

School of Public Health

Subjective Hunger Sensations and Prospective Food Intake in Obese and **Overweight Subjects: Influence of Ethnicity**

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ABSTRACT

The thesis assessed the potential impact of ethnicity on subjective sensations of hunger and food intake. Eighteen Asian and twenty six Caucasian origin resided in Australia who were overweight/obese with or without weight fluctuation in the last 6 months had their hunger/satiety sensations tracked before and for 2h after an oral glucose load, which then followed by recorded food intake during lunch buffet meal and over 24h. There were no differences in hunger/satiety sensations and food intake between ethnic groups. Weight stability interacted with ethnicity to influence hunger/satiety sensations and food intake during buffet lunch.

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ABBREVIATIONS

A-MESH : alpha melanocyte-stimulating hormone

ANCOVA : Analysis of covariance

ARC : Arcuate nucleus
BMI : Body Mass Index
CCK : Cholecystokinin

CNS : Central Nervous System

DEXA : Dual Energy X-Ray Absorptiometry

GIP : Glucose dependent insulinotropic polypeptide

GLP-1 : Glucagon Peptide-1
GLUT : Glucose transporter

-h : hour

HOMA-IR: Homeostatic Model Assessment Insulin Resistance

iAUC : incremental Area Under the Curve

IGT : Impaired Glucose ToleranceIFG : Impaired Fasting Glycemia

IQR : Inter Quartile Range

kg : kilogram
mg : milligram
min : minutes

MONW : metabolic obese with normo-weight

NGT : Normal glucose tolerance

OGIS : Oral glucose insulin sensitivity
OGTT : Oral Glucose Tolerance Test
PYY : Peptide tyrosine-tyrosine

SEM : Standard Error

T2DM : Type 2 Diabetes Mellitus

TEFQ: Total Emotional Factor Questionnaire

tAUC : total Area Under the Curve

VAS : Visual Analog scale

WHO : World Health Organization
WHR : Waist-circumference ratio

CHAPTER ONE

INTRODUCTION

1.1 Statement of problem

The Australian Bureau of Statistics showed that prevalence of overweight and obesity among adults in Australia had increased from 56.3% in 1995 to 61.2% in 2007/8 and to 62.8% in 2012 (ABS, 2013). The latter year comprised of 35.4% overweight and 27.4% obese. In parallel, prevalence of diabetes in Australia had doubled since 1981 to 2000 and is predicted to reach around 1.8 million in 2030 (Whiting et al., 2011, Shaw et al., 2010).

Body weight and body energy content remains quite stable in most adults for long periods of time despite daily fluctuations in energy intake and energy expenditure. However, this requires the presence of regulatory processes able to match fuel supply to energy requirements to ensure stability of weight (Galgani and Ravussin, 2008, Martinez, 2000). The maintenance of a physiological set point for body weight is complex and includes aminostatic and glucostatic controls of feeding, metabolic or nutrient partitioning, input from the sympathetic nervous system, signals from adipose tissue as well as additional behavioral influences (Martinez, 2000). The interaction to maintain body weight focuses on an axis in which three different self-regulated components, namely food intake, nutrient turnover, thermogenesis and body fat store, interacts. Genetics, the environment and psychosocial factors also play a role in influencing these regulatory processes (Hill, 2006, Martinez, 2000).

Overweight and obesity has been viewed as secondary to appetite dysfunction (Speechly and Buffenstein, 2000). Increased food intake is closely related to increased calorie intake (Ello-Martin et al., 2005, Blundell and Gillett, 2001) which is then stored in the body as fat and be accumulated over certain period of time (Ello-Martin et al., 2005). Fat mass in the body was correlated with the release of hunger stimulating hormones, such as Ghrelin (Benedict et al., 2014). This leads to a vicious cycle in overweight and obese subjects.

Asian ethnicities, such as Indian and Chinese origin had a higher risk of accumulating body adipose tissue which could lead to obesity and other metabolic diseases in later life as compared to Caucasians (Schmiegelow et al., 2015, Al Rifai et al., 2015, Lu et al., 2014, Rasmussen-Torvik et al., 2012, Borai et al., 2009, Yajnik et al., 2003, Lubree et al., 2002, Mayer-Davis et al., 1997, Chiu et al., 2000). The above-mentioned ethnic groups have also been studied to have increased risk of developing insulin resistance at a lower BMI (Yajnik et al., 2015, Bavdekar et al., 1999). However, studies to investigate the role of ethnicity in influencing subjective hunger and satiety sensations as proxy indicator for food intake are lacking. We therefore assessed subjective hunger/satiety sensations after Oral Glucose Tolerance (OGTT) in relation to an acute and subsequent 24-h food intake among overweight and obese Asians and Caucasians. The advantages of using OGTT as a pretreatment condition are it provides a comparable amount of normal breakfast which is around 300 kcal and at the same time also serves as an indicator of Impaired Fasting Glucose or Impaired Glucose Tolerance. As glucose is one of the main drivers of hunger, an impaired response to glucose could play a role in influencing the subjective hunger /sensations and subsequent food intake, in addition to the potential role of ethnicity.

1.2 Aim and objectives of the study

The primary objective is:

to assess subjective appetite sensations on hunger, satiety and prospective food intake among Asian and Caucasian overweight/obese individuals residing in Australia.

The secondary objectives are:

- 1. to understand the influence of age, body composition, weight stability and impaired glycemia in modulating subjective hunger and food intake.
- to understand the relation between subjective hunger, satiety and prospective and 24-h food intake.
- 3. To understand the interaction between ethnicity and weight stability in modulating subjective hunger and food intake.

1.3 Relevance of the study

The prevalence of overweight/obesity in Australian adults has increased significantly during the last decade (ABS, 2013). Regulation of appetite which is influenced by insulinglucose physiology is an important factor in the energy balance equation (Batterham et al., 2007). Recent studies showed that insulin sensitivity was determined by ethnicity (Borai et al., 2009, Rasmussen-Torvik et al., 2012). The emerging ethnic mix resulted into a multicultural Australia with around 27% of the population now born outside the country from China, India, Malaysia and the Philippines (ABS, 2013). A greater understanding of the role of ethnicity in subjective sensations and food intake has important ramifications for weight control of the population.

1.4 Definition of terms

Ethnicity is defined by people who come from similar origin. In this study, we include study volunteers whose parents have similar ethnicity (Bhopal, 2006). Asian ethnicity in this study includes all ethnic groups in Asia starting from Northern Asia (China, Taiwan, Korea), South east Asia (Malaysia, Indonesia, Singapore) and South Asia (Indian, Sri Lanka and Pakistan). Caucasian ethnicity in this study includes all ethnic groups which originated in Europe which includes the United Kingdom and the rest of Europe.

Impaired glucose response is defined in accordance with the American Diabetes Association which classified study participants into Type 2 Diabetes Mellitus (T2DM), Impaired Glucose Tolerance (IGT) and Impaired Fasting Glucose (IFG) based on fasting and 2-h glucose level (Expert Committee on the and Classification of Diabetes, 2003).

1.5 Research overview

This research investigated the influence of ethnicity on subjective hunger and fullness sensations and its relation to prospective food intake. Chapter 2 comprises a literature review to discuss food intake regulation, food intake indicators, influencing factors of food intake which includes glucose-insulin metabolism, body composition, ethnicity & other environmental factors. The study methodology is outlined in Chapter 3, the results are presented in Chapter 4 and discussion and interpretation of these results is outlined in Chapter 5. Conclusions and recommendations for further research are presented in Chapter 6.

CHAPTER TWO

LITERATURE REVIEW

The literature review comprises the following sections: regulation of food intake, biomarkers for satiation, factors influencing food intake, appetite disruption and food preference in overweight and obese subjects, the role of ethnicity. A literature search for each section was conducted to follow some aspects of systematic review. Relevant English publication from 2005-2015 using PubMed, Scopus and Google scholar data base were retrieved. For regulation of food intake, following key words were included in the search: "eating" AND "food" AND "intake" OR "food intake" AND "social control" OR "social" AND "control" AND "formal" OR "formal social control" OR "regulation" AND" humans" or "human". Manual selection was conducted to exclude: anti-obesity drugs, pharmaceutical preparations, disorders, metabolic disturbance, childhood, overweight and obesity, anorexia nervosa, uremic anorexia, breastmilk, hyperthyroidism, hyperaldosteronism, nutrition intervention, cancer, neoplasms. Around 9491 publications were retrieved and 62 publications were reviewed.

2.1 Regulation of food intake

The pattern of eating in humans is episodic and includes main meals, snacks and drinks (Bisogni et al., 2007, de Graaf et al., 2004, Gibney and Wolever, 1997). People will generally eat until they are comfortably full which is termed satiation, and this is interspersed with periods of time with no eating, i.e. satiety (de Graaf et al., 2004). The drive to eat is usually low after a meal and builds up until the moment of the next eating episode (de Graaf et al., 2004, Gibney and Wolever, 1997). Episodes of hunger and satiety are closely related with the amount of glucose, presence of satiety-related hormones such as Ghrelin, Leptin, CCK and environmental factors such as cognition, emotion and rewards (de

Graaf et al., 2004, Austin and Marks, 2009, Peneau et al., 2009, Schwartz et al., 2000, Batterham et al., 2007).

The internal driving force for the search, choice and ingestion of food is defined as appetite (de Graaf et al., 2004) which is controlled by short and long term chemical and mechanical processes (Schwartz, 2006). The mechanical process starts in the mouth and also, at the same time, activates the chemical process by releasing several enzymes and hormones related to digestion and satiety. Several hormones are related to the chemical part of food digestion and satiety (Schwartz, 2006) such as insulin which acts as a short term signal of satiety (Verdich et al., 2001, VanderWeele, 1994, de Graaf et al., 2004). Insulin could directly work through the insulin receptors in the hypothalamus (Verdich et al., 2001) or indirectly through satiety-inducing peptides, such as CCK, GLP-1 and PYY (Riedy et al., 1995, Schmid et al., 1989, Fernandez-Garcia et al., 2013, Vrang et al., 2006). A reduction in post prandial glucose and an increase in post-prandial insulin promote satiety and suppressed hunger, thereby changing food intake (Tamam et al., 2012, Friedman and Tordoff, 1986, Tordoff and Friedman, 1986).

The role of glucose in food intake regulation/energy metabolism is divided into Central Nervous System (CNS) related and non–CNS related (de Graaf et al., 2004, Schwartz, 2010). The CNS mechanism evolved from the glucostatic theory postulated by Mayer et al in 1950s (Mayer, 1955) followed by several studies showing different glucose concentrations in influencing hunger through glucoreceptors in the brain. These receptors detected changes in the rate of glucose utilization and thus regulated short-term appetite (Gielkens et al., 1998, Chapman et al., 1998, Andrews et al., 1998a, Andrews et al., 1998b, Welle et al., 1980). The non-CNS mechanism involved stimulation of glucoreceptor or osmoreceptor in the small intestine which further influenced the release of insulin, incretin peptides or both (Schwartz et al., 2000).

In summary, glucose modulates short-term food intake. A decrease in glucose utilization and blood glucose concentration leads to transient and dynamic declines of blood glucose to which activate meal requests (Melanson et al., 1999).

Gut hormones such as Ghrelin and Cholecystokinin (CCK) influence the long-term regulation of food intake by involving the central nervous system regulation (Schwartz, 2006). Ghrelin is secreted by the stomach and is indirectly influenced by its stretch-receptor (de Graaf et al., 2004). It is known as orixegenic (appetite stimulant) hormone and actively participates in the control of food intake, meal size and meal termination (Woods, 2004, de Graaf et al., 2004). In contrast to Ghrelin, Cholecystokinin (CCK) is an anorexigenic hormone and is secreted by enteroendocrine cells into the intestinal lumen of the stomach and the small intestine. CCK reaches its circulating peak around 25 minutes after food ingestion and stays elevated for three hours (Woods, 2004, de Graaf et al., 2004).

Other anorexigenic hormones are Glucagon-Peptide 1 (GLP-1), Peptide tyrosine-tyrosine (PYY) and Glucose dependent insulinotropic polypetide (GIP). GLP-1 is secreted in a biphasic pattern once the nutrients enter the ileum and colon (de Graaf et al., 2004, Woods, 2004). The first phase starts 10-15 min after ingestion followed by subsequent sustained release around 30-60 min post-ingestion. PYY is secreted at the distal segment of small intestine and prolongs inter-meal interval (de Graaf et al., 2004). GIP is synthesized in the mucosa of duodenum and jejunum and also induces insulin secretion (Woods, 2004, de Graaf et al., 2004).

2.2 Biomarker of satiation

2.2.1 Visual Analog Scale for subjective hunger/satiation sensations

Appetite in humans could be assessed by two common methods, namely subjective ratings and measurement of actual food intake (Horner et al., 2014, de Graaf et al., 2004, Blundell et al., 1996). As satiation and satiety involves different functions of the brain and

different hormones release, a combination of both methods could provide improved sensitivity and specificity to assess satiation (de Graaf et al., 2004).

Visual analogue scale (VAS) of hunger and satiety is a validated measure to assess subjective states of motivation to eat before and in response to food in normal and obese subjects (Horner et al., 2014, Stubbs et al., 2000, de Graaf, 1993). The VAS measurement is a 100 mm in length, anchored at the most negative rating at one end and the most positive at the other end. It consists of 4 different questions which were designed to record subjective sensations of hunger, satiety, fullness, prospective food consumption and desire to eat." Hunger" is a proxy marker for appetite for a meal, while "Fullness" refers to fullness sensation in the stomach. "Hunger" could be considered as the opposite of "Fullness", while "Prospective food to be consumed" could refer to preoccupied thought of food and "desire to eat" may refer to a feeling for eating (Blundell et al., 1996).

