# Kiwifruit and Guar Gum Modulation of Postprandial Blood Glucose and its Cognitive Effects

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## ABSTRACT

Glucose is the main source of energy for the brain, and blood glucose levels have been shown to have a significant impact on cognitive performance. This thesis research examined the effects of kiwifruit and guar gum (a soluble fiber) on the postprandial blood glucose response, and to what extent glucose manipulation can influence cognitive performance. Twenty healthy participants took part in a within subjects trial, with each individual consuming one of four breakfast diets per week (Weet-Bix, Weet-Bix + Kiwifruit, Weet-Bix + Guar Gum, and Weet-Bix + Kiwifruit + Guar gum). Each breakfast was separated by at least a 1-week washout period. It has been shown that kiwifruit and its interaction with guar gum decreases blood glucose peaks during the postprandial phase, and maintains a glucose level above fasting baseline measures over a 3-hour time period. In the present study there were no main effects of Breakfast Type across the cognitive tasks, or for interactions between Breakfast Type and Testing Time. However, there was a significant effect for time for each task, collapsed across each breakfast. (Time refers to the three points the cognitive tests were administered, one pre-breakfast, and two post-breakfast at 90 and 180 mins). Trends in the blood glucose response data indicated that when blood glucose levels were controlled and maintained (by the kiwifruit and guar gum), performance on some cognitive tasks improved and was largely sustained across the 3-hour time period, although the effects of practice could not be ruled out.

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# INTRODUCTION

Over the years sugar has been given a damaging reputation and is directly related to unhealthy foods and increased weight gain. However, one type of sugar, called glucose, is vital to everyday physical and mental functioning. Glucose sugar is an essential ingredient of the blood in the human body, as its primary responsibility is to provide energy to the brain (McNay, McCarty, & Gold, 2001). Glucose is involved in additional physiological functions, such as respiration, muscle contraction and relaxation, heart rhythm and the regulation of body temperature (Parker, Doyle, & Peppas, 2001). Within the brain, neurons and developing red blood cells predominantly use glucose for energy, using about twice as much as any other cells in the body (American Academy of Neurology, 2013). If the supply of glucose is low, then cognitive processes can be impaired (Scholey, Harper, & Kennedy, 2001). People often find that actively using their brain for thinking or problem solving can be mentally draining. Various studies have found that the level of glucose in the brain decreases rapidly during mental activity when high levels of energy are required (McNay et al., 2001; Scholey et al., 2001). Most of the time our blood glucose levels naturally fluctuate between 3.5 – 6 mmol/L (millimoles per litre) (Brand-Miller, Foster-Powell, & Mendosa, 2004). Lam, Gutierrez-Juarez, Pocai, and Rossetti (2005) state that it is essential for normal health that blood sugar be maintained at this level, and furthermore, that it should not fall below this level for periods longer than an hour. After a meal, blood glucose levels rise at once, usually reaching up to 7.8 mmol/L in non-diabetic individuals. After one to two hours the concentration then gradually falls to the previous fasting level (McNay et al., 2001).

Glucose from carbohydrates has been found to be the only fuel normally used by brain cells. Neurons cannot store unused glucose; therefore, they depend on the bloodstream to continually supply sufficient levels (De Vivo et al., 1991). Neurons always have a high need for glucose as they are constantly metabolically active, including during times of rest or sleep when the neurons work to repair and rebuild structural components in the brain (Parker et al., 2001). However, there is a fine line between too little and too much sugar and

refined carbohydrates at any one point in time. It has been shown that too much sugar can actually deprive the brain of glucose, compromising a person's ability to concentrate (McNay et al., 2001; Melanson, 1999; Parker et al., 2001). High levels of sugar from carbohydrates can cause a rapid rise in blood glucose levels quickly followed by a crash below baseline level, resulting in even lower glucose levels than before. This sugar crash is caused by the body responding to the rapid rise in glucose by quickly producing insulin to allow the body to either store the glucose or use it for energy. Therefore, glucose levels consequently fall leaving the individual feeling fatigued and irritable (Brand-Miller et al., 2004). In contrast, if glucose intake is inadequate, the body will then draw on glycogen stores to supply the brain with energy (American Academy of Neurology, 2013). Glycogen stores are converted back into glucose by the body through the use of an enzyme called glucagon, which is secreted when there is an energy deficiency (Parker et al., 2001). In rare cases where this store also fails the body will automatically seek glucose elsewhere, such as by breaking down muscle tissue to form into glucose (McNay et al., 2001).

**High and Low Blood Glucose Levels**

Normally the human body keeps its blood glucose levels stable by regulation with hormones, such as insulin and glucagon (Baron, 1998). High blood glucose levels are symptomatic of hyperglycemia; those who have a consistent blood glucose level above 7.8 mmol/L of blood are viewed as suffering from this disorder (Mizock, 2001). Most people can occasionally experience temporary hyperglycemia without significant permanent effects; however, prolonged hyperglycemia can result in diabetes and neurological damage (Del Prato, 2003; Yki-Jarvinen, 1990). Chronic symptoms of high sugar levels include fatigue, weight loss, erectile dysfunction, stupor (Yki-Jarvinen, 1990) and cardiac arrhythmia, effectively putting people at risk of heart disease and strokes (Mizock, 2001). Stress and eating large portions of refined sugars and carbohydrates have been found to increase blood glucose levels (De Vivo et al., 1991). Furthermore, heavier and obese people are also at risk as they need to create more insulin to maintain healthy glucose levels (Bruning et al., 2000).

In contrast, hypoglycemia is a condition where the glucose levels in the bloodstream drop dangerously low. This state can lead to an inadequate supply of glucose to the brain, causing excessive levels of insulin to be produced in the body (Diabetes New Zealand, 2008). Plesman (2011) notes that there are two types of non-diabetic hypoglycemia, fasting and reactive. Fasting hypoglycemia occurs when an individual has no food for eight or more hours. Those at risk of experiencing fasting hypoglycemia are individuals on medication, such as antibiotics or for pain relief, those who consume heavy amounts of alcohol or binge drink, and also those with low levels of hormones, such as insulin, glucagon, and cortisol (Diabetes New Zealand, 2008). Reactive or postprandial (after eating a meal) hypoglycemia occurs two to four hours after a meal, primarily caused by an excessive insulin release following a meal. That is, insulin continues to be released after the digestion of the glucose derived from the meal. Individuals who experience reactive hypoglycemia can be considered as pre-diabetic or at risk for developing diabetes (Diabetes New Zealand, 2008).

Low blood sugar levels have been found to result in limited energy for muscles and brain cells (American Academy of Neurology, 2013). Donnelly et al. (2005) found that low blood sugar resulted in deteriorated attention and markedly slower processing of auditory and visual information. Their study also investigated impairment at different blood sugar levels. They found that when blood sugar falls to 3.3 – 3.8 mmol/L of blood, symptoms are mild and include slight headaches, faintness, weakness and irritability. Symptoms become more severe when blood sugar levels fall to 2.8 – 3.2 mmol/L of blood; migraines, dizziness, unsteady gait, faintness, weakness, fatigue, marked irritability, sweating, tremors and general nervousness can all be experienced (McNay et al., 2001). Low sugar levels in the brain may also produce impairment of judgment, moodiness, depression, negativism, personality change, confusion, automatic behavior, ataxia and memory loss (Amiel, Dixon, Mann, & Jameson, 2008; Cryer, 2002). Severe hypoglycemia may also cause seizures and/ or coma. The consumption of adequate amounts of carbohydrate foods can help normalize the body's glucose levels and symptoms quickly resolve (Cryer, 2002).

Abnormal glucose levels are seen to cause various health issues, such as diabetes (Cryer, 2002), slow cognitive development (Lucia et al., 2013) and brain damage (McNay et al., 2001). Diabetes develops when the bloodstream is overloaded with simple sugars (processed foods, candy, cakes, fruit juices) and refined carbohydrates (pasta, white rice, white bread), which create rapid swings in glucose levels and effectively impair the body's ability to respond to insulin (Diabetes New Zealand, 2008). Diabetics are more likely to suffer a faster decline in mental ability than non-diabetics as they age, specifically in regards to processing speed, and they have also been found to be more vulnerable to depression and dementia (Lucia et al., 2013).

Diabetes is characterized by persistent hyperglycemia or hypoglycemia, where intermittent episodes may be present in pre-diabetic states. The three most common symptoms found in this disease are polyphagia (frequent, pronounced hunger), polydipsia (frequent thirst), and polyuria (increased volume of urination) (Baron, 1998). The World Health Organization (2006) recognizes two main forms of diabetes: Type 1 and Type 2. Type 1 is associated with abnormally low insulin levels in the body, such that blood glucose levels cannot be kept in the normal range. The body's own immune system destroys insulin-producing cells that are located in the pancreas, resulting in a complete deficiency of the insulin hormone (Diabetes New Zealand, 2008). Type 1 diabetics are at risk of developing hypoglycemia, most commonly being caused by the medication they are given to lower high blood glucose levels (Cryer, 2002). That is, the treatment amount is not exactly matched with what the body needs, and the response is to drive glucose levels too low. If too much insulin is administered, the excess negatively reacts with already compromised glucose regulation (Diabetes New Zealand, 2008). In contrast, Type 2 is characterized by a marked resistance to insulin. That is, the body is still able to produce insulin, but it is either not enough or the body is unable to recognize what it is and use it properly (Amiel et al., 2008). Therefore, glucose cannot get into the body's cells to be used for energy. Resistant or low insulin levels also prevent the body from converting glucose into glycogen, which makes it difficult to remove excess glucose from the blood (De Vivo et al., 1991). This then causes individuals with Type 2 diabetes to develop hyperglycemia, as glucose levels in the blood remain

high without the working mechanisms of insulin to control the surges after meals (Brand-Miller et al., 2004).

**Food, Nutrients and Glucose Levels**

The food we eat is made up of carbohydrates, protein and fat; although humans can get sufficient energy from protein and fat, the brain requires carbohydrates to function, primarily because neurons cannot burn fat (Brand-Miller et al., 2004). However, as the body changes 100% of carbohydrates consumed into sugar, all foods containing carbohydrates will cause blood glucose levels to rise, within an hour or two after eating. Thus, carbohydrates are not always a beneficial source of energy, as some foods can make blood glucose levels rise more rapidly than others by producing more glucose than our body actually needs (Benton & Parker, 1998). The impact of carbohydrates on blood glucose levels depends on what foods have been consumed, the size of the meal and also the time of day it is eaten (Dye & Blundell, 2002). The amount of carbohydrates that individuals should consume depends largely on their overall calorie intake per day. For example, an average-weight woman should consume around 60 grams of carbohydrates per meal (daily intake 2000 calories), whereas an overweight individual should aim to eat around 45 grams of carbohydrates per meal (daily intake 1200 calories) (Brand-Miller et al., 2004). Humans tend to have a higher blood glucose level in the morning, as the body draws on its glycogen stores over night to ensure the body has enough energy in anticipation of its wakening needs (Dye & Blundell, 2002).

One way of determining how certain foods impact on blood glucose is to use the Glycaemic Index (GI). The GI is a measure of how quickly carbohydrates enter the bloodstream as glucose (Brouns et al., 2005). Foods are ranked on the GI scale from 0-100, and pure glucose has a rating of 100 (Brand-Miller et al., 2004). Low GI foods (less than 55) cause a slower and steadier rise in blood sugar after eating. Some example foods are mixed-grain bread, fresh fruit, pasta, milk, noodles and sweet potatoes (Jenkins et al., 2002). Foods high on the GI scale (70 or more) result in a faster and higher rise in glucose levels. For instance, white bread, instant rice, fries and cornflakes all have a high GI

(Jenkins et al., 2002). Limiting the intake of high GI foods will lower the average GI of a meal and stop swings in glucose levels. High GI foods cause a rapid increase in blood glucose levels, followed by a relatively fast glucose disposal. This causes the levels of glucose in the body to decrease below the fasting concentration in the later postprandial period (Brouns et al., 2005). Low GI foods have been found to supply slow, continuous energy over the day and as they are digested slower in the stomach, they make people feel fuller for longer (Jenkins, Kendall, Axelsen, Augustin, & Vuksan, 2000). Low GI foods result in a more moderate peak and may also maintain a prolonged net increment in blood glucose above fasting concentration (Brouns et al., 2005), as shown in Figure 1.

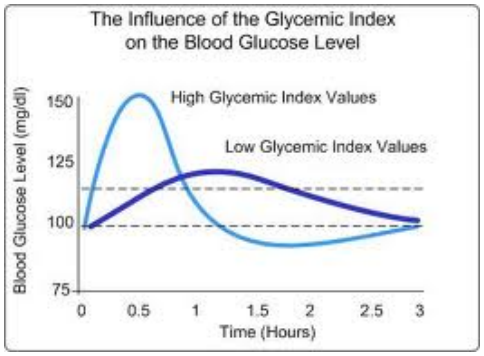



*Figure 1*. Blood glucose levels in response to high and low glycaemic foods (Diabetes Nutrition, 2014).

However, the GI alone does not make a certain food good or bad; many high GI foods contain beneficial nutrients. It is the amount eaten that makes the difference to how they impact on the body. Individuals vary in their insulin response to food; some may have a higher tolerance for high GI foods than others (Jenkins et al., 2002).

Liljeberg, Akerberg, and Bjorck (1999) found that when a low GI cereal was consumed at breakfast the body had an overall lower demand for insulin and also an improved sensitivity to insulin. An important finding within this study was that in the hours between breakfast and lunch the fasting glucose state was delayed and a greater glucose increment was found up to four hours after the breakfast. This also resulted in a lower glucose response after a high GI lunch (fries, meatballs and instant powder-mashed potatoes) on the same day,

emphasizing the notion that low GI foods create a more lasting, stable glucose level after food consumption. A following study by Nilsson, Ostman, Holst, and Bjorck (2008) looked at how long this improved fasting concentration can last in the body. They found that after a low GI meal was consumed at night there was still an improved glucose tolerance at breakfast, 10.5 hours later. The authors concluded that their low GI meal reduced the rate of digestion and absorption over a more substantial postprandial period than previously believed possible.

Food supplements have also been added to low GI foods in order to see if they can improve glucose tolerance. A supplement frequently used is guar gum, a soluble fiber that can be added to various mixtures and foods, working to lower the GI of a meal. The effects of guar gum have been identified in studies with diabetic and healthy participants. Groop, Aro, Stenman, and Groop (1993) found that when guar gum was added to a bread breakfast participants showed an improved level of glycaemic control and postprandial glucose tolerance compared to a control group who were given a similar breakfast with no guar. Although this study did not choose a breakfast based on the GI, the bread used was considered to have a low GI value. Wood, Braaten, Scott, Riedel, and Poste (1990) also found that the use of guar gum was most effective in decreasing the overall peak of blood glucose levels, coupled with a lowered insulin response. In other words, by slowing the rate of digestion they found a lowered peak in glucose levels. However, not all studies have yielded such conclusive findings. For example, although Vourinen-Markkola, Sinisalo, and Koivisto (1992) did find that guar gum reduced postprandial glycaemia, this was only found in large meal consumption but not in smaller, frequent snacks. In addition, the blood glucose response was improved even more when guar gum was combined with low GI carbohydrates, in comparison to carbohydrates with a high GI value.

**Glucose Regulation in the Brain and Body**

Once glucose has been absorbed through the stomach and the intestine, it passes into the liver and then throughout the body (Berdanier, 1996). Mechanisms in the body maintain a minimal level of glucose in the blood and also regulate surges of glucose after meal consumption (Schneider, 2013). In the resting state

the level of glucose in the blood is around 3.9 mmol/L and the body works to stop it from exceeding 7.8 mmol/L (Berdanier, 1996). Normally, around two hours after a meal is consumed, blood glucose levels will be at their highest concentration. A rise in blood glucose levels signals the pancreas to release the hormone, insulin, from the beta cells to allow the glucose to become available to the cells in the body (Gropper & Smith, 2013). Insulin is secreted immediately to control the initial glycaemic response, and from then on a steady supply of insulin is produced to manage on-going digestion after food (Berdanier, 1996). The main responsibility of insulin is to transport glucose to cells around the body to be used for energy, and in turn, this works to lower blood glucose levels (Schneider, 2013). However, Nordlie, Foster, and Lange (1999) argue that the centre of the glucose regulatory mechanism is the liver, where glycogen is stored.

The importance of glycogen is that it is readily mobilized, that is, it is capable of quickly turning into glucose when the body's blood sugar level drops below its normal fasting level. Glycogen is developed from the process of digestion and from what is available from digestion in the stomach (Berdanier, 1996). Therefore, the amount of glycogen that can be stored depends largely on the nutritional value of the food that is consumed over the day. For example, a well-balanced carbohydrate meal within individuals' daily calorie intake will allow for beneficial glycogen storage, one factor that individuals themselves are able to control. Cells and organs in the body are never in a resting state; therefore, the demand for glucose is constant. As a result, glycogen is also constantly being used and broken down into glucose and transported around the body via the blood stream (Schneider, 2013).

For everyday functioning, blood glucose levels need to be carefully maintained to ensure blood sugar levels are held reasonably constant. Swings in blood sugar levels can have detrimental effects on an individual's health (Nordlie et al., 1999) and may interfere with the sufficient supply of energy to keep vital organs functioning (Berdanier, 1996). Maintaining an adequate level of glucose can be achieved through a balance of various factors, including the rate of food consumption, the absorption of carbohydrates in the intestine, the rate of

utilization of glucose, the loss of glucose through the kidney tubule (that is, what is lost due to normal bodily waste), and finally, the removal or release of glucose by the liver (Jiang & Zhang, 2003). The liver plays an important role in glucose homeostasis by maintaining a balance between the uptake and storage of glucose (glycogenesis) and the release or breakdown of glucose (glycogenolysis) (Nordlie et al., 1999). These metabolic pathways are regulated by four hormones: insulin, glucagon, adrenalin, and cortisol (Berdanier, 1996). Insulin levels in the blood have been found to be minimal when limited food has been consumed. Glucagon is then responsible for breaking down the glycogen stores from which glucose can be manufactured (Jiang & Zhang, 2003). The body then uses insulin and glucagon to avoid reactive and postprandial hypoglycemia (Berdanier, 1996). During these periods, cortisol and adrenalin cause an increase in glucose levels. The pancreas automatically responds by secreting more insulin to counter-balance this action thereby maintaining low (normal) glucose levels (Schneider, 2013).

**Glucose and its Effects on Cognition**

There are numerous studies supporting the idea that glucose levels have a direct effect on cognition. The brain's supply of energy from glucose is received intermittently throughout the day. Scholey et al. (2001) state that prolonged and demanding tasks have been found to deplete glucose concentrations, emphasizing the idea that cognitive performance relies on the continuous supply of blood sugar to the brain. Although the majority of research has focused on the effects of glucose metabolism and memory, a number of other studies have examined enhancements in other cognitive domains. Glucose administration has been found to be beneficial in choice reaction time studies (Fischer, Colombani, Langhams, & Wenk, 2001; Benton & Owens, 1993), serial sevens (Kennedy & Scholey, 2000) which assesses attention/ concentration, mental tracking and visual information processing (Benton, Owens, & Parker, 1994; Donohoe & Benton, 1999), immediate recall (Donohoe & Benton, 1999), word lists (Benton & Parker, 1998; Benton & Owens, 1993), selective attention (Holmes, Koepke, & Thompson, 1986) and vigilance (Green, Taylor, Elliman, & Rhodes, 2001), the Stroop test (Benton et al., 1994), and verbal fluency (Craft, Murphy, &

Wemstrom, 1994). A governing factor consistently found across these studies is that the tasks need to be demanding in order to observe a significant enhancement from glucose. Complex tasks have repeatedly been found to deplete glucose levels, whereas simpler tasks have often been reported to have no or little effect (Benton et al., 1994; Feldman & Barshi, 2007).

Traditionally, it was believed that there is an equal distribution of glucose throughout the brain, but recent studies have found that extracellular glucose concentrations vary across brain structures. It has also been shown that the average level of glucose in the brain does not follow the average levels in the bloodstream (Lund-Andersen, 1979; Warren & Frier, 2005). Specific behavioral tasks have been found to cause fluctuations in glucose levels within areas of the brain involved in the processing of that task (Messier & Gagnon, 1996; Ragozzino, Unick, & Gold, 1996). McNay, Fries, and Gold (2000), working with rats, found glucose levels in the hippocampus decreased during the performance of a spatial working memory task, with a more pronounced decrease when the task became more complex. When the rats were administered glucose, the depletion in the hippocampus was reversed and memory was enhanced. Ragozzino and Gold (1995) injected rats with glucose directly into the hippocampus finding a profound improvement in mnemonic functioning.

This same effect has been found in studies with humans following glucose administered in an oral form. Messier and Gagnon (1996) investigated the relationship between glucose and insulin levels in the brain and cognitive performance. PET scans revealed that individuals with Alzheimer's disease metabolized less glucose in the cerebellum and hippocampus in comparison to those of similar age without the disease. The degree to which cognitive performance is affected depends in part on the degree of hypoglycemia. For example, Gold (1986) showed that with moderate hypoglycaemia, short and long-term memory and working memory all deteriorated significantly. Choice reaction time was also impaired in moderate hypoglycaemia, but not in participants with mild hypoglycaemia. Neuroimaging examining the long-term effects of hypoglycemia has found profound brain damage, particularly in the

frontal lobes and hippocampus. Cerebral blood flow was found to be redistributed during hypoglycemia, with an increase in the amount of blood going through the frontal lobe (Szablewski, 2011). Such findings demonstrate that some areas of the brain are more susceptible to the influence of glucose than others (Duelli & Kuschinsky, 2001; Mayer, Nitsch, & Hoyer, 1990).

Poor glucoregulation (regulation of blood glucose levels) has been found to induce a slow degradation of neurons, and therefore, can result in severe cognitive impairment over time (Messier & Gagnon, 1996). Neuronal death can occur when neuronal energy mechanisms are compromised by ischemia (restricted blood supply to tissues), which causes a shortage of oxygen and glucose to the brain. In a study with healthy (non-diabetic) individuals, Gerozissis (2002) determined each person's glucose regulation abilities and then had them all perform cognitive tests on memory and information processing. It was found that the individuals with good glucoregulation out-performed the poor glucoregulators on every cognitive task. Greenwood (2003) completed a similar study using young and old participants, concluding that although age does have an effect on cognitive decline, the older individuals with good glucoregulation performed better than the older individuals with poor regulation. Surprisingly, the older individuals with good glucoregulation had an overall better performance on memory and attention tasks than the younger individuals with poor glucoregulation. These findings not only emphasize the relationship between glucose and cognition but also the role an individual's ability to metabolize glucose plays, despite varying factors such as age.

Of all cognitive tasks those involving memory most clearly show the effects of glucose on cognitive performance. Research has found that glucose improves memory through facilitating acetylcholine (ACh) synthesis and release in the brain. ACh is a neurotransmitter widely recognized for its influence on learning and memory (Sims et al., 1980). When the brain metabolizes glucose, one of its functions is to produce acetyl-CoA, which is a precursor of ACh (Ragozzino et al., 1996). However, the effect of ACh synthesis only seems to be apparent when cholinergic neurons are also activated. Glucose concentrations in the brain

can directly influence the rate of ACh synthesis by modifying the breakdown of acetyl-CoA (Ragozzino et al., 1996). When blood glucose levels are low (hypoglycaemia) the brain is unable to produce sufficient acetyl-CoA, and ACh synthesis is significantly decreased. Abnormalities in the cholinergic system have been shown to influence behaviour, learning and memory (Mayer et al., 1990).

It has also been suggested that glucose may be involved in the process of storing new information in the brain. Messier and Gagnon (1996) found that depleted levels of glucose in the blood stream had a significant effect on short-term memory, more so than long-term memory. Low glucose levels have also been related to Glut-1 deficiency. Glut-1 is a glucose transport protein that is used to carry glucose molecules through the blood-brain barrier (Gjedde & Crone, 1981). This then means when blood glucose levels are low, the amount of glucose metabolized in the brain to fuel cognitive performance is limited, primarily resulting in learning difficulties. For example, Type 1 diabetic rats revealed a decreased capacity to transport glucose across the blood-brain barrier, suggesting that low blood sugar levels were responsible for the reduction in carriers for facilitated glucose transport into the brain (Ragozzino & Gold, 1995).

There is a strong correlation between neurological impairment and glucose intolerance. Roozendaal's (2003) study with middle-aged, healthy adults found that increasing fasting glucose levels were significantly associated with hippocampal and amygdala atrophy over a 4-year period. These brain structures are part of the limbic system and are involved in memory, decision-making, emotional reactions and spatial navigation (Lucia et al., 2013). Taylor and MacQueen (2007) argue that glucose metabolism is directly related to psychological disorders. They found that individuals with depression had a 60% higher risk of developing diabetes than those without depression. High levels of glucose were also found in individuals with schizophrenia; however, this particular relationship may have been influenced by antipsychotics (Gibson & Green, 2002). Glucose intolerance is also linked to neurodegenerative disorders,

including dementia, Huntington's and Parkinson's diseases (Gibson & Green, 2002; Taylor & MacQueen, 2007). These diseases have a high correlation with Type 2 diabetes and also obesity in older adults (Amiel et al., 2008).
It is important to note, though, that findings on the link between neurological impairment and glucose are only correlational studies. At this stage, no causal relationship has been established.

