EFFECT OF VARIETY AND COOKING METHOD ON RESISTANT STARCH CONTENT OF WHITE RICE AND SUBSEQUENT POSTPRANDIAL GLUCOSE RESPONSE AND APPETITE IN HUMANS

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ABSTRACT

Rice is a staple carbohydrate throughout the world. Previous work has indicated that the resistant starch (RS) content of rice consumed in India varied with rice variety and cooking method. This study quantified RS in four white rice varieties (jasmine, long grain, medium grain, and short grain) cooked in three manners (baked, conventional rice cooker, and pressure cooker). The rice varieties with the highest and lowest RS content were selected for a pilot-scale trial to characterize postprandial glycemic response and appetite ratings in healthy adults ($n = 21$). The results showed refrigerated long-grain rice cooked in a conventional rice cooker had the highest RS content (HRS, $2.55 \text{ g RS 100 g}^{-1}$) and refrigerated short-grain rice cooked in a pressure cooker had the lowest RS content (LRS, $0.201 \text{ g RS 100 g}^{-1}$). The areas under the curves for glycemic response were significantly lower with HRS and LRS than with glucose beverage; however, there was no difference between HRS and LRS. Glycemic indices did not differ significantly between HRS and LRS. Subjects reported an overall increased feeling of fullness and decreased desire to eat based on the incremental area under the curve for both HRS and LRS compared to control. In conclusion, we found that RS naturally occurring in rice had minimal impact on postprandial glycemic response and appetite.
# TABLE OF CONTENTS

ACKNOWLEDGEMENTS ................................................................................................................................. ii

ABSTRACT .......................................................................................................................................................... iii

LIST OF TABLES ................................................................................................................................................ v

LIST OF FIGURES ................................................................................................................................................. vi

LIST OF ABBREVIATIONS ............................................................................................................................... vii

CHAPTER 1: LITERATURE REVIEW ................................................................................................................... 1

1.1 INTRODUCTION ........................................................................................................................................... 1

1.2 DIABETES MELLITUS .................................................................................................................................. 3

1.2.1 Diagnosis of Diabetes ............................................................................................................................ 4

1.2.2 Blood Glucose Regulation ................................................................................................................... 6

1.2.3 Types of Diabetes .................................................................................................................................. 6

1.3 GLYCEMIC INDEX AND GLYCEMIC LOAD .............................................................................................. 15

1.4 DIETARY FIBER .......................................................................................................................................... 17

1.5 RESISTANT STARCH .................................................................................................................................. 19

1.5.1 Definition and Chemistry ....................................................................................................................... 19

1.5.2 General Physiological Effects of Resistant Starch ................................................................................ 21

1.5.3 Impact on Glycemic Response and Insulin Sensitivity .......................................................................... 21

1.5.4 Food Sources of Resistant Starch ........................................................................................................ 23

CHAPTER 2: EFFECT OF VARIETY AND COOKING METHOD ON RESISTANT STARCH CONTENT OF WHITE RICE AND SUBSEQUENT POSTPRANDIAL GLUCOSE RESPONSE AND APPETITE IN HUMANS .................................................................................................................. 26

2.1 INTRODUCTION ........................................................................................................................................... 26

2.2 MATERIALS AND METHODS ................................................................................................................... 28

2.2.1 Determination of RS in Rice ................................................................................................................ 28

2.2.2 Determination of Postprandial Glucose Response and Appetite Ratings ........................................... 30

2.2.3 Statistical Analysis ............................................................................................................................... 33

2.3 RESULTS .................................................................................................................................................... 33

2.3.1 Effect of Cooking Method and Variety on RS Content of Rice ................................................................. 33

2.3.2 Effect of RS on Glycemic Response and Appetite .................................................................................. 34

2.4 DISCUSSION .............................................................................................................................................. 35

2.5 CONCLUSION ............................................................................................................................................ 40

APPENDIX A: QUESTIONAIRES ................................................................................................................... 46

APPENDIX B: FOOD RECORD ..................................................................................................................... 53

APPENDIX C: APPETITE SURVEY .................................................................................................................. 57

REFERENCE .................................................................................................................................................... 58
LIST OF TABLES

Table 1-1. The diagnostic criteria of diabetes .............................................................. 25

Table 1-2. Classification of resistant starch................................................................. 25

Table 2-1. Resistant starch content in 4 varieties of white rice, prepared by different cooking methods. ........................................................................... 41

Table 2-2. Demographics ............................................................................................. 42

Table 2-3. Mean blood glucose concentrations .......................................................... 42

Table 2-4. Dietary intake during 24 hours prior to study visit ..................................... 43
LIST OF FIGURES

Figure 2-1. Mean appetite rating in response to glucose beverage and rice treatments over time .............................................................................................................. 44

Figure 2-2. Total appetite sensation AUC ............................................................................. 45
**LIST OF ABBREVIATIONS**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adequate intake</td>
<td>AI</td>
</tr>
<tr>
<td>Area under the curve</td>
<td>AUC</td>
</tr>
<tr>
<td>Cardiovascular diseases</td>
<td>CVD</td>
</tr>
<tr>
<td>Codex Alimentarius Commission</td>
<td>CAC</td>
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<tr>
<td>Dietary fiber</td>
<td>DF</td>
</tr>
<tr>
<td>Fasting blood glucose</td>
<td>FBG</td>
</tr>
<tr>
<td>Food and Agriculture Organization</td>
<td>FAO</td>
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<tr>
<td>Gestational diabetes mellitus</td>
<td>GDM</td>
</tr>
<tr>
<td>Glucose transport protein</td>
<td>GLUT</td>
</tr>
<tr>
<td>Glycemic index</td>
<td>GI</td>
</tr>
<tr>
<td>Glycemic load</td>
<td>GL</td>
</tr>
<tr>
<td>High resistant starch</td>
<td>HRS</td>
</tr>
<tr>
<td>Impaired fasting blood glucose</td>
<td>IFG</td>
</tr>
<tr>
<td>Incremental area under the curve</td>
<td>iAUC</td>
</tr>
<tr>
<td>Low resistant starch</td>
<td>LRS</td>
</tr>
<tr>
<td>Oral glucose tolerance test</td>
<td>OGTT</td>
</tr>
<tr>
<td>Rapidly digestible starch</td>
<td>RDS</td>
</tr>
<tr>
<td>Resistant starch</td>
<td>RS</td>
</tr>
<tr>
<td>Resistant starch type 1</td>
<td>RS I</td>
</tr>
<tr>
<td>Resistant starch type 2</td>
<td>RS II</td>
</tr>
<tr>
<td>Resistant starch type 3</td>
<td>RS III</td>
</tr>
<tr>
<td>Resistant starch type 4</td>
<td>RS IV</td>
</tr>
<tr>
<td>Short chain fatty acids</td>
<td>SCFAs</td>
</tr>
<tr>
<td>Type 1 diabetes mellitus</td>
<td>T1DM</td>
</tr>
<tr>
<td>Type 2 diabetes mellitus</td>
<td>T2DM</td>
</tr>
</tbody>
</table>
CHAPTER 1
LITERATURE REVIEW

1.1 INTRODUCTION

The prevalence of diabetes is rapidly rising in the United States and throughout the world. Diabetes was the seventh leading cause of death in the United States in 2010 [1]. In the same year, the Centers for Disease Control and Prevention reported that diabetes mellitus affected 25.8 million people [2]. Approximately 8.3% of the U.S. population suffers from diabetes mellitus [2]. Diabetes may be influenced by race or ethnicity. In Hawai‘i, diabetes is the fourth leading cause of death in Native Hawaiians, and they are at particularly high risk of diabetes compared to other ethnic groups [3]. Cultural differences and racial and ethnic disparities in health care and social determinants of health status are considered to be factors that contribute to the onset of diabetes and secondary complications [4]. These chronic diseases reduce the quality of life and raise medical costs in the United States. There is a need for more health promotion programs that address diabetes complications and encourage behavior that may help delay the onset of diabetes among high-risk groups.
Diabetes mellitus is a chronic disease and has three major forms. Type 2 diabetes (T2DM) is the most common form and represents 90% to 95% of diagnosed diabetes cases [3]. The risk of T2DM is associated with not only family history, aging, and obesity, but also lifestyle, dietary intake, and physical inactivity. However, the causes of T2DM are complicated and not yet clarified. If blood glucose concentrations are uncontrolled, complications such as cardiovascular disease and kidney failure may develop [5-6]. High consumption of refined starchy foods and low consumption of fruits and vegetables are believed to be factors responsible for the rising prevalence of diabetes and obesity. Dietary Guidelines for Americans 2010 encouraged consuming more dietary fiber throughout the day. The average dietary fiber intake of Americans is only 15 g/day, which is only about half of the recommended amount based on a diet consuming 2000 kcal/day. The Adequate Intake (AI) for dietary fiber is 28 g for female adults and 35 g for male adults.