VAS can also be used to track evolution of these sensations over a certain period of time or to assess pre- and post-effects of certain meal intervention (Flint et al., 2000, Gregersen et al., 2008). Reproducibility of VAS to measure hunger/satiety sensations has been shown with sample sizes of 17 subjects, provided there was standardization of diet. Such samples were capable of detecting a difference of 500 kJ in energy intake (Gregersen et al., 2008). Horner et al in 2014 also showed reproducibility of VAS among obese and overweight subjects with similar sample size to measure appetite sensations (Horner et al., 2014).

2.2.2 Food intake

2.2.2.1 Dietary methods for food intake assessment

There are several validated methods to assess food intake depending on the objectives of the study (Block, 1982). This review will discuss dietary assessment method used in the study. Food Weighing is one of the most accurate techniques which considered as "golden standards" in assessing food intake which involved weighing food before and after the study

intervention (Lassale et al., 2009). This validated approach will allow actual measurement of food intake at one time point by subtracting the left-over food with the food prepared before consumption. The advantage of food weighing is it ensures the amount of food consumed by the study participants in a controlled environment, is accurately measured without the influence of memory bias and possibility of omission for certain foods (Lassale et al., 2009, Murakami et al., 2005). However, some of the disadvantages are food weighing requires extensive work for both the researchers and study participants and thus is not a preferred method to asses dietary intake in a large study population (Illner et al., 2010). Food weighing still poses possibility for under- and over-reporting despite ample time it required to weigh the food (Myers et al., 1988).

Prospective dietary food record is another technique to assess nutrient intake in which the study participants record all the food and its portions during a certain period of time (Biro et al., 2002). This technique involve adequate training prior to the dietary recording on how to fill in the food record in each meal time and to record the portion size using household items i.e plates, spoon or food models (Biro et al., 2002). The advantages of this method are minimal memory bias and a higher compliance rate by the study participants provided if they don't have to record their food intake over a long duration (Kelemen, 2007). The disadvantage of food record is a potential of under- and over-reporting of nutrient intake due to incorrect estimation of the portion size, its inability to capture the food pattern over longer period of time and the compliance of study participants to record every meals consumed (Knudsen et al., 2011). In order to improve its accuracy in assessing total nutrient intake, food record usually involves 3 days of reporting which should include both weekdays and weekend to cover seasonal and daily variation as most people had a quite different food preference/intake during the weekend as compared to weekdays (Cheng et al., 2013, Kobayashi et al., 2011, Biro et al., 2002, Gersovitz et al., 1978).

2.2.2.2 The socio-culture influence in measuring actual food intake

Measuring the relationship between appetite and actual food intake should take into account several internal and external factors as food is usually being consumed within certain context (de Graaf et al., 2004). The external factors which influence food intake could vary from socio-cultural to age, gender, food availability and hedonic properties of food (de Graaf et al., 2004).

Studies among young children showed that family culture determines food choices and feeding style (de Wit et al., 2014, Santiago-Torres et al., 2014). There was an inverse relationship between hunger and fullness present among young adults as compared to those present among elderly (Wurtman, 1988, Rolls, 1994, Clarkston et al., 1997). Rolls et al in 1991 showed that men consumed more calories than women as the latter experienced more inhibitions for food intake such as body dissatisfaction and other socio-cultural pressure (Rolls et al., 1991a). In addition, there was a different environmental influence between men and women. Changes in income and food price influenced the food intake of men but not women (Buttet and Dolar, 2014).

Cognitive function is one of the key internal drivers which influence food intake (de Graaf et al., 2004, Batterham et al., 2007). Individuals who consciously restrained her or himself towards certain foods would have different food habit which would influence the total consumption of nutrients (Stunkard and Wadden, 1990). Stunkard and Messick et al in 1985 has developed the Three Factors Eating Questionnaire to measure the total elements of restraint eating and its components, namely Cognitive, Uncontrolled and Emotional eating (Stunkard and Messick, 1985). This questionnaire has been validated across different ethnic populations and for normo-weight and overweight/obese populations (Karlsson et al., 2000). Restraint eaters were closely related with having weight fluctuation/cycling which poses a higher risk to cardio-vascular diseases, T2DM and psychological disturbances (Brownell and Rodin, 1994, Montani et al., 2015).

2.3 Ethnicity

Relevant English publication from 2005-2015 using PubMed, Scopus and Google scholar data base were retrieved. For the role of ethnicity in influencing body composition, food intake and appetite regulation and metabolic health, following key words were included in the search: "ethnicity" AND "body composition". Manual exclusion was conducted using following criteria: puberty, exercise, supplementation, disease, diagnosis, treatment, child*, hospital*, disorder*. Around 2333 publication are retrieved and 241 publications were reviewed.

2.3.1 Influence of ethnicity on body composition and metabolic health

Several studies showed ethnicity plays a role in determining the relationship between central obesity and insulin resistance (Kruger et al., 2015, Marinou et al., 2014, Ntuk et al., 2014, Nazare et al., 2012, Ross et al., 2002). Asians are known to have higher body fat mass as compared to their Caucasian counterparts with similar Body Mass Index (BMI) (Jowitt et al., 2014, Stults-Kolehmainen et al., 2013, Tan et al., 2004, Flowers et al., 2013). In addition, ethnicity also plays an important role in influencing the fat deposition in the body. Chinese, Puerto-Rican and Mexican men had higher abdominal fat as compared to the Non-Hispanic White men (Stults-Kolehmainen et al., 2013). Chinese and Hispanic descent had higher percentage of android fat than gynoid fat as compared to those of African American and Non-Hispanic men (Stults-Kolehmainen et al., 2013).

Total abdominal fat and total visceral fat among Indian men were progressively increased from those with normal glucose tolerance (NGT), impaired glucose tolerance (IGT) and type 2 diabetes mellitus (T2DM), although there was no correlation found between subcutaneous fat and glucose tolerance (Indulekha et al., 2011). Visceral fat has been closely related with production of different adipokines which causes an increased risk on metabolic health risk among different ethnic groups in Asia (Indulekha et al., 2011, Yuan et al., 2010, Wasim et al., 2006). Increased abdominal adipose tissue as indicated by larger

waist circumference had more metabolic consequence among Asians, as compared to total fat mass (Shah et al., 2012, Shen et al., 2006). World Health Organization (WHO) recommends lower cut-off of waist circumference to determine risk of metabolic disease among Asians as compared to Caucasians (Health, 2008). Asian men have a higher risk of metabolic disease with a waist circumference of more than 90 cm, while for Caucasian men it is more than 102 cm. Asian women had a higher risk of this disease with waist circumference of more than 80 cm, while Caucasian women with more than 88 cm.

Although BMI is not a sensitive measure of body composition, it is still a good indicator to measure fat mass and risk of metabolic diseases in the general population. Studies in adults and children have consistently reported that at similar BMI, Asians had more body fat mass as compared to Caucasians (Thomas et al., 2012, Lakshmi et al., 2012, Wulan et al., 2010). Among Asians, South Asians (Indians) had the highest risk, followed Southeast (Malays) and East (Chinese) Asians (Wulan et al., 2010, Stults-Kolehmainen et al., 2013). Data from 27 cohort studies in Asia, Australia and New Zealand which contributed around 1,244,793 person-years of follow-up showed that each reduction of 2 kg/m² among Asians contributed to 37% lower risk of developing T2DM, while the same amount of reduction among Australians only contributed to 25% lower risk (Ni Mhurchu et al., 2006).

There was a different association between BMI and insulin resistance among African, Asian, Hispanic American and Non-Hispanic white, which could be due to different levels of adiponectin and leptin (Rasmussen-Torvik et al., 2012).

2.3.2 Influence of ethnicity on food intake and appetite regulation

Ethnicity influenced insulin and GLP-1 release after an OGTT as shown by studies among healthy, young South Asian men who had a higher insulin response, higher Matsuda Index and higher AUC for GLP-1 after 6-h OGTT as compared to their Caucasian counterparts (Sleddering et al., 2013) and a study among a group of lean, non-diabetic young

adults of Indian and Chinese origins (Dickinson et al., 2002). Those of Indian descent had 100% and those of Chinese descent in this study had 50% higher iAUC for capillary glucose following carbohydrate load as compared to their Caucasian counterparts (Dickinson et al., 2002). Asians also had higher post-prandial glycemic response as compared to Caucasians when challenged with food of similar amount of carbohydrate (Venn et al., 2010, Henry et al., 2008).

2.3.3 The influence of ethnicity and migration on BMI in Australia

Australia is a melting pot of people from different ethnicities from all over the world, being a culturally diverse country with around 27% of the population born overseas (ABS, 2013). Several studies conducted in Europe and USA showed influence of assimilation to the culture of the new country on BMI (Hauck et al., 2011, Lindstrom and Sundquist, 2005, Sundquist and Winkleby, 2000). Astell-Burt et al in 2013 showed in a cross sectional in New South Wales that overseas-born groups had lower BMI especially the Chinese born in China as compared to native Australian, and so does English and Scottish. However, this was not the case for Italian and Greeks (Astell-Burt et al., 2013b). Further study from Victoria Health Survey from 2003-2005 involving around 15,000 by Hauck et al conducted in 2011 showed that the BMI between 1st and 2nd generation immigrants of Asian and those of native Australian was different. The 1st generation migrants had lower BMI as compared to native Australians, however the 2nd generation migrants had a comparable BMI compared to the latter. There was less impact of assimilation on the BMI of East, North-West Europe migrants. This study concluded that the changes were more related to the change of environment rather than the intrinsic factors of genetics or other ethnic-related factors among Asians. As for those of Caucasians descent, the less impact of assimilation on BMI could be due to similar heritage, culture and genetic traits between Australians and Europeans as the earlier generation of native Australians emigrated from Europe (Hauck et al., 2011). Study from the US showed that duration of stay was significantly correlated with increased BMI

(Wang et al., 2006). These study results clearly showed the interplay between genetic variation and environmental determinants in influencing adipose tissue and muscularity deposition even in similar ethnic groups.

2.4 Metabolic and food preference disruption in overweight and obese subjects

2.4.1 Metabolic disruption in overweight and obese subjects

Adipose tissue is an active endocrine organ which is closely related to risk for metabolic diseases (Kershaw and Flier, 2004). In early 1980, Krotkiewski et al elucidated that overweight men were more vulnerable to metabolic disarrangement than women due to different fat deposition (Krotkiewski et al., 1983). Men and women with men-type abdominal adiposity in this study were more susceptible to the effect of excess body fat on lipid and carbohydrate metabolism. In parallel with increasing body fat, there was an increased level of triglycerides, fasting insulin and glucose in both men and women (Krotkiewski et al., 1983).

A study among 1000 healthy White and African American men and women showed that waist circumference had the strongest correlation to metabolic syndrome, followed by BMI, as compared to the percentage of body fat (Shen et al., 2006). Women had more peripheral fat as compared to visceral fat, thus waist circumference became a more sensitive marker among women as compared to men (Shen et al., 2006). Obese women with different regional body fat had different plasma glucose at baseline and throughout the different time line after the OGTT. Those with high percentage of deep abdominal fat have consistently higher plasma glucose as compared to those with lower percentage (Sparrow et al., 1986, Ross et al., 1996). Despite abundance of data showing increased abdominal or visceral fat related with insulin resistance, the pathophysiology of this regional fat in influencing insulin resistant is still inconclusive. It was postulated that greater lipolytic activity of centrally located adipocytes contributed significantly to milieu-favouring fatty acid substrate

competition. The elevation of fatty acids in portal venous decreased hepatic insulin extraction and promoted production of Apo lipoprotein B lipoproteins (Conus et al., 2007).

Waist-hip ratio (WHR) has been established as a crude indicator of upper-body fat mass and also correlated with impaired glucose tolerance (Sekikawa et al., 1999, Unwin et al., 1997, Ostlund et al., 1990), which could be commonly used using Oral Glucose Tolerance Test (OGTT) (ADA, 2010). People having an elevated blood glucose but still below the given threshold for Type 2 Diabetes Mellitus (T2DM) in accordance to American Diabetes Association (ADA) classification could be categorized as either having Impaired Fasting Glucose (IFG) and or Impaired Glucose Tolerance (IGT) (ADA, 2010).

Impaired glucose tolerance based on OGTT could also be a surrogate index for insulin sensitivity (George et al., 2011). Other surrogate indexes includes HOMA-IR (Matthews et al., 1985), Matsuda-de Fronzo Insulin Sensitivity Index Composite (Matsuda and DeFronzo, 1999), Strumvoll Index; OGIS Index (Oral Glucose Insulin Sensivity) (Mari et al., 2001). Another method to measure insulin resistance is through euglycemic insulin clamp. As it requires glucose utilization by the muscle the insulin clamp is a good parameter for skeletal muscle insulin sensitivity (Pratt-Phillips et al., 2015).

The data from 725 Japanese adult further showed that elevated 2-h postprandial glucose post 75 gram glucose load was related to metabolic syndrome regardless of the normal fasting glucose value (Kanauchi et al., 2006). Henrikssen et al in 1994 showed that insulin resistant was also found in normo-glycemic borderline overweight subjects with first degree relatives of Type 2 Diabetes Mellitus (T2DM) as measured by Insulin Sensitivity Index (Henriksen et al., 1994). In addition, not every overweight and obese subject was insulin resistant. (Straznicky et al., 2009).

It was estimated that the worldwide prevalence of impaired glucose response was around 8% in 2007, (Gerstein et al., 2007a, Jagannathan et al., 2014). People with impaired glucose

response were predicted to have an average of 5-10 times higher risk in developing Type 2 Diabetes Mellitus (T2DM) as compared to those with normo-glycemia. In contrast, they had 30 lower relative risks to develop normoglycemia within one year (Gerstein et al., 2007b).

Insulin resistance influences the GIP hormones in which T2DM and obese subjects were not responsive to and had lower levels of GIP secretion after meals as compared to healthy subjects (Skrha et al., 2010, Verdich et al., 2001). This irresponsiveness and inadequate GIP secretion could lead to overeating in overweight and obese person.