**Effects of Glucose on Attention**

Attention is a complex mental ability involving various processes that relate to everyday life and functioning. Attention can be defined as a state of consciousness characterized by an individual's capacity to maintain selective (the ability to focus on a particular object while ignoring other irrelevant information) or sustained (the ability to focus on specific stimuli over a prolonged period of time) concentration. For this reason, attention has recently become a focus point in regards to the effects of blood glucose levels. These effects have been widely demonstrated on psychomotor tasks, such as reaction time, selective attention and fine motor activity (Feldman & Barshi, 2007). Studies have shown that there is a noticeable decrease in performance on certain tasks when glucose levels are low (3.8 mmol/L or less). Low glucose levels have a marked effect on executive functioning processes, such as reasoning, planning and problem solving (Feldman & Barshi, 2007). In order to assess specific attention abilities, studies have used various measures, including the Test of Everyday Attention (McAulay, Deary, Ferguson, & Frier, 2001), the Trail-making test (Hoffman et al., 1989), the Stroop test and the Letter Cancelation task (Benton et al., 1994). These tests have been used to determine the level of sustained, visual, selective and divided attention in individuals with diabetes and acute hypoglycaemia.

Reaction times on selective attention tasks are significantly influenced by low blood glucose levels. Hoffman et al. (1989) completed a study assessing reaction time and divided attention in 18 Type 1 diabetic patients. The Trail-making test was used to assess sensory motor skills and higher-cortical functioning. This test has two parts (A and B), with Trail B being more

cognitively demanding. Consistent with findings in other studies, a slower motor response was observed during hypoglycaemia (Gjedde & Crone, 1981; Wolever, 2003). However, Hoffman et al. also reported a decrease in sustained concentration, suggesting that hypoglycaemia does more than disrupt mechanisms of motor control. Hypoglycaemia did not alter performance on simple visual reaction time, but significant impairments were found on more complex reaction time tasks that required decision making and sensory/ response discrimination, supporting Feldman & Barshi's (2007) assertion that the effects of hypoglycaemia are likely to be apparent only on complex cognitive tasks. An additional measure in their study was a driving simulator task. Interestingly, though individuals in a hypoglycaemic state did perform slightly poorer, the results failed to reach statistical significance. If a task is overly familiar to the individual, then selective attention and concentration may be less affected by low glucose levels (Feldman & Barshi, 2007; Gropper & Smith, 2013). Lobmann et al. (2000) used a selection of healthy and diabetic (Type 1) individuals in a study using event-related brain potentials to assess performance on a selective attention task. Healthy participants were induced into a hypoglycaemic episode by the use of a hyperinsulinemic (high insulin) euglycemic (normal blood sugar) clamp. The clamp is used to quantify sensitivity to insulin, and in this particular study insulin was administered to the participants at an increased rate until individual blood glucose levels decreased to 2.8 mmol/L. Within the selective attention task, participants were exposed to a sequence of 200 randomly ordered coloured letters and were asked to respond using either their left or right hand, to 2 letters of a particular colour (i.e., a red 'H' and 'D') when they appeared on the screen. Participants were instructed to respond as fast and accurately as they could, and their response times to correct and incorrect targets were recorded. The cognitive testing was completed before and after the use of the hyperinsulinemic euglycemic clamp; that is, when glucose levels were considered in the normal range and then when they were in the hypoglyceamic range. In accordance with other studies, Lobmann et al. found cognitive disturbances began to occur when blood glucose levels dropped below 3 mmol/L. This finding was noted in both experimental groups, with no difference in diabetic and non-medical hypoglycaemia. In both groups, the induction of hypoglycaemia delayed the

selection of stimuli to a relevant colour, motor processing and reaction time. When blood glucose levels were restored to normal, reaction time improved for the diabetic group but not the control group; however, colour selection did return to the normal selection rate. For the control group, reaction time delays remained, suggesting that individuals with Type 1 diabetes have a higher ability to cope with hypoglycaemic states. This study used a simple cognitive task (Feldman & Barshi, 2007); therefore, the test used may not have been sensitive to the stronger effects of hypoglycaemia.

A large number of studies involving glucose and attention have focused on the effects of hypoglycaemia and various levels of low blood glucose. In contrast, Benton et al. (1994) examined the effects of increased blood glucose levels on attention in young adults by giving the participants either two glucose drinks (50 g and then 25 g 25 mins later) or a placebo. Participants were required to complete the Rapid Information Processing Task (RIPT) and the Stroop test, the latter containing both congruent and incongruent components. It was found that falling blood glucose levels between the two glucose drinks was associated with faster reaction times and improved memory in the RIPT. This suggests that the cognitive demands of the task were increasing the brain's use of glucose at that time, reflecting the individual's ability to regulate glucose (Gerozissis, 2002). Benton et al. also found that the participants who had consumed the placebo drinks, and whose blood glucose stayed relatively high between the two measurement times, had slower overall reaction times during the RIPT task. Over the course of the two placebo drinks it was assumed that the participants' blood glucose levels would drop dramatically, particularly as the tasks completed were considered to be cognitively demanding. However, after observing that some participants' blood glucose levels stayed relatively high, Benton et al. hypothesized that this reflected a poor ability to regulate glucose, as blood glucose may not have been transported to the cells where it was needed. Interestingly, improved performance was observed during the incongruent Stroop task when blood glucose levels were rising instead of decreasing. Although there was no mention of performance on the Stroop test when glucose levels were falling, this suggests that the brain's initial uptake of glucose also provides sufficient energy for optimal cognitive performance.

McAulay et al. (2001) conducted a study that experimentally induced hypoglycaemia in healthy individuals. Blood glucose levels were lowered to 2.6 mmol/L, just under the level that has been found to cause hypoglycaemic symptoms. The Test of Everyday Attention was used to measure and discriminate among the types of attention, including visual selective attention, auditory selective attention, sustained attention and divided attention (Feldman & Barshi, 2007). When hypoglycaemia was induced participants displayed a significant deterioration in visual selective attention, being unable to ignore irrelevant information. However, accuracy of identifying objects was preserved but at the expense of reaction time (McAulay et al., 2001). Reaction time significantly increased, suggesting that responses were slowed with the increased need to concentrate. Sustained attention stayed the same across both groups, a result also obtained by Donnelly et al. (2005). Other studies have found sustained attention to be affected by low blood sugar over a prolonged period of time (Amiel et al., 2008; Cryer, 2002). McAulay et al. found no significant difference in the sustained attention tasks; however, participants induced into a hypoglycaemic state did take an overall longer time to complete the tasks.

Examining attention in relation to hypoglycemia is highly important, as it directly relates to the ability to carry out every-day activities. Individuals with insulin-treated diabetes are at a high risk for developing hypoglycaemia (Szablewski, 2011), which may result in attention deficits (Lobmann et al., 2000). Practical problems that may occur during periods of low blood sugar include difficulty filling out and interpreting forms, driving in heavy traffic and finding particular items that are mixed with others (Donnelly et al., 2005). These aspects of hypoglycaemia could have important practical implications on an individual's life.

**Effects of Glucose on Memory**

Although memory has traditionally been the primary focus in studies of the cognitive effects of blood glucose levels, some discrepancies still exist regarding the types of memory that are affected by glucose. Working memory, spatial memory and verbal declarative memory have all been shown to improve with

increased glucose, whereas procedural memory has not (Feldman & Barshi, 2007). However, this may be due to the studies being inconsistent with the type of memory being tested, that is, procedural memory has different functions in comparison to other types of memory, and possibly should not be compared against other forms. Other research has shown that glucose can improve memory in elderly subjects (Manning, Hall, & Gold, 1997), but the same has not been consistently found in younger adults. For instance, age has been found to be an important factor in regards to performance abilities on verbal recall (Benton & Owens, 1993; Benton et al., 1994). Gold (1986) also found that individuals had a better chance of recalling a memory if they had a higher level of blood glucose at the time of storing the information, emphasizing that adequate glucose levels are required throughout the entire process of forming memories and not just at retrieval.

To date, word lists have been the most commonly used measure of glucose and cognitive performance during the assessment of memory, and a better recall has also been highly associated with blood glucose levels in diabetic and non-diabetic individuals. Benton and Owens (1993) conducted a study examining the influence of increasing blood glucose on memory. Young, healthy adults consumed a 50 g glucose drink before beginning the tasks after an overnight fast. In their first experiment they used a word list and a spatial memory test, where participants were shown 16 pictures of everyday objects (e.g., hammer, cat, doll) in a 4x4 grid for 30 s and then asked to arrange the pictures in their previous position. It was reported that increasing blood glucose levels from the glucose drink condition were associated with the recall of more words from the list in comparison to those who received a placebo, but no significant difference was found for the spatial memory test. In a second experiment, Benton and Owens examined the effects of raising glucose levels over a period of two hours. Participants received an initial glucose drink of 50 g, and then two subsequent drinks of 25 g at 45 and 75 mins later in an attempt to keep blood glucose levels from falling after an initial rise. Again, a significant difference was found in the ability to recall the word list, but not with the recall of a Wechsler story. This finding is inconsistent with a similar study conducted by Manning et al. (1990), although these findings may be the result of Manning et al. using college

students as participants, whom presumably had above average intelligence; therefore, the test may not have been demanding enough. In a subsequent study, Benton et al. (1994) used the word list to assess memory and gave their participants two glucose drinks (50 g and 25 mins later another 25 g). They also found that relative to the placebo group, the glucose drink was associated with a better recall of words, even after completing another cognitively demanding task in between.

The effects of glucose have also been shown in relation to recognition memory in non-diabetic individuals. Green et al. (2001) used an immediate verbal recognition memory task and the Bakan vigilance task (a measure of sustained attention and reaction time) in a study completed by young healthy adults. Each participant completed four experimental sessions where cognitive performance was tested. A glucose drink was consumed at two of the sessions prior to testing and on the other two sessions a placebo drink was given. In one of each drink condition the participants were accurately told what drink they were being given, and in the other two they were told they were incorrectly informed about the drink's content. This procedure aimed to limit and distinguish between expectancy effects. When given the glucose drink on both the accurate and inaccurate occasions, an improvement of recognition times was found; however, the number of words correctly recognized did not improve. A similar result was obtained by Azari (1991), who concluded that although glucose administration did improve reaction time, it did not influence the ability to recall more words. However, Manning et al. (1990) found that the number of words recognized did actually increase when glucose was administered to participants with brain deficits, such as dementia. Following a glucose drink, performance also improved on the Bakan task, but this was only observed when the participants were told they were receiving a glucose drink.

Studies examining older adults have displayed stronger effects of glucose than those using younger adults. Manning et al. (1990) used healthy older adults (62-84 years of age) to examine the effects of glucose on memory performance. They found that a glucose drink benefited performance on a declarative memory task but not for "non memory" processes. That is, cognitive functioning (IQ

test), attention (letter cancellation task and digit span) and motor tasks (finger-tapping). Glucose consumption was found to enhance memory on a selective reminding task and a logical memory task (a modified version of the Wechsler memory test). The selective reminding task examined the ability to recall non-contextual verbal information (Feldman & Barshi, 2007), whereas the logical memory task measured contextual verbal information (Manning et al., 1990), suggesting that increased blood glucose actually does influence more than one aspect of declarative memory. An important point about these findings is that these two tasks have previously been found to be particularly difficult for older adults (Feldman & Barshi, 2007). In a similar study Craft et al. (1994) found that older adults displayed better memory after receiving a glucose drink when their blood glucose levels were decreasing after an initial rise. Those participants whose blood glucose levels stayed above baseline one hour after the first test were found to have a higher rate of word recall than those whose blood glucose levels dropped below their usual fasting baseline.

An important outcome of research on glucose and memory is the finding that consuming a breakfast to increase blood glucose levels may lead to different results from when the same amount of glucose is consumed in just a drink. Benton and Parker (1998) found that after participants had consumed a breakfast in the morning, performance on spatial memory, word list recall and Wechsler story retention tasks were all significantly improved in comparison to a fasting breakfast period. These findings are inconsistent with other studies where glucose levels were manipulated by a glucose drink only (Gold, 1986; Green et al., 2001). Breakfast has been found to increase glucose levels in the blood after consumption, which in turn appears to improve memory performance (Riby et al., 2009).

Overall, it has been shown that memory is highly susceptible to the influence of glucose and glucose-enhancing breakfasts. Memory is a cognitive function that is used daily for various tasks; therefore, it is important to have a consistent supply of glucose to the brain in order to keep memory processes working effectively.

**Effects of Glucose on Reaction Time**

Reaction time (RT) is defined as the interval time between the initiation of a stimulus presentation and the initiation of a subsequent behavioral response. To date, RT has been incorporated into a large number of cognitive tests, and is considered to be an indicator of speed of processing. That is, how fast the participant can execute the mental operations of the task. RT tests have used visual and auditory stimuli, such as a specific colour, spatial position or the frequency with which an object appears (Feldman & Barshi, 2007). Simple RT tasks require participants to hit a key or button as fast as they can when a stimulus appears, where in comparison, Choice Reaction Time tasks involve multiple stimuli that must be discriminated from each other before the individual responds. Studies of the effect of blood glucose levels on RT have produced conflicting results. The interaction between glucose and RT has been found to be a complex process that is influenced by several factors, including the time of day and what is consumed to alter glucose levels (Wolever, 2003).

To date, the relationship between RT performance and blood glucose has been most commonly examined when glucose levels were low, or hypoglycaemic, and has been displayed in diabetic and non-diabetic individuals. Strachan et al. (2001) used the Digit Symbol Substitution Test (DSST) to assess decision and movement time in non-diabetic individuals who were made hypoglycaemic (2.5 mmol/L). They found that performance significantly deteriorated when the DSST was completed at hypoglycaemic levels, compared to the test being completed at a normal glucose range (5.0 mmol/L +) by the same participants on a different morning. Similarly, Kerr, MacDonald, and Tattersall (1989) measured the RT of healthy participants on a finger-tapping task. They found that RT began to slow at 3.0 mmol/L, which is just above the 2.8 mmol/L cut-off for medical hypoglycaemia (Brand-Miller et al., 2004), suggesting that low blood glucose levels still within the healthy range can impair RT.

Driesen, Cox, Gonder-Frederick, and Clarke (1995) examined cognitive processing speed on adults with insulin-dependent diabetes. Participants were required to complete various simple, choice and complex RT tasks in normal

resting glucose states (4.4 – 6.7 mmol/L), mild (3.1 – 3.9 mmol/L), moderate (1.8 – 2.8 mmol/L) and severe hypoglycemic states (1.5< mmol/L). Driesen et al. reported that during moderate and severe states, RT was significantly delayed on all cognitive tasks. However, during periods of mild hypoglycaemia, RT varied across participants, and simple RT was not significantly affected. This finding has been consistent across various studies, indicating that individuals are differentially affected by the symptoms of mild hypoglycaemia. One reason for this may be due to an individual's ability to regulate glucose, with some people being more sensitive to the physiological effects of low blood sugar levels, whereas others have the ability to function adequately even when glucose drops into the hypoglycaemic range (Jian & Zhang, 2003; Yki-Jarvinen, 1990).

Maassen et al. (1990) examined a number of RT tasks, also using participants with insulin-dependent diabetes, including the Number Connections Test (connecting circles as fast as possible), an Aiming Center Test (placing marks inside circles for 60 s), a Line Tracing Time Test (time taken to draw a line between two parallel lines), and a Reaction Time Test (pressing a button every time a light is presented). Interestingly, in this study a significant increase in RT was only observed in the Reaction Time Test, and none of the other measures. A study completed by Holmes et al. (1986) concluded that simple RT, such as the Reaction Time Test completed by Maassen et al. (1990), was not affected by any level of glucose at hypo- or hyperglycaemic levels. Holmes et al. did find that choice and complex RT tasks were significantly slowed with glucose levels at 3.1 mmol/L or less, indicating that RT is affected by the type of task and how demanding it is (Feldman & Barshi, 2007).

RT has also been examined in regards to increasing blood glucose levels, largely by having participants consume a glucose drink. Gropper and Smith (2013) reported a glucose-induced RT improvement across various levels of blood glucose. Higher levels of glucose (4.5 – 6.5 mmol/L) significantly correlated with improved RT (Manning et al., 1990). RT on a computerized version of Serial Sevens (counting backwards in sevens from 100) has also been found to improve after the consumption of a glucose drink, considerably comparable to the placebo control group (Scholey et al., 2001). Benton et al. (1994) found

similar results, in that increasing blood glucose levels resulted in faster decision times. Such results suggest that speed of processing information becomes faster when there is a larger amount of glucose available for the brain to use.

However, although increased blood glucose levels within a normal range have been found to improve RT, levels that reach hyperglycaemia impair an individual's ability on RT tasks. Sommerfield, Deary, and Frier (2004) found that when they induced hyperglycaemia in 20 participants (blood glucose levels of 16.5 mmol/L +), RT on a digit symbol test and on the Trail Making B test slowed significantly compared to when the same tests were completed at a normal blood glucose level of 4.5 mmol/L. Cox et al. (2005) concluded from their own study that hyperglycaemia begins to impair RT and cognitive performance at a glucose level of 15 mmol/L. However, they did argue that the negative impairment found was highly individualized, with only 55% of the affected participants demonstrating slowed RT and increased error rates.

Various studies have also used meals to increase blood glucose levels and subsequently examine RT. Dye and Blundell (2002) reported that improvements on RT were more pronounced in the morning after the consumption of a medium GI breakfast in comparison to any other mealtime of the day. This finding has been repeated in similar studies where participants were required to consume a standard dinner the night before, followed by a 12-hour fast overnight, and then were given a specific test breakfast (Fischer et al., 2001; Lloyd, Rodgers, Hedderley, & Walker, 1996). Bellisle et al. (1998) found that missing breakfast significantly slowed RTs on a variety of cognitive tasks. Dye and Blundell also reported that foods that enhance glucose availability (low glycaemic foods) facilitated the speed of RTs more than food that had little effect on the glycaemic response. However, this study did not clearly identify the actual level of blood glucose; therefore, it is difficult to make assumptions as to whether the food consumed increased glucose levels and if so, for how long. In comparison, Kaplan, Greenwood, Winocur, and Wolever (2001) found similar improvements in RT on a memory task after elderly participants had consumed either a high or low GI carbohydrate meal on separate occasions, indicating that RT can be improved from any food that increases blood glucose levels. However, Nilsson,

Radeborg, and Bjorck (2012) found no significant difference in RT between two groups that either consumed a carbohydrate only breakfast or carbohydrate breakfast mixed with guar gum.

To date, carbohydrate meals have produced mixed results, indicating that various factors may be contributing to RT performance. Wolever (2003) argued that the type of food consumed has a large impact on RT. For example, a meal that is high in fat has consistently been found to slow RT, irrespective of what time of day it was consumed (Taylor & MacQueen, 2007; Wolever & Mehling, 2002). Craig (1986) found that after a high-fat lunch, participants' RTs were markedly delayed, known as the post lunch dip. On the other hand, a low-to-medium fat lunch was found to improve performance for up to two hours after the meal. In a later study, Markus, Panhuysen, Jonkman, and Bachman (1999) found that in stress-prone participants, who had relatively low blood glucose levels (but not hypoglycaemic), a carbohydrate-rich diet improved RT and also reduced errors on a demanding recognition task. In support of high carbohydrate diets, D'Anci, Watts, Kanarek, and Taylor (2009) completed a study with 19 healthy females, with half being put on a low-carbohydrate diet and the other half on a low-calorie diet. RT was assessed at various points over a 2-week period and those eating the limited carbohydrate diet had significantly slower RTs, particularly in comparison to their own baseline assessment results.

It is apparent that blood glucose levels can have a significant effect on individuals' RTs, no matter if they are considered healthy or diabetic. RT has been found to be slowed on a variety of cognitive tests, including digit substitution, finger tapping, trail making, and drawing (psychomotor) tasks, suggesting that low glucose levels may cause a global impairment affecting numerous activities. Interestingly, a glucose reading of 3.0 mmol/L, considered to be within an abnormal range, has been shown to slow RTs. However, previous research has shown that mild hypoglycaemia does not consistently have a negative impact on RT. Higher glucose levels after the consumption of a balanced carbohydrate meal have led to faster RTs, and more accurate responses. Yet, it is crucial that this increase in glucose does not exceed the 7.8 mmol/L limit for healthy glucose levels, as this can result in a negative impact on RT.

**Guar Gum and Glucose**

Recent studies have supported the view that soluble dietary fiber is a beneficial component of our daily diet, irrespective of whether an individual is healthy, obese, or diabetic. In general, soluble fibers have been most effective in reducing the rise in blood glucose and insulin (Gropper & Smith, 2013). It has been consistently observed that the higher the viscosity of the soluble fiber, the better the glycaemic control. The most important attribute of soluble fiber is its potential ability to lower the postprandial glycaemic response (Torsdottir, Alpsten, Andersson, & Einarsson, 1989). To date, guar gum (a water-soluble galactomannan made from the seeds of the guar plant) has been the most effective supplement studied in this area of research. However, the effect of guar gum on the increase in blood glucose concentrations after a meal has produced conflicting findings. Studies that have administered guar gum by mixing it with glucose, milk, soup or mashed potato have shown the most beneficial effects by decreasing the peak of the postprandial glucose response, and also by maintaining glucose levels above participants' fasting baseline levels in healthy and diabetic participants (Feldman & Barshi, 2007). In comparison, when guar gum is administered by means of sprinkling over everyday meals most studies have failed to find an effect (Blackburn & Johnson, 1981; Schmidl & Abuza, 2000). So it seems that guar gum is most effective at reducing blood glucose when it is completely mixed with food (Groop et al., 1993).

The exact mechanism by which guar gum reduces the peak of blood glucose levels is not known, but various theories have been put forward. A common explanation is that the main factor slowing absorption in the stomach is guar gum's viscosity (Blackburn & Johnson, 1981; Malmlof, Nunes, & Askbrant, 1989). That is, guar gum works to inhibit the absorption of foods and nutrients by increasing the thickness of the unstirred water layer in the intestine, resulting in a reduced rate of glucose uptake (Schmidl & Abuza, 2000). It has also been suggested that the viscosity of soluble fibers actually reduces the rate of absorption due to impaired convection in the intestine (Braaten et al., 1991), which in turn, lowers the glycaemic index of the food consumed. Blackburn and Johnson (1981) concluded that as a meal's fiber content and viscosity increase,

gastric emptying is slowed. Research on diabetes shows that guar gum works to slow down the absorption of sugars in the small intestine (Ebeling et al., 1988). However, even though absorption is slowed, the amount of sugars eventually absorbed is still the same (Schmidl & Abuza, 2000). Thus, reducing the rate of absorption appears to only impact on the glucose peak. Yet, none of these theories are conclusive; it is possible that a number of other mechanisms are at work.

Generally, it has been widely accepted that guar gum is beneficial for glucose regulation. However, the best way to administer and consume the guar gum is a focus of current research. For example, Jenkins, Leeds, Gassull, Cochet, and Alberti (1977) conducted a study with healthy participants who after a 14-hour fast consumed either a portion of bread containing guar flour or a liquid glucose drink mixed with guar flour. Jenkins et al. found that the glycaemic response was markedly reduced in the bread condition in comparison to the liquid glucose and control (white bread only) conditions. Blood glucose levels after the meal of bread containing guar were significantly reduced below the control condition at 15, 45 and 60 mins. At 120 mins, when glucose levels had also markedly dropped in the control group, glucose levels stayed slightly above baseline concentrations in the guar gum - bread condition. These findings are similar to those of Braaten et al. (1991). They examined the effect of oat gum on plasma glucose and insulin levels, compared to the effects of guar gum. The study used healthy participants who consumed either a 50 g glucose drink, 50 g glucose mixed with oat gum or 50 g glucose mixed with guar gum. Mean plasma glucose levels above baseline were greater for the glucose drink than the oat gum and guar gum conditions at 20 and 60 mins, but when the glucose drink was consumed, the glycaemic response fell below baseline following the rise. Guar gum was also found to reduce insulin secretion, which in turn, would have acted to decrease the blood glucose peak and stabilize its concentration over the trial (Torsdottir et al., 1989). More recently, Nilsson et al. (2012) found that guar gum mixed with white wheat bread improved the glycaemic response by lowering the blood glucose peak, and enhancing cognitive performance on a selective attention task.

The amount of guar gum that needs to be consumed (either as food or liquid) to produce a significant improvement of blood glucose levels by reducing the postprandial peak has also been examined in several studies. Wolever, Jenkins, Nineham, and Alberti (1979) varied both the form and dose in which guar gum might be given to be most effective in lowering glucose levels and preventing excessive blood glucose increases. Meals containing guar gum in a bread (solid) or soup (liquid) were given with either 5 g or 10 g of guar gum. Wolever et al. found that 5 g of guar gum in bread reduced blood glucose levels at 30 mins by 10% and at 45 mins by 15%, whereas 5 g of guar gum in soup had an improved effect by reducing blood glucose levels at 30 mins by 23% and 45 mins by 20%. The larger amount (10 g) of guar gum in both food conditions had an even greater improvement on glucose responses compared to the 5 g and control groups. This dose not only reduced blood glucose levels, but also reduced the peak after meal consumption. Torsdottir et al. (1989) examined the effects of both small and large doses of guar gum when it was added to liquid meals, finding that dose size had little or no effect. The presence of guar gum (at any dose amount) significantly reduced the rise in blood glucose levels compared to a meal without guar gum. However, insulin levels were lowered with a larger dose of guar gum, suggesting that guar gum might have beneficial effects beyond just regulating blood glucose levels.

The effects of guar gum have also been observed in relation to medical issues, including obesity and diabetes. Guar gum has previously been identified for its ability to regulate glucose metabolism and increase feelings of satiety (Schmidl & Abuza, 2000). Poor glucoregulation has also been associated with weight gain and obesity. Krotkiewski (1984) looked at the effects of guar gum on postprandial blood glucose levels in obese individuals over a period of eight weeks. When 10 g of guar gum was consumed the glucose peak was significantly reduced at 30 mins. Body weight and fat decreased in all participants, suggesting that a lowered glycaemic response could be directly related to weight loss.

