THE RELATIONSHIPS AMONG THE EFFECT OF CARBOHYDRATES ON BLOOD GLUCOSE, APPETITE, FOOD INTAKE, MOOD AND MEMORY

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ABSTRACT

The hypothesis of this research was that there is a relationship between the effect of carbohydrates on blood glucose and their effects on appetite, food intake, mood and memory. Experiment 1 examined the effects of 300kcal preloads of sucrose, polycose, amylose and amylopectin on blood glucose over one hour. Experiment 2 examined the effect of the same treatments on appetite, food intake, mood and memory. The final experiment examined the relationships among the glycemic response to sucrose, polycose, glucose, fructose/glucose mixture and sucralose preloads and their effects on appetite, food intake, mood and memory.

A relationship was observed between the high glycemic carbohydrates, sucrose, polycose and glucose and decreased mealtime energy intake at one hour. No relationship was found between the glycemic response to carbohydrates and mood and memory. Sucrose preloads were found to improve memory performance.

It is concluded that the glycemic response to carbohydrates is associated with their effect on appetite and food intake but not mood and memory.

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I. INTRODUCTION

The complex interactions of psychosocial and cultural factors associated with obesity indicate that the mechanisms underlying the disease are complex and deeply rooted in biologic systems. Obesity is an imbalance between energy intake and energy expenditure resulting in an excessive storage of energy in the form of fat. Obesity has multiple causes and creates an enormous psychological burden, not to mention increased mortality rates and susceptibility to certain risk factors. One area of investigation that may contribute to a greater understanding of the etiology of obesity, is the role of macronutrients in satiety and food intake.

Carbohydrates comprise the main energy source in our diets (Asp 1994), but in addition to providing energy, their ingestion affects many aspects of brain function. The concept that glucose regulates satiety and food intake is the basis for the glucostatic theory of food intake regulation (Mayer 1953). Carbohydrate ingestion and the glucose derived from it also play a role in cognitive processes such as mood and memory.

This literature review explores the effects of carbohydrates on satiety, food intake, mood and memory. A more in-depth review of the relationship between the glycemic response to carbohydrates and satiety, food intake, mood and memory is presented. Finally, the interrelationships between carbohydrate intake and satiety, food intake, mood and memory are discussed.

II. LITERATURE REVIEW

A. FOOD INTAKE REGULATION

1.Overview

Understanding how the complex processes of hunger, appetite and satiation lead to energy balance is essential in determining the etiology of obesity. One important dietary determinant of food intake is the macronutrient composition of foods.

The primary mechanism by which carbohydrates are thought to regulate satiety is through the postprandial increase in blood glucose, however not all studies support this hypothesis. The cause of the discrepancies may lie in the failure of many investigators to control for the physio-chemical properties of the carbohydrate treatments. It is shown that the glycemic response is affected by many factors such as the structure, form and extent of processing of the carbohydrate treatment (Brown 1998; Goddard et al 1984 ; Holt & Brand Miller 1995).

Thus, the primary focus of this section of the literature review is to examine the influence of carbohydrates on appetite and food intake and discuss the role of blood glucose as a physiological mechanism by which carbohydrates regulate energy intake.

2. Satiety

With the initiation of food ingestion a progression of physiological and psychological responses occur leading to satiation and termination of food intake (Anderson 1994). Technically, satiety can be defined as an inhibition of hunger that arises as a consequence of food ingestion, whereas satiation is the process that brings a period of eating to a halt. Satiety is described as the ability of a food to affect hunger and reduce food intake at a future meal (Kissileff et al 1984). Of the many experimental paradigms

employed to investigate the satiating capacity of a food, the preload paradigm is the most widely used. The effect of a treatment on the amount of energy subsequently consumed from a food, beverage or meal is used as a simple and convenient assay to determine the effect of a nutrient or food on appetite.

It has been suggested that a cascade of satiety signals interact together to determine human motivation and food ingestion behaviour (Blundell et al 1994). There are four main mediators influencing appetite and food consumption including sensory, cognitive, preabsorptive and postabsorptive factors.

2.a. Cognitive and Sensory Cues

The amount of food ingested during a period of eating depends upon sensory and cognitive cues as well as the energy and nutrient content of the food (Castonguay et al 1986). The sensory or cephalic phase is initiated by the sight, smell and even the thought of food (Feldman & Richardson 1986). This results in the activation of salivary glands, secretion of gut hormones and the subjective feeling of hunger. Cognitive factors involve social and learned behaviours whereby food intake and selection are influenced by what is deemed acceptable (Herman 1996). If hunger is weak, energy intake may be enhanced by pleasant sensory and cognitive factors. Presentation of a novel or palatable food has been found to override feelings of satiety and increase food intake. Conversely, even when hunger signals are strong, food that is unfamiliar, unpleasant or forbidden may result in failure to respond to hunger signals (Castonguay et al 1986).

2. b. Preabsorptive signals

Peripheral signals such as receptors in the mouth detect taste, temperature and texture of the meal, while receptors in the stomach detect the presence of food and relay

information on the bulk, osmolarity and chemical qualities along the vagus nerve to the central nervous system (CNS) (Anderson 1994, Rolls 1995). The vagus nerve transmits information to the hypothalamus, which is known to be the brain region responsible for integration of peripheral signals regarding food intake and energy balance. There are two major areas within the hypothalamus known to regulate food intake; the ventral medial nucleus is the brains' satiety centre while the lateral hypothalamic area is classed as the hunger centre (Martin & Mullen 1987).

The rate of gastric emptying has been implicated as a factor in food intake regulation, with higher gastric emptying rates resulting in greater appetite (McHugh & Moran 1985). The mechanism proposed for this response is determined in part by the change in stomach volume monitored by stretch receptors and the rate of entry of nutrients into the duodenum. The delivery of nutrients to the small intestine stimulates the release of several peptides shown to mediate energy intake, such as cholecystokinin (CCK), bombesin and glucagon-like peptides (GLP-1) (Gibbs & Smith 1986, 1988, Silver et al 1989).

Cholecystokinin slows gastric emptying through contraction of the pyloric sphincter. Further evidence suggests that CCK mediates food intake suppression through a peripheral neurocrine mechanism. Upon its release, CCK is thought to directly bind to CCK-A receptors situated on the gastric afferent vagus, which projects to the ventromedial hypothalamus to decrease food intake (Gutzwiller 2000). Antagonists specific to the CCK-A receptor have been shown to block the food intake suppression observed following a protein preload in rats (Trigazis 1997).

Glucagon-like peptide is a biologically active product of the prohormone, proglucagon released from enteroendocrine L-cells in the distal gut in response to carbohydrate ingestion (Gutzwiller et al 1999, Flint et al 1998). GLP-1 in both the brain and periphery functions as a mediator of food intake suppression in the rat. More recent evidence has shown that intravenous infusion of GLP-1 decreases food intake in humans (Gutzwiller et al 1999).

2. c. Postabsorptive Signals

Post-absorptive signals are those that arise after the absorption and storage of nutrients. The liver is the first organ of passage after nutrients are absorbed from the gut and enter the portal circulation. Satiety signals following absorption of nutrients such as glucose (glucostatic theory) and amino acids (aminostatic theory) may be sent via the liver to the brain via the vagus nerve through their metabolic action (Anderson 1994). Heat produced from metabolism of these nutrients increases body temperature which may inhibit feeding (thermostatic theory) (Westerterp-Plantega et al 1994; Biernacka 1995). Short-term regulation may be related to the quantity of glycogen stores in muscle and the liver, whereas the size of adipose tissue (lipostatic theory) may reflect long-term regulation (Westerterp-Plantega 1994).

Because of the close linkage between the absorption of carbohydrate and insulin release, insulin has been proposed as a regulator of food intake (Woods et al 1996). Highly specific, receptor-mediated transport mechanisms exist for passage of insulin from the circulation to the interstitial fluid of the brain. Furthermore, areas of the brain known to be involved in food intake regulation contain neurons with numerous insulin receptors on their cell membranes (Woods et al 1996). Administration of insulin into the

animal brain under a glucose clamp results in a decrease in food intake and body weight (Schwarz et al 1992).

3. Macronutrients and Food Intake Regulation

Not only energy intake, but also the macronutrient composition, palatability and energy density of the diet play a key role in the development of satiety and energy balance (Raben & Holst 1996).

Food intake selection studies indicate that there are specific regulatory mechanisms governing the intake of fat, protein and carbohydrate. Animal studies have shown that protein is more satiating than carbohydrate, which in turn is more satiating than fat (Trigazis 1998). The evidence for such a hierarchy of satiety with macronutrients in humans has not been as clearly defined. In humans, protein produces the greatest effect on satiety (de Castro 1987; Poppitt et al 1998). There are reports that carbohydrate is more satiating than fat (Woodend 2000; Blundell et al 1994; Rolls et al 1988) or that they are both equally satiating (de Graaf et al 1992; Rolls et al 1994).

Because carbohydrates constitute a large portion of the diet it is of interest to further explore their role in energy regulation.

4. Carbohydrates and Food Intake Regulation

The effect of dietary carbohydrates on energy intake has received considerable attention. Previously, it was thought that complex carbohydrates such as starches were more satiating than sugars. Because of the diversity in availability for digestion, it can be expected that the source of carbohydrate is an important determinant of the physiological effects of carbohydrates. For example, sugars which are readily available for absorption

can be predicted to have different physiological effects than carbohydrates that are more complex (starch) and less rapidly absorbed.

Sugars in the diet are primarily in three forms: sucrose, glucose and fructose and each of these has received investigation for their effects on appetite and food intake.

4.a. Sucrose and Food Intake

In addition to providing energy, sugars provide sweet taste. Because of its hedonic value, sucrose has been labeled unique among carbohydrates. It is suggested that its hedonic properties override normal regulatory mechanisms controlling appetite and as a result contribute to excess energy intake (Anderson 1995). However, survey data have found an inverse relationship between obesity and sugar (Hill & Prentice 1995).

The belief that sucrose contributes to excess energy intake through bypassing regulatory systems is based in part on experimental designs that have not adequately tested the effects of sugar on energy intake (Anderson 1995). In preload studies in which a dose of sucrose is given and appetite and food intake measured, the quantity consumed and the interval between treatment and measurement of appetite and food intake is crucial. By increasing the time interval between the preload and test meal, there is a greater chance of not detecting an effect of treatment on hunger. For example, a recent study found no difference in total energy intake following a sugar free beverage versus a sugar rich beverage (41g) in eleven, young males after 1hour and 50 minutes (Holt et al 2000).

For these reasons a detailed examination of the relationships between the quantity of sucrose in preloads (25g, 50g and 75g) and appetite and food intake was undertaken (Woodend 2000). From the study, it was clear that all treatments including the smallest

dose of sucrose (25g) suppressed appetite and food intake at one hour compared with the water control. Thus sucrose intake is detected by food intake regulatory mechanisms and suppresses but does not increase food intake.

Because sucrose is composed of glucose and fructose, one or both of these monosaccharides may explain the effect of sucrose on regulatory mechanisms.

4.b. Glucose and Fructose

In humans, ingestion of glucose (50g) is shown to suppress food intake within one hour, (Rogers & Blundell 1989; Rogers et al 1988; Blundell et al 1994). The effect of fructose on food intake compared with glucose is not clear.

Several studies have demonstrated that 50g fructose suppresses energy intake to a greater extent than equicaloric preloads of glucose at test meals from 38 minutes (Rodin 1990) to 2.25 hours later (Spitzer & Rodin 1987; Rodin et al 1988; Rodin 1991).

Other studies report that the suppressive effect of fructose is not so robust (Guss et al 1994; Kissileff & Gruss 1989; Stewart et al 1997). No significant difference was observed between cereals containing 30g fructose or 33.5g glucose on meal time energy intake either at 30 minutes or 120 minutes post consumption even though blood glucose responses were significantly different (Stewart et al 1997). It is possible that the presence of starch in the cereals masked the subtle treatment effect. For example, the addition of 15g starch abolishes the suppressive effect of 50g fructose at 2.25 hours (Rodin 1991). Similarly, no differences in food intake are observed between 50g fructose and 50g glucose at 2.25 hours when given in a mixed nutrient meal including starch (Rodin 1988).

4.c. Starch and Food Intake Regulation

Evidence that the composition of starches is an important determinant of food intake derives from studies that have compared high amylose to high amylopectin starch. Amylose is the minor component of starch and is primarily a linear glucose polymer in which the individual monomers are connected solely by alpha (1-4) glycosidic linkages. Amylopectins are the major components of starch (~70%), containing alpha (1-4) and alpha (1-6) linkages to form a branched structure. The branched form of amylopectin allows a greater surface area for digestive enzymes and is subsequently more rapidly absorbed. It is this factor which is proposed to determine the differential effects of these two forms of starches on energy intake.

It has been shown that a high amylose containing meal induces more prolonged satiety over six hours than a high amylopectin meal (van Amelsvoort & Westrate 1992). The low, sustained increase in postprandial blood glucose following amylose ingestion has been proposed as the cause of the prolonged satiety.

B. GLUCOSE METABOLISM, FOOD INTAKE REGULATION AND THE GLYCEMIC RESPONSE

Of the many mechanisms by which carbohydrates regulate food intake, glucose utilisation and uptake have been the focus of many studies. This originates from the glucostatic theory proposed by Mayer (1953) that decreased glucose utilisation is detected by the brain at glucosensitive sites and is the signal initiating a period of feeding.

1. Glucostatic Theory

The glucostatic theory of feeding proposes that blood glucose levels are closely monitored as they reflect the availability of energy to the brain and other tissues (Mayer 1953). More recent evidence suggests that a decrease in glucose utilisation is the primary

stimulus for meal initiation rather than the absolute level of blood glucose in both animals and humans (Campfield & Smith 1985; 1986; 1990; Campfield et al 1996).

Transient declines in blood glucose of the correct magnitude and time course which induce meal initiation are hypothsised to be detected by peripheral and central glucoreceptive elements and mapped into feeding behaviour (Campfield et al 1996). When the blood glucose and meal patterns were continuously measured in free feeding rats, there was a smooth, gradual decline in blood glucose concentration 12 minutes before onset of feeding which decreased 11.6 percent below baseline. Further research demonstrated that meal requests and changes in hunger rating were related to spontaneous and insulin-induced transient declines in blood glucose concentration in human subjects isolated from food and time cues (Campfield et al 1996).

Based on the glucostatic hypothesis and the different satiating capacities of carbohydrates, the effect of carbohydrates on postprandial blood glucose may reflect a mechanism controlling satiety and energy intake.

2. Glycemic Response

Carbohydrates are only absorbed as their constituent monosaccharides; i.e glucose, fructose or galactose. The glycemic response to carbohydrates has been defined as the area under the blood glucose curve when blood glucose concentrations are plotted against time (Wolever 1990). To provide a basis for comparing glycemic responses to foods, the glycemic index (GI) was developed.

2.a. Glycemic Index

The glycemic index is a classification of the potential of foods to raise blood glucose (Jenkins et al 1981). This index compares the incremental area under the blood

glucose response curve of a 50g carbohydrate portion of a test food expressed as a percent of the response to the same amount of carbohydrate from a standard food for example white bread, ingested by the same subject (Wolever 1990). Since the GI standardises the glycemic response to a test food, it is corrected for between subject variation thereby allowing glycemic responses from different studies to be compared.

The bell shaped form of the glycemic response to carbohydrates is affected by two opposing factors: increasing blood glucose due to entry of glucose from the gut and liver into the peripheral circulation, versus the uptake of glucose through the action of insulin.

The glycemic index reflects the digestion and absorption rates of carbohydrates. Rapid and high increases in postprandial blood glucose, observed with high GI foods, represent fast digestion, compared to slow and maintained increases in blood glucose which represent slow digestion of low GI foods (Brand Miller 1994, Wolever 1990).

2.b. Type of Carbohydrate

Postprandial insulin and glucose responses are dependent upon the source and amount of carbohydrate. Differences in glycemic responses are observed not only between simple sugars and complex carbohydrates, but also within each group. Contrary to popular belief, the glycemic response to many common starches is similar to that of high glycemic sugars.

2.b.i Sugars

A range of glycemic responses are observed upon ingestion of various sugars (Lee & Wolever 1998). Glucose produces a rapid and high increase in postprandial blood glucose and insulin compared to a slower and more gradual response to fructose (Moyer & Rodin 1993; Rodin 1991; Rodin et al 1988). Sucrose tends to elicit a lower increase in

postprandial blood glucose concentrations than glucose. Indeed, fructose and sucrose have lower GI values than most common starchy foods (Foster-Powell & Brand Miller 1995). For example, it has been shown that replacing starch with 21g and 43g sucrose in a high GI breakfast cereal lowers the glycemic and insulinemic responses (Brand Miller & Lobbezoo 1994).

2.b. ii. Starch

One of the most important factors influencing the rate of starch digestion and the subsequent glycemic response is the ratio of amylopectin to amylose (Truswell 1992).

The open branched structure of amylopectin starch makes it easier to digest than the linear amylose starch and as a result, meals made with high amylose starch are expected to induce a lower postprandial plasma glucose response than meals made from high amylopectin starches (Byrnes 1995). Indeed, high amylose starch meals have been shown to produce lower postprandial glucose and insulin responses (van Amelsvoort and Westrate 1992, Behall et al 1988) and improved fasting triglyceride and cholesterol concentrations in healthy and hyperinsulinemic individuals compared to a high amylopectin containing starch meal (Zhou and Kaplan 1997, Behall and Howe 1995).

3. Glycemic Response, Satiety and Food Intake

The glucostatic mechanism proposes that low blood glucose levels trigger the onset of a period of feeding and high blood glucose levels signal satiety and the termination of feeding (Mayer 1953). However, it is not merely the level of blood glucose that triggers hunger but the rate of glucose utilisation. Campfield et al's research implies that it may be the shape or the rate of change over time (slope) of the glycemic curve that determines energy intake. The effect of the shape of the postprandial blood glucose curve

may potentially be extrapolated to situations where a subject is fed a preload in order to manipulate the glycemic response. Accordingly, the relationship between glycemic response and food intake may be examined.

3.a. Relationship between Glycemic Response and Satiety

Although the literature suggests an inverse relationship between the glycemic response and satiety, the majority of studies that have attempted to test this hypothesis have failed to control for all the necessary dimensions required to make a definitive conclusion. For example, the time intervals for measuring subjective satiety and/or food intake have varied between 30 minutes and 6 hours. The test meals have not always been balanced for energy, fibre and macronutrient content. Finally, the tools used to measure satiety and energy intake are inconsistent among investigators.

Among the studies that have attempted to control for these variables, a greater satiating effect of low glycemic carbohydrate meals has been shown over 2 to 6 hours (van Amelsvoort 1992; Holt & Brand Miller 1995). For example, consumption of a high amylose starch mixed meal produced a stronger decrease in hunger and increased feelings of fullness for up to 6 hours post consumption compared to a low amylose meal (van Amelsvoort 1992). Similarly, ingestion of high amylose puffed rice (986 kcal) increased satiety and decreased energy intake at a meal at 2 hours compared to ingestion of low amylose puffed rice (957 kcal) (Holt & Brand Miller 1995).

It has also been shown that peak satiety scores are inversely related to the glycemic and insulin index for 3 hours following consumption of seven breakfasts (Holt 1992). Although multivariate analysis specified fibre and GI as significant predictors of satiety, the relationship still held when the high fibre treatment was removed from the

analysis, suggesting that a high glycemic response is associated with decreased satiety (Holt et al 1992). However the satiety ratings were correlated to the energy density of the breakfasts. In other words, as the glycemic response was higher and peak satiety scores decreased, the energy content of the cereals also decreased. Therefore the satiety ratings may be attributed to the energy content of the cereals and not necessarily the glycemic response.

One of the mechanisms by which low glycemic carbohydrates may mediate satiety is through their slower digestion rate and subsequent increased contact with intestinal glucose receptors and prolonged stimulation of putative satiety peptides. For example, an increase in satiety and a decrease in hunger is observed when absorption of a 300kcal glucose beverage was decreased by addition of 5g guar gum (Lavin & Read 1995). No difference was observed on gastric emptying rates when guar gum was added to the glucose drink. It was therefore proposed that the viscous properties of guar gum increased satiety through delayed absorption of glucose and increased contact of glucose with receptors in the small intestine and subsequent release of putative satiety peptides.

Not all researchers support a relationship between the glycemic response and satiety perhaps due to the many variables introduced within a mixed meal study. For example, the glycemic response to breakfast cereals containing either 30g fructose or 33.5g glucose was examined on a test meal, 30 minutes or 120 minutes post consumption (Stewart et al 1997). Although significantly different blood glucose responses were observed, there was no differences between the low (fructose) and high (glucose) glycemic treatments on energy intake. Similarly, no relationship was observed between the glycemic response to 50g of carbohydrate from various sources and satiety in

sedentary and active individuals measured at 30 and 120 minutes (Krishnamachar & Mickelsen 1987). The presence of other nutrients and the lack of control for fibre, palatability and energy density within mixed meals may prevent detection of satiety cues.

In contrast, when a meta analyses was performed on data from three experiments that examined the effects of various preloads on plasma glucose, insulin and subjective satiety, a positive relationship was found between plasma glucose and insulin area under the curves (AUC) and increased satiety AUC (Raben et al 1996). However, because other variables such as GIP levels and carbohydrate content were strongly intercorrelated, it was not possible to distinguish their independent roles in appetite regulation.

The question remains, do blood glucose levels affect appetite? Some studies have addressed this question by taking a closer look at the mechanisms by which carbohydrates influence appetite and energy intake.

For example, earlier studies have demonstrated either no effect (Woo et al 1984) or an increase in hunger and food intake under hyperinsulinemic and hyperglycemic (10 mmol/L) conditions (Rodin et al 1985). In contrast, more recent studies have shown that acute hyperglycemia (15 mmol/L) induces satiety over 240 minutes (Gielkens et al 1998) and decreases food intake at 140 minutes (Chapman et al 1998) and has been related to the satiety peptide, GLP-1 (Lavin et al 1998). It is therefore unclear whether the stimulus for satiety after carbohydrate ingestion is directly related to blood glucose or is secondary to blood glucose though the release of satiety peptides or insulin.

While it is evident that the effect of carbohydrates on blood glucose and satiety is specific to their composition, the hypothesis that a relationship exists between glycemic response and satiety has not been adequately tested.

B. MOOD

1. Overview

Behavioural change following consumption of carbohydrate rich foods has been documented (Spring et al 1983*).* Many studies reporting the effect of carbohydrates on behaviour have been based on the hypothesis that carbohydrate intake increases brain serotonin synthesis. Serotonergic neurons have been reported to participate in sleep, pain, mood and pain sensitivity (Lieberman et al 1983; Hartmann 1983) and it has been suggested that a deficiency in serotonin release may characterise some depressive illnesses (van Praag 1982*).* Decreased arousal and increased ratings of sleepiness have been reported in some studies following consumption of carbohydrate rich foods (Lieberman et al 1986; Pivonka & Grunwald 1990; Spring et al 1983). Others have shown no effect (Woodend 2000; Reid & Hammersley 1995).

The following discussion will explore some of the possible pathways through which carbohydrate consumption can influence an individual's mood.

2. Dietary Modulation of Mood

The ingestion of carbohydrate-rich foods releases insulin, which increases the availability of tryptophan for brain uptake. Because the enzyme converting tryptophan to serotonin is not saturated at normal brain tryptophan concentrations, increased tryptophan uptake leads to more serotonin synthesis.

A high carbohydrate, low protein meal stimulates insulin secretion, which in turn lowers the plasma level of most amino acids with the exception of tryptophan that remains bound to albumin in the plasma. As a result the ratio of tryptophan to the other

large neutral amino acids (LNAA) in the plasma increases. Tryptophan competes with the LNAA's for entry into the brain, so as the tryptophan to LNAA ratio increases the tryptophan gains a competitive advantage and crosses the blood brain barrier where it increases serotonin synthesis.

2.a. Tryptophan and Mood

Support for the serotonin hypothesis has been obtained through experiments that increase or decrease brain tryptophan concentrations (Lieberman et al 1986). For example, a significant improvement in dysphoria, mood swings, tension and irritability has been observed 17 days after supplementation with L-tryptophan in patients with premenstrual dysphoric syndrome (PMS). Similarly, short term administration of (500mg) tryptophan in a high carbohydrate meal increased feelings of lethargy and somnolence over 3 hours (Leathwood & Pollet 1982/3).

Conversely, dietary treatments that deplete brain tryptophan are associated with negative mood states (Delgado et al 1990; Benkelfat et al 1994; Smith et al 1999). Tryptophan depletion decreases brain levels of serotonin to levels associated with those found in depressed subjects producing a model whereby mood can be studied directly. Many studies have proved the efficacy of this method; the tryptophan depletion method is found to cause: significant declines in central serotonin turnover (Carpenter et al 1998), increases in the scores in depression scales on the Multiple Affect Adjective Checklist (Young et al 1995) and reappearance of depression in subjects who had previously been successfully treated for depression by anti-depressant drugs (Delgado et al 1990).

2.b. Carbohydrates and Mood

There is substantial evidence to support the theory that carbohydrate consumption improves mood in affected populations such as depression, premenstrual syndrome and carbohydrate cravers (Sayegh et al 1995; Wurtman et al 1989; Lieberman et al 1986). The effect of carbohydrate consumption on mood in healthy adults is not clear for a number of reasons. Food induced changes in mood in a normal population are subtle and hard to measure. Furthermore, the majority of the experiments designed to test the hypothesis that carbohydrates regulate mood have failed to control for many confounding variables, including subject population, time of day and the macronutrient content of the test meal.

Daytime alertness is influenced by circadian rhythms, often resulting in an energy peak in the morning, which declines until early evening (deCastro 1986). Increased alertness has been observed following high carbohydrate breakfasts, whereas increased fatigue has been found following high carbohydrate lunches. A decline in fatigue/dysphoria is observed 3 hours after a low fat, high carbohydrate breakfast (Lloyd et al 1996). Similarly, a high fibre, carbohydrate rich breakfast meal is shown to increase ratings of alertness compared to fat rich, low fibre carbohydrate meals (Holt et al 1999). Conversely, a high carbohydrate (105g), low protein (0.7g) mixed meal at lunchtime has been shown to increase ratings of fatigue after 2 hours (Spring et al 1989). Non-carbohydrate cravers report feeling less alert, more fatigued, sleepy and more depressed for two hours following a high carbohydrate lunch (104g wheat starch) (Lieberman et al 1986).

The macronutrient content of breakfasts has small but significant effects on mood state (de Castro 1987). Meals with different compositions may produce different psychological effects consistent with their various physiological actions. The

consumption of a preload similar in macronutrient composition to that habitually eaten tends to enhance mood independent of macronutrient effects (Rogers & Hedderly 1996). Likewise, deviation from usual intake has been observed to produce a decline in mood.

The time frame of measurement after a preload may also have an effect on the outcome. Previous studies have shown a treatment effect as early as 30 minutes (Christensen & Redig 1993; Smith et al 1988) and as late as two hours (Lieberman et al 1986) after a meal.

Studies that have attempted to address the role of specific nutrients in mood have suggested that carbohydrate ingestion either has no regulatory effect on mood or leads to increased fatigue.

For example, ingestion of a 400kcal maltodextrin preload was found to increase fatigue over 4 hours (Cunliffe et al 1997). Similarly, increased sleepiness is observed one hour following consumption of 50g sucrose (Pivonka & Grunewald 1990). In contrast, other studies have found no effect of pure nutrient preloads on mood. For example, a range of sucrose doses (25g, 50g, 75g) had no effect on mood in young healthy males over one hour (Woodend 2000). Similarly, 100g sucrose had no effect on mood 20 minutes and 4 hours post preload (Reid & Hammersley 1995).

Further examination of the relationship between carbohydrate consumption and mood regulation is required. By eliminating the many confounding variables shown to affect the measure of mood, the mechanisms by which carbohydrate consumption affects mood may become clearer.

D. MEMORY

1. Overview

Glucose is the primary fuel for the brain and for this reason many investigators have studied the role of carbohydrates in cognitive functioning. An improvement in memory upon administration of glucose is most easily demonstrated in subjects with memory deficits including the elderly and those with Alzheimer's Disease and Downs Syndrome (Manning et al 1993, 1998). It would appear that the effect of carbohydrates on cognition in the young is subtle and may be related to their ability to deal with a glucose load rather than an underlying pathological disorder (Benton et al 1994). There is substantial evidence supporting a relationship between blood glucose regulation and cognition, with recent evidence suggesting multiple sites through which glucose and other sugars such as fructose may affect memory (Rodriguez et al 1999). Understanding the integration of both central and peripheral pathways may present a more realistic picture of the mechanisms underlying memory modulation (Messier & White 1987, Gold 1991). A review of the literature is presented with specific emphasis on the role of blood glucose in memory regulation and the role of carbohydrates such as fructose and sucrose on memory.

2. Memory

Experiences are remembered through the modulation of memories and it is understood that certain experiences are more vividly remembered than others. The reasoning behind this phenomenon has been implicated to involve hormonal control, specifically, noradrenergic and cholinergic systems (Gold 1995; Messier et al 1990). Memories are formed readily if hormones are released at the time of the experience as in a

stressful situation, thereby enhancing the formation of the memory. One of the first studies to examine the effects of adrenaline induced memory enhancement was in rats trained to avoid a brief foot shock through one trial passive avoidance tasks (Hall et al 1989). Systemic injections of adrenaline immediately after training, to mimic the hormonal response to a foot shock i.e. adrenaline release, resulted in memory performance similar to that observed with a higher foot shock. Adrenaline is a hormone released from the adrenal gland in response to stress and is not present in the brain under most conditions (Axelrod et al 1959). The inability of adrenaline to cross the blood brain barrier suggests a peripheral action on memory (Gold et al 1986). One such peripheral action of adrenaline is an increase in circulating levels of glucose.

3. Carbohydrates and Memory

Glucose produced from the digestion and absorption of dietary carbohydrates is essential to normal functioning of the nervous system. The brain depends on glucose as its major source of fuel and has therefore developed a highly regulated system to ensure that an excess of the nutrient is available (Sieber & Trastman 1992). The role of glucose in mediating memory has been extensively examined in both rodents and humans. More recent evidence supports the presence of multiple pathways through which carbohydrates such as glucose and perhaps fructose and sucrose may affect memory.

3.a. Glucose

A number of investigations have suggested an important role for glucose in modulating memory. Earlier studies found that post-training injections of glucose restored memory in memory deficient mice (Hall and Gold 1986). Adrenaline enhances

performance in fed rats but not rats deprived of food for 24 hours, suggesting that hyperglycemia subsequent to adrenaline injections contributes to the memory enhancing effects of adrenaline (Talley et al 2000). Glucose administration produces a bimodal dose response function, both in rodents (Messier and White 1987; Gold 1986; Kopf et al 1993; Rodriguez et al 1994) and elderly humans (Parsons &Gold 1992), implicating the presence of two or more independent mechanisms by which glucose may affect memory. However there is substantial evidence to suggest that the modulation of memory in the young is uniquely different from that observed in the elderly.