2.5.1 Appetite and food intake disruption among overweight and obese subjects

A meta-analysis by Flint et al in 2000 showed that an increase in post-prandial insulin, and not glucose, was associated with short-term appetite regulation in normo-weight adults (Flint et al., 2000). However, this mechanism maybe disrupted in obese and overweight subjects due to several mechanisms including a decrease in central and peripheral insulin sensitivity and in satiety-related hormone release (Fernandez-Garcia et al., 2013, Viardot et al., 2008).

Adipose tissues secrete Leptin which has been known to influence the neuroendocrine regulation of obesity and is closely related to glucose metabolism. In an animal model, injection of leptin has been shown to reduce food intake and promote weight loss (Seeley et al., 1996, Schwartz et al., 1996). Schwartz et al in 2000 argued there were several mechanisms of leptin in influencing food intake. The mechanisms involved :1) leptin deficiency which led to hyperphagia that persisted even with high insulin level (Schwartz et al., 2000). A high level of leptin occurred only in obese and not normo-weight subjects (Considine et al., 1996, Abdullah et al., 2009) Leptin release is in parallel with the amount of total fat mass and superficial subcutaneous fat (Bougoulia et al., 1999). Leptin is a hunger suppressant mediator and although insulin does not influence the secretion of leptin (Dagogo-Jack et al., 1996), studies showed that insulin-leptin response may be disrupted in

overweight and obese subjects (Bougoulia et al., 1999, Considine et al., 1996, Dagogo-Jack et al., 1996) . 2) secretion of α - melanocyte-stimulating hormones (α -MSH) which leads to excessive food intake such as (Schwartz, 2006) and several dipocytokines—such as adiponectin and visfatin which are related to low-grade inflammation, insulin sensitivity, glucose metabolism (Cancello and Clement, 2006).

Insulin had a negative correlation with subsequent food intake in lean men but not in obese men (Speechly and Buffenstein, 2000, Verdich et al., 2001). Basal concentration of insulin and leptin were significantly higher in obese subjects as compared to their normoweight counterparts. There was no correlation between insulin and leptin at basal concentration in obese subjects, in contrary to a strong correlation in normo-weight subjects. At 30- and 60- minutes after OGTT, there was an increase of not only insulin, but also leptin concentration among obese subjects. However, the leptin level in normo-weight subjects was constant over the same time duration (Bougoulia et al., 1999, Cakir et al., 2005). These results were in contrast to previous studies which showed that there was no increment of leptin concentration in both normal and obese subjects after different meal regimens of standardized breakfast and lunch (Considine et al., 1996) or standardized lunch only (Dagogo-Jack et al., 1996). The absence of this acute surge of leptin in normo-weight subjects was then postulated to be related to leptin resistance/ insensitivity which also has been associated with insulin resistance in obese individuals (Cakir et al., 2005).

Disruption of appetite regulation has been shown among overweight and obese individuals through provision of pre-load meals (Speakman et al., 2002, Brennan et al., 2012, Chapman et al., 1998). Unfortunately, regardless of the calories given in the different pre-load meals, these subjects were not able to detect these differences. Overweight and obese subjects who received high calorie pre-load meals, still consumed more calories in the subsequent meal, thus the pre-load did not adequately compensate. After having high –fat

preload meals overweight and obse men consumed ~ 56% more energy than their lean counterparts in the subsequent meals (Speechly and Buffenstein, 2000).

Overweight and obesity are also related to incentive reward systems which involved dopaminergic rewards system in the brain. Individuals with high food—cue responsiveness tended to be vulnerable to over-eating thus more prone to obesity (Nijs et al., 2010). Among food-deprived obese adults, it is shown that they gave longer attention to food cues as compared to the normal-weight counterparts (Castellanos et al., 2009).

Nijs et al in 2010 also attempted to show the association of VAS score and external food cues, bogus task and amount of food consumed during this task. In the study involving 40 normo-weight and 26 overweight obese female participants, the study found that hungry overweight/obese subjects were more prone to food-related stimuli as compared to normal weight counterparts. During the experiment these hungry individuals were also more prone to select more snacks (Nijs et al., 2010). This study also showed that although normo-weight subjects had higher hunger scores as compared to obese subjects when they had better control of their appetite. The findings seems in line with earlier findings by Stunkard et al in 1990 which showed that physiological signals of hunger corresponded with subjective hunger in normo-weight subject but not in obese who were less sensitive to recognize this internal hunger and satiety cues (Stunkard and Wadden, 1990).

2.5.2 Food preference among overweight and obese subject

An earlier study by Strain GW et al in 1992 showed that overweight and obese subjects had total calorie intake in proportion with their body weight. Obese subjects maintained their excessive calorie intake by consuming food with high-energy density in combination with binge eating.

This finding is in accordance to several other studies comparing obese and normo-weight women showed that obese women preferred high-fat food (Strain et al., 1992) and they were

not able to compensate the energy intake in the subsequent meal which may be caused by the fact that obese subjects were relatively insensitive to the satiety effect of fat (Rolls et al., 1991b, Rolls et al., 1992, Rolls et al., 1994). High-fat foods were believed to cause highest satiety effect, however, it has been shown that fat increased hedonic preference and thus increased total food intake rather than directly causing satiety (Blundell et al., 1996).

Taking into account the above-mentioned understanding, assessment on the influence of ethnicity and weight fluctuation on appetite sensations and food intake were further explored with a study methodology as described in Chapter 3.

CHAPTER THREE

METHODOLOGY

3.1. Study Protocol

This study is a cross-sectional study involving overweight and obese Asian and Caucasians-Australian participants of who fulfil the following inclusion criteria: Age older than 35 years of age, BMI \geq 25 kg/m², non-smokers, consumption of less than 2 standard alcoholic drinks per day, no GI tract problems, not on medication affecting metabolic rate, body composition or taste/ appetite sensations (i.e. anti-depressant) and non-shift workers. The cut-off age of 35 years is determined from an earlier study by Dunstan et al in 2002 which showed that the incidence of T2DM among Australians started to increase at 35 years old and above (Dunstan et al., 2002). The inclusion criteria in this study only allow minimum intake of alcohol to less than 2 standards drinks per day, thus excluded excessive alcohol drinkers because of a dose-dependent relationship between alcohol intake and appetite (Caton et al., 2004) and potential alcohol in influencing appetite and food intake through an opioid, serotonergic, and GABAergic pathways (Schrieks et al., 2015).

Personal history of chronic disease, weight stability/fluctuation for the last 6 months, blood pressure measurement and medication use was obtained. Study participants were recruited by advertisement in the local media or by personal approach until November 2014.

One day prior to the study day, all study participants received ready-to-eat meals for dinner and were requested to arrive at the School of Public Health, Curtin University after a minimum of 10-12 hours of overnight fasting and an 8- hour sleep, with minimum physical active. They were then accommodated in a dedicated room where they could relax and conduct activities with minimal physical effort (reading, knitting, solving cross words, listening to music) for the subsequent 2 hours between OGTT and buffet lunch. Participants

were requested not to read any materials related to food or cooking i.e food books and cooking magazines.

All study participants underwent an OGTT with 75 g glucose in 300 ml (GLUCAID TM). OGTT has commonly being used as a screening tool for impaired glucose response/insulin resistance(ADA, 2010). : Glucose via insulin may directly affect hunger/ satiety in short duration. Having single nutrients creates less issue with gastric emptying and palatability than mixed breakfast test meals. In effect, the study will have more output using the OGTT as test meal. OGTT has also commonly being used as a screening tool for impaired glucose response/insulin resistance (ADA, 2010).

Capillary blood glucose samples were taken four times during the study at the following: baseline, 30-,60-, 90-and 120-minutes after OGTT. Questionnaires on habitual food habit, habitual physical activities and restrained eating behaviours were administered during the study to further understand the characteristics of the study participants. They were also allowed small sips of water during the study time until lunch time.

3.2 Sample Size

The main objective is to detect an ethnic difference in appetite sensations among overweight and obese subjects. A total sample size of 52 completed subjects (n=26 subjects per group) was required to detect a moderate effect of 0.40 according to Cohen's effect conventions for the main effect of ethnic group and weight stability and possible interaction between the ethnicity and weight stability, with a power of 80% at 5% significance level.

At the time of the study design, there was no publication analysing the difference between subjective sensations of hunger and satiety in response to glucose. Therefore a moderate effect size of 0.40 was selected. Gregersen et al in 2008 showed that a sample size of 25 is sufficient to detect a difference of 100 mm in VAS of subjective hunger sensations with a difference in energy intake of 500 kJ (Gregersen et al., 2008).

3.3 Study Instruments

Ratings of subjective feelings of hunger, appetite and fullness

The validated visual analogue scale (VAS) was used as it is a reliable method of measuring subjective states of motivation to eat before and in response to meals (Flint et al., 2000, Gregersen et al., 2008, Stubbs et al., 2000).

Drapeau et al in 2005 further showed that VAS on appetite sensations administered before standardized test meal, immediately after and every 10 minute after the meal was a marker of overall Energy Intake (EI) (Drapeau et al., 2005), while Doucet et al in 2003 showed AUC fullness using VAS was correlated with post-prandial energy intake among overweight and obese subjects participating in long-term weight loss program (Doucet et al., 2003). In addition, there were no earlier publications which assessed subjective hunger/satiety sensations using VAS between two different ethnic groups which is the novelty of this study. The VAS used in this study was shown in Appendix 5.

The study participants were informed on how to use VAS on four different subjective parameters at baseline, 30-, 60-, 90- and 120-min after OGTT. The VAS were filled before finger pricked for blood glucose was taken. The VAS questionnaires consist of following questionnaires: "How hungry do you feel?" (Not hungry at all = 0 mm to extremely hungry = 100 mm, "How full do you feel?" (Not full at all = 0 mm to extremely full = 100 mm), "How strong is your desire to eat?" (None = 0 mm to very strong = 100 mm) and "How much food do you think you can eat" (nothing at all = 0 mm to an extremely large quantity = 100 mm). They were also requested to place an "I" at any point along the scale, and scores were then converted to continous variables from 0 to 100 mm (Stubbs et al., 2000) VAS score was reported up to 1 decimal in mm from the left to the place where an "I" was located. The higher the VAS score in hunger /desire to meat/amount of food to be consumed means that the participants had more intense sensation of hunger / desire to eat/amount of

food to be consumed. The higher the VAS score for satiety means that the participants had more intense sensation of fullness which could translate in less food intake.

The palatability of the test meal was also measured with a VAS using 100-millimetre line scales with following questions "How does the meal taste?" (not pleasant = 0 mm to very pleasant = 100 mm) as shown in Appendix 6. The questionnaire was administered right after the participants consumed the glucose solution as a breakfast meal and after the subsequent lunch. Palatability score was reported up to 1 decimal in mm from the left to the place where an "I"was located.

Food Restrained Questionnaire

The validated shortened version of Three-Factor Eating Questionnaire (TFEQ) was administered (Karlsson et al., 2000). The original TFEQ was developed by Stunkard and Messick in 1985 which consists of 51 questionnaires (Stunkard and Messick, 1985). These questionnaires was categorized into three scales namely the Cognitive restraint (21 items), Disinhibition (16 items) and Emotional eating (14 items). In the shortened version, the questionnaire consists of 18 questions which were then categorized into cognitive restraint (6 items), uncontrolled eating (9 items) and emotional eating (3 items) as shown in Appendix 10. There were no clear cut-off points for the score of each category. Studies showed that those with higher score of cognitive restraint had the lower energy intake, in contrast with those with higher score of emotional eating (de Lauzon et al., 2004, Karlsson et al., 2000).

Physical Activity Diary

Physical activity questionnaires developed and validated by Baecke et al in 1982 was administered as it had been validated among an adult population and was able to provide information of physical during different time of the day as shown in Appendix 9 (Baecke et al., 1982). The questionnaire was filled in between the administration of VAS and finger blood pricks.

Lunch meal

A simple lunch was served approximately 2.5 hours after OGTT. The buffet lunch consisted of a pre-selected hot meal with following choices ready-to-eat meals from Coles supermarket: Indian butter chicken & rice, Honey beef, Beef stroganoff, Rice and black pepper beef and two vegetarian options which were Tuscan Tomato Fusili and Neopolitan Penne. The nutrition information of these meals is described in Table 1 below. In addition a variety of other food such as slices of bread, vegetables (tomatoes or cucumber), apples, oranges, potato chips, fruit juice and chocolate bars were offered for consumption ad libitum. These foods were selected based on high acceptance from a previous study at the same centre (Ping-Delfos and Soares, 2011).

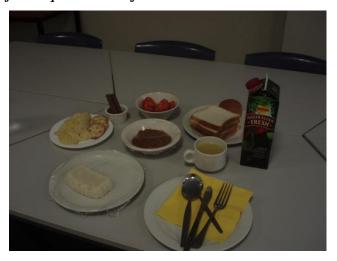
Every food item was weighed using food weighing scales up to 0.1 gram prior to being served. The difference between the weighed and left-over food was calculated as actual food intake for lunch which was then recorded for the study (Lassale et al., 2009) as shown in Appendix 7- Food intake form during lunch. The nutrients intake was calculated from the manufacturers' product information and the Food Works ^R nutrient analysis .version 7 (Xyris Software, Brisbane, Queensland, Australia).