Some studies have shown that resistant starch (RS), a type of dietary fiber, has favorable physiological effects on glycemic and insulin response [7-8]. Although the mechanism of RS on glycemic control has remained unclear, increased RS consumption may improve risk factors for diabetes [8].
Rice is a staple carbohydrate source in many Asian countries and worldwide [9]. In the United States, rice is consumed in significant amounts by the adult population [10]. Rice is considered a carbohydrate with low to moderate dietary fiber content, including varying amounts of RS. Rice is considered a high glycemic index food [11] and recommended for moderate consumption by diabetics [12]. However, the ratio of RS in rice depends on the rice varieties, heating/cooling treatments, and cooking processes [13-14]. Rice with a higher RS content may cause a reduced postprandial blood glucose response and serve the goals of better controlling blood glucose and delaying diabetes onset or progression [15].

Understanding the additional physiological effects of rice consumption may reveal implications for those people with diabetes who commonly consume rice as a main dish. This literature review will provide background information on diabetes, resistant starch, and rice.

**1.2 DIABETES MELLITUS**

Insulin and glucagon are hormones that regulate blood glucose concentrations. Insulin is released from the pancreas when blood glucose concentrations are
elevated. Insulin triggers the uptake of glucose from the blood into the cells for use as energy or storage. The removal of glucose from the bloodstream causes blood glucose concentrations to return to the normal range. However, diabetics have compromised blood glucose regulation because of having either cellular resistance to insulin or insufficient insulin secretion.

1.2.1 Diagnosis of Diabetes Mellitus

The diagnosis of diabetes is based on an individual’s glucose tolerance. Fasting blood glucose (FBG) and the oral glucose tolerance test (OGTT) are the common diagnostic tests for diabetes.

For the determination of FBG, an individual fasts for 8–10 hours prior to blood sampling. A normal fasting blood glucose concentration is less than 100 mg/dl (Table 1-1). According to the criteria of the American Diabetes Association (ADA), an individual with a fasting blood glucose greater than 100 mg/dl (5.6 mmol/l) but below 125 mg/dl (6.9 mmol/l) is classified as having impaired fasting blood glucose (IFG), and fasting blood glucose greater than or equal to 126 mg/dl (7 mmol/l) is indicative of diabetes.
The OGTT evaluates postprandial blood glucose concentration two hours after the consumption of a beverage containing 75 g of glucose. After ingesting the glucose beverage, a healthy individual’s blood glucose concentration is expected to rise rapidly during the first 30–45 minutes and decline gradually thereafter, eventually returning to normal. Impaired blood glucose regulation results in elevated postprandial blood glucose concentrations that remain high during the 2-hour test period. Impaired glucose tolerance is diagnosed when the 2-hour plasma glucose level is between 140 mg/dl and 199 mg/dl (Table 1-1). Diabetes is diagnosed when the 2-hour plasma glucose level is greater than 200 mg/dl [16]. The hemoglobin A1C test is a widely used biomarker of chronic glycemia, reflecting the average of blood glucose concentrations over two to three months. The threshold of the hemoglobin A1C test is 6.5%; diabetes is diagnosed when the hemoglobin A1C is greater than 6.5%. An A1C value of less than 5.7% is a normal blood glucose concentration. Pre-diabetes is diagnosed when the A1C value is more than 5.7% but less than 6.4%.
1.2.2 Blood Glucose Regulation

Insulin is a peptide hormone secreted by the pancreatic β-cells located in the islets of Langerhans. Endogenous insulin is the principal hormone responsible for regulating glucose uptake. Insulin triggers the movement of insulin-responsive glucose transport proteins (GLUT) from the cytosol to the surface of the cell membrane. When active GLUTs shift to the plasma membrane, glucose moves across from the blood stream into the cells [17]. Dysfunction of GLUTs may result in insulin resistance and failure to regulate the glucose uptake [18]. Under normal conditions, pancreatic β-cells secrete insulin when plasma glucose rises above 5 mmol/l. Insulin secretion is suppressed when blood glucose levels decline to below 81 mg/dl. When blood glucose drops to approximately 68 mg/dl, glucagon secretion from pancreatic α-cells is stimulated. Glucagon is another key regulatory hormone for glucose homeostasis. Glucagon induces hepatic glucose output to raise blood glucose concentrations into the normal range.

1.2.3 Types of Diabetes Mellitus

Pre-diabetes

Pre-diabetes is an abnormal state of glucose regulation that does not meet the
diagnostic criteria of diabetes, but is characterized by higher than normal blood glucose concentrations (Table 1-1) [16, 19]. Individuals with pre-diabetes may have either IFG or IGT conditions alone or combined. Pre-diabetes typically precedes the diagnosis of T2DM. Early diagnosis and maintenance of blood glucose control may delay the progression to T2DM and the development of other complications. Appropriate lifestyle interventions and exercise can improve IGT in obese individuals [20]. In one study, caloric restriction combined with aerobic exercise training increased insulin sensitivity among overweight and obese postmenopausal women with IGT, thus reducing the risk of T2DM development [20]. Ryan et al. suggested promoting lifestyle modifications including bodyweight loss and physical activity to increase insulin sensitivity and prevent glucose intolerance and T2DM in older adults [21].

In the United States, the crude prevalence of either IFG or IGT was approximately 29.5% among individuals aged above 20 years old. The pre-diabetes crude prevalence reached 46.7% in the segment of the population aged above 75 years old [22]. Aging is an important factor that contributes to metabolism diseases, which steadily increase in prevalence among people as they age. The incidence of hyperglycemia has been found to be much higher in men than in women [22].
Pre-diabetes indicates abnormal glucose homeostasis and results in high risk of developing T2DM, cardiovascular diseases, and other diabetic complications [19].

Type 1 diabetes mellitus

Type 1 diabetes mellitus (T1DM) accounts for approximately 5–10% of diagnosed diabetes. This type of diabetes was previously termed insulin-dependent diabetes mellitus or juvenile-onset diabetes. Type 1 diabetes results from primary failure of the pancreatic β-cells. The pancreas is unable to produce insulin, resulting in inability to maintain normal blood glucose concentrations. The etiology of this type of diabetes is complex, but it is better understood than T2DM. Type 1 diabetes is considered an autoimmune disease, in which the immune system destroys insulin-producing pancreatic β-cells. Additionally, environmental factors and genetic susceptibility have been implicated in the pathogenesis of T1DM [23-24]. For instance, viral infection may cause β-cell failure. The infected pancreatic β-cell may have increased endoplasmic reticulum (ER) stress, which make the β-cells susceptible to apoptosis. Elevated ER stress may also cause collateral damage to β-cell proteins, which may invoke an autoimmune response to pancreatic β-cell destruction [23]. Dysfunction of pancreatic β-cells leads toward onset of T1DM.
Currently there are no preventative treatments for individuals at risk for T1DM [24].

Although T1DM can develop at any age, it is commonly diagnosed in childhood and adolescence [16]. The destruction of β-cells can rapidly accelerate in infants and children, rendering the β-cells unable to produce insulin. Lack of insulin secretion causes hyperglycemia and ketoacidosis, and without effective interventions, can cause coma and death [25]. Individuals with T1DM are dependent on daily insulin injections or an insulin pump to regulate blood glucose levels and maintain normal glucose metabolism.

**Type 2 diabetes mellitus**

In 2010, the Centers for Disease Control and Prevention (CDC) reported that about 25.6 million people in the United States had diabetes [2]. Approximately 90–95% of diagnosed cases of diabetes are T2DM [3]. This disease was previously referred to as non-insulin dependent diabetes mellitus or adult onset diabetes. Type 2 diabetes develops gradually over a long time period and is considered a chronic disease. Type 2 diabetes is often asymptomatic in its early stages and can remain undiagnosed for many years. Because the signs and symptoms are subtle, they may be neglected. These symptoms include polyuria, polydipsia, polyphagia, weight loss,
and blurred vision. If untreated, T2DM results in severe complications, e.g. impaired vision, heart diseases, and hypertension [26]. Type 2 diabetes is caused by a defective intracellular response to insulin and reduced glucose transportation activity [27]. Insulin resistance (e.g. muscle and adipose tissue) and obesity have shown strong associations with risk of developing T2DM [28]. Most people suffering from T2DM are either overweight or obese. Excessive adipose tissue is considered a risk factor for developing insulin resistance. Ros Perez et al. indicated that increased storage of adipose tissue is related to the inflammatory response leading to the development of insulin resistance [29]. However, the cause of T2DM is not completely understood.

Type 2 diabetes usually begins with insulin resistance. Insulin resistance occurs when insulin’s target receptors fail to respond to insulin or the response is diminished, thus reducing insulin sensitivity. The potential causes of insulin resistance include aging, pregnancy, certain medications, low physical activity level, and obesity.