Groop et al. (1993) examined the effects of guar gum on glucose tolerance over one year with diet-treated participants with non-insulin dependent diabetes

mellitus. Guar gum was administered in either a liquid or solid food form. Groop et al. found that guar gum improved long-term glycaemic control and postprandial glucose tolerance in these participants. Similarly, Ebeling et al. (1988) investigated the influence of dietary guar on the metabolic control and insulin sensitivity in patients with Type 1 diabetes. Over a 4-week period participants ingested either 5 g of guar or a placebo before daily meals. Compared to the glucose condition, lowered postprandial blood glucose levels were observed in the guar gum condition, with a reduced postprandial rise after breakfast and lunch. Ebeling et al. found that the beginning baseline of fasting blood glucose was similar in both groups, with a peak rise at 90 mins. After four weeks, it was seen that fasting blood glucose levels were comparable in both groups; however, the rise in glucose was significantly reduced after consuming the guar gum. Additionally, a significant decline in insulin requirements was found, suggesting improvement over time in the body's sensitivity to insulin. Findings from these studies of the effects of guar gum on obesity and diabetes suggest that the supplement of guar may be considered as an additional tool to improve weight loss and glycaemic control.

However, there are important dangers of self-administering guar gum, as the supplement has been found to have various side effects. Aro et al. (1981) stated that the amount consumed needs to be carefully controlled, as guar gum can swell up to 20 times its original size and there is a risk of blocking the gastrointestinal tract and obstructing other nutrients from being absorbed. Guar gum has also been found to have laxative effects, and cause abdominal discomfort (Aro et al., 1981).

**Kiwifruit and Glucose**

In recent years, the nutritional value of kiwifruit has been promoted all around the world, particularly within New Zealand. Kiwifruit contain large amounts of vitamins, minerals, antioxidants and fibers that can directly benefit and maintain a healthy body function (Zespri Kiwifruit, 2013). Kiwifruit have a low glycaemic index, are low in fat and also have a high fiber content, which can effectively improve digestive functioning.

Green kiwifruit reportedly contain levels of vitamins and minerals that are slightly higher than that of many other types of fruit. Kiwifruit are known to have high levels of vitamin C, which plays a significant role in healthy well-being (Zespri Kiwifruit, 2013). Gaining adequate amounts of vitamin C relies completely on an individual's diet, as the body cannot produce vitamin C itself. This particular vitamin plays an important role in the functioning of the immune system, metabolizing energy, reducing tiredness and fatigue, and increasing iron absorption (Will & Byers, 1996). Vitamin C is structurally similar to glucose, in that they both use the Glut-1 receptor to enable transportation into cells around the body. However, high levels of blood glucose can take over the Glut-1 receptors and limit the amount of vitamin C that enters the body (Will & Byers, 1996). This finding was supported by Afkhami-Ardekani and Shojaoddiny-Ardekani (2007) who found that diabetic patients had a decreased level of vitamin C when experiencing a hyperglycaemic state. As a result, it has been hypothesized that low GI foods (such as kiwifruit) may be beneficial in transporting vitamin C throughout the body, as they create a slow and controlled rise in blood glucose levels after a meal. However, it is not known whether the low GI food consumed needs to contain sufficient vitamin C itself or a low GI can be consumed alongside any food that has the vitamin (such as kiwifruit).

Carbohydrates are a major component of kiwifruit, with the available carbohydrates being a mixture of glucose, fructose and sucrose, in proportions of 2:2:1 (Monro, 2013). Of these sugars, glucose has a GI of 100%, fructose 19% and sucrose 68%. According to Monro (2013), the value for the GI of kiwifruit can be calculated from the proportions of sugars in the fruit and their published GI values to be 60.6%, placing them in the medium range for GI value. However, additional *in vivo* clinical studies have measured the GI for kiwifruit, giving the fruit a GI value of 49 (a low GI rating is <55) (Chen, Wu, Weng, & Liu, 2011). Nevertheless, this discrepancy may be caused by the fact that Monro used *in vitro* measures of GI, with calculations being based on the combining sugars. Clinical studies have used *in vivo* measures, calculating the GI value from actual human digestion and blood glucose responses. This suggests that there may be other factors operating within the body that reduce the glycaemic

response to kiwifruit, lowering the overall measure of GI. Kiwifruit have been found to be a natural digestive aid, with their high level of fiber working to keep a low glycaemic impact per serving of food. The cell wall of a kiwifruit remains undigested in the stomach and small intestine. Here, the dietary fiber can swell to up to four times its original size, which may be important in adjusting carbohydrate digestion and absorption. When the digested contents have then settled into the gut, they can reduce the rate of glucose absorption into the bloodstream by 40% (Monro, 2013), and can reduce the digestive mixing. By reducing the rate of absorption of nutrients, kiwifruit can work to control the glycaemic response as well as increasing feelings of satiety.

The effects of kiwifruit have been examined in conjunction with guar gum, when both components are mixed together. Monro (2013) used an *in vitro* digestive system to test the retardation of mixing of kiwifruit and kiwifruit and gaur combined, modeling what would usually occur in the stomach after consumption. To represent the intestine, a plastic tube (26 cm in length) was used and the asymmetrical moving constriction was obtained by rolling a 7.5 g ball down the tube, replicating the velocity of what occurs in the small intestine. The digestion-resistant remnants of kiwifruit, guar gum, and a combination of the two have been shown to retard processes in the gut that influence the rate of sugar absorption (Mishra & Monro, 2012). These processes include digestion rate, mixing, and diffusion, and are influenced by the rheological (thickening) effects of the kiwifruit remnants and guar on gut contents. As a result of the slowing of digestion, mixing and diffusion, the rate at which sugars reach the gut wall for absorption is reduced. Therefore, the rate of transfer of sugars into the blood is reduced, and so the measured blood glucose response is correspondingly reduced. Overall, both of these natural supplements have been found to have functional attributes of potential importance to health.

Overall, both kiwifruit and guar gum have been found to have important health benefits. They both have similar mechanisms that work to considerably lower the postprandial glycaemic response to food, and are completely natural and easily accessible products.

**Inconsistent Findings: Potential Contributing Factors**

Many of the studies examining the effects of glucose levels on cognitive performance have produced inconsistent results. For example, some studies have found glucose to significantly impair cognitive performance, where others have found no effects at all. Some of these inconsistencies may be caused by various moderating factors that have not been taken into account, including participant age, task difficulty, quantity of glucose consumed or the type of carbohydrate that was consumed to influence glucose levels. The following section explores some of these possible reasons for replication failures and other inconsistent findings.

A predominant finding in numerous studies is that the effect of glucose on cognitive performance is dependent on the level of task difficulty. Several investigations have demonstrated that more demanding and complex cognitive tasks more rapidly deplete glucose levels in the brain, resulting in an *improvement* in performance (Feldman & Barshi, 2007). However, others have criticized this finding, concluding that complex and demanding tasks are cognitively challenging because there is insufficient glucose available to perform such tasks (McCrimmon, Deary, & Frier, 1997). Simpler tasks usually have been found to be less sensitive to the effects of glucose (Driesen et al., 1995). The finding that cognitive performance is enhanced when blood glucose levels are falling suggests that cognitive processes are fueled by fully utilizing the available glucose. Scholey et al. (2001) argued that intelligence tests should be administered to participants prior to testing in order to gauge how difficult the cognitive tasks need to be to achieve a reliable measure of the effects of glucose. Older adults, whose cognitive ability declines with age, and individuals with learning disabilities, have been found to show glucose sensitivity to simpler tasks (Manning et al., 1990), supporting the idea that tasks need to be assigned relative to the individual's abilities. Simple tasks have also been criticized for their lack of external validity. That is, the various cognitive tasks used in laboratory-based studies often are not applicable to real-life situations, limiting the extent to which results can be generalized to everyday settings (Feldman & Barshi, 2007).

Additionally, the effects of glucose have been observed to be more prominent when distracting stimuli are present in cognitive tasks. For example, the ability to recall words while being distracted by a dull tone was impaired when participants' blood glucose levels were low (below 3.8 mmol/L) (McCrimmon et al., 1997). In effect, hypoglycemia slows down the brain's ability to gather information through the auditory system, and/or how efficiently it is organized and stored in memory (Scholey et al., 2001).

The time of day a study is completed and task duration have also been found to affect glucose levels and cognitive performance. Research findings have varied in regards to the most optimal time to administer glucose before completing the cognitive tests. Some studies conduct cognitive tests straight after glucose consumption and others hours later. These times are related to rising and falling blood glucose levels. Benton and Owens (1993) found that rising blood glucose levels improved memory on word recall, within one hour of glucose consumption. Benton et al. (1994) found that RT was faster when blood glucose levels were rising (5.2 to 6.5 mmol/L) than when they were falling (6.0 to 5.1 mmol/L). In comparison, Rodgers and Lloyd (1994) found that verbal memory performance was improved over the course of the morning when blood glucose was maintained, with the greatest difference at 210 mins. However, these findings then raise the question as to whether a smaller, slower rise in glucose (such as with low GI food) or a rapid rise (high GI) are more beneficial for cognitive performance levels. Feldman and Barshi (2007) argue that testing should be conducted at several time points after food consumption to ascertain an optimal time for testing. This may well be the case, but few investigations have the time and resources to carry out such an exhaustive procedure. In any event, it has already been established that cognitive performance is most sensitive to the effects of glucose in the morning, possibly as a result of the overnight fast (Dye & Blundell, 2002). The positive glucose response of eating low glycaemic foods for breakfast has also been seen to carry over to lunch (Jenkins et al., 2000). If this factor is not taken into consideration, cognitive performance after a lunch meal may be inaccurately attributed to the effects of glucose at that time.

Smith, Kendrick, and Maben (1992) reported that inconsistencies in their study may have been the result of certain tasks not being long enough, or alternatively, not being completed at various time points over the testing period. Some studies did not observe an effect at 20 mins post consumption (Smith et al., 1992); however, others have at 30 mins (Green et al., 2001). Although investigations with prolonged cognitive tasks are limited in number, those that have been conducted show an increased sensitivity to glucose to tasks that are longer (Groop et al., 1993). As cognitive tasks go on, glucose levels have been found to deplete more rapidly, indicating that the brain is using up the glucose (energy) to perform the tasks (Scholey et al., 2001). Studies that have failed to find an effect may not have considered this factor, and completed tasks before an adequate amount of time had been given to allow sufficient uptake of glucose to the brain.

The age of participants varies considerably across studies, often without age being considered as an independent variable. Manning et al. (1990) reported that glucose levels in older adults (65+) influence cognitive performance, especially memory, more than any other age group. Age has been found to relate directly to changes in an individual's ability to effectively regulate and metabolize glucose in the body and brain. Individuals whose glucose levels fall well below baseline, or stay relatively high after peaking, are generally perceived to have poor glucose regulation, which in turn, is associated with poorer performance on cognitive tasks. Therefore, it is important that the interaction between age and glucoregulation are taken into consideration when analyzing results from similar studies. Bellisle et al. (1998) identified impaired glucose regulation as a predictor of cognitive impairment in older adults. However, an individual's ability to regulate glucose at any age has been found to impact on cognitive performance (Berdanier, 1996). Younger individuals with poor glucoregulation have been found to perform worse on memory tasks than older adults with a superior ability to regulate glucose (Messier & Gagnon, 1996). Another factor that is frequently not considered is that an individual's glucoregulation occurs at different times in different people (Gropper & Smith, 2013), meaning that each individual's body responds to glucose at a unique rate, with varying intensity. Therefore, blood glucose levels can peak and fall at different time points over the

period of a study. This may be overcome by identifying individuals' glucose regulation over the day before commencing the study, and potentially aiming to group together those with similar regulation responses. However, reducing this variability by choosing a more homogeneous sample may be futile as group research deals with this issue. For example, the statistical analysis using an ANOVA deals with individual variance.

The nature of food used in research examining glucose levels and cognitive performance is a further variable that may influence research results. Donohoe and Benton (2000) concluded that controlling what participants have to eat prior to glucose administration is essential. They found that there was no association between glucose and cognitive performance when breakfast was consumed by participants but without the content of the meal being controlled. It was noted that the breakfast consumed varied between nothing to cereal or bread. Coffee, also consumed by some participants, has been found to cause a rapid rise in glucose levels, quickly followed by a crash below baseline levels (Smith, Clark, & Gallagher, 1999). However, when a breakfast or other meal is provided as part of a study, it is often the case that it includes items some participants would not normally eat, potentially resulting in an abnormal glucose reading for them, as their body is adapting and responding to a meal they are not used to (Donohoe & Benton, 2000). Examples of such foods are soup, bread, potatoes, and supplements such as saccharine (Feldman & Barshi, 2007). Carbohydrates have been found to be less tolerated by some participants later in the day, and are highly associated with fatigue and a post-lunch dip in performance (Gibson & Green, 2002). Additionally, the amount of food needed to provide the body with sufficient energy varies across people and across genders. Women are found to eat less and to be more sensitive to the negative effects of carbohydrates and fats after lunch, effects such as fatigue and a rapid increase in blood glucose levels (Wolever, 2003). Therefore, determining the optimal type and size of meal for a group of participants can be challenging. Berdanier (1996) asserts that researchers should consider body mass in relation to meals and glucose regulation. Overweight people are at a higher risk of diabetes, and also have a higher tolerance of carbohydrates and additional heavy foods.

Contextual factors in regards to an individual's general health and daily living have also been overlooked in a large percentage of studies to date, including alcohol intake, caffeine, exercise, stress and medications. Various factors have been found to influence blood glucose levels, which may then influence an individual's ability to regulate glucose and adequately perform cognitive tasks, aside from medical and health problems. Alcohol has been found to decrease blood glucose levels by inhibiting the production and release of glucose from the liver, limiting the overall amount of glucose that is supplied to the brain (Smith et al., 1999). Exercise can also decrease blood glucose levels, with the effects lasting up to 48 hours. Therefore, any exercise completed leading up to testing time can impact on the body's ability to produce glucose (Feldman & Barshi, 2007). In comparison, stress causes a significant increase in glucose levels, which have been found to have a higher and longer lasting peak (Scholey et al., 2001). Numerous studies have demonstrated that prolonged peaks in glucose levels are associated with a decline in cognitive performance from baseline performance (Benton & Owens, 1993; Lobmann et al., 2000; Yki-Jarvinen, 1990). Lastly, caffeine and certain medications have varying effects on glucose levels, sometimes with profound effects on accompanying cognitive tasks (Smith et al., 1999).

Scholey et al. (2001) suggest that the reason performance on demanding cognitive tasks is easily influenced by the body's current level of glucose may be explained by physiological mechanisms that occur in the body due to the more intense nature of the given task. Cognitively demanding tasks have been associated with an increased heart rate, for example, which in turn increases the rate at which glucose reaches particular areas of the brain (Messier & Gagnon, 1996; Kennedy & Scholey, 2000). Physiological symptoms of hypoglycemia, including shakiness, dizziness, and hunger pains, may account for poorer performance due to these distracting symptoms (Mizock, 2001). Such findings suggest that there are various mechanisms at work when blood glucose levels are low; poor cognitive abilities may not be due only to reduced availability of glucose.

Finally, a lack of standardization of participants in studies, beyond being considered healthy or being free from diabetes, is another potentially confounding variable. Ranges of samples have been used in research, including varying ethnicities and participants with natural or induced hypoglycemia (Feldman & Barshi, 2007). Induced hypoglycemia sometimes produces different behaviour to low glucose levels in participants with natural, diabetic hypoglycemia (Lobmann et al., 2000). For example, individuals who have been diagnosed with natural hypoglycaemia are found to be more resistant and used to the symptoms they experience, whereas individuals who have hypoglycemic symptoms induced are more likely to be highly sensitive to it effects (Feldman & Barshi, 2007). Therefore, the two groups cannot be validly compared (Driesen et al., 1995).

Global studies have reported that certain ethnic groups and minorities have an increased risk of developing diabetes (Friedrich, 2012; Herman & Cohen, 2012). For instance, individuals from a South Asian ethnicity are six times more likely to develop Type 2 diabetes; African individuals are three times more likely than any other population (Friedrich, 2012). In New Zealand, diabetes is most common among Maori and Pacific Islanders, being three times more likely than for other New Zealanders (Ministry of Health, 2013).

A large number of studies have concluded that there are no significant differences between gender and blood glucose responses (Benton & Owens, 1993); however, gender has been identified as a confounding factor in research by various critics (Craft et al., 1994; Feldman & Barshi, 2007). Craft et al. (1994) found that there was a significant gender difference in their study, with middle-aged men being more susceptible to the negative effects of low glucose levels than middle-aged women and younger males. It has been argued that there are prominent gender differences in blood glucose levels and regulation (Faerch, Borch-Johnsen, Vaag, Jorgensen, & Witte, 2010), mainly due to differences in physiology between men and women. For example, body size and the daily amount of food and calories consumed tends to be higher in males than females (Brand-Miller et al., 2004). Such studies should be taking into account

when considering the quantity of glucose/ food administered during trials and whether it should be the same for both genders.

**Summary**

Significantly low and high blood glucose levels are not only found in those with diabetes these days, but are also seen in healthy people due to the food that they eat. Individuals can eat high levels of refined sugars and carbohydrates, not realizing the effect it may be having on their blood glucose levels. High and low blood glucose levels can have detrimental effects on overall health and well-being, including fatigue, headaches, weakness, faintness, irritability, sweating and it can also impair mood and judgment. Low blood glucose levels limit the brain's main source of energy for everyday functioning. Seriously low levels have been found to cause diabetes, to slow cognitive development and may also lead to brain damage.

Brand-Miller et al. (2004) argue that what an individual eats should be determined by their body weight and height, in order to gauge the amount of calories they should consume. In addition, the food that is consumed should be evaluated on GI values. Low GI foods have been found to cause a slow and steady increase in blood glucose levels, lowering the overall peak, and maintaining glucose over prolonged period of time. In comparison, foods that are high in GI cause rapid glucose swings, resulting in a 'sugar crash' below baseline. Low GI foods make people feel fuller for longer, and have also had a positive impact on following meals (Liljeberg et al., 1999). Moreover, the benefits of low GI foods are increased when nutritional supplements are added to them, such as guar gum.

The effects of glucose levels have been found to influence cognitive performance, with previous studies covering various areas of performance, including attention, memory, and RT. Low blood glucose levels were consistently found to increase RT on attention tasks, impairing concentration and also motor control. Attention was improved when blood glucose levels were

rising, but only when they stayed within the normal range (below 7.8 mmol/L). Glucose levels rising into the hyperglycaemic range again began to negatively affect attention abilities. Memory and the way it can be influenced by blood glucose levels has been a focal point for many studies, with many consistently finding significant changes in memory in research with older aged adults, but not in those with middle-aged or younger. What was used to manipulate glucose levels has also been varied in studies involving memory. Benton and Owens (1993) found that only word recall improved after a glucose drink, while Manning et al. (1990) found that after a low GI carbohydrate breakfast participants improved on spatial memory, and word list recall. Lastly, the measure of RT is included in numerous cognitive tests, and has been found to be dependent on the time of day and also what is consumed to alter memory. It has been shown that an increased blood glucose level decreases RT, suggesting that the higher level of glucose available to the brain facilitates optimal performance. Similar to findings in attention studies, RT is significantly increased by low (hypoglycaemic) and high (hyperglycaemic) glucose levels. Feldman and Barshi (2007) argue that, ultimately, performance comes down to the individual's ability to regulate glucose.

When added to low GI carbohydrate meals, guar gum has consistently been found to lower the postprandial glycaemic response and effectively control glucose levels. Guar gum works to slow the absorption of food in the stomach, creating a slow release of glucose over a longer period of time. It has also been found to increase feelings of satiety, which makes the supplement an ideal natural method to aid weight loss and control diabetes. Kiwifruit is also a natural food that has been found to have various benefits to health, including vitamin C, antioxidants and a good source of fiber. Kiwifruit has a low GI value, and similar to guar gum, has a slow release rate when digested. Non-digested remnants of kiwifruit can swell up to four times original size, and works to retard mixing of food in the small intestine. As a consequence, glucose diffusion is slowed and individuals report feeling fuller for longer.

Although many studies have shown the effects of glucose on cognition, others have obtained no effects. This inconsistency can be explained by a number of factors, including task difficulty, timing of cognitive tests, length of task, participant age and contextual factors. A large number of studies have concluded that cognitive tests need to be relatively demanding and complex to show any effects of glucose. It has been shown that performance on demanding tasks improves when glucose levels in the brain are depleting, suggesting that participants were using up the energy from supplied glucose for optimal performance. Scholey et al. (2001) concluded that the type and level of a cognitive task should be relative to each individual's abilities.

Morning has been found to be the best time of day to complete testing, possibly due to the fact that participants are able to complete an adequate amount of fasting time overnight. In regards to what is the best time lapse between consumption and testing, this still remains unclear. Improvements have been found straight after consumption (when blood glucose is rising) and also as long as 210 mins after (when blood glucose levels have dropped but leveled off above baseline).

The age of participants has been another factor that could have contributed to inconsistent results, with many studies indiscriminately using participants of all ages. Older adults have been found to be more susceptible to the effects of glucose, particularly in regards to memory. However, Manning et al. (1990) state that age is related to glucoregulation, with older adults having a poorer ability to adequately regulate glucose.

Contextual factors such as stress, exercise, medications and alcohol consumption have all been found to influence glucose levels and are hard to control and standardize in large sample groups. Ethnicity is also another factor that has not been controlled for in previous research, even though it is widely known that some ethnicities have a higher risk of developing blood disorders, such as diabetes. For example, New Zealand Maori and Pacific Islanders have a higher percentage of diabetes and high blood pressure in comparison to others.

A further difficulty is that people with diabetes have reportedly been found to have a higher level of tolerance to the symptoms of high and low blood glucose levels compared to normals. Thus, their cognitive performance may be differentially affected.

Finally, the amount of food given to alter blood glucose levels and increase energy can vary between men and women. As a result of their body size and calorie intake, men often need a larger portion of food than women. So, where this has not been accounted for, blood glucose levels taken may not actually be an accurate rating of normal levels for that individual. Hence, arguments for why cognitive performance changes in these groups may sometimes be invalid.

Overall, there are a number of factors that can account for the inconsistencies found in the literature so far, with many critics still claiming that while glucose can be directly correlated with energy, it has little effect on cognitive performance. Cognitive performance can be influenced by various factors at work in the human body; it may be difficult to show definitively that changes are the result of particular glucose levels.

**The Present Study**

The present study is a replication and extension of the work by Nilsson, Radeborg, and Bjorck (2012). Nilsson et al. evaluated cognitive performance in the postprandial period after two types of bread breakfasts (one including guar gum), resulting in significantly different postprandial glucose responses. Their study used 40 participants, 28 women and 12 men, aged between 49 and 71 years. The participants were initially examined for normal fasting blood glucose concentrations and also normal-to-overweight body mass index. Any individuals who were considered over-weight or who had an abnormal BMI would have been excluded from the study.

On the first visit, fasting glucose concentrations and individual glucoregulation were determined after an overnight fast, using finger-prick capillary blood

samples. One blood sample was taken at 8 a.m. (baseline glucose level), and then a glucose drink (50 g glucose in 250 ml water) was consumed straight after. Following this, blood samples were taken at 15, 30, 45, 60, 90, 120 and 150 mins. In the second and third sessions the participants ingested either a white wheat bread (WWB) or a white wheat bread with the added supplement of guar gum (G-WWB). The same participants consumed both breakfasts on two separate days at least one week apart following an overnight fast. Every participant had each breakfast once, with 20 randomly chosen to have the WWB breakfast first and the G-WWB breakfast second, with the reverse order for the remaining 20 participants.

Each session tested performance on working memory (WM) and selective attention (SA). The WM test was completed four times over each testing day, at 90, 135, 180 and 225 mins. The SA test was also completed four times each day at 75, 120, 165 and 210 mins. Breakfast consumption was considered to be 0 mins. It was found that the G-WWB breakfast resulted in a lower postprandial blood glucose peak followed by a low but steady net increment in glucose, above the fasting value, from 60 – 240 mins. In the late postprandial phase (210 – 240 mins after the meal) the G-WWB participants had a higher blood glucose concentration in comparison to the WWB participants. The SA test showed better performance at 120 min following the G-WWB breakfast, and differences in cognitive performance became more pronounced in the second half of the SA test, considered to be more demanding than the first half. Though, as the two types of tests were not identified, there is some doubt on its difficulty level. There were no significant differences for type of breakfast or RT for the working memory test. However, individuals with superior glucoregulation performed better on the cognitive tasks in both the WWB and G-WWB groups in comparison to those with poorer glucoregulation. Moreover, individuals who had superior glucoregulation also performed better on the second part if the SA test after consuming G-WWB.

Overall, this study found cognitive performance on SA improved in the later postprandial stage (75 – 225 mins) after the G-WWB meal was consumed. The present study sought to replicate the effects of guar gum on cognitive

performance, but by using kiwifruit in the place of bread. The reason kiwifruit was used was to add to the previous *in vitro* laboratory measurements that had suggested kiwifruit would help in the regulation of blood glucose responses to foods. Kiwifruit is considered to be an important export crop for New Zealand, and any scientific findings that may contribute to its "health halo", such as a less acute blood glucose response, reduced hypoglycaemic overshoot, and improved cognition would be beneficial in marketing.

**Objectives**

The present study aimed to determine:

a) The effects of kiwifruit and guar gum (individually and combined) on reducing the postprandial glycaemic response to carbohydrate foods, and whether this reduced response allowed cognitive performance to be maintained.

b) The effects of kiwifruit and guar gum (individually and combined) on perceived levels of hunger at 180 mins from 0 mins (when breakfast was consumed).

# METHOD

## Participants

Twenty participants, 4 males and 16 females, aged between 26 and 66 years (*M* = 36.30, *SD* = 10.94) volunteered to take part in the present study. All participants were recruited from advertisements placed around the Massey University Campus, Palmerston North and The New Zealand Institute of Plant and Food Research, or by word-of-mouth. Participants were reimbursed for their time with a $20 supermarket voucher for each trial completed.

All materials and procedures used in the present study were approved by the Health and Disability Ethics Committee, Wellington, New Zealand (Protocol 14/STH/77).

