.76
How many hours do you play a week?	< 1 hour	0.5
	1-2 hours	1.5
	3-4 hours	3.5
	> 4 hours	4.5
How many months do you play in a year?	< 1 month	0.04
	1-3 months	0.17
	4-6 months	0.42
	7-9 months	0.67
	> 9 months	0.92

Where: • The sport intensity is divided into 3 levels: (1) low level (billiards, sailing, bowling, golf etc) with an average energy expenditure of 0.76 MK/h; (2) middle level (badminton, cycling, dancing, swimming, tennis) with an average energy expenditure of 1.26 MJ/h; (3) high level (boxing, basketball, football, rugby, rowing) with an average energy expenditure of 1.76 MJ/h.

Data on Second Most Frequently Played Sport	Finding	Value
What sport do you play most frequently	Low intensity	0.76
	Medium intensity	1.26
	High intensity	1.76
How many hours do you play a week?	< 1 hour	0.5
	1-2 hours	1.5
	3-4 hours	3.5
	> 4 hours	4.5
How many months do you play in a year?	< 1 month	0.04
	1-3 months	0.17
	4-6 months	0.42
	7-9 months	0.67
	> 9 months	0.92

Simple sports score = ((value for intensity of most frequent sport) * (value for weekly time of most frequent sport) * (value for yearly proportion of most frequent sport)) * ((value for intensity of second sport) * (value for weekly time of second sport) * (value for yearly proportion of second sport)) Sport index = (SUM(points for all 4 parameters)) / 4

Appendix F: Rating of Perceived Exertion

Rating of perceived exertion (RPE)

Subject # _____

Date _____

Time _____

15 point scale

6 -	20% effort	
7 -	30% effort -	Very, very light (Rest)
8 -	40% effort	
9 -	50% effort -	Very light - gentle walking
10 -	55% effort	
11 -	60% effort -	Fairly light
12 -	65% effort	
13 -	70% effort -	Somewhat hard - steady
pace		
14 -	75% effort	
15 -	80% effort -	Hard
16 -	85% effort	
17 -	90% effort -	Very hard
18 -	95% effort	
19 -	100% effort -	Very, very hard
20 -	Exhaustion	

I certify that these data are complete and correct

Researcher's name: _____ **Date:** _____

Researcher's signature: _____

Appendix G: Consent Form

Consent Form (version 1, 21/07/2014)

Title of Project: Effect of blackcurrant consumption and physical fatigue on cognitive performance

Principal Investigator: Dr Suzanne Hurst

1

English	I wish to have an interpreter.	Yes	No
Maori	E hiahia ana ahau ki tetahi kaiwhakamaori/kaiwhaka pakeha korero.	Ae	Kao
Cook Island	Ka inangaro au i tetahi tangata uri reo.	Ae	Kare
Fijian	Au gadreva me dua e vakadewa vosa vei au.	Io	Sega
Niuean	Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu.	E	Nakai
Samoan	Ou te mana'o ia i ai se fa'amatala upu.	loe	Leai
Tokelaun	Ko au e fofou ki he tino ke fakaliliu te gagana Peletania ki na gagana o na motu o te Pahefika.	loe	Leai
Tongan	Oku ou fiema'u ha fakatonulea.	Io	Ikai

(circle one)

I have read and I understand the information sheet dated _____ for volunteers taking part in the study designed to assess the effects of blackcurrant consumption and physical fatigue on cognitive performance. Yes / No

I have had the opportunity to discuss this study and I am satisfied with the answers I have been given. Yes / No

I have had the opportunity to use family, whānau support or a friend to help me ask questions and understand the study. Yes / No

I have had the opportunity to discuss with my family, whānau about the use of my blood in this study and agree that it should only be used in this study and all samples be destroyed afterwards. Yes / No

I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time without giving a reason. Yes / No

- I understand that data will be stored until a maximum of 10 years and will be the responsibility of the principal investigator, after which it will be destroyed. Yes / No
- I understand that at the end of the study all blood samples will be destroyed, and health, fitness and subjective forms and questionnaires will either be destroyed or returned. Yes / No
- I understand that my health questionnaire will **only** be assessed by a medically trained employee, Julia Crosby (NZRN Comp Dip OHP, Otago) of the Waikato Occupational Health consultancy and advise me about my suitability to take part in this study. No fellow co-workers, including the principal applicant, Dr Suzanne Hurst, will see or have access to this information. Yes / No
- I understand that the investigation will be stopped if it should appear harmful to me. Yes / No
- I understand the compensation provisions for this study. Yes / No
- I have had time to consider whether to take part. Yes / No
- I know who to contact if I have any side effects during the study. Yes / No
- I know who to contact if I have any questions about the study. Yes / No
- I would like the researcher to discuss the outcomes of the study with me. Yes / No
- I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. Yes / No

I _____ (full name) hereby consent to take part in this study.

Date: _____

Signature: _____

Researchers contact details:

Dr Dominic Lomiwes (Trial coordinator) Tel. 06 355 6224, Email.
Dominic.Lomiwes@plantandfood.co.nz

Dr Suzanne Hurst (Principal Investigator) Tel. 06 355 6231, Email.
Suzanne.Hurst@plantandfood.co.nz

Unita Harold (Co-investigator) Tel.