High blood glucose concentration, also known as hyperglycemia, is a common characteristic found in all diabetic conditions. Chronically elevated blood glucose concentrations induce pancreatic β-cells to produce excessive insulin in order to
return abnormally high blood glucose levels back to normal range. Excessive insulin secretion damages pancreatic β-cells and can eventually lead to β-cell failure and reduced insulin production over time. Chronic hyperglycemia plays a major role in the initiation of diabetic microvascular complications, including retinopathy, neuropathy, and nephropathy. Hyperglycemia can also increase the risk of cardiovascular diseases and cause hyperglycemia-induced cardiomyopathy [30-31]. These metabolic dysregulations may increase the risk of T2DM, along with other long-term complications.

Epidemiological research has demonstrated that the rates of T2DM vary by race and ethnicity. According to the 2007 National Diabetes Fact Sheet, African Americans, Hispanic/Latino Americans, American Indians, and some Asian Americans are particular ethnic groups with a high risk of T2DM and its complications [3]. After prevalence is adjusted for group age differences, 2004–2006 national survey data for adults showed that 10.4% of Hispanics, 6.6% of non-Hispanic whites, 7.5% of Asian Americans, and 11.8% of non-Hispanic blacks had diabetes [3]. Furthermore, Native Hawaiians and other Pacific Islanders are particular ethnic groups with a high risk of diabetes and its secondary complications [6]. The prevalence of diabetes, obesity, and hypertension is higher in Native
Hawaiians than in other ethnic groups in Hawai‘i, such as Filipinos and Japanese.

Diabetes is the fourth leading cause of death in the Native Hawaiian population. The age-adjusted death rate for diabetes was 38.8 (per 100,000) for Native Hawaiians, which was much higher than for other ethnic groups in Hawai‘i; for example, it was 20.7 for Filipinos and 12.0 for Japanese [5]. The prevalence of T2DM among Native Hawaiian adults was 19% to 24%, and approximately 15% to 47% for individuals with IGT or IFT [6]. Native Hawaiian adults also have a higher prevalence of diabetic complications that include cardiovascular diseases, hypertension, renal diseases and kidney failure, and retinopathy [32].

*Gestational diabetes mellitus*

Gestational diabetes mellitus (GDM) has been defined as “carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy” [33]. Several temporary alterations have been observed during pregnancy including decreased insulin sensitivity and altered hepatic glucose and/or adipose tissue metabolism. Gestational diabetes mellitus affects 2–5% of pregnant women, and GDM typically resolves after delivery [34]. The incidence of GDM is higher among African American, Hispanic, American Indian, and Asian women than among
Caucasian women [34]. According to the National Diabetes Fact Sheet, 2011, the CDC indicated that approximately 5–10% of pregnant women with GDM were immediately diagnosed with diabetes after pregnancy. Type 2 diabetes was found to be the most common type among them [2]. Moreover, women who have had GDM during pregnancy have a 40% to 60% incidence of developing diabetes in the following 10 to 20 years [2-3]. The American Diabetes Association stated that, as there is an ongoing epidemic of obesity and diabetes, which are significantly associated with the development of T2DM in women of childbearing age, the number of undiagnosed cases of T2DM in pregnant women has increased [16].

The pathophysiology of GDM is controversial. Retnakaran et al. suggested that both GDM and T2DM have a similar pathophysiology. Metabolic defects involve insulin resistance in target cells, decreased insulin activity, and insufficient pancreatic β-cell insulin secretion [35].

Although the onset of GDM is difficult to predict, various risk factors may be associated with the development of GDM during pregnancy. The risk factors that are associated with developing GDM include family history of diabetes, certain ethnicities, overweight (body weight in pregnancy > 110% of ideal body weight) or obesity (body mass index > 30), age above 25 years old, persistent glucosuria,
polycystic ovarian syndrome, hypertension or pregnancy-related hypertension, a
history of macrosomia (birth weight > 4 kg), and a history of GDM in a previous
pregnancy. These risk factors are identified in approximately 50% of individuals
with GDM, and other risk factors remain unknown.

After delivery, GDM resolves for most women. However, affected women are
at risk of developing metabolic syndromes in later life [36-37]. Vrachnis et al.
indicated that women with a previous history of GDM are at increased risk of
cardiovascular diseases, stroke, dyslipidemia, insulin resistance, and T2DM [36].

Daily self-monitoring of glucose and urinary ketones is recommended for
women with GDM. Moderate exercise may also improve insulin sensitivity. Insulin
therapy may be prescribed if individuals have uncontrolled blood glucose values,
where the value is 5 mmol/l for fasting blood glucose and/or 7.2 mmol/l for 1-hour
postprandial glucose [38]. Follow-up programs for women with GDM can reduce
their potential risk of developing T2DM and cardiovascular diseases.

Overall, the prevalence of diabetes continues to rise worldwide. The crude
prevalence of diagnosed diabetes in individuals 20 years old and above was 7.7%
[22]. The prevalence was 17.6% in individuals aged between 60 and 74 years, and
slightly less, 14.9%, in those aged 75 years old and above. The combined crude
prevalence of diabetes (included diagnosed and undiagnosed) and pre-diabetes was 42.3% in the population aged above 20 years old, and 75.7% in the population aged above 75 years old [22]. Wild et al. estimated that the global prevalence of diabetes was 2.2% in 2000, and this report predicted that the global prevalence for all age groups will reach 4.4% in 2030 [39]. Consequently, screening, prevention, and intervention are required in order to delay the onset of diabetes and its complications.

1.3 GLYCEMIC INDEX AND GLYCEMIC LOAD

Glycemic index (GI) is a ratio of the blood glucose response to a test food to that of a reference food. Pure glucose or white bread is commonly used as the reference food. Glycemic index is based on continuous measurement of postprandial blood glucose response for 2 hours after consumption of a portion of food containing 50 g of carbohydrate. Complex carbohydrates require longer digestion time and delay the glycemic response. Foods containing certain simple carbohydrates, such as glucose and maltose, induce a more rapid rise in blood glucose concentrations than complex carbohydrates, over the postprandial period.
High GI foods trigger an enhanced insulin response, which may result in a sharp drop in blood glucose concentrations postprandially [40].

Glycemic index is also affected by meal composition (e.g. protein and/or fat present), starch chemical structure (e.g. amylopectin and amylose), food processing, and dietary fiber content. Glycemic index can be a useful nutritional tool to evaluate the relationship between carbohydrate-rich food and glycemic response. Glycemic index may be a helpful reference for individuals with T2DM to use as a dietary guideline [41]. Riccardi et al. showed that GI is available and useful for clinical practice in diabetics. Low-GI foods have consistently shown beneficial effects on glycemic control in both the short term and the long term [42]. Low GI foods may also improve blood lipid concentration and prevent further diabetic complications [43].

Glycemic load (GL) takes into account the total amount of carbohydrate consumed. The GL calculation of a food is its GI multiplied by the number of grams of available carbohydrate of one serving, then divided by 100 [41]. Some researchers believe that the GL better predicts the postprandial blood glucose response and is a better tool for meal planning than GI alone. Previous studies have indicated that long-term consumption of a diet with high GI and high GL foods is
associated with increased risk of developing T2DM and other chronic diseases, such as heart disease [44-46].

Area under the curve (AUC) is a calculation of glycemic response for 2 to 3 hours after consuming an examined food. Glycemic index and GL are calculated based on the area under the glycemic response curve, whereas the portion above basal glucose value to the blood glucose peak value. The AUC value affects the variability and mean of GI values [47-48].

1.4 DIETARY FIBER

The current definition of dietary fiber (DF), accepted by the Codex Alimentarius Commission, is carbohydrate polymers with ten or more monomeric units, which are not hydrolyzed by the endogenous enzymes in the small intestine of humans [49]. Dietary fibers are derived from plant cell walls. Common dietary sources of DF include whole grains, fruits, legumes, and vegetables [50]. In addition to being resistant to digestion in the small intestine, some DFs may be viscous and/or are fermented in the colon by bacteria [51].

Generally, DFs have been classified as either soluble or insoluble based on
their solubility in water. Soluble fibers are often viscous and able to form gels in the GI tract. This property slows gastric emptying and may slow nutrient absorption from the small intestine. Insoluble fibers often reduce the transit time in the small intestine and increase the fecal bulk. Dietary fiber is recognized as a factor in the prevention of chronic diseases. The physiochemical properties of DFs contribute to many physiological effects, including the reduction of GI transit time, decreased blood cholesterol, improved regulation of blood glucose [52], and enhanced laxation [51]. By these mechanisms, increased consumption of DFs is significantly associated with reduced risk of hypertension, cardiovascular diseases, diabetes, obesity, and gastrointestinal disorders [53-54].

Dietary fibers are fermented by colonic bacteria and produce short-chain fatty acids (SCFAs), such as acetate, propionate, and butyrate [55]. Butyrate is the primary energy source for the colonocytes [56]. Short-chain fatty acids are associated with improving lipid homeostasis [57], increasing the gene expression of specific receptors (e.g. GPR41 and GPR43) [58] and improving colon health.