Table 1. Nutrition composition of ready-to -eat meals

	Indian	Beef	Honey	Beef	Tuscan	Neopolitan
	Butter	stroganoff	beef	black	Tomato	Pasta
	Chicken		rice	pepper	Fussili	(vegetarian)
				and	(vegetarian)	
				rice		
Serving size (g)	300	300	320	320		
Energy (kJ)	1370	1490	1977	1817	1630	1500
Protein (g)	18.9	20	17.5	20.7	12.2	14.4
Total fat (g)	3.6	4.2	8	5.2	15.2	6
Total saturated	3	3	2.8	2.4	2.6	1.2
fat (g)	3	3	2.0	2.4	2.0	1.2
Carbohydrate g	53.7	58.8	75.8	69.1	48	59
Sugar (g)	4.8	4.5	11.8	3.6	8	3.6
Sodium (mg)	639	618	639	784	1020	980

An example of a meal was shown in Figure 1.

Figure 1. Example of subsequent lunch after OGTT



Food record

Study participants were requested to record all the foods that they consumed after leaving the study site for 24 hour after the buffet lunch. The food record was derived from the food record booklet developed by the Fred Hutchinson National Cancer Research and contained information on portion sizes . Study participants were given thorough information on how to record their food intake including portion sizes via face-to-face discussion. No scales were given to the study participants as the information printed in the 24-h food record booklet had adequately included the portion sizes

This 24-h food recorded will be able to provide participants' acute food intake (Block et al., 1990). The Food Works nutrient analysis version 7 (Xyris Software, Brisbane, Queensland, Australia). and the manufacturers' product information were used to compute the nutrients intake from the food record as shown in Appendix 8.

Anthropometrics assessment

Height was measured barefoot by a portable stadiometer (Holtain, Crymych, United Kingdom) to within 0.5 cm (Ogle et al., 1995, Dobbelsteyn et al., 2001). Weight was measured by digital scale to within 100 g, without heavy clothing (Ogle et al., 1995, Dobbelsteyn et al., 2001). Mid-Upper-Arm –Circumference (MUAC) was measured on the right arm at the midpoint between the tip of the acromion and the tip of olecranon, elbow bent at 90 using flexible tape (Lean et al., 1996).

Waist circumference (WC) was measured mid-way between the lowest rib and the iliac crest with the subject standing at the end of gentle expiration (Dobbelsteyn et al., 2001). Hip circumference was measured at the greater trochanters (Dobbelsteyn et al., 2001). All circumferences were measured to within 1 mm with plastic tapes. BMI was calculated as the ratio of weight to height squared (kg/m²). WHR was calculated as WC (cm) divided by hip circumference (cm) (Dobbelsteyn et al., 2001).

DEXA measurement

Fat mass, fat-free mass and bone mineral content was determined using the Lunar Prodigy DEXA instrument (GE Medical System, Madison, WI, USA) together with Encore software. All scans were performed when subjects were wearing a light gown and had removed all metal objects. The scans were performed by one trained operator. Scan time period was around 5 minutes depending on the body size of the study participants. Fat mass, fat-free mass and bone mineral content were reported in term of percentage to body weight (Ogle et al., 1995, Van Der Ploeg et al., 2003).

Blood pressure measurement

Upon arrival at the School of Public Health, Curtin University, study participants were requested to lie down on a bed for 10 minutes. Blood pressure was measured by auscultation of brachial artery using digital sphygmomanometer (Omron ^R) (Tolonen et al., 2015).

Readings of systolic and diastolic blood pressure were taken twice. An average value was recorded in the data.

3.4 Ethics Approval

The study received Ethics Approval from the School of Public Health, Curtin University, Perth, Western Australia number HR 109/2010 under a study title "Endothelial Function and Food Intake (EFFI). As the focus of the study is addressing the food intake of EFFI, both studies had similar selection criteria.

All subjects were given a thorough explanation in the Patient Information Sheet on the course of the study and signed informed consent prior to study enrolment. They had been informed that they had the right to withdraw from the study at any time. Each subject was assigned to an identification code which was used for all data collection thus de-identifying all the subject-specific information. Subjects' names and ID codes were stored separately. Information regarding the study participants was kept confidential. All data are stored in the locked cabinet with the supervisor for 15 years. All e-data were accessible only by the researcher and supervisors. All data had been aggregated and no subject was identifiable in any publication.

3.5 Data Analysis

Dependent variables for this study are the four subjective sensations, namely hunger, fullness, desire to eat and amount of food able to consume and food intake. These main research outcome variables were further presented as the total area under the curve (tAUC) calculated using a trapezoidal rule based on the five repeated measurements collected at baseline, 30, 60, 90 and 120 minutes following the glucose load. The analysis using tAUC, is preferred over a conventional mixed design repeated measures ANOVA due to tAUC ability detect total/ cumulative changes/response towards an intervention as compared to differences at individual time point (Jacobsen et al., 2005, Sibbald et al., 2008, Pruessner et

al., 2003, Wolever, 2004). The Independent variables for this study are ethnicity, age, glucose response, fat mass and fat-free mass.

As weight fluctuation affects glucose homeostasis (Kajioka et al., 2002), we hypothesis it could also affect the hunger/satiety sensations and food intake in this study. Analyses using two-way ANOVA were further conducted with a focus on the interactive effect of weight stability status and ethnicity. An independent two-samples T-test was conducted to further compare the difference between Asians and Caucasians within both weight stable and non-weight stable groups.

Descriptive statistics were obtained for variables of interest. Continuous variables were presented as mean ± standard error. Categorical variables were presented as frequency (%) for defined categories. Chi-square and Pearson's correlation coefficient were used to assess the association between variables of interest. An independent samples t-test was conducted to assess possible difference in the tAUC of subjective hunger/satiety between the two different ethnic groups. Furthermore different analyses of covariates (ANCOVA) models, implemented with a general linear model, were developed to explore the effect of ethnicity and weight fluctuation on the main research outcomes (the four subjective sensations, namely hunger, fullness, desire to eat and amount of prospective food to be consumed) after controlling for fasting (baseline) values or other confounders and independent variables. If the weight fluctuation was found to be a significant effect modifier, the difference in the mean AUC between the two ethnic groups were further compared within each level of the weight fluctuation status, namely weight stable group and weight unstable group. All data analyses were carried out using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp. Released 2010 Armonk, NY). All tests were two-sided and a p value of less than 0.05 was considered as statistically significant.

3.6 Summary

This chapter provides an overview of the methodology used in the study. A validated visual analogue scale was administered to assess subjective hunger, fullness, desire to eat and amount of prospective food. Standard statistical tests were conducted to examine the potential difference between the two ethnic groups in each variable of interest and to further explore the interplay between ethnicity and weight fluctuation in influencing these subjective sensations and subsequent food intake. These study results are discussed in Chapter 4.

CHAPTER FOUR

RESULTS

4.1 Demographics and metabolic health characteristics

This cross-sectional study involved 44 participants. About 40% (n=18) were Asians comprised of South-east Asians (Malaysia and Singapore) and South Asians (Bangladesh, Sri Lanka and India). The Caucasians were of European descent. Sixty four percent (n=28) of the study participants were women and 81% (n=22) of them were post-menopausal. Further demographic characteristics of the study participants are described in Table 2.

Weight stability was defined as not having weight fluctuation more than 2 kg in the last 6 months in this study (Osborn et al., 2011, Venditti et al., 1996). There was no statistical difference on number of participants who reported to be weight stable weight and those who reported to be non-weight stable in each ethnic group (χ2 test,p=0.802). As weight instability could affect the glucose homeostasis (Kajioka et al., 2002), and we hypothesize that it could also affect the hunger/satiety sensations and food intake. Analyses using two-way ANOVA were further conducted with a focus on the interactive effect of weight stability status and ethnicity. An independent two-sample T-test was further used to compare the difference between Asians and Caucasians within both weight stable and non-weight stable groups. Due to some missing data, the number of participants varied for each analysis.

There was a significant correlation between ethnicity and age, but no interaction on main effect of weight stability or on interaction between ethnicity and weight stability as shown in Table 2. Asians on average were statistically younger than the Caucasians (mean (SEM)= 43)

(2.4) vs 54 (11.9) years, p=0.002) regardless of the weight stability status. The Independent two-way samples T-Test found that non-weight stable Asians, on average, were younger than non-weight stable Caucasians (mean (SEM) = 43 (2.0) vs 57 (3.0) years, p<0.005). However, there was no significant difference in mean age between Asians and Caucasians within the weight stable group.

Table 2. Demographic characteristics of the study participants

	Asians	(n= 18)		ians (n=		p values	
					Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction
	weight- stable (n=9)	non weight stable (n=9)	weight- stable (n=12)	non weight stable (n=14)			
Age (y)	43 (4.6)	43(2.0)	51 (3.6)	57 (3.0)	0.002*	NS	NS
Duration living in Australia (y) #	17 (5.8)	14 (4.0)	49 (3.9)	46 (3.8)	<0.001*	NS	NS
Living in Australia for more than 10 years (%)	4 (44)	4 (44)	12 (100)	11 (79)	NS	NS	NS
Males (%)	2 (22)	6 (66)	4 (33)	4 (28)	NS	NS	NS

Data are mean (SEM), # Two-way ANOVA, ap< 0.05 Independent two samples T-Test: significant different from Caucasians within weight stable group,

A significant effect of ethnicity on duration of living was found based on the two-way ANOVA, indicating Asians on average had a shorter (p<0.001) residence in Australia as compared to Caucasians (overall mean (SEM)= 15 (3.4) vs 47 (13.6) years, p<0.001) regardless of the weight stability status.

Overall Asians, on average, were lighter (mean (SEM) = mean (SEM) = 80 (3.4) vs 90 (17.3) kg; p =0.044) regardless of their weight stability condition. Asians who were weight stable, on average, tended to be lighter than their Caucasian counterparts (mean (SEM) = 74 (2.6) vs 89 (6.4) kg; p= 0.062), however the difference was not statistically significant. The

difference in average body weight between Asians and Caucasians within the non-weight stable group was also not statistically significant as shown in Table 3.

Asians were also significantly shorter (overall mean (SEM) = 1.6 (0.10) vs 1.7 (0.10) m; p=0.018). Weight-stable Asians (mean (SEM) = 1.6 (0.10) m) on average were found to be shorter as compared to weight-stable Caucasians (mean (SEM) = 1.7 (0.06) m). There was no significant difference in mean height between non-weight stable Asians and Caucasians.

There was no significant difference in mean hip circumference between Asians and Caucasians within the weight stable group. However, non-weight stable Asians (overall mean (SEM) = 108 (2.0) cm) on average had smaller hip circumference as compared to non-weight stable Caucasians (overall mean (SEM) = 114 (8.8) cm). Overall, Asians, on average, had a smaller hip circumference (mean (SEM) = 108 (2.0) vs 114 (8.8) cm; p=0.021).

Those who were weight stable on average had smaller waist circumference as compared to those who were non-weight stable, regardless of the ethnicity, (overall mean (SEM) = 95 (2.4) vs 104 (2.4) cm). The same phenomenon was also found for mid-upper arm circumference (MUAC). Those who were weight stable (overall mean (SEM) = 31(3.3) cm) on average had smaller MUAC (p=0.036) as compared to those who were non-weight stable (overall mean (SEM) = 34(3.9) cm).

Table 3. Metabolic health characteristics of the study participants

	Asia	ns (n= 18)	Cauc	casians (n= 26)		p values	
					Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction
	weight- stable (n=9)	non weight stable (n=9)	weight- stable (n=12)	non weight stable (n=14)			
Weight (kg)	74 (2.6) ^a	86 (5.6)	89 (6.4)	91 (3.5)	0.044	NS	NS
Height (m)	1.6 (0.10) ^a	1.7 (0.10)	1.7 (0.06)	1.7 (0.07)	0.018	NS	NS
BMI (kg/m2)	28 (0.6)	32 (1.4)	32 (2.6)	31 (1.1)	NS	NS	NS
Waist circumference(cm)	90 (2.6)	102 (3.9) ^b	98 (3.3)	15 (3.2)	0.094	0.009	NS
Hip circumference (cm)	100 (1.6) ^a	108 (2.0)	110 (5.3)	114 (8.8)	0.021	NS	NS
WHR	0.9 (0.02)	0.9 (0.03)	0.9 (0.05)	0.9 (0.0)	NS	NS	NS
Mid-upper arm circumference(cm)	31 (0.5)	35 (1.1) ^b	31 (1.4)	33 (1.1)	NS	0.036	NS
Fat Mass- DXA (kg)	27 (2.4) ^a	33 (7.2)	36 (3.5)	37 (2.1)	0.045	NS	NS
Bone Mass - DXA (kg)	2.6 (0.16)	$2.6(0.11)^{b}$	2.9 (0.11)	2.9 (0.11)	0.016	NS	NS
Lean Mass- DXA (kg)	42 (2.9)	48 (3.6)	50 (3.6)	50 (2.2)	NS	NS	NS
Fat Free Mass- DXA (kg)	45 (3.0)	51 (3.6)	53 (3.5)	53 (2.3)	NS	NS	NS
Total Body Fat %	38 (3.0)	41 (2.0)	41 (1.5)	41 (1.6)	NS	NS	NS
Systolic Blood Pressure (mmHg)	113 (5.7)	129 (4.4)	124 (7.3)	131 (4.0)	NS	0.069	NS
Diastolic Blood Pressure (mmHg)	71 (2.9)	75 (1.3) ^b	76 (3.7)	83 (2.7)	0.033	0.050	NS

Data are mean (SEM), # Two-way ANOVA, ap< 0.05 Independent two samples T-Test: significant different from Caucasians within weight stable group, NS: not significant at 5% significance level.

The main effect of ethnicity, weight stability and interaction between ethnicity and weight stability on lean mass, fat-free mass and total percentage of body fat were found insignificant as shown in Table 3 indicating Asians, on average had a comparable lean mass, fat-free mass and total percentage of body fat as Caucasians. Asians (overall mean (SEM) = 30 (1.8) kg) on average had less fat mass as compared to Caucasians (overall mean (SEM) = 36 (1.9) kg) regardless of the weight stability condition as shown in Table 3. Overall, Asians also, on average, had significantly less bone mass (overall mean (SEM) = 2.5 (0.09) vs 2.9 (0.08) kg; p=0.016) regardless of their weight stability condition.