3.b. Memory in the Young and Aged

There is a large body of work demonstrating a beneficial effect of carbohydrate consumption, specifically glucose on memory in the aged. Young adults appear to be less sensitive to the modulating effects of carbohydrates. In one respect, consumption of 50g glucose is reported to enhance performance in young (Gonder-Frederick et al 1987: Hall et al 1989) and old subjects (Manning et al 1990, 1992). A fasting state or failure to eat breakfast results in a decline in performance of word list recall, which is reversed upon administration of a 50g glucose-supplemented drink (Benton et al 1998). Administration of 50g glucose to young and aged humans improves performance on the prose passage (logical memory), a test of contextual verbal memory, and for a composite memory score across tests in the elderly (Hall et al 1989). In contrast, older patients demonstrate dose-dependent (0g, 25g, 50g, 75g) improvements in delayed recall performance and reaction time compared to younger patients who demonstrate a decline in attentional performance at 75g dose compared to placebo (Fucetola et al 1999). Similarly, young subjects demonstrate no improvement in memory 30 minutes after consuming a 30g and 100g

glucose beverage (Azari 1991) or following a 50g glucose preload (Winder & Borrill 1998). More challenging tasks and a more rigid dietary fast may have been required to detect an effect. Fasting state and time of day are important factors affecting cognition and behaviour.

Quality of memory decreases with age in humans and animals. The cognitive deficits observed in the elderly may be related to either a problem in the regulatory mechanisms such as a deficiency in neuroendocrine regulators or a loss of structural, chemical or electrical components of neurons. The ability to distinguish between both alternatives has important clinical and experimental relevance (Korol and Gold 1998). The age-related change in anatomical use of the brain in memory processing may reflect dynamic re-allocation of networks of the brain, for young and old good performers of a memory task use different regions of the brain. Specifically, young adults activate frontal regions, whereas elderly performers rely on occipital regions (Hazlett et al 1998). The uncertainty surrounding the effects of glucose on memory in healthy adults may be partially explained by the notion that some of these participants are working at their optimal physiological and cognitive efficiency, thereby functioning at or near ceiling level. This would explain the greater sensitivity for memory enhancement in the elderly with cognitive deficits. Any benefits observed in the young may only be observed when engaged in demanding cognitive tasks. A more physiological explanation is the difference in glucose metabolism between the aged and young (Stone et al 1989). Circulating glucose levels may play a more important role in regulating brain glucose metabolism under

conditions of impaired brain glucose utilisation compared to when brain glucose utilisation is normal (Hall et al 1989).

4. Blood Glucose Regulation and Memory Performance

Evidence suggests that the elderly, with lower glucose regulation are more sensitive to a glucose preload than young healthy adults with normal glucose regulation (Hall et al 1989). For example, a Wechsler story is more easily remembered following moderate increases in blood glucose in the elderly whereas a similar but smaller effect is observed in young subjects (Gonder-Frederick 1987, Manning et al 1990). Glucose improves word learning tasks and verbal declarative memory (e.g. a prose passage) in young adults with poor glucose regulation (Messier et al 1999) and older males with good glucose regulation (Craft et al 1994). Those subjects with higher baseline blood glucose levels post-consumption perform better on tests of delayed recall of a prose passage and attention tests (Korol and Gold 1998). Furthermore, a positive correlation between baseline blood glucose levels and forgetting in both glucose and placebo drinkers is observed. Those with an initially high blood glucose level retain information better and have faster reaction times in a rapid information processing task (RIPT) (Benton et al 1994). The emphasis on regulation may be more of a reflection of the rates of recovery and amplitude of the blood glucose concentrations rather than the blood glucose levels per se.

4.a. Blood Glucose Dynamics

Findings consistently implicate a mechanism related to rising and falling blood glucose concentrations as a determinant of memory improvement in young adults. Lists of

words or a story are more easily learned by subjects with high (>7.2 mmol/L) rather than low (<4.4 mmol/L) blood glucose concentrations (Lapp1981, Hall et al 1989). Indeed, a significant correlation with blood glucose concentration and delayed recall performance following 25g glucose in young healthy individuals is observed. However, the improvement was independent of the individuals' differences in baseline blood glucose concentrations (Foster et al 1998). It is proposed that those with higher blood glucose levels will have higher brain glucose levels, consistent with the observation that memory improvement is associated with high brain glucose levels (Benton et al 1994). A decrease in blood glucose levels between one reading of a cognitive task and a second may reflect the replenishment of intra-cellular glucose stores required for cognitive function. Higher levels of blood glucose would allow for greater passage of glucose into the brain to fuel the memory process. It is therefore reasonable to assume that those subjects with low blood glucose will be disadvantaged when engaged in a memory task (Donohoe & Benton 1999). It is consistently observed that those whose blood glucose levels are increasing following a glucose preload, remember significantly more words from a word list than those whose blood levels are falling (Benton & Owens 1993, Benton, Owens & Parker 1994). However, a review of the literature indicates that the majority of evidence supporting a relationship between blood glucose levels and memory in young adults is based on one group of investigators in particular (Benton and Owens 1993, Benton et al 1994, Benton & Sargeant 1992). Based on the methodologies employed in these studies, it is unreasonable to assume that the relationship between blood glucose and memory performance was adequately addressed. For example, their experimental designs are severely impeded by a lack of dietary restrictions in their subjects. When examining the

effect of a dietary nutrient within an experimental paradigm, it is imperative to control for the fasting state for the presence of other nutrients and environmental influences occludes the outcome. By reducing the level of noise within an experimental design, there is a greater chance of observing an effect specific to the intervention or preload, in this case. Secondly, it is unreasonable to assume arbitrary levels of high ($\geq$5 mmol/l) and low (<4.9 mmol/L) blood glucose levels as a basis for the effect of blood glucose levels on memory. The normal physiological range for fasting blood glucose levels is between 4.0 mmol/L and 6.0 mmol/L. More specifically, the main hypothesis proposed by these studies is that a change of 0.5 mmol/L in blood glucose levels is directly proportional to the effect on memory performance. Considering the error level of the portable blood glucose meter is greater than the arbitrary change of 0.5 mmol/L, there is no physiological basis for these assumptions. The question still remains; is there an association between blood glucose and memory in humans? If so, is it the level of glucose or the rate of change that is important? Do carbohydrates other than glucose improve memory? Would a high glycemic carbohydrate produce a more beneficial effect than a low glycemic carbohydrate?

It is not altogether unreasonable to assume that there is in fact no relationship between blood glucose levels and cognitive performance (Green et al 1997). The body is well developed to prevent potentially detrimental declines in glucose supply to the brain i.e. in the face of insulin administration or extreme starvation (Amiel 1994). In healthy individuals, blood glucose levels are maintained at ~5mmol/L even after an overnight fast. In the postabsorptive state, glucose enters the blood stream almost exclusively from the liver due to the process of gluconeogenesis and glycogenolysis, however, the rise in blood

glucose is rapidly counteracted by insulin release, causing the uptake of glucose into the liver, muscle and adipose tissue (Frayn & Kingman 1995). The brain is therefore impervious to serious fluctuations in glucose flow and is an improbable explanation for glucose mediated memory. It is possible however, that high baseline blood glucose levels or rapidly rising glucose levels (Benton & Owens 1993), reflects an increase in sympathetic activity, including increased cortisol and adrenaline secretion. Certainly, incentive motivation significantly improves reaction times in young adults, independent of blood glucose levels, suggesting greater sympathetic arousal (Rogers et al 1995). A more specific explanation may be that glucose regulation, utilisation and physiological arousal during cognitive demand are linked, in that certain processes associated with physiological arousal may serve as mechanisms to increase the delivery of substrates to the brain. A high cognitive load induces physiological arousal such as increased salivary cortisol and urinary catecholamines and cardiac output (Fibiger et al 1986). Indeed, performance on a Serial Sevens test following consumption of 25g glucose is correlated with the magnitude of change in both blood glucose and heart rate (Kennedy & Scholey 2000).

5. Sucrose and Memory

There is a general consensus among the lay public that sucrose consumption may negatively affect health, contribute to excess energy intake and cause hyperactivity or aggressive behaviour. Because sugar is one of the main dietary components, any relation between sugar and behaviour is of great concern. Regardless of popular opinion, it is of interest that clinical investigations to date have failed to observe a relationship between sucrose and negative behaviour (White & Wolraich 1995). Only recently has the role of sugar as a positive reinforcer and cognitive enhancer been proposed. Sucrose is a

disaccharide of fructose and glucose and produces a moderately rapid increase in blood glucose post consumption. If there is indeed a role for blood glucose in cognition, then the release of glucose through the digestion of most rapidly absorbed carbohydrates should affect memory. Preliminary evidence in our laboratory supports a positive role for sucrose in memory in young adults. A dose response experimental paradigm in young males, demonstrated that consumption of 300kcal sucrose prevents a decline in memory for word lists between 15 and 60 minutes post consumption (Hui 1998 unpublished). Considering that sucrose is composed of both glucose and fructose monomers, there may be a beneficial effect of fructose in memory. Recent evidence supports a positive role for fructose in modulating memory in animals, however, the role of fructose in regulating memory processes in human adults is yet to be addressed.

6. Fructose and Memory

Fructose is a 2-ketohexose that is metabolised almost exclusively in the liver (Henry & Crapo 1991) and does not readily cross the blood brain barrier. Fructose is metabolised preferentially over glucose with a half-life of 18 minutes in the blood compared to glucose, which has a half life of 43 minutes (Henry & Crapo 1991). It is proposed that the enhancement of memory observed by both endogenous and systemically administered glucose is through a centrally mediated mechanism (Gold 1991, Wenk 1989). However, because the time dependent effects of fructose on memory parallel those observed with glucose and because fructose does not readily cross the blood brain barrier, a peripheral action is postulated (Horne et al 1997).

A passive avoidance to active avoidance negative transfer paradigm was used to investigate the effects of glucose and fructose on recently acquired memories in rats. Equimolar 10, 32, 100 and 200 mg/kg subcutaneous doses of both sugars impaired acquisition of the reversal task, 3.2 mg/kg had no effect and 320 mg/kg enhanced subsequent performance (Rodriguez et al 1994). The authors went on to replicate these results in a more recent study whereby glucose and fructose treatment dose-dependently (100mg/kg and 2000mg/kg) enhanced memory for a passive avoidance response. Although the dose response functions for the effects of glucose and fructose on memory where indistinguishable, a combined 1000mg/kg glucose plus 1000mg/kg fructose dose did not improve memory to the same extent as a singular 2000mg/kg dose of fructose or glucose alone (Rodriguez et al 1999). These results suggest a different mode of action for fructose and glucose. However, the similar cubic dose response functions for glucose and fructose combined with the observation that the memory modulating effects parallel the effects of these sugars on hepatic blood glucose concentrations, suggests a common peripheral mechanism (White 1991, Messier & Gagnon 1996). The many pathways by which carbohydrates may modulate memory peripherally is addressed below.

7. Peripheral Mechanism for Memory Enhancement

Gastrointestinal hormones can modulate central behaviour such as memory through a number of mechanisms. They may pass directly through the blood brain barrier, or produce a secondary effect through pituitary hormone release, alter circulating metabolites such as glucose or free fat or alter blood flow directly to the brain (Morley 1986, Flood & Morley 1989; 1988). Peripherally administered or released substances that

modulate memory storage, but do not freely enter the brain, may produce their effects on memory by activating peripheral receptors that send messages centrally through the vagus nerve. Vagal afferents as compared to vagal efferents are suggested to carry messages about the peripheral states that lead to the modulation of memory (Clark et al 1998). These findings are extended to humans whereby vagus nerve stimulation administered after learning significantly enhances retention (Clark et al 1999). Early studies demonstrate that feeding mice immediately following training, enhances memory retention and that cholecystokinin (CCK), a gastrointestinal hormone released during a meal, also enhances retention after peripheral administration (Flood & Morley 1988). The memory enhancing effect of CCK-8 is blocked when the vagus nerve is cut, indicating that CCK-8 may regulate memory retention and meal induced enhancement of memory through ascending fibres in the vagus nerve (Flood et al 1987, 1989). The CCK-A receptor is positively associated with learning and memory functions (Nomoto et al 1999) as compared to the CCK-4 which may exert a negative influence on memory consolidation and retrieval (Shlik et al 1998). The facilitating effect of post-training administration of GRP and bombesin on memory is blocked by vagotomy (Flood & Morley 1988), suggesting that the vagus nerve is one pathway by which systemic gastrointestinal peptides influence storage processes. Based on these findings, it is probable that the enhanced memory retention associated with feeding is secondary to the release of gastrointestinal hormones (Morley et al 1992). Glucose may also interact with transport receptors on the liver leading to the production of a neural signal, which is relayed to the brain to modulate memory.

8. Central Mechanism for Memory Enhancement

It is proposed that circulating glucose interacts with central cholinergic systems to enhance memory (Durkin 1992).

8.a. Acetylcholine and Memory

Evidence that glucose facilitates the actions of cholinergic neurons derives from the observation that memory impairment, sleep deficits and hyperactivity induced by muscarinic cholinergic antagonists can be attenuated through systemic administration of glucose. One mechanism proposed to underlie the memory enhancements of glucose is that an increase in the availability of central glucose may increase its metabolism through pyruvate to acetyl-Co A, a substrate for acetylcholine (Durkin et al 1992). It is suggested that in resting animals, an increase in the availability of glucose has little effect (Messier et al 1990), however, when there is a high demand for acetycholine (Ach) as in learning, a high availability of glucose increases the rate of acetyl CoA production thereby increasing synthesis of the transmitter. Durkin et al (1992) were the first to demonstrate that raised glucose levels facilitate acetylcholine synthesis during conditions of increased neuronal activity. More recent evidence suggests that cholinergic drugs alter the recall of the primacy part of word lists, consistent with an effect of glucose on memory through an interaction with brain cholinergic systems (Messier et al 1998). Indeed, systemic administration of glucose potentiates the release of hippocampal Ach and enhances spontaneous alternation scores (Ragozzino et al 1996), an indication that glucose may act in the hippocampus to augment Ach release and thereby improve memory. Further research found that glucose infusion into the hippocampal formation, potentiated the

release of Ach during a behavioural condition that was not observed in the resting state (Ragazzino et al 1998). The Ach potentiation was seen both ipsilateral and contralateral to the glucose infusion and was found to increase the performance of rats in a four-arm maze. This evidence supports the theory that cholinergic neurons that project to the hippocampal formation are activated during times of learning and memory, consistent with those found previously (Ragozzino 1996). Systemic administration of glucose into other neural areas also affects performance, indicating that the increase in Ach output observed with systemic glucose may be derived from the action of glucose on multiple brain sites.

Overall, there is consistent evidence showing a relationship between ingestion of sugars and improved memory. However the mechanism underlying this relationship is not clear. Peripheral blood glucose levels are associated with memory performance in some studies but this does not necessarily mean that there is a direct relationship to central regulation of memory. Perhaps there are many mechanisms that overlap to regulate memory depending on the substrate dose, structure and the timing of administration?

Research to date has documented a beneficial effect of 50g glucose on memory between 15 and 30 minutes post consumption, corresponding to the peak increase in postprandial blood glucose. Considering that different carbohydrates produce varying blood glucose responses, there may be a relationship between the structure of carbohydrate employed and the effect on memory. Indeed, as discussed earlier, recent evidence points towards a role for sucrose and fructose in memory modulation. Further research is required on the relationship between blood glucose utilisation and memory in young adults following a carbohydrate preload other than glucose.

E. Interrelationships among Food intake, Mood and Memory

There is substantial evidence supporting the interrelation among mechanisms involved in the regulation of food intake, mood and memory. Food intake can be affected by mood as a form of self-medication as in carbohydrate craving, obesity and premenstrual syndrome (Lieberman et al 1986). In addition, a depressed or lethargic state of mind may affect food intake through a lack of desire to prepare and consume food. Studies have also shown that cognition is negatively affected in the fasting state or by skipping breakfast (Benton & Parker 1998).

It is unlikely that one common mechanism explains the interaction among carbohydrate consumption and the responses to appetite, food intake, mood and memory. However, one of the primary signals proposed to underlie the effects of carbohydrates on brain function is the postprandial increase in blood glucose.

F. SUMMARY

In summary, there is substantial evidence to support a relationship between the postprandial increase in blood glucose following carbohydrate consumption and appetite, food intake, mood and memory. However the effect of carbohydrates with specific physio-chemical properties on these measures has not been considered. Thus, the purpose of this thesis is to investigate the effect of selected carbohydrates on blood glucose, appetite, food intake, mood and memory.

G. HYPOTHESIS

The effect of carbohydrates on satiety, food intake, mood and memory is associated with their glycemic response.

H. OBJECTIVES

1) To determine the effect of selected carbohydrates on postprandial blood glucose

2) To determine the effect of selected carbohydrates on satiety, food intake, mood and memory

3) To examine the relationship between the glycemic response and satiety, food intake, mood and memory

I. Outline of Experimental Studies

In total, three experiments were conducted. Experiment one was designed to examine the effect of four carbohydrate treatments, polycose, sucrose, amylose and amylopectin on blood glucose. Experiment two examined the effect of five carbohydrate treatments, polycose, sucrose, amylose, amylopectin and a sweet control, sucralose on subjective appetite, short term food intake, mood and memory. Experiment three was designed to investigate the relationship between the glycemic response to polycose, sucrose, glucose, fructose/glucose mixture and sucralose and their effects on appetite, food intake, mood and memory.

III. MATERIALS AND METHODS

A. SUBJECTS

Healthy, non-smoking males were recruited, aged between 18-35 years with a body mass index (BMI) between 20 and 25 kg/m^2 (WHO 1997). Diabetics, breakfast skippers and those dieting and taking medicine were excluded. Those who scored 11 or more on the Eating Habits Questionnaire (Appendix I) were identified as restrained eaters (Herman and Polivy 1980) and excluded from all studies. Subjects for each study were recruited through advertisements posted around the University of Toronto campus.

Individuals meeting the initial screening requirements completed baseline questionnaires, signed a consent form and were given an outline of the study (Appendix I). Subjects were required to maintain consistent eating, sleeping and exercise habits the day previous to and morning of each session.

Eight subjects were recruited for experiment one and all participants completed the study.

Power analysis for the primary endpoint, food intake, indicated that 12 participants would be an adequate sample size to investigate the effects of food intake compensation to a preload in a within subject design. Power analysis was based on previous studies investigating the dose dependent effect of sucrose on appetite and short term food intake in young males (Hui 1998; Woodend 2000). Eighteen subjects were recruited for experiment two and 15 subjects completed all sessions. In experiment three, eighteen subjects were recruited and 16 subjects completed all sessions.

Subject characteristics for experiment 1 are shown in Table 1. The average BMI for the eight participants was 23.6 kg/m^2 and average age was 24.1 yr.

Subject characteristics for experiment 2 are shown in Table 2. The average BMI for the fifteen participants was 22.8 kg/m^2 and the average age was 26.7 yr. Subject 13 was dropped from the analysis due to inconsistent completion of visual analogue scales perhaps due to language barriers.

Subject characteristics for experiment 3 are shown in Table 3. The average BMI was 22.9 kg/m^2 and the average age was 22.6 yr. Subject nine was dropped from the final analysis due to a very low food intake as a result of a dislike for the test meal.

Table 1: Subject Characteristics for Experiment I

Subject No.	Age	Weight(kg)	Height(m)	BMI[1]
1	26	59.0	1.68	20.9
2	30	78.2	1.80	24.1
3	23	74.1	1.78	23.3
4	33	81.8	1.80	25.2
5	23	71.4	1.75	23.3
6	20	77.3	1.80	23.8
7	19	83.6	1.80	25.8
8	19	68.2	1.75	22.3
Mean	24.1	74.2	1.77	23.6
SEM[2]	1.41	2.83	0.01	0.55

[1] BMI = Body Mass Index (kg/m^2)
[2] SEM = standard error of the mean; n=8

Table 2: Subject Characteristics for Experiment II

Subject No.	Age	Weight(kg)	Height(m)	BMI[1]
1	19	83.6	1.80	25.7
2	24	69.9	1.70	24.2
3	33	77.0	1.82	23.2
4	21	72.7	1.75	23.7
5	19	68.2	1.75	22.3
6	23	68.2	1.70	23.5
7	35	75.0	1.86	21.7
8	30	70.0	1.85	20.5
9	23	65.9	1.78	20.8
10	31	79.5	1.80	24.4
11	35	54.5	1.63	20.4
12	20	68.2	1.75	22.3
13	30	70.4	1.70	24.4
14	30	80.0	1.81	24.4
15	27	60.8	1.70	21.0
Mean	26.7	70.9	1.76	22.8
SEM[2]	1.48	1.94	0.02	0.43

[1] BMI = Body Mass Index (kg/m^2)
[2] SEM = standard error of the mean; n=15

Table 3: Subject Characteristics for Experiment III

Subject No.	Age	Weight(kg)	Height(m)	BMI[1]
1	20	75.0	1.78	23.7
2	35	70.0	1.75	22.8
3	28	68.2	1.70	23.6
4	20	79.5	1.78	25.1
5	26	68.5	1.78	21.6
6	21	65.9	1.70	22.8
7	21	82.5	1.82	24.9
8	25	81.8	1.83	24.5
9	21	59.1	1.73	19.7
10	23	84.0	1.93	22.6
11	19	72.0	1.93	19.3
12	20	63.6	1.75	20.7
13	22	77.3	1.85	22.5
14	18	70.4	1.75	23.0
15	19	86.4	1.85	25.2
16	24	79.5	1.83	23.8
Mean	22.6	74.0	1.80	22.9
SEM[2]	1.1	2.0	0.02	0.45

[1] BMI = Body Mass Index (kg/m^2)
[2] SEM = standard error of the mean; n=16

B. EXPERIMENTAL DESIGN

1. Experiment One: The Effect of Carbohydrate Composition on Blood Glucose

Four treatments were administered in a counterbalanced order 1) polycose, 2) sucrose, 3) amylopectin and 4) amylose (Appendix IV). All treatments were provided as 200ml beverages containing 75g carbohydrate. An additional 100ml of water was given in a separate glass to minimise after taste.

All treatments, polycose (Abott Laboratories), amylose (Amioca, National Starch and Chemical Company, Bridgewater, NJ), amylopectin (Hylon, National Starch and Chemical Company, Bridgewater, NJ), and sucrose (Redpath sugar, Tate and Lyle North American Sugars, Toronto.ON, Canada) were prepared one hour prior to the experimental session and stored in the refrigerator. Sucralose, a non-caloric sweetener was added to amylose, amylopectin and polycose to equalise sweetness with that of the sweeter sucrose treatment (Appendix V). Lemon from concentrate (Appendix V) was added immediately prior to consumption to further equalise the palatability of the treatments.

1.a. EXPERIMENTAL PROCEDURE

Subjects practiced the procedure of collecting finger-prick blood samples prior to the experimental study. Once participants felt comfortable with the procedure and understood the protocol, they chose a time between 7:00-10:00 am at which to participate in the study. Subjects arrived at the same time for each session to the Department of Nutritional Sciences. Subjects were asked to fast for 10-12 hours before a session with the exception of water, which was allowed up to one hour before the session. Once a baseline blood sample was taken, subjects were given one of four treatments and asked to

consume it within 5 minutes. Blood samples were taken every fifteen minutes after consumption of the treatment for one hour. Subjects remained seated throughout the experimental session.

1.b. SAMPLE COLLECTION

Finger-prick blood samples were obtained using a Monojector Lancet Device (Sherwood Medical, St. Lois, MO, U.S.A.). Before and after each fingerprick, the subjects' cleaned their finger with an alcohol swab (Ingram and Bell Medical, Don Mills, Ont. Canada). Immediately after a fingerprick, the first drop was wiped off and one drop was placed on a glucometer test strip of a portable blood glucose monitoring system (Fast Take™. One Touch®, LifeScan Canada Ltd, Burnaby, B.C.) for an immediate reading of glucose content. Three drops were then placed into an epindorf tube coated with potassium oxalate/ sodium fluoride anticoagulent (Vacutainer, Becton Dickinson Vacutainer Systems, Rutherford, NJ, U.S.A) and frozen. Blood glucose samples were stored less than two weeks and further analysed on an automated analyser (YSI 2300 STAT, Yellow Springs, OH, U.S.A).

1.c. DATA ANALYSIS

All statistical analyses were conducted using SAS version 7.1 (Statistical Analysis Systems, SAS Institute Inc., Carey, NC). The GLM (general linear models) procedure was used to perform ANOVA on data for which there was missing values.

One-way repeated measures analysis of variance (ANOVA) was conducted on blood glucose concentrations at each timepoint and on the incremental area under the curve (AUC) to test for the effect of treatment.

Duncans' posthoc tests were performed when treatment effects were statistically significant.

Linear regression analyses was carried out for the blood glucose values obtained from the portable glucose monitoring system versus those obtained through the automated analyser.

2. Experiment Two: The Effect of Carbohydrates on Appetite, Food Intake, Mood and Memory

Five treatments were served 1) polycose, 2) sucrose, 3) amylopectin, 4) amylose and 5) control (sucralose) (Appendix IV). Each 75g treatment was dissolved in 200ml cold spring water. An additional 200ml spring water was consumed in a separate glass, bringing the total volume to 400ml. The additional 200ml spring water was given to minimise after taste. Treatments were equalised in sweetness by the addition of sucralose, a noncaloric sweetener provided by McNeil Speciality Products Company (New Brunswick, NJ, U.S.A) (Appendix V). Sucralose was chosen as it is recognised by the body as an inert substance, having no central effect or interaction with carbohydrate metabolism or glucose or insulin secretion. A pre-experiment taste test established that subjects could not distinguish the difference in perceived sweetness among the treatments. Lemon from concentrate was added to improve palatability (Appendix V). All treatments were prepared one hour prior to consumption, stored in the refrigerator and served chilled. All treatments were consumed in 5 minutes or less.

A within subject, repeated measures design was employed and the treatments and memory tests were administered in a counterbalanced order.

2.a. EXPERIMENTAL PROCEDURE

Subjects chose a time between 7:00 - 10:00 am to arrive at the Department of Nutritional Sciences and were asked to arrive at the same time for each session. Subjects were required to fast for 10-12 hours previous to each visit except for water, which was

allowed up to one hour before the session. Upon arrival, subjects were carefully instructed on how to complete visual analogue scales and memory tests. Those participants who admitted to feeling ill, atypical fatigue or stress were asked to reschedule. Subjects then filled out baseline Visual Analogue Scale (VAS) questionnaires measuring motivation to eat, physical comfort and mood. Upon completion of the questionnaires, subjects were taken to the taste panel room to consume one of the five treatments. Subjects were asked to consume a treatment within 5 minutes. A timer was started immediately after complete consumption of the preload.

Subjects returned to the original room and completed VAS questionnaires designed to assess the sweetness and palatability of the treatment (Appendix II). VAS for mood and motivation to eat were completed at 15, 30, 45 and 60 minutes (Appendix II). Physical comfort questionnaires were completed at baseline and 60 minutes; immediately prior to the test meal (Appendix II). Each page of the questionnaire was folded out of view after each rating. Subjects remained seated throughout the study period.

Memory tests were given at 15, 45 and 60 minutes after the treatment. Subjects were distracted between 15 and 45 minutes completing trailmaking, audiovisual tasks (Appendix III) and completing a sleep / stress questionnaire to determine any unusual events, illness and compliance with the fast (Appendix I). At 30 minutes, the audiovisual task was paused to complete VAS mood and motivation to eat questionnaires.

Immediately following completion of the 60 minute physical comfort and motivation to eat questionnaires, subjects returned to the taste panel room and were served a pizza lunch and bottled spring water (1.5L,Crystal Springs, Quebec, Canada).

Upon termination of the test meal, subjects rated the palatability of the test meal and completed the post meal motivation to eat questionnaire.

2.b. DEPENDENT MEASURES

i. Food Intake

Four varieties of pizzas (McCain Deep 'N Delicious; 5" diametre: Deluxe, Pepperoni, Three Cheese and Deli Lovers) were available. Subjects ranked the pizzas according to their preference prior to the sessions. Participants were served two pizzas of their first choice and one each of their second and third choice per tray. Pizza meals were served approximately 66 minutes following consumption of the preload treatments. Subjects were specifically instructed to eat until they were "comfortably full" and that a second identical hot tray would be presented in 6 minutes. After six minutes, the second tray of four identical hot pizzas was presented to the subject and the first tray removed.

The cooked pizzas were weighed prior to serving and the amount left over after the meal was calculated as a measure of food intake. An advantage of using these pizzas was the lack of crust, which results in a pizza with a more uniform energy content and eliminated the possibility of the subject eating the more energy dense filling and leaving the outside crust of the pizza.

Each variety of pizza was weighed separately and the energy consumed (kcal) was estimated by converting the net weight consumed to kilocalories consumed using the information provided by McCain Foods Ltd. (Florenceville, New Brunswick) (Appendix V). The bottled water was also weighed prior to and following the test meal to calculate the net amount ingested during the meal.

ii. Average Appetite

Subjective appetite was measured by a VAS questionnaire measuring motivation to eat. Each VAS is a 100 mm line anchored at the beginning and end by opposing statements (Rogers & Carlyle et al 1988). Subjects marked an 'X' on the line to depict their feelings at that given moment in time. Scores were determined by measuring the distance in mm from the left starting point of the line to the intersection of the 'X'. The motivation to eat questionnaire was comprised of four questions (Appendix II):

1) How strong is your desire to eat? ('Very weak' to 'Very strong');

2) How hungry do you feel? ('Not hungry at all' to As hungry as I've ever felt')

3) How full do you feel? ('Not full at all' to 'Very full')

4) How much food do you think you could eat? ('Nothing at all' to 'A large amount')

iii. Subjective Physical Comfort

A physical comfort questionnaire was employed to assess the subjects' well being and consisted of the VAS question; How well you feel?: 'Not well at all' to 'Very well'(Appendix II).

iv. Subjective Palatability

The palatability of each treatment as well as the palatability of the test meal was assessed by the VAS question; 'How pleasant have you found the food?': 'Very pleasant' to 'Not at all pleasant'(Appendix II).

v. Perceived Sweetness

The sweetness of each treatment was assessed by the VAS question; 'How sweet have you found the drink?': 'Not at all sweet' to 'Extremely sweet' (Appendix II).

vi. Mood

Mood was measured using VAS questions designed to detect changes in mood and subjective activation (Monk 1987). The method measures Global Vigour and Global Affect. Four VAS questions assessed global vigour, which is concerned with subjective activation or vigour (alertness, sleepiness, motivation and weariness). Four VAS questions rated global affect, which is concerned more with feelings or affective state (happiness, sadness, calmness and tension) (Appendix II).

The eight VAS questions were presented to subjects in exact order:

1. How alert do you feel? ('very little' to 'very much')

2. How sad do you feel? ('very little' to 'very much')

3. How tense do you feel? ('very little' to 'very much')

4. How much of an effort is it to do anything? ('very little' to 'very much')

5. How happy do you feel? ('very little' to 'very much')

6. How weary do you feel? ('very little' to 'very much')

7. How calm do you feel? ('very little' to 'very much')

8. How sleepy do you feel? ('very little' to 'very much')

vii. Memory

The memory tests consisted of ten different sets of 20 flashcards upon which were written words specifically selected for word list presentation tests (Thorndike & Lorge 1944). The 20 flashcards were presented to the subject in one second intervals after which subjects had to write down as many of the words as they could recall. The procedure was repeated three times. The ten sets of word lists were randomly allocated to the subjects over the five treatment periods (Appendix III). Upon completion of each memory test, the

sheets containing the words were removed. The subjects were told at baseline that they would be asked to recall the word list again at 45 minutes without being shown the flashcards. Memory performance was based on the number of words correctly recalled from the 20 flashcards presented three times at 15 minutes and 60 minutes. Delayed recall was measured as the number of words correctly recalled at 45 minutes.

viii. Trail Making Test

The Trail Making Test is a test of speed for visual search, attention, mental flexibility and motor function (Reitan & Wolfson 1985). It requires the connection, by making pencil lines, between 25 encircled numbers, randomly arranged on a page in proper order (Part A) and of 25 encircled numbers and letters in alternating order (Part B). Part A was presented at every session as a measure of visuo-motor speed. Five different variations of the Part B test were randomly presented at each session to measure a treatment effect. For both forms, scoring was expressed in terms of the time required to complete Part A and Part B of the test.

ix. Audio-visual Task

The audio/visual task was a visual search and attention task employed to distract the participants' attention from rehearsal of the memory test at 15 minutes to the delayed recall at 45 minutes. It required the subject to concentrate on a series of country music videos and fill out a table pertaining to objects and words in the video. For every object (visual) and word (audio) the subjects' see/hear, they placed a tally mark within the corresponding box (Appendix III).