According to the Dietary Reference Intakes, the Adequate Intake (AI) of dietary fiber for adults ages 19 years and above is 25 g/day for females and 38 g/day for males. Adequate Intake is based on 14 g per 1000 kcal. However, the average
consumption of DF in the United States is about 15 g per day [59]. Inadequate consumption of whole grains, legumes, vegetables, and fruits may contribute to low DF intake. Thus, the 2010 Dietary Guidelines for Americans encouraged increased consumption of these foods to meet DF requirements and prevent chronic diseases.

1.5 RESISTANT STARCH

1.5.1 Definition and Chemistry

The definition of resistant starch (RS) is “the sum of starch and products of the starch degradation not absorbed in the small intestine of healthy individuals” [60]. Resistant starch is resistant to digestive enzymes in the small intestine and enters the large intestine, where some types are fermented [61]. Considering its chemical nature and poor digestibility, RS has been classified as a type of dietary fiber. Resistant starch content varies in starchy foods and has been correlated with altering the digestibility and sensory properties of foods, such as rice varieties.

Resistant starch is categorized into four main subtypes (Table1-2) [62-64]. Resistant starch type 1 (RS I) is a closely wrapped starch granule that is dehydrated, minimizing physical accessibility to digestive enzymes, such as in partly milled
grains and sesame seeds. More extensive milling increases the accessibility and decreases the resistance of RSI. Resistant starch type 2 (RS II) is the raw, poorly gelatinized native starch molecule in granular form, which is slowly hydrolyzed, such as raw potato or green bananas. Resistant starch type 3 (RS III) is a retrograded starch. This type of starch is commonly found in many heat-processed starchy foods. Gelatinized starchy foods that have undergone a cooling process have an increase in RS III. Foods containing RS III have relatively reduced digestible carbohydrate content, while DF content is relatively increased. Resistant starch type 4 (RS IV) is a chemically modified form with new glycosidic linkages; this new structure cannot be further broken down by digestive enzymes [7-8]. All RS types have various granule structures and solubility.

Several factors affect the resistant starch quantities of starchy foods during food processing. These factors include water content, pH, food processing (e.g. cooking methods, heating temperature, and time), the presence of other nutrient components (e.g. protein and lipid), heating and cooling cycles, freezing, and source [13-14]. Different cooking methods change the solubility of starchy food during the heating and cooling cycling. The new structure of retrograded starch that occurs in the food may reduce the digestibility and the nutrient absorption in the human GI
tract and may affect physiological properties of the food.

1.5.2 General Physiological Effects of Resistant Starch

The physiological effects of resistant starch have been studied in animals and human clinical trials [13, 60, 65]. Systemic effects in humans include improving insulin sensitivity [15, 66]; decreasing postprandial blood glucose and insulin response [15, 67-69]; increasing absorption of minerals, such as calcium and iron [70-71]; changing the microflora composition and reducing symptoms of diarrhea [72]; reducing blood cholesterol and fat oxidation [65]; and aiding in weight loss [60]. Some types of RS are fermented by gut bacteria, which increase production of SCFAs. Short-chain fatty acids mediate fecal water content and lower the colonic pH [61]. Although in vivo studies have demonstrated health benefits from RS consumption, the mechanism of RS remains unclear.

1.5.3 Impact on Glycemic Response and Insulin Sensitivity

Previous studies have reached no consistent agreement on the effects of RS on improving insulin sensitivity and decreasing postprandial glycemic and insulin responses [73-74]. High RS consumption (60 g/d of Novelose 260) lowered not
only the postprandial glycemic response, but also the postprandial insulin concentrations of 10 healthy subjects [15]. The mechanism of action may reflect the physiological properties of SCFA produced by fermentation. Robertson et al. inferred that increased SCFA production from RS consumption contributed to the improvement in insulin sensitivity via altered signal transduction [15]. A long-term intervention of 40 g RS/day (Hi-Maize 260) showed a significant improvement in tissue insulin sensitivity over 12 weeks, in healthy subjects [66]. Yamada et al. demonstrated that 6 g RS from tapioca reduced postprandial blood glucose and insulin responses of 20 subjects with and without normal blood glucose levels [67]. Subjects were divided into two groups based on fasting blood glucose levels (100–110 mg/dl and 111–140 mg/dl). Resistant starch significantly decreased glycemic response and insulin secretion in those subjects who had an abnormal blood glucose level (fasting blood glucose level between 111 and 140 mg/dl) [67]. In healthy individuals, RS showed no risk of inducing hypoglycemia or any other harmful down-regulation effects.

However, the beneficial effects of RS on glycemic response and insulin concentration have not been consistently found. The amount of RS used among studies is as varied as the types of RS. For instance, Novelose 260 and Hi-Maize
260 belong to the RSII type, unlike tapioca starch, which mostly contains RSIII, or retrograded starch. Each type of RS has unique characteristics such as varied degrees of digestibility and fermentability; these variations may contribute to inconsistent results.

The mechanism of RS action on suppression of postprandial glycemic and insulin response and on improvement of insulin sensitivity requires further investigation. Future studies on the role of SCFA in enhancing insulin sensitivity as well as to identify the optimal dose and RS type are necessary. Until these studies are conducted, no conclusions can be drawn regarding the benefit of consuming RS for blood glucose regulation.

1.5.4 Food Sources of Resistant Starch

Rice consists of RS and digestible starch. Current data show that RS content in rice is inconsistent due to the variety of rice cultivars. The RS content varies widely, from 0.8 g to 26.1 g per cup of cooked rice [75-76]. Reports show that the amount of RS content in parboiled rice ranges from 72 g per 100 g short grain brown rice to 86.32 g per 100 g parboiled long grain brown rice after cooking.

Rice is a staple food in both developed and developing countries. Rice
consumption is gradually increasing within the U.S., even though rice is not a staple food in the American diet [77]. The carbohydrates in rice are starch and DF, and the digestibility of the starch is greatly influenced by the rice cultivar. Resistant starch content varies with rice cultivar and is correlated with an alteration of the digestibility and the sensory properties of the rice. Starch digestibility depends on the chemical structures of the starch and is influenced by processing and storage conditions [78].

Consumption of dietary carbohydrates is associated with the increased risk of developing metabolic disorders (e.g. diabetes), certain cancers, and other chronic diseases [12, 44-46, 79-80]. A meta-analysis found that higher white rice consumption is associated with a significantly elevated risk of T2DM, especially among Asian populations who consume rice as a staple food [9]. A dose-response analysis showed that each serving per day of white rice was associated with an 11% increase in risk of diabetes in the overall population [9]. Sun et al. suggested that substituting whole grains, such as brown rice, for white rice may reduce the risk of T2DM [81].
### Table 1-1. The diagnostic criteria of diabetes

<table>
<thead>
<tr>
<th></th>
<th>Pre-diabetes</th>
<th>Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>FBG</td>
<td>100–125 mg/dl (5.6–6.9 mmol/l)</td>
<td>≥ 126 mg/dl (7 mmol/l)</td>
</tr>
<tr>
<td>OGTT</td>
<td>140–199 mg/dl (7.8–11.0 mmol/l)</td>
<td>≥ 200 mg/dl (11.1 mmol/l)</td>
</tr>
<tr>
<td>Hemoglobin A1C</td>
<td>5.7–6.4%</td>
<td>≥ 6.5%</td>
</tr>
</tbody>
</table>

### Table 1-2. Classification of resistant starch

<table>
<thead>
<tr>
<th>Types</th>
<th>Description</th>
<th>Examples of Food Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>RS I</td>
<td>Physically inaccessible to digestive enzymes</td>
<td>Partly milled grains and seeds</td>
</tr>
<tr>
<td>RS II</td>
<td>Ungelatinized and slowly enzymatic hydrolyzed starches</td>
<td>Raw potato, green banana, legumes, and high amylose starches</td>
</tr>
<tr>
<td>RS III</td>
<td>Retrograded starches</td>
<td>Cooked and cooled potato, bread, cornflakes</td>
</tr>
<tr>
<td>RS IV</td>
<td>Chemically modified starches (processed starches)</td>
<td>Modified starches through esterification, etherization, crosslinking, or transglycosylation</td>
</tr>
</tbody>
</table>

* Modified classifications from Bird et al. [64] and Topping et al. [62]
CHAPTER 2
EFFECT OF VARIETY AND COOKING METHOD ON RESISTANT STARCH CONTENT OF WHITE RICE AND SUBSEQUENT POSTPRANDIAL GLUCOSE RESPONSE AND APPETITE IN HUMANS

The following chapter is under review for publication in the Journal of the Science of Food and Agriculture, October 2012.

2.1 INTRODUCTION

Resistant starch (RS) is naturally found in starchy foods such as potato, corn, and rice. Due to its chemical nature and low digestibility, RS is considered a type of dietary fiber. Resistant starch is classified into four subtypes based on its physicochemical properties. Type 1 (RS I) is physically inaccessible starch granules, such as seeds. Type 2 (RS II) is native granular starch, such as that found in potato and banana. Type 3 (RS III) is retrograded starch made by cooking/cooling processes on starchy materials. Type 4 (RS IV) is chemically modified starch. Foods containing RS III have relatively lower digestible carbohydrate content, while their dietary fiber content is relatively higher.