Kirsty Lyall (Trial coordinator) Tel. 07 959 4468, Email.
Kirsty.Lyall@plantandfood.co.nz

Assoc.Prof. John Podd (Co-investigator) Tel.

Prof. Roger Hurst (Co-investigator) Tel. 06 953 7677 E mail.
Roger.Hurst@plantandfood.co.nz

Project details explained by: (print name) --- _____

Project role: _____

Date: _____

Signature: _____

Appendix H: List of Foods to Avoid

List of foods to avoid 2 days before the main trial .

Here's a list of common foods that are high in nutritional polyphenolic compounds and antioxidants

The trial co-ordinator will ask you to avoid eating these foods 2 days before and during the main part of the trial

Fruit & Fruit juice: Blackcurrants, Blueberries, Kiwifruit, Raspberry, Strawberry, Apples (green and red), Plums, Blackberries, Cherries, Cranberries, Citrus fruit (Oranges, grapefruit, lemons etc.), Grapes (black and Red), Figs.

Vegetables and Vegetable Juice: Aubergine, Beans (red, kidney), Potato/Sweet potato (red, purple), Onion (red), Cabbage (red), Broccoli (purple), Beetroot, Corn (purple), Olives (black), Avocado.

Miscellaneous: Wine (red), Coffee, Chocolate (dark), Tea (Black, green).

Dietary supplements (e.g. all supplements containing berries, vitamin C, Vitamin E)

If you have any queries about other foods that you may eat as part of your normal diet please ask the trial co-ordinator (Dominic Lomiwes).

Appendix I: ANCOVA Tables of Between-Subjects Effects

ANCOVA Table for the Stroop Overall

Tests of Between-Subjects Effects

Source	Sum of Squares	<i>df</i>	Mean Squares	<i>F</i>	Sig.	Partial Eta Squared
Stroop T1	1403.983	1	1403.983	61.290	.000	.044
Intervention	17.790	1	17.790	.777	.381	.478
Condition	9.006	1	9.006	.393	.533	.006
Intervention *Condition	3.156	1	3.156	.138	.712	.002
Error	1534.789	67	22.907			

ANCOVA Table for the Stroop Congruent

Tests of Between Subjects Effects

Source	Sum of Squares	<i>df</i>	Mean Squares	<i>F</i>	Sig.	Partial Eta Squared
StroopC1	504.379	1	504.379	16.824	.000	.201
Intervention	13.375	1	13.375	.446	.506	.007
Condition	3.490	1	3.490	.116	.734	.002
Intervention* Condition	1.312	1	1.312	.004	.835	.001
Error	2008.627	67	29.980			

ANCOVA Table for the Stroop Non-congruent
Tests of Between Effects Subjects

Source	Sum of Squares	<i>df</i>	Mean Squares	<i>F</i>	Sig.	Partial Eta Squared
Stroop Non-Congruent	894.750	1	894.750	27.830	.000	.293
Intervention	.208	1	.208	.006	.936	.000
Condition	25.554	1	25.554	.795	.376	.012
Intervention* Condition	.099	1	.099	.003	.956	.000
Error	2154.078	67	32.150			

ANCOVA Table for the Digit Span Backwards
Tests of Between Subjects Effects

Source	Sum of Squares	<i>df</i>	Mean Squares	<i>F</i>	Sig.	Partial Eta Squared
DSBT1	77.460	1	77.460	49.047	.000	.423
Intervention	.182	1	.182	.115	.736	.002
Condition	.000	1	.000	.000	.986	.000
Intervention* Condition	.384	1	.384	.220	.640	.003
Error	105.812	67	1.579			

ANCOVA Table for Digit Symbol Substitution

Test of Between Subjects Effects

Source	Sum of Squares	<i>df</i>	Mean Square	<i>F</i>	Sig.	Partial Eta Squared
DSSspeedT1	6782.961	1	6782.961	114.964	.000	.632
Intervention	.005	1	.005	.000	.992	.000
Condition	66.161	1	66.161	1.121	.293	.016
Intervention* Condition	5.846	1	5.846	.099	.754	.001
Error	3953.056	67	59.001			

ANCOVA Table for Digit Symbol Substitution

Test of Between Subjects Effects

Source	Sum of Squares	<i>df</i>	Mean Square	<i>F</i>	Sig.	Partial Eta Squared
TrailspeedT1	4933.135	1	4933.135	53.736	.000	.445
Intervention	155.579	1	155.579	1.695	.197	.025
Condition	177.711	1	177.711	1.936	.169	.028
Intervention* Condition	156.063	1	156.063	1.700	.197	.025
Error	6150.852	67	91.804			

ANCOVA Table for Complex Reaction Task

Test of Between Subjects Effect

Source	Sum of Squares	<i>df</i>	Mean Squares	<i>F</i>	Sig.	Partial Eta Squard
CRTt1	133140.805	1	133140.805	220.309	.000	.767
Intervention	1560.478	1	1560.478	2.582	.113	.037
Condition	496.065	1	497.065	.821	.368	.012
Intervention* Condition	116.408	1	116.408	.193	.662	.003
Error	40490.627	67	604.338			