2.c. DATA ANALYSIS

One-way repeated measure analyses of variance (ANOVA) were performed to test the effect of treatments on the dependent variables: food intake, memory, palatability, perceived sweetness and physical comfort.

A two-way repeated measures ANOVA was used to test for the effect of treatment and time on absolute and change from baseline scores for overall mood and average appetite scores and their individual questions.

Duncan's post-hoc tests were performed where treatment effects were statistically significant. The General Linear Models (GLM) procedure was used to conduct analysis of variance on data sets when data were missing. All values are presented as a mean $\pm$ standard error of the mean (SEM). The p-value of less than 0.05 was considered to indicate statistical significance for all tests in the study.

Specific calculations applied to the dependent variables as follows:

i. Food Intake

Food intake (kcal) at the pizza test meal after all five treatments was analysed. The percent compensation for the 300kcal preload was calculated using the formula:

kcal consumed at test meal after control - kcal consumed at test meal after treatment

/ kcal in preload x 100

ii. Subjective Appetite

To assess the effect of treatments on subjective appetite, the summary measure, average appetite, was calculated for each timepoint from the four individual questions pertaining to motivation to eat, using the formula:

Average appetite = Question 1 + Question 2 + (100-Question 3) + Question 4 / 4

Question 3 is rated opposite to the other questions and was therefore subtracted from 100. The absolute values and the change from baseline were calculated for average appetite and the individual questions. A two-way ANOVA was then applied to assess the main effect of treatment and time.

iii. Subjective Physical Comfort

Ratings of well being at baseline and prior to the test meal were analysed. The higher the score, the greater the feeling of well being. The changes from baseline scores were also analysed to determine if there was a significant change in physical comfort after each preload treatment.

iv. Subjective Palatability

The palatability scores for the treatments were analysed to determine if there was a difference between the palatability of the preloads. The palatability of the pizza meal was also analysed to assess the subjects' 'liking' of the pizza meal.

v. Perceived Sweetness

The intensity of the sweetness of the treatments was assessed to determine if subjects perceived the sweetness of each of the preloads differently.

vi. Mood

The following formulas were applied to the eight VAS questions pertaining to mood to produce a summary measure for global vigour and global affect (Monk 1989):

Global vigour = [(alert) + 300- (sleepy) - (effort) - (weary)] / 4

Global affect = [(happy) + (calm) + 200 - (sad) - (tense)] / 4

The absolute values and the change from baseline scores for global vigour, global affect and the individual questions were assessed. A two-way repeated measures ANOVA was conducted to test for an effect of time and treatment.

vii. Memory

The individual (score at each trial) 15, 45 and 60 minute memory scores and total (sum of the score of the three trials) 15 and 60 minute memory scores were analysed separately for a treatment effect at each time point. Total immediate recall (total 15 and 60 minutes score) was analysed to test for a treatment effect over one hour.

The difference among all three trials at both 15 and 60 minutes was analysed to test for a change in performance between trials. Assuming that each subjects' score after the sucralose treatment represents a baseline score, the effect of treatment on performance was also assessed using the scores expressed as the difference from control.

Analysis of the 45 minute score and the difference from both control and 15 minute score was also completed to test for an effect of treatment on delayed recall. All analyses were performed by one way repeated measures ANOVA.

viii. Trail Making Test

The scores for Part A and B of the trailmaking test were analysed by one-way repeated measures ANOVA to test for an effect of treatment.

ix. Correlations

Correlation analysis was conducted to determine if there were any relationships between the following variables (Appendix VI):

1. Food intake versus sixty minute average appetite; global vigour (and all individual questions); global affect (and individual questions); total memory score at sixty minutes

2. Sixty minute Average appetite versus sixty minute global vigour (and individual questions); global affect (and individual questions); total memory score at sixty minutes

3. Sixty minute Global vigour versus global affect (and individual questions); total memory score at sixty minutes

4. Sixty minute Global vigour (and individual questions) versus individual memory scores at sixty minutes

5. Thirty and sixty minute Alert versus sixty minute memory score (third trial)

3. Experiment Three: The Relationship between the Glycemic Response to Carbohydrates and Appetite, Food Intake, Mood and Memory

The experimental design for experiment three was a within-subject design employing the procedures described in experiment one and two. Those aspects of the protocol that differed between experiment two and three are presented below.

3.a. EXPERIMENTAL DESIGN

Five treatments were served: 1) polycose, 2) sucrose, 3) fructose/glucose, 4) glucose and 5) control (sucralose). The fructose/glucose treatment contained 80% fructose and 20% glucose (Appendix IV). Glucose was added to decrease the extent of malabsorption observed upon consumption of high doses (>50g) of fructose (Rumessen & Gudmand-Hoyer 1986). Each treatment was a 75g dose dissolved in 200ml spring water. An additional 200ml spring water was consumed in a separate glass, bringing the total volume to 400ml. Sucralose and lemon concentrate was added to equalise sweetness and improve palatability (Appendix V). Sucralose was added to polycose, sucrose and glucose beverages to equate the sweetness with that of the sweeter fructose treatment. A test taste identified no discernable difference in sweetness between treatments. All treatments were prepared one hour prior to consumption, stored in the refrigerator and served chilled. All treatments were consumed in 5 minutes or less.

Each treatment and dependent measure was administered in a counterbalanced order.

3.b.EXPERIMENTAL PROCEDURE

The experimental procedure in experiment three was similar to that followed in experiment two with the exception of the frequency and timing of measurement of mood and physical comfort and the addition of blood glucose measures.

Consistent with experiment one, subjects practiced the procedure of collecting finger-prick blood samples prior to the experimental study. Blood glucose was measured at baseline and 20, 37 and 65 minutes post consumption. These times were chosen to reduce interference of blood sampling with VAS ratings and memory tests.

Mood was measured at baseline and 30 minutes post consumption. The 30 minute timepoint was deemed acceptable based on the significant treatment effect observed in experiment two. Measurement of delayed recall of the 60 minute word list was made immediately following the test meal. Physical comfort questionnaires were completed every 15 minutes to assess possible discomfort caused by the fructose/glucose treatment.

3.c.DATA ANALYSIS

Data analysis for all dependent measures was similar to experiment II.

Correlations

Correlation analyses was conducted using the Pearson Correlation coefficient. Those correlations of significance were further analysed using the Partial Pearson Correlation, controlling for subject (Appendix VI).

IV. RESULTS AND DISCUSSION

EXPERIMENT I:

THE EFFECT OF CARBOHYDRATE COMPOSITION ON BLOOD GLUCOSE

The purpose of this experiment was to examine the effect of sucrose, polycose, amylose and amylopectin preloads on postprandial blood glucose over one hour.

A. RESULTS

Treatment affected blood glucose concentrations measured over one hour [$F=25.85$; $p<0.0001$] (Table 4). A post hoc Duncans' test revealed a treatment effect 15, 30, 45 and 60 minutes following preloads (Table 4). A significant time [$F=30.93$; $p<0.0001$] and time by treatment interaction [$F=7.44$; $p<0.0001$] was found.

Polycose and sucrose produced the greatest increase in blood glucose, peaking at 30 minutes and returning to baseline by 60 minutes. Amylopectin demonstrated an intermediate increase in blood glucose, which remained significantly higher over the hour. Amylose did not significantly increase blood glucose compared to baseline (Table 5).

There was an effect of treatment [$F=19.24$; $p<0.001$], time [$F=9.59$; $p=0.0003$] and a time by treatment interaction [$F=4.32$; $p=.0002$] on blood glucose concentrations when expressed as the difference from baseline (Figure 1). A post hoc Duncans' test revealed a treatment effect 15, 30, 45 and 60 minutes following preloads [$p<0.05$] (Table 6). Polycose and sucrose demonstrated the greatest increase in incremental blood glucose, with an intermediate effect of amylopectin and no effect with amylose. The time effect was demonstrated by a pattern of increase in

blood glucose to 15 minutes which was sustained to 30 minutes and then decreased to 45 and 60 minutes after the carbohydrate treatments.

There was a treatment effect on area under the curve (AUC) for blood glucose [F=21.2 ; p<0.0001] (Table 7 ; Figure 2). A Duncan's post hoc analysis revealed that all treatments were significantly different. Amylose demonstrated the smallest AUC, followed by amylopectin and sucrose. Polycose demonstrated the greatest AUC over one hour.

Linear regression analyses confirmed a positive correlation between blood glucose values obtained from the portable glucose monitoring system versus those obtained through the automated analyser [r^2=0.96 ; p<0.02].

Table 4. Exp. I Effect of Treatment on Blood Glucose Concentrations At Each Time[1]

Time (mins)	Amylopectin	Amylose	Polycose	Sucrose	F	P
0	4.98 ± 0.1^a	5.05 ± 0.2^a	4.96 ± 0.1^a	5.20 ± 0.1^a	0.6	0.6
15	5.96 ± 0.1^a	5.38 ± 0.2^a	8.15 ± 0.4^b	7.81 ± 0.1^b	39.2	<0.0001
30	6.58 ± 0.2^a	5.31 ± 0.2^b	8.54 ± 0.6^c	8.36 ± 0.6^c	16.0	<0.0001
45	6.64 ± 0.3^a	5.40 ± 0.2^b	7.89 ± 0.7^c	6.91 ± 0.5^{ac}	8.5	<0.0007
60	6.25 ± 0.3^a	5.15 ± 0.2^b	6.43 ± 0.3^a	5.86 ± 0.3^{ab}	5.1	<0.009

[1] Mean ± SEM (mmol/L); n=8.
[a] Means with different superscripts within a row are significantly different; $p<0.05$

Table 5. Exp. I. Effect of Time on Blood Glucose Concentrations For Each Treatment[1]

Time (mins)	Amylopectin	Amylose	Polycose	Sucrose
0	4.98 ± 0.1^a	5.05 ± 0.2^a	4.96 ± 0.1^a	5.20 ± 0.1^a
15	5.96 ± 0.1^b	5.38 ± 0.2^a	8.15 ± 0.4^{bc}	7.81 ± 0.1^b
30	6.58 ± 0.2^b	5.31 ± 0.2^a	8.54 ± 0.6^b	8.36 ± 0.6^b
45	6.64 ± 0.3^b	5.40 ± 0.2^a	7.89 ± 0.7^{bc}	6.91 ± 0.5^{bc}
60	6.25 ± 0.3^b	5.15 ± 0.2^a	6.43 ± 0.3^{ac}	5.86 ± 0.3^{ac}
F	8.579	0.61	9.91	12.42
P	<0.001	0.66	<0.001	<0.001

[1] Mean ± SEM (mmol/L); n=8.
[a] Means with different superscripts within a column are significantly different; $p<0.05$

Table 6. Exp. I. Effect of Treatment on Incremental Blood Glucose Concentrations at each Time[1]

Time (mins)	Amylopectin	Amylose	Polycose	Sucrose	F	P
15	1.11 ± 0.5^a	0.39 ± 0.3^b	3.19 ± 0.9^c	2.6 ± 0.4^d	42.4	<0.0001
30	1.73 ± 0.6^a	0.26 ± 0.4^b	3.58 ± 1.7^c	3.16 ± 1.6^c	13.7	<0.0001
45	1.79 ± 0.9^{ab}	0.35 ± 0.5^c	2.93 ± 1.8^b	1.70 ± 1.4^a	7.3	0.002
60	1.28 ± 0.8^a	0.10 ± 0.4^b	1.48 ± 0.9^a	0.93 ± 1.1^a	4.7	0.011

[1] Mean ± SEM (mmol/L); n=8.
[a] Means with different superscripts within a row are significantly different, p<0.05.

Table 7. Exp. I. Blood Glucose Area Under the Curve after Treatments [1]

Treatment	AUC (mmol/min/L)
Amylose	18.19 ± 18.4^a
Amylopectin	73.35 ± 10.0^b
Sucrose	117.7 ± 19.1^c
Polycose	156.6 ± 18.4^d
F	21.2
P	<0.0001

[1] Mean ± SEM (mmol/L); n=8.
[*] Means with different superscripts within a row are significantly different.
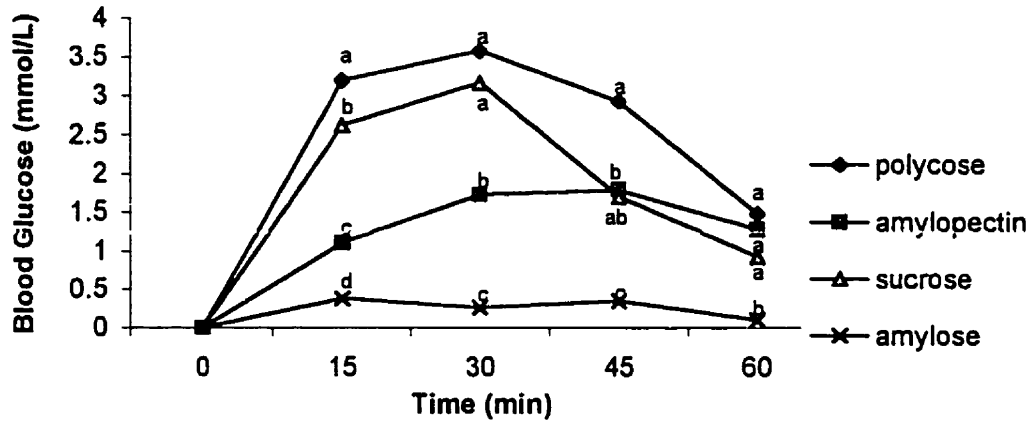
Figure 1. Incremental blood glucose concentrations after four treatments. Values with different letters, within the same timepoint are significantly different (p<0.05; n=8).
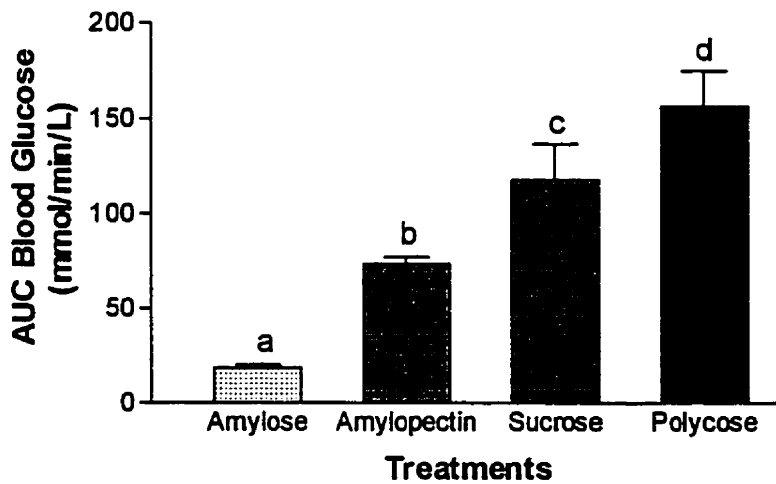
Figure 2. Area Under the Curve Blood Glucose Concentration after four treatments. Treatments with different letters are significantly different (p<.0001 ; n=8).

B. DISCUSSION

This experiment demonstrated that pure carbohydrate preloads of different physio-chemical properties elicited very different responses in blood glucose.

The glycemic response to the carbohydrate treatments was proposed from their underlying chemical structure (Appendix IV). The results confirmed that the carbohydrates produced the desired glycemic responses over one hour.

Based upon the branched structure of amylopectin, which renders a greater surface area for digestive enzymes, a higher glycemic response was predicted compared to the linear form of amylose. In the present study, the increase in blood glucose was negligible upon amylose consumption and slow with amylopectin consumption. These results are similar to those observed in a study that examined the glycemic response to high amylose and high amylopectin starch crackers (Behall et al 1988).

Polycose demonstrated a rapid and high increase in blood glucose that was expected based upon its composition of rapidly digestible short-chain glucose polymers. Sucrose elicited a lower glycemic response compared to polycose that corresponds with previous studies which have found sucrose to have a lower glycemic index than glucose (Foster-Powell & Brand Miller 1995). Both polycose and sucrose demonstrated a peak increase in blood glucose 30 minutes post consumption, which declined to baseline within 60 minutes. Thus, the glycemic response to polycose and sucrose is completed in 60 minutes whereas measurement over two hours may give a clearer picture of the glycemic response to amylose and amylopectin.

Because a range of glycemic responses were produced by these carbohydrate treatments, it was concluded that the treatments were appropriate for testing the

hypothesis that the effect of carbohydrates on appetite, food intake, mood and memory is defined by their glycemic response.

EXPERIMENT II:

THE EFFECT OF CARBOHYDRATE TREATMENTS ON SUBJECTIVE APPETITE, SHORT TERM FOOD INTAKE, MOOD AND MEMORY

The results and discussion have been divided by dependent measures into three sections based on the effect of treatment on; satiety and food intake, mood and memory.

PART I:

THE EFFECT OF CARBOHYDRATE TREATMENT ON SUBJECTIVE APPETITE AND FOOD INTAKE

A. RESULTS

1. FOOD AND WATER INTAKE

One hour after preloads there was an effect of treatment on mealtime energy intake ($F=4.14$, $p=0.006$) (Table 8). Sucrose, and polycose significantly decreased food intake compared to the control, sucralose and amylopectin. Amylose was not significantly different from all other treatments (Table 8).

There was a treatment effect on percent compensation for the calories consumed in the 300kcal preload [$F=3.56$, $p<0.03$] (Table 8). Compensation for a 300kcal preload of polycose and sucrose was significantly higher than for amylopectin.

The amount of water consumed with the test meal was not affected by treatment [$F= 1.59$; $p= 0.19$] (Table 8).

Table 8. Exp. II Food and Water Intake after Treatments [1]

Treatment	Test Meal [2] (kcals)	% Compensation [3]	Water Intake (grams)
Sucralose	1017 ± 69.9 [a]		347.6 ± 51.6
Amylose	946 ± 56.8 [ab]	23.7 ± 15.6 [ab]	338.1 ± 49.8
Amylopectin	1018 ± 92.4 [a]	-0.2 ± 21.7 [b]	327.4 ± 33.5
Polycose	823 ± 87.1 [b]	64.9 ± 19.4 [a]	302.9 ± 39.5
Sucrose	884 ± 83.9 [b]	44.4 ± 13.1 [a]	387.0 ± 53.8
F	4.14	3.56	1.59
P	0.006	0.03	0.19

[1] Mean ± SEM, n=14

[2] Energy Consumed (kcal) in a test meal 60 minutes following preload

[3] Calories Consumed after Control - Calories Consumed Treatment/ Calories in Preload x 100

[a] Means with different superscripts, within a column, are significantly different

2. AVERAGE APPETITE

No treatment effect was observed upon analysis of the absolute values [F=0.82 ; p=0.52]. Average appetite increased with time [F=6.15 ; p=0.0004] and a time by treatment interaction occurred because the time effect was stronger after amylose and amylopectin [F=2.58 ; p=0.001] (Table 9).

When the data were analysed as change from baseline, an effect of treatment [F=2.56 ; p=0.049], time [8.78 ; p=0.0001] and a time by treatment interaction [F = 1.85 ; p=0.045] was observed (Table 10 ; Figure 3). A post hoc Duncans' test revealed a treatment effect 30 minutes post preload (Figure 4). At this time, polycose and amylopectin reduced average appetite more than sucralose or amylose treatment [F=3.74 ; p=0.009]. The response to sucrose was not different from any other of the treatments.

2.a. Individual Average Appetite Questions:

No treatment effect was observed on the absolute values for hunger [F=0.65 ; p=0.63]. Hunger was significantly affected by time [F=5.37 ; p=0.001] but a time by treatment interaction [F=2.52 ; p=0.0015] was observed due to the decreased hunger after polycose, amylose, amylopectin compared to sucrose and sucralose (Table 9).

When hunger scores were based on change from baseline, there was again no effect of treatment [F=2.05 ; p=0.1]. The increase in hunger with time [F= 7.87 ; p=0.0003] and the time by treatment interaction [F = 2.99 ; p=0.0009] was due to the decreased hunger following polycose and amylopectin treatments (Table 10).

No treatment effect was observed upon analysis of the absolute values for fullness [F=0.68 ; p=0.61]. Scores for fullness increased immediately following all preloads then

decreased over the remaining hour resulting in a significant effect of time [F=5.35 ; p=0.0011] but no time by treatment interaction [F=1.61 ; p=0.068] (Table 9).

When the data expressed as change from baseline was analysed, there was a near treatment effect [F=2.35 ; p=0.066]. At 30 and 45minutes, post preload, amylopectin and polycose were associated with increased ratings of fullness. A significant effect of time [F= 6.19 ; p=0.0015], but no time by treatment interaction was observed [F = 1.30 ; p=0.22] (Table 10).

No treatment effect was observed upon analysis of the absolute values for desire to eat [F=0.82 ; p=0.52]. A significant effect of time [F=5.63 ; p=0.0008] and a time by treatment interaction was observed [F=2.16 ; p=0.007]. Desire to eat decreased immediately following the amylose, amyloepctin and polycose treatments and then increased with time (Table 9).

When the data expressed as change from baseline was analysed again, no treatment effect was found [F=1.36 ; p=0.26]. Ratings for desire to eat increased over time [F=9.55 ; p<0.0001] but a time by treatment interaction [F=3.32 ; p=0.0003] because the increase with time was less after polycose and sucrose than after amylose and amylopectin treatments (Table 10).

No treatment effect was observed upon analysis of the absolute values for amount [F=0.54 ; p=0.71]. Ratings of amount increased over time [F=3.85 ; p=0.008] but no time by treatment interaction [F=1.57 ; p=0.079] (Table 9).

When the scores were based on change from baseline, there was no effect of treatment on amount [F=1.59 ; p=0.19]. Scores for amount increased over time [F= 6.76 ; p=0.0009]. No time by treatment interaction was observed [F = 1.55 ; p=0.11] (Table 10).

Table 9. Exp II Absolute Average Appetite Scores [1,2]

Question	Time (min)	Polycose	Sucrose	Amylo-pectin	Amylose	Sucralose	F ; p
Average Appetite	0	71.5 ± 3.0	70.5 ± 4.5	72.0 ± 2.8	62±4 5.8	63.6 ±4.4	1.54; 0.2
	15	59.1 ± 6.7	64.8 ± 5.3	56.0± 5.6	48.3 ±5.3	60.1± 4.9	2.76; 0.04
	30	56.6 ± 7.6	62.8 ± 5.1	58.9 ± 5.5	57.7 ± 5.6	63.6± 5.0	0.79; 0.54
	45	60.5 ± 6.8	66.0 ± 4.5	64.7 ± 4.6	60.1 ± 6.0	66.7± 5.1	0.55; 0.7
	60	62.6 ± 6.2	68.4 ± 4.6	70.1 ±3.3	64.3 ± 5.0	67.4 ±5.0	0.63; 0.64
1. Hunger	0	69 ± 3.1	67.4 ± 5.8	66.1 ± 3.4	60.3 ± 5.5	57.2 ± 6.0	1.4; 0.25
	15	54.9 ± 7.3	62.2 ± 6.3	51.4± 6.5	42.4 ± 5.8	59.3 ±4.7	3.37; 0.02
	30	55.2 ± 7.5	59.5 ± 6.4	53.9 ± 6.4	55.2 ± 6.4	59.8 ± 5.3	0.5; 0.73
	45	57.3 ± 7.1	60.5 ±5.6	60.9 ± 4.7	56.9 ±5.9	61.6± 6.0	0.24; 0.91
	60	59.1± 7.2	65.6 ± 5.8	67.3 ±3.6	60.6 ± 5.6	62.6± 5.6	0.6; 0.66
2. Full [3]	0	80.9 ± 2.8	78.6 ± 5.3	83.4 ± 3.2	71.9 ± 6.8	70.9 ± 5.8	1.48; 0.22
	15	69.2 ± 7.1	72.1 ± 5.8	65.5± 7.5	53.5 ± 8.0	64.9 ± 7.5	1.7; 0.16
	30	61.3 ± 8.8	68.2 ± 5.6	65.9 ±6.8	65.9 ± 7.0	72.9 ±5.8	0.7; 0.59
	45	68.8 ± 6.3	77 ± 4.0	71.9 ± 5.9	69.4 ± 6.7	78.9 ± 4.2	1.10; 0.37
	60	74.5 ±5.4	75.9 ±4.3	74.5 ± 4.9	75.6 ± 5.4	78.2 ± 4.7	0.19; 0.94
3. Desire	0	68.4 ± 4.9	69.6 ±6.4	72.9 ±4.8	58.9 ±6.8	61.1± 6.6	1.17; 0.33
	15	57.4 ± 6.6	64.1 ± 6.8	50.1± 6.6	48.1± 6.2	57.7 ±5.5	2.03; 0.1
	30	54.9 ± 7.3	63.1± 6.2	55.1 ±5.8	55.4 ±6.8	58.0 ±6.0	0.69; 0.6
	45	57.5 ± 7.1	65.1 ±5.8	60.9 ±5.0	56.5 ±7.2	60.8± 6.5	0.48; 0.75
	60	55.3 ± 7.9	68.2 ±5.8	70.6 ± 3.0	60.9 ± 6.1	63.6± 6.2	1.49; 0.22

[1] Mean ± SEM; n=14

[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100

[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100

Table 9. Continued

Question	Time (min)	Polycose	Sucrose	Amylo-pectin	Amylose	Sucralose	F ; p
4.Amount	0	67.6 ± 3.8	66.4 ± 5.0	66.2 ± 3.2	57.8 ± 7.2	65.1 ± 4.6	0.98; 0.42
	15	54.6 ± 7.3	60.6 ± 5.5	55.5 ± 4.5	51.5 ± 7.0	61.7 ± 5.1	1.17; 0.33
	30	55.1 ± 7.3	60.1 ± 5.8	58.1 ± 5.7	56.9 ± 6.1	63.9 ± 4.6	0.75; 0.56
	45	58.3 ± 7.4	61.1 ± 6.3	63.3 ± 3.8	59.2 ± 6.4	65.4 ± 5.2	0.43; 0.79
	60	61.7 ± 6.5	63.9 ± 6.0	67.1 ± 3.9	61.1 ± 5.5	65.4 ± 5.1	0.37; 0.83

[1] Mean ± SEM; n=14
[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100

Table 10. Exp II Change from Baseline Average Appetite Scores [1,2].

Question	Time (min)	Polycose	Sucrose	Amylo-pectin	Amylose	Sucralose	F ; p
Average Appetite	15	-12.4 ± 5.0	-5.75 ± 2.6	-16.2 ± 6.9	-13.3 ± 4.7	-2.7 ± 4.3	1.93; 0.12
	30	-14.9 ± 5.8[b]	-7.8 ± 2.9[ab]	-13.9 ± 6.0[b]	-3.8 ± 4.3[a]	0.07 ± 4.3[a]	3.74; 0.009
	45	-11 ± 4.9	-4.6 ± 1.8	-7.9 ± 5.3	-1.7 ± 5.0	3.1 ± 4.0	2.14; 0.09
	60	-8.8 ± 4.3	-2.1 ± 1.7	-2.3 ± 4.1	2.3 ± 5.6	3.9 ± 4.4	2.08; 0.1
1. Hunger	15	-14.1± 6.2	-5.2 ± 3.1	-14.7 ± 6.4	-17.9 ± 5.5	2.1 ± 5.6	2.81; 0.04
	30	-13.8 ± 6.5	-7.9 ± 4.0	-12.3 ± 6.5	-5.1 ± 4.7	2.6 ± 5.4	2.37; 0.06
	45	-11.7± 5.6	-6.9 ± 2.5	-5.3 ± 5.2	-3.4 ± 5.2	4.4 ± 5.4	1.71; 0.16
	60	-9.9 ± 5.6	-1.8 ± 2.5	1.14 ± 4.5	0.4 ± 5.1	5.4 ± 5.3	1.82; 0.14
2. Full [3]	15	-11.7 ± 6.2	-6.5 ± 3.6	-18.9 ± 7.0	-18.4 ± 7.3	-6.1 ± 9.5	1.07; 0.38
	30	-19.6 ± 7.4	-10.4 ± 4.9	-17.6 ± 6.2	-6.1 ± 5.0	2.0 ± 7.5	2.8; 0.04
	45	-12.1± 5.8	-1.6 ± 3.1	-11.5 ± 6.8	-2.5 ± 5.3	7.9 ± 6.6	2.64; 0.12
	60	-6.4 ± 4.2	-2.7 ± 3.7	-8.9 ± 4.7	3.7 ± 6.9	7.3 ± 7.2	1.86; 0.14
3. Desire	15	-10.9 ± 5.0	-5.5 ± 3.3	-22.9 ± 1.0	-10.8 ± 6.7	-3.4 ± 5.0	1.76; 0.15
	30	-13.5± 5.8	-6.5 ± 2.6	-17.8 ± 8.9	-3.4 ± 5.7	-3.1 ± 4.5	1.89; 0.13
	45	-10.9 ± 4.7	-4.6 ± 1.9	-12.0 ± 8.1	-2.4 ± 6.9	-0.2 ± 4.5	1.01; 0.41
	60	-13.1± 6.0	-1.4 ± 1.6	-2.4 ± 5.6	2.0 ± 6.7	2.6 ± 5.2	1.78; 0.15
4. Amount	15	-12.9 ± 5.3	-5.7 ± 3.2	-10.7± 4.1	-6.3 ± 4.8	-3.4 ± 3.0	1.75; 0.15
	30	-12.5 ± 5.5	-6.3 ± 3.7	-8.1± 5.0	-0.9 ± 4.9	-1.2 ± 3.4	2.16; 0.09
	45	-9.2 ± 5.6	-5.4 ± 3.3	-2.9 ± 3.5	1.4 ± 5.0	0.35 ± 3.2	1.64; 0.18
	60	-5.9 ± 4.5	-2.6 ± 3.6	0.9 ± 3.6	3.4 ± 6.4	0.35 ± 3.3	0.94; 0.45

[1] Mean ± SEM; n=14
[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100
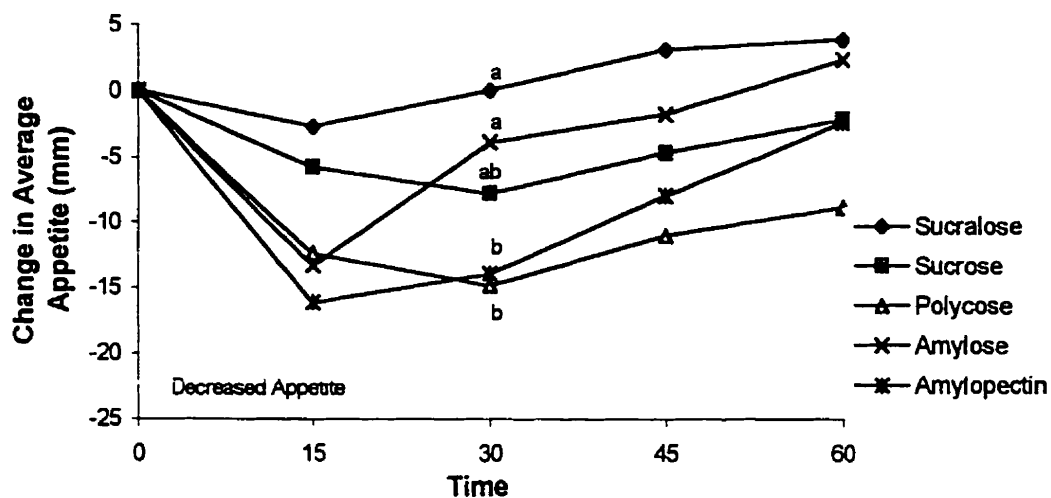
Figure 3. Average Appetite Change from Baseline scores after five treatments. Values within the same timepoint with different letters are significantly different (p<0.05; n=14).
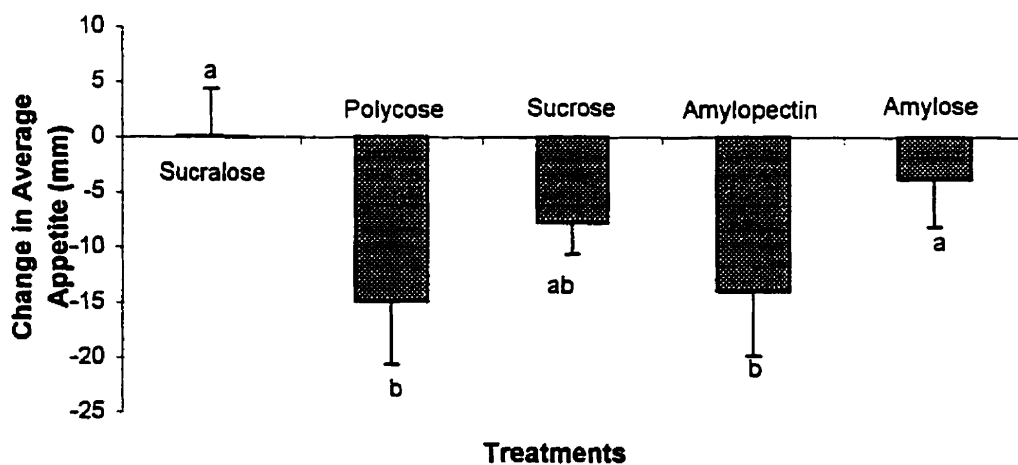
Figure 4. Average Appetite Change from Baseline scores 30 minutes after five treatments. Treatments with different letters are significantly different (p<0.05; n=14).