## Group Assignment

The present study was an intervention study, and used a within-subjects design. Thus, each participant took part in each of the four experimental diets. Each participant received the four breakfasts in a counterbalanced, random order (see Table 1), with each breakfast type being consumed five times each week.

Table 1

*A Visual Representation of the Within-subjects Experimental Design. Each Breakfast was Consumed Five Times each Week with the Order of Breakfasts Randomised.*

| | | | |
|---|---|---|---|
| WB | WB + KF | WB + GG | WB + Both |
| WB + KF | WB + GG | WB + Both | WB |
| WB + GG | WB + Both | WB | WB + KF |
| WB + Both | WB | WB + KF | WB + GG |
| 1 | 2 | 3 | 4 |

TRIAL WEEKS

*Note.* WB = Weet-Bix; KW = Kiwifruit; GG = Guar Gum; Both = Kiwifruit + Guar Gum

The criteria for inclusion in the study were the absence of any current or pre-existing medical conditions such as diabetes, obesity, blood disorders, and physical or neurological diseases. If a participant disclosed they had any previous illnesses, or an immediate family member had, this was discussed to check for any medications or activities that would interfere with the study. If any serious concerns were raised, then that participant was excluded from the study. These factors were assessed through a screening questionnaire during initial contact with the participant.

**Apparatus**

The cognitive tasks were completed on a 15-inch HP laptop supplied by Plant & Food Research. Upon opening each task, a set of instructions was displayed and participants could continue when they were ready by pressing the space bar, as indicated. Scores on each task were recorded on the computer and saved to the coded file of each individual participant. Soundproof headphones were worn for each task to block out any distractions. A computer mouse was also used for tasks where participants were required to move between certain objects on the screen.

The glucose monitor (HemoCue Glucose 201 DM RT Analyzer) was used for measurement of blood glucose levels. A blood sample was obtained from the participants by finger-prick, and the blood transferred to a glucose-sensing strip that was fed into the monitor. The monitor then returned a reading of the amount of glucose in the peripheral blood supply. When testing the accuracy of the HemoCue over two days, using five blood samples each day, results were almost identical. Day one had a mean of 5.2, where day two had a mean of 5.1. The coefficient of variation for the two days was 2.2% and 1.4%, respectively.

## Procedure

### *Screening/ Training Session*

Participants were tested in the Electrophysiological Laboratory at the School of Psychology, Massey University, Palmerston North and the Blood Collection room at the Plant & Food Research building, Turitea, Palmerston North. Two participants were tested in their own office for their convenience and to limit the walking distance between blood samples.

After initial contact, where potential participants expressed interest in volunteering for the study, an information sheet (see Appendix C) was emailed out to each person regarding the details of the study. Those who were still interested were screened for eligibility and a time was made for an initial meeting and practice session. At the first meeting participants were given a consent form (see Appendix D) and health questionnaire (see Appendix E) to read over, fill out and sign. Once completed, participants were given the opportunity to ask any questions, and asked if they would like to keep their small blood samples or have them disposed of.

At the initial meeting a blood glucose sample by finger-prick was taken first. A small lancet was used to make a small puncture wound in the finger and the drop of blood obtained was transferred to a glucose-sensing micro-cuvette, which was then inserted into the monitor. The blood glucose level reading was noted and used to make sure no participant was diabetic (none were).

Practice versions of the cognitive tests then followed, in order to limit practice effects in the main trials. Participants were asked to take the time to read over the test instructions carefully and to ask any questions. Once they had completely run through a test, participants had the opportunity to repeat the test, with the exception of the word recall task, which could be completed only once. In any event, all participants chose to complete each test once only in this practice run.

Once all the tests had been completed, the screening/ practice session was at an end. The participants were thanked and asked to make appointments for one morning a week over the next four weeks. They were asked to fast for 12 hours overnight before the arrival at each session.

***Experimental Procedure***

On arrival participants were required to have a 15-min rest period before starting the trial, to bring glucose levels back to a resting state after morning activity (e.g., getting to the session). After the rest period the first blood sample was taken, giving the baseline reading for that morning. Between blood samples and cognitive tests participants were able to do light reading or work that was not overly demanding.

Following the initial blood sample, participants completed the first round of all five cognitive tests. Next, participants were given their experimental breakfast for that day, presented in a cereal bowl with all ingredients crushed up and mixed together. Each breakfast was made as similar as possible to all others and to conceal what participants were consuming. However, due to the colour and consistency of the kiwifruit, this was harder to disguise. The time point at which breakfast was given was considered as 0 mins on the session timeline, and participants were given 10 mins to consume the breakfast. Each person was given 200 ml of room temperature water in a clear plastic cup to have with the breakfast.

Twenty mins after the start of breakfast the second blood glucose reading was taken. After another 20 mins the third blood sample was taken at 40 mins, and the fourth at 60 mins. Blood samples were then delayed out to 30 min intervals. At the 90 min time point the fifth blood sample was taken, and then participants completed their second round of cognitive tests. The tests were given in a random order each time, and care was taken not to use the same order more than once for each participant. The cognitive tests took an average 15 min to complete. Another 30 mins after the last blood sample (around 15 mins after the cognitive tests were completed), the sixth blood sample was taken (120 mins).

The seventh blood sample was taken at 150 mins, and the last sample at 180 mins. Then the third round of cognitive tests was completed, again in a randomised order. Finally, participants completed the satiety scale and were then provided with lunch for that day. Before participants left each session they were thanked, and the time for the following week was confirmed.

**Cognitive Measures**

The five cognitive tasks used in the present study consisted of Word Recall, Serial Sevens, Trail Making B, the Stroop Task and a Choice Reaction Time task (see Appendix A). All tasks chosen for this study have previously been used in similar studies, and have produced significant results (Benton et al., 1994; Donohoe & Benton, 1999; Fischer et al., 2001; Holmes et al., 1986; Kennedy & Scholey, 2000).

***Choice Reaction Time***

The Choice Reaction Time test was a 4-choice test, where participants had to choose from four stimuli and press the key matching their choice as fast as they could. At the beginning of each trial participants were instructed to fixate on an 'X' presented on the centre of the screen. After one second, four boxes appeared in a line across the screen, with three being yellow and one being orange. Participants were required to identify the new position of the orange box on each trial as quickly as possible using the 'Z', 'X', 'N', and 'M' keys on the laptop keyboard. So, for example, the Z key was pressed if the left-most box was orange, the X key of the second from left box was orange, and so on. Participants were instructed to use their left middle finger and index finger for 'Z' and 'X', and their right index and middle finger for 'N' and 'M'. Three of the participants were left-handed.

At the beginning of each test participants were given a short practice version, following the initial instructions. The practice trials had a beep for any incorrect responses; however, during the main trial there was no sound feedback. Participants pressed the spacebar when they were ready to begin the main trial.

***Word Recall***

The word recall task consisted of 20 words, that were selected from a list of four-letter words occurring with equal frequency in the English language (Kucera & Francis, 1967). Word recall is considered an immediate free recall task and predominately measures short-term memory. At the beginning of the task, participants were presented with a set of instructions informing them that a series of 20 words would appear on the screen for three s each, and they should try to remember as many as possible. Following the presentation of the words, participants were asked to recall as many words as they could by typing them into a blue box on the screen, and to press the key F12 when they were sure they could not remember any more.

The computer programme did not record subsequent typings of the exact same word, and any words typed that were not on the original list were still recorded but at the bottom of the page that was saved for that individual participant.

***Trail Making B***

As previous studies had found the Trail Making A task to be too simplistic for research on cognition and blood glucose levels, it was left out of the present study and only Trail Making B was used (Kennedy & Scholey, 2000). Trail Making B is a measure of visual attention and task switching consisting of scattered circles on the screen, with half containing letters A - L and the other half numbers 1 - 12. Participants were required to connect the circles in the ascending sequence A to 1, 1 to B, B to 2, 2 to C and so on, until a line connected all the circles. If a mistake was made, the computer programme would not connect the line to the next circle. There were 24 circles altogether, with 12 letters, and 12 numbers. The time taken to complete the task and the number of incorrect moves were recorded.

Participants were given a series of instructions on the first screen and when they were ready they had the opportunity to use a short practice version of the task before moving on to complete the main task.

***Serial Sevens***

Serial sevens is a mental arithmetic task, measuring information processing speed, concentration and short-term memory, where participants are timed on how long it takes them to count backwards from a given number until they reach zero. In the present study participants were required to complete two versions of the task, firstly counting backwards from 100 by seven, and secondly, counting backwards from 104 by seven. Two versions were used to limit practice effects. If participants got lost on this task they were given one opportunity to start again for each task. If a second chance was used their time automatically started again from zero seconds. If they failed a second time, then they were given a time score of zero. All scores of zero were included in the analysis.

At the beginning of each test, participants were initially presented with a set of instructions and were not given any practice trials. A short computer-generated 'ding' sound was used to indicate if a number was typed correctly or incorrectly. If an answer was incorrect, the participant could not move forward until the correct number had been typed in. After the first task was completed, participants were instructed to start the next serial sevens task.

***Stroop***

The Stroop task examines the relationship between interference and RT, and is thought to be a measure of concentration and attention (Lezak, Howieson, Loring, Hannay, & Fischer, 2004). There are two subgroups of trials in the Stroop, congruent and incongruent. The congruent trials occur when the word printed on the screen is the same as the colour it is presented in. For example, the word 'green' is written in the colour, green. The incongruent trials are when the word printed on the screen does not match the colour it is presented in. For example, the word 'green' is written in the colour, red. The incongruent tasks are expected to take longer to respond to, as participants have to distinguish between the two forms of colour presented (word or colour).

Participants were instructed to identify as quickly as possible the colour of the word, not the word text. To do this, they responded by using the 'R' (red), 'G' (green), and 'B' (blue) keys. The response keys were arranged in a straight line on the keyboard (keys 'V', 'B', and 'N'), which had 'R', 'G', and 'B' stickers attached to the respective keys. Participants were given a short practice run and the opportunity to complete another series of practice trials if they felt the need to (by pressing the F12 key). They then moved on to the 72 main trials. During the trials, if participants did not respond fast enough to fall within the pre-determined response time window (2 s), a 'Too slow' message was flashed on the screen, but the remaining trials continued without pause. RT (to the nearest ms) was recorded for the congruent, incongruent, and overall trials.

**Breakfast Diets**

All breakfast diets were organised and prepared by The Institute of Plant and Food Research. Each component was initially measured and stored into small containers, ready to be mixed on the morning of each trial. All diets were consumed within a 10-min period. Table 2 displays the components of each diet.

Table 2

*Diet Components of each of the Four Experimental Breakfasts*

| | Diet 1<br>WB | Diet 2<br>WB+KF | Diet 3<br>WB+GG | Diet 4<br>WB+KF+GG |
|---|---|---|---|---|
| Weet-Bix | WB | WB | WB | WB |
| Kiwifruit | - | 200 g KF | - | 200 g KF |
| Guar gum | - | - | 10 g | 10 g |
| Sucrose | + | - | + | - |
| Fructose | + | - | + | - |
| Glucose | + | - | + | - |
| Water (180 ml) | + | - | + | - |

*Note.* WB = Weet-Bix, KF = Kiwifruit, GG = Guar Gum

The '+' sign indicates that the relative component was included in the diet, whereas, the '-' sign indicates the component was excluded from the diet.

***Preparation of Diet Components***

Kiwifruit at a ready-to-eat ripeness were peeled, halved, and frozen. When a sufficient amount had been collected for the whole trial they were disintegrated in a 3 L Hallde food processor with no added water, and 200 g portions were weighed into plastic containers and frozen. The Weet-Bix were purchased at a local supermarket and 47.3 g portions were pre-weighed into plastic containers. A bulk mixture of glucose (dextrose monohydrate): fructose: sucrose (2:2:1) was prepared and 19 g portions pre-weighed into containers. Lastly, 10 g portions of guar gum were pre-weighed into plastic vials.

As shown in Table 2 the breakfast without kiwifruit have an added sugar component of glucose, fructose and sucrose. This was determined by the total amount of sugar in kiwifruit, as a means to standardize the level of sugar intake across each breakfast. The following tables show the weights and portions of the combined sugars (Table 3), the available carbohydrates from the Weet-Bix (Table 4), and overall weights of the diet components used (Table 5).

*Sugar Analysis of Kiwifruit*

Total sugars = 9.15 g/100 g = 18.30 g /200 g kiwifruit.
Ratio of sugars in kiwifruit: glucose: fructose: sucrose (2:2:1)

Table 3

*Combined Sugars that are Equivalent to Kiwifruit Sugars*

| Sugars | Sugar ratios | Weight/diet (g) | Using dextrose monohydrate | |
|---|---|---|---|---|
| | | | Weight/diet (g) | For 50 diets (g) |
| Glucose | 2 | 7.32 | 8.04 | 402 |
| Fructose | 2 | 7.32 | 7.32 | 360 |
| Sucrose | 1 | 3.66 | 3.66 | 183 |
| Total | | 18.30 | 19.02 | 585 |

*Note.* Dextrose monohydrate is glucose with one molecule of water attached (91% CHO). Therefore, the required glucose weight (7.32 g) was multiplied by 100/91 = 8.04 g.

For 50 diets containing added sugars (Weet-Bix, and Weet-Bix + Guar Gum), using dextrose monohydrate as glucose, require 402 g dextrose + 360 g fructose + 183 g sucrose (see Table 3).

*Weet-Bix Carbohydrate*

Weet-Bix = 67% available CHO (nutrient information panel). Thus, 31.70 g Weet-Bix CHO = 31.70*100/67 g = 47.31 g Weet-Bix. Two Weet-Bix = 30 g, so, 47.31 g Weet-Bix = (47.31/30)*2 = 3.15 Weet-Bix approximately (see Table 4 and Table 5).

Table 4

*Total Available Carbohydrates in Diets*

| | Diet 1<br>WB | Diet 2<br>WB+KF | Diet 3<br>WB+GG | Diet 4<br>WB+KF+GG |
|---|---|---|---|---|
| WeetBix | 31.70 | 31.70 | 31.70 | 31.70 |
| Kiwifruit | - | 18.30 | - | 18.30 |
| Guar gum | - | - | + | + |
| Glucose | 7.32 | - | 7.32 | - |
| Fructose | 7.32 | - | 7.32 | - |
| Sucrose | 3.66 | - | 3.66 | - |
| Total | 50 | 50 | 50 | 50 |

Table 5

*Weights of Diet Components used (g)*

| | Diet 1<br>WB | Diet 2<br>WB+KF | Diet 3<br>WB+GG | Diet 4<br>WB+KF+GG |
|---|---|---|---|---|
| WeetBix | 47.3 | 47.3 | 47.3 | 47.3 |
| Kiwifruit | - | 200 | - | 200 |
| Guar gum | - | - | 10 | 10 |
| Sugar mix | 19 | - | 19 | - |
| Water | 180 ml[2] | - | 180 ml | - |

*Note.* An additional 200 ml water was drunk with all diets, allowing for the approximate water content in 200 g of fresh kiwifruit.

*Calculation of Guar Gum dose*

The guar gum dose used by Nilsson et al. (2012) was estimated in two ways; firstly, based on New Zealand flour composition, and secondly, based on the proportions used by Nilsson et al. given in a supplementary information paper.

The guar gum content of the test diets was based on the experiment of Nilsson et al. in which participants were fed "a low GI test bread made from white wheat flour supplemented with guar gum (15 % on dry weight basis)" (p. 1040). As the recipe for the bread was not given, the actual guar intake was estimated from the only information that was given for the composition of the guar bread: a dose of 179 g of the guar bread delivered 50 g available starch. If one assumes that all of the carbohydrate was derived from flour, and given that white wheat flour contains 75.8 % starch (monosaccharide equivalents) (Monro & Humphrey-Taylor, 1994), the amount of flour consumed was: 50 x 100/75.8 = 65.96 g flour. This amount of flour had been 15% substituted with guar gum on a dry weight basis. As flour is 86.7 % dry matter, the dry weight of flour used was: 65.96 x 0.867 = 57.19 g. As the guar gum was supplemented at 15% on a dry weight basis, the amount of guar gum per participant in the Nilsson experiment was estimated to be 15% x 57.19 = 8.58 g guar.

Based on the proportions used by the Nilsson et al. study, 71 g of guar was used per 400 g of flour. To get the same proportion for the present study: (65.96/400) * 71 = 11.7 g of guar gum. After obtaining the mean of the two calculations ((8.58 + 11.7)/2 = 10.14 g guar), it was determined that the present study would need to use a guar gum dose of 10 g per meal consumed.

***Preparation of Diets***

The dry ingredients for each diet were thoroughly mixed, as summarized in Table 4. The moist component (kiwifruit pulp or 180 ml water) was then added with rapid mixing to prevent formation of lumps. The diets were consumed with 200 ml water.

**Hunger Scale**

A satiety scale was administered at the end of each session to ascertain the participant's level of hunger after each test breakfast. The 11-point Likert Scale used ranged from 0 (not at all hungry) to 11 (extremely hungry) (see Appendix B).

**Design and Analysis**

The design for the present study was a 4 (Breakfast Type) x 3 (Testing Time) repeated-measures ANOVA, based on a completely within-subjects design (see Table 1). A planned approach was used to analyse the data, where a specific set of objectives (research questions) were formulated before the study began. Therefore, the data collected were analysed specifically with the research questions in mind. This approach lessens the risks of chance significant outcomes due to a large number of multiple comparisons where all possible statistical tests are conducted. This approach also increases the statistical power (SP) with which to detect any effects of the primary research questions (Pallant, 2010).

On initial analysis outliers were identified over the five cognitive tests, and in accordance with Allen and Bennett (2012), these outliers were modified by one unit higher than the largest non-outlier in the sample. (Re-running the analysis with the modified data showed that the outliers were having very little effect on the outcome).

All statistical analyses were completed using the statistical package SPSS for Mac, version 20.0. The *F* values and associated statistics are reported in the text. The dominant measures in the present study were the effect size and the significance level, and were calculated in accordance with the guidelines set by Cohen (1988). The effect size ($n^2$) was estimated using partial eta squared ($\eta^2_p$); available in SPSS, and the family-wise significant level was set at .05. ANOVA tables can be found in Appendix F.

# RESULTS

The first comparison examined was the manipulation of blood glucose levels over the four experimental breakfasts across the 180-min time frame. The second comparison examined the effect of the post-prandial glycaemic response on Choice Reaction Time, Word Recall, Trail Making B, Serial Sevens, and Stroop. The two versions of the Serial Sevens task (100 and 104) produced similar results; therefore, scores were collapsed across the two tasks. Finally, the third comparison examined the effect of perceived hunger on the cognitive tasks, and the influence of each breakfast.

## Postprandial Blood Glucose Response

The first analysis investigated the effects of the four breakfasts on participants' blood glucose levels. Specifically, the analysis looked at how each breakfast influenced the rise, peak value, and subsequent fall in blood glucose, and also which diet allowed blood glucose levels to be maintained across the time period. It was expected that three of the breakfasts (Weet-Bix + Kiwifruit, Weet-Bix + Guar Gum, and Weet-Bix + both) would result in improved glucose control, in comparison to the breakfast of just Weet-Bix. Figure 2 shows the recorded blood glucose measurements across all 20 participants.

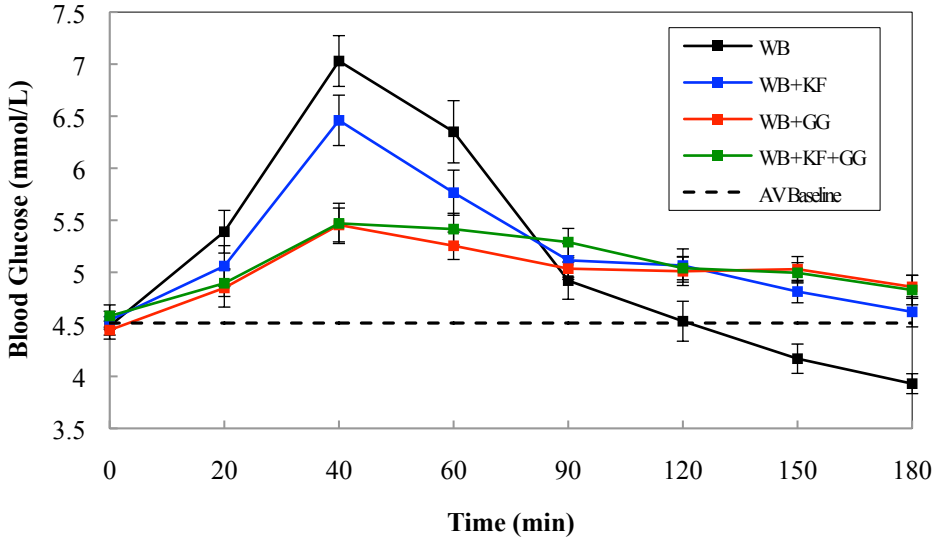



*Figure 2.* Blood glucose levels across all breakfasts, over 180 mins. Error bars are standard error bars. AV Baseline = average baseline.

From this manipulation check it can be seen that the breakfasts had a classic effect on glucose levels. Weet-Bix caused the highest peak (7.0 mmol/L), and the largest crash below baseline (3.9 mmol/L) in comparison to the other three breakfasts. Weet-Bix + Kiwifruit had the second highest peak (6.5 mmol/L); however, the decrease of glucose was slowed over 60 – 90 mins, and between 90 min to 180 min the glucose response leveled out to stay above baseline. Glucose response levels from Weet-Bix + Guar Gum and Weet-Bix + Kiwifruit + Guar Gum showed the largest effect, with a considerable decrease in the glucose peak at 40 mins post consumption. Glucose levels in the Weet-Bix + Guar Gum condition had a slight decrease at 90 mins, leveling out across 90 mins to 180 mins and staying well above baseline at 4.9 mmol/L. In comparison, the glucose peak of the Weet-Bix + Kiwifruit + Guar Gum diet lasted over 40 mins to 90 mins, before it had a slight drop at 120 mins. Similarly, blood glucose levels in this condition stayed above baseline even at 180 mins.

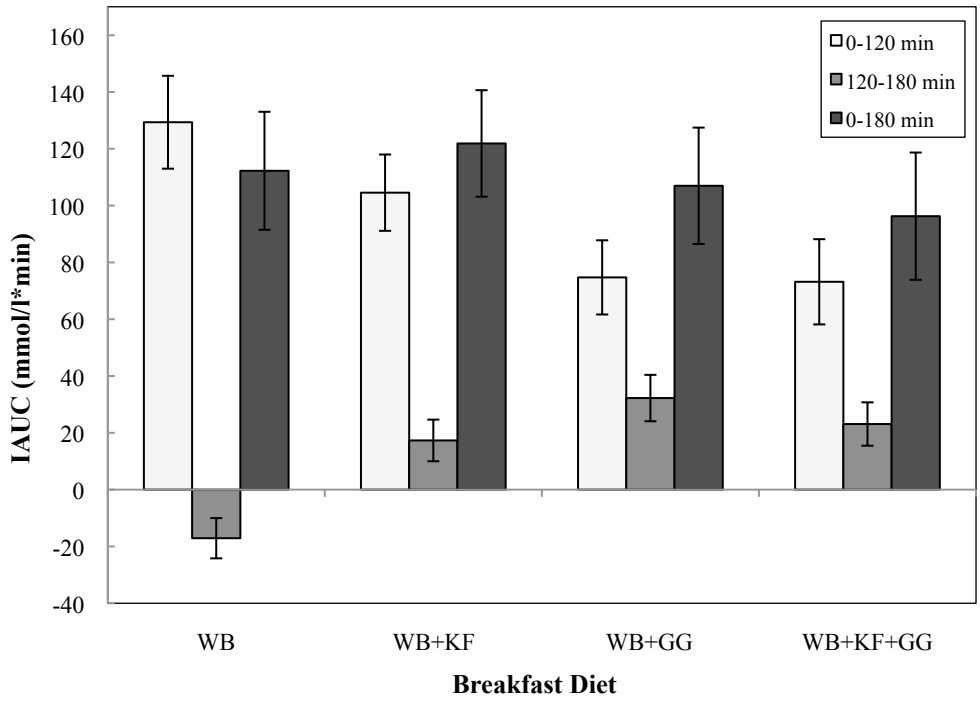



*Figure 3.* Incremental area under the curve (IAUC) over each of the breakfast diets at 0 – 120 mins, 120 – 180 mins, and 0 – 180 mins.

Following the glucose manipulation the incremental area under the curve (IAUC) was determined, examining the increase in blood glucose concentration

over baseline x time. The IAUC is a measure of the area under the glucose response curve for each breakfast diet, and is calculated by the method of trapezoid summation. The IAUC was examined over the total measurement period, and for the 0 – 120 mins and 120 – 180 mins periods separately, as shown on Figure 3. Although the total area under the curve was not altered much by the different breakfast diets, the distribution changed. The Weet-Bix diet was the only one that showed a negative IAUC between 0 and 120 mins; all of the other breakfasts were positive in this time period. Of particular interest here is the low but sustained blood glucose response caused by kiwifruit and guar gum combined compared to Weet-Bix alone.

Moreover, during the 0 – 120 min period Weet-Bix + Guar Gum has a much smaller area that the Weet-Bix alone, though it is almost identical to the response shown by Kiwifruit + Guar Gum combined, indicating that that the addition of kiwifruit had little effect. In comparison to the Kiwifruit + Guar Gum diet, the kiwifruit diet has a much larger area difference, suggesting that guar gum is having the main effect.

**ANOVA Assumptions**

Prior to beginning the main analysis, the general assumptions underlying the ANOVA technique were examined. Although the study's experimental trials were completed in groups of up to three, the assumption of independence of observation was met. All cognitive tests were completed privately on an individual computer, where each participant could not be influenced by the presence of others. The Hunger Scale scores were also kept private and confidential.