Resistant starch escapes digestion in the stomach and small intestine and enters into the large intestine, where it may be fermented by colonic microbiota to produce
SCFAs, which lower the colonic pH [61]. Additionally, systemic effects of RS include improving insulin sensitivity [15, 66] and decreasing the postprandial blood glucose and insulin response in healthy subjects and subjects with elevated fasting glucose [15, 67-69]. However, not all studies have shown reduction in glycemic response or fasting glucose concentrations after RS consumption [65, 82]. These differences may be attributed to food form, source of RS, and subject characteristics.

Rice is a staple carbohydrate source in many Asian countries. The consumption of rice has gradually increased within the United States during the past decade, even though rice is not a staple carbohydrate of the American diet [10]. Rice cultivars vary in RS content; RS content is dependent on original amylose content. Resistant starch content may be increased during food processing, cooling, and storage [78]. Current data on RS content of rice are inconsistent, ranging from 0.8 g to 26.1 g per cup of cooked rice [83]. Although the GI of rice has been studied by several groups, this is the first study to consider RS content when evaluating postprandial glycemic response to rice [11, 84]. Resistant starch from rice has the potential to improve human health; however, RS content must first be better characterized, and subsequent clinical trials are necessary to confirm a physiological benefit. The
objectives of the present study were to (1) determine the effect of cooking method and refrigeration on the RS content of four varieties of commonly consumed white rice, and (2) assess the impact of RS on postprandial glucose response and appetite ratings in 21 healthy adults. This study hypothesizes that rice variety will significantly impact RS content, and rice with a high RS content will result in a lower postprandial glycemic response.

2.2 MATERIALS AND METHODS

2.2.1 Determination of RS in Rice

A survey of available rice varieties was conducted in four major supermarkets in Honolulu, Hawai‘i. The most common brands of short-grain, medium-grain, long-grain, and jasmine rice were selected for analysis. Each rice variety was cooked using three different methods (oven baked, conventional rice cooker, and pressure cooker). The resistant starch content of fresh rice was measured immediately after preparation, while the RS content of refrigerated rice was measured after three days of storage at 4°C.
Resistant starch was measured by the AOAC Official Method 2002.02, using a commercially available assay kit (Megazyme International, Ireland). All samples were analyzed in duplicate. Unless otherwise noted, all reagents were obtained from Sigma-Aldrich (St. Louis, MO, USA). Each rice sample was ground using a mortar and pestle. The sample (0.5 g) was combined with 4 mL pancreatic α-amylase (10 mg mL\(^{-1}\)) containing amyloglucosidase (3 U mL\(^{-1}\)). Samples were incubated in a shaking water bath at 37°C, with continuous shaking for 16 hours. Upon removal from the water bath, ethanol (4 mL, 99% v v\(^{-1}\)) was added to each sample, and samples were vortexed. The samples were then centrifuged at 2,000 g for 10 minutes. Supernatants were decanted. Pellets were re-suspended with 2 mL ethanol (50% v v\(^{-1}\)) and vortexed. Additional ethanol (6 mL, 50% v v\(^{-1}\)) was added, and the tubes were vortexed and then centrifuged at 2,000 g for 10 minutes. The suspension, vortex, and centrifuge steps were repeated twice. A magnetic stir bar and 2 mL potassium hydroxide (2 M) were added to each remaining pellet. Samples were stirred and incubated in an ice water-bath for 20 minutes. Sodium acetate buffer (8 mL, 1.2 M, pH 3.8) was added to each tube, and 0.1 mL amyloglucosidase (3300 U mL\(^{-1}\)) was added immediately after. The tubes were mixed and placed in a water bath at 50°C for 30 minutes. Samples were centrifuged at 2,000 g for 10 minutes.
Aliquots (0.1 mL) of supernatants were transferred into clean glass test tubes. GOPOD reagent was added to the test tubes and the tubes were incubated in a water bath at 50°C for 20 minutes. Immediately upon removal, absorbance was measured at 510 nm compared with a reagent blank (0.1 mL of 100 mM sodium acetate buffer (pH 4.5) with GOPOD reagent) by spectrophotometer (Shimadzu UV-160U, Kyoto, Japan). Based on the RS analysis, the rice samples with the highest and lowest RS content were selected for use in the clinical study.

2.2.2 Determination of Postprandial Glucose Response and Appetite Ratings

Twenty-one healthy adults (12 men and 9 women) were recruited from the University of Hawai‘i at Mānoa and nearby communities via flyers and posters. Eligibility criteria included: (1) being aged between 18 and 65 years old; (2) being a non-smoker, non-vegetarian, and not taking any medication(s); (3) eating breakfast regularly; and (4) being able to fast for 12 hours and attend three morning study visits. Exclusion criteria included: (1) being pregnant or lactating; (2) having been diagnosed with disease(s) or taking medication(s); (3) being a smoker, and/or vegetarian; (4) having gained or lost weight greater than 10 lbs in the past three months; (5) being currently enrolled in other clinical studies; (6) having a restricted
eating habit; or (7) having a fasting blood glucose level > 6.99 mmol L\(^{-1}\). The research project was approved by the University of Hawaiʻi Committee on Human Studies (CHS approval number 17457), and written consent was obtained from the subjects after a full explanation of consent and the methods of the study. Prior to the first study visit, subjects completed an eating habits questionnaire to identify any restricted eating habits [85] and a health history questionnaire to confirm health status (see Appendix B). Fasting blood glucose concentration was measured with a OneTouch® Ultra blood glucose meter (LifeScan) to ensure subjects were not diabetic (blood glucose < 6.99 mmol L\(^{-1}\)). Study visits were completed no less than two days apart. Subjects completed a 1-day diet record (see Appendix B) on the day prior to each study visit. On the morning of a study visit, subjects arrived having fasted for the prior 12 hours. The study was a randomized, single-blind crossover study.

At each study visit, subjects consumed one of three treatments with one cup of water: glucose beverage (control, 50 g glucose), high RS rice (HRS), and low RS rice (LRS). The cooked rice portions contained 50 g available carbohydrate each (HRS: 4.4 g RS 50 g available carbohydrate\(^{-1}\), LRS: 0.4 g RS 50 g available carbohydrate\(^{-1}\)). Subjects’ fasting blood glucose was measured at the beginning of
the study visit (Time 0). Subjects were immediately presented with the test rice or glucose beverage and were required to consume the treatment within 15 minutes.

Blood glucose measurements were taken at 0, 15, 30, 45, 60, 90, and 120 minutes during the 2-hour study visit. Blood glucose area under the curve (AUC) was calculated using the trapezoidal rule. Glycemic index for HRS and LRS was calculated based on the glucose response AUC [86]. Participants’ response on an appetite survey (see Appendix C) were assessed with a 100 mm visual analogue scale (VAS). The subjects answered the following appetite survey questions at 0, 15, 30, 45, 60, and 120 minutes: “How hungry are you?” 0 = Not hungry at all and 100 = I have never been more hungry; “How satisfied do you feel?” 0 = I am completely empty and 100 = I cannot eat another bite; “How full do you feel?” 0 = Not full at all and 100 = Totally full; “How much do you think you can eat?” 0 = Nothing at all and 100 = A lot. The VAS ratings were quantified by measuring the distance from the left end of the scale to the point marked by the participant. All measurements were reported in millimeters. Appetite AUC was calculated using the trapezoidal rule. Dietary intake data were collected and analyzed using the Nutrition Data System for Research software version 4 (2010), developed by the Nutrition Coordinating Center, University of Minnesota. Total energy, fat, protein,
carbohydrate, saturated fatty acids, monounsaturated fatty acids, polyunsaturated fatty acids, total dietary fiber, soluble dietary fiber, and insoluble fiber were analyzed.

2.2.3 Statistical Analysis

Data were analyzed with SAS statistical software (Version 9.1.3, North Carolina, USA). Results are presented as mean ± standard deviation. Resistant starch content was compared among rice varieties and cooking methods using ANOVA (PROC GLM). Effect of refrigeration was determined using a t-test. Treatment effects on blood glucose concentrations and appetite ratings were determined using PROC MIXED to control for subject variation. Significant differences were determined at $p < 0.05$.