3. PHYSICAL COMFORT

There was no difference between treatments on the ratings of well being at

baseline [F=0.11 ; p=0.98] and 60 minutes [F=0.85 ; p=0.49]. Similarly, no effect of

treatment was observed when data describing the difference between scores of well being

at 60 minutes and baseline [F= 0.61; p= 0.66] were analysed (Table 11).

Table 11. Experiment II Physical Comfort Scores [1,2]

Treatment	Time 0 min	Time 60 min	Change[3] (60 - 0)
Sweetener	72.7 ± 3.7	71.3 ± 4.3	1.35 ± 3.7
Sucrose	73.9 ± 4.6	75.0 ± 4.0	-1.14 ± 3.2
Polycose	73.8 ± 4.3	76.7 ± 2.2	-2.86 ± 3.5
Amylose	74.9 ± 3.4	71.86 ± 4.3	3.07 ± 3.9
Amylopectin	72.4 ± 4.1	72.7 ± 3.8	-0.36 ± 3.6
F	0.11	0.85	0.61
P	0.98	0.49	0.66

[1] Mean ± SEM (mm); n = 14
[2] Question: How well do you feel? Scoring: Not well at all=0; Very well=100
[3] Change in physical comfort score from baseline to 60 minutes

4. PALATABILITY

There was no significant differences in subjective ratings of palatability for the test meal [F=0.69 ; p=0.6] (Table 12).

However, the rated palatability of the preloads was different among the treatments [F= 13.2 ; p=0.001]. Amylose and amylopectin were rated as less palatable than sucrose, sucralose and polycose (Table 12).

Table 12. Experiment II Palatability Ratings of Beverage and Pizza after each Treatment[1,2]

Treatment	Beverage	Pizza
Sweetener	60.9 ± 8.3^{a}	80.7 ± 3.5
Sucrose	57.8 ± 8.6^{a}	78.4 ± 4.0
Polycose	54.9 ± 7.9^{a}	78.4 ± 2.8
Amylose	27.5 ± 7.9^{b}	80.9 ± 3.7
Amylopectin	20.2 ± 6.2^{b}	82.0 ± 2.8
F	13.20	0.69
P	0.001	0.60

[1] Mean ± SEM (mm); n = 14
[2] Question: How pleasant have you found the beverage or food?
 Scoring: Very Pleasant = 0; Not pleasant at all = 100
[a] Means with different superscripts within a column are significantly different; $p < 0.05$

5. PERCEIVED SWEETNESS

The subjective ratings of perceived sweetness also varied among treatments (Table 13). Sucrose was rated as sweeter than amylose and amylopectin [F=5.44 ; p=0.001]. Amylopectin was perceived to be less sweet than control and polycose. There was no significant difference in sweetness among amylose, polycose and control.

Table 13. Experiment II   Sweetness Ratings of Preloads[1,2]

Treatment	Perceived Sweetness
Sucralose	79.9 ± 3.3[ab]
Sucrose	87.5 ± 3.2[a]
Polycose	80.3 ± 3.3[ab]
Amylose	72.4 ± 5.3[bc]
Amylopectin	60.6 ± 5.9[c]
F	5.44
P	0.001

[1] Mean ± SEM (mm); n = 14
[2] Question: How sweet have you found the beverage?
    Scoring: Not sweet at all = 0;   Extremely sweet = 100
[a] Means with different superscripts are significantly different

6. CORRELATIONS

No relationships were observed among the perceived sweetness and palatability of the preloads and both food intake and 30 minute average appetite. Similarly, no relationship was observed between 60 minute physical comfort ratings and food intake. A positive relationship was observed between subjective ratings of appetite and subsequent food intake [r=0.39 ; p<0.001] (Appendix VI; Table i).

B. DISCUSSION

Polycose and sucrose significantly decreased food intake compared to control and amylopectin, whereas neither amylose nor amylopectin decreased food intake from control values. Average appetite was suppressed to the greatest extent 30 minutes following polycose and amylopectin treatments.

The results support the validity of the VAS questionnaire as a tool for tracking the effect of treatment with time on appetite (Rolls et al 1988; Stewart et al 1997; Brown 1998). Average appetite immediately prior to the test meal was strongly associated with energy intake at the meal (Appendix VI; Table ii).

The discrepancy between the observed decrease in appetite observed 30 minutes after the amylopectin treatment and the failure to suppress energy intake may lie with the decreased palatability of the amylopectin treatment. However, it is unlikely that the difference in perceived sweetness or palatability of the treatments was associated with the treatment effect on subjective ratings of appetite or food intake as no direct relationship was found among the dependent measures (Appendix VI; Table i). Indeed, it has been shown that the 'liking' of a preload does not influence energy intake one hour later (Graff 1999).

It was the high glycemic sources that produced the greatest decrease in food intake, suggesting that a rapid increase in blood glucose may be important in regulating energy intake one hour post preload. The lack of effect of amylose and amylopectin on energy intake supports the hypothesis that low glycemic carbohydrates result in greater short term energy intake. However, the effect of slowly digestible starches such as amylose and amylopectin over a longer period of time may offer a different perspective.

A study employing a within subject design measuring the glycemic response to carbohydrates and their effects on energy intake is required to directly address the relationship between blood glucose and energy intake. Therefore, experiment III was designed to investigate the relationship between the glycemic response to sucrose, polycose, glucose and a high fructose preload and their effects on satiety and energy intake over one hour.

PART II:

THE EFFECT OF CARBOHYDRATE TREATMENTS ON MOOD

A. RESULTS:

1. Global Vigour and Global Affect

No treatment effect was observed on the mean absolute values of global vigour [F=1.14 ; p=0.35]. Overall global vigour decreased with time [F=3.36 ; p=0.016], but no time by treatment interaction was observed [F=0.86 ; p=0.61] (Table 14).

When expressed as the difference from baseline, there was again no effect of treatment [F=1.15 ; p=0.34]. Global vigour decreased with time [F=3.05 ; p=0.039]. No time by treatment interaction was found [F=0.58 ; p=0.86] (Table 15).

There was no treatment effect on absolute scores for global affect over one hour [F= 1.46 ; p=0.23]. A strong effect of time was observed [F=24.26 ; p<0.0001] with no time by treatment interaction [F=1.42 ; p=0.13]. Duncans' post hoc test revealed a strong increase in global affect at 60 minutes; immediately prior to the test meal (Table 14).

When the scores for global affect were expressed as the difference from baseline, there was no treatment effect [F=1.14 ; p=0.34] Ratings of global affect increased with

time [F=26.44 ; p<0.0001] with no time by treatment interaction [F=1.65 ; p=0.08] (Table 15).

2. Individual Mood Questions

There was no effect of treatment on ratings of sleepy, weary, effort, calm, happy, sadness or tense. However, there was a treatment effect on ratings of alertness and sadness when expressed as the change from baseline (Table 15).

2.a. Alert

No treatment effect was found for the absolute values for alertness [F=0.68 ; p=0.61]. A time effect was observed [F=2.86 ; p=0.03] but no time by treatment interaction was found [F=1.38 ; p=0.15] (Table 14).

However, when the change from baseline data were analysed, a treatment effect was observed [F=2.57, p=0.048]. A near effect of time [F=2.79 ; p=0.053] but no time by treatment interaction [F=0.84 ; p=0.61] was observed (Table 15 ; Figure 5). Post hoc Duncans' test revealed a significant treatment effect at 30 minutes. Amylose, amylopectin and sucrose increased ratings of alert compared to control at 30 minutes post preload [F=3.61 ; p=0.01] (Figure 6). Amylose increased ratings of alertness compared to polycose at 30 minutes.

2.b. Sad

When the absolute values for sadness were analysed, no treatment effect was found [F=2.12 ; p=0.09]. Ratings of sadness increased over time [F=5.32 ; p=0.001] but no time by treatment interaction was found [F=1.88 ; p=0.02] (Table 14).

A near treatment effect was observed when the change from baseline scores were analysed [F=2.27 ; p=0.07] (Table 15). Amylopectin increased ratings of sadness at 45 minutes [F=2.35 ; p=0.06] compared to amylose and polycose. At 60 minutes, there was a trend for amylopectin and sucrose to increase sadness compared to amylose [F=2.46 ; p=0.057]. Ratings of sadness increased with time [F=4.75 ; p=0.006] but no time by treatment interaction was found [F=1.63 ; p=0.087].

Table 14. Exp II Absolute Mood scores after treatments[1,2]

Question	Time (min)	Sucralose	Sucrose	Polycose	Amylose	Amylo-pectin	F;p
Global Vigour	0	64.4 ± 5.0	59.1 ± 5.8	59.1 ± 4.2	53.7 ± 5.1	57.0 ± 5.5	1.13; 0.35
	15	60.8 ± 4.3	56.8 ± 6.0	52.9 ± 6.3	53.4 ± 5.5	55.5 ± 4.8	0.92; 0.46
	30	52.5 ± 2.9	49.4 ± 4.1	49.1 ± 3.9	50.4 ± 3.5	45.5 ± 3.1	1.38; 0.25
	45	56.9 ± 4.9	53.8 ± 7.2	47.3 ± 6.0	52.1 ± 5.4	48.2 ± 6.3	1.26; 0.30
	60	57.9 ± 5.1	54.2 ± 7.2	52.2 ± 5.9	55.2 ± 5.8	50.9 ± 6.2	0.68; 0.61
1. Alert	0	63.9 ± 5.3	63.4 ± 6.3	57.8 ± 5.4	50.9 ± 6.3	54.6 ± 5.6	1.7; 0.6
	15	56.1 ± 4.5	53.6 ± 6.1	46.7 ± 7.6	50.1 ± 6.3	53.9 ± 5.5	0.75; 0.56
	30	47.0 ± 6.2	52.2 ± 7.1	44.4 ± 6.7	50.6 ± 7.0	52.1 ± 4.9	0.74; 0.57
	45	45.9 ± 6.7	52.0 ± 7.3	40.4 ± 6.8	50.1 ± 6.2	42.6 ± 6.9	1.44; 0.23
	60	48.0 ± 6.4	49.0 ± 8.2	44.6 ± 7.3	51.4 ± 6.1	46.9 ± 6.9	0.3; 0.88
2. Sleepy	0	44.4 ± 7.5	49.5 ± 8.4	45.6 ± 6.9	60.6 ± 6.8	60.1 ± 6.9	1.81; 0.14
	15	49.7 ± 6.2	52.5 ± 8.2	54.6 ± 8.5	55.9 ± 7.3	57.9 ± 6.8	0.36; 0.84
	30	47.9 ± 6.8	51.6 ± 8.2	50.1 ± 7.4	48.4 ± 7.9	60.5 ± 6.6	1.24; 0.3
	45	44.2 ± 7.0	51.6 ± 9.1	63.4 ± 7.5	60.9 ± 6.1	65.3 ± 7.4	3.67; 0.01
	60	49.3 ± 6.6	52.9 ± 8.8	57.3 ± 8.2	53.6 ± 7.1	64.5 ± 7.4	1.7; 0.16
3. Weary	0	33.1 ± 5.4	44.5 ± 6.6	44.6 ± 7.3	42.5 ± 6.8	42.1 ± 6.5	1.05; 0.39
	15	35.4 ± 4.9	38.2 ± 6.5	42.9 ± 8.0	42.8 ± 6.7	46.4 ± 6.6	0.82; 0.52
	30	37.9 ± 6.0	43.3 ± 6.8	33.3 ± 6.0	35.0 ± 4.5	41.6 ± 4.4	1.03; 0.4
	45	43.2 ± 7.1	44.8 ± 7.4	44.6 ± 7.0	43.4 ± 6.3	43.7 ± 6.1	0.12; 0.97
	60	40.7 ± 6.4	44.4 ± 6.9	43.7 ± 6.7	38.1 ± 5.0	41.3 ± 6.7	0.37; 0.83
4. Effort[3]	0	28.8 ± 5.2	33.1 ± 6.1	31.1 ± 5.3	32.1 ± 4.8	25.3 ± 5.9	0.63; 0.64
	15	27.9 ± 4.9	35.6 ± 6.6	37.7 ± 6.8	36.6 ± 6.1	28.9 ± 5.9	1.42; 0.24
	30	23.6 ± 5.1	31.6 ± 6.8	39.4 ± 7.3	34.8 ± 6.5	35.1 ± 6.0	1.87; 0.13
	45	31.1 ± 5.5	39.2 ± 8.1	43.2 ± 7.2	36.0 ± 6.9	39.9 ± 7.3	1.36; 0.26
	60	26.2 ± 5.4	38.3 ± 8.7	34.9 ± 7.5	41.3 ± 6.9	35.1 ± 8.5	1.37; 0.26

Table 14. Continued

Question	Time (min)	Sucralose	Sucrose	Polycose	Amylose	Amylo-pectin	F ; p
Global Affect	0	71.2 ± 3.8	71.8 ± 4.6	72.8 ± 3.6	74.7 ± 3.6	75.8 ± 3.2	1.13; 0.35
	15	74.1 ± 3.6	72.9 ±3.9	76.9 ± 3.1	76.5 ± 3.0	75.7 ± 4.9	0.92; 0.46
	30	72.0 ± 4.5	73.5 ± 3.8	79.3 ± 2.5	74.9 ± 3.7	76.8 ± 4.1	1.38; 0.25
	45	72.4 ± 4.2	73.3 ± 3.9	78.9 ± 2.8	76.0 ± 3.1	70.1 ± 4.5	1.26; 0.29
	60	83.9 ± 1.8	84.4 ± 2.0	86.1 ± 1.4	85.5 ± 1.7	86.2 ± 1.6	0.68; 0.61
5. Sad	0	14.1 ± 3.3	19.4 ± 4.7	18.0 ± 3.8	23.6 ± 5.2	14.1 ± 3.4	3.00; 0.03
	15	15.6 ± 2.9	23.8 ± 5.4	21.9 ± 4.6	20.3 ± 4.1	18.7 ± 4.6	1.90; 0.12
	30	19.4 ± 4.5	24.0 ± 4.8	19.3 ± 4.3	19.6 ± 3.7	19.4 ± 3.9	0.61; 0.65
	45	18.5 ± 4.3	29.5 ± 6.1	19.3 ± 3.9	24.7 ± 5.7	27.6 ± 6.2	2.63; 0.04
	60	20.9 ± 5.8	28.7 ± 6.9	18.8 ± 3.7	21.0 ± 4.6	23.6 ± 5.4	1.98; 0.13
6. Calm	0	63.1 ± 5.9	67.4 ± 4.9	70.4 ± 4.6	76.0 ± 3.5	75.6 ± 3.5	3.89; 0.01
	15	69.9 ± 5.3	71.9 ± 4.8	79.3 ± 3.2	78.6 ± 2.7	74.6 ± 5.4	1.83; 0.14
	30	67.6 ± 6.6	73.2 ± 5.6	80.2 ± 3.1	76.4 ± 4.1	74.2 ± 4.6	2.11; 0.09
	45	67.4 ± 6.0	79.2 ± 3.5	80.1 ± 3.2	79.7 ± 3.4	71.4 ± 6.1	2.36; 0.07
	60	73.7 ± 3.9	71.7 ± 5.6	80.7 ± 3.2	78.0 ± 4.9	70.4 ± 4.6	1.24; 0.31
7. Happy	0	65.8 ± 5.3	65.9 ± 4.5	63.6 ± 4.9	68.0 ± 4.9	63.0 ± 4.2	0.46; 0.77
	15	66.1 ± 4.9	67.1 ± 3.8	70.5 ± 4.4	69.8 ± 4.7	66.6 ± 6.1	0.53; 0.71
	30	63.5 ± 6.3	66.6 ± 4.3	70.3 ± 4.1	63.0 ± 6.8	70.4 ± 5.4	1.29; 0.28
	45	66.2 ± 5.8	65.1 ± 5.3	69.7 ± 4.1	65.6 ± 5.1	62.3 ± 6.4	1.07; 0.38
	60	66.0 ± 6.1	64.2 ± 5.7	70.7 ± 4.0	66.9 ± 5.0	62.9 ± 6.4	0.93; 0.45

[1]Mean ● SEM; n=14
[2]Question: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3]Question 4 : How much effort is it to do anything? Scoring: Very little = 0; Very much = 100

Table 14. Continued

Question	Time (min)	Sucralose	Sucrose	Polycose	Amylose	Amylo-pectin	F ; p
8. Tense	0	25.4 ± 5.9	26.6 ± 6.8	24.7 ± 4.0	21.6 ± 3.4	20.4 ± 4.5	0.74; 0.57
	15	23.9 ± 4.7	23.9 ± 5.5	20.1 ± 4.3	22.3 ± 3.5	19.6 ± 5.3	0.42; 0.79
	30	23.6 ● 5.3	21.8 ± 4.2	14.1 ± 2.7	20.1 ± 4.7	18.2 ± 4.2	2.26; 0.08
	45	25.6 ± 5.4	20.7 ± 6.2	14.9 ± 2.7	16.5 ± 3.1	19.6 ± 5.2	2.83; 0.03
	60	20.7 ± 3.9	25.4 ± 5.9	17.2 ± 3.7	15.3 ± 4.4	21.9 ± 6.4	0.81; 0.52

[1]Mean ± SEM; n=14
[2]Question: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3]Question 4 : How much effort is it to do anything? Scoring: Very little = 0; Very much = 100

Table 15. Exp II Change from Baseline Mood after five treatments[1,2].

Question	Time (min)	Polycose	Sucrose	Amylo-pectin	Amylose	Sucralose	F ; p
Global Vigour	15	6.3 ± 4.3	2.3 ± 3.6	1.6 ± 3.9	0.2 ± 2.5	3.6 ± 4.2	0.74; 0.57
	30	9.9 ± 3.0	6.7 ± 4.0	11.2 ± 3.9	3.5 ± 4.1	11.9 ± 4.3	0.95; 0.44
	45	11.8 ± 4.4	5.3 ± 4.7	7.3 ± 4.8	1.4 ± 5.3	7.5 ± 6.1	1.17; 0.33
	60	6.9 ± 4.3	4.9 ± 4.7	5.3 ± 4.0	-0.7 ± 4.9	6.4 ± 5.6	0.96; 0.44
1. Alert	15	-11.1 ± 6.3	-9.7 ± 7.1	-0.6 ± 5.4	-0.7 ± 2.4	-7.7 ± 6.4	1.66; 0.17
	30	-13.4 ± 7.2[ab]	-11.1 ± 7.4[bc]	-2.5 ± 6.6[bc]	-0.3 ± 4.6[c]	-10.3 ± 6.5[a]	3.61; 0.01
	45	-17.4 ± 7.4	-11.4 ± 8.9	-12 ± 6.8	-0.7 ± 5.9	-8.7 ± 7.4	1.7; 0.17
	60	-13.2 ± 6.7	-14.4 ± 9.2	-7.6 ± 6.2	0.5 ± 5.2	-10.4 ± 6.8	1.78; 0.15
2. Sleepy	15	-9.0 ± 6.1	-3.0 ± 4.4	2.1 ± 5.7	4.8 ± 2.9	-5.3 ± 6.9	1.67; 0.17
	30	-4.4 ± 5.6	-2.1 ± 5.1	-0.4 ± 5.9	12.2 ± 6.8	-3.5 ± 7.4	1.81; 0.14
	45	-17.8 ± 6.1	-3.4 ± 5.6	-3.6 ± 4.6	-0.2 ± 7.6	0.2 ± 8.9	1.94; 0.12
	60	-11.6 ± 6.1	-0.1 ± 6.4	-4.4 ± 6.1	7.0 ± 7.2	-4.9 ± 8.0	1.86; 0.13
3. Weary	15	-1.6 ± 3.3	-6.3 ± 4.4	4.3 ± 4.4	0.3 ± 4.5	2.3 ± 4.3	1.00; 0.4
	30	-11.3 ± 6.6	-1.2 ± 4.4	-0.6 ± 5.2	-7.5 ± 7.1	4.8 ± 4.7	1.36; 0.26
	45	0 ± 3.7	0.3 ± 3.4	-1.5 ± 5.5	0.9 ± 6.2	10.1 ± 6.5	1.14; 0.34
	60	-0.9 ± 4.4	-0.14 ± 3.6	-0.9 ± 4.9	-4.4 ± 5.5	7.6 ± 5.4	1.46; 0.23
4. Effort[3]	15	-6.6 ± 5.1	-2.6 ± 5.1	-3.6 ± 6.7	-4.4 ± 3.7	0.9 ± 4.2	0.42; 0.79
	30	-8.3 ± 6.0	1.4 ± 6.9	-9.8 ± 4.7	-2.6 ± 5.2	5.2 ± 7.2	1.86; 0.13
	45	-12. ± 6.2	-6.1 ± 5.1	-14.6 ± 5.7	-3.8 ± 6.1	-2.3 ± 6.9	1.39; 0.25
	60	-3.8 ± 5.9	-5.2 ± 6.6	-9.9 ± 5.0	-9.1 ± 5.7	2.6 ± 7.0	1.11; 0.36

[1]Mean ± SEM; n=14
[2]Question: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3]Question 4 : How much effort is it to do anything? Scoring: Very little = 0; Very much = 100
[a] Means with different superscripts within a row are significantly different; $p<0.05$

Table 15. Continued

Question	Time (min)	Polycose	Sucrose	Amylo-pectin	Amylose	Sucralose	F ; p
Global Affect	15	4.1 1.6	1.1 2.2	-0.04 3.0	1.8 1.3	2.9 2.8	0.53; 0.71
	30	6.5 2.2	1.7 2.1	0.9 2.2	0.2 1.6	0.8 2.9	1.31; 0.28
	45	6.1 1.6	1.5 2.5	-4.1 3.6	1.3 1.5	1.2 3.7	1.84; 0.14
	60	13.3 2.4	12.6 2.8	10.5 1.8	10.8 1.9	12.7 2.2	0.7; 0.6
5. Sad	15	-3.9 ± 2.3	-4.3 ± 3.6	-5.3 ± 1.9	3.35 ± 2.9	-1.5 ± 1.5	1.46; 0.22
	30	-1.2 ± 2.7	-4.6 ± 4.5	-13.5± 5.3	4.0 ± 2.8	-5.3 ± 2.6	1.87; 0.13
	45	-1.3 ± 1.9	-10.1 ± 5.3	-9.5 ± 4.6	-1.07 ± 3.3	-4.4 ± 2.1	2.35; 0.07
	60	-0.8 ± 2.7	-9.3 ± 4.8	-3.9 ± 2.3	2.6 ± 2.2	-6.8 ± 3.4	2.46; 0.06
6. Calm	15	-8.9 ± 3.9	-4.6 ± 4.6	-0.07 ± 4.3	-2.6 ± 2.7	-6.7 ± 6.8	0.64; 0.63
	30	-9.9 ± 3.6	-5.9 ± 3.8	0.36 ± 4.0	-0.4 ± 2.8	-4.5 ± 7.5	0.94; 0.45
	45	-9.8 ± 3.7	-11.8 ± 4.5	1.7 ± 5.5	-3.7 ± 2.5	-4.3 ± 9.3	1.12; 0.36
	60	-10.4 ± 4.3	-4.4 ± 6.1	4.5 ± 6.6	-2.0 ± 3.2	-10.6 ± 7.1	1.33; 0.27
7. Happy	15	6.9 ± 4.4	1.3 ± 2.9	3.6 ± 4.7	1.8 ± 1.9	0.4 ± 2.8	0.58; 0.68
	30	6.6 ± 4.9	0.8 ± 3.5	7.4 ± 4.3	-5.0 ± 3.1	-2.3 ± 4.1	2.07; 0.1
	45	6.1 ± 3.7	-0.7 ± 4.0	-0.7 ± 5.2	-2.4 ± 2.4	0.4 ± 2.5	1.19; 0.33
	60	7.1 ± 4.3	-1.6 ± 4.3	-0.07 ± 5.0	-1.1 ± 3.0	0.2 ± 3.7	1.02; 0.41
8. Tense	15	4.6 ± 2.6	2.8 ± 2.8	0.8 ± 2.1	-0.7 ± 2.6	1.5 ± 3.1	0.62; 0.65
	30	10.6 ± 3.7	4.9 ± 4.2	2.1 ± 1.2	1.5 ± 3.1	1.8 ± 2.6	1.46; 0.23
	45	9.8 ± 3.1	4.9 ± 5.1	0.5 ± 3.3	5.1 ± 2.5	-0.3 ± 3.9	1.16; 0.34
	60	7.5 ± 2.9	5.9 ± 5.2	-1.5 ± 3.4	6.3 ± 3.0	4.6 ± 4.7	0.84 0.5

[1] Mean ± SEM; n=14
[2] Question phrase: How (adjective) do you feel?   Scoring: Very little = 0; Very much = 100
[3] Question 4 phrased differently: How much of an effort is it too do anything
   Scoring: Very little = 0; Very much = 100
[a] Means with different superscripts within a row are significantly different; p<0.05
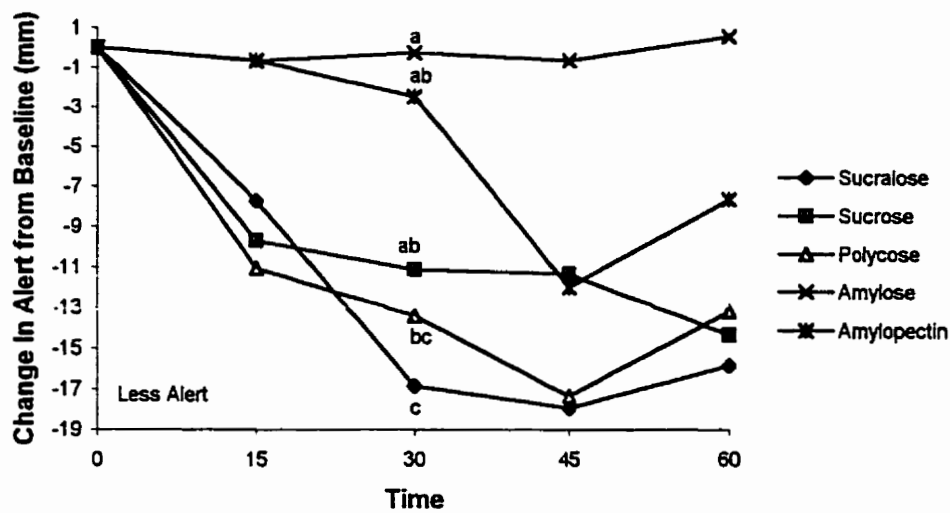
Figure 5. Change in Alert from Baseline after five treatments. Values within the same timepoint with different letters are significantly different (p<0.02 ; n=14).
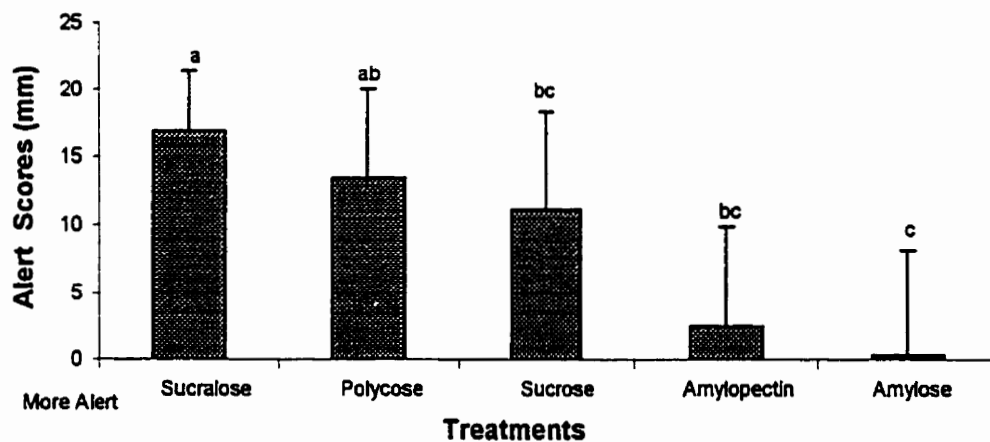
Figure 6. Change from Baseline Alert scores at 30 minutes after five treatments. Treatments with different letters are significantly different (p<0.02 ; n=14).

B. DISCUSSION

No effect of treatment on overall global vigour and global affect was found. However, amylose, amylopectin and sucrose increased ratings of alertness compared to control 30 minutes post preload. Of the few studies that have examined the effect of pure nutrients on mood, either the opposite effect or no effect at all has been shown. For example, consumption of 50g sucrose has been shown to increase ratings of sleepiness after one hour (Pivonka & Grunewald 1990), whereas a recent study in our own group found no effect of 25g, 50g or 75g preloads of sucrose on mood over one hour (Woodend 2000). It is possible that a more sensitive sample population, such as depressed individuals or a longer experimental time frame is required to detect a subtle treatment effect on mood.

Although the lower glycemic treatments, amylose and amylopectin were found to sustain alertness at 30 minutes compared to the high glycemic treatments, polycose and sucrose, there is only a weak argument for a positive relationship between the glycemic response and alertness. Considering, sucralose the lowest glycemic treatment decreased alertness similar to polycose, this would rule out a direct role for blood glucose in mood regulation. However, to clarify the role of blood glucose in mood regulation, both the glycemic response and ratings of mood need to be compared in a within subject design.

Therefore experiment III was designed to include treatments with a range of glycemic responses to investigate the relationship between the glycemic response to carbohydrates and mood.