Various scores on the dependent measures were slightly skewed; however, due to a reasonable sample size (20 within-subjects), it was decided not to do any transformations on the data, as the ANOVA is considered robust to moderate violations of normality (Pallant, 2010). In addition, initial analysis on the Serial Sevens 100 and 104 identified that these two measures had similar scores; therefore, they were combined after the removal of outliers. The following main

analysis utilized a 4 (Breakfast Type) x 3 (Testing Time) repeated-measures ANOVA to determine the effect of each breakfast condition on blood glucose levels and cognitive performance over 3-hours. Greenhouse-Geisser was used, with its corrections to the degrees of freedom. Unequal variances were checked by the Levine's test, and if any serious violations occurred they would be reported.

## Cognitive Measures

ANOVAs were individually conducted for each the five cognitive tasks, across each breakfast and the three time periods. It was expected that kiwifruit and guar gum (alone and combined) would improve cognitive performance on all tasks post-consumption.

### *Choice Reaction Time*

Table 6

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Performance on the Choice Reaction Time Task*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 437.40 | 39.59 | 451.90 | 67.95 | 453.65 | 70.37 | 454.95 | 60.96 |
| 2 | 444.10 | 58.09 | 443.65 | 64.63 | 442.05 | 58.47 | 446.50 | 54.42 |
| 3 | 445.60 | 54.41 | 446.50 | 67.13 | 446.70 | 68.39 | 438.80 | 51.80 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values for Choice Reaction Time are given in ms.

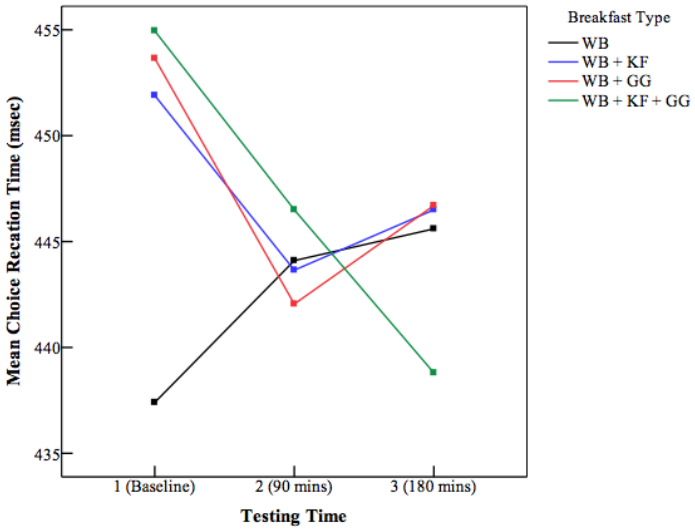



*Figure 4.* Mean scores for Choice Reaction Time for each breakfast over each time point.

From Table 6, it can be seen that, overall, there appears to be a small decrease in RT across Testing Time, which was close to statistical significance, $F(1.81, 34.35) = 2.67$, $p = .09$, $n^2_p = .12$, $SP = .47$. Partial eta squared had a moderate effect size, explaining 12% of the variance in performance, suggesting that with a better SP a statistically significant effect of Testing Time might have been observed. There was no main effect for Breakfast Type, $F<1$, and no significant interaction between Testing Time and Breakfast Type, $F(3.92,74.48) = 1.92$, $p = .12$, $n^2_p = .09$, $SP = .55$, suggesting no difference in the effectiveness of the four diets. Importantly, though, there was a small effect size suggesting that the study had insufficient power to produce a statistically significant outcome. For ease of interpretation *M*s are also shown graphically in Figure 4. It can be seen that kiwifruit and guar gum combined had the greatest improvement across all three Testing Times; however, the remaining breakfasts produced a slightly slower RT across Testing Times 2 and 3.

***Word Recall***

Table 7

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for the Word Recall Task*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 15.15 | 3.68 | 15.40 | 4.27 | 14.95 | 4.55 | 14.90 | 3.74 |
| 2 | 17.20 | 2.46 | 16.85 | 3.12 | 16.45 | 3.39 | 17.05 | 2.52 |
| 3 | 17.95 | 2.39 | 17.50 | 2.70 | 17.70 | 2.56 | 16.95 | 2.70 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values are the number of words recalled.

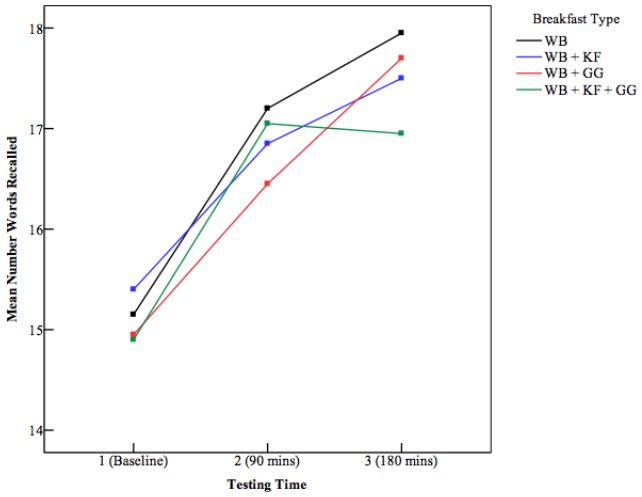



*Figure 5.* Mean scores for Word Recall for each breakfast over each time point.

From Table 7, it can be seen that there is a substantial increase in the number of words recalled across all three Testing Times, an increase that was statistically significant, $F(1.66, 31.44) = 70.16$, $p = <.005$, $n^2_p = .79$, $SP = 1.00$. Testing Time showed a large effect size, explaining 79% of the variance in performance. However, there was no main effect for Breakfast Type, $F<1$, and no significant interaction between Testing Time and Breakfast Type, $F<1$.

Post-hoc tests using a Bonferroni test revealed the average recall score increased substantially from Testing Time 1 ($M= 15.10$) to 2 ($M= 16.89$) (p= <.005), as

well as from Testing Time 2 to 3 (*M*= 17.53) (p= <.005). This suggests that performance on Word Recall improved significantly post-consumption over 90 mins and 180 mins. Because the memory recall task would have been subject to practice effects, the rate of change was investigated, but little difference was observed across each Breakfast Type and Testing Time (as shown in Figure 5). It can be assumed that the practice effect associated with each breakfast was constant because the order of breakfasts was randomized. Therefore, any differences in the rate of change would be due to Breakfast Type.

***Trail Making***

Table 8

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Time to Complete Trail Making B*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 52.89 | 20.81 | 53.33 | 22.94 | 52.12 | 13.70 | 54.62 | 15.97 |
| 2 | 45.66 | 13.77 | 48.10 | 19.28 | 47.64 | 15.91 | 47.48 | 12.92 |
| 3 | 43.07 | 12.28 | 41.25 | 13.49 | 42.37 | 14.29 | 41.48 | 10.12 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values are given in s.

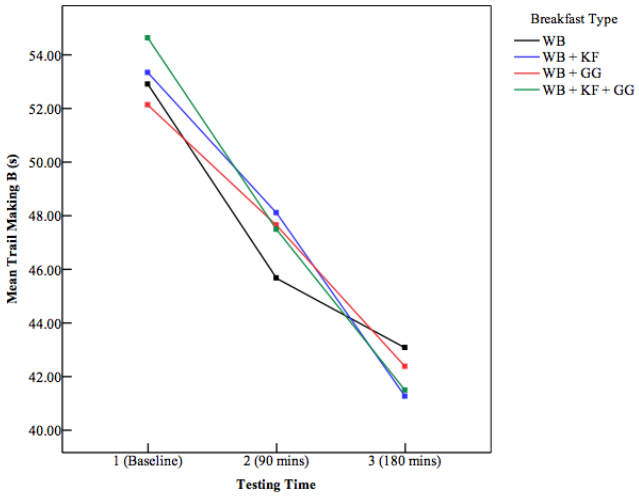



*Figure 6.* Mean scores for the time taken (nearest s) to complete the Trail Making B for each breakfast over each time point.

It can be seen from Table 8 that RT scores got substantially faster across all three Testing Times, a decrease in RT that was statistically significant, $F(1.50, 28.57) = 37.68$, $p = <.005$, $n^2_p = .67$, $SP = 1.00$. There was no main effect for Breakfast Type, $F<1$, and no significant interaction between Testing Time and Breakfast Type, $F<1$. Performance on the Trail Making task produced a large effect for Testing Time. Testing Time explained 67% of the variance in performance on the task, while Breakfast Type and the interaction between Breakfast Type and Time explained little of the variance. Similarly to the Word Recall task, this task was subject to large practice effects, most likely accounting for most of the decrease in time across Testing Time. The differences between Breakfast Types were too small in this task to analyze the rate of change across times to see if learning speed was affected. Again, for ease of interpretation this can be observed in Figure 6.

Post-hoc tests using a Bonferroni test revealed substantial changes between all three of the Testing Time periods, all reaching statistical significance ($p= <.005$). The average score decreased between Testing Time 1 ($M= 53.24$) and 2 ($M= 47.22$), as well as from Testing Time 2 to 3 ($M= 42.04$), suggesting that performance on the Trail Making task substantially improves post-consumption up to 180 mins.

Table 9

*Accuracy Scores on the Trail Making B, across Breakfast Type and Testing Time*

| | Breakfast Type | | | |
|---|---|---|---|---|
| Time | WB | WB + KF | WB + GG | WB + KF + GG |
| 1 | 3 | 10 | 8 | 8 |
| 2 | 7 | 10 | 7 | 8 |
| 3 | 4 | 5 | 4 | 3 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum.

Accuracy was also recorded for the Trail Making B task; interestingly, Weet-Bix + Kiwifruit had the highest number of errors (incorrectly selecting a number or letter) at Testing Time 1, 2 and 3 (see Table 9). Guar gum alone and combined

had similar accuracy scores, whereas, Weet-Bix alone had the least amount of mistakes. However, the number of errors was small and little faith should be attached to the small differences observed.

***Serial Sevens***

Table 10

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Performance on Serial Sevens Collapsed across the 100 and 104 Starting Values*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 93.26 | 42.13 | 86.41 | 36.56 | 88.48 | 38.09 | 89.27 | 30.81 |
| 2 | 78.73 | 28.04 | 79.61 | 39.70 | 81.90 | 35.07 | 93.75 | 32.81 |
| 3 | 85.93 | 40.07 | 74.07 | 29.24 | 82.40 | 35.29 | 84.20 | 25.12 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values for Serial Sevens are given in s.

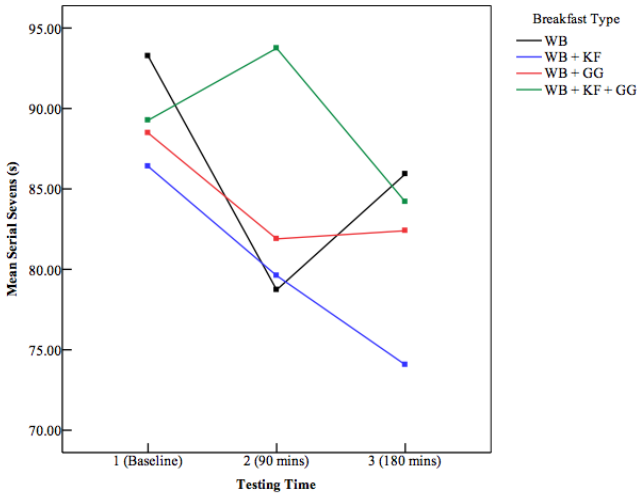



*Figure 7.* Mean scores for Serial Sevens for each breakfast over the three testing times.

From Table 10, it can be seen that performance improved substantially from Testing Time 1 to 2 on all diets, apart from Weet-Bix + Kiwifruit + Guar Gum.

The two breakfasts that included kiwifruit both resulted in improved performance at Testing Time 3, whereas the other two breakfasts did not (see Figure 7 for a visual interpretation). There was a statistically significant difference across Testing Time, $F(1.67, 31.68) = 5.63$, $p = .01$, $n^2_p = .23$, $SP = .78$, which had a large effect size, explaining 23% of the variance in performance. There was no statistically significant difference for Breakfast Type, $F(2.40, 45.54) = 1.12$, $p = .34$, $n^2_p = .06$, $SP = .25$, and no significant interaction between Testing Time and Breakfast Type, $F(4.08, 77.50) = 1.90$, $p = .12$, $n^2_p = .09$, $SP = .56$. Breakfast Type and the interaction between Breakfast Type and Testing Time displayed smaller effect sizes, with Breakfast Type explaining 6% of the variance, while the interaction between Breakfast Type and Testing Time accounted for 9% of the variance.

Post-hoc Bonferroni tests revealed that the average score decreased significantly from Testing Time 1 ($M$= 89.35) to 2 ($M$= 83.49) ($p$= .012), but the small change from Testing Time 2 to Testing Time 3 ($M$= 81.65) was not significant ($p$= 1.00).

***Stroop***

Separate analyses were undertaken for congruent scores, incongruent scores, and these two scores combined.

Table 11

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Time to Complete the Stroop (congruent)*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 542.25 | 58.59 | 532.65 | 50.54 | 544.40 | 63.92 | 556.90 | 45.71 |
| 2 | 539.00 | 49.45 | 542.30 | 69.20 | 542.80 | 46.69 | 540.10 | 42.47 |
| 3 | 542.90 | 55.64 | 538.65 | 64.25 | 543.15 | 60.07 | 533.80 | 51.15 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values are given in ms.

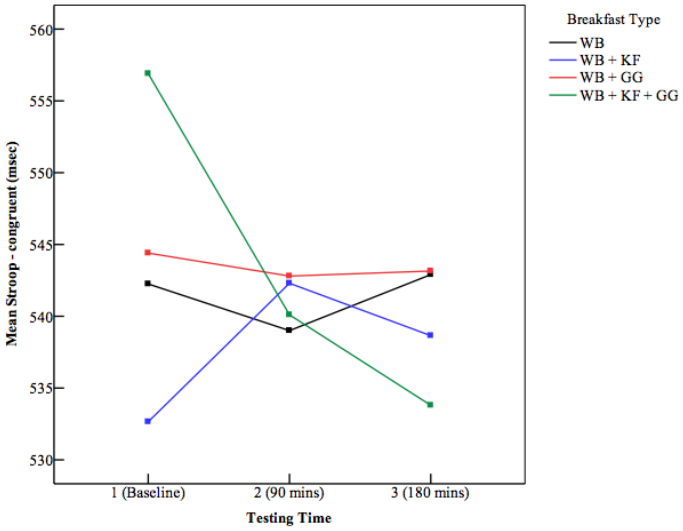



*Figure 8.* Mean scores for Stroop (congruent) for each breakfast over each time point.

From Table 11, it can be seen that there is little difference between performance on the Stroop (congruent) across Testing Time and Breakfast Type. Though, the Weet-Bix + Kiwifruit + Guar Gum diet showed quite a large and consistent improvement in performance across the three testing times, unlike the other breakfasts. There was no significant main effect for Testing Time, $F<1$, for Breakfast Type, $F<1$, and also no significant interaction between Testing Time and Breakfast Type, $F(4.66, 88.47) = 1.33$, $p = .26$, $n^2_p = .07$, $SP = .43$. Figure 8 gives a visual representation of each of the scores across Breakfast Type and Testing Time.

Performance on the Stroop (congruent) condition showed small to moderate effects across Testing Time, Breakfast Type, and the interaction between Testing Time and Breakfast Type. Testing Time explained 7% of the variance, while Breakfast Type explained 2% of the variance, and the Breakfast Type and Testing Time interaction explained 7% of the variance in performance.

Table 12
*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Time to Complete the Stroop (incongruent)*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 583.40 | 69.57 | 574.30 | 61.51 | 577.45 | 73.41 | 588.95 | 40.35 |
| 2 | 565.30 | 66.85 | 575.65 | 74.33 | 562.15 | 61.76 | 567.50 | 42.61 |
| 3 | 568.80 | 56.44 | 568.55 | 74.33 | 568.95 | 67.18 | 571.65 | 40.62 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values are given in ms.

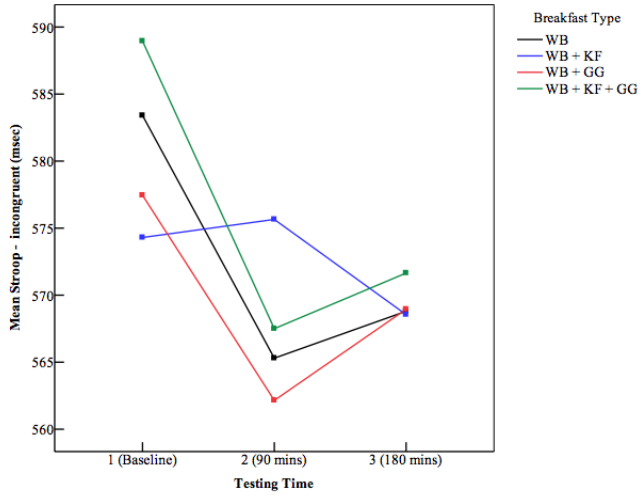



*Figure 9.* Mean scores for Stroop (incongruent) for each breakfast over each time point.

From Table 12 and Figure 9, it can be seen that, there appears to be a substantial improvement across Testing Time 1 to 2 across all Breakfast Types, apart from kiwifruit alone, which had an improvement across Testing Time 2 to 3. This decrease in RT across Testing Times was statistically significant, $F(1.91, 36.27) = 5.17$, $p = .01$, $n^2_p = .21$, $SP = .78$, and had a large effect size, explaining 21% of the variance on performance. There was no main effect for Breakfast Type, $F<1$, and no significant interaction between Breakfast Type and Testing Time, $F<1$.

Post-hoc Bonferroni tests revealed a significant decrease from Testing Time 1 (*M*= 581.03) to 2 (*M*= 567.65) (*p*= .02). There was only a small change from Testing Time 2 to 3 (*M*= 569.49), where the mean time actually showed a small increase. RT underwent a relatively large decrease initially and then appeared to stabilize (see Figure 9).

Table 13

*Means (M) and Standard Deviations (SD) as a Function of Breakfast Type and Testing Time for Time to Complete the Stroop (overall)*

| | Breakfast Type | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Time | WB | | WB + KF | | WB + GG | | WB + KF + GG | |
| | *M* | *SD* | *M* | *SD* | *M* | *SD* | *M* | *SD* |
| 1 | 567.15 | 62.84 | 560.80 | 57.00 | 566.20 | 68.63 | 577.65 | 39.58 |
| 2 | 555.95 | 57.96 | 564.45 | 65.80 | 555.20 | 59.54 | 556.15 | 35.77 |
| 3 | 560.00 | 53.76 | 556.05 | 66.17 | 560.00 | 61.73 | 555.50 | 42.19 |

*Note.* WB = Weet-Bix; WB + KF = Weet-Bix and Kiwifruit; WB + GG = Weet-Bix + Guar Gum; WB + KF + GG = Weet-Bix + Kiwifruit + Guar Gum. Mean values are given in msec.

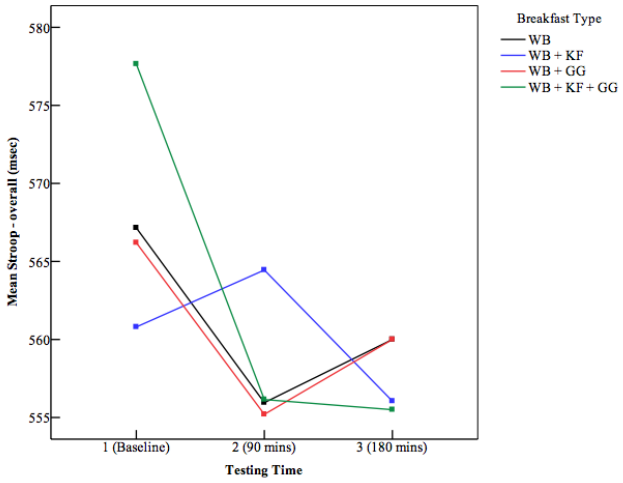



*Figure 10.* Mean scores for Stroop (overall) for each breakfast over each time point.

Table 13 shows that there appears to be a decrease in RT across Testing Time 1 to 2 (apart from kiwifruit alone), whereas, performance slowed again at Testing Time 3 on Weet-Bix and Weet-Bix + Guar Gum. Interestingly, the two

breakfasts containing kiwifruit had similar scores at Testing Time 3. Overall there was a statistically significant effect for Testing Time, $F(1.67, 31.72) = 4.04$, $p = .03$, $n^2_p = .18$, $SP = .63$. However, there was no main effect for Breakfast Type, $F<1$, and no significant interaction between Testing Time and Breakfast Type, $F(4.84, 91.94) = 1.12$, $p = .35$, $n^2_p = .06$, $SP = .38$.

Performance on the Stroop (overall) showed a large effect for Testing Time, and small effects for the interaction between Breakfast Type and Time of Testing. Testing Time explained 17% of the variance, Breakfast Type explained none, and the Breakfast Type and Testing Time interaction explained 5% of the variance. Post-hoc Bonferroni tests revealed a significant decrease from Testing Time 1 to 2, ($M$= 567.95 and $M$= 557.94, respectively), $p$=.05. However, there was little change between Testing Time 2 and 3 ($M$= 557.89), as shown in Figure 10.

**Hunger Scale**

Table 14

*Means (M) and Standard Deviations (SD) for the Hunger Scale across all Breakfasts*

| Breakfast Type | *M* | *SD* |
|---|---|---|
| Weet-Bix Only | 9.23 | 1.30 |
| Weet-Bix + Kiwifruit | 7.18 | 1.92 |
| Weet-Bix + Guar Gum | 6.53 | 1.64 |
| Weet-Bix + Kiwifruit + Guar Gum | 5.56 | 2.27 |

*Note:* Hunger scores was obtained at the end of each session for all participants.

As shown in Table 14, there is a substantial difference across the Hunger Scale means for each Breakfast Type. Particularly when comparing Weet-Bix (9.23) to Weet-Bix + Kiwifruit + Guar Gum (5.56). Interestingly, kiwifruit and guar gum individually had similar means, with only a slight difference between them.

A one-way ANOVA showed a difference between Breakfast Type on the Hunger Scale, $F(2.44, 46.41) = 20.13$, $p$= <.005, $n^2_p = .51$, $SP$= 1.00. This suggests that there was a difference between diets in regards to perceived hunger at the end of

each trial. The ANOVA table is given in Appendix F. A post hoc Bonferroni test revealed a significant difference only between Weet-Bix alone and the remaining three breakfasts ($p$= <.005). There was no significant difference found for the diets containing kiwifruit and guar gum, suggesting that these breakfasts had a similar effect on satiety.

A bivariate correlation was used to examine the relationship between hunger and cognitive performance at Testing Time 3 (180 mins) on the experimental trials. Pearson correlation and Spearman's *rho* produced similar results, but Spearman's *rho* was used due to the small sample size (Pallant, 2010). Spearman's *rho* was used to examine all of the correlations between the Hunger Scale of each breakfast and the dependent measures. Conventional effect sizes are small (0.1), medium (0.3), and large (0.5) (Cohen, 1988).

Table 15

*Bivariate Correlations between Hunger Ratings and Cognitive Performance at Time 3 after each Experimental Breakfast*

| | WB Only | WB + FK | WB + GG | WB + KF + GG |
|---|---|---|---|---|
| Choice Reaction Time | .02 | -.35 | -.05 | -.01 |
| Word Recall | .19 | .18 | .13 | -.21 |
| Serial Sevens 100 | .03 | -.47 | -.20 | -.08 |
| Serial Sevens 104 | .04 | -.25 | -.17 | .04 |
| Trail Making B | .07 | -.33 | .06 | -.09 |
| Stroop (overall) | -.21 | -.09 | -.04 | -.04 |
| Stroop (congruent) | -.13 | -.14 | .02 | -.19 |
| Stroop (incongruent) | -.32 | .02 | -.02 | -.24 |

*Note:* Significance level was 0.05 (2-tailed).

Scores on the Hunger Scale were not significantly correlated with cognitive performance on any of the five tasks used (as shown in Table 15). Small effect sizes were found across Weet-Bix (apart from Stroop (incongruent) which had a moderate effect size), Weet-Bix + Guar Gum, and Weet-Bix + Kiwifruit + Guar Gum. However, the Weet-Bix + Kiwifruit diet did display three moderate effect sizes (.3 and above), despite the lack of statistical significance.

# DISCUSSION

The present study aimed to determine the effects of kiwifruit and guar gum (alone and combined) on reducing the postprandial glycaemic response to carbohydrate foods, and examined whether the reduced glucose response enabled cognitive performance to be maintained over a 3-hour time period, compared to baseline.

The study also investigated the relationship between perceived hunger and each breakfast three hours after the consumption of the experimental breakfasts at 8.30 a.m.

## Postprandial Blood Glucose Response

The first analysis of the present study was to determine the effects of kiwifruit and guar gum on postprandial glycaemia after consuming each of the four experimental breakfasts (Weet-Bix, Weet-Bix + Kiwifruit, Weet-Bix + Guar Gum and Weet-Bix + Kiwifruit + Guar Gum). On the basis of previous research it was hypothesized that kiwifruit and guar gum (individually) would lower the glucose peak after meal consumption and also work to maintain adequate blood glucose levels over a 3-hour time frame. It was expected that when kiwifruit and guar gum were combined, the two together would have an even greater impact on lowering the postprandial glucose peak.

Following a manipulation check across all four breakfasts it was observed that each intervention had a glucose peak at 40 mins, suggesting that kiwifruit and guar gum do not delay the peak as previous research has concluded (Feldman & Barshi, 2007); however, they were both effective in lowering the overall peak, where Weet-Bix alone did not. Kiwifruit did not lower the glucose peak as much as was expected, with glucose levels still rising to an average of 6.5 mmol/L (the control measure had a peak of 7 mmol/L), suggesting that there may have been a delay in the kiwifruit working to aid the digestion of the Weet-Bix. However, the kiwifruit did work to maintain glucose levels at a later postprandial time (90

mins to 120 mins) at 5.1 mmol/L, where in comparison by this stage the control intervention had already dropped to 3.9 mmol/L. The final average reading for kiwifruit at 180 mins was 4.6 mmol/L, which was still above the initial baseline level (4.5 mmol/L).