There were no significant main effects of ethnicity, weight stability and interaction between ethnicity and weight stability on systolic blood pressure indicating Asians, on average, also had a comparable systolic blood pressure as Caucasians. A different phenomenon was found for diastolic blood pressure. Asians, on average, had significantly lower diastolic blood pressure (overall mean (SEM) = 73 (1.6) vs 80 (2.3) mmHg; p=0.033) regardless of the weight stability condition.

Asians, on average, were found to have a comparable cognitive restraint, emotional eating and disinhibiting score as Caucasians. A main effect of ethnicity, weight stability and interaction between ethnicity and weight stability on cognitive restraint score, emotional eating score and disinhibiting score were found insignificant as shown in Table 4.

Overall, those who were weight stable on average had longer duration of daily walking as compared to those who were non-weight stable (overall mean (SEM)= 53(14.2) vs 23 (3.5) minutes, p=0.041) regardless of their ethnicity.

Table 4. Physical activity and restraint eating score of the study participants

	Asians (n= 18)	Caucasia	ns (n= 26)		p values	
					Ethnic (ETH)	Weight stability(WS)	ETH x WS interacti on
	weight- stable (n=9)	non weight stable (n=9)	weight- stable (n=12)	non weight stable (n=14)			
Cognitive restraint score	12 (0.9)	13 (0.4)	13 (0.6)	13 (0.5)	NS	NS	NS
Number of participants with high cognitive restraint (%) b	6 (66.7)	9 (100)	12 (100)	13 (93)	NS ^c	NS ^c	
Disinhibition score	26 (1.7)	25 (0.9)	27 (1.2)	26 (1.1)	NS	NS	NS
Emotional eating	8 (1.2)	8 (0.7)	8 (0.9)	9 (0.7)	NS	NS	NS
Duration of daily walking (min)	44 (14.1) ^a	23 4.5)	60 (22.8)	23 (5.1)	NS	0.041	NS

Data are mean (SEM), $^{a}p<0.05$ Independent two samples T-Test: significant different from Caucasians within weight stable group, b Data are presented in n (%), $^{c}\chi^{2}$ test, NS: not significant at 5% significance level.

4.2 Correlation between glycemia/ glucose response and subjective sensations

A correlation analysis between blood glucose and subjective sensations; and between subjective sensations and food intake during lunch was conducted by calculating Pearson's correlation coefficient. Among Asians who were weight stable, there was a significant negative strong association between glucose at 2-hour post OGTT and AUC hunger, satiety, desire for food and amount of prospective food to be consumed (r= -0.869, p=0.005; r=-0.875, p=0.004; r=-0.915, p=0.001; r=-0.878, p=0.004 respectively). However, there was no significant association observed among other pairs listed in Table 5.

Table 5. Pearson's correlation coefficient between glucose response and subjective sensations

			weigh	t stable			non- weig	ht stability	
		Asian	s (n=9)	Caucasia	ans (n=12)	Asian	s (n=9)	Caucasia	ans (n=14)
		r	p value	r	p value	R	p value	r	p value
	tAUC of hunger	0.095	NS	0.033	NS	-0.560	NS	0.138	NS
	tAUC of satiety	-0.209	NS	-0.341	NS	0.282	NS	0.130	NS
Fasting Glucose	tAUC desire for food	0.008	NS	-0.174	NS	-0.370	NS	0.439	NS
	tAUC of amount of food	0.037	NS	-0.019	NS	-0.508	NS	0.229	NS
	tAUC of hunger	-0.869	0.005*	0.116	NS	-0.261	NS	-0.048	0.870
	tAUC of satiety	-0.875	0.004*	0.002	NS	0.175	NS	-0.05	0.865
Glucose at 120 min	tAUC desire for food	-0.915	0.001	0.341	NS	-0.226	NS	-0.004	0.989
	tAUC of amount of								
	food	-0.878	0.004	0.158	NS	-0.285	NS	-0.018	0.951
	tAUC of hunger	-0.559	NS	0.233	NS	-0.245	NS	-0.140	NS
	tAUC of satiety	-0.553	NS	0.350	NS	-0.114	NS	-0.136	NS
AUC glucose	tAUC desire for food	-0.458	NS	0.327	NS	-0.371	NS	-0.124	NS
	tAUC of amount of food	-0.454	NS	0.326	NS	-0.242	NS	-0.035	NS

^{*}p<0.05 Pearson's correlation coefficient, NS: not significant at 5% significance level: tAUC: total AUC.

4.3 Correlation on glycemia/glucose response and macronutrient intake during subsequent lunch.

Among Asians who were weight stable, glucose at 2-hour post OGTT was found to have significant negative association with fat intake during lunch (r= - 0.808, p=0.015) as shown in Table 6. There was no significant association observed between glucose at 2-hour post OGTT and fat intake observed among Asians who were non-weight stable. Among Caucasians who were non-weight stable, there was a significant negative moderate association between AUC glucose and protein intake(r= - 0.545, p=0.044); AUC glucose and fat intake during lunch (r=-0.652, p=0.012) as shown in Table 6. However, there was no similar correlation observed among Caucasians who were weight stable, nor among Asians.

4.4 Correlation on subjective sensations and macronutrient intake during subsequent lunch.

Among Asians who were weight stable, AUC hunger was found to be positively associated with fat intake during lunch (r= 0.832, p=0.005) as shown in Table 7. There were a significant negative strong association found between AUC satiety and calorie(r=-0.820,p=0.007), AUC satiety and carbohydrate intake (r=-0.672,p=0.047), AUC satiety and protein intake, AUC satiety and fat intake (r=-0.805,p=0.009);and AUC satiety and fiber intake during lunch (r=-0.739,p=-0.023).

Table 6. Pearson's correlation on glucose response and macronutrient intake during subsequent lunch.

			weight	stable			non- weig	ht stability	
		Asian	s (n=9)	Caucasia	ans (n=12)	Asian	s (n=9)	Caucasia	nns (n=14)
		r	p value	r	p value	r	p value	r	p value
	Energy (Kcal)	0.35	NS	0.346	NS	0.047	NS	-0.046	NS
	Carbohydrate (g)	0.379	NS	0.270	NS	0.106	NS	-0.077	NS
Fasting Glucose	Protein (g)	0.224	NS	-0.223	NS	-0.295	NS	-0.057	NS
	Fat (g)	-0.036	NS	0.342	NS	0.077	NS	0.020	NS
	Fiber (g)	0.275	NS	0.494	NS	0.007	NS	-0.427	NS
	Energy (Kcal)	-0.642	0.086	0.184	NS	-0.125	NS	-0.299	NS
	Carbohydrate (g)	-0.464	NS	0.355	NS	0.111	NS	0.104	NS
Glucose at 120 min	Protein (g)	-0.564	NS	0.437	NS	-0.343	NS	-0.257	NS
	Fat (g)	-0.808	0.015	-0.340	NS	-0.443	NS	-0.494	0.073
	Fiber (g)	-0.665	0.072	-0.091	NS	0.163	NS	-0.187	NS
	Energy (Kcal)	-0.522	NS	-0.241	NS	0.158	NS	-0.467	0.093
	Carbohydrate (g)	-0.411	NS	0.218	NS	0.277	NS	0.072	NS
AUC glucose	Protein (g)	-0.517	NS	0.229	NS	0.033	NS	-0.545	0.044
	Fat (g)	-0.502	NS	-0.588	0.096	-0.095	NS	-0.652	0.012
	Fiber (g)	-0.628	0.095	-0.368	NS	-0.061	NS	-0.250	NS

^{*}Pearson's correlation coefficient; NS: not significant at 5% significance level.

There was positive association between AUC desire to eat and fat intake (r=0.859, p=0.003) and between AUC amount of prospective food to be consumed and protein intake (r=0.259, p=0.005). There was no significant associations among Asians who were non-weight stable.

Among Caucasians, this study found that AUC satiety and carbohydrate intake during lunch was positively associated (r= 0.707, p= 0.010) in those who were weight stable and a positive moderate association was also observed between AUC prospective amount of food to be consumed and protein intake during lunch (r= 0.558, p=0.038) among those who were non-weight stable as shown in Table 7.

4.5 Oral Glucose Tolerance Test

Three statistical models (analysis of covariance (ANCOVA))were developed to compare the mean glucose response before- and after-OGTT between the two ethnic groups, with adjustment of baseline value (Model 1); of age, fat mass and far-free mass (Model 2), and of age, fat mass and far-free mass, baseline value (Model 3) as shown in Table 8. In all models, the interaction between ethnicity and weight stability status was considered. For fasting glucose level, only the unadjusted and Model 2 were further analyzed.

No significant main effect of ethnicity, weight stability and interactive effect between ethnicity and weight stability were found in fasting blood glucose, 120-min after OGTT blood glucose and AUC blood glucose even after controlling for confounders.

Table 7. Pearson's correlation between subjective sensations and macronutrient intake during subsequent lunch.

			weig	ht stable			non- we	ight stability	
		Asia	ns (n=9)	Caucas	ians (n=12)	Asia	ans (n=9)	Caucas	ians (n=14)
		r*	p value	r*	p value	R*	p value	R*	p value
	Energy (Kcal)	0.363	NS	0.215	NS	0.053	NS	0.282	NS
	Carbohydrate (g)	0.183	NS	0.103	NS	0.125	NS	0.192	NS
AUC of hunger	Protein (g)	0.345	NS	0.206	NS	0.075	NS	0.414	NS
	Fat (g)	0.832	0.005	0.214	NS	0.100	NS	0.142	NS
	Fiber (g)	0.640	0.064	0.200	NS	0.440	NS	0.290	NS
	Energy (Kcal)	-0.820	0.007	0.607	0.083	0.152	NS	0.031	NS
	Carbohydrate (g)	-0.672	0.047	0.797	0.010	0.053	NS	0.256	NS
AUC of satiety	Protein (g)	-0.805	0.009	0.605	0.084	0.176	NS	0.095	NS
	Fat (g)	-0.734	0.024	0.179	NS	0.313	NS	0.320	NS
	Fiber (g)	-0.739	0.023	0.617	0.076	0.109	NS	0.272	NS
	Energy (Kcal)	0.312	NS	0.186	NS	0.069	NS	0.334	NS
AUC of desire	Carbohydrate (g)	0.119	NS	-0.004	NS	0.112	NS	0.331	NS
to eat	Protein (g)	0.351	NS	0.053	NS	0.129	NS	0.372	NS
	Fat (g)	0.859	0.003	0.281	0.096	0.026	NS	0.089	NS

	Fiber (g)	0.431	NS	0.169	NS	-	NS	0.275	NS
						0.458			
	Energy (Kcal)	0.246	NS	0.242	NS	-	NS	0.514	0.060
						0.380			
	Carbohydrate (g)	0.059	NS	-0.064	NS	-	NS	0.435	NS
ALIC of our ount						0.350			
AUC of amount	Protein (g)	0.259	0.005	-0.012	NS	-	NS	0.558	0.038
of prospective food to be consumed	_					0.225			
to be consumed	Fat (g)	0.834	NS	0.428	NS	-	NS	0.215	0.461
						0.319			
	Fiber (g)	0.272	NS	0.255	NS	-	NS	0.252	NS
	-					0.435			

^{*}Pearson's correlation coefficient; NS: not significant at 5% significance level.

Using the American Diabetes Association 2010 criteria (ADA, 2010) , there were around 42% of the Asian participant and 53 % of Caucasian participant who had Impaired Glucose Response (either Impaired Fasting Glucose, with glucose value \geq 5.6 and Impaired Glucose Tolerance with 2-h glucose value> 7.8). However, this difference was not statistically significant (χ^2 test, p=0.111).

Table 8. Blood glucose level by ethnicity and weight stability

	Asia	ns (n=17)	Caucasians (na	=25)		p values#	
	Weight	Non-weight	Weight stable	Non-weight	Ethnic	Weight	Ethnic*
	stable (n=9)	stable (n=9)	(n=10)	stable (n=14)		stability	weight stability
Fasting Glucose							
Unadjusted [†]	5.7 (0.11)	5.2 (0.17)	5.8 (0.21)	5.8 (0.18)	0.090	NS	NS
Model 2	5.7 (0.20)	5.2 (0.16)	5.9 (0.21)	5.8 (0.18)	NS	NS	NS
120-min after OGTT							
Unadjusted†	7.1 (1.40)	7.8 (2.50)	6.5 (0.85)	7.6 (0.58)	NS	NS	NS
Model 1	7.1 (0.94)	7.8 (1.02)	6.5 (0.80)	7.6 (0.73)	NS	NS	NS
Model 2	7.2 (1.00)	8.4 (0.88)	6.2 (0.77)	7.3 (0.73)	NS	NS	NS
Model 3	7.3 (1.03)	8.3 (0.95)	6.3 (0.78)	7.3 (0.74)	NS	NS	NS
AUC Glucose							
Unadjusted†	740 (58.4)	766 (55.0)	736 (52.2)	774 (44.1)	NS	NS	NS
Model 1	740 (58.9)	740 (53.1)	753 (59.1)	779 (45.1)	NS	NS	NS
Model 2	793 (67.2)	803 (56.1)	717 (51.5)	734 (48.9)	NS	NS	NS
Model 3	806 (67.4)	780 (58.2)	724 (51.3)	736 (48.5)	NS	NS	NS

Data is mean (SEM), #ANCOVA, †Two-way ANOVA, NS: not significant at 5% significance level.

Model 1: adjusted for respective baseline value

Model 2:adjusted for age, fat mass and fat free mass

Model 3: Model 1+ Model 2.

4.6. Subjective sensations of hunger, satiety, desire for food and amount of prospective food to be consume.

A Two way ANOVA was conducted to assess the main effect of ethnicity, weight stability and interactive effect between ethnicity and weight stability on the average sensation scores of hunger, satiety, desire to eat and prospective amount of food to be consumed as shown in Table 9.