PART IV

THE EFFECT OF CARBOHYDRATE TREATMENTS ON MEMORY

A. RESULTS:

1. Immediate Recall

There was no effect of treatment on the total number of words recalled at 15 minutes [F=0.85 ; p=0.5] (Table 16). However, there was a treatment effect on a measure of learning at 15 minutes [F=3.55 ; p=0.012] which was expressed as the mean number of words recalled at the third presentation of a word list compared to the mean number of words recalled at the first presentation of the word list (Table 16). When expressed as the difference from control, polycose resulted in the least number of words recalled compared to all other treatments [F=4.29 ; p=0.01] (Table 17).

A treatment effect was observed for total immediate recall at 60 minutes [F=2.88, p=0.03] (Table 16). Sucrose significantly increased the total number of words recalled at 60 minutes (total number of words recalled in all three presentations of the word list) compared to polycose, amylopectin and amylose.

When expressed as the difference from control, sucrose significantly increased the total number of words recalled at 60 minutes compared to all other treatments [F=3.92 ; p=0.015].

The total number of words recalled at 15 minutes and 60 minutes is a summary measure of the effect of treatment on immediate recall over one hour. Sucrose improved total immediate recall for a word list compared to amylose, amylopectin and polycose [F=2.63 ; p=0.04] (Table 16). When the data for total immediate recall was expressed as

the difference from control, sucrose was found to significantly improve immediate recall compared to all other treatments (F=3.04 ; p=0.04) (Table 17).

2. Delayed Recall

No effect of treatment was observed on delayed recall for a word list, 45 minutes post preload [F=0.71 ; p=0.59] (Table 16).

3. Trail Making Test

A treatment effect was observed on Part B of the trail making test [F=2.59 ; p=0.047]. Amylopectin significantly decreased the time to complete the trail making test compared to control (Table 16). No treatment effect was observed when values were expressed as the difference from control [F=1.00 ; p=0.404]. There was no treatment effect on the absolute values for Part A of the trail making test [F=0.39 ; p=0.82] or when expressed as the difference from control [F=0.45 ; p=0.72].

Table 16. Experiment II. Memory Scores 15, 45 and 60 minutes Post Preload[1]

Time	Sucralose	Sucrose	Polycose	Amylose	Amylopectin	F;p
15-1st Trial	6.4 ± 0.5	6.9 ± 0.6	7.0 ± 0.6	6.6 ± 0.6	5.9 ± 0.6	0.87; 0.49
15-2nd Trial	9.9 ± 0.9	10.4 ± 0.8	9.3 ± 0.6	9.8 ● 0.8	9.3 ± 0.7	0.72; 0.58
15-3rd Trial	12.5 ± 0.7	12.9 ± 0.8	10.9 ± 0.6	12.1 ± 0.8	11.9 ± 0.8	1.80; 0.14
15-(3rd1st)	6.14 ± 0.6^a	6.0 ± 0.5^a	3.9 ± 0.6^b	5.4 ± 0.5^a	5.9 ± 0.4^a	3.55;0.01
Total 15 Score	28.7 ± 1.9	30.1 ± 2.0	27.1 ± 1.6	28.5 ± 2.0	27.1 ± 1.9	0.86; 0.49
45 Score	8.5 ± 0.7	8.1 ± 0.8	7.3 ± 0.6	7.2 ± 0.8	8.1 ± 0.8	0.71; 0.59
60-1st Trial	6.3 ● 0.6	5.9 ± 0.4	6.3 ± 0.6	7.2 ± 0.7	7.0 ± 0.7	2.21; 0.08
60-2nd Trial	9.4 ± 0.7	9.6 ± 0.9	9.6 ± 0.7	11.1 ● 0.9	9.8 ± 1.0	1.91; 0.12
60-3rd Trial	12.1 ± 0.7	11.9 ± 1.0	12.1 ± 0.9	13.2 ± 1.0	12.0 ± 1.0	1.57; 0.20
Total 60 Score	28.8±2.6^{ab}	31.5 ± 2.4^a	28.1 ±2.0^b	27.8 ±1.8^b	27.4 ±2.1^b	2.88; 0.03
Total 15+60 Score	56.3±4.2^{ab}	60.4 ± 4.1^a	54.1 ±2.9^b	55.3 ±3.6^b	53.6 ±3.4^b	2.63;0.04
TrailMaking Test B	67.3±5.7^a	59.3±4.6^{ab}	57.6±4.4^a	59.6±4.9^b	52.9±3.3^{ab}	2.59;0.04
Test A	50.2±3.9	49.2±4.6	53.9±6.4	46.9±5.3	50.4±4.5	0.39;0.82

[1] Mean ± SEM (number of correct words recalled); n = 14

^{a} Means within a row with different letters are significantly different ;p<0.05

Table 17. Experiment II. Memory Scores Expressed as the Difference from Control[1]

Time	Sucrose	Polycose	Amylose	Amylopectin	F ; p
15-(3rd1st)	0.14 ± 0.8^a	2.3 ± 0.7^b	0.7 ± 0.8^a	0.2 ± 0.7^a	4.29;0.01
Total 60 Score	2.5 ± 1.1^a	-0.6 ± 1.3^b	-1.1 ± 1.5^b	-1.1 ± 1.3^b	3.66;0.02
Total 15+60 Score	4.1 ± 1.7^a	-1.1 ±2.4^b	-2.2 ±2.4^b	-2.7 ± 2.1^b	3.04;0.04

[1] Mean ● SEM (number of correct words recalled); n = 14

^{a} Means within a row with different letters are significantly different ;p<0.05

B. DISCUSSION

In the present study, sucrose was found to improve immediate recall compared to amylose, amylopectin and polycose. Polycose decreased memory at 15 minutes compared to all other carbohydrate treatments except control.

The results do not support a relationship between the glycemic response to the carbohydrates and memory performance. For example, the carbohydrate treatments, amylose, amylopectin and polycose, which are composed solely of glucose polymers, demonstrated no effect or impairment on memory over one hour. Furthermore, the decrease in memory observed with the high glycemic carbohydrate, polycose, does not lend support to a relationship between high blood glucose levels and memory improvement. This is in contrast to previous studies that have suggested that the postprandial increase in blood glucose is responsible for the memory enhancing effects of glucose. For example, failure to eat breakfast results in a decline in performance of word list recall and memory tests, which can be reversed upon administration of a 50g glucose-supplemented drink (Benton & Owens 1993). It is possible that the 75g dose employed in the present study was not in the optimum dose range observed for a beneficial effect on memory. It is shown that the effect of glucose on memory follows an inverted U shape dose response curve whereby doses lower or higher than 50g do not show an improvement in performance (Gold 1986; Messier & White 1987).

The results suggest a beneficial effect of 75g sucrose on memory for a word list, supporting a preliminary study, which found that 75g sucrose prevented a decline in memory between 15 and 60 minutes (Hui 1998, unpublished). Conversely, a more recent

study found no effect of a range of sucrose doses (25g, 50g and 75g) on memory for a word list in young adults over one hour (Woodend 2000).

The mechanism(s) by which sucrose may improve memory is unclear. However, a hypothesis can be formulated for a beneficial effect of fructose on memory based on the observation that sucrose, which is composed of glucose and fructose improved immediate recall over one hour. The fructose component of sucrose has been implicated in animal studies to improve memory (Rodriguez et al 1994, 1999), however, no human studies to date have investigated the role of fructose in memory.

Based on the results of the present study, experiment III was designed to explore the relationship between the glycemic effect of sucrose, polycose, glucose and a high fructose preload on memory over one hour.

PART V

RELATIONSHIPS BETWEEN CARBOHYDRATES AND APPETITE,

FOOD INTAKE, MOOD AND MEMORY

The relationships between all individual dependent measures for experiment II are shown in the Appendix. The statistically significant relationships are presented below.

A. RESULTS

A positive correlation was found between 60 min average appetite and mealtime food intake for all five treatments [$r=0.39$; $p=0.0006$] (Figure 7).

No correlation was found between mealtime food intake and 60 min global vigour and global affect. A significant positive correlation between average appetite and global vigour was observed [$r=0.4$; $p=0.0007$] (Figure 8).

A positive correlation was observed between global vigour and memory at 60 minutes for a word list shown third times [$r = 0.25$; $p<0.04$] .

A positive correlation was observed between ratings of Alert at 30 minutes and memory at 60 minutes for a word list shown three times [$r=0.38$; $p=0.001$] (Figure 9).
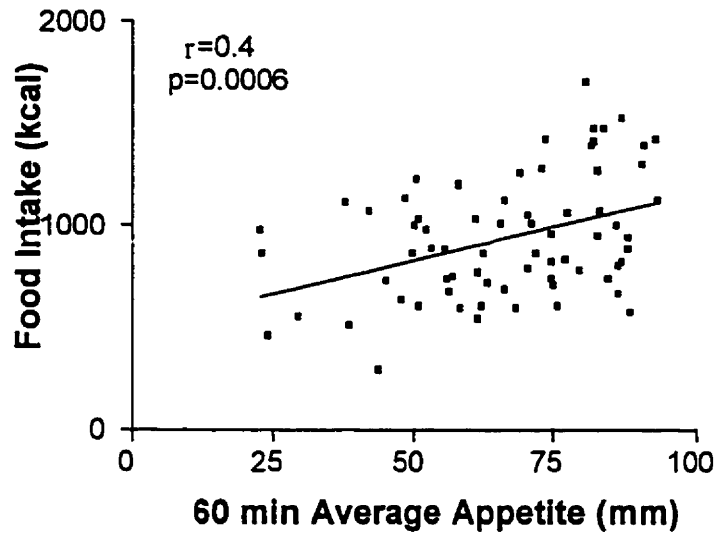
Figure 7. Mealtime Food Intake versus Average Appetite at 60 minutes after five treatments.
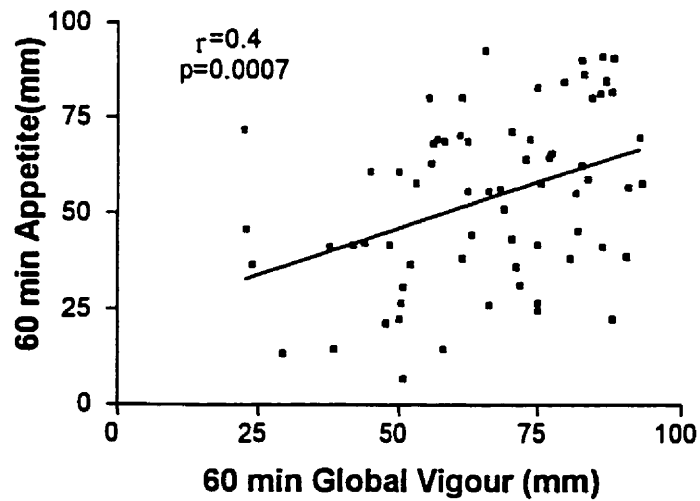
Figure 8. Global Vigour at 60 minutes versus Average Appetite at 60 minutes after five treatments.
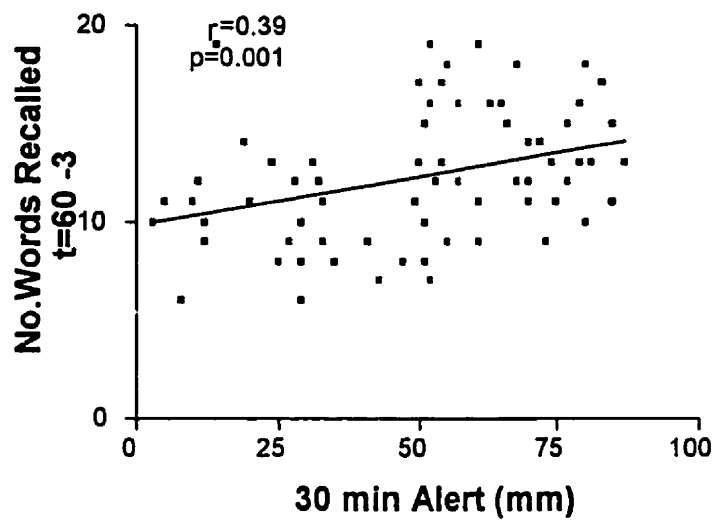
Figure 9. Ratings of Alert at 30 minutes versus number of words recalled for a word list after third showing at 60 minutes after five treatments.

B. DISCUSSION

This experiment demonstrated significant interactions between the effect of carbohydrate preloads on appetite, food intake, mood and memory in young adults.

Higher average appetite immediately prior to the test meal was associated with increased mealtime energy intake and ratings of global vigour at 60 minutes. Furthermore, ratings of alertness at 30 minutes were associated positively with 60 minute memory performance at the third trial.

The positive relationship observed between appetite and food intake is expected based on the assumption that increased appetite immediately prior to a meal results in greater intake at that meal (Appendix VI ; Table ii). The results support the validity of visual analogue scales as a useful tool for measuring the effect of treatment over time on appetite (Woodend 2000; Brown 1998; Stewart et al 1997).

The positive relationship between global vigour and appetite at 60 minutes suggests that the more vigorous the participants felt, the more stimulated was their appetite. This is evident in the significant relationship between alertness and average appetite (Appendix VI; Table iii). Subsequently, increased ratings of negative feelings such as weariness, sleepiness and effort were correlated with a decrease in appetite at 60 minutes.

Consistent with an expected relationship between mood and memory, increased ratings of alertness at 30 minutes was correlated with increased retention for a word list at 60 minutes (Figure 9). This suggests that the more alert a subject feels, the better their performance on a memory test.

It is unlikely that one common mechanism explains the interaction among the responses to appetite, food intake, mood and memory. However, one of the primary signals proposed to underlie the effects of carbohydrates on brain function is the postprandial increase in blood glucose. Therefore, experiment III was designed to investigate the relationships among all dependent variables including blood glucose in the same population.

EXPERIMENT III

THE EFFECT OF CARBOHYDRATES ON BLOOD GLUCOSE, APPETITE, FOOD INTAKE, MOOD AND MEMORY

The effect of carbohydrate treatments on blood glucose is presented first, followed by their effect on appetite and food intake. Then the effect of the carbohydrate preloads on mood and on memory is presented in the third and fourth sections. In the final section, the interrelationships between all dependent measures are explored.

PART I

THE EFFECT OF CARBOHYDRATE TREATMENTS ON BLOOD GLUCOSE

A. RESULTS

Mean blood glucose concentrations measured over the one hour were affected by treatment [F=36.14 ; p<0.0001]. A time [F= 58.51; p<0.0001] and a time by treatment interaction [F= 15.85; p<0.0001] was also found (Table 18). Polycose and glucose produced the greatest increase in blood glucose at all times, remaining elevated above baseline levels at 65 minutes. Sucrose demonstrated a rapid increase in blood glucose between baseline and 20 minutes and was significantly higher than baseline at 65 minutes. The fructose/glucose-combined treatment elicited a smaller increase in blood glucose than all other carbohydrate treatments that returned to baseline by 65 minutes.

The time effect was demonstrated by a pattern of increase in blood glucose to 20 minutes, which was sustained at 37 minutes and then decreased to 65 minutes after carbohydrate treatments. Sucralose demonstrated no significant increase in blood glucose over time (Table 19).

The incremental area under the blood glucose curve was affected by treatment [F=39.5; p<0.0001]. Glucose and polycose produced the greatest AUC, followed by sucrose and the fructose/glucose mixture. Sucralose produced the lowest AUC (Table 20).

The changes in blood glucose concentration between each of the times of measurement are summarised in Table 21.

The mean change in blood glucose concentrations when expressed as the difference from baseline was affected by treatment [F=53.13; p<.0001]. A time [F=16.9 ; p<.0001] and time by treatment interaction was found [F=4.15 ; p=0.0002]. As with the absolute values, the treatment effect was shown by an increase in blood glucose concentrations above baseline that was greatest for polycose and glucose and intermediate for sucrose and lowest for the fructose/glucose preload at each of the timepoints (Figure 10).

The increase in blood glucose concentration between baseline and 20 minutes [F=38.71 ; p<.0001], 37 minutes [F=34.08 ; p<.0001] and 65 minutes [F=21.96 ; p<.0001] was affected by treatment. Polycose and glucose demonstrated the greatest rise in blood glucose concentration over time. The rise in blood glucose concentration for fructose/glucose was significantly lower, with sucralose demonstrating no increase in blood glucose with time (Table 21).

The fall in blood glucose expressed as the difference between blood glucose concentrations 37 minutes and 65 minutes after preloads was affected by treatment [F=3.42 ; p=0.01] (Table 21). All carbohydrate treatments produced a decline in blood glucose between 37 and 65 minutes compared to control.

Table 18. Exp. III Effect of Treatment on Blood Glucose Concentrations At Each Time[1]

Treatment	0	Time (mins) 20	37	65
Sucralose	5.14 ± 0.1	5.15 ± 0.1^a	5.18 ± 0.1^a	5.16 ± 0.1^a
Fructose	5.24 ± 0.1	7.12 ± 0.3^b	6.90 ± 0.2^b	5.60 ± 0.2^a
Sucrose	5.22 ± 0.1	8.30 ± 0.3^c	7.80 ± 0.4^c	6.40 ± 0.3^b
Polycose	5.18 ± 0.1	8.70 ± 0.4^c	9.02 ± 0.5^d	7.73 ● 0.3^c
Glucose	5.08 ± 0.1	8.37 ± 0.3^c	9.0 ± 0.5^d	7.67 ± 0.4^c
F	0.34	32.53	30.83	18.52
P	0.85	<.0001	<.0001	<.0001

[1] Mean ± SEM (mmol/L) ; n=15

^{a} Means with different superscripts within a column are significantly different ; p<0.0001

Table 19. Exp. III Effect of Time on Blood Glucose Concentrations For Each Treatment[1]

Treatment	0	Time (mins) 20	37	65	F ; p
Sucralose	5.14 ± 0.1	5.15 ± 0.1	5.18 ± 0.1	5.16 ± 0.1	0.1 ; <.9585
Fructose	5.24 ± 0.1^a	7.12 ± 0.3^b	6.9 ± 0.2^b	5.60 ± 0.2^a	25.68 ; <.0001
Sucrose	5.22 ± 0.1^a	8.30 ± 0.3^b	7.8 ± 0.4^b	6.40 ± 0.3^c	26.01 ; <.0001
Polycose	5.18 ● 0.1^a	8.70 ± 0.4^b	9.02 ± 0.5^b	7.73 ± 0.3^c	48.2 ; <.0001
Glucose	5.08 ± 0.1^a	8.37 ± 0.3^{bc}	9.0 ● 0.5^c	7.67 ± 0.4^b	34.75 ; <.0001

[1] Mean ± SEM (mmol/L) ; n=15

^{a} Means with different superscripts within a row are significantly different ; p<0.0001
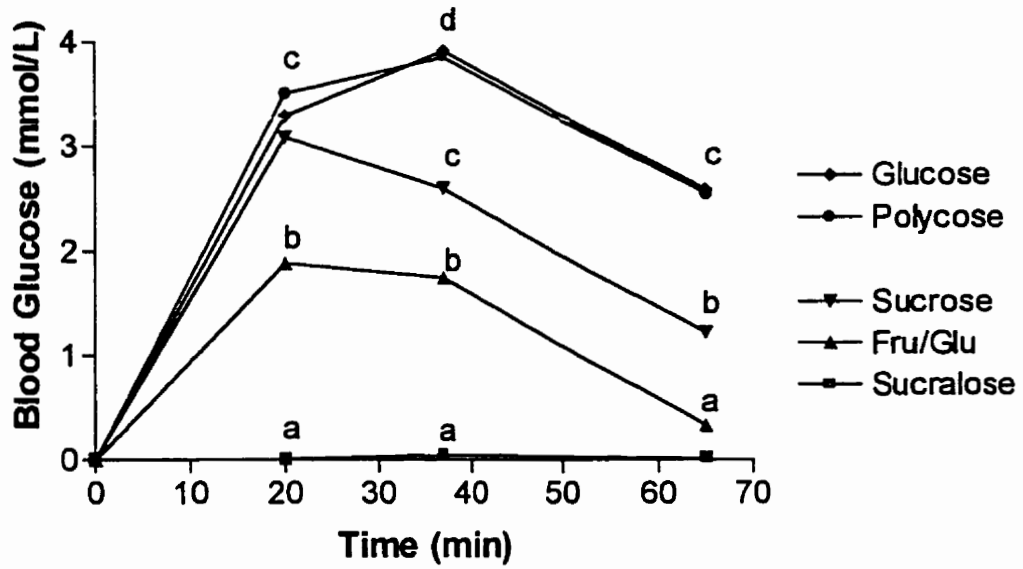
Figure 10. Change from Baseline Blood Glucose concentration after five treatments. Values with different letters, within the same timepoint are significantly different (p<.0001 ; n=15).

Table 20. Exp. III. Blood Glucose Area Under the Curve after Treatments[1]

Treatment	Incremental AUC (mmol/L/min)
Sucralose	7.97 ± 2.2 [a]
Fru/Glu	71.5 ± 8.3 [b]
Sucrose	131.6 ± 16.9 [c]
Polycose	177.5 ± 19.4 [d]
Glucose	190.5 ± 19.1 [d]
F	39.5
p	<.0001

[1] Mean ± SEM, n=15

[a] Means with different superscripts, within a column, are significantly different.

Table 21. Exp III. Effect of Treatment on Blood Glucose Concentrations Between Timepoints[1]

Blood Glucose (mmol/L)	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F ; p
20min-0min	0.01±0.1 [a]	1.89±0.2 [b]	3.09±0.3 [c]	3.51±0.3 [c]	3.29±0.3 [c]	38.71; <.0001
37min-0min	0.05±0.1 [a]	1.74±0.2 [b]	2.59±0.5 [c]	3.84±0.4 [d]	3.91±0.5 [d]	34.08; <.0001
65min-0min	0.02±0.1 [a]	0.33±0.2 [a]	1.22±0.3 [b]	2.55±0.3 [c]	2.59±0.4 [c]	21.96; <.0001
37min-20min	0.03±0.1 [abc]	-0.14±0.3 [bc]	-0.44±0.4 [c]	0.34±0.3 [ab]	0.62±0.3 [a]	3.15; 0.02
65min-20min	0.01±0.1 [a]	-1.63±0.3 [bc]	-1.86±0.3 [c]	-0.95±0.3 [bc]	-0.70±0.4 [ab]	5.44; .0009
65min-37min	-0.03±0.1 [a]	-1.44±0.2 [b]	-1.43±0.5 [b]	-1.29±0.3 [b]	-1.33±0.5 [b]	3.42; 0.01

[1] Mean ± SEM, n=15

[a] Means with different superscripts, within a row, are significantly different.

B. DISCUSSION

The carbohydrate treatments selected for this experiment resulted in a wide range in blood glucose responses. Glucose demonstrated the greatest increase in AUC blood glucose, followed by polycose and then sucrose. The fructose/glucose preload produced the lowest AUC blood glucose response compared to the other carbohydrate treatments whereas a minimal increase in blood glucose was observed with the control, sucralose.

The postprandial increase in blood glucose for sucrose and glucose observed in the present study is consistent with the literature. Glucose produces a rapid and high increase in postprandial blood glucose and has a high glycemic index (GI) of 100 (Foster-Powell & Brand Miller 1995). Polycose, which is readily digested also produced a high glycemic response consistent with the data in experiment one. The intermediate glycemic response to sucrose reflects the presence of the two monosaccharides, lying between that of the higher GI of glucose and the lower GI of fructose (Lee & Wolever 1998).

Calculations can be made to estimate the glycemic response for the 80%:20% fructose/glucose preload based on the glycemic response obtained with the glucose and sucrose treatments. The AUC of 100% fructose can be calculated first:

(2 x (131.6)-190.5 = 72.7mmol/min/L) which allows the AUC for the 80%fructose / 20% glucose preload to be calculated:

(0.8 (72.7) + 0.2 (190.5) = 96.3mmol/min/L).

Thus, the actual value obtained for the fructose/glucose treatment (71.5 mmol/min/L) lies reasonably close to the estimated value (96.3 mmol/min/L) when based on a sample size of n=15. However, because the GI of glucose (100) is based on a much larger scale, the

decrease in variation allows for a more accurate calculation of 78.16 when substituted for the glucose AUC (Appendix IV).

It is therefore accurate to say that the blood glucose response obtained for the fructose/glucose preload was realistic.

PART II:

THE EFFECT OF CARBOHYDRATE TREATMENTS ON SUBJECTIVE

APPETITE AND SHORT TERM FOOD INTAKE

A. RESULTS

1.FOOD AND WATER INTAKE

Treatment affected mealtime energy intake at one hour [F=2.56, p=0.049]. Glucose and sucrose decreased food intake compared to control but food intake after fructose/glucose and polycose treatments was not different from all other treatments (Table 22).

There was no significant effect of treatment on the compensation (in percent) at meal time for the calories consumed in the 300kcal preload [F=1.53; p=0.22]. (Table 22). However, an average of 40% compensation was observed for all treatments except for the fructose/glucose mixture which resulted in less than 12% compensation.

The amount of water consumed with the test meal was not affected by treatment [F= 0.31 ; p= 0.86] (Table 22).

Table 22. Exp III Food and Water Intake after Treatments[1]

Treatment	Test Meal [2] (kcals)	% Compensation[3]	Water (grams)
Sucralose	998 ± 69.9 [a]		451 ± 70.4
Fru/Glu	964 ± 77.3 [ab]	11.52 ± 15.7	441 ± 46.7
Sucrose	871 ± 78.9 [b]	42.34 ± 14.3	416 ± 55.2
Polycose	889 ± 72.3 [ab]	36.38 ● 16.05	416 ± 53.2
Glucose	854 ± 81.5 [b]	48.14 ± 25.5	438 ± 49.6
F	2.56	1.53	0.31
P	0.048	0.22	0.87

[1] Mean ± SEM, n=15

[2] Energy Consumed (kcal) in a test meal 60 minutes following preload

3 Calories Consumed after Control – Calories Consumed Treatment/ Calories in Preload x 100

[a] Means with different superscripts, within a column, are significantly different

2. AVERAGE APPETITE

No effect of time [F= 2.13 ; p=0.09], treatment [F= 1.11 ; p=0.36] or a time by treatment interaction [F= 1.09 ; p=0.367] was observed for absolute average appetite over one hour following consumption of treatments (Table 24). When the data are expressed as the difference from baseline, there was an effect of time [F= 3.78 ; p=0.02] but no treatment effect [F=0.62 ; 0.65] or a time by treatment interaction [F= 1.47 ; p=0.14] observed (Table 25).

2.a. Individual Average Appetite Questions

The absolute values for desire to eat were not affected by time [F= 1.9 ; p=0.12], treatment [F= 0.45 ; p=0.77] or a time by treatment interaction [F= 0.7 ; p=0.79] (Table 23). When the data were expressed as change from baseline, there was no treatment [F=0.56 ; p=0.69] or time by treatment interaction [F=0.91 ; p=53]. An effect of time was

found as desire to eat increased with time after all treatments[F=4.32 ; p=0.0096] (Table 24).

The absolute values for hunger were not affected by time [F= 1.55 ; p=0.2], treatment [F= 1.62 ; p=0.18] or a time by treatment interaction [F= 1.58 ; p=0.07] (Table 23). When the data was expressed as change from baseline, there was no time [F= 2.36 ; p=0.09], treatment [F= 0.8 ; p=0.52] or time by treatment interaction [F= 0.75 ; p=0.7] (Table 24).

The absolute values for fullness were not affected by treatment [F=1.16 ; p=0.33] or a time by treatment interaction [F=0.91 ; p=0.55]. The time effect [F=4.0 ; p=0.006] was demonstrated by a decrease in ratings of fullness with time (Table 23). When the data was expressed as the change from baseline, there was no effect of treatment [F=0.86 ; p=0.49], time [0.64 ; p=0.59] or time by treatment interaction [F = 0.74 ; p=0.7] (Table 24).

The absolute values for amount were not affected by time [F= 2.3 ; p=0.09], treatment [F= 0.67 ; p=0.6] or a time by treatment interaction [F= 0.99 ; p=0.45] (Table 23). When the data was expressed as change from baseline, there was no time [F= 1.59 ; p=0.19], treatment [F= 0.99 ; p=0.41] or time by treatment interaction [F= 1.07 ; p=0.38] (Table 24).