2.3 RESULTS

2.3.1 Effect of Cooking Method and Variety on RS Content of Rice

Within the fresh rice, cooking method did not significantly affect RS content of long grain, medium grain, or short grain rice varieties (Table 2-1). Pressure cooking
significantly reduced the RS content of fresh jasmine rice, compared to the rice cooker and oven baking. Within the pressure cooked and oven baked rice, rice variety had a significant effect on the RS content of fresh rice. Resistant starch content of refrigerated rice was significantly impacted by cooking method, with the rice cooker consistently producing higher RS content within a variety. Rice variety significantly influenced the RS content of refrigerated rice when rice was prepared with a rice cooker and when baked in the oven. Refrigeration significantly decreased the RS content of pressure cooked and oven baked jasmine rice, but had no effect on the other rice varieties or cooking method. Refrigerated long grain rice prepared with the rice cooker had the highest RS content (2.554 g RS 100 g as-eaten rice\(^{-1}\)). Refrigerated short grain rice prepared with the pressure cooker had the lowest RS content (0.201 g RS 100 g rice\(^{-1}\)). These two rice varieties were selected for use in the glycemic response trial.

**2.3.2 Effect of RS on Glycemic Response and Appetite**

Baseline demographics of the study subjects are shown as average and range of each characteristic in Table 2-2. Mean fasting blood glucose concentrations (\(t = 0\)) were not different among treatment groups (Table 2-3).
Blood glucose concentrations differed significantly at 30 min, 45 min, and 60 min, with glucose control resulting in higher concentrations than HRS and LRS. Glucose AUCs were significantly lower for HRS and LRS compared to glucose control. There was no statistical difference between the GI of the two rice treatments. Macronutrient intake and energy intake did not differ among treatment groups (Table 2-4). Appetite ratings did not differ significantly at each individual time point (Figure 2-1). Subjects reported significantly increased fullness and significantly lower desire to eat after consuming HRS and LRS compared to glucose control based on the total AUC (Figure 2-2).

2.4 DISCUSSION

Rice has been considered a carbohydrate with low to moderate dietary fiber content, based on total dietary fiber analysis (0.6–3.5 g dietary fiber 1 cup prepared rice$^{-1}$) [87] and resistant starch analysis (1.9–3.4 g RS 1 cup prepared rice$^{-1}$) [88]. However, resistant starch content varies with cooking process (steamed, boiled, strained, or pressure cooked) and rice cultivar, ranging from 0.8 g–26.1 g RS 1 cup prepared rice$^{-1}$ [83]. Results similar to this study’s were found by another research
group, Rashmi et al., although the range of RS content that they reported was smaller than that in the present study [83, 89]. Results from the study and cooling process were similar to or lower than those presented in previous reports. This may be attributed to cooking methods and/or rice cultivar.

Carbohydrate digestibility is influenced by intrinsic factors (e.g. food forms, granule shape, crystalline structure, etc.) and extrinsic factors (e.g. other food components). The rate of digestion in different rice varieties is modified due to the effect of cooked-rice particle size, preparation, cooking procedures (e.g. cooking time, heat treatment), and storage methods on their chemical structures (e.g. amylose) [83, 90-91]. Rice varieties with similar amylose content can result in different starch digestion rates and glycemic response due to different physicochemical properties (e.g. gelatinization) and the factors previously mentioned [91]. Jung et al. [92] reported that uncooked rice with less gelatinization resulted in lower glycemic and insulin responses than cooked rice. Gelatinization is correlated with digestibility of starch and metabolism responses.

Besides the cooling and drying processes, another confounding factor that has been considered to alter the RS content and the degree of RS formation is the length of cold storage. Ma et al. [93] indicated that the grain structure of various cooked
rice varieties was significantly affected by cold storage time. The cold room cooling method, a longer cooling process, resulted in a higher rate of retrogradation than air blast cooling (immediate cooling). In addition, Ma et al. also claimed that amylose content alters the retrogradation that can easily occur with high-amylose content rice varieties at 0–4°C [93]. In the present study, the state of gelatinization was not measured. The rice in this study was stored for 24–28 hrs at 4°C prior to RS analysis or consumption. Longer storage has the potential to increase RS content; however microbial growth could render the rice unfit for consumption. Including these characteristics in future studies may provide a better understanding of RS formation in foods.

White rice is typically considered a high GI food, as previously discussed by Atkinson et al. in a review article [94]. While the mean GI reported in this review was 73, the range of published GI values was quite wide: 43–94. This emphasizes the importance of considering rice variety and cooking method when making a generalization about the glycemic response to rice. Recently, GI for three Indian varieties of rice was evaluated using glucose as the reference food (50 g available carbohydrate) [84]. The GIs of the Indian rice were slightly lower than those reported in our study, ranging from 70.2 to 77. As early as 1992, amylose content of
rice was identified as a characteristic that could reduce the glycemic index of rice [11]. Further research on high-amylose varieties of rice is necessary to fully understand the variability in rice GI. The high GI of white rice is associated with higher risk of type 2 diabetes, particularly in Asian populations. The dose-response analysis showed that each serving a day of white rice consumption was significantly associated with an 11% increase in risk of diabetes in the overall population. Therefore, identification of lower GI rice may help protect populations against chronic disease development [9].

Increasing consumption of RS has shown beneficial effects on postprandial blood glucose and insulin concentrations in people with either normal or impaired blood glucose concentrations. Yamada et al. [67] reported that a single ingestion of bread containing 6 g RS significantly inhibited postprandial glucose and insulin responses in subjects with fasting blood glucose > 6.11 mmol L\(^{-1}\). The treatment had no effect on subjects with fasting blood glucose < 6.11 mmol L\(^{-1}\). Behall et al. [52] reported that the consumption of test muffins providing nine different combinations of different levels of β-glucan (averages of 0.3, 0.9, or 3.7) and RS (averages of 0.9, 3.4, or 6.5 g) resulted in a reversal relationship of postprandial blood glucose and insulin responses. The combination of resistant starch with β-glucan showed a
greater decrease in glucose and insulin than RS or β-glucan consumed alone.

Moreover, in another study, subjects who continuously consumed breads containing 8–13.4 g RS showed a significant reduction of glucose and insulin responses [95]. Maize-derived RS needed to be consumed at the 15–30 g level to improve insulin sensitivity in overweight or obese men, and this effect was not seen in women [96]. A clinical trial showed that a RS supplement (48 g RS) had no significant effect on the appetite and postprandial glycemic response in healthy adults [73]. In the present study, it is likely that the dose of 4.4 g RS was not sufficient to observe a acute change in glycemic response, particularly in healthy adults. The amount of rice consumed in the present study was a realistic serving size (1.25 cups cooked rice), and the RS dose in this study was realistically obtainable from foods as part of a normal, healthy diet.

The present study found no differences in appetite ratings between rice treatments; however, subjects felt more full and desired to eat less after consuming the rice than after consuming the glucose beverage. A similar finding was reported by Ranawana et al.: Basmati rice made subjects feel significantly less hungry, more full, and with a lower desire to eat compared to how they felt after consuming a sucrose sweetened beverage [97]. The increased fullness and satisfaction are likely
due to the solid nature of the rice. However, RS has been linked with appetite suppression and decreased energy intake [73, 98]. Resistant starch content of rice could influence appetite ratings, but further work is necessary to explore this mechanism. The maximum dose consumed in this study (4.4 g RS) may not be sufficient, and long-term studies would be more appropriate for evaluating this effect.

2.5 CONCLUSION

This study demonstrated that the RS content of rice is variable, depending on rice variety and cooking method. Although the rice samples studied had significantly different RS contents, these differences did not result in physiological differences in postprandial glycemic response in healthy adults. The study was limited by the small sample size and the short study period. Future work should evaluate RS content of novel varieties of rice and determine postprandial glucose response in a higher-risk group, such as adults with elevated fasting blood glucose concentrations.
### Table 2-1. Resistant starch content in 4 varieties of white rice, prepared by different cooking methods

<table>
<thead>
<tr>
<th>Variety</th>
<th>Jasmine</th>
<th>Long Grain</th>
<th>Medium Grain</th>
<th>Short Grain</th>
<th>p-value within cooking method§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fresh</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pressure</td>
<td>0.528 ± 0.011&lt;sup&gt;aAB&lt;/sup&gt;</td>
<td>0.575 ± 0.019&lt;sup&gt;A&lt;/sup&gt;</td>
<td>0.368 ± 0.080&lt;sup&gt;AB&lt;/sup&gt;</td>
<td>0.294 ± 0.023&lt;sup&gt;B&lt;/sup&gt;</td>
<td>0.0278</td>
</tr>
<tr>
<td>Rice cooker</td>
<td>0.923 ± 0.018&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.076 ± 0.2676</td>
<td>0.494 ± 0.117</td>
<td>0.378 ± 0.057</td>
<td>0.0746</td>
</tr>
<tr>
<td>Oven</td>
<td>0.953 ± 0.024&lt;sup&gt;bA&lt;/sup&gt;</td>
<td>0.723 ± 0.009&lt;sup&gt;B&lt;/sup&gt;</td>
<td>0.271 ± 0.010&lt;sup&gt;C&lt;/sup&gt;</td>
<td>0.375 ± 0.013&lt;sup&gt;D&lt;/sup&gt;</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td>p-value</td>
<td>within fresh varieties&lt;sup&gt;#&lt;/sup&gt;</td>
<td>0.0008</td>
<td>0.2057</td>
<td>0.296</td>
<td>0.3163</td>
</tr>
</tbody>
</table>

| Refrigerated  |               |               |               |               |                                 |
| Pressure      | 0.409 ± 0.019<sup>a*</sup> | 0.471 ± 0.230<sup>a</sup> | 0.257 ± 0.514<sup>a</sup> | 0.201 ± 0.068<sup>a</sup> | 0.0798                           |
| Rice cooker   | 0.744 ± 0.118<sup>bA</sup> | 2.554 ± 0.738<sup>bB</sup> | 0.863 ± 0.325<sup>bA</sup> | 0.809 ± 0.260<sup>bA</sup> | 0.0009                           |
| Oven          | 0.384 ± 0.033<sup>aA*</sup> | 0.667 ± 0.081<sup>aB</sup> | 0.342 ± 0.092<sup>aA</sup> | 0.234 ± 0.067<sup>aA</sup> | 0.0004                           |
| p-value       | within refrig. varieties<sup>#</sup> | 0.0011 | 0.0021 | 0.0045 | 0.0069 |

<sup>#</sup> Within a column of fresh or refrigerated samples, cells with different lower case superscripts are significantly different (<i>p</i> < 0.05).