At baseline, main effect of ethnicity, weight stability and interaction between ethnicity and weight stability on average score for hunger, satiety, desire to eat and amount of food to be consumed were found insignificant. There were no significant differences found in mean values for all these four main outcomes between two ethnic groups for both weight stable and unstable groups.

For Hunger, there were also no main effects of ethnicity, weight stability and interaction effect on the average score for hunger sensation found at 30-, 90- and 120- min after OGTT, indicating that Asians and Caucasians have similar hunger status after 30-, 90- and 120- min OGTT. However at 60- min after OGTT, an significant interaction between ethnic and weight stability (p = 0.027) was found. Asians who reported a stable weight during the last 6 months had a lower hunger score than Caucasians (mean (SEM) = 27 (8.4) vs 44 (8.7) mm). In contrast, Asians who reported a unstable weight during the last 6 months had a higher hunger score than Caucasians (mean (SEM) = 56 (8.3) vs 36 (6.6) mm) on average.

There were also no main effects of ethnicity, weight stability and interaction effect on the average score for satiety sensation found at 30-, 90- and 120- min after OGTT, indicating that Asians and Caucasians have similar satiety status after 30-, 90- and 120- min OGTT regardless of the weight stability status.

Asians and Caucasians had similar desire to eat status after 30-, 60- and 120- min OGTT as there were no significant main effects of ethnicity, weight stability and interaction effect were found on the average score for desire to eat sensation at 30-, 60- and 120- min after OGTT. At 90- min after OGTT, however, a significant interaction between ethnic and weight stability (p = 0.004) was found. The two ethnic groups had almost similar desire to eat mean score (mean (SEM) = 39 (8.0) vs 40 (7.2) mm) in the weight stable group. While in the non-weight stable group, Caucasians had a lower desire to eat score than Asians (mean (SEM) = 41 (5.7) vs 70 (7.5) mm) on average.

For Amount of food to be consumed, there were no main effects of ethnicity, weight stability and interaction effect on the average score for amount of food to be consumed found at 30- and 120- min after OGTT. Asians and Caucasians have similar score for amount of food to be consumed after 30- and 120- min OGTT. However at 60- min and 90- minutes after OGTT, significant interactions between ethnic and weight stability was found (p =0.023, p=0.031 respectively), suggesting the difference in the amount of food to be consumed sensations between Asians and Caucasians for weight stable and non-weight stable groups. More specific, among those who reported weight stable, Asians had a lower amount of food to be consumed sensation at 60- minutes after OGTT) than Caucasians at 60 minutes: mean (SEM) = 31 (7.2) vs 41 (7.0) mm, at 90 minutes: 37(8.0) mm vs 39 (6.9) mm, respectively. However among those who reported non-weight stable, Caucasians had a lower average score of amount of food to be consumed than Asians(at 60-minutes: mean (SEM) = 61 (7.7) mm vs 37 (6.8) mm; at 90-minutes: mean (SEM) = 70 (8.0) mm vs 40 (5.6) mm; respectively).

Table 9. Subjective sensation score by ethnicity and weight stability

		Asians (n=17)		Caucasians	(n=25)		p values	#
		Weight stable (n=9)	Non- weight stable (n=9)	Weight stable (n=12)	Non-weight stable (n=14)	Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction
Hunger	at baseline	45 (9.1)	49 (8.5)	36 (6.6)	37 (23.3)	NS	NS	NS
	30- min after OGTT	28 (6.5)	42 (9.3)	37 (7.1)	31 (7.0)	NS	NS	NS
	60-min after OGTT	27 (8.4)	56 (8.3)	44 (8.7)	36 (6.6)	NS	NS	0.027*
	90-min after OGTT	43 (9.3)	72 (7.8)	43 (8.6)	43 (5.8)	0.083	NS	0.077
	120-min after OGTT	49 (14.7)	70 (6.8)	43 (10.4)	49 (6.3)	NS	NS	NS
Satiety	at baseline	22 (5.7)	42 (8.8)	38 (9.0)	49 (6.9)	NS	0.071	NS
	30- min after OGTT	63 (8.2)	45 (9.2)	57 (8.2)	54 (7.3)	NS	NS	NS
	60-min after OGTT	54 (8.7)	44 (10.1)	52 (8.1)	48 (6.6)	NS	NS	NS
	90-min after OGTT	43 (10.5)	38 (8.7)	46 (7.9)	51 (5.5)	NS	NS	NS
	120-min after OGTT	36 (12.2)	46 (10.7)	47 (6.7)	39 (5.9)	NS	NS	NS
Desire to eat	at baseline	41 (9.0)	39 (9.1)	39 (5.5)	37 (6.8)	NS	NS	NS
	30- min after OGTT	27 (7.2)	49 (9.1)	34 (8.2)	31 (7.0)	NS	NS	NS
	60-min after OGTT	31 (6.9)	58 (8.8)	39 (6.7)	37 (7.3)	NS	NS	0.059

	90-min after OGTT	39 (8.0)	70 (7.5)	40 (7.2)	41 (5.7)	0.063	0.098	0.004*
	120-min after OGTT	49 (12.6)	73 (6.8)	43 (9,7)	46 (6.7)	0.051	NS	NS
Amount of	at baseline	41 (8.2)	56 (8.6)	41 (5.7)	42 (5.5)	NS	NS	NS
prospective food to be consumed	30- min after OGTT	26 (6.0)	47 (7.6)	33 (7.8)	29 (5.5)	NS	NS	0.090
	60-min after OGTT	31 (7.2)	61 (7.7)	41 (7.0)	37 (6.8)	NS	NS	0.023*
	90-min after OGTT	37 (8.0)	70 (8.0)	39 (6.9)	40 (5.6)	0.084	0.085	0.031*
	120-min after OGTT	44 (10.6)	70 (7.1)	40 (8.4)	47 (6.3)	0.086	0.076	NS

Data are mean (sem), # ANCOVA, NS: not significant at 5% significance level: AUC: total

Three statistical analysis of covariance (ANCOVA) models were developed to compare the AUC subjective sensations between the two ethnic groups, controlling for baseline value (Model 1); for age, fat mass and fat-free mass (Model 2) and for age, fat mass, fat-free mass and glucose (Model 3) as shown Table 10. In all models, the interaction between ethnicity and weight stability status was considered.

On the amount of prospective food to be consumed sensation, when no adjustment for confounders was considered, a significant interactive effect between ethnic and weight stability was found. Asians who reported a stable weight during the last 6 months had a lower AUC sensation for amount of prospective food to be consumed than Caucasians (mean (SEM) = 4010 (720.5) vs 4366 (737.5); p =0.034 respectively). In contrast, Asians who reported a unstable weight during the last 6 months had a higher AUC sensation for amount of prospective food to be consumed than Caucasians (mean (SEM) = 7145(880.0) vs 4510 (694.9) p= 0.034 respectively) on average.

After controlling for confounders, a significant interactive effect of ethnicity and weight stability was found on all AUC of subjective sensations, except the AUC of satiety, in all the three models.

For the total response to hunger, this study found that Asians reported less hunger sensation (lower value of AUC of hunger) compared to Caucasians (Model 1: Asians 4168 vs Caucasians 4697, Model 2: Asians 4347 vs Caucasians 4510, Model 3: Asians 4342 vs Caucasians 6461) if the participants had a stable weight during last 6 months. However among those were non-weight stable, Caucasians were found to be less hungry sensations (Model 1: Asians 6752 vs Caucasians 4553, Model 2: Asians 6457 vs Caucasians 4714, Model 3: Asians 6461 vs Caucasians 4700).

A significant interaction on ethnicity and weight stability was also found for total response to desire to eat (Model 1: p=0.044; Model 2: p=0.019, Model 3: p=0.017), suggesting among participants who reported to be weight stable during the last 6 months, Asians had a lower sensation for desire to eat (Model 1: Asians 4293 vs Caucasians 4350, Model 2: Asians 4347 vs Caucasians 4510, Model 3: Asians 4517 vs Caucasians 4630). Among those who reported their weight has been changed more than 2 kg during the last 6 months, Asians had a higher desire to eat as compared to Caucasians (Model 1: Asians 6862 vs Caucasians 4525, Model 2: Asians 6457 vs Caucasians, Model 3: Asians 6741 than Caucasians 4460).

This study also found that among those who reported weight stability in the last 6 months, Asians had a less sensation on the amount of prospective food to be consumed compared to Caucasians (Model 1: Asians 4552 vs Caucasians 4448, Model 2: Asians 4507 vs Caucasians 4332 Model 3: Asians 4500 vs Caucasians 4636).

Among those who reported to have weight changes more than 2 kg in the last 6 months, again Asians were found to have higher sensations for amount of food to be consumed as compared to Caucasians (Model 1: Asians 6488 vs Caucasians 4492, Model 2: Asians 6711 vs Caucasians 4592, Model 3: Asians 6178 vs Caucasians 4466).

Table 10. AUC subjective sensations by ethnicity and weight stability adjusted for model of metabolic profiles

		Asians (n=17)	Caucasians	(n=25)		p values#	
	Weight stable (n=9)	Non-weight stable (n=9)	Weight stable (n=10)	Non-weight stable (n=14)	Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction
tAUC of hunger							
Unadjusted	4041 (839.8)	6878 (884.9)	4658 (846.3)	4586 (634.3)	NS	NS	0.076
Model 1	4168 (601.4)	6752 (601.4)	4697 (623.6)	4553 (577.3)	NS	NS	0.032*
Model 2	4347 (604.3)	6457 (561.9)	4510 (539.2)	4714 (497.6)	NS	0.049*	0.005*
Model 3	4342 (638.8)	6461 (589.4)	4737 (589.6)	4700 (492.9)	NS	NS	0.005*
tAUC of satiety	- 1						-
Unadjusted	5402 (891.9)	5700 (748.7)	5142 (880.0)	5876 (613.4)	NS	NS	NS
Model 1	5920 (859.1)	4623 (859.2)	6079 (514.5)	5550 (475.6)	NS	NS	NS
Model 2	5885 (569.7)	5065 (531.1)	6002 (498.3)	5616 (459.1)	NS	NS	NS
Model 3	5885 (599.5)	5062 (558.6)	6556 (492.8)	5590 (411.8)	NS	NS	NS
tAUC of desire t	o eat	,		•			,

Unadjusted	3808 (781)	7231(881.2)	4403 (723)	4532 (616.7)	NS	NS	0.060
Model 1	4293 (476.0)	6862 (476.0)	4350 (627.6)	4525 (581.0)	NS	0.064	0.044*
Model 2	4491 (567.0)	6736 (528.1)	4245 (572.9)	4614 (528.7)	NS	0.026*	0.019*
Model 3	4517 (553.3)	6741 (515.4)	4630 (562.4)	4460 (470.0)	NS	NS	0.017*
tAUC of prospe	ective amount of food					,	
Unadjusted	4010 (720.5)	7145 (880)	4366 (737.5)	4510 (694.9)	NS	0.072	0.034*
Model 1	4552 (260.6)	6488 (260.6)	4448 (561.8)	4492 (520.1)	NS	NS	0.042*
Model 2	4507 (273.4)	6711 (254.3)	4332 (563.2)	4592 (519.7)	NS	0.041*	0.022*
Model 3	4500 (285.7)	6178 (265.0)	4636 (610.6)	4466 (510.3)	NS	NS	0.022*

Data are mean (sem), #ANCOVA†Two-way ANOVA. tAUC: total AUC, NS: not significant at 5% significance level.

Model 1: adjusted for respective baseline value; Model 2: Model 1 + adjusted for age, fat mass and fat free mass.; Model 2 + adjusted for AUC glucose

4.7 Amount of food consumed during lunch by ethnicity adjusted by metabolic profiles

Again, three ANCOVA models were developed to compare the mean energy and macronutrient intake during subsequent lunch between the two ethnic groups, adjusting for baseline value (Model 1); for age, fat mass and fat-free mass (Model 2) and for age, fat mass, fat- free mass and glucose (Model 3) as shown in Table 11. In all models, the interaction between ethnicity and weight stability status was considered.

Before the afore-mentioned three models, a two-way ANOVA analysis was carried out to compare the unadjusted difference in the mean energy and macronutrient intake between Asians and Caucasians. There was a significant interaction between ethnic and weight stability found on total calorie intake and also on total carbohydrate intake during subsequent lunch. The interactions suggested that in the weight stable group Asians, on average, were found to have a significantly higher total calorie intake (730 vs 498 kcal, p =0.039 respectively) and total carbohydrate intake (133 vs 81 gram, p =0.046 respectively) than Caucasians. However in adjusted model, the non-stable weight group Caucasians had a higher total calorie intake (615 vs 529 kcal, p= 0.039 respectively) and total carbohydrate intake (95 vs 87 gram; p= 0.046 respectively) than Asians.

For total calorie intake, an interaction of ethnicity and weight stability was also found after controlling for age, fat mass, fat free mass and AUC glucose with a p value just on the boundary of significance (Model 2: p=0.050). It suggested that after adjustment of age, body composition and glucose, Asians who reported a stable weight during the last 6 months tended to have a higher calorie intake (mean (SEM) = 698 (97.8) kcal) than Caucasians (mean (SEM) = 439 (75.3) kcal) while Asians who reported a unstable weight during the last 6 months tended to have a lower calorie intake (mean (SEM) = 553 (82.2) kcal) than Caucasians (mean (SEM) = 599 (71.1) kcal) on average.

There was no significant effect of ethnicity on protein intake found after adjustment of age, fat mass, fat-free mass (Model 1) and age, fat mass, fat-free mass, AUC glucose (Model 2). However after controlling for one more variation (lunch energy intake), a significant main effect of ethnicity (p=0.019) on protein intake was observed (Model 3). This finding suggested overall Asians consumed less protein (mean (SEM)= 16 (1.8) gram) as compared to Caucasians (mean (SEM) =19 (1.3) gram) on average regardless of their weight stability status.