Table 23. Exp. III Absolute Average Appetite after five treatments[1,2]

Question	Time (min)	Sucralose	Fruc/Glu	Sucrose	Polycose	Glucose	F;p
Average Appetite	0	71.1 ● 3.4	69.2 ± 3.4	71.0 ± 3.8	70.4 ± 2.4	69.4 ● 3.5	0.14 ; 0.97
	15	67.4 ± 3.4	64.8 ± 3.3	64.8 ± 3.8	64.8 ● 2.7	64.8 ● 3.2	0.25 ; 0.91
	30	70.8 ± 3.6	68.9 ± 3.2	70.3 ± 3.7	66.8 ± 3.5	65.1 ± 3.3	1.05 ; 0.39
	45	72.7 ± 3.9	66.2 ± 3.7	68.0 ± 4.1	60.1 ± 3.8	65.0 ± 4.0	1.71 ; 0.16
	60	76.5 ± 4.1	70.6 ± 3.4	67.2 ± 4.5	70.5 ± 3.7	68.2 ± 4.4	2.36 ; 0.06
1. Desire	0	67.1 ± 5.4	61.9 ± 5.7	68.3 ± 5.3	68.1 ± 4.3	65.3 ± 4.7	0.69 ; 0.60
	15	63.5 ± 5.0	61.8 ± 4.4	61.2 ± 5.0	63.3 ± 3.2	62.9 ● 4.6	0.13 ; 0.97
	30	68.7 ± 4.8	65.9 ± 3.8	68.3 ± 4.6	65.5 ± 4.2	65.9 ± 3.7	0.70 ; 0.60
	45	69.7 ± 4.9	65.6 ± 4.2	66.3 ± 5.0	67.5 ± 4.5	66.1 ± 4.6	0.35 ; 0.84
	60	72.9 ● 5.2	68.7 ± 3.8	65.7 ± 5.3	70.3 ± 4.4	67.0 ± 5.9	0.94 ; 0.45
2. Hunger	0	67.2 ± 4.0	63.9 ± 4.8	65.7 ● 4.7	66.2 ± 3.5	63.6 ± 4.0	0.38 ; 0.82
	15	62.4 ± 4.0	61.1 ± 3.9	63.7 ± 4.4	56.8 ± 4.5	59.6 ± 3.9	0.91 ; 0.46
	30	67.2 ± 4.3	64.2 ± 3.9	69.1 ± 4.4	60.7 ± 5.0	63.2 ± 3.7	1.35 ; 0.26
	45	69.1 ± 4.5	62.2 ± 4.0	65.0 ± 5.1	58.3 ± 5.6	59.9 ± 5.2	2.46 ; 0.06
	60	74.7 ± 4.9	66.3 ± 3.8	62.1 ± 5.8	65.6 ± 4.6	63.1 ± 5.3	2.48 ; 0.05
3. Full[3]	0	18.9 ± 3.4	17.4 ± 4.1	19.3 ± 3.8	20.1 ± 3.4	18.8 ± 5.3	0.10 ; 0.98
	15	22.5 ± 3.8	29.1 ± 5.1	28.9 ± 5.6	25.1 ± 3.4	26.8 ± 5.4	0.61 ; 0.66
	30	20.3 ± 4.3	23.1 ± 3.3	24.1 ± 5.1	23.8 ± 3.3	31.4 ± 5.5	1.61 ; 0.18
	45	19.4 ± 4.1	26.9 ± 4.0	25.3 ± 5.2	24.5 ± 4.1	28.4 ± 5.0	1.42 ; 0.24
	60	15.9 ± 3.9	22.3 ± 3.6	24.1 ± 4.7	21.5 ± 3.7	23.7 ± 4.7	2.09 ; 0.09

[1] Mean ± SEM; n=15
[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100

Table 23.Continued

Question	Time (min)	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F;p
4. Amount	0	68.9 ± 3.2	68.5 ± 3.8	69.3 ± 3.5	67.5 ± 3.2	67.7 ± 3.1	0.08 ; 0.99
	15	66.3 ± 3.4	65.6 ± 3.6	62.9 ± 3.5	64.2 ± 3.5	63.3 ●3.2	0.32 ; 0.87
	30	67.6 ± 3.9	68.4 ± 4.0	68.1 ± 3.6	66.7 ± 4.2	62.7 ± 3.5	1.16 ; 0.34
	45	71.5 ± 3.9	64.1 ± 4.9	65.7 ± 4.5	63.1 ± 4.4	62.5 ± 4.6	1.80 ; 0.14
	60	74.3 ●4.5	69.5 ± 4.0	65.1 ± 5.4	67.6 ± 4.6	66.5 ± 4.4	0.84 ; 0.50

[1] Mean ± SEM; n=15
[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100

Table 24. Exp III Change from Baseline Average Appetite after five treatments[1,2]

Question	Time (min)	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F;p
Average	15	-3.67 ± 2.7	-4.38 ± 3.2	-6.25 ± 2.7	-5.63 ± 2.4	-4.68 ± 3.5	0.18 ; 0.94
Appetite	30	-0.28 ± 2.7	-0.37 ± 4.2	-0.67 ± 2.7	-3.63 ± 3.1	-4.35 ± 3.8	0.51 ; 0.73
	45	1.65 ± 3.4	-2.98 ± 4.8	-3.05 ± 3.4	-4.37 ± 3.1	-4.42 ± 4.6	0.77 ; 0.55
	60	5.4 ± 3.9	1.33 ± 4.0	-3.8 ± 4.1	0.08 ± 3.1	-1.23 ± 4.5	1.55 ; 0.20
1. Desire	15	-3.7 ± 4.1	4.5 ± -0.07	-5.9 ● 2.5	-4.7 ± 3.1	-1.7 ± 4.6	0.73 ; 0.51
	30	1.6 ± 3.5	5.3 ± 3.9	0.1 ± 3.3	-4.5 ± 4.8	1.8 ± 3.9	0.56 ; 0.74
	45	3.2 ± 5.0	4.4 ± 5.8	-1.4 ± 3.6	-0.6 ± 4.6	0.9 ± 5.2	0.85 ; 0.34
	60	5.7 ± 4.8	7.3 ± 5.7	-2.6 ± 5.0	2.3 ± 4.8	1.7 ± 6.0	0.87 ; 0.48
2. Hunger	15	-4.8 ± 3.3	-2.8 ± 4.0	-2.0 ± 2.7	-9.4 ± 4.5	-4.0 ± 4.9	0.87 ; 0.49
	30	0.0 ± 3.3	0.3 ± 5.7	3.3 ± 3.0	-5.1 ± 4.0	-0.4 ± 4.8	0.86 ; 0.49
	45	1.2 ± 4.1	-1.7 ± 5.7	-0.7 ± 4.6	-7.9 ± 4.4	-3.9 ± 4.5	1.17 ; 0.33
	60	2.3 ± 6.7	2.3 ± 4.9	-0.5 ± 4.4	-0.6 ± 3.8	-1.2 ± 4.7	0.23 ; 0.92
3. Full[3]	15	3.7 ± 3.0	11.7 ± 4.6	9.6 ± 4.6	4.0 ± 2.8	2.1 ± 4.6	1.12 ; 0.36
	30	1.5 ± 3.0	5.5 ± 4.9	5.1 ± 3.4	3.7 ± 2.5	6.1 ± 5.8	0.92 ; 0.22
	45	1.7 ± 2.8	9.5 ± 5.5	6.5 ± 3.6	4.5 ± 3.4	4.5 ± 5.9	0.66 ; 0.62
	60	-3.0 ± 3.2	4.9 ± 4.9	4.9 ± 3.4	3.9 ± 2.4	6.9 ± 4.6	1.41 ; 0.24
4. Amount	15	-1.3 ± 2.3	-2.9 ± 3.1	-6.3 ± 3.8	-3.3 ± 2.8	-4.3 ± 2.5	0.78 ; 0.43
	30	-0.6 ± 3.4	-0.1 ± 4.3	-1.2 ± 3.4	-0.8 ± 3.5	-5.0 ● 3.3	0.78 ; 0.44
	45	2.0 ● 3.7	-4.5 ± 5.2	-3.5 ± 3.9	-4.5 ± 3.3	-5.1 ● 5.1	0.78 ; 0.54
	60	5.3 ± 4.1	1.0 ± 2.9	-4.1 ± 5.1	0.1 ± 3.6	-1.2 ± 4.2	1.32 ; 0.27

[1] Mean ± SEM; n=15
[2] Question phrase: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question phrase: How Full do you feel? Scoring : Not Full at all = 0; Very Full = 100

3. PHYSICAL COMFORT

No treatment [F=0.93 ; p=0.45], time [F=0.8 ;p=0.53] or time by treatment

interaction [F= 1.22 ; p=0.25] was observed for ratings of well being over one hour after

consumption of each treatment (Table 25).

Table 25. Exp III. Physical Comfort[1,2]

Treatment	Time (mins)					
	0	15	30	45	60	F;p
Sucralose	74.07 ± 3.73	71.6± 3.3	72.0± 3.1	72.7± 3.9	69.1± 5.2	0.97; 0.43
Fru/Glu	68.0± 4.8	67.3± 4.8	67.1± 4.9	64.3± 6.2	68.2± 4.6	0.82; 0.51
Sucrose	68.5± 4.5	68.7± 4.1	72.5± 3.0	68.3± 3.5	71.2± 3.9	1.04; 0.39
Polycose	74.1± 2.9	69.4± 3.9	70.9± 3.8	70.0± 4.1	69.0± 4.0	1.72; 0.16
Glucose	73.6± 3.1	70.8± 4.0	75.2± 3.0	72.9± 3.2	74.9± 3.4	1.14; 0.3

[1] Mean ± SEM; n=15

[2] Question: How well do you feel? Scoring: Not well at all = 100; Very well = 100

4. PALATABILITY

There was no significant differences between treatments for palatability ratings

for the test meal [F=1.91 ; p=0.12].

The palatability of the preloads were rated significantly different among the

treatments [F= 5.79 ; p=0.0006]. The fructose/glucose treatment was rated more palatable

than sucralose. Fructose, glucose and sucrose were rated more palatable than polycose

(Table 27).

Table 26. Experiment III Palatability Ratings of Beverage and Pizza after each
Treatment[1,2]

Treatment	Beverage	Pizza
Sucralose	38.8 ± 6.1^{bc}	77.4 ± 3.6
Fru/Glu	54.9 ± 6.3^{a}	76.4 ± 3.0
Sucrose	50.7 ± 6.3^{ab}	76.4 ± 4.0
Polycose	28.6 ± 5.1^{c}	78.7 ± 3.0
Glucose	52.3 ± 6.1^{ab}	71.0 ± 4.9
F	5.79	1.91
p	0.0006	0.12

[1] Mean ± SEM (mm); n = 15
[2] Question: How pleasant have you found the beverage or food?
  Scoring: Very Pleasant = 0; Not pleasant at all = 100
[a] Means with different superscripts within a column are significantly different; $p<0.05$

5. PERCEIVED SWEETNESS

There was an effect of treatment [F=5.54 ; p=0.0008] on the subjective ratings of

sweetness (Table 27). Sucralose was rated as less sweet compared to all other treatments.

Table 27. Experiment III Sweetness Ratings of Beverage Preloads[1,2]

Treatment	Perceived Sweetness
Sucralose	64.1 ± 4.1[b]
Fru/Glu	77.5 ± 3.2[a]
Sucrose	77.7 ± 3.7[a]
Polycose	72.2 ± 3.9[a]
Glucose	79.8 ± 3.2[a]
F	5.54
P	0.0008

[1] Mean ± SEM (mm); n = 15
[2] Question: How sweet have you found the beverage?
  Scoring: Not sweet at all = 0; Extremely sweet = 100
[a] Means with different superscripts within a column are significantly different

B. DISCUSSION

A significant effect of treatment was observed on mealtime energy intake one hour after consumption of glucose, polycose, sucrose, fructose/glucose and sucralose preloads. Glucose and sucrose decreased food intake compared to sucralose, whereas energy intake after polycose and the fructose/glucose preload was not different from control.

The decrease in energy intake observed with glucose and sucrose is consistent with other studies in the literature. Large (>50g) carbohydrate preloads of glucose and sucrose are shown to suppress food intake 1 to 1.5 hours post preload (Green & Blundell 1996; Rogers & Blundell 1989; Rogers & Carlyle et al 1988; Woodend 2000). Although the suppression of food intake following sucrose consumption in young adults appears to be dose dependent at much lower doses, the dose of 75g was chosen for these experiments because it has been previously shown to decrease short term food intake compared to a sweet control containing sucralose (Woodend 2000).

The lack of effect of the high fructose treatment may result in part from the fact that food intake was measured at one hour. Previous reports have demonstrated that fructose (~50g) decreases energy intake when measured at 1.5 to 2.5 hours (Spitzer & Rodin 1987; Rodin et al 1988 ; Rodin 1991), however there is a strong possibility that the lack of effect of the fructose/glucose mixture was associated with its low glycemic response.

The results support an inverse relationship between the glycemic response to the carbohydrate treatments and meal time energy intake at one hour. The high glycemic preloads, sucrose and glucose decreased food intake, whereas the low glycemic

fructose/glucose preload did not suppress energy intake. Indeed, only 11.5% compensation for the calories derived from the preload was observed with the low glycemic fructose/glucose preload compared to the 36%-40% compensation observed with the high glycemic carbohydrates, polycose, glucose and sucrose treatments.

Although the high glycemic treatment, polycose did not decrease energy intake in the present experiment, the strong suppressive effect observed in experiment II combined with the 36% compensation observed in the present experiment suggests that a larger sample size was perhaps required to detect the suppressive effect of polycose.

It is unlikely that energy intake was affected by physical discomfort due to fructose malabsorption as the ratings of physical comfort were not rated differently among treatments. Similarly, no relationship was observed between the palatability or sweetness of the preloads and meal time energy intake (Appendix VI; Table vi).

PART III

THE EFFECT OF CARBOHYDRATE TREATMENTS ON MOOD

A. RESULTS

There was no treatment effect on global vigour at baseline [F=2.28 ; p=0.07], 30 minutes [F=1.88 ; p=0.13] or on the difference between 30 minutes global vigour scores and baseline [F=0.67 ; p=0.62] (Table 28).

There was no treatment effect on global affect at baseline [F=0.61 ; p=0.65], 30 minutes [F=0.77 ; p=0.55] or on the difference between 30 minute global affect ratings and baseline [F=1.3 ; p=0.28] (Table 28).

No treatment effect was observed for the individual mood questions for global vigour and global affect (Table 28).

Table 28. Exp. III Scores on individual Mood Questions[1,2]

Question	Time (min)	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F;p
Global Vigour	0	60.7 ± 4.6	57.3 ± 4.6	59.2 ± 3.5	54.1 ± 4.7	62.2 ± 4.2	1.89; 0.13
	30	64.2 ± 4.3	60.3 ± 3.4	64.0 ± 3.5	61.9 ± 4.0	67.3 ± 3.8	1.83; 0.14
	30-0	3.6 ± 2.7	3.0 ± 3.9	4.9 ± 2.3	7.8 ± 2.4	5.2 ± 2.7	0.56; 0.69
1. Alert	0	64.5 ± 4.2	62.1 ± 4.7	64.1 ± 3.7	55.3 ± 6.2	62.1 ± 4.7	1.00; 0.42
	30	65.3 ± 4.5	58.3 ± 3.8	64.7 ± 3.8	65.5 ± 3.4	62.4 ± 5.2	0.76; 0.56
	30-0	0.7 ± 3.5	-0.4 ± 4.5	0.6 ± 3.6	10.2 ± 5.7	0.3 ± 5.6	1.24; 0.30
2. Sleepy	0	45.5 ± 5.9	51.9 ± 6.4	47.9 ± 4.8	56.5 ± 6.7	47.1 ± 6.0	2.77; 0.4
	30	42.3 ± 5.7	43.4 ± 5.0	41.3 ± 4.4	47.5 ± 5.4	34.9 ± 4.4	3.26; 0.02
	30-0	-3.2 ± 3.4	-8.5 ± 5.0	-6.6 ± 3.5	-9.0 ± 3.2	-12.1 ± 3.9	1.05; 0.39
3. Weary	0	36.2 ± 5.9	41.5 ± 6.0	44.4 ± 4.6	41.1 ± 5.0	33.6 ± 5.5	1.94; 0.12
	30	33.7 ± 5.4	36.7 ± 4.9	32.1 ± 4.9	32.1 ± 4.9	27.5 ± 4.1	2.29; 0.07
	30-0	-2.5 ± 3.8	-4.9 ± 4.4	-12.9 ± 2.9	-9.0 ± 4.1	-6.1 ± 3.1	1.17; 0.33
4. Effort[3]	0	40.2 ± 5.3	36.3 ± 5.2	35.1 ± 4.3	32.7 ± 5.3	32.7 ± 4.7	1.32; 0.27
	30	32.4 ± 5.3	37.1 ± 4.9	35.7 ± 5.5	38.3 ± 4.9	30.6 ± 5.5	1.12; 0.36
	30-0	-7.8 ± 3.9	0.9 ± 5.0	0.5 ± 4.5	-2.9 ± 3.2	-2.1 ± 3.3	0.88; 0.48
Global Affect	0	70.0 ± 4.2	69.2 ± 2.9	66.9 ± 4.4	73.0 ± 3.6	71.3 ± 4.1	0.69; 0.60
	30	72.2 ± 3.6	68.6 ± 3.8	68.3 ± 3.6	69.8 ± 4.2	72.5 ± 3.7	0.72; 0.58
	30-0	2.2 ± 1.7	-0.6 ± 1.5	1.4 ± 3.0	-3.2 ± 2.2	1.2 ± 1.2	1.11; 0.36
5. Sad	0	21.6 ± 4.4	26.5 ± 4.9	26.6 ± 5.8	21.9 ± 4.4	25.3 ± 5.0	0.39; 0.81
	30	22.2 ± 4.2	25.7 ± 4.7	24.2 ± 4.0	23.1 ± 4.3	24.7 ± 4.9	0.24; 0.91
	30-0	0.6 ± 2.3	-0.7 ± 4.7	-2.4 ± 5.1	1.1 ± 3.8	-0.6 ± 2.3	0.14; 0.97
6. Calm	0	64.1 ± 4.8	67.2 ± 4.5	64.6 ± 5.2	68.7 ± 4.8	67.9 ± 4.9	0.30; 0.88
	30	67.2 ± 4.2	64.0 ± 5.3	62.5 ± 5.3	64.7 ± 5.1	73.3 ± 3.6	1.42; 0.24
	30-0	3.1 ± 2.9	-3.2 ± 4.6	-2.1 ± 3.6	-3.9 ± 3.4	5.3 ± 3.2	1.33; 0.27

[1] Mean ± SEM; n=15
[2] Question: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question 4: How much effort is it to do anything? Scoring: Very little = 0; Very much = 100

Table 28. Continued

Question	Time (min)	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F;p
7. Happy	0	67.2 ± 4.4	65.4 ± 3.9	62.9 ± 3.9	67.3 ± 4.2	67.0 ± 4.0	0.45; 0.77
	30	69.3 ± 4.0	67.0 ± 4.1	67.7 ± 2.9	65.8 ± 4.2	68.1 ± 4.3	0.24; 0.91
	30-0	2.1 ± 2.1	1.6 ± 3.3	4.8 ± 3.3	-1.5 ± 1.8	1.1 ± 1.7	0.92; 0.46
8. Tense	0	29.8 ± 5.4	29.5 ± 4.2	33.1 ± 5.6	22.3 ± 3.6	24.5 ± 5.2	1.45; 0.23
	30	25.7 ± 4.0	30.9 ± 5.2	32.7 ± 5.4	28.4 ± 4.7	26.7 ± 4.6	0.80; 0.53
	30-0	-4.1 ± 2.5	1.5 ± 4.1	-0.5 ± 4.4	6.1 ± 3.0	2.2 ± 2.5	1.22; 0.31

[1] Mean ± SEM; n=15
[2] Question: How (adjective) do you feel? Scoring: Very little = 0; Very much = 100
[3] Question 4: How much effort is it to do anything? Scoring: Very little = 0; Very much = 100

B. DISCUSSION

The present results do not support a relationship between carbohydrate

consumption and mood regulation. Consistent with experiment II, no treatment effect

was observed on ratings of global vigour and global affect. Furthermore, the effect of

sucrose on ratings of alertness was not reproduced.

It is possible that the present experimental paradigm was not adequate to detect an

effect on mood for it was designed to measure a treatment effect on the primary outcome,

energy intake. It is suggested that dietary regulation of mood occurs at least 2 hours post

preload (Lieberman et al 1986; Spring et al 1989) coinciding with peak serotonin (Sayegh

et al 1995) and cholecystokinin levels (Wells et al 1997).

Furthermore, the effect of dietary carbohydrates on mood in young, healthy males

is perhaps more subtle than what would be observed in affected populations such as

depressed or obese individuals. The very subjective nature of mood makes it difficult to

quantify and measure. Perhaps, examining the effect of the selected carbohydrate prelaods on mood over an extended period of time will allow the subtle treatment effects to emerge.

PART IV

THE EFFECT OF CARBOHYDRATE TREATMENTS ON MEMORY

A. RESULTS

1. Immediate Recall

The total number of words correctly recalled at 15 minutes was affected by treatment [F=3.4 ; p=0.0148]. Sucrose increased the number of words correctly recalled 15 minutes post preload, compared to fructose/glucose, polycose and the control, sucralose (Table 29).

The number of correct words recalled at the first 15 minute trial was affected by treatment [F=2.53; p=0.05]. Sucrose increased the number of words correctly recalled compared to polycose and fructose/glucose (Table 29). The number of incorrect words recalled at the first 15 minute trial was not affected by treatment [F=0.33 ; p=0.86].

The number of words correctly recalled at the second presentation of the word list was affected by treatment [F=4.21 ; p=0.005]. Sucrose increased the number of words correctly recalled compared to fructose/glucose, sucralose (control) and polycose (Table 29). Glucose increased the number of words correctly recalled compared to polycose. The number of words incorrectly recalled at the second presentation of the word list was not affected by treatment [F=0.48 ; p=0.75].

The number of words correctly recalled at the third presentation of the word list was not affected by treatment [F=2.04 ; p=0.1] (Table 29). The number of words incorrectly recalled was affected by treatment [F=2.57 ; p=0.048]. The fructose/glucose preload increased the number of words incorrectly recalled at the third presentation of the word list compared to sucrose and sucralose.

The total number of words correctly recalled for a word list presented 60 minutes post preload was not affected by treatment [F=0.64 ; p=0.64]. A treatment effect was observed for the number of incorrect words recalled at the first 60 minute trial [F=3.02 ; p=0.03]. Polycose demonstrated the greatest number of incorrect words recalled compared to sucrose, fructose/glucose and control, sucralose (Table 29).

2. Delayed Recall

Delayed recall was affected by treatment [F=2.63 ; p=0.04]. Glucose and sucrose increased the number of words correctly recalled at 45 minutes compared to fructose/glucose (Table 29). The number of words incorrectly recalled at 45 minutes was not affected by treatment (Table 29).

The number of words correctly [F=0.35 ; p=0.85] and incorrectly [F=0.95 ; p=0.44] recalled post test meal was not affected by treatment (Table 29).

3. Trail Making Test

The absolute values for Part A [F=0.27 ; p=0.89] or Part B [F=1.57 ; p=0.19] of the trail making test was not affected by treatment.

Table 29. Experiment III Memory Scores [1]

Time (mins)	Correct/ [2] Incorrect	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F ; p
15-1st Trial	Correct	6.9 ± 0.4^{ab}	6.4 ± 0.4^{a}	7.9 ± 0.5^{b}	6.2 ± 0.6^{a}	7.1 ± 0.6^{ab}	2.53;0.05
	Incorrect	0.1 ± 0.1	0.1 ± 0.1	0.2 ± 0.1	0.3 ± 0.1	0.1 ± 0.1	0.33;0.86
15-2nd Trial	Correct	9.5 ± 0.5^{ab}	10.1 ± 0.8^{ab}	11.7 ± 0.6^{c}	9.3 ± 0.7^{a}	10.9 ± 0.5^{bc}	4.2;0.0048
	Incorrect	0.0 ± 0.0	0.07 ± 0.07	0.07 ± 0.07	0.09 ± 0.13	0.07 ± 0.07	0.48; 0.75
15-3rd Trial	Correct	11.8 ± 0.6	11.7 ± 0.7	13.3 ± 0.6	12.2 ± 0.7	12.5 ± 0.8	2.04;0.10
	Incorrect	0.0 ± 0.0^{a}	0.33 ± 0.2^{b}	0.0 ± 0.0^{a}	0.2 ± 0.1^{ab}	0.1 ± 0.1^{ab}	2.57;0.05
Total 15 Score	Correct	28.1 ± 1.4^{a}	28.1 ± 1.8^{a}	32.9 ± 1.6^{b}	27.7 ± 1.8^{a}	30.5 ± 1.8^{ab}	3.40;0.01
	Incorrect	0.09 ± 0.13	0.21 ± 0.6	0.14 ± 0.2	0.19 ± 0.6	0.12 ± 0.3	2.43;0.059
45 min Score	Correct	8.3 ± 0.8^{ab}	7.3 ± 0.9^{a}	9.2 ± 1.0^{b}	7.6 ± 0.8^{ab}	9.3 ± 1.1^{b}	2.36;0.04
	Incorrect	0.1 ± 0.1	0.13 ± 0.1	0.1 ± 0.1	0.2 ± 0.1	0.1 ± 0.1	0.65;0.63
60-1st Trial	Correct	6.9 ± 0.3	6.3 ± 0.6	6.7 ± 0.3	6.3 ± 0.6	6.7 ± 0.8	0.61;0.66
	Incorrect	0.0 ± 0.0^{a}	0.0 ± 0.0^{a}	0.1 ± 0.1^{a}	0.4 ± 0.2^{b}	0.1 ± 0.1^{ab}	3.02;0.03
60-2nd Trial	Correct	0.8 ± 10.9	10.9 ± 0.7	10.5 ± 0.7	10.3 ± 0.9	10.1 ± 0.9	0.44;0.77
	Incorrect	0.1 ± 0.1	0.1 ± 0.1	0.0 ± 0.0	1.0 ± 1.0	0.1 ± 0.1	0.94;0.45
60-3rd Trial	Correct	13.7 ± 0.8	12.8 ± 0.7	13.1 ± 0.8	12.3 ± 0.9	12.9 ± 0.9	0.75;0.56
	Incorrect	0.1 ± 0.1	0.1 ± 0.1	0.0 ± 0.0	0.1 ± 0.1	0.1 ± 0.1	0.68;0.61
Total 60 Score	Correct	31.5 ± 1.8	30.1 ± 1.8	30.3 ± 1.8	28.8 ± 2.1	29.7 ± 2.3	0.64;0.64
	Incorrect	0.09 ± 0.13	0.12 ± 0.27	0.11 ± 0.2	0.21 ± 0.4	0.07 ± 0.07	1.06 ; 0.38
Post Meal [3]	Correct	9.1 ± 0.9	8.3 ± 0.9	7.9 ± 0.8	8.7 ± 1.1	8.3 ± 1.1	0.35 ; 0.84
	Incorrect	0.4 ± 0.3	0.1 ± 0.1	0.0 ± 0.0	0.2 ± 0.1	0.3 ± 0.2	0.95 ; 0.44

[1] Mean ±SEM (number of words recalled); n = 15
[2] Correct : Number of words correctly recalled from word list
Incorrect: Number of words recalled not present in word list
[3] Number of words recalled from 60 minute word list after consumption of pizza meal

Table 29. Continued

Time (mins)	Correct/ [2] Incorrect	Sucralose	Fru/Glu	Sucrose	Polycose	Glucose	F ; p
60 – 15 [4]	Correct	3.4 ± 1.7	1.9 ± 1.3	-2.6 ± 2.1	1.3 ± 1.5	-0.8 ± 1.5	2.13;0.09
15 – 45 [5]	Correct	19.9 ± 0.9	20.9 ± 1.4	23.7 ± 1.1	20.1 ± 1.5	21.2 ± 0.8	2.0 ; 0.10
15 3 – 45 [6]	Correct	3.5 ± 0.4	4.4 ± 0.7	4.1 ± 0.7	4.6 ± 0.6	3.2 ± 0.5	1.13 ; 0.35
Trail Making	Part B	51.6±2.7	50.9±4.3	56.2±3.9	48.4±4.1	55.1±4.8	1.57;0.19
Trail Making	Part A	44.7±3.9	44.1±3.1	41.1±2.5	41.8±2.7	43.0±4.8	0.27;0.89

[1]Mean ±SEM (number of words recalled); n = 15

[2] Correct : Number of words correctly recalled from word list

Incorrect: Number of words recalled not present in word list

[3] Number of words recalled from 60 minute word list after consumption of pizza meal

[4] Total no. of words recalled at 60 mins compared to the total no. words recalled at 15 mins

[5] Total no. words recalled at 15 mins compared to the no. words recalled at 45 mins

[6] No. words recalled at 15-3rd trial compared to the no. words recalled at 45 mins

B. DISCUSSION

Consistent with experiment two, the present results support a beneficial effect of sucrose on memory. Sucrose significantly improved immediate recall for a word list at 15 minutes compared to the fructose/glucose, polycose and control treatment. A significant effect of glucose and sucrose was also observed on delayed recall of the word list at 45 minutes.

This is one of the first studies to systematically examine the effect of pure carbohydrate preloads other than glucose on memory in young adults. The results suggest that 75g sucrose improves memory in young adults independent of its constituent monosaccharides, glucose and fructose. The beneficial effect of sucrose on memory observed in the present study is consistent with the results from experiment two and with preliminary results in our lab (Hui 1998 unpublished). However the results are not always consistent among investigators, for a similar preload paradigm failed to observe an effect of a range of sucrose (25g, 50g and 75g) doses on memory in young adults (Woodend 2000).

The results suggest that the ratio of glucose and fructose within a treatment is of some importance. For example, an equal ratio of glucose: fructose in the form of sucrose appears to be optimum for improving immediate recall in young adults, whereas a high or low glucose treatment fails to enhance performance consistent with an inverted U shaped dose response curve (Gold 1986). Future studies examining the effect of preloads with various fructose:glucose ratios may allow a greater understanding of the role of sucrose and its monosaccharides on memory in young adults.

PART V

RELATIONSHIPS BETWEEN BLOOD GLUCOSE, APPETITE, FOOD INTAKE, MOOD AND MEMORY

The relationships between the glycemic response to the five carbohydrate treatments and their effects on subjective appetite, short term food intake, mood and memory are tabulated in Appendix VI. Statistically significant relationships between the dependent variables are presented below.

A. RESULTS

1. Food Intake and Average Appetite

Subjective average appetite at 60 minutes and short term food intake were positively associated [r=0.45 ; p<0.0001] (Figure 11; Appendix VI ; Table vi).

2. Food Intake, Average Appetite and Mood

Food intake was positively associated with increased ratings of alertness from baseline [r=0.25 ; p=0.029] and decreased ratings of calm from baseline [r=0.248 ; p=0.033] (Appendix VI ; Table xv). Food intake was negatively associated with ratings of tense at 30 minutes [r=0.28 ; 0.015] (Appendix VI ; Table xii).

Average appetite at sixty minutes and ratings of weary at 30 minutes showed a positive relaitonship[r=0.269 ; p=0.02] (Appendix VI ; Table xiii).

3. Mood and Memory

Memory at 15 minutes was positively associated with a decrease in sadness from baseline [r=0.229 ; 0.049] (Appendix VI ; Table xvii). A negative relationship was

found for memory at 15 minutes and increased ratings of happiness [r=-0.277 ; p=0.017] from baseline (Appendix VI ; Table xvii).

Increased ratings of alertness was negatively associated with memory at 60 minutes [r=-0.3 ; p=0.008] and total 15 and 60 minute memory score [r=-0.27 ; 0.019] (Appendix VI ; Table xvii).

4. Blood Glucose, Food Intake and Average Appetite

Average appetite [r=-0.23 ; p=0.045] and food intake [r=-0.24 ; p=0.04] were negatively associated with AUC blood glucose concentrations (Figure 12). Similarly, an inverse relationship was found between blood glucose concentration at 37 minutes [r =-0.24 ; 0.046] (Figure 13) and 67 minutes [r=-0.22 ; 0.06] and meal time energy intake (Appendix VI ; Table viii).

5. Blood Glucose and Memory

No relationships were observed between absolute, rising or falling blood glucose concentrations and immediate and delayed recall for a word list (Appendix VI; Table ix).

6. Blood Glucose and Mood

Baseline blood glucose values were positively associated with baseline [r=0.258 ; p=0.027] and 30 minute [r=0.294 ; 0.012] Sad ratings (Appendix VI; Table x).

Similarly, baseline blood glucose values were positively associated with baseline [r=0.258 ; p=0.027] and 30 minute [0.294 ; 0.012] Tense ratings (Appendix VI; Table xi).
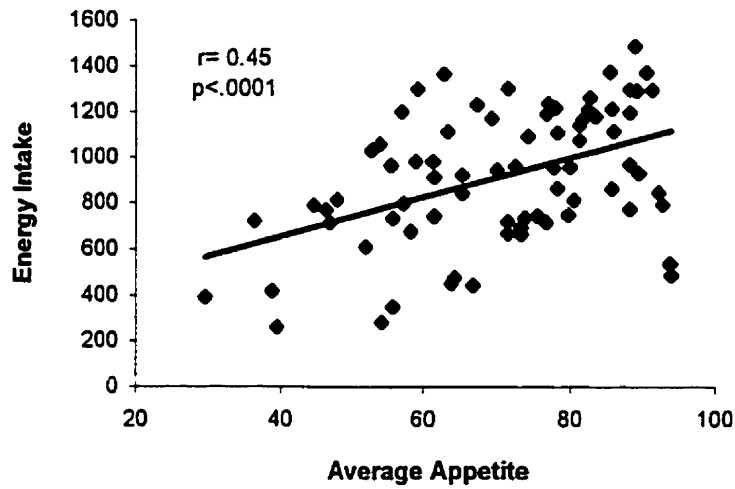
Figure 11. Relationship between 60 min Average Appetite and Food Intake after five treatments
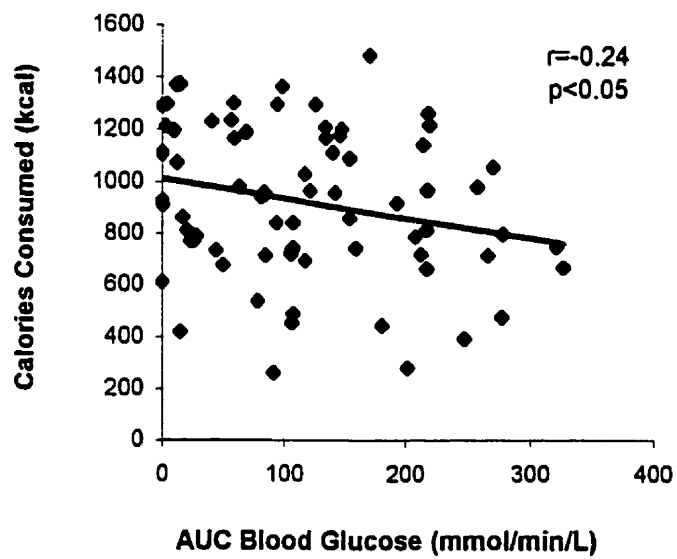
Figure 12. Relationship between AUC blood glucose and Food Intake after five treatments
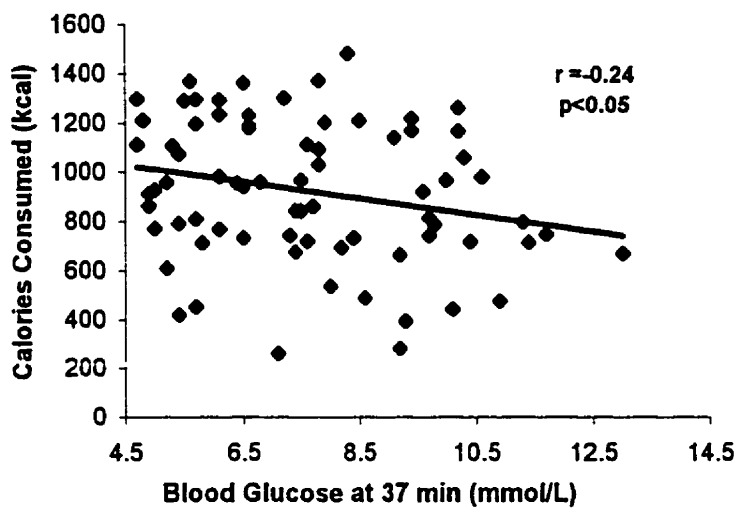
Figure 13. Relationship between Blood Glucose concentrations at 37 minutes and Food Intake after five treatments

B. DISCUSSION

This experiment demonstrated that the effect of the selected carbohydrates on blood glucose, average appetite, food intake, mood and memory are interrelated.