ANOVA Model RS = cooking method, data grouped by variety and fresh/refrigerated, <i>n</i> = 2

<sup>§</sup> Within a row, cells with different upper case superscripts are significantly different (<i>p</i> < 0.05), ANOVA Model RS = variety, data grouped by cooking method and fresh/refrigerated, <i>n</i> = 2.

* indicates that resistant starch was significantly different between fresh and refrigerated samples (within variety and cooking method), t-test <i>p</i> < 0.05.
### Table 2-2. Demographics

<table>
<thead>
<tr>
<th></th>
<th>Male/Female (n)</th>
<th>Age (yr)</th>
<th>Height (m)</th>
<th>Weight (kg)</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>12/9</td>
<td>29.33</td>
<td>1.70</td>
<td>66.2</td>
<td>22.88</td>
</tr>
<tr>
<td>Range</td>
<td>--</td>
<td>22–57</td>
<td>1.47–1.90</td>
<td>49.1–102.7</td>
<td>18.53–30.12</td>
</tr>
</tbody>
</table>

### Table 2-3. Mean blood glucose concentrations

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Glucose beverage</th>
<th>High RS rice</th>
<th>Low RS rice</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Min</td>
<td>5.36 ± 0.13</td>
<td>5.33 ± 0.13</td>
<td>5.14 ± 0.11</td>
</tr>
<tr>
<td>15 Min</td>
<td>8.13 ± 0.28</td>
<td>7.67 ± 0.20</td>
<td>7.38 ± 0.24</td>
</tr>
<tr>
<td>30 Min</td>
<td>9.29 ± 0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.27 ± 0.26&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.93 ± 0.21&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>45 Min</td>
<td>8.98 ± 0.41&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.66 ± 0.28&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.57 ± 0.24&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>60 Min</td>
<td>8.07 ± 0.37&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.38 ± 0.31&lt;sup&gt;b&lt;/sup&gt;</td>
<td>6.71 ± 0.26&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>90 Min</td>
<td>6.92 ± 0.35</td>
<td>6.58 ± 0.27</td>
<td>5.99 ± 0.23</td>
</tr>
<tr>
<td>120 Min</td>
<td>5.06 ± 0.28</td>
<td>5.76 ± 0.22</td>
<td>5.55 ± 0.18</td>
</tr>
<tr>
<td>Total AUC</td>
<td>264.34 ± 24.36</td>
<td>211.16 ± 14.10</td>
<td>181.01 ± 12.00</td>
</tr>
<tr>
<td>Glycemic Index</td>
<td>--</td>
<td>83.95 ± 6.61</td>
<td>78.00 ± 10.62</td>
</tr>
</tbody>
</table>

*Different letters indicate that the data in the same column were significantly different between control and rice treatments, ANOVA (p < 0.05)*
<table>
<thead>
<tr>
<th></th>
<th>Glucose Beverage</th>
<th>High RS Rice</th>
<th>Low RS Rice</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy Intake (kJ)</strong></td>
<td>9755.03 ± 912.22</td>
<td>9917.48 ± 917.20</td>
<td>8662.11 ± 738.84</td>
<td>0.12</td>
</tr>
<tr>
<td><strong>Total Fat (g)</strong></td>
<td>102.33 ± 13.86</td>
<td>97.74 ± 12.61</td>
<td>82.15 ± 10.43</td>
<td>0.43</td>
</tr>
<tr>
<td>Saturated FA</td>
<td>31.61 ± 4.12</td>
<td>31.63 ± 4.36</td>
<td>29.78 ± 4.17</td>
<td>0.48</td>
</tr>
<tr>
<td>MUFA</td>
<td>40.24 ± 6.58</td>
<td>37.36 ± 5.10</td>
<td>29.94 ± 4.23</td>
<td>0.5</td>
</tr>
<tr>
<td>PUFA</td>
<td>21.77 ± 3.26</td>
<td>20.30 ± 2.88</td>
<td>15.28 ± 1.85</td>
<td>0.27</td>
</tr>
<tr>
<td><strong>Total Carb (g)</strong></td>
<td>253.29 ± 20.71</td>
<td>263.59 ± 22.14</td>
<td>243.23 ± 20.46</td>
<td>0.05</td>
</tr>
<tr>
<td><strong>Total Protein (g)</strong></td>
<td>104.32 ± 11.88</td>
<td>108.32 ± 11.39</td>
<td>93.01 ± 7.37</td>
<td>0.27</td>
</tr>
<tr>
<td><strong>Total Dietary Fiber (g)</strong></td>
<td>21.03 ± 2.39</td>
<td>18.91 ± 2.25</td>
<td>18.47 ± 2.30</td>
<td>0.24</td>
</tr>
<tr>
<td>Soluble Fiber</td>
<td>5.92 ± 1.00</td>
<td>5.25 ± 0.55</td>
<td>5.57 ± 0.68</td>
<td>0.19</td>
</tr>
<tr>
<td>Insoluble Fiber</td>
<td>15.00 ± 1.84</td>
<td>13.27 ± 1.82</td>
<td>14.25 ± 2.28</td>
<td>0.77</td>
</tr>
</tbody>
</table>

*\( n = 21 \), ANOVA was used to compare dietary outcomes across treatment
Figure 2-1. Mean appetite rating in response to glucose beverage and rice treatments over time.
Figure 2-2. Total appetite sensation AUC. *Data are presented as mean ± SD. Within a descriptor, values with different letters are significantly different ($p < 0.05$).
APPENDIX A: QUESTIONNAIRES

Name___________________________        Date_________

**Eating Habit Questionnaire**
The Glycemic Response to Rice Study

**Part 1**
Based on your personal eating habits, please check *True* or *False* after reading each statement.

<table>
<thead>
<tr>
<th>Statement</th>
<th>True</th>
<th>False</th>
</tr>
</thead>
<tbody>
<tr>
<td>When I have eaten my quota of calories, I am usually good about not eating any more.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I deliberately take small helpings at meals as a means of controlling my weight.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Life is too short to worry about dieting.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I have a pretty good idea of the number of calories in common food.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I enjoy eating too much to spoil it by counting my calories or watching my weight.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I often stop eating when I am not really full as a conscious means of limiting the amount that I eat.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I consciously hold back at meals in order not to gain weight.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I eat anything I want, anytime I want.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I count calories as a conscious means of controlling my weight.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I do not eat some foods because they make me fat.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I pay a great deal of attention to changes in my figure.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Part 2
Based on your personal eating habits, please circle the number that best answers the question.

How often are you dieting in a conscious effort to control your weight?

1 Rarely 2 Sometimes 3 Usually 4 Always

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

1 Not at all 2 Slightly 3 Moderately 4 Very much

Do your feelings of guilt about overeating help you to control your food intake?

1 Never 2 Rarely 3 Often 4 Always

How conscious are you of what you are eating?

1 Not at all 2 Slightly 3 Moderately 4 Extremely

How frequently do you avoid “stocking up” on tempting foods

1 Almost never 2 Seldom 3 Usually 4 Almost always

How likely are you to shop for low calorie foods?

1 Unlikely 2 Slightly unlikely 3 Moderately unlikely 4 Very likely

How likely are you to consciously eat slowly in order to cut down on how much you eat?
1 Unlikely
2 Slightly unlikely
3 Moderately unlikely
4 Very likely

How likely are you to consciously eat less than you want?

1 Unlikely
2 Slightly unlikely
3 Moderately unlikely
4 Very likely

On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never ‘giving in’), what number would you give yourself?