Kiwifruit has been identified as having a low GI and being high in fiber; for this reason it has been hypothesized that kiwifruit can also lower the overall GI of the food it is consumed with. A number of factors have been reported that enable kiwifruit to influence blood glucose levels, some or all of which may be responsible for the present results. Firstly, when kiwifruit is consumed it has the capacity to swell to four times its size in the stomach and small intestine; the remnants reduce the mixing of carbohydrates in the gut and slow down the rate of digestion (Monro, 2013). A further factor is that the soluble fiber in kiwifruit balances out the effects of sugar from carbohydrates by blocking them and limiting access to the blood stream (Will & Byers, 1996). Both of these factors work to slow down the digestion of carbohydrates, and substantially reduce the rate glucose is released into the bloodstream. The present study supports these findings as kiwifruit was found to delay and slow the decline in blood glucose over 180 mins. It is important to note that even though kiwifruit did not eliminate the glucose peak entirely, the peak after consuming kiwifruit stayed well within the normal and recommended range for healthy glucoregulation.

In comparison, guar gum worked to control the glucose peak at 40 mins better than kiwifruit, with the glucose response rising to only 5.4 mmol/L, followed by a very slight decrease to 5.0 mmol/L across the next 50 mins. Glucose levels were then maintained for the remaining 90 mins of the trial at 4.9 mmol/L. The observed response was expected from the guar gum intervention, and it replicates the findings of Nilsson et al. (2010). Previous studies have found that guar gum is most effective when it is mixed with food (Feldman & Barshi, 2007). In line with this finding, when guar gum was mixed thoroughly with Weet-Bix and water substantial control was exerted over blood glucose across the entire 180 mins. Moreover, the blood glucose readings taken across the guar gum trials in the present study are in accordance with those recorded from previous research (Braaten et al., 1991; Jenkins et al., 1977), indicating that the

guar gum administered produced the expected effects. Of further interest is the amount of guar gum used to exert effects on the postprandial glycaemic response. The present study supports the theory that approximately 10 g of guar gum per serving is needed is produce significant effects, which is consistent with previous studies that found anything less than 10 g of guar failed to produce any changes when using healthy participants (Wolever et al., 1979).

According to Blackburn and Johnson (1981) guar gum influences blood glucose levels by slowing the rate of digestion in the stomach, slowing the release of glucose to the blood stream. Groop et al. (1993) suggest that this is because guar gum lowers the overall GI of a meal, slowing the absorption rate. The blood glucose results of the present study supports this theory as glucose levels were maintained across a substantial period of time (without any additional food consumption), suggesting that glucose is slowly, but consistently being released into the bloodstream. Wolever et al. (1979) found that the effects of guar gum only worked when it was consumed with a low GI food; however, the present study shows that this is not the case. Weet-Bix has a medium GI value, with two Weet-Bix biscuits having a GI of 57 (Auckland District Health Board, 2013). Blackburn and Johnson reported that gastric emptying is slowed when guar gum is mixed with carbohydrates. Therefore, the effects of the high carbohydrate content of Weet-Bix was moderated by adding guar gum in the present study, resulting in better glucose control than was achieved by Weet Bix alone.

In addition, adding gaur gum to the carbohydrate meal may have contributed to the body's ability to store glycogen. That is, by allowing a controlled level of glucose to be released into the blood stream, there would have been more available glucose to be stored into glycogen. In turn, mobilized glycogen is ready to be turned back into glucose for energy, helping maintain an adequate level (between 5 – 6 mmol/L) of glucose.

Kiwifruit and guar gum combined had a similar blood glucose response as the guar gum breakfast. The overall peak at 40 mins was small, just 5.5 mmol/L, and glucose levels were maintained over 40 to 90 mins between 5.3 mmol/L and

5.0 mmol/L, dropping to 4.8 mmol/L at 180 mins. The observed glucose response for kiwifruit and guar gum combined suggests that guar gum may aid the digestion of kiwifruit, slowing the absorption rate in the stomach, allowing a slower release of the natural sugars found in kiwifruit. Soluble fiber has been found to limit the sugars that enter the bloodstream; therefore, guar gum may be working to inhibit the response to the natural sugars from the kiwifruit to alter the glucose peak.

**Cognitive Measures**

The main analysis determined the influence of each Breakfast Type on cognitive performance across the three Testing Time points. Improved performance from time 1 to time 2 was expected, with kiwifruit and guar gum working to maintain performance across times 2 and 3. Cognitive performance was tested on five tasks: Choice Reaction Time, Word Recall, Trail Making B, Serial Sevens, and Stroop. No statistically significant differences were found for Breakfast Type or the interaction between Testing Time and Breakfast Type. However, every task, apart from Choice Reaction Time and the Stroop (congruent), found a statistically significant effect for Testing Time, indicating that there was a change in performance over the three time points for each trial. Thus, the present results are inconsistent with the findings of Nilsson et al. (2012) in regards to cognitive performance. They found significant improvements on a selective attention task after a breakfast containing guar gum, in comparison to a control. However, Nilsson et al. failed to find any significant results for time (and for breakfast type) on RT and working memory.

Previous research has examined the interaction between blood glucose levels and cognitive performance, sometimes focusing on high (hyperglycaemic), sometimes low (hypoglycaemic), and sometimes normal glucose responses. Thus, there is little previous research to compare the present results to. Furthermore, few studies have examined the effect of guar gum, and even fewer kiwifruit. Therefore, the present results should be considered preliminary until more research has been done.

***Choice Reaction Time***

Previous studies on RT have focused on how cognitive performance is affected by low blood glucose, finding that, generally, RT deteriorated when participants were hypoglycaemic (Kerr et al., 1989; Strachan et al., 2001). However, the level of low blood glucose that produces negative effects has been inconsistent across studies. Mild hypoglycaemia (3.5 mmol/L +) has failed to produce any cognitive deficits, with various studies concluding that glucose needs to be 3.0 mmol/L or under (Kerr et al., 1989; Lobmann et al., 2000). In the present study, the Weet-Bix diet was the closest to reaching hypoglycaemic blood glucose levels (3.8 mmol/L) with the final average reading at 180 mins being 3.9 mmol/L. Performance on the Choice Reaction Time task changed little from time 2 to time 3 with the Weet-Bix diet, despite the rapid drop in blood glucose below the fasting baseline rate at 180 mins, consistent with the idea that performance on RT is only altered in moderate to severe hypoglycemia (Strachan et al. 2001).

The present study found that performance on Choice Reaction Time was most improved during the second round of cognitive tests, and all (apart from kiwifruit and guar gum combined) had a slight increase in RT at time 3. Increasing and higher blood glucose levels (under 7.8 mmol/L) have been found to improve RT in various studies. Strachan et al. (2001) found that once glucose levels had been restored to normal (5 mmol/L +) participants had a significantly improved RT. Benton et al. (1994) also concluded that increasing blood glucose levels resulted in a faster ability to process information. These findings were supported by the present study, as performance on the Choice Reaction Time task improved when the glucose response across all diets was between 4.7 and 5.5 mmol/L. However, a discrepancy was that the Weet-Bix condition was found to have a comparable RT at the last round of testing, even though glucose levels had dropped below baseline. Choice Reaction Time performance was maintained at 180 mins, and Weet-Bix + Guar Gum + Kiwifruit was found to elicit an even better performance time. These results, though only showing small effects, support the notion that sustaining blood glucose levels can provide the brain with adequate energy to maintain RT performance over several hours.

While some studies have found RT to be improved by a medium GI breakfast (e.g., Dye & Blundell, 2002), others have found that low GI foods have elicited a higher performance rate (Kaplan et al., 2004). Dye and Blundell (2002) concluded that foods that have a positive influence on glucose, that is, effectively controlling and maintaining the postprandial glucose response, facilitated RT, particularly in comparison to foods that caused unfavorable effects on glucose (e.g., swings in glucose levels). Although the differences found in regards to breakfast in the present study were small, relative scores from the Choice Reaction Time task are consistent with Dye and Blundell's theory. Results from the present study show that the Weet-Bix + Kiwifruit + Guar Gum breakfast improved RT across all three time points, also producing the most stable and controlled blood glucose response across the 180 min time frame. However, the Weet-Bix intervention displayed a large swing in the postprandial glycaemic response, yet this did not appear to have any negative influence on Choice Reaction Time. This finding may be explained by the fact that the peak and drop in blood glucose levels for Weet-Bix stayed within the considered healthy range for postprandial blood glucose. Previous studies have not shown any influence on performance within this glucose range, though there are at present too few studies to base a firm conclusion on.

The statistically nonsignificant effect found on RT for selective attention in the Nilsson et al. (2012) study was not discussed in their report, therefore, no comparisons or conclusion can be made between that and the present study.

***Word Recall***

Currently, research on the interaction between glucose and memory has displayed inconsistent results. Various studies have concluded that glucose levels can influence memory (Benton & Owens, 1993; Manning at al., 1990; Craft et al., 1994), where others have found no difference in memory when blood glucose levels have been manipulated (Green et al., 2001; Nilsson et al., 2012). The present study found no significant effect of Breakfast Type or the interaction between Breakfast Type and Testing Time on Word Recall; however, there was a significant main effect for Testing Time.

Word Recall has been one of the most extensively examined memory tasks in this area of research. Benton and Owens (1993) found that increasing blood glucose levels resulted in a better ability to recall words. Glucose in the form of a drink given at two time points had an even greater recall performance rate, suggesting that the uptake of glucose was influencing performance. A following study by Craft et al. (1994) found that recall was improved, but only when blood glucose levels were maintained above baseline after one hour. The finding from Craft et al. was partially supported by the present study with recall improving after blood glucose was increased; however, the Breakfast diet of Weet-Bix alone had the highest level of recall at time 3 even though blood glucose levels had dropped below baseline.

It has been found that during periods of cognitive demand blood glucose levels decline at a more rapid rate, which has been hypothesized to be caused by the increased uptake of glucose from the blood by neurons (Scholey et al., 2001). Glucose seems to improve memory by facilitating acetylcholine (ACh) synthesis and release in the brain. Low blood glucose levels are unable to produce acetyl-CoA (a precursor to ACh), and ACh synthesis is decreased (Ragozzino et al., 1996), therefore, limiting memory performance. However, this theory is not supported in the present study, with low blood glucose levels (from the Weet-Bix diet) having the same level of performance as the remaining breakfasts. Nilsson et al. (2012) also failed to produce any significant results for performance on working memory. Unfortunately, they failed to discuss the possible reasons behind this finding.

A highly likely confounding factor for the Word Recall task in the present study is the effects of practice; thus, the results need to be interpreted with caution. Previous research examining recall have not clearly identified the specific type of test used and how it was conducted. Therefore, in the present study one word list was used with the same 20 words, meaning that every time the task was repeated there was a high chance of remembering some words from the previous presentations. This aspect also limits the level of task difficulty, a factor that has been a crucial part of various studies that have found a significant relationship between glucose and cognitive performance (Feldman & Barshi, 2007). Benton

and Owens (1993) found that recall became harder when a distracting task was set between word presentation and recall, overall limiting the amount of time to memorize the words. This may have been beneficial in the present study in order to minimize learning.

For their working memory task Nilsson et al. (2012) conducted two versions of the test, and each task was randomly ordered; yet they still failed to find a significant result from either intervention used in their study. Their finding suggests that previous studies may have falsely concluded significant effects occurred by ignoring the possibility of practice effects. Alternatively, the present study and the study completed by Nilsson et al. may not have used tasks that were difficult enough; or the breakfast interventions chosen did not influence blood glucose levels enough to elicit an accurate effect.

Previous studies that have found significant results in regards to memory and glucose have been most consistent with older aged adults. Participants in the current study were aged between 26 and 66, with a mean age of 36. Maybe an older group might have responded differently. It has been hypothesized that older aged adults are more likely to produce a glucose enhanced effect due to deficiencies in glucose metabolism and memory regulators in the brain (Korol & Gold, 1998; Manning et al., 1990), where consuming glucose or a glucose-influencing nutrient allows a higher availability of glucose to reach the bloodstream (Korol & Gold, 1998).

All would not have been lost in the present study had there been a greater difference in performance across the experimental breakfasts. Because the order of these breakfasts was counterbalanced, one could assume that practice effects were equal for all breakfasts. Any differences showing up across breakfasts could then be assumed to be a learning effect. Unfortunately, differences were too small to allow for an assessment of this possibility.

***Trail Making B***

As for the recall task, the Trail Making B Task revealed few differences across Breakfast Type, yet there was a significant main effect for Testing Time across all three time points. Performance on the Trail Making B task has produced mixed results in previous research, although usually showing significant impairments at low blood glucose levels. Past studies have found that hypoglycaemia produces a slower motor response, which effectively increases RT on the task. Pramming, Thorsteinsson, Theilgaard, Pinner, and Binder (1986) only found impairments on the Trail Making B task when glucose levels dropped to below 3.0 mmol/L. The present study found no difference in performance from when blood glucose levels had dropped below baseline to when they were maintained across the 180 min time frame, which is in line with previous studies (Evans, Pernet, Lomas, Jones, & Amiel, 2000; Hoffman et al., 1989). However, the Weet-Bix condition did produce a slightly slower RT overall at time 3, compared to the remaining breakfasts.

Evans et al. (2000) found that there was no difference on the Trail Making B at any level of blood glucose manipulation. The study concluded that any changes found were solely due to individual differences on the task. This finding was later supported by Cox et al. (2005) who also determined that performance on the task was highly individualized, with only 55% of participants showing impaired performance at a mild hypoglycaemic state. The theory behind individualized performance has been explained by various factors. Cox et al. argue that each participant has individualized levels of being able to physiologically deal with hypoglycaemia, with some bouncing back from its effects when blood glucose levels return to normal faster than others. Trail Making B task has been used to examine various areas of cognitive performance, and many past studies do not define what type of task they are focusing on. For example, Trail Making B has been used to test visual scanning, motor control, attention, judgment and decision-making, where individuals have natural strengths and weaknesses across these tasks (Feldman & Barshi, 2007). Additionally, this task has been found to be insensitive to individuals with a

marked ability to regulate and metabolize glucose efficiently (Evans et al., 2000).

Although the Trail Making B has been identified for being cognitively demanding (Evans et al., 2000), participants in the present study reported that that the task became easier over time, due to recalling where certain letters/ numbers were located. In other words, a large component of performance across the three testing times may well be due to a practice effect. As for the previous tasks, there was little difference in performance across breakfast types, suggesting that the type of breakfast was not affecting speed of learning. Previous studies have not gone into detailed explanation of how the Trail Making task was presented; however, as the task was completed numerous times over the four trials in the present study, in hindsight it would have been wise to randomly change the location of the 24 circles each time the test was completed. Feldman and Barshi (2007) noted that when tasks become overly familiar attention and planning become less affected by glucose, suggesting that the results of the Trail Making B task in this study cannot be explained by the influence of glucose manipulation.

***Serial Sevens***

Previous studies have commonly used the Serial Sevens task to assess working memory through math calculation performance at varying blood glucose levels. Researchers have concluded that Serial Sevens is a highly cognitively demanding task that is markedly affected by blood glucose levels.

It has been found that when glucose levels drop below 3.0 mmol/L performance on the Serial Sevens task declines significantly (Hale, Margen, & Rabak, 1981; Pramming et al., 1986). However, when blood glucose levels have been manipulated to be within a healthy range, performance has significantly improved. Scholey et al. (2001) found that performance on a computerized version of Serial Sevens improved after consuming a glucose drink, when the glycaemic response had risen to 5.5 mmol/L. They also found that glucose enhancement generated more responses in a 5-min time period, in comparison to

a placebo control. In the present the study, analysis of the Serial Sevens task found a significant improvement between time 1 and time 2 of testing, but there was little difference between time 2 and time 3. This finding follows previous research, in that improvement of performance was observed when glucose levels had been initially enhanced, particularly in comparison to baseline glucose readings.

Although, there was no significant difference across Breakfast Type in the present study, Weet-Bix and Weet-Bix + Kiwifruit had the fastest average scores at Testing Time 2, suggesting that the increased uptake in glucose available from the higher peak at 40 mins may have had some influence on performance. Unexpectedly, Weet-Bix + Kiwifruit + Guar Gum had the smallest effect. However, any effects were very small and may well be due to experimental error. Kiwifruit and guar gum (individually and combined) maintained performance over 180 mins, with kiwifruit performance scores actually improving. These findings further support previous studies that have shown that available glucose within a healthy range positively enhances Serial Sevens performance.

A potential confounding factor that may have influenced the results found on the Serial Sevens task is the perceived level of difficulty. Previous research has used various versions of Serial Sevens, including counting back by seven from any number between 800-999 for a time period of five minutes (Scholey et al., 2001), counting backwards from three different numbers (100, 99, and 98) by seven until reaching zero (Pramming et al., 1986), and alternating between Serial Sevens and Serial Threes (Kennedy & Scholey, 2000). Previous studies have reported no effects for Serial Threes, thought to be because the task is not cognitively demanding enough. The present study used two versions of Serial Sevens, starting from 100 and 104. Pramming et al. (1986) found no statistically significant difference between the three Serial Seven tasks they used, believed to be because the starting numbers were too close together, which made the task easier. Therefore, 100 and 104 were used, as neither are a multiple of seven. As with the other cognitive tasks used, Serial Sevens was required to be completed numerous times over the trial, leading to the concern of practice effects.

However, although there may be a small practice effect, this is unlikely to have had any major effect. Had there been large practice effects, one would expect that performance would have consistently improved across all three testing times.

***Stroop***

Previous research has found that glucose manipulation more often than not influences performance on the Stroop task. Evans et al. (2000) found that hypoglycaemic levels of blood glucose significantly impaired RT on the Stroop. It is important to note that this only occurred when glucose levels were 2.7 mmol/L or less, indicating moderate to severe hypoglycaemia, where prominent physiological symptoms are usually apparent, symptoms such as dizziness, blurred vision, confusion, headaches, shakiness and irritability.

Additional studies have focused on manipulating blood glucose by increasing the level across the study. Increasing glucose has produced a faster RT on the Stroop task (Craft et al., 1994; Evans et al., 2000), with other investigations identifying that glucose needs to be maintained above baseline levels to exert effects (Benton et al., 1994). Benton et al. (1994) failed to find a significant effect of glucose level change on RT, although they did observe that in both their experimental (glucose drink) and control groups that when glucose level was falling, but staying above baseline, performance on the Stroop improved. The present study partially replicated this finding, with overall scores showing improved and sustained performance over 180 mins. However, there was no statistically significant difference across Breakfast Type, indicating that even though the glucose response to Weet-Bix at time 3 had dropped below baseline, performance was still on par with the other breakfasts. This finding may be explained by the fact that the drop in glucose in this condition still left it within the normal range rather than at a hypoglycaemic level.

The majority of studies using the Stroop had not clearly distinguished between the different difficulty levels of the task. Some used just the congruent part, others the incongruent part, and yet others the overall score. However, Brandt,

Gibson, and Rackie (2013) split the Stroop into its congruent and incongruent components, concluding that glucose enhancement only occurred for the incongruent condition, the most cognitively demanding of the two. This finding was replicated in the present study, with no significant difference being found over time on the congruent condition, but there was on the incongruent condition, and overall. This outcome lends support to the contention that glucose enhancement is most sensitive to tasks with a certain level of difficulty.

**Hunger Scale**

The final comparison determined the effect of hunger post-consumption of each breakfast, and whether this factor was associated with cognitive performance. No statistically significant correlations between hunger and the cognitive tests were found, though the set of correlations obtained revealed small to moderate associations between cognitive performance and Breakfast Type.

From the hunger scale it was apparent that each breakfast had substantially different levels of satiety at the end of each trial (see Figure 11 below).

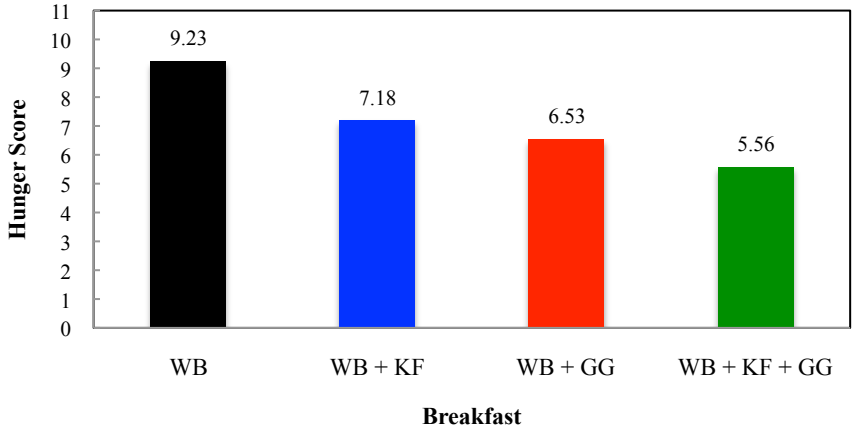



*Figure 11.* Hunger Scale scores across the four experimental breakfasts.

As expected, Weet-Bix had the highest hunger rating at 180 mins, although the elevated rating was somewhat surprising due to the high fiber content of Weet-Bix. This finding may suggest that the amount of food given was not substantial enough for some participants, and that having a standardized breakfast across all

participants, irrespective of size and weight, also meant that the type of food varied from their usual routine and eating habits. It may have been useful to have participants complete the hunger scale immediately after consumption as well as at the end of the experimental session. Even so, there is usually a lag between eating food and the highest feeling of "fullness", not to mention individual variability. So, the time between finishing a meal and completing the satiety scale is also problematic.

Weet-Bix and kiwifruit had the next highest satiety score, but it was substantially less than the Weet-Bix condition. Kiwifruit has previously been identified for its ability to increase satiety and make people feel fuller for longer (Zespri Health Communication, 2013), primarily because the cell wall of the kiwifruit remains undigested in the stomach and small intestine, where the dietary fiber can swell up to four times it original size. The digested part of the meal settle in the gut, which reduces the rate of digestive mixing, which in turn, increases satiety. This theory is supported in the present study, with participants rating the kiwifruit diet more satisfying after three hours than the Weet-Bix alone diet.

Weet-Bix and guar gum had a slightly lower hunger score than kiwifruit, in agreement with the results of previous studies (Krotkiewski, 1996; Schmidl & Abuza, 2000). A predominant theory behind the reason guar gum can increase satiety for longer periods is because it has been found to slow the absorption of food by increasing the thickness of the unstirred water layer in the intestine (Schmidl & Abuza, 2000). Previous studies have also reported a significant relationship between guar gum and weight loss, providing some support to the theory that it increases satiety (Blackburn & Johnson, 1981; Krotkiewski, 1996).

Overall, kiwifruit and guar gum combined had the lowest hunger scale rating out of the four breakfasts. This finding was expected given the mechanisms of each supplement once consumed. It was hypothesized that guar gum would increase the viscosity of the kiwifruit, essentially thickening the contents in the stomach, reducing the rate of mixing and diffusion, and slowing the rate of digestion, leading to the participants feeling fuller for longer.

**Practical Implications**

Manipulation of blood glucose levels using four experimental breakfast diets have shown that kiwifruit and guar gum can be consumed to help control and maintain blood glucose levels over a 3-hour period. Kiwifruit and guar gum are both easily accessible natural supplements, which can potentially contribute to maintaining healthy blood glucose levels, limiting the health risk of diabetes and obesity. Furthermore, kiwifruit and guar gum were found to increase levels of satiety, and for this reason, may also be beneficial for weight loss.

The findings from the present study examining the interaction between glucose and cognitive performance indicate that maintaining a postprandial blood glucose profile within a healthy boundary over an extended period of time can positively enhance performance on some cognitive tasks, such as working memory, attention and choice reaction time. Overall, consuming low GI meals that effectively control glucose can maintain performance and increase energy for everyday tasks.

**Limitations of the Present Study and Suggested Further Research**

On completion of the present study, various limitations became apparent that have weakened some of the results.

One major limitation was the difficulty level of each cognitive task used in the present study. Earlier studies that have examined the level of difficulty have previously focused on either easy or hard conditions. However, the majority of these studies have failed to find any statistically significant effects between glucose and cognitive performance on easy tasks (Feldman & Barshi, 2007; Kennedy & Scholey, 2000; Korol & Gold, 1998). Therefore, in the present study it was decided to use only harder versions of each task. For example, just Trail Making B was used, and Serial Sevens was given two different starting points. However, due to the repetitive nature of the testing, the cognitive tasks had a substantial risk of practice effects. Current inconsistencies in the literature may be partly explained by the lack of clear guidelines as to how certain tasks are to

be conducted. The level of difficulty for each task has not clearly been outlined in previous research, making it difficult to determine what tests were to be used, or whether parallel forms should be used in studies using a repetitive testing regime. Clearly outlining what is involved for each task will help further research in this area. In hindsight, both the Word Recall task and the Trail Making B task would have benefitted by using parallel forms. Unfortunately, the degree of learning in these tasks (over and above the practice effects) was largely unaffected by Breakfast Type. For tests that may be subject to strong practice effects, future studies need to develop parallel forms of these tests that have been shown to produce the same results as the original tests.

Feldman and Barshi (2007) concluded that similar (if not the same) tasks should be used to determine the effects of glucose in order to be able to accurately compare findings validly across previous studies. Currently, in the literature various forms of tasks have been used that have been criticized for the fact that they may measure different cognitive abilities. For example, various tasks of memory have been used, which may measure short-term, long-term or working memory. Invalid comparisons are then made across studies using a "memory" task, where the types of memory may be different.