The greater the response in blood glucose after the carbohydrates, the less food intake consumed at a test meal at one hour. A high AUC for blood glucose was associated with decreased average appetite and mealtime energy intake. As well, blood glucose concentration at 37 minutes was associated with decreased mealtime food intake.

Consistent with experiment two, a positive relationship was found between sixty minute average appetite and mealtime energy intake. Relationships were also found between average appetite, food intake and mood. Increased ratings of alertness and decreased ratings of calm, resulted in greater mealtime energy intake, suggesting that the more vigorous subjects felt, the more food they consumed.

It would seem logical that a fasting state or hypoglycemia would naturally decrease mood, so the finding that high baseline ratings of blood glucose were positively associated with a negative mood state such as increased ratings of tense and sadness is unclear. Perhaps those subjects who felt more tense had increased circulating levels of adrenaline, which would result in increased blood glucose concentrations.

No relationships were observed between absolute, rising or falling blood glucose concentrations and memory performance over one hour, suggesting that the beneficial effect of sucrose on memory was unrelated to blood glucose dynamics.

In summary, the results suggest that the mechanisms by which the selected carbohydrates regulated appetite and food intake, but not mood and memory were associated with their glycemic response.

V. GENERAL DISCUSSION

The results of this study support the hypothesis that the glycemic response to the selected carbohydrates is associated with their effect on appetite and energy intake at one hour. The hypothesis that the glycemic response to the carbohydrates is associated with their effect on mood and memory was not supported.

The selected carbohydrate treatments resulted in a wide range of blood glucose responses. A high AUC for blood glucose was associated with decreased average appetite and mealtime energy intake at one hour. No relationship was observed between the glycemic response to the carbohydrate treatments and their effects on mood or memory. However, a beneficial effect of sucrose was found on memory performance. The results of this study and the conclusions are dependent on the time frame of the measurements. It is evident that measurement over one hour is optimal to detect a suppressive effect of high glycemic carbohydrates on appetite and food intake. A longer time frame is perhaps required to observe a treatment effect on mood or a suppressive effect of low glycemic carbohydrates on appetite and energy intake.

The relationship among the glycemic response to the carbohydrates and appetite, food intake, mood and memory is discussed in sequence. Firstly, the relationship between postprandial blood glucose and appetite and food intake is discussed, this is followed by a discussion of the relationship between the glycemic response to the carbohydrates and mood and memory. Finally, the interrelationships among the glycemic response to the the carbohydrate treatments and their effect on appetite, food intake, mood and memory is discussed.

PART A : RELATIONSHIPS AMONG CARBOHYDRATE COMPOSITION, GLYCEMIC RESPONSE AND SUBJECTIVE APPETITE AND SHORT TERM FOOD INTAKE

The results of these three experiments support the hypothesis that the effect of the carbohydrates on short term food intake can be predicted from their effect on blood glucose. Specifically, the greater the elevation in blood glucose after the carbohydrates, the more food intake is reduced 60 minutes later.

A range of glycemic responses was observed following consumption of 75g preloads of selected carbohydrates. The high glycemic carbohydrates, sucrose, polycose and glucose decreased energy intake at one hour. Although the suppressive effect of polycose observed in experiment II was not repeated in experiment III, a negative correlation was found between postprandial blood glucose and appetite and short term food intake in young, healthy males (Figure 12).

The suppression of food intake one hour after a 300 kcal (75g) preload of sucrose and glucose is consistent with other studies reported in the literature. Glucose and sucrose (≥50g) suppress short-term food intake 1 to 1.5 hours post preload (Rogers & Blundell 1989; Rogers & J. Carlyle et al 1988; Green & Blundell 1996; Woodend 2000).

Measurement of food intake at one hour appears to be appropriate for detecting the effects of 75g of sucrose and glucose on satiety, but this interval may not be suitable for providing a picture of the effect of consumption of all carbohydrates on food intake.

For example no suppression of food intake was observed following the 300kcal preloads of fructose/glucose, amylose and amylopectin.

It is possible that our ability to detect an effect of amylose and amylopectin on energy intake was compromised by measurement at one hour. The rate at which a meal moves down the small intestine and the subsequent length of its intestinal exposure influences nutrient receptors in the ileum. Nutrients are likely to alter visceral sensations by releasing chemical transmitters from enteroendocrine cells in the small intestinal epithelium (Read et al 1994). These transmitters may then influence perception by circulating in the blood as hormones or by stimulating afferent nerves. In recent years, the role of GLP-1 has received considerable attention as a putative satiety peptide involved in regulating carbohydrate induced satiety (Gutzwiller et al 1999). Therefore, slower digestion of amylose and amylopectin may increase satiety not through a lower glycemic response as previously thought, but through prolonged stimulation of satiety peptides.

There are several possible explanations for the failure of the fructose/glucose mixture to affect short term energy intake in experiment three. These include the effect of fructose on plasma glucose and insulin, absorption in the intestinal tract, tissue utilisation and liver metabolism.

Because of the differential absorption rates of glucose and fructose, the effect of the two monosaccharides on satiety may be time-dependent. Glucose is absorbed via the sodium-dependent SGLT-1 and GLUT-2 transporters, and fructose is absorbed via a less well characterised sodium independent transport system (Levin 1994). Following absorption, fructose is transported in the portal circulation to the liver and metabolised to glucose, glycogen, lactate and triglycerides (Chandramouli et al 1993), which may individually influence satiety. Therefore, a duration exceeding one hour may be required to observe the satiating capacity of fructose. Indeed, some studies show 50g fructose to

have little effect on food intake when administered in a preload 60 min before a test meal

(Lambert 1991, Guss et al 1994). However, when the time between preload and test meal

is extended to 1.5 to 2.25 hours, fructose preloads have been reported to suppress food

intake (Blundell et al 1994, Rayner et al 2000, Spitzer & Rodin 1987, Rodin et al 1988,

Rodin 1991). The slower expression of the effect of fructose on energy intake in

comparison to other carbohydrates may also reflect its delayed effect on thermogenesis

and oxidation (Schwarz et al 1989, Biernacka 1995).

It is also possible that the weak satiety effect observed with the fructose/glucose

preload in experiment three is due to the addition of glucose. A mixture of fructose and

glucose was given because approximately 50% of the population are limited in their

absorptive capacity of fructose, presenting with symptoms of nausea and diarrhea with as

little as 25g of fructose (Rumessen & Gudmand-Hoyer 1986, Henry & Capo 1991).

Glucose administration dose dependently increases the uptake of fructose thereby

decreasing the extent of malabsorption (Truswell et al 1992). For this reason, 20%

glucose was added to the fructose preload. However, previous studies have shown that

the satiating effect of 50g fructose at a meal 2.25 hours post consumption disappears once

an equal source of glucose in the form of starch is added (Rodin 1991). However, this is

unlikely for other studies have also failed to demonstrate an effect of fructose on energy

intake (Guss et al 1994; Kissileff et al 1989).

It is therefore possible that the lack of effect of the fructose/glucose mixture on

food intake at one hour was a reflection of the low glycemic response.

The glucostatic hypothesis states that a rise in blood glucose concentrations

signals satiety and the termination of feeding (Mayer 1953). The results from the three

experiments are consistent with this hypothesised relationship between glycemic response and satiety. The greater the response in blood glucose after sucrose, glucose and polycose, the less energy consumed at a test meal at one hour. The blood glucose concentration at 37 minutes postprandial was most strongly associated with decreased mealtime energy intake. It is possible that blood glucose acts as a direct signal to feeding centres in the hypothalamus to signal satiety or mediates satiety through peripheral signals such as circulating hormones and gut peptides.

Fluctuations in the blood glucose concentrations may be seen as a primary indication of the availability of glucose to nervous tissue and glucose utilisation by brain cells (Mayer 1953). This is supported by previous studies which have found that acute hyperglycemia (>10mmol/L) increases satiety and decreases food intake (Gielkens et al 1998 ; Lavin et al 1998; Chapman 1998). In addition to brain cellular energy utilisation serving as an indicator of glucose availability, it has been proposed that glucose availability in the liver influences glucoreceptors, which provide signals to the brain via the vagus nerve (Russek 1970).

In view of the close linkage between the blood glucose released following carbohydrate consumption and the subsequent release of insulin, the role of insulin as a proposed mediator of satiety is possible. A number of studies have observed enhanced satiety when modest and sustained increases in insulin have been experimentally achieved (VanderWeele et al 1985, 1994, Woods et al 1996). Similarly, insulin is implicated in the suppressive effect of fructose on energy intake (Rodin et al 1988; 1991) however, there is still some debate as to whether insulin released following mixed meals

signals increased (Holt et al 1996) or decreased satiety (Holt & Brand Miller 1994; 1995).

This is one of the first studies demonstrating an inverse relationship between the glycemic response to pure nutrients and energy intake at one hour. The results conflict with the majority of earlier studies, which support a relationship between a low glycemic response to high carbohydrate meals and increased satiety. Important distinctions are apparent between the present study design and previous investigations that may account for this discrepancy. For example, an important determinant in measuring the satiating capacity of a preload is the time interval between preload and test meal. Because a cascade of satiety signals are produced upon ingestion of food (Blundell et al 1994), measurement of satiety at one hour will reflect different satiety signals from measurement at 2 or 4 hours. Indeed, the majority of previous studies have employed a time frame of 2 to 6 hours between preload and test meal (Leathwood & Pollet 1988; Holt et al 1992; van Amelsvoort & Westrate 1992; Holt & BrandMiller 1995). It is therefore possible that the lack of effect of amylose and amylopectin on food intake in the present study was a result of the short time frame of 1 hour, between preload and test meal. It is shown that high amylose meals which are slowly digested, lead to greater satiety 2 to 6 hours postprandial compared to low amylose meals (Granfeldt et al 1994; van Amelsvoort & Westrate 1992). Because raw amylose and amylopectin were employed in experiment II, the effect on satiety may not have begun to occur within the first hour due to slow transit from the stomach (Mourot et al 1988).

Of the many investigations that have found no relationship or an association between high blood glucose and decreased satiety (Ludwig 2000), a large majority of

them have failed to control for fibre, energy content and the presence of other satiating nutrients (Krishnamachar & Mickelson 1987; Raben et al 1994; Holt & Brand Miller 1994; Barkeling et al 1995; Holt et al 1996; Stewart et al 1997). In conjunction with a lack of uniformity in study designs including the time frame, endpoints (subjective satiety or food intake), subject population and extent of processing, it is unreasonable to assume that the relationship between blood glucose and satiety was adequately addressed in these studies. Indeed, the results of the present study strongly support the validity of the preload paradigm and the use of pure carbohydrates when investigating the satiating capacity of nutrients.

Conclusion

A significant relationship was observed between the glycemic response to the carbohydrates and short term food intake at one hour. The carbohydrate treatments producing the greatest postprandial increase in blood glucose decreased mealtime energy intake at one hour. Further studies are required to examine the relationship between glycemia and food intake regulation over extended time intervals.

PART B: RELATIONSHIPS AMONG THE GLYCEMIC RESPONSE TO CARBOHYDRATES AND MOOD

The results of the present study did not consistently demonstrate an effect of carbohydrates on mood and did not support the hypothesis that the effect of carbohydrates on mood is defined by their glycemic response.

In experiment two and three, no consistent effect was found between carbohydrate ingestion and subjective measures of global vigour and global affect over one hour. However, in experiment II it was found that 300 kcal preloads of sucrose, amylose and amylopectin increased ratings of alertness compared to control, 30 minutes post preload. Although attempts were made to minimise any interaction between mood ratings and finger prick blood glucose measures in experiment III, it is possible that the subjects' expectancy of blood glucose measures occluded the reproducibility of a treatment effect on ratings of alertness.

The lack of reproducibility between sucrose consumption and increased ratings of alertness between experiment II and III, does not provide strong support for a role of sucrose in mood regulation. Several other studies have examined the effect of sucrose preloads on mood over one hour with inconsistent results. Consumption of 40g sucrose was found to have no effect on mood 30 and 60 minutes post consumption (Reid & Hammersley 1995). Similar preload paradigms utilising a range of sucrose doses (25g, 50g and 75g) have also failed to observe an effect on mood in young, healthy males over one hour (Hui 1998, Woodend 2000).

Because of the inconsistent findings observed between experiment II and III on only one component of mood at one time point, the theory that carbohydrates regulate

mood cannot be confirmed. However, it is important to acknowledge the role of the glycemic response in mood and the possible mechanisms by amylose and amylopectin may have sustained alertness.

It is possible that the slower digestion and absorption of amylose and amylopectin induced strong and sustained stimulation of post-absorptive factors, such as gut peptides, insulin and neurotransmitters associated with mood regulation. For example, a recent study found that consumption of a high fibre, carbohydrate rich meal was associated with higher post meal alertness ratings compared to a fat rich, low fibre breakfast (Holt et al 1999). It is possible that the low glycemic response observed with these treatments mediates an increase in alertness. However, previous studies have found no relationship between postprandial blood glucose concentrations and mood (Wells et al 1997; Spring et al 1989). For example, no relationship was observed between glycemia and mood, 20 minutes and 4 hours following consumption of 100g sucrose (Brody & Wolitzky 1983). The increased ratings of fatigue observed in young females following ingestion of high carbohydrate lunch bars, was not attributed to hypoglycemia for blood glucose levels remained elevated (Spring et al 1986).

The low glycemic and subsequent insulin response observed with amylose and amylopectin may reflect decreased serotonin synthesis, which may alleviate feelings of sleepiness thereby increasing alertness. The ingestion of carbohydrate rich foods releases insulin and increases the availability of tryptophan, enhancing brain uptake and saturating the enzyme converting tryptophan to serotonin. The subsequent increase in serotonin synthesis is thought to combat negative feelings and induce satiety and fatigue (Spring et al 1989). Those carbohydrates demonstrating a high glycemic response, e.g polycose,

would perhaps result in increased serotonin synthesis and decreased alertness. Because the effect of polycose was not different from the low glycemic control, sucralose, a role for blood glucose or insulin in modulating mood is unlikely. Indeed, the relationship between plasma glucose and insulin levels at 30 minutes, may be too short a time interval to capture the effects of carbohydrate consumption on tryptophan availability and serotonin synthesis. It has been suggested that food induced mood alterations occur at least 2 hours post preload (Lieberman et al 1986; Spring et al 1989) coinciding with peak serotonin (Sayegh et al 1995), tryptophan (Spring et al 1989) and cholecystokinin levels (CCK) (Wells & Read 1996; Wells et al 1997).

Overall, the inability to detect a treatment effect on mood in experiment three, may have been a result of experimental interference or time restrictions. Mood ratings were obtained prior to blood sampling, however the overriding effect of each subjects' expectancy cannot be ruled out. Furthermore, the treatments employed in experiment three were not equivalent to the carbohydrate treatments in experiment one and two. By eliminating the carbohydrates that demonstrated the greatest effect on alertness, i.e amylose and amylopectin, the treatment effect was possibly removed. However, because the effect of sucrose on only one component of mood, alertness was not reproduced in experiment III, it is not accurate to say that the present study supported a role for carbohydrates in regulating mood over one hour. Because the present studies were designed based on the primary endpoint, food intake, the effect of the selected carbohydrates on mood was perhaps confined. Future studies may benefit by focusing on the effect of the selected carbohydrates on mood over an extended period of time.

PART C: RELATIONSHIPS AMONG THE GLYCEMIC RESPONSE TO CARBOHYDRATES AND MEMORY

The results of these experiments support a beneficial effect of sucrose on memory. However, the results do not support a relationship between the glycemic response to carbohydrates and memory enhancement.

This is one of the first studies to examine the effect of pure carbohydrate preloads other than glucose on memory in young adults. Experiment two demonstrated a positive effect of 300kcal sucrose on memory for a word list, 15 and 60 minutes post consumption. Experiment three confirmed the positive effect of a 300kcal sucrose preload on immediate recall for a word list at 15 minutes. Preloads of polycose and fructose/glucose were associated with a decline in performance over one hour.

No relationship was observed between the glycemic response to carbohydrate treatments and memory in young adults over one hour. For example, glucose and polycose demonstrated the greatest area under the curve blood glucose values, with peak blood glucose concentrations >7.2mmol/L. Not only was there no significant improvement in memory recall with these carbohydrate treatments over one hour, polycose treatment resulted in a greater number of words incorrectly recalled 65 minutes later. Similarly, no relationship was observed between rising or falling blood glucose levels and cognitive performance over one hour (Appendix VI; Table ix). Previous studies have reported a beneficial effect of glucose on memory based on the hypothesis that higher blood glucose levels allow for greater passage of glucose into the brain to fuel memory processes (Donohoe & Benton 1999, Benton & Owens 1993, Benton et al 1994).

Lists of words or a story are more easily learned with high (>7.2 mmol/L) rather then low (<4.4 mmol/L) blood glucose concentrations (Lapp 1981, Hall et al 1989). In contrast, the present results demonstrated that high blood glucose levels >8mmol/L observed with polycose and glucose did not result in a beneficial effect on memory.

The effect of glucose on memory appears to be dose dependent. Several studies have suggested that an optimum dose of approximately 50g may be necessary to observe a positive effect of glucose on memory in young adults, with lower or higher doses producing a decline in performance (Gonder-Frederick et al 1987, Hall et al 1989). It is possible that the lack of effect of a 75g preload of glucose on memory in both experiment two and three was because it was higher than the optimum dose. Similarly, preloads of 30g and 100g glucose failed to improve memory at 30 minutes in young adults (Azari 1991).

The positive effect of 75g (300kcal) sucrose on immediate recall in young adults observed in the present study supports preliminary evidence in our laboratory whereby consumption of 300kcal sucrose was observed to prevent a decline in memory for word lists between 15 and 60 minutes post consumption (Hui 1998 unpublished).

It was somewhat surprising to find that the high fructose treatment failed to enhance memory, but this may have been due to the presence of glucose. A dose-dependent enhancement of memory for a passive avoidance test in rats was found upon administration of 1000mg/kg and 2000mg/kg of either glucose or fructose. However, a combined 1000mg/kg glucose plus 1000mg/kg fructose dose failed to improve memory (Rodriguez et al 1999). Why the presence of both fructose and glucose decreases the memory enhancing effects of each monosaccharide alone is unclear. It is possible that

memory modulation is species specific. Further examination of the effect of dose and timing of fructose, glucose and sucrose preloads on memory in rats and humans may aid in understanding the results found in the present study.

It is possible that optimal insulin stimulation in the face of high sucrose preloads may explain the memory enhancement observed in experiment two and three. The insulinemic response to sucrose is greater than would be expected from its glycemic response due to the presence of the fructose moiety. However, a positive role of insulin is unlikely considering that the insulinemic response to 100g of sucrose is significantly lower than 100g glucose (Lee & Wolever 1998).

Central behaviour such as memory may be regulated through a number of mechanisms including the release of peptide hormones after carbohydrate ingestion. They may pass directly through the blood brain barrier, produce a secondary effect through pituitary hormone release, alter circulating metabolites such as glucose and free fat or alter blood flow directly to the brain (Morley 1986; Morley & Flood 1992). Peripherally administered or released substances that modulate memory storage, but do not freely enter the brain, may activate peripheral receptors that send messages centrally through the vagus nerve. Vagal afferents as compared to vagal efferents are shown to carry messages about the peripheral states that lead to the modulation of memory (Clark et al 1998). For example, vagus nerve stimulation administered after learning in human subjects significantly enhances retention (Clark et al 1999).

In summary, a beneficial effect of sucrose was found on memory performance in young adults, however opposite to the literature, preloads of glucose had no effect on memory. A review of the literature suggests that a beneficial effect of glucose on

memory is more apparent in the elderly (Hall et al 1989, Messier et al 1997, Craft et al 1994), or those with pre-existing memory deficits and poor blood glucose regulation (Messier et al 1999). Young adults have a high baseline performance on low-level cognitive tests, which may interfere with the beneficial effects of glucose on memory (Korol & Gold 1998). A more difficult task including a longer time frame between immediate and delayed recall may be necessary to detect an effect on cognitive processes in the young. It is also possible that sucrose ingestion may have a stronger effect in the elderly and should be tested.

Conclusion

The lack of a relationship between the glycemic response to the selected carbohydrate treatments and memory performance suggests that a mechanism other than blood glucose may explain the beneficial effect of sucrose on memory. Future studies examining the role of a range of sucrose doses and its monosaccharides, glucose and fructose in memory in young adults may provide a clearer picture of the mechanisms by which sucrose may improve performance.

PART D: RELATIONSHIPS AMONG THE GLYCEMIC RESPONSE TO CARBOHYDRATES AND APPETITE, FOOD INTAKE, MOOD AND MEMORY

The role of blood glucose in the modulation of appetite, food intake, mood and memory has been discussed in detail for each individual parameter. However, the considerable overlap between the mechanisms suggested to govern these behaviours, implies that changes in one variable do not occur independently of the others. For example, it is suggested than an increase in serotonin synthesis decreases vigour (Wurtman et al 1989) and energy intake (Leibowitz & Alexander 1998). Similarly, an increase in CCK is proposed to decrease vigour (Fara et al 1969), reduce energy intake (Blundell 1991) and enhance memory (Dauge & Lena 1998).

The present evidence supports the presence of interrelationships among the effect of carbohydrate ingestion on appetite, food intake, mood and memory. For example, appetite immediately prior to the test meal was a significant factor in determining the amount of calories ingested at that meal (Figure 12). Furthermore, an increase in sleepiness, and weariness was associated with a decrease in appetite in both experiment two and three (Appendix VI; Table iii & xii). The positive association between alertness and food intake in experiment three, suggests that the more vigorous subjects feel, the greater their appetite and energy intake (Appendix VI ; Table xv).

A significant amount of interaction was observed between high blood glucose levels and decreased average appetite and food intake (Appendix VI ; Table viii). Furthermore, baseline blood glucose ratings were positively associated with feelings of sad and tense (Appendix VI; Table x & xi). The evidence infers that low baseline blood

levels are associated with lower ratings of sadness and tenseness. Whether these relationships hint at an association between blood glucose regulation and or circadian serotonin levels is unknown.

Perhaps the rate of contact of nutrients with gut peptides and the stimulation of circulating hormones such as insulin are the essential peripheral stimuli to the central nervous system for regulation of mood, memory and energy intake. The glycemic response may only serve to depict the absorption characteristics required to stimulate specific satiety signals within certain time intervals. For example, rapidly digestible carbohydrates such as sucrose, polycose and glucose may stimulate insulin and gut peptides to a greater extent within one hour thereby suppressing energy intake through the vagus nerve and decreasing alertness through increased serotonin synthesis. Decreased insulin levels observed with the slowly digestible treatments would have less influence on energy intake at one hour but increase alertness through decreased serotonin synthesis. However, because of the lack of consistency between the effect of the selected carbohydrates and mood, it is not reasonable to assume that only one variable, namely blood glucose is responsible for the interactions observed between the dependent measures.

E. OVERVIEW

Overall, the results from the present study support the hypothesis that the glycemic response to the selected carbohydrates is associated with their effect on subjective appetite and short term food intake. The effect of the carbohydrates on mood and memory was not associated with their glycemic response. A beneficial effect of

sucrose was observed on memory performance. The interactions between all dependent measures suggests that the effects of carbohydrates on appetite, food intake, mood and memory are interrelated.

An inverse association was observed between the glycemic response to the selected carbohydrates and their effect on appetite and energy intake at a test meal at one hour. The greater the response in blood glucose after carbohydrate consumption, the less energy consumed at a test meal. Blood glucose concentrations 37 minutes following ingestion of the carbohydrates were positively associated with energy intake at one hour. Whether high blood glucose concentrations act directly on central feeding centres to suppress food intake or act through secondary peripheral signals is unclear. Because insulin is implicated in energy intake and is closely associated with glucose release (Rodin et al 1988; VanderWeele et al 1985; Woods et al 1996), further investigations into the relationship between the glycemic and insulinemic response to the carbohydrate treatments and energy intake is of interest.

It is important to acknowledge that the results are specific to the carbohydrate treatments employed and the time interval between preload and test meal. Further research is required to determine the role of blood glucose in appetite regulation over an extended period of time. It is possible that a series of integrated postabsorptive factors unrelated to blood glucose levels come into play several hours after ingestion of slowly digestible carbohydrates such as amylose and amylopectin. Similarly, the carbohydrate treatments were found to have no effect on ratings of mood over one hour. It is possible that the one hour time restriction compromised the ability to detect an effect of the carbohydrate treatments on mood.

An important observation is the positive impact sucrose was found to have on energy intake, mood and memory. Previous reports have suggested that sucrose consumption contributes to negative health through excess energy intake, and aggressive or hyperactive behaviour (Anderson 1995). The present results refute these unfounded beliefs and support further research into the positive effects of sucrose, perhaps through the formulation of satiating foods designed to aid in a weight loss program or enhance cognitive performance in children, young adults and the elderly.

A possible criticism of the present study is that the experimental design did not allow for adequate testing of the dependent measures, mood and memory as the focus was on the primary dependent measures, appetite and food intake. The results did however present some insight into the mechanism underlying these measures and their possible interactions.

In summary, the present evidence strongly supports the utilisation of pure carbohydrates in preload designs. In this respect, any effect observed can be attributed to a specific carbohydrate of known structural composition. Understanding the physiological parameters of each carbohydrate, such as the glycemic and insulinemic response can aid in the understanding of the mechanisms underlying carbohydrate modulation of behaviour.

VI. SUMMARY AND CONCLUSIONS

A. SUMMARY

1. The glycemic response after 75g preloads was of the general order:
    glucose > polycose > sucrose > amylopectin > fructose/glucose > amylose.

2. Sucrose, polycose and glucose preloads decreased mealtime energy intake at one
    hour. Only sucrose preloads improved immediate recall of a word list. None of the
    treatments affected mood over one hour.

3. An inverse relationship was observed between subjective appetite and short term food
    intake and area under the curve blood glucose. No relationship was observed between
    the glycemic response to the selected carbohydrates and mood and memory.

4. The effects of sucrose, polycose, glucose, fructose/glucose, amylose and amylopectin
    preloads on blood glucose, appetite, food intake, mood and memory were interrelated.

B. CONCLUSION

The effect of the selected carbohydrates on appetite and food intake but not mood
and memory is inversely associated with their glycemic response over one hour.

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APPENDIX 1

SCREENING QUESTIONNAIRES:

BASELINE INFORMATION QUESTIONNAIRE

FOOD ACCEPTABILITY LIST

EATING HABITS QUESTIONNAIRE

EFFECT OF CARBOHYDRATE SOURCES ON BLOOD GLUCOSE, APPETITE, MOOD AND COGNITION

CONSENT FORM

BASELINE INFORMATION QUESTIONNAIRE

NAME: _____   AGE: _____

ADDRESS: _____

_____

PHONE #: (____)_____

HEIGHT: _____   WEIGHT: _____   BMI: _____

PARTICIPATION IN ATHLETICS/EXERCISE:

ACTIVITY: _____   HOW OFTEN?: _____   HOW LONG? (HRS): _____

Do you usually eat breakfast?   YES _____   NO _____

 If YES, what do you usually eat for breakfast? _____

HEALTH STATUS

Do you have diabetes?   YES _____   NO _____

Do you have any other major diseases?   YES _____   NO _____

If YES please specify _____

Are you taking any medications?   YES _____   NO _____

Do you have reactions to any foods?   YES _____   NO _____

If YES please specify _____

Are you on a special diet?   YES _____   NO _____

If YES please specify _____

Do you smoke?   YES _____   NO _____

Have you gained or lost weight recently?   YES _____   NO _____

How many alcoholic beverages do you consume per day / week?   _____

EATING HABITS QUESTIONNAIRE

Choose the appropriate answer to best describe your personal situation.

1. How often are you dieting?

    never ___ rarely ___ sometimes ___ often ___ always ___

2. What is the maximum amount of (weight in pounds) that you have ever lost within one month?

    1-4 ___ 5-9 ___ 10-14 ___ 15-19 ___ 20+ ___

3. What is your maximum weight gain within one week?

    0-1 ___ 1.1-2 ___ 2.1-3 ___ 3.1-5 ___ 5.1+ ___

4. In a typical week, how much does your weight fluctuate?

    0-1 ___ 1.1-2 ___ 2.1-3 ___ 3.1-5 ___ 5.1+ ___

5. Would a weight fluctuation of 5 lb affect the way you live your life?

    Not at all ___ slightly ___ moderately ___ very much ___

6. Do you eat sensibly in front of others and splurge alone?

    Never ___ rarely ___ often ___ always ___

7. Do you give too much time and thought to food?

    Never ___ rarely ___ often ___ always ___

8. Do you have feelings of guilt after overeating?

    Never ___ rarely ___ often ___ always ___

9. How conscious are you of what you are eating?

    Not at all ___ slightly ___ moderately ___ extremely ___

10. How many pounds over your desired weight were you at your maximum weight?

    0-1 ___ 2-5 ___ 6-10 ___ 11-20 ___ 21+ ___

FOOD ACCEPTABILITY LIST

At each session you will receive a lemon flavoured, sweet beverage that may be high in sugar.

Please indicate whether you will be able to drink the beverage provided:

Yes _____   No _____

During each of the five sessions, you will also be provided with pizza. In order to provide you with a meal that you will enjoy, we ask that you rank the following pizzas according to your personal preference (i.e. **1,2,3**) in the space provided. If you do **NOT** like a particular type of pizza, then do not rank it, but place an **"X"** in the space provided.