0 eat whatever you want, whenever you want it
1 usually eat whatever you want, whenever you want it
2 often eat whatever you want, whenever you want it
3 often limit food intake, but often ‘give in’
4 usually limit food intake, rarely ‘give in’
5 constantly limiting food intake, never ‘giving in’
Glycemic Response to Rice

Health History Questionnaire

Thank you for your interest in participating in our study. Before we can determine if you meet the criteria to participate in the study, we need to ask you some questions about your health history. You will be answering a health history questionnaire, but you do not need to answer any questions which you do not feel comfortable answering. If you do not meet the criteria for the study, we will destroy the information collected during this interview.

NAME: ____________________________________________________________

DATE ___________________

DATE OF BIRTH _______________ AGE _______________

HT____ WT_____ (office use : BMI ____)

Do you smoke or chew tobacco? ____  ____

For women, are you currently pregnant or lactating? ____  ____

For women, have you been pregnant or lactating within the last 6 months? ____  ____

For women, have you missed a menstrual cycle in the last 6 months? ____  ____

Are you a vegetarian? ____  ____

Do you eat rice? ____  ____

If YES, how often?

Do you have any food or other allergies? ____  ____

If YES, what are they?

Are you taking any medications to do the following: Yes No

Control blood sugar ____  ____

Control insulin ____  ____

Control appetite ____  ____

Lose weight ____  ____
Control depression  _____  _____

Antibiotics  _____  _____

Have you taken any medication in the past month (over the counter or prescription) or are you currently taking any medication?

Medications:  Dose/Frequency:

Have you ever been diagnosed with the following diseases or conditions:

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes (type I or type II)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperglycemia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperinsulinemia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoglycemia?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anorexia nervosa?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulimia nervosa?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Binge eating disorder?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any gastrointestinal conditions?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

If so, please explain:

Have you lost or gained weight in the past 3 months?  _____  _____

If yes, how much?

Do you consume alcohol?  _____  _____

If YES, how many drinks per week do you typically consume? (One drink = 12 oz beer or 4 oz wine or 1 oz hard liquor) _____

In the past 6 months, have you sought treatment for drug or alcohol abuse?  _____  _____
How would you rate your present state of health compared to other people about your age?
Excellent___ Good ___ Fair ___ Poor ___

Have you participated in a research study before? Yes No

If yes, when?

This study requires you to consume rice or a sweet beverage and give blood samples every 15 min for 2 hours. Are you willing and able to do this? Yes No

This study will include 3 morning visits to the University of Hawai‘i at Mānoa campus. Each visit will last approximately two and a half hours.

Are you willing to come to the University of Hawai‘i at Mānoa campus in a fasted state (nothing to eat for 12 hours) to have your blood drawn on 3 occasions? Yes No

This study requires you to record all the food that you eat for 24 hours before your study visit. This will be repeated 3 times.

Are you willing and able to do this? Yes No

Do you have reliable transportation? _______ _______

Do you travel out of the Honolulu area frequently? _______ _______

Are there specific dates you will not be available? _______ _______

Are you planning on living in the Honolulu area for the next 3 months? _______ _______

Are you able and willing to give blood samples? _______ _______

Have you ever had problems with giving blood? _______ _______

If yes, describe:

After hearing about the study, how do you feel about the time commitment and effort involved to complete the study?
ADDRESS_______________________________________________________

CITY____________________ STATE________ ZIP___________

TELEPHONE NUMBERS: Work (daytime):_____________________

Home (evening):_______________________________________

Email: ________________________

Best time of day to be reached ______________________

Comments:
APPENDIX B: FOOD RECORD

FOOD RECORD

Name: __________________________

Phone: __________________________

Date: ___________________________

SPECIAL INSTRUCTIONS:

Begin completing this food record on ___________________at 7:00 am.
Record your food intake until you arrive at the study visit.

The Glycemic Response to Rice
PI: Dr. Maria Stewart
808-956-9114
mstew@hawaii.edu
FOOD RECORD INSTRUCTIONS

In order to calculate your nutrient intake, a complete record must be kept of your food and beverage consumption for the period specified. Following are some rules in recording food intake that will help you to provide accurate intake data.

1. All meals and snacks MUST be recorded, whether they are eaten at home or away.

2. Be as specific as possible in recording food items. If the brand name of the product is available, please include it.

3. Measure food items when possible or estimate the quantities closely. You should estimate portions either in weight, such as grams or ounces, or in volume, such as teaspoons, tablespoons, cups, etc.

4. Be sure to specify the method of preparation for each food item. If additional products are used in preparation, such as oil to pan-fry a food, be sure they are included.

5. Make sure that all condiments, such as ketchup, gravy, or sauces, are recorded.

6. The abbreviations used for food intake weight and measures are as follows:
   
   tsp = teaspoon  
   Tbsp = tablespoon  
   c = cup  
   oz = ounce  
   ml = milliliter

7. Please use the following sample food record as a guide.
<table>
<thead>
<tr>
<th>Time</th>
<th>Food Eaten</th>
<th>Amount</th>
<th>Food Description and/or Preparation Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:30am</td>
<td>Frosted Mini Wheats cereal</td>
<td>1 ¼ cup</td>
<td>Kellogg’s</td>
</tr>
<tr>
<td></td>
<td>Milk</td>
<td>1/3 cup</td>
<td>Lucerne</td>
</tr>
<tr>
<td></td>
<td>POG juice drink</td>
<td>12 oz</td>
<td>Meadow Gold</td>
</tr>
<tr>
<td>10:00am</td>
<td>Coffee</td>
<td>12 oz</td>
<td>from break room</td>
</tr>
<tr>
<td></td>
<td>Equal</td>
<td>1 pkt</td>
<td>Original flavor</td>
</tr>
<tr>
<td></td>
<td>Coffeeemate</td>
<td>1 tsp</td>
<td></td>
</tr>
<tr>
<td>12:00pm</td>
<td>Roast beef sandwich</td>
<td>3 slices</td>
<td>deli slices, thin, roasted, no salt</td>
</tr>
<tr>
<td></td>
<td>Beef</td>
<td>2 slices</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Whole wheat bread</td>
<td>1 tsp</td>
<td>Promise margarine</td>
</tr>
<tr>
<td></td>
<td>Margarine</td>
<td>1 leaf</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lettuce</td>
<td>20 oz</td>
<td>snack size bag, Nacho Cheese</td>
</tr>
<tr>
<td></td>
<td>Diet Coke</td>
<td>1 bag</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Doritos</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6:00pm</td>
<td>Chicken Stirfry</td>
<td>6 pc</td>
<td>At friends house</td>
</tr>
<tr>
<td></td>
<td>Chicken pieces</td>
<td>½ c</td>
<td>cooked in canola oil</td>
</tr>
<tr>
<td></td>
<td>Broccoli</td>
<td>¼ c</td>
<td>White meat, ~ size of thumb</td>
</tr>
<tr>
<td></td>
<td>Onion</td>
<td>2 Tbsp</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Bamboo shoots</td>
<td>1 ½ c</td>
<td>White, cooked in rice cooker</td>
</tr>
<tr>
<td></td>
<td>Carrots</td>
<td>1 tsp?</td>
<td>Chinese 5-spice, garlic</td>
</tr>
<tr>
<td></td>
<td>Rice</td>
<td>3 Tbsp</td>
<td>Aloha brand</td>
</tr>
<tr>
<td></td>
<td>Seasoning</td>
<td></td>
<td>Miller Lite</td>
</tr>
<tr>
<td></td>
<td>Shoyu</td>
<td></td>
<td>Cadbury Milk Chocolate</td>
</tr>
<tr>
<td></td>
<td>Beer</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Chocolate bar</td>
<td>2-12 oz cans</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 oz</td>
<td></td>
</tr>
<tr>
<td>9:00pm</td>
<td>Water</td>
<td>16 oz</td>
<td>Tap, no ice</td>
</tr>
<tr>
<td>Time</td>
<td>Food Eaten</td>
<td>Amount</td>
<td>Food Description and/or Preparation Method</td>
</tr>
<tr>
<td>------</td>
<td>------------</td>
<td>--------</td>
<td>--------------------------------------------</td>
</tr>
</tbody>
</table>

S M T W T F S

Date: ________________________________
APPENDIX C: APPETITE SURVEY

Survey 1 - Appetite Assessment

Subject ID_____
Date_____
Time __ minutes

How hungry do you feel?

Not hungry at all ____________________________ I have never been more hungry

How satisfied do you feel?

I am completely empty ____________________________ I can’t eat another bite

How full do you feel?

Not at all full ____________________________ Totally full

How much do you think you can eat?

Nothing at all ____________________________ A lot
20. Ryan AS, Ortmeyer HK, Sorkin JD: Exercise with calorie restriction improves insulin sensitivity and glycogen synthase activity in obese postmenopausal women with impaired glucose tolerance. Am J Physiol Endocrinol Metab 2012,


60. Mary MM, Judith Spungen D, Anne B: Resistant Starch Intakes in the United...


78. Liu Q, Donner E, Yin Y, Huang RL, Fan MZ: The physicochemical properties


95. Behall KM, Hallfrisch J: Plasma glucose and insulin reduction after