The study failed to find any significant difference in the mean of fat intake between ethnic groups, based on the unadjusted model and all three adjusted models.

For total fibre intake, no significant interactions were found based on all models considered as shown in Table 10. However there was a significant main effect of weight stability on fibre intake (Model 1: p=0.025, Model 2: p<0.001, Model 3: p<0.001). After adjustment of confounders, overall those who reported weight stable consumed less fibre on average (Model 1: mean (SEM) = 5.7 (2.4) gram, Model 2: mean (SEM) = 2.7 (2.3) gram, Model 3: mean (SEM) = 4.9 (2.1) gram) as compared to those who were non-weight stable (Model 1: mean(SEM)=11 (2.2) gram, Model 2: mean (SEM) = 12 (1.9) gram, Model 3: mean (SEM) = 10 (1.8) gram) regardless of their ethnicity.

4.8 Palatability of food consumed during lunch by ethnicity

Participants were asked in a 100 mm Visual Analog Scale (0 equals to not pleasant and 10 equals to very pleasant) their feedback on the amount, taste and smell of the buffet lunch. There was no difference on average palatability score of the buffet lunch between the Asian and Caucasian as shown in Table 12 in Appendix 1.

Table 11. Energy and macronutrient intake during buffet lunch by ethnicity and weight stability adjusted to metabolic profiles

	Asia	Asians (n=17)		Caucasians (n=25)		p values #		
	Weight stable (n=9)	Non-weight stable (n=9)	Weight stable (n=10)	Non-weight stable (n=14)	Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction	
Total calorie intal	ka (kaal)							
		700 (01.0)	400 (70.2)	(17 (64.0)	NG	NG	0.020*	
Unadjusted	730 (81.0)	529 (81.0)	498 (70.2)	615 (64.9)	NS	NS	0.039*	
Model 1	704 (97.7)	529 (82.5)	489 (52.8)	604 (71.0)	NS	NS	0.063	
Model 2	698 (97.8)	553 (82.2)	439 (75.3)	599 (71.1)	NS	NS	0.050*	
Total carbohydra	te intake (gram)							
Unadjusted†	133 (15.8)	87 (15.8)	81 (13.7)	95 (12.6)	NS	NS	0.046*	
Model 1	132 (18.1)	91 (15.3)	77.9 (12.9)	87 (13.2)	NS	NS	0.079	
Model 2	130 (18.5)	93 (15.5)	67.6 (14.3)	88 (13.5)	NS	NS	0.059	
Model 3	108 (9.1)	97 (7.5)	89 (7.1)	82 (6.4)	0.097	NS	NS	
Total protein inta	ke (gram)							
Unadjusted†	19 (2.3)	14 (2.3)	18 (2.0)	21 (1.9)	NS	NS	0.087	
Model 1	19 (2.9)	14 (2.5)	17 (2.1)	21 (2.1)	NS	NS	NS	
Model 2	19 (3.1)	15 (2.6)	17 (2.6)	20 (2.3)	NS	NS	0.095	
Model 3	16 (2.0)	16 (2.0)	20 (1.6)	18 (1.1)	0.019*	NS	NS	
Total fat intake (g	gram)							
Unadjusted†	14 (3.8)	14 (3.8)	11 (3.3)	17 (3.0)	NS	NS	NS	

Model 1	11 (4.8)	12 (4.0)	12 (3.4)	20 (3.5)	NS	NS	NS			
Model 2	12 (4.6)	13 (3.9)	11 (3.6)	19 (3.3)	NS	NS	NS			
Model 3	8 (4.0)	14 (3.3)	15 (3.2)	18 (2.9)	NS	NS	NS			
Total fiber intake (g	Total fiber intake (gram)									
Unadjusted†	10 (2.8)	14 (2.8)	7 (2.4)	10 (2.2)	NS	NS	NS			
Model 1	5 (3.4)	12 (2.5)	7 (2.4)	12 (2.5)	NS	0.025*	NS			
Model 2	4 (3.1)	14 (2.7)	5 (2.4)	13 (2.3)	NS	< 0.001*	NS			
Model 3	2 (2.8)	14 (2.4)	7 (2.3)	12 (2.0)	NS	<0.001*	NS			

Data is mean (se), # ANCOVA, † Two-way ANOVA, NS: not significant at 5% significance level Model 1: adjusted for age, fat mass and fat free mass.

Model 2: Model 1 + adjusted for AUC glucose Model 3: Model 2 + lunch energy intake.

4.9 Amount of food consumed during 24-h post OGTT adjusted by model of metabolic profiles.

The study again used the three ANCOVA model to compare the energy and macronutrient intake during 24-h post OGTT between the two ethnic groups, with a adjustment of age, fat mass and fat-free mass (Model 1); of age, fat mass and fat-free mass and glucose (Model 2) and of age, fat mass, fat- free mass, glucose and calorie during dinner (Model 3) as shown in Table 12. In all models, the interaction between ethnicity and weight stability status was considered.

The study failed to find any statistically significant main effects and interactive effects on all four energy and macronutrient intake during 24-h food intake based on the unadjusted two-way ANOVA and all three ANCOVA models.

There were also no main effects of ethnicity, weight stability and interaction effect on most of nutrient intake within 24-h after OGTT indicating that Asians and Caucasians had similar consumption of these nutrients within 24-h after OGTT as shown in Table 13 in the Appendix 1. However a significant effect of weight stability on saturated fat intake (p= 0.017) was found suggesting those who reported a stable weight during the last 6 months had a lower saturated fat intake (overall mean (SEM) = 19 (2.9) gram) than those who were non-weight stable (overall mean (SEM) = 34 (4.3) gram). Asians who reported a stable weight during the last 6 months had a less saturated fat consumption (mean (SEM) = 12 (7.9) gram) than Caucasians (mean (SEM) = 23 (5.9) gram) on average. However, there was no significant difference on saturated fat consumption among those who were non weight stable regardless of their ethnicity.

There was also a significant effect of ethnicity on MUFA intake (p=0.034) was found suggesting Asians, on average, had a lower MUFA intake (overall mean (SEM) = 18 (3.5) gram) than the Caucasians (overall mean (SEM) = 29 (3.5) gram).

Table 12. Energy and macronutrient intake during 24-h food record by ethnicity and weight stability adjusted to metabolic profiles

	Asia	Asians (n=14)		Caucasians (n=22)		p values		
	Weight stable (n=5)	Non-weight stable (n=9)	Weight stable (n=9)	Non- weight stable (n=13)	Ethnic (ETH)	Weight stability(WS)	ETH x WS interaction	
Total calorie intake	e (kcal)	•			1			
Unadjusted†	1372 (295.1)	1988 (220.0)	1793 (220.0)	2060 (183.1)	NS	0.092	NS	
Model 1	1636 (366.6)	2082 (237.2)	1776 (226.6)	1906 (220.8)	NS	NS	NS	
Model 2	1569 (386.3)	2057 (243.1)	1790 (230.2)	1939 (229.5)	NS	NS	NS	
Total carbohydrate	Total carbohydrate intake (gram)						•	
Unadjusted†	188 (41.3)	220 (30.8)	197 (30.8)	198 (25.6)	NS	NS	NS	
Model 1	214 (51.2)	228 (33.1)	194 (31.6)	185 (30.9)	NS	NS	NS	
Model 2	208 (54.2)	196 (32.3)	196 (32.3)	187 (32.2)	NS	NS	NS	
Model 3	240 (37.6)	207 (23.6)	205 (22.2)	181 (22.1)	NS	NS	NS	
Total protein intak	e (gram)							
Unadjusted†	78 (19.4)	88 (14.6)	97 (14.5)	101 (12.0)	NS	NS	NS	
Model 1	102 (23.6)	95 (15.2)	95 (15.2)	88 (14.1)	NS	NS	NS	

Model 2	96 (24.7)	97 (14.7)	93 (15.5)	90 (14.7)	NS	NS	NS
Model 3	111 (16.5)	84 (10.4)	102 (9.7)	88 (9.7)	NS	NS	NS
Total fat intake (gran	<u> </u> n)						
Unadjusted†	32 (16.5)	76 (12.3)	61 (12.3)	89 (10.2)	NS	NS	NS
Model 1	36 (20.1)	78 (13.0)	61 (12.5)	86 (12.1)	NS	0.060	NS
Model 2	32 (21.2)	76 (13.4)	62 (12.7)	88 (12.6)	NS	0.062	NS
Model 3	45 (14.9)	67 (9.3)	69 (9.3)	86 (8.8)	NS	NS	NS
Total fiber intake (gra	am)						
Unadjusted†	27 (6.2)	35 (4.6)	30 (4.6)	23 (3.9)	NS	NS	NS
Model 1	31 (7.4)	38 (4.8)	29 (4.8)	20 (4.4)	0.071	NS	NS
Model 2	29 (7.8)	29 (4.6)	37 (4.8)	21 (4.6)	NS	NS	NS
Model 3	31 (7.7)	36 (4.8)	30 (4.5)	21 (4.5)	NS	NS	NS

Data is mean (sem), #ANCOVA, †two-way ANOVA, NS: not significant at 5% significance level.

Model 1: adjusted for age, fat mass and fat free mass.

Model 2: Model 1 + adjusted for AUC glucose

Model 3: Model 1 + dinner energy intake

CHAPTER FIVE

DISCUSSION

This study was designed to observe subjective appetite sensations on hunger, satiety and prospective food intake among Asian and Caucasian overweight/obese individuals residing in Australia as primary objectives. As several participants provided a history of weight instability, we choose to assess this factor as secondary objective and so statistical aanalyses were conducted with a focus on the interactive effect of weight stability status and ethnicity. Due to difficulties in recruiting study participants of Asians origins thus the study was not able to reach the required sample size and had only 73% power to detect differences in the primary objectives. Therefore this study could serve as a pilot for further study in the future

5.1. Influence of ethnicity and weight stability on subjective hunger/satiety sensations

There was significant main interactive effect between ethnic group and weight stability found on three of the subjective appetite sensations namely: hunger sensation, desire to eat sensation and amount of prospective food to be consumed sensation in this study. Results indicated that after controlling for age, fat mass, fat-free mass and total glucose response, non-weight stable Asians had more sensations of hunger and desire to eat as compared to non-weight stable Caucasian, weight stable Asians and weight stable Caucasians (Table 10).

Weight-stable Asians had less sensation for amount of prospective food to be consumed as compared to the weight-stable Caucasians and that non-weight stable Asians had a more sensation for amount of prospective food to be consumed as compared to the non-weight stable Caucasians. These effects persisted despite adjustment for a variety of confounders including, age, fat mass, fat-free mass and baseline value and glucose response through the use of different models for analysis (Table 10). This result indicates that weight instability per se among Asians could be an important modifier of subjective hunger/satiety sensations.

5.2 The influence of ethnicity and weight fluctuation on oral glucose response

Although the impact of weight fluctuation defined as weight gain and weigh loss over short period of time in affecting resting energy expenditure and body composition still inconclusive (Wadden et al., 1996), the impact of weight fluctuation over longer time periods on insulin levels had been shown by a study among Japanese adults (Yatsuya et al., 2003). Those with larger weight fluctuation had a higher fasting insulin concentration as compared to those who were weight stable (Yatsuya et al., 2003).

In this study, Asians, on average, tended to have a lower fasting glucose level as compared to the other Caucasians despite similar instructions to all participants to fast between 10-12 hours prior to the study day and the similar pre-OGTT condition.

It is a well-accepted technique to provide pre-load meal in order to assess satiety (Rolls, 1995). In this study, all participants were requested to consume ready-to-eat meals during dinner prior to OGTT and then received OGTT as pre-load meal. The use of OGTT as pre-load meals in assessing satiety has several advantages and limitations. Studies have shown that macronutrient composition of pre-load meal affected hunger and satiety sensations (Zhu et al., 2013, Heden et al., 2013, Rolls, 1995, Kissileff, 1984). Thus by having OGTT, the study participants were only exposed to glucose as source of carbohydrate and not to other type of macro-nutrients such as protein or fat. In addition, OGTT serves as a good indicator for glucose response. OGTT can also be used as a good indicator for meal stimulation (Belfiore et al., 1998, Matsuda and DeFronzo, 1999, Reaven, 1988). Despites all of these advantages, there are several limitations to use OGTT as compared to standards breakfast

such as: 1) a liquid meal has been known to provide less satiety as compared to solid meal which could lead to compensation in the subsequent meal (Jones et al., 2013, Almiron-Roig et al., 2003, Heden et al., 2013) and 2) high palatability of sweeten food could trigger overeating (Bellisle et al., 2012, Anderson and Woodend, 2003).

In this study the blood glucose levels after OGTT were also comparable between Asians and Caucasians regardless of their weight stability condition. This is in contrast with study conducted by Venn et al in 2010 who showed that 2-h incremental AUC for glucose among Asians was higher as compared to Caucasians after consuming similar amount of glucose beverage and cereals, suggesting different insulin-glucose mechanism between the two ethnic groups (Venn et al., 2010).

Absolute glucose concentration does not have a straightforward relationship with satiety (de Graaf et al., 2004) which is further explained by study among healthy normo-weight Caucasians in which glycemic response to a standardized meal was not related to appetite sensation, while insulin response was associated (Flint et al., 2006). There were negative correlations between glucose at 2-h after OGTT and subjective sensations for hunger, satiety, desire to eat and amount of food to be consumed in this study. This could be due the influence of dynamic and transient declines of blood glucose as postulated by de Graaf et al in 2004 (de Graaf et al., 2004). However these dynamic and transient change need to be confirmed by frequently monitoring of blood glucose such as 8-10 times in one minute, which was quite difficult to achieve with current blood glucose methodology (de Graaf et al., 2004).