Pepperoni (cheese; pepperoni) _____

Deluxe (cheese; pepperoni; peppers; mushrooms) _____

Three Cheese (mozzarella; cheddar; parmesan) _____

Deli Lovers (cheese; pepperoni; salami; bacon) _____

EFFECT OF CARBOHYDRATE SOURCES ON BLOOD GLUCOSE, APPETITE, MOOD AND COGNITION
Outline of Participant's Role

INITIAL SCREENING INTERVIEW:
    Each participant will provide the interviewer with basic information (anthropometric, health status) and answer questionnaires pertaining to food habits, in addition to completing a food acceptability list.

SESSIONS: 4 (Experiment 1). 5 (Experiment II and III)

NIGHT BEFORE EACH SESSION:
    Fast from 9:00 p.m., except for water. No water should be consumed for one hour prior to arrival.

SCHEDULE OF EACH SESSION:

7:45 a.m.	Participants will arrive at the Department of Nutritional Sciences, Room 329. Participant expected to stay within the department for the duration of the experiment (approx. 1½ hours).
7:50 a.m.	Participant will complete an appetite, physical comfort and mood questionnaire and a fingerprick blood sample will be taken.
8:00 a.m.	Participant will be given one of the 5 treatments (sweetened carbohydrate beverage). Participant has 5 minutes in which to consume the drink, after which he will be asked to rate its sweetness and palatability.
8:15 a.m.	Participant will take fingerprick blood samples and complete appetite. mood and physical comfort questionnaires every 15 minutes for 1 hour. Memory tests will be completed 15, 60 minutes and 45 minutes after the start of the treatment. A sleep and stress questionnaire, as well as audio/visual and trail making tests will be completed between 15 and 45 minutes after drinking the beverage.
9:00 p.m.	After the completion of questionnaires, a pizza meal will be served. Following the meal, participants complete a physical comfort, appetite and palatability questionnaire.

If you have any questions regarding this study, please contact:

Nicole Catherine	Investigator	(416) 978-3700
Dr. G. Harvey Anderson (Principal investigator)		(416) 978-1832

BLOOD GLUCOSE, APPETITE, MOOD AND COGNITION
Consent Form

The purpose of this study is to determine the effect of carbohydrates on blood glucose, appetite, mood and cognition. I have been fully informed of what is expected of me as a participant in this research project and I have been provided with a typewritten copy of these expectations as outlined in the attachment to this consent form.

I am aware that my participation will not involve any health risk to me; that personal information will remain confidential; and that my name will not appear in any published document.

I understand that for the purposes of the research project, it is hoped that I will complete all 5 sessions. However, I may choose to withdraw at any time without prejudice, whereupon I will receive the prorated portion of the total payment of $75. If I should complete all 5 sessions, I will receive a bonus amount of $15. Upon completion of the study, a summary of the results will be available for me to pick up from the Department of Nutritional Sciences.

I hereby agree and give my authorized consent to participate in the study.

DATE:_____

PARTICIPANT'S NAME:_____

PARTICIPANTS SIGNATURE:_____

WITNESS' SIGNATURE:_____

APPENDIX II

STUDY DAY QUESTIONNAIRES

SLEEP AND STRESS FACTORS QUESTIONNAIRE

VAS-MOTIVATION TO EAT

VAS-PHYSICAL COMFORT

VAS-PALATABILITY

VAS-PERCEIVED SWEETNESS

MEMORY TEST SHEET

SLEEP HABITS AND STRESS FACTORS QUESTIONNAIRE

NAME: _____

DATE: _____

1. Did you have a normal night's sleep last night?   YES _____   NO _____

2. How many hours of sleep did you have?   _____

3. Are you under unusual stress?   e.g. exams/report deadlines, work deadlines, personal
life stress.

   Today: YES ____ NO ____   Last 24 hours: YES ____ NO ____

   If YES, please describe briefly: _____

   _____

4. Have you been engaged in any physical activity, unusual to your normal routine,

   within the past 24 hours?   YES ____   NO ____

   If YES, please describe briefly: _____

   _____

5. Have you had anything to eat or drink, other than water since 9:00 p.m. last night?

   YES ____   NO ____

   If YES, please describe briefly: _____

6. At what time did you eat your last meal yesterday? _____

7. Please describe the contents of your last meal yesterday: _____

   _____

Visual Analogue Scale
Motivation to Eat

NAME: _____

DATE: _____

These questions relate to your motivation to eat at this time. Please rate yourself by placing a small "x" across the horizontal line at the point which best reflects your present feelings.

1. How strong is your desire to eat?

Very
WEAK _____ Very
STRONG

2. How hungry do you feel?

NOT
Hungry
At all _____ As hungry as
I have ever
Felt

3. How full do you feel?

NOT
Full
At all _____ VERY
Full

4. How much food do you think you could eat?

NOTHING
At all _____ A LARGE
Amount

Visual Analogue Scale
Mood

NAME: _____

DATE: _____

These questions relate to your mood state at this time. Please rate yourself by placing a small "x" across the horizontal line at the point which best reflects your present feelings.

1. How alert do you feel?

very little _____ very much

2. How sad do you feel?

very little _____ very much

3. How tense do you feel?

very little _____ very much

4. How much of an effort is it to do anything?

very little _____ very much

5. How happy do you feel?

very little _____ very much

Visual Analogue Scale
Mood (Continued)

6. How weary do you feel?

very little _____ very much

7. How calm do you feel?

very little _____ very much

8. How sleepy do you feel?

very little _____ very much

Visual Analogue Scale
Physical Comfort

NAME: _____

DATE: _____

These questions relate to your comfort level at this time. Please rate yourself by placing a small "x" across the horizontal line at the point which best reflects your present feelings.

How well do you feel?

NOT   VERY
well at all _____ well

Visual Analogue Scale
Palatability

NAME: _____

DATE: _____

This question relates to the palatability of the beverage you just consumed. Please rate the pleasantness of the beverage by placing a small "x" across the horizontal line at the point which best reflects your present feelings.

How pleasant have you found the beverage?

Not at all   Very
Pleasant _____ Pleasant

Visual Analogue Scale
Sweetness

NAME: _____

DATE: _____

This question relates to the sweetness of the beverage you just consumed. Please rate the sweetness of the beverage by placing a small "x" across the horizontal line at the point which best reflects your present feelings.

How sweet have you found the beverage?

Not sweet   Extremely
at all   _____   Sweet

DATE: _____
NAME: _____

Please write down as many words as you can remember:

APPENDIX III

WORD LISTS

AUDIO VISUAL TASK

Word Lists: Set 1

#1	#2	#3	#4	#5
Cottage	Barrel	Closet	Dealer	Flower
Rider	School	Series	City	Willow
Frontier	Basket	Spaniard	Marble	Service
Hammer	Weather	Culture	Refuge	Stocking
Husband	Olive	Income	Darkness	Leather
Herald	System	Wisdom	Weapon	Matter
Distress	Motion	Moisture	Career	Compass
Salad	Oven	Bargain	Copy	Sunshine
Battle	Morning	Diamond	Demand	Industry
Cabin	Riches	Candle	Tribute	Detail
Laughter	Shiver	Victim	Eagle	Repeat
Pillow	Errand	Message	Neighbour	Fountain
Prayer	Trouble	Contract	Escape	Rifle
Insult	Delay	Pattern	Cotton	Witness
Officer	Music	Sulphur	Quarrel	Dismay
Captive	Attempt	Mercy	Unit	Ideal
Handle	Shipping	Vapour	Spider	Servant
Mother	Kitten	Valley	Cancel	Rubber
Menace	Decrease	Season	Lion	Temple
Notion	Nephew	Stable	Motor	Drama

#6	#7	#8	#9	#10
Body	Paper	Merchant	Chairman	Teacher
Consent	Section	Timber	Contest	Puzzle
Gesture	Value	Function	Standing	Spirit
Outline	Agent	Lady	Prairie	Father
Luncheon	Offer	Ankle	Liquid	Owner
Doctrine	Desire	Product	Reply	Negro
Pleasure	Navy	Current	Leader	Disgrace
Assault	Cable	Conflict	Oyster	Orange
Wedding	Elbow	Slipper	Scholar	Journey
Aspect	Button	Kitchen	Maker	Garment
Prospect	Appeal	Fabric	Rabbit	Tunnel
Amount	Result	Theory	Marvel	Latin
Parent	Manner	Total	Dragon	Highway
Disease	Hunter	Content	Jersey	Muscle
Extent	Formal	Language	Squirrel	Figure
Device	Rebel	Million	Darling	Empire
Interest	Heaven	Virtue	Kingdom	Devil
Cousin	Bushel	Jewel	Return	Reverse
Sentence	Debate	Pupil	Market	Design
Enemy	Decay	Slumber	Surprise	Factor

Word Lists: Set 2

#1	#2	#3	#4	#5
station	banker	twenty	water	journal
cancer	despair	guitar	insight	vulture
transfer	nation	steamer	stumble	europe
dollar	carpet	basin	winter	armour
warrant	banner	women	exhaust	receipt
courage	level	beneath	bottom	midnight
hybrid	washer	venom	weaver	wallet
office	village	moment	lesson	visit
letter	football	sandwich	keeper	cleveland
center	nature	party	table	people
monarch	trousers	resource	traitor	carbon
tower	account	castle	harvest	welcome
doorbell	summer	outlaw	outbreak	clutter
escort	warehouse	commerce	grammer	concert
title	organ	standard	wagon	power
effort	record	evening	sister	daylight
research	underneath	costume	speaker	murder
solid	lawyer	bishop	insect	abuse
irish	prophet	mankind	doorway	signal
survey	needle	rival	velvet	ticket

#6	#7	#8
farewell	household	passion
oval	habit	quaker
colour	crystal	bubble
penny	museum	frozen
cluster	flavour	practise
limit	lover	fortune
steamship	utensil	college
union	public	public
sickness	daughter	butcher
island	pistol	degree
mischief	acre	compound
pocket	wedlock	progress
vampire	soldier	quicksand
eyebrow	product	mansion
treaty	garden	purchase
mountain	uncle	duty
coward	nibble	intent
bureau	poison	triumph
talent	movie	instinct
layer	retreat	object

AUDIO/VISUAL TASK

<u>Instructions</u>

You will be shown a series of music videos and will be asked to fill out the following table. For every object and word you see/hear, place a tally mark in the corresponding box.

<u>Example:</u>

Title	Visual / Objects	# Observations	Audio / Words	# Observations
"Wannabe" Spice Girls				
	London Bus	I I I I	Lover	I I I I I I
	Union Jack	I I	Friends	I I I I I I I I
	Policeman	I		
	Lollipop	I I I I		

APPENDIX IV

TREATMENTS:

CARBOHYDRATE COMPOSITION

FRUCTOSE CALCULATION

Experiment I and II. Characteristics of Treatments

Carbohydrate	Class (Degree of Polymerisation)†	Ratio of Monomers	Structure‡	GI*	Predicted Glycemic response
Sucrose	Sugars (1-2)	50% Glucose, 50% Fructose	Disaccharide, Glycosidic bonds	~84	Moderate
Hydrolysed Corn Starch	Modified Corn Starch (2-4),(5-9), (>9)	2% Free Glucose 98% Glucose chains	Linear, α(1-6) linkages	~100	Rapid
High Amylose Corn Starch	Starch (>9)	70% Amylose	Linear, α (1-4) linkages	~59[1]	Slow
High Amylopectin Corn Starch	Starch (>9)	70% Amylopectin	Branched, α(1-4) and α(1-6) linkages	~88[2]	Moderate

† FAO/WHO (1997) Expert Consultation Rome
‡ Human Nutr Rev. (1989) Ed. John Dobbing Springer-Verlag Berlin Heidelberg
* Foster-Powell & Brand Miller (1995) Am.J.Clin. Nutr. 62:871S-93S

[1]Mean value for high amylose white rice (Bread =100)
[2]Mean value for low amylose white rice (Bread = 100)

Experiment III. Characteristics of Treatments

Treatment	Class (Degree of Polymerisation)†	Ratio of Monomers	Structure‡	G.I.*	Predicted Glycemic response
Sucrose	Sugars (1-2)	50% Glucose : 50% Fructose	Disaccharide, Glycosidic bonds	87	Moderate
Hydrolysed Corn Starch (Polycose)	Modified Corn Starch (2-4),(5-9), (>9)	2% Free Glucose : 98% Glucose chains	Linear, α(1-6) linkages	~100	Rapid
Fructose / Glucose	Sugars (1-2)	80% Fructose : 20% Glucose	Mono-saccharide	16	Slow
Glucose	Sugars (1-2)	100% Glucose	Mono-saccharide	149	Rapid

*Glycemic Index. Standard White Bread = 100.Lee BM, Wolever TM Eur J Clin Nutr 1998 52(12): 924-8

Experiment III: Fructose Calculation

To find Fructose (y):

If Glucose (x) =190.5 mmol/min/L and Sucrose (x/2 + y/2) =131.6 mmol/min/L

then; $x + y = 2 \ (131.6) = 263.2$

$y = 263.2 - 190.5$

$y = 72.7$

If fructose (y) = 72.7 and GI of glucose = 100*, then GI of 80% fructose and 20% glucose :

$$= (0.8 \times 72.7) + (0.2 \times 100) = \mathbf{78.16}$$

Therefore the glycemic index of an 80% fructose, 20% glucose mixture would be expected to have the value of 78.16.

*Foster-Powell & Brand Miller 1995 ; Glucose = 100

APPENDIX V

SUCRALOSE ADDITIONS

PIZZA COMPOSITION

Experiment I and II. Sucralose and Lemon from Concentrate Additions

Treatment	Sucralose	Lemon
Sucrose	0mg	1 tsp
Hydrolysed Corn Starch (Polycose)	170mg	1 tsp
High Amylose Corn Starch	550mg	2 tsp
High Amylopectin Corn Starch	550mg	2 tsp
Sucralose	250mg	½ tsp

Experiment III. Sucralose and Lemon from Concentrate Additions

Treatment	Sucralose	Lemon
Sucrose	150mg	½ tsp
Hydrolysed Corn Starch (Polycose)	1.5 g	½ tsp
Fructose/ Glucose	0 mg	½ tsp
Glucose	750mg	½ tsp
Sucralose	1 g	¼ tsp

Pizza Composition [1]

Nutritional Information per 100g	Pepperoni	Deluxe	Three Cheese	Deli Lovers
Protein (g)	11.0	9.1	13.0	11.0
Total Fat (g)	7.7	6.2	8.4	8.9
Carbohydrate (g)	28.0	27.0	29.0	27.0
Energy (kcal)	219.0	195.0	237.0	230.0

[1] McCain Foods: Deep and Delicious, 5' Pizza

APPENDIX VI

CORRELATIONS: EXPERIMENT II

EXPERIMENT III

THE RELATIONSHIP BETWEEN BLOOD GLUCOSE AND APPETITE, FOOD INTAKE, MOOD AND MEMORY

EXPERIMENT II

Table i. Exp II Relationships Between Appetite, Food Intake, Sweetness and Palatability

Correlated Variables	r ; p[1]
Food Intake	
Palatability	-0.006;0.96
Sweetness	-0.03; 0.79
60 min Physical Comfort	-0.00; 0.99
30 min Average Appetite	
Palatability	0.203;0.09
Sweetness	0.15;0.23

[1]Pearson Correlation Coefficients, Probability under the null hypothesis: r=0

Table ii. Exp II Relationships Between Appetite, Food Intake and Mood

Correlated Variables	r ; p[1]
Food Intake	
Average Appetite at 60 mins	0.399 ; 0.0006*
Hunger at 60 mins	0.417 ; 0.0003*
Amount at 60 mins	0.455 ; <.0001*
Desire at 60 mins	0.42 ; 0.0003*
Full at 60 mins	0.087 ; 0.47
Global Vigour	0.065 ; 0.59
Alert at 60 mins	0.07 ; 0.56
Weary at 60 mins	0.009 ; 0.93
Sleepy at 60 mins	-0.057 ; 0.63
Effort at 60 mins	-0.09 ; 0.44
Global Affect	-0.04 ; 0.73
Calm at 60 mins	-0.06 ; 0.6
Happy at 60 mins	-0.12 ; 0.33
Tense at 60 mins	0.002 ; 0.98
Sad at 60 mins	0.12 ; 0.33

[1]Pearson Correlation Coefficients, Probability under the null hypothesis: r=0
*Correlation is significant at the 0.01 level (2-tailed)

Table iii . Exp II Relationships Between Appetite and Mood

Correlated Variables	r ; p[1]
Average Appetite	
Global Vigour	0.395 ; 0.0007**
Sleepy at 60 mins	-0.35 ; 0.003**
Weary at 60 mins	-0.27 ; 0.02*
Effort at 60 mins	-0.31 ; 0.009**
Alert at 60 mins	0.39 ; 0.0008**
Alert at 30 mins	0.204 ; 0.09

[1]Pearson Correlation Coefficients, Probability under the null hypothesis: r=0
*Correlation is significant at the 0.05 level (2-tailed)
**Correlation is significant at the 0.01 level (2-tailed)

Table iv . Exp II Relationships Between Appetite, Food Intake and Memory

Correlated Variables	r ; p[1]
Food Intake	
Memory score at 60-1	0.198 ; 0.1
Memory score at 60-2	0.183 ; 0.13
Memory score at 60-3	0.087 ; 0.47
Total Memory score at 60	0.16 ; 0.18
Average Appetite	
Memory score at 60-1	0.014 ; 0.9
Memory score at 60-2	0.09 ; 0.42
Memory score at 60-3	0.14 ; 0.24
Total Memory score at 60	0.09 ; 0.4

[1]Pearson Correlation Coefficients, Probability under the null hypothesis: r=0

Table v. Exp II Relationships Between Mood and Memory

Correlated Variables	$r ; p$[1]
Global Vigour	
Memory score at 60-1	0.21 ; 0.08
Memory score at 60-2	0.15 ; 0.21
Memory score at 60-3	0.25 ; 0.04
Total Memory score at 60	0.22 ; 0.07
Global Affect	
Memory score at 60-1	-0.01 ; 0.9
Memory score at 60-2	-0.02 ; 0.86
Memory score at 60-3	0.11 ; 0.35
Total Memory score at 60	0.03 ; 0.78

[1] Pearson Correlation Coefficients, Probability under the null hypothesis: r=0

EXPERIMENT III

Table vi.Exp III Relationships between Appetite, Food Intake, Sweetness and Palatability

Correlated Variables	$r : p$[1]
Food Intake Average Appetite	0.45 ; <.0001**
Food Intake and Preload Palatability	0.15 ; 0.21
Food Intake and Preload Sweetness	-0.17 ; 0.14
Average Appetite and Preload Palatability	0.11 ; 0.34
Average Appetite and Preload Sweetness	-0.15 ; 0.21

[1] Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
** Correlation is significant at the 0.01 level (2-tailed)

Table vii . Exp III Relationships between Blood Glucose, Appetite and Food Intake

Correlated variables	r ; p[1]
Average Appetite	
Baseline Blood Glucose	0.03 ; 0.79
20min Blood Glucose	-0.17 ; 0.14
37min Blood	-0.207 ; 0.07
67min Blood Glucose an	-0.17 ; 0.13
Area Under the Curve	-0.233 ; 0.045*
Food Intake	
Baseline Blood Glucose	-0.119 ; 0.31
20min Blood Glucose	-0.157 ; 0.18
37min Blood Glucose	-0.239 ; 0.04*
67min Blood Glucose	-0.219 ; 0.06
Area Under the Curve and Food Intake	-0.236 ; 0.04*

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
*Correlation is significant at the 0.05 level (2-tailed)
**Correlation is significant at the 0.01 level (2-tailed)

Table viii. Exp III Partial Correlations Between Blood Glucose, Appetite and Food Intake

Correlated variables	Food Intake r ; p	Average Appetite r ; p
37 min Blood Glucose	-0.235; 0.046*	-0.22 ; 0.058
Area Under the Curve	-0.237 ; 0.043*	-0.239 ; 0.041*

[1]Pearsons' Partial Correlation Coefficient ; Controlling for subject
*Correlation is significant at the 0.05 level (2-tailed)

Table ix. Relationships between Blood Glucose and Memory

Correlated variables	r ; p[1]
Total 15 min Memory score and 0 min Blood Glucose	-0.007; 0.95
Total 15 min Memory score and 20 min Blood Glucose	0.59 ; 0.61
Total 15 min Memory score and AUC Blood Glucose	0 176 ; 0 14
Total 15 min Memory score and (20 –0 min) Blood Glucose [2]	0.86 ; 0.46
Total 60 min Memory score and 0 min Blood Glucose	0.003; 0.98
Total 60 min Memory score and 66 min Blood Glucose	0.11 ; 0.92
Total 60 min Memory score and AUC Blood Glucose	0.014 ; 0.91
Total 60 min Memory score and (66 –37 min) Blood Glucose [3]	-0.055 ; 0.65
15 and 60 min Memory score and 0 min Blood Glucose	-0.007; 0.95
15 and 60 min Memory score and AUC Blood Glucose	0.13 ; 0.23
45 min Memory score and 37 min Blood Glucose	0.138; 0.24
45 min Memory score and AUC Blood Glucose	0.192 ; 0.1
45 min Memory score and (20 –0 min) Blood Glucose	0.124 ; 0.29
45 min Memory score and (66 –37 min) Blood Glucose	-0.06 ; 0.62
15 min Incorrect Memory score and 0min Blood Glucose	-0.159 ; 0.17
15 min Incorrect Memory score and AUC Blood Glucose	-0.75 ; 0.53
15 min Incorrect Memory score and (20 - 0min) Blood Glucose	-0.56 ; 0.633

[1] Pearsons' Correlation Coefficient ; r= 0 ; significance at 0.01 levels (2 tailed)
[2] Rise in Blood Glucose between baseline and 20 mins
[3] Fall in Blood Glucose between 37 min and 66 min

Table ix. Continued

Correlated variables	r ; p[1]
15 −1 min Incorrect Memory score and 0min Blood Glucose	0.051 ; 0.66
15 −1 min Incorrect Memory score and AUC Blood Glucose	0.033 ; 0.78
15 −1 min Incorrect Memory score and (20-0min) Blood Glucose	-0.087 ; 0.46
15 −3 min Incorrect Memory score and 0min Blood Glucose	-0.22 ; 0.058
15 −3 min Incorrect Memory score and AUC Blood Glucose	-0.166 ; 0.16
15 −3 min Incorrect Memory score and (20-0min) Blood Glucose	-0.051 ; 0.66
60 −1 min Incorrect Memory score and 0min Blood Glucose	-0.038 ; 0.75
60 −1 min Incorrect Memory score and AUC Blood Glucose	0.002 ; 0.99
60 −1 min Incorrect Memory score and (66-37min) Blood Glucose	-0.14 ; 0.24
60 −2 min Incorrect Memory score and 0min Blood Glucose	-0.13 ; 0.26
60 −2 min Incorrect Memory score and AUC Blood Glucose	0.00 ; 0.99
60 −2 min Incorrect Memory score and (66-37min) Blood Glucose	-0.74 ; 0.54

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0

Table x. Relationships between Blood Glucose and Ratings of Sadness

Correlated Variables	r ; p[1]
Baseline Sad rating and 0min Blood Glucose	0.258 ; 0.027*
Baseline Sad rating and 20min Blood Glucose	0.016 ; 0.89
Baseline Sad rating and 37min Blood Glucose	0.037 ; 0.75
Baseline Sad rating and (20-0min) Blood Glucose	-0.05 ; 0.66
30min Sad rating and 0min Blood Glucose	0.294 ; 0.012*
30min Sad rating and 20min Blood Glucose	0.029 ; 0.81
30min Sad rating and 37min Blood Glucose	-0.009 ; 0.94
30min Sad rating and (20-0min) Blood Glucose	-0.479 ; 0.69
30-0 min Sad rating and 0min Blood Glucose	-0.006 ; 0.96
30-0min Sad rating and 20min Blood Glucose	0.012 ; 0.92
30-0min Sad rating and 37min Blood Glucose	-0.058 ; 0.62
30-0min Sad rating and (20-0min) Blood Glucose	0.014 ; 0.91

[1] Pearsons' Partial Correlation Coefficient ; Controlling for subject
* Significance at 0.05 level (2 tailed)

Table xi. Relationships between Blood Glucose and Ratings of Tense

Correlated variables	r ; p[1]
Baseline Tense rating and 0min Blood Glucose	0.285 ; 0.015*
Baseline Tense rating and 20min Blood Glucose	0.07 ; 0.55
Baseline Tense rating and 37min Blood Glucose	0.027 ; 0.82
Baseline Tense rating and (20-0min) Blood Glucose	-0.002 ; 0.99
30min Tense rating and 0min Blood Glucose	0.233 ; 0.049*
30min Tense rating and 20min Blood Glucose	0.14 ; 0.23
30min Tense rating and 37min Blood Glucose	0.103 ; 0.39
30min Tense rating and (20-0min) Blood Glucose	0.08 ; 0.48
30-0 min Tense rating and 0min Blood Glucose	-0.09 ; 0.45
30-0min Tense rating and 20min Blood Glucose	0.09 ; 0.42
30-0min Tense rating and 37min Blood Glucose	0.107 ; 0.37
30-0min Tense rating and (20-0min) Blood Glucose	0.12 ; 0.3

[1] Pearsons' Partial Correlation Coefficient ; Controlling for subject
* Correlation is significant at 0.05 level (2 tailed)

Table xii . Exp III Relationship Between Appetite, Food Intake and Mood at 30 minutes

Correlated Variables	r ; p[1]	
Individual Mood Questions	Food Intake	Appetite
Alert score at 30 mins	0.09 ; 0.44	-0.1 ; 0.37
Sad score at 30 mins	-0.97 ; 0.41	0.19 ; 0.09
Tense score at 30 mins	-0.289 ; 0.012*	-0.7 ; 0.5
Happy score at 30 mins	0.241 ; 0.037*	-0.75 ; 0.5
Effort score at 30 mins	-0.15 ; 0.19	0.19 ; 0.09
Weary score at 30 mins	-0.58 ; 0.62	0.267 ; 0.02*
Calm score at 30 mins	0.228 ; 0.49*	0.043 ; 0.72
Sleepy score at 30 mins	0.018 ; 0.88	0.364 ; .001**

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
*Correlation is significant at the 0.05 level (2-tailed)
**Correlation is significant at the 0.01 level (2-tailed)

Table xiii. Exp III Partial Correlation between Appetite, Food Intake and Mood (30 mins)

Correlated Variables	r ; p[1]	
Individual Mood Questions	Food Intake	Appetite
Tense score at 30 mins	-0.281 ; 0.015*	-0.11 ; 0.37
Happy score at 30 mins	0.226 ; 0.052	-0.018 ; 0.88
Weary score at 30 mins	-0.06 ; 0.63	0.269 ; 0.021*

[1]Partial Pearson Correlation Coefficient, controlling for subject
*Correlation is significant at the 0.05 level (2-tailed)

Table xiv . Exp III Relationship Between Appetite, Food Intake and Mood (Change from Baseline)

Correlated Variables	r ; p[1]	
Change from Baseline Mood	Food Intake	Appetite
Alert score (30-0)mins	0.248 ; 0.032*	0.16 ; 0.17
Sad score (30-0)mins	-0.39 ; 0.74	0.13 ; 0.27
Tense score (30-0)mins	-0.118 ; 0.31	-0.06 ; 0.64
Happy score (30-0)mins	0.15 ; 0.21	-0.09 ; 0.43
Effort score (30-0)mins	-0.09 ; 0.47	0.025 ; 0.83
Weary score (30-0)mins	0.06 ; 0.6	0.09 ; 0.42
Calm score (30-0)mins	0.238 ; 0.04*	0.223 ; 0.054
Sleepy score (30-0)mins	-0.1 ; 0.38	-0.15 ; 0.19

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
*Correlation is significant at the 0.05 level (2-tailed)

Table xv. Exp III Partial Correlation Between Appetite, Food Intake and Mood (Change from Baseline)

Correlated Variables	r ; p[1]	
Change from Baseline Mood	Food Intake	Appetite
Alert score (30-0) mins	0.254 ; 0.029*	0.155 ; 0.19
Calm score (30-0) mins	0.248 ; 0.033*	0.21 ; 0.07

[1]Partial Pearson Correlation Coefficient, Controlling for subject
*Correlation is significant at the 0.05 level (2-tailed)

Table xvi. Exp III Relationship Between Immediate recall and Mood (Change from
Baseline)

Correlated Variables	r ; p[1]	
Change from Baseline Mood	Total 15 min score	Total 60 min score
Alert score (30-0)mins	-0.16 ; 0.18	-0.294 ; 0.01*
Sad score (30-0)mins	0.24 ; 0.038*	0.074 : 0.53
Tense score (30-0)mins	0.08 ; 0.51	0.04 ; 0.72
Happy score (30-0)mins	-0.281 ; 0.015*	0.029 ; 0.81
Effort score (30-0)mins	-0.62 ; 0.59	0.15 ; 0.19
Weary score (30-0)mins	0.07 ; 0.56	-0.009 ; 0.94
Calm score (30-0)mins	-0.07 ; 0.54	-0.06 ; 0.64
Sleepy score (30-0)mins	0.06 ; 0.61	0.15 ; 0.21

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
*Correlation is significant at the 0.05 level (2-tailed)

Table xvii. Exp III Partial Correlations Between Immediate recall and Mood (Change from Baseline)

Correlated Variables	r ; p[1]
Change from Baseline Sad score and memory at 15mins	0.229 ; 0.049*
0min Sad score and memory at 15 mins	-0.01 ; 0.92
30min Sad score and memory at 15 mins	0.19 ; 0.09
Change from Baseline Happy score and memory at 15mins	-0.277 ; 0.017*
0min Happy score and memory at 15 mins	-0.01 ; 0.93
30min Happy score and memory at 15 mins	-0.19 ; 0.09
Change from baseline Alert score and memory at 60 mins	-0.3 ; 0.008**
0min Alert score and memory at 60mins	0.09 ; 0.42
30min Alert score and memory at 60mins	-0.229 ; 0.05*

[1]Partial Pearson Correlation Coefficient, Controlling for subject
*Correlation is significant at the 0.05 level (2-tailed)
** Correlation is significant at the 0.01 level (2-tailed)

Table xviii . Exp III Relationship Between Memory and Mood (Change from Baseline)

Correlated Variables	r ; p[1]	
Mood (Change from Baseline)	Total Immediate recall (15 +60 min score)	Delayed recall (45 min score)
Alert score (30-0)mins	-2.61 ; 0.24*	-0.15 ; 0.19
Sad score (30-0)mins	0.14 ; 0.23	-0.27 ; 0.82
Tense score (30-0)mins	0.95 ; 0.42	0.16 ; 0.18
Happy score (30-0)mins	-0.12 ; 0.3	-0.27 ; 0.82
Effort score (30-0)mins	0.025 ; 0.83	-0.1 ; 0.39
Weary score (30-0)mins	0.008 ; 0.95	-0.76 ; 0.51
Calm score (30-0)mins	-0.1 ; 0.39	-0.16 ; 0.17
Sleepy score (30-0)mins	0.11 ; 0.35	0.02 ; 0.86

[1]Pearson Correlation Coefficient, Probability under the null hypothesis, r=0
*Correlation is significant at the 0.05 level (2-tailed)

Table xix. Exp III Partial Correlations Between Total Immediate recall and Mood (Change from Baseline)

Correlated Variables	r ; p[1]
Change from Baseline Alert score and Total (15+60)min score	-0.271 ; 0.019*
0min Alert score and Total (15+60)min score	0.092 ; 0.44
30min Alert score Total (15+60)min score	-0.19 ; 0.09

[1]Partial Pearson Correlation Coefficient, Controlling for subject
*Correlation is significant at the 0.05 level (2-tailed)