Sample size was another limitation in the present study. Previous studies have used varying sample sizes, with Nilsson et al. (2012) basing their findings on 40 middle-aged adults, while Groop et al. (1993) used 15 men and women. However, in the present study it is apparent that more statistical power was needed to find significant effects across Breakfast Type and for the interaction between Breakfast Type and Testing Time. Further research will need to use previous studies to evaluate the effect sizes for breakfast effects, and determine the sample size needed to detect these effects. The sample size chosen for the present study was largely due to the time constraints imposed by having to run each participant over at least four weeks in multiple testing sessions.

A further limitation to the present study is that the amount of guar gum used in the breakfast diets may have been different to that used by Nilsson et al. (2012). This may explain some of the discrepancies between the findings of the two

studies, and possibly why the present study found no significant effect of the guar gum breakfasts on any of the cognitive performance tasks. Nilsson et al. did not include the exact measurements of guar within their study; therefore, calculations of guar gum amount were based on limited information.

An additional limitation to the study was the inability to include the covariate of glucose tolerance in the analysis. This was also largely due to time restraints imposed on the present research. Previous research has shown that the ability to regulate glucose significantly impacts on an individual's sensitivity to glucose and also the effect it can have on cognitive performance. However, acknowledging this factor, in the initial stages of recruitment for the present study a preliminary blood sample was taken after participants had eaten their usual standard lunch to ensure no abnormal glucose readings were observed. Initial baseline tests were also taken every morning to allow for varying glucose levels after fasting. Yet, it still may have been beneficial to determine each participant's glucoregulation, as was done by Nilsson et al. (2012).

A final limitation is the age distribution of the sample group. Participants ranged from 26 to 66 years and were examined collapsed across the entire group. As previous studies have found a significant difference in regards to the effects of glucose and age on cognitive performance, such as memory (Kaplan et al., 2000), it may have been beneficial to split the sample into young and older adults and compare the difference of age. However, in the present study recruiting two age groups proved difficult due to the time constraints of the study, and also because the trials required participants to volunteer considerable amounts of time each week, which most potential participants could not commit to. The majority of studies have used either young or old participants with only a few including age as an independent variable. Future studies should examine the difference between younger and older participants.

**Summary and Conclusions**

The present study investigated the effects of kiwifruit and guar gum on glucose manipulation, and how the postprandial glucose response affects cognitive

performance. The cognitive tasks used were Choice Reaction Time, Word Recall, Serial Sevens, Trail Making B and Stroop. Satiety was also examined to establish the level of hunger after each breakfast, and to determine if this had any influence on cognitive performance.

The present study was unable to replicate the findings of Nilsson et al. (2012), showing that guar gum enhances performance on cognitive tasks relative to a control. However, trends in the data did show that a controlled and sustained glycaemic response did lead to enhanced performance on Choice Reaction Time, Serial Sevens, and Stroop. Enhanced performance on the remaining tasks, Word Recall and Trail Making B, was most likely due to practice effects; therefore, caution should be taken when interpreting the data gathered for those tasks.

Overall, there was no significant effect of Breakfast Type across all tasks, inconsistent with the findings of Nilsson et al. (2012). However, small to moderate effect sizes on these interventions suggest that a larger sample size may be needed to achieve statistically significant effects. This is supported by the fact that there were substantial differences on the postprandial blood glucose response between each breakfast. That is, kiwifruit and guar gum were found to control the glucose peak and maintain blood glucose concentrations above baseline over 180 mins.

The Choice Reaction Time task displayed higher levels of performance at time 2 compared to baseline, which was maintained at time 3. This finding indicated that healthy blood glucose levels could enhance RT abilities over a period of around three hours.

In comparison, performance on the Word Recall task improved significantly over all three Testing Times. However, it is concluded that these findings are largely due to practice effects and not the influence of the glucose response. A harder version of the task, not subject to practice effects, may have elicited effects. Previous studies have found a significant relationship between glucose and recall (Korol & Gold, 1998).

Significant results were also found across all three time periods for the Trail Making B task, but this is also likely due to practice effects. Thus, no interpretation can be accurately and validly made from the results. This is further strengthened by the fact that previous research using normal blood glucose levels have failed to find any difference in this task.

The Serial Sevens task followed the findings of previous research, in that maintained blood glucose can enhance RT performance on this task. Trends in the data showed that kiwifruit and guar gum (individually and combined) enhanced and sustained performance over the 180 min time frame.

Lastly, performance on the Stroop task was also found to be influenced by glucose; however, as for previous studies, this was not the case on the easier congruent condition. In comparison, the incongruent task had the most improved performance at time 2, indicating that the initial uptake of glucose may be the reason for improved RT.

Although no statistically significant results were found across the four breakfast diets, small to medium effect sizes for some cognitive tests are apparent. Across all tasks not influenced by practice effects (Choice Reaction Time, Serial Sevens, and Stroop), the Weet-Bix diet yielded better performance at time 2 of testing compared to baseline with a slight decrease at time 3. These results suggest that the initial rise in glucose availability may have facilitated performance, and the slight decline in performance to the drop in blood glucose levels. The remaining three breakfast diets were more likely to prolong performance on these tests, displaying comparable results across time 2 and 3. Thus, it is proposed that the sustained increment of blood glucose may be beneficial in maintaining cognitive abilities for up to a period of three hours.

The present study is the first to integrate kiwifruit and guar gum into an investigation of the effects of glucose and cognitive performance. An interesting finding was the observation of the effect they both had on the postprandial glycaemic response, when consumed individually and combined. This manipulation shows that both products are capable of controlling the glucose

response to food, and effectively work to maintain levels above baseline over a prolonged period of time. Additionally, the health benefits are further supported by the finding that both work to increase satiety. Further research may benefit from examining the impact of kiwifruit and guar gum over a longer period of time.

In conclusion, the presence of statistically significant enhancement effects of kiwifruit and guar gum has not been confirmed by this study. However, the significant differences in Testing Time collapsed across Breakfast Type indicate that the manipulation of glucose post-consumption is having an effect on some aspects of cognitive performance. The present study questions the conclusions drawn by Nilsson et al., (2012), who claimed that selective attention was enhanced by the consumption of guar. Further research needs to focus on the possible interactions between breakfast diets and the type of cognitive task. More informed knowledge of the components that make up these tasks used is needed if the effects of kiwifruit and guar gum on cognitive performance are to be fully understood.

## REFERENCES

Afkhami-Ardekani, M., & Shojaoddiny-Ardekani, A. (2007). Effect of vitamin C on blood glucose, serum lipids & serum insulin in type 2 diabetes patients. *The Indian Journal of Medical Research, 16,* 471-474.

Allen, P., & Bennett, K. (2012). *SPSS statistics. A practical guide: Version 20*. Australia: Cengage Learning Australia Pty Limited.

American Academy of Neurology (2013, October). *Lower blood sugars may be good for the brain.* Retrieved from www.sciencedaily.com/10/131023165016.htm

Amiel, S. A., Dixon, T. D., Mann, R., & Jameson, K. (2008). Hypoglycaemia in Type 2 diabetes. *Diabetic Medicine, 25*, 245-254. doi: 10.1111/j.1464-5491.2007.02341

Aro, A., Uustitupa, M., Voutilainen, E., Hersio, K., Korhonen, T., & Siitonen, O. (1981). Improved diabetic control and hypocholesterolaemic effect induced by long-term dietary supplementation with guar gum in type 2 (insulin-dependent) diabetes. *Diabetologia, 21*, 29-33.

Auckland District Health Board. (2013). *The glycaemic index (GI) of foods.* Retrieved from www.health.govt.nz

Azari, N. P. (1991). Effects of glucose on memory processes in young adults. *Psychopharmacology, 105*, 521-524.

Baron, A. D. (1998). Postprandial hyperglycaemia and a-glucosidase inhibitors. *Diabetes Research and Clinical Practice, 40*, 51-55.

Bellisle, F., Blundell, J. E., Dye, L., Fantino, M., Fern, E., Fletcher, R. J., Lambert, J., Roberfroid, M., Specter, S., Westenhofer, J., & Westerterp-Plantenga, M. S. (1998). Functional food science and behaviour and psychological functions. *British Journal of Nutrition, 80*, 173-193.

Benton, D., & Owens, D. S. (1993). Blood glucose and human memory. *Psychopharmacology, 113*, 595-607.

Benton, D., Owens, D. S., & Parker, P. Y. (1994). Blood glucose influences memory and attention in young adults. *Neuropsychologia, 32*, 595-607. doi: 10.1016/0028-3932(94)90147-3

Benton, D., & Parker, P. Y. (1998). Breakfast, blood glucose, and cognition. *American Journal of Clinical Nutrition, 67*, S772-S778.

Berdanier, C. D. (1996). Advanced nutrition: macronutrients. *American Journal of Clinical Nutrition, 63*, 141-142.

Blackburn, N. A., & Johnson, I. T. (1981). The effect of guar gum on the viscosity of the gastrointestinal contents and on glucose uptake from the perfused jejunum in the rat. *British Journal of Nutrition, 46*, 239-246. doi: 10.1079/BJN19810029

Braaten, J. T., Wood, P. J., Scott, F. W., Riedel, K. D., Poste, L. M., & Collins, M. W. (1991). Oat gum lowers glucose and insulin after an oral glucose load. *American Journal of Clinical Nutrition, 53*, 1425-1430.

Brand-Miller, J., Foster-Powell, K., & Mendosa, R. (2004). *The new glucose revolution. The glycemic index solution for optimum health. What makes my blood glucose go up and down?* Sydney, Australia: Griffin Press.

Brandt, K. R., Gibson, E. L., & Rackie, J. M. (2013). Differential facilitative effects of glucose administration on Stroop task conditions. *Behavioural Neuroscience, 127*, 932-935. doi:10.1037/a0034930

Brouns, F., Bjorck. I., Frayn, K. N., Gibbs, A. L., Lang, V., Slama, G., & Wolever, T. M. S. (2005). Glycaemic index methodology. *Nutrition Research Reviews, 18*, 145-171.

Bruning, J. C., Gautam, D., Burks, D. J., Gillette, J., Schubert, M., Orban, P. C., Klein, R., Krone, W., Muller-Wieland, D., & Kahn, C. R. (2000). Role of brain insulin receptor in control of body weight and reproduction. *Science, 289*, 2122-2125. doi: 10.1126/289.5487.2122

Chen, Y. Y., Wu, P. C., Weng, S. F., & Liu, J. F. (2011). Glycaemia and peak incremental indices of six popular fruits in Taiwan: Healthy and type 2 diabetes subjects compared. *Journal of Clinical Biochemistry and Nutrition, 49*, 195 – 199. doi: 10.3164/jcbn.11-11

Cohen, J. (1988). *Statistical power analysis for the behaviour sciences* (2nd ed.). NJ: Lawrence Erlbaum Associates.

Cox, D. J., Kovatchev, B. P., Gonder-Frederick, L. A., Summers, K. H., McCall, A., Grimm, K. J., & Clarke, W. L. (2005). Relationships between hyperglycemia and cognitive performance among adults with type 1 and type 2 diabetes. *Diabetes Care, 28*, 71-77. doi: 10.2337/diacare.28.1.71

Craft, S., Murphy, C., & Wemstrom, J. (1994). Glucose effects on complex memory and nonmemory tasks: The influence of age, sex, and glucoregulatory response. *Psychobiology, 22*, 95-105. doi: 10.3758/BF03327086

Craig, A. (1986). Acute effects of meals on perceptual and cognitive efficiency. *Nutrition Reviews*, 163-171.

Cryer, P. E. (2002). Hypoglycaemia: The limiting factor in the glycaemic management of Type 1 and Type 2 Diabetes. *Diabetologia, 45*, 937-948. doi: 10.1007/s00125-002-0822-9

D'Anci, K. E., Watts, K. L., Kanarek, R. B., & Taylor, H. A. (2009). Low-carbohydrate weight-loss diets. Effects on cognition and mood. *Appetite, 52*, 96-103.

Del Prato, S. (2003). Loss of early insulin secretion leads to postprandial hyperglycaemia. *Diabetologia, 46*, 2-8. doi: 10.1007/s00125-002-0930-6

De Vivo, D. C., Trifiletti, R. R., Jacobson, R. I., Ronen, G. M., Behmand, R. A., & Harik, S. I. (1991). Defective glucose transport across the blood-brain barrier as a cause of persistent hypoglycorrhachia, seizures and developmental delay. *New England Journal of Medicine, 325*, 703-709. doi: 10.1056/NEJM199109053251006

Diabetes New Zealand. (2008). *Low blood glucose (hypoglycaemia) for Type 1 diabetes.* Retrieved from www.diabetes.org.nz

Diabetes Nutrition. (2014). *Glycaemic Index – What's it all about*. Retrieved from www.diabetes.org.uk

Donnelly, L. A., Morris, A. D., Frier, B. M., Ellis, J. D., Donnan, P. T., Durrant, R., Band, M. M., Reekie, G., & Leese, G. P. (2005). Frequency and predictors of hypoglycaemia in Type 1 and insulin-treated Type 2 diabetes: A population-based study. *Diabetic Medicine, 22*, 749-755. doi: 10.1111/j.1464-5491.2005.01501.x

Donohoe, R. T., & Benton, D. (1999). Cognitive functioning is susceptible to the level of blood glucose. *Psychopharmacology, 145*, 378-385. doi: 10.1007/s002130051071

Donohoe, R., & Benton, D. (2000). Glucose tolerance predicts performance on tests of memory and cognition. *Physiology & Behavior, 71*, 395-401. doi: 10.1016/S0031-9384(00)00359-0

Driesen, N. R., Cox, D. J., Gonder-Frederick, L., & Clarke, W. (1995). Reaction time impairment in insulin-dependent diabetes: Task complexity, blood glucose levels, and individual differences. *Neuropsychology, 9*, 246-254. doi: 10.1037/0894-4105.9.2.246

Duelli, R., & Kuschinsky, W. (2001). Brain glucose transporters: Relationship to local energy demand. *News in Physiological Sciences, 16*, 71-76.

Dye, L., & Blundell, J. (2002). Functional foods: Psychological and behavioural functions. *British Journal of Nutrition, 88*, 187-211. doi: 10.1079/BJN2002684

Ebeling, P., Yki-Jarvinen, H., Aro, A., Helve, E., Sinisalo, M., & Koivisto, V. A. (1988). Glucose and lipid metabolism and insulin sensitivity in type 1 diabetes: The effect of guar gum. *American Journal of Clinical Nutrition, 48*, 98-103.

Evans, M. L., Pernet, A., Lomas, J., Jones, J., and Amiel, S. A. (2000). Delay in onset of awareness of acute hypoglycemia and of restoration of cognitive performance during recovery. *Diabetes Care, 23*, 893-898.

Faerch, K., Borch-Johnsen, K., Vaag, A., Jorgensen, T., & Witte, D. R. (2010). Sex differences in glucose levels: A consequence of physiology or methodological convenience? The inter99 study. *Diabetologia, 53*, 858-865. doi: 10.1007/s00125-010-1673-4

Feldman, J., & Barshi, I. (2007). The effects of blood glucose levels on cognitive performance: A review of the literature. *National Aeronautics and Space Administration*. Retrieved from www.nasa.gov

Fischer, K., Colombani, P.C., Langhams, W., & Wenk, C. (2001). Cognitive performance and it relation to postprandial metabolic changes after different macronutrient ingestion in the morning. *British Journal of Nutrition, 85*, 393-405. doi: 10.1079/BJN2000269

Friedrich, M. J. (2012). Ethnic blood glucose variations. *The Journal of the American Medical Association, 308*, 1746-1748. doi: 10.1001/2012.jama.11946

Gerozissis, K. (2002). Brain insulin: Regulation, mechanisms of action and functions. *Cellular and Molecular Neurobiology, 23*, 1-25. doi: 10.1023/A:1022598900246

Gibson, E. L., & Green, M. W. (2002). Nutritional influences on cognitive function: Mechanisms of susceptibility. *Nutrition Research Reviews, 15*, 169-206. doi: 10.1079/NRR200131

Gjedde, A., & Crone, C. (1981). Blood-brain glucose transfer: Repression in chronic hyperglycemia. *Science, 214*, 456-457. doi: 10.1126/science.7027439

Gold, P. E. (1986). Glucose modulation of memory storage processing. *Behavioural and Neural Biology, 45*, 342-349.

Green, M. W., Taylor, M. A., Elliman, N. A., & Rhodes, O. (2001). Placebo expectancy effects in the relationship between glucose and cognition. *British Journal of Nutrition, 86*, 173-179. doi: 10.1079/BJN2001398

Greenwood, C. E. (2003). Dietary carbohydrate, glucose regulation, and cognitive performance in elderly persons. *International Life Sciences Institute, 61*, 68-74.

Groop, P., Aro, A., Stenman, S., & Groop, L. (1993). Long-term effects of guar gum in subjects with non-insulin dependent diabetes mellitus. *American Journal of Clinical Nutrition, 58*, 513-518.

Gropper, S., & Smith, J. (2013). *Advanced nutrition and human metabolism* (6th ed.). Belmont, CA: Wadsworth Cengage Learning.

Hale, F., Margen, S., & Rabak, D. (1982). Postprandial hypoglycaemia and "psychological" symptoms. *Biological Psychiatry, 17*, 125-130.

Herman, W. H., & Cohen, R. M. (2012). Racial ethnic differences in the relationship between HbA1c and blood glucose: Implications for the diagnosis of diabetes. *Journal of Clinical Endocrinology and Metabolism, 97*, 1067-1072. doi: 10.1210/jc.2011-1894

Hoffman, R. G., Speelman, D. J., Hinnen, D. A., Conley, K. L., Guthrie, R. A., & Knapp, R. K. (1989). Changes in cortical functioning with acute hypoglycemia and hyperglycemia in type 1 diabetes. *Diabetes care, 12*, 193-197. doi: 10.2337/diacare.12.3.193

Holmes, C., Koepke, K. M., & Thompson, R. G. (1986). Simple versus complex performance impairments at three blood glucose levels. *Psychoneuroendocrinology, 11*, 353-357. doi:10.1016/0306-4530(86)90021-1

Jenkins, D. J. A., Kendall, C. W. C., Axelsen, M., Augustin. L. S. A., & Vuksan, V. (2000). Viscous and nonviscous fibres, nonabsorbable and low glycaemic index carbohydrates, blood lipids and coronary heart disease. *Current Opinion in Lipidology, 11*, 49-56.

Jenkins, D. J. A., Kendall, C. W. C., Augustin, L. S. A., Franceschi, S., Hamidi, M., Marchie, A., Jenkins, A. L., & Axelsen, M. (2002). Glycaemic index: Overview of implications in health and disease. *American Journal of Clinical Nutrition, 76*, 266-273.

Jenkins, D. J. A., Leeds, A. R., Gassull, M. A., Cochet, B., & Alberti, G. M. M. (1977). Decrease in postprandial insulin and glucose concentrations by guar and pectin. *Annals of Internal Medicine, 86*, 20-23.

Jiang, G., & Zhang, B. B. (2003). Glucagon and regulation of glucose metabolism. *American Journal of Physiology, 284*, 671-678. doi: 10.1152/ajpendo.00492.2002

Kaplan, R. J., Greenwood, C. E., Winocur, G., & Wolever, T. M. S. (2001). Dietary protein, carbohydrate, and fat enhance memory performance in the healthy elderly. *American Journal of Clinical Nutrition, 74*, 687-693.

Kennedy, D. O., & Scholey, A. B. (2000). Glucose administration, heart rate and cognitive performance: effects of increasing mental effort. *Psychopharmacology, 149*, 63-71. doi: 10.1007/s002139900335

Kerr, D., MacDonald, I. A., & Tattersall, R. B. (1989). Adaption to mild hypoglycemia in normal subjects despite sustained increases in counter-regulatory hormones. *Diabetologia, 32*, 249-254. doi: 10.1007/BF00285293

Korol, D. L., & Gold, P. E. (1998). Glucose, memory, and aging. *American Journal of Clinical Nutrition, 67*, S764-S771.

Krotkiewski, M. (1984). Effect of guar gum on body-weight, hunger ratings and metabolism in obese subjects. *British Journal of Nutrition, 52*, 97-105.

Kucera, H., & Francis, W. N. (1967). *Computational analysis of present-day American English*. Providence: Brown University Press. doi: 10.1126/science.1112085

Lam, T. K. T., Gutierrez-Juarez, R., Pocai, A., & Rossetti, L. (2005). Regulation of blood glucose by hypothalamic pyruvate metabolism. *Science, 309*, 943-947. doi: 10.1126/science.1112085

Lezak, M. D., Howieson, D. B., Loring, D. W., Hannay, H. J., & Fischer, J. S. (2004). *Neuropsychological assessment* (4th ed.). New York: Oxford University Press.

Liljeberg, H., Akergerg, A., & Bjorck, I. (1999). Effect of the glycaemic index and content of indigestible carbohydrates of cereal-based breakfast meals on glucose tolerance at lunch in healthy subjects. *The American Journal of Clinical Nutrition, 69*, 647-655.

Lloyd H.M., Rodgers, P.J., Hedderley, D.I., & Walker, A.F. (1996). Acute effects on mood and cognitive performance of breakfasts differing in fat and carbohydrate content. *Appetite, 27*, 151-164. doi: 10.1006/appe.1996.0042

Lobmann, R., Smid, H. G. O. M., Pottag, G., Wagner, K., Heinze, H. H., & Lehnert, H. (2000). Impairment and recovery of elementary cognitive function induced by hypoglycemia in type-1 diabetic patients and healthy controls. *The Journal of Clinical Endocrinology and Metabolism, 85*, 2758-2766.

Lucia, K. Witte, V., Winkler, A., Grittner, U., Rujescu, D., & Floel, A. (2013). Higher glucose levels associated with lower memory and reduced hippocampal microstructure. *Neurology, 81*, 1746-1752.

Lund-Andersen, H. (1979). Transport of glucose from blood to brain. *Physiological Review, 59*, 305-352.

Maassen, M. M., Lingenfelser, T., Gluck, H., Renn, W., Eggstein, M., & Jakober, B. (1990). Cognitive and psychomotor function during hypoglycemia: A comparison between porcine and human insulin. *Neuropsychobiology, 24,* 30-36. doi: 10.1159/000119039

Manning, C. A., Hall, J. L., & Gold, P. E. (1990). Glucose effects on memory and other neuropsychological tests in elderly humans. *Psychological Science, 1*, 307-311. doi: 10.1111/j.1467-9280.1990.tb00223.x

Markus, C. R., Panhuysen, G., Jonkman, L. M., & Bachman, M. (1999). Carbohydrate intake improves cognitive performance of stress-prone individuals under controllable laboratory stress. *British Journal of Nutrition, 82*, 457-467. doi: 10.1017/S0007114599001713

Malmlof, K., Nunes, C. S., & Askbrant, S. (1989). Effects of guar gum on plasma urea, insulin and glucose in the growing pig. *British Journal of Nutrition, 61*, 67-73. doi: 10.1079/BJN19890093

Mayer, G., Nitsch, R., & Hoyer, S. (1990). Effects of changes in peripheral and cerebral glucose metabolism on locomotor activity, learning and memory in adult male rats. *Brain Research, 532*, 95-100. doi: 10.1016/0006-8993(90)91747-5

McAulay, V., Deary, I. J., Ferguson, S. C., & Frier, B. M. (2001). Acute hypoglycemia in humans causes attentional dysfunction while nonverbal intelligence is preserved. *Diabetes Care, 24*, 1745-1750. doi: 10.2337/diacare.24.10.1745

McCrimmon, R., Deary. I., & Frier, B. (1997). Auditory information processing during acute insulin-induced hypoglycemia in non-diabetic human subjects. *Neuropsychologia, 35*, 1547-1553. doi: 10.1016/S0028-3932(97)00080-8

McNay, E. C., Fries, T. M., & Gold, P. E. (2000). Decreases in rat extracellular hippocampal glucose concentration associated with cognitive demand during a spatial task. *Proceedings of the National Academy of Sciences, 97*, 2881-2885. doi: 10.1073/pnas.050583697

McNay, E. C., McCarty, R. C., & Gold, P. E. (2001). Fluctuations in brain glucose concentration during behavioural testing: Dissociations between brain areas and between brain and blood. *Neurobiology of Learning and Memory, 75*, 325-331. doi: 10.1006/nlme.2000.3976

Melanson, K. J. (1999). Blood glucose patterns and appetite in time-blinded humans: Carbohydrate versus fat. *American Journal of Physiology Regulatory Integrative and Comparative Physiology, 277*, 337-345.

Messier, C., & Gagnon, M. (1996). Glucose regulation and cognitive functions: Relation to Alzheimer's disease and diabetes. *Behavioural Brain Research, 75*, 1-11. doi: 10.1016/0166-4328(95)00153-0

Ministry of Health (2013). *Diabetes.* Retrieved from www.health.govt.nz

Mishra, S., & Monro, J. (2012). Kiwifruit remnants from digestion in vitro have functional attributes of potential importance to health. *Food Chemistry, 135*, 2188-2194. doi: 10.1016/j.foodchem.2012.06.102

Mizock, B. A. (2001). Alterations in fuel metabolism in critical illness: hyperglycaemia. *Best Practice and Research Clinical Endocrinology and Metabolism, 15*, 533-551. doi: 10.1053/beem.2001.0168

Monro, J. A., & Humphrey-Taylor, V. (1994). *Composition of New Zealand foods 5. Bread and flour*. Christchurch, New Zealand: New Zealand Institute for Crop and Food Limited.

Monro, J. A. (2013). Kiwifruit, carbohydrate availability, and the glycaemic response. In M. Boland & P. J. Moughan, Advances in food and nutrition research (vol 68). Nutritional benefits of kiwifruit. (pp. 257-271). Burlington: Academic Press.

Nilsson, A. C., Ostman, E. M., Holst, J. J., & Bjorck, I. (2008). Including indigestible carbohydrates in the evening meal of healthy subjects improves glucose tolerance, lowers inflammatory markers, and increases satiety after a subsequent standardized breakfast. *The Journal of Nutrition, 138*, 732-739.

Nilsson, A., Radeborg, K., & Bjorck, I. (2012). Effects on cognitive performance of modulating the postprandial blood glucose profile at breakfast. *European Journal of Clinical Nutrition, 66,* 1039-1043. doi: 10.1038/ejcn.2012.80

Nordlie, R. C., Foster, J. D., & Lange, A. L. (1999). Regulation of glucose production by the liver. *Annual Reviews: Nutrition, 19*, 379-406.

Pallant, J. (2001). *SPSS survival manual: A step-by-step guide to data analysis using SPSS*. Australia: Allen & Unwin.

Parker, R. S., Doyle, F. J., & Peppas, N. A. (2001). The intravenous route to blood glucose control. *Engineering in Medicine and Biology Magazine, 20*, 65-69. doi: 10.1109/51.897829

Plesman, J. (2011). What is hypoglycaemia. *Hypoglycaemic Health Association.* Retrieved from www.hypoglycemia.asn.au

Pramming, S., Thorsteinsson, B., Theilgaard, A., Pinner, T. M., & Binder, C. (1986). Cognitive function during hypoglycaemia in Type 1 diabetes mellitus. *British Medical Journal, 292*, 647-650.

Ragozzino, M. E., & Gold, P. E. (1995). Glucose injections into the medial septum reverse the effects of intraseptal morphine infusions on hippocampal acetylcholine output and memory. *Neuroscience, 68*, 981-988. doi: 10.1016/0306-4522(95)00204-V

Ragozzino, M. E., Unick, K. E., & Gold, P. E. (1996). Hippocampal acetylcholine release during memory testing in rats: Augmentation by glucose. *Proceedings of the National Academy of Sciences, 93*, 4693-4698.

Riby, L. M., Marriott, A., Bullock, R., Hancock, J., Smallwood, J., & McLaughlin, J. (2009). The effects of glucose ingestion and glucose regulation on memory performance in older adults with mild cognitive impairment. *European Journal of Clinical Nutrition, 63*, 566-571. doi: 10.1038/sj.ejcn.1602981

Rodgers, P. J., & Lloyd, H. M. (1994). Nutrition and mental performance. *Proceedings of the Nutrition Society, 53,* 443-456. doi: 10.1079/PNS19940049

Roozendaal, B. (2003). The hippocampus mediates glucocorticoid-induced impairment of spatial memory retrieval: Dependence on the basolateral amygdala. *Proceedings of the National Academy of Sciences, 100*, 1328-1333. doi: 10.1073/pnas.0337480100

Schmidl, M. K., & Abuza, T. P. (2000). *Essentials of functional foods*. Gaithersburg, MD: Aspen Publishers, Inc.

Schneider, C. (2013). How does your body naturally regulate blood glucose? *Diabetes Care*. Retrieved from www.diabetescare.net

Scholey, A. B., Harper, S., & Kennedy, D. O. (2001). Cognitive demand and blood glucose. *Physiology and Behavior, 73*, 585-592. doi: 10.1016/S0031-9384(01)00476-0

Sims, N. R., Smith, C. C. T., Davison, A. N., Bowen, D. M., Flack, R. H. A., Snowden, J. S., & Neary, D. (1980). Glucose metabolism and acetylcholine synthesis in relation to neuronal activity in Alzheimer's disease. *The Lancet, 315*, 333-336. doi: 10.1016/S0140-6736(80)90884-3

Szablewski, L. (2011). Glucose homeostasis – mechanism and defects. *Diabetes: Damages and Treatments*. Retrieved from www.intechopen.com/books/diabetes-damages-and-treatments

Smith, A. P., Kendrick, A. M., & Maben, A. L. (1992). Effects of breakfast and caffeine on performance and mood in the late morning and after lunch. *Neuropsychobiology, 26,* 198-204. doi: 10.1159/000118920

Smith, A. P., Clark, R., & Gallagher, J. (1999). Breakfast cereal and caffeinated coffee: Effects on working memory, attention, mood and cardiovascular function. *Physiology & Behaviour, 67*, 9-17. doi: 10.1016/S0031-9384(99)00025-6

Sommerfield, A. J., Deary, I. J., & Frier, B. M. (2004). Acute hyperglycemia alters mood state and impairs cognitive performance in people with type 2 diabetes. *Diabetes Care, 27*, 2335-2340. doi: 10.2337/diacare.27.10.2335

Strachan, M. W. J., Deary, I. J., Ewing, F. M. E., Ferguson, S. S. C., Young, M. J., & Frier, B. M. (2001). Acute hypoglycemia impairs the functioning of the central but not peripheral nervous system. *Physiology & Behaviour, 72*, 83-92. doi: 10.1016/S0031-9384(00)00380-2

Taylor, V. H., & MacQueen, G. M. (2007). Cognitive dysfunction associated with metabolic syndrome. *Obesity Review, 8*, 409-418. doi: 10.1111/j.1467-789X.2007.00401.x

Torsdottir, I., Alpsten, M., Andersson, H., & Einarsson, S. (1989). Dietary guar gum effects on postprandial blood glucose, insulin and hydroxyproline in humans. *American Institute of Nutrition, 119,* 1925-1929.

Vuorinen-Markkola, H., Sinisalo, M., & Koivisto, V. A. (1992). Guar gum in insulin-dependant diabetes: effects on glycaemic control and serum lipoproteins. *The American Journal of Clinical Nutrition,* 56, 1056-1060.

Warren, R. E., Frier, B. M. (2005). Hypoglycaemia and cognitive function. *Diabetes, Obesity and Metabolism, 7*, 493-503. doi: 10.1111/j.1463-1326.2004.00421.x

Will, J. C., & Byers, T. (1996). Does diabetes mellitus increase the requirement for vitamin C? *Nutrition Review, 54*, 193-202. doi: 10.1111/j.1753-4887.1996.tb03932.x

Wolever, T. M. (2003). Carbohydrate and the regulation of blood glucose and metabolism. *Nutrition Review, 61*, 40-48. doi: 10.1301/nr.2003.may.S40-S48

Wolever, T. M. S., Jenkins, D. J. A., Nineham, R., & Alberti, K. G. M. M. (1979). Guar gum reduction of post-prandial glycaemia: Effect of incorporation into solid food, liquid food, both. *British Journal of Nutrition, 41*, 505-510. doi: 10.1079/BJN19790065

Wolever, T. M. S., & Mehling, C. (2002). High-carbohydrate-low-glycaemic index dietary advice improves glucose disposition index in subjects with impaired glucose tolerance. *British Journal of Nutrition, 87*, 477-487. doi: 10.1079/BJN2002568

Wood, P. J., Braaten, J. T., Scott, F. W., Riedel, D., & Poste, L. M. (1990). Comparisons of viscous properties of oat and guar gum and the effects of these and oat bran on glycaemic index. *Journal of Agricultural and Food Chemistry, 38*, 753 – 757. doi: 10.1021/jf00093a036

World Health Organisation. (2006). *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia.* Geneva, Switzerland: WHO Press.

Yki-Jarvinen, H. (1990). Acute and chronic effects of hyperglycaemia on glucose metabolism. *Diabetologia, 33*, 579-585. doi: 10.1007/BF00400200

Zespri Kiwifruit. (2013, May). *Health communications toolkit*. New Zealand: Author.

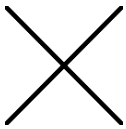
# APPENDICES

# Appendix A

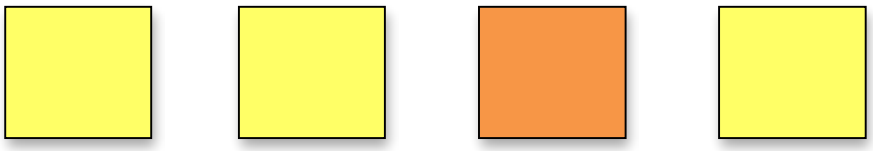
Examples of Cognitive Tasks: Choice Reaction Time, Word Recall, Trail Making B, Serial Sevens, and Stroop

## CHOICE REACTION TIME

'X' displayed that participants are required to focus on between responses.



Example of the boxes that appeared on the screen. Participants were required to respond to the orange box.



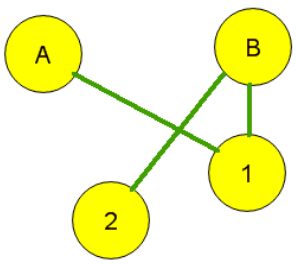
First box – 'Z' key, second box – 'X' key, third box – 'N' key, fourth box – 'M' key.

## WORD RECALL

Mule, Rail, Leaf, Hire, Twin, Drag, Flew, Alas, Loom, Cage, Seed, Cape, Code, Bond, Dull, Folk, Deed, Drug, Soup, Ugly

## TRAIL MAKING B

Example: A – 1, 1 – B, B – 2



## SERIAL SEVENS

From 100: 100, 93, 86, 79, 72, 65, 58, 51, 44, 37, 30, 23, 16, 9, 2

From 104: 104, 97, 90, 83, 76, 69, 62, 55, 48, 41, 34, 27, 20, 13, 6

## THE STROOP TEST

Congruent example: GREEN, BLUE, RED.

Incongruent example: GREEN, BLUE, RED.

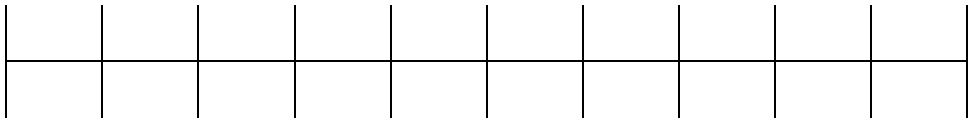
Note: Participants are required to respond to the colour the word is presented in, not the text of the word.

**Appendix B**

Satiety Scale

**SATIETY SCALE**

*Please indicate on the scale below how hungry you feel at this present time.*



Not at all Extremely

## Appendix C

Information Sheet

**PARTICIPANT INFORMATION SHEET**

| | | | |
|---|---|---|---|
| Study title: | ***Kiwifruit modulation of post-prandial glycaemia and its cognitive effects*** | | |
| Locality: | **The NZ Institute for Plant and Food Research, Palmerston North** | Ethics committee ref.: | **14/STH/77** |
| Lead investigator: | **John Monro** | Contact phone number: | **06 355 6137** |

You are invited to take part in a study of the effects of consuming kiwifruit and weetbix, with and without soluble dietary fiber, on blood glucose responses to the meal and cognitive performance. If you think you want to take part now, but change your mind later, you can pull out of the study at any time.

This Participant Information Sheet will help you decide if you'd like to take part. It sets out why we are doing the study, what your participation would involve, what the benefits and risks to you might be, and what will happen after the study ends. We will go through this information with you and answer any questions you may have. You do not have to decide today whether or not you will participate in this study. Before you decide you may want to talk about the study with other people, such as family, whānau, friends, or healthcare providers. Feel free to do this.

If you agree to take part in this study, you will be asked to sign the Consent Form on the last page of this document. You will be given a copy of both the Participant Information Sheet and the Consent Form to keep.

This document is 4 pages long. Please make sure you have read and understood all the pages.

## What is the purpose of the study?

The purpose of this study is to examine the effects of kiwifruit on blood glucose levels after a meal and the influence of these levels on cognitive performance, such as memory, attention and reaction time. The study will also examine how kiwifruit can interact with the dietary fiber, guar gum, to reduce blood glucose peaks after a meal, possibly resulting in a more sustained or improved cognitive performance. The impact of blood glucose levels on cognitive performance has previously been demonstrated in numerous studies; however, investigating the interaction of natural nutrients such as kiwi fruit and soluble fiber is a relatively

new approach. The study will use participants in the age bracket of 25-60 years, a population group that have a high rate of blood glucose-related health problems (e.g., diabetes and obesity). By examining ways to reduce blood glucose responses to food, the study aims to improve physical and mental wellbeing.

This study focuses on the use of natural foods and supplements to control blood glucose levels, and does not use any official medications. You will receive four types of breakfast, once each, throughout the study. They will be breakfast cereal (Weetbix™), Weetbix™ plus kiwifruit, Weetbix™ plus guar gum (soluble fiber), and Weetbix™ plus kiwifruit plus guar gum. You will be told at the end of the study which meal had the greatest improvement on blood glucose levels and cognitive performance.

The New Zealand Institute of Plant and Food Research in Palmerston North, alongside Massey University, are conducting this study. The two investigators, John Monro (Plant and Food Limited) and Haley Edwards (Massey University), will be contactable during the study by phone and email.

**What will my participation in the study involve?**

Individuals will be chosen to participate in the study if they are within the age bracket (25-60) and considered to be generally healthy. Health status will be determined initially by the use of a health questionnaire, which asks about your current and past health issues.

During the study you will be required to refrain from eating anything over night (12 hours) and then you will be given one of the four test breakfasts (once a week), and blood glucose levels will be monitored over the course of the morning. Blood samples will be taken five times over the day by the use of a small finger prick machine (similar to those used by diabetics). You will also complete five short cognitive tests. During the three hours you will be able to continue with your normal work if it is not strenuous (e.g., office work)

If you agree to take part in the study, you will be required to be available one morning a week, over four weeks, with each visit lasting a maximum of four hours. Lunch will be provided after the testing has been completed. A follow up meeting will also be included to feedback the results of the study and answer any questions. You will also be reimbursed for your time with a $20 petrol or supermarket voucher, at each of the four meeting times during the study.

You will have the opportunity to discuss with us what you would prefer to do with your blood samples after the reading has been taken. Any support or family members are welcome to join you during the study.

The cognitive tests will include tests of memory, selective attention, reaction time, processing speed and the evaluation of executive functions. All tests will be completed on a computer and instructions will be given at the time of testing. Before beginning the study you will be given a consent form to participate, and the health questionnaire. There will be various opportunities for you to raise any questions you may have. We will ask you if you need any information to be translated or given in another language and this will be provided.

**What are the possible benefits and risks of the study?**

Due to the natural methods used for intervention in this study, there are minimal foreseeable risks and side-effects. You may experience a small discomfort from the finger prick to draw blood; however, the finger prick device is used daily by many people with diabetes. If any discomfort occurs, you will be given the opportunity to take a break, or if necessary stop participation in the study.

Adverse health issues caused by blood glucose swings can occur in healthy individuals, as well as those with pre-existing health problems. Therefore, various groups in the population can benefit from the findings of this study. Kiwifruit and guar gum are easily accessible supplements, and the participants of this study may potentially immediately benefit from the effects of the meals in controlling blood glucose responses.

**Who pays for the study?**

The study is supported by The NZ Institute of Plant and Food Research, and no participating person will incur any costs at any stage.

**What if something goes wrong?**

If you were injured in this study, which is highly unlikely, you would be eligible for compensation from ACC just as you would be if you were injured in an accident at work or at home. You will need to lodge a claim with ACC, which may take some time to assess. If your claim is accepted, you will receive funding to assist in your recovery.

If you have private health or life insurance, you may wish to check with your insurer that taking part in this study won't affect your cover.

**What are my rights?**

Participation in this study is completely voluntary, and you have the right to decline to participate, or to withdraw from the research at any stage, without the need to give reason and also without experiencing any disadvantage.

As personal information is disclosed during this study, you have the right to access the information that we have collected about you at any stage. You will also be informed of your own blood glucose readings as soon as they become available; we would tell you if there was reason for concern. Information obtained from the questionnaire and blood samples will be kept completely confidential at all times. Only the investigators of the study will have access to these records.

If any new information arises about adverse or beneficial effects related to this study that may have an impact on health, then you will be informed straight away.

**What happens after the study or if I change my mind?**

On completion of the study, the researcher will provide the overall findings of the study. This will be given to you in written form so you can keep a copy for future reference. You will also have the opportunity to ask any questions or raise any concerns during this meeting. As stated earlier, you will have the right to withdraw from the study at any time.

The findings of the study will be written into a thesis report and may be available for future use. This formal report will also be available to you, if you are interested, by the end of 2014. Confidential health information must be securely stored for up to ten years; however, this information will not be available to access for future studies without your written permission.

All blood samples taken during the study will be destroyed immediately after the reading is recorded.

**Who do I contact for more information or if I have concerns?**

If you have any questions, concerns or complaints about the study at any stage, you can contact:

*Haley Edwards*
*Haley.e@hotmail.com*

*John Monro*
*john.monro@plantandfood.co.nz*

If you want to talk to someone who isn't involved with the study, you can contact an independent health and disability advocate on:

Phone: 0800 555 050
Fax: 0800 2 SUPPORT (0800 2787 7678)
Email: advocacy@hdc.org.nz

For Maori health support please contact :

*Daniel Kawana*
*d_m_kawana@hotmail.com*

You can also contact the health and disability ethics committee (HDEC) that approved this study on:

Phone: 0800 4 ETHICS
Email: hdecs@moh.govt.nz

## Appendix D

Consent Form

**PARTICIPANT CONSENT FORM**

Kiwifruit modulation on post-prandial glycaemia and its cognitive effects

**If you need an INTERPRETER, please tell us.**

**Please tick to indicate you consent to the following**

| | | |
|---|---|---|
| I have read, or have had read to me in my first language, and I understand the Participant Information Sheet. | Yes ☐ | No ☐ |
| I have been given sufficient time to consider whether or not to participate in this study. | Yes ☐ | No ☐ |
| I have had the opportunity to use a legal representative, whanau/ family support or a friend to help me ask questions and understand the study. | Yes ☐ | No ☐ |
| I am satisfied with the answers I have been given regarding the study and I have a copy of this consent form and information sheet. | 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 this affecting my medical care. | Yes ☐ | No ☐ |
| I consent to the research staff collecting and processing my information, including information about my health. | Yes ☐ | No ☐ |
| If I decide to withdraw from the study, I agree that the information collected about me up to the point when I withdraw may continue to be processed. | Yes ☐ | No ☐ |
| I understand that my participation in this study is confidential and that no material, which could identify me personally, will be used in any reports on this study. | Yes ☐ | No ☐ |
| I understand the compensation provisions for this study. | Yes ☐ | No ☐ |
| I know who to contact if I have any questions about the study in general. | Yes ☐ | No ☐ |
| I understand my responsibilities as a study participant. | Yes ☐ | No ☐ |
| I wish to receive a summary of the results from the study. | Yes ☐ | No ☐ |

**Declaration by participant:**

I hereby consent to take part in this study.

Participant's name: _____

Signature: _____ Date: _____

**Declaration by member of research team:**

I have given a verbal explanation of the research project to the participant, and have answered the participant's questions about it.

I believe that the participant understands the study and has given informed consent to participate.

Researcher's name: _____

Signature: _____ Date: _____

**Appendix E**

Health Questionnaire

# HEALTH SCREENING FORM

## Personal details

Name: _____

Age: _____

Contact details: _____

_____

*Emergency contact*

Name: _____

Contact details: _____

_____

*Family doctor*

Name: _____

Contact details: _____

_____

## Health history

1. Have you or anyone in your family (Grandparents, parents and siblings) ever experienced any of the following (tick for yes):
   - High blood pressure
   - Low blood pressure
   - Heart problems
   - Stroke
   - Breathing problems
   - Cancer or tumour
   - Obesity
   - Migraines/headaches
   - Diabetes
   - Epilepsy
   - Arthritis
   - Kidney/bladder disorders
   - Gastrointestnal disorders
   - Hernia
   - Allergies/asthma

- Blood disorder/diseases e.g. hepatitis
- Chronic condition e.g. lupus, arthritis
- Other (please identify) _____

If yes to any of the above, please explain and list any regular medications being taken **by you** (not family members)
e.g: High Blood pressure diruetics such as frusemide; ACE inhibitors such as Acupril; Calcium channel blockers such as nifedipine; beta blockers such as atenolol as well as Aspirin, blood disorders being treated by anti-coagulants; again aspirin, or warfarin or heparin.

_____

_____

_____

_____

2. Are you currently or have been taking any antacids, laxatives or supplements (what/why?)

_____

_____

_____

3. Have you had any antibiotics recently? If yes, when?

_____

_____

4. Do you any have food sensitivities/allergies, and in particular do you know if you are allergic to any fruits or vegetables (if yes, please give details)?

_____

_____

5. Do you smoke (yes/no)?

_____

6. Do you drink any alcohol (if yes please select from frequency options below)?
   No [ ] Yes, less than 5 units / week [ ] 5-14 units/ week [ ] 14-21 units / week [ ] more than 21 units / week [ ]

_____

_____

7. Do you drink any caffine (e.g., coffee, energy drinks) (if yes please select from frequency options below)?
   No [ ] Yes, less than 5 units / week [ ] 5-14 units/ week [ ] 14-21 units / week [ ] more than 21 units / week [ ]

_____

_____

8. Weight (kg)

   _____

9. Height (cm)

   _____

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

_____

_____

_____

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

Signature: _____ Date: _____

Researcher: _____ Date: _____

# Appendix F

ANOVA Tables for the Hunger Scale, Choice Reaction Time Task, Word Recall Task, Trail Making B Task, Serial Sevens Task, and Stroop Task

**ANOVA Table for the Choice Reaction Time Task**

**Test of Within-Subjects Effects**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 1467.23 | 1.80 | 811.53 | 2.67 | .09 | .12 | 4.83 |
| Breakfast | 1064.17 | 2.28 | 465.98 | .46 | .66 | .02 | 1.06 |
| Time*Breakfast | 3970.93 | 3.92 | 1013.00 | 1.92 | .12 | .09 | 7.53 |
| Error (Time) | 10443.10 | 34.35 | 304.00 | | | | |
| Error (Breakfast) | 43578.67 | 43.39 | 1004.33 | | | | |
| Error (Time*Breakfast) | 39294.73 | 74.48 | 527.59 | | | | |

**ANOVA Table for the Word Recall Task**

**Test of Within-Subjects Effects**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 252.858 | 1.66 | 152.77 | 70.16 | .00 | .79 | 116.13 |
| Breakfast | 8.15 | 2.66 | 3.06 | .16 | .90 | .01 | .43 |
| Time*Breakfast | 12.14 | 3.56 | 3.40 | .92 | .45 | .05 | 3.29 |
| Error (Time) | 68.48 | 31.45 | 2.18 | | | | |
| Error (Breakfast) | 960.44 | 50.53 | 19.01 | | | | |
| Error (Time*Breakfast) | 250.53 | 67.81 | 3.69 | | | | |

**ANOVA Table for the Trial Making B Task**

**Test of Within-Subjects Effects**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 5024.89 | 1.50 | 3342.14 | 37.68 | .00 | .67 | 56.65 |
| Breakfast | 13.98 | 2.82 | 4.95 | .03 | .99 | .00 | .07 |
| Time*Breakfast | 162.15 | 4.45 | 36.48 | .59 | .69 | .03 | 2.61 |
| Error (Time) | 2533.95 | 28.57 | 88.70 | | | | |
| Error (Breakfast) | 10247.13 | 53.64 | 191.04 | | | | |
| Error (Time*Breakfast) | 5240.42 | 84.46 | 62.05 | | | | |

**ANOVA Table for the Serial Sevens (both) Task**

**Test of Within-Subjects Effects**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 2588.71 | 1.67 | 1552.67 | 5.63 | .01 | .23 | 9.39 |
| Breakfast | 2559.60 | 2.40 | 1067.99 | 1.12 | .34 | .06 | 2.67 |
| Time*Breakfast | 2500.22 | 4.08 | 612.95 | 1.90 | .12 | .09 | 7.76 |
| Error (Time) | 8736.85 | 31.68 | 275.80 | | | | |
| Error (Breakfast) | 43605.56 | 45.54 | 957.60 | | | | |
| Error (Time*Breakfast) | 24972.34 | 77.50 | 322.22 | | | | |

**ANOVA Table for the Stroop Task**

**Test of Within-Subjects Effects (Overall)**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 5373.51 | 1.67 | 3217.98 | 4.04 | .03 | .18 | 6.74 |
| Breakfast | 284.98 | 2.85 | 100.03 | .05 | .98 | .00 | .13 |
| Time*Breakfast | 4194.39 | 4.84 | 866.79 | 1.12 | .36 | .06 | 5.42 |
| Error (Time) | 25297.49 | 31.73 | 797.35 | | | | |
| Error (Breakfast) | 119023.18 | 54.13 | 2198.70 | | | | |
| Error (Time*Breakfast) | 71133.942 | 91.94 | 773.691 | | | | |

**Test of Within-Subjects Effects (Congruent)**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 816.30 | 1.71 | 478.19 | .40 | .64 | .02 | .69 |
| Breakfast | 1284.28 | 2.63 | 488.60 | .23 | .85 | .01 | .60 |
| Time*Breakfast | 6039.87 | 4.66 | 1297.18 | 1.33 | .26 | .07 | 6.21 |
| Error (Time) | 38675.20 | 32.43 | 1192.42 | | | | |
| Error (Breakfast) | 107077.22 | 49.94 | 2144.06 | | | | |
| Error (Time*Breakfast) | 86058.63 | 88.47 | 972.78 | | | | |

**Tests of Within-Subjects Effects (Incongruent)**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Time | 8410.16 | 1.91 | 4406.11 | 5.17 | .01 | .21 | 9.87 |
| Breakfast | 1278.05 | 2.62 | 488.37 | .16 | .90 | .01 | .42 |
| Time*Breakfast | 3373.24 | 4.91 | 686.79 | .64 | .67 | .03 | 3.14 |
| Error (Time) | 30898.34 | 36.27 | 851.99 | | | | |
| Error (Breakfast) | 152487.70 | 49.73 | 3066.81 | | | | |
| Error (Time*Breakfast) | 1000198.26 | 93.32 | 1073.70 | | | | |

**ANOVA Table for the Hunger Scale**

**Test of Within-Subjects Effects**

| Source | Sum of Squares | *df* | Mean Squares | *F* | Sig. | Partial Eta Squared | Power |
|---|---|---|---|---|---|---|---|
| Breakfast | 138.93 | 2.44 | 56.88 | 20.13 | .00 | .51 | 1.00 |
| Error (Breakfast) | 131.13 | 46.41 | 2.83 | | | | |