5.3 Association of subjective sensations and energy/macronutrient intake by ethnicity and weight stability

Subjective sensations of hunger, fullness, desire to eat and amount of prospective food to be consumed had been commonly assessed by a validated visual analogue score (Flint et al., 2000, Gregersen et al., 2008).

Asians in this study, on average, had higher subjective hunger sensations as compared to Caucasians. In addition to glucose, age might be another possible explanation for this phenomenon as Asians in this study were significantly younger the Caucasians. Several studies have shown that after an overnight fast, older subjects were less hungry than younger adults (Donini et al., 2003, Rolls et al., 1995, Shide et al., 1995). A negative association between subjective sensations and food intake was commonly present among those older than 65 years of age which lead to reduction of appetite and energy intake (Parker et al., 2004, Donini et al., 2013).

There were different associations between subjective sensations of hunger, fullness, desire to eat and amount of prospective food to be consumed with subsequent macronutrient intake during lunch among weight stable Asians, non-weight stable Asians and non-weight stable Caucasians in this study as shown in Table 7. There was no association between any of subjective sensations with subsequent food intake during lunch among weight-stable Caucasians.

Among weight-stable Asians there was a significant positive association between hunger sensation and fat intake during lunch; negative association between satiety sensation and calorie, carbohydrate, protein, fat and fiber intake during lunch. There were also significant positive associations between desire to eat sensation and fat intake; and between the amount of prospective food to be consumed sensation and protein intake. However, there were no significant associations among non-weight stable Asians. In contrast, there were significant positive associations between amount of prospective food to be consumed and protein intake among non-weight stable Caucasians and between satiety sensation and carbohydrate intake among weight-stable Caucasians These findings further indicates that weight stability could have a stronger influence on subjective sensation and food intake among Asians but not

among Caucasians.

The associations between subjective sensations and food intake were also in accordance with a study conducted by Flint et al in 2007 which found that subjective appetite cues in overweight and obese subjects were associated with subsequent food intake (Flint et al., 2007). However to date, we are not aware of any other study to assess the relationship between appetite cues in overweight/obese subjects from different ethnic groups or from those with different weight stability condition.

The served lunch consisted of pre-selected main ready-to-eat warm meals in addition to other food items such as slices of vegetables, fruits, biscuits, chocolate bar and fruit juice. This buffet meal concept has been used by earlier study (Ping-Delfos and Soares, 2011) and allowed study participants to select and consume the meal to their liking and in accordance to their hunger/satiety sensations. Although the study was conducted in a laboratory setting in which each individual consumed their individual meal, and not in accordance to the normal socio-cultural setting where individual could choose their own meal and consume it within their social context (de Graaf et al., 2004, Woods, 1991), both ethnic groups, on average, consumed comparable amount of calorie during lunch. In addition, most of the study participants found that the meal served was acceptable. The palatability of the food is quite an important factor as it drives satiation and thus influences food intake (De Graaf et al., 1999).

The amount of macro-nutrients intake based on 24-h food record in this study was quite similar with those reported by Conway et al in 2003 (Conway et al., 2003) which assessed the accuracy of two different dietary methods; 24-h food recall and 5 multiple-steps approach, among overweight and obese women in the US. It is interesting to note that the total energy intake for both Asians and Caucasians in this study were still within the recommended daily allowance. Thus the possibility of under-reporting among overweight

and obese men or women which have been frequently reported previously could not be eliminated (Pietilainen et al., 2010, Lichtman et al., 1992, Weber et al., 2001, Lindroos et al., 1999, Bandini et al., 1990).

5.4 The influence of ethnic and weight stability on energy and macronutrient intake

There were different preferences of macronutrient intakes between Asians and Caucasians in relation to weight stability in this study. During the subsequent lunch, Asians, on average, tended to consume more carbohydrate, while Caucasians tended to consume fat as contributor to the total energy intake. Those who were weight stable had a lower fiber intake regardless of the ethnicity. This was in contrast from earlier study comparing native vs migrant Australians from different ethnic groups which found that there higher percentage among migrants Asians who consumed more than five servings of vegetable per day as compared to migrants Caucasians (Astell-Burt et al., 2013a).

During the 24-h food intake, after adjusting to confounders, non-weight stable Asians, on average, tended to consume more calorie and carbohydrate, while weight stable Asians, tended to consume less protein and less fat as compared to those who were non-weight stable. However the differences were not significant. This effect of weight stability condition on fat intake was more prominent during 24-h food intake. Those who were non-weight stable tended to consume more total fat as compared to those who were weight stable, regardless their ethnicity. This could further indicate weight stability as a driver for fat consumption. Interestingly, during lunch, Asians who were weight stable, on average, had a higher calorie intake compared to the other three groups. This phenomenon were reversed during 24-h after OGTT as Asians who were weight stable then tended to consume lower calorie intake. This suggests that short term changes in food intake are counter balanced by 24h intake.

5.5. The influence of ethnicity on body composition and food intake

Asian participants in this study had smaller body build as characterized by having less body weight, less body height and smaller waist-circumference than Caucasians. This is similar with findings from study by Stults-Kolehmainen et al in 2013 which showed Asian men had the smallest build in term of height, weight and BMI as compared to other ethnicities. This study involved 800 participants from four different ethnic groups in the US namely Asian, African-American, Hispanics and non-white Hispanics (Stults-Kolehmainen et al., 2013). The Asians in the study had smaller built, a comparable BMI and percentage of fat mass to the Caucasians. This was in contrast with findings from Hauck et al in 2011 which showed that the 1st generation migrants had lower BMI as compared to native Australians (Hauck et al., 2011).

All of the Asians participants in this study were born outside Australia with average duration of living in Australia about 1/3 of their age indicating that there were 1st generation of migrants. While, many of the Caucasians participants in this study were born in Australia as their average duration of living about 4/5 of their age indicating that most of them were either native or 2nd generation of migrants. The importance of duration of living in influencing the prevalence of obesity and overweight among migrants was shown by Roshania et al in 2008 which analyzed the data from around 6,000 adult immigrants in the United States (Roshania et al., 2008). This study found that immigrants who were younger than 20 years of age at the time of arrival and had lived in the USA for more than 15 years had 11 times higher risk of developing obesity as compared to those who arrived at the same age and had only lived in the USA for about 1 year (Roshania et al., 2008). Although, the particular information on the age of arrival was not collected in this study, we estimated that the Asian participants in this study could possibly arrived in Australia around their early 20s and had lived in the country, on average, around 15 years. This could allow sufficient time to assimilate to the new country environment and culture.

In this study, different body compartments were assessed using DEXA. Earlier study showed that this technique allowed accurate assessment on fat mass and fat-free mass (Williams et al., 2006). There was a negative association between Fat Free Mass and satiety among weight-stable Asians. There was a positive association between Fat Mass and satiety among non-weight-stable Caucasians. The associations between body composition with subjective hunger/satiety sensations were similar to findings from early studies which showed that fat mass and fat-free mass influenced appetite regulation, although these studies did not further analyze the influence of ethnicity and weight stability condition (Benedict et al., 2014, Verdich et al., 2001, Fernandez-Garcia et al., 2013).

5.6 Study limitations

There were difficulties in recruiting study participants of Asians origins thus the study was not able to reach the required sample size and power to test the hypothesis. Difficulties in recruitment for study participation in medical research among certain ethnic groups, especially the minority, had been widely documented (Mohiuddin and Hilleman, 1993, Roberson, 1994, Hussain-Gambles, 2004). Hussain-Gambles in 2004 postulated that participant's culture, social status and gender were key influencing factors for participating (Hussain-Gambles, 2004).

With the current number of participants, the study had only 73% power to detect differences in subjective appetite sensations including hunger and satiety sensations as compared to the initial sample size calculation which would provide 80% power to detect these differences. This would explain why we found several marginal differences such as the differences on unadjusted fasting glucose level, hunger sensations, desire to eat and amount of prospective food to be consumed between Asians and Caucasians regardless of their weight stability status (Table 10). However there were some significant differences too which indicates a much greater effect size than the moderate (0.40) we had envisaged for our

sample size calculations. Clearly with the required sample size, we would have been better positioned to confirm the role of ethnicity in influencing subjective sensations and prospective food intake.

No association between impaired glucose response to OGTT and disruption of subjective hunger/satiety sensations was found in this study. Insulin was not measured in this study due to limited resources which had hindered further exploration on the influence of insulin resistance, in addition to impaired glucose tolerance, on subjective sensations and subsequent food intake. An earlier study showed that insulin concentrations increase hunger ratings independent of changes in blood glucose (Woods and Porte, 1983). However, further studies found that insulin does not directly affect appetite when studies were conducted at euglycemic or hyperglycemic condition (Woo et al., 1984, Holt and Miller, 1995).

The magnitude of weight fluctuation was also not further determined. As weight fluctuation has been shown to influence glucose homeostasis (Lagerpusch et al., 2012), more in-depth information on insulin and weight fluctuation could allow further confirmation of their influence on appetite and food intake.

In this study, despite the differences in characteristic, we managed to have the study conducted in highly controlled situation with minimal impact from external surrounding. Rolls et al in 1995 reviewed that as studies related with food intake were easily influenced by different factors; several conditions must be fulfilled to be able to detect subtle differences in food intake which includes careful selection of pre-load and a thorough screening of study participants in order to ensure homogeneity of their characteristics (age, sex, body weight), (Rolls and Hammer, 1995)

There were no normal/lean subjects in either ethnic group. This could have served as a control group in this study. Having this information would have allowed the study to confirm

the influence of body size as well since studies have shown a disrupted appetite sensation among overweight/obese (Speakman et al., 2002, Brennan et al., 2012). Different ethnicities across Asia were combined together in one group of interest as Asians in this study. Thus the differences in each individual ethnic group were not able to be further distinguished. A further complexity is that several Asian countries have a cultural mix in them as well. For example Singapore and Malaysia have Indians, Malays, Chinese, and Indigenous groups. Such a sample selection was beyond the scope of our study.

CHAPTER SIX

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion

The present study could serves to highlight the possible influence of ethnicity and weight stability on subjective hunger/satiety sensations and subsequent food intake. We found Asians who were weight stable planned to eat less food and had a lower subjective hunger rating, when compared to Caucasians who were weight stable. In contrast, Asians who were non-weight stable planned to eat more food and showed a greater hunger score than their Caucasians' counterpart.

Asians who weight-stable tended to have lower calorie consumption during buffet lunch as compared to the other three groups (weight-stable Caucasian, non-weight stable Asians and non-weight stable Caucasians). However, this phenomenon were reversed during 24-h after OGTT as Asians who were non-weight stable then tended to consume more calorie although the difference was not significant. Asians, on average, tended to consume more carbohydrate, while Caucasians tended to consume fat as contributor to the total energy intake.

Among Asians who were weight-stable, there was significant positive association between hunger sensation and fat intake during lunch so after a high fat lunch these Asians were hungrier as time progressed. When carbohydrate, protein and fiber were considered in addition to fat intake during lunch for weight-stable Asians, a negative association with satiety sensations was found and so if the lunch included carbohydrate, protein, fat and fiber, the feelings of fullness were greater over time.

There was an inverse association between blood glucose response and subjective sensations between the two ethnic groups. Blood glucose response was associated with

subjective sensations only among Asians who were weight stable and Caucasians who were non-weight stable as shown by following findings: 2-h after OGTT was significantly correlated with subjective hunger/satiety sensations among weight-stable Asians but not among the other. Blood glucose response at 2-h after OGTT was negatively correlated with the amount of fat and fibre intake during lunch among Asians who were weight stable, while. AUC glucose response had a significant negative correlation with protein and fat intake among Caucasians who were non-weight stable.

6.2 Recommendation for future research

Future studies on the role of ethnicity in influencing subjective sensations for hunger and satiety should include normal/lean subjects in both ethnic groups to be able to confirm whether there is a disruption of appetite signals in obesity. These studies should also include other potential influencing factors such as insulin, lipid profiles, and other satiety related hormones such as CCK, PYY and Ghrelin, in addition to weight fluctuation. The inclusion of these biomarkers could bring a more holistic overview on the indirect or direct influencing factors on food intake. The impact of migration on weight fluctuation, subjective sensations and food intake should also be considered. This could be achieved through epidemiological studies between Asians in Asia and those who have migrated to other countries including Australia, or in recently-migrated Asians and those who have migrated for a longer period of time.

Finally based on our findings of weight stability, a more detailed report of how amount of weight lost or gained influenced hunger/satiety and prospective food intake would be very useful to the world literature. This will especially valuable if the ethnicity was confirmed as an effect modifier of such outcomes.

Appendix 1-Additional Tables

Table 13. Absolute palatability score of buffet lunch by ethnicity and weight stability

	Asians (n=14)		Caucasians (n=22)		p values#		
	Weight stable (n=5)	Non-weight stable (n=9)	Weight stable (n=9)	Non-weight stable (n=13)	Ethnic	Weight stability	Ethnic* weight stability
Amount of meal (mm)	75 (7.0)	70 (7.0)	65 (6.7)	66 (5.9)	NS	NS	NS
Taste (mm)	68 (8.3)	60 (8.3)	73 (7.9)	70 (6.9)	NS	NS	NS
Smell (mm)	64 (8.9)	56 (8.9)	74 (8.4)	70 (7.7)	NS	NS	NS

Data are mean (SEM), # Two-way ANOVA, NS: not significant at 5% significance level.

Table 14. Nutrients intake within 24-h after OGTT by ethnicity and weight stability

	Asia	Asians (n=14)		(n=22)	p values#		
	Weight stable (n=5)	Non-weight stable (n=9)	Weight stable (n=9)	Non-weight stable (n=13)	Ethnic	Weight stability	Ethnic* weight stability
Saturated fat (g)	12 (7.9)	34 (5.9)	23 (5.9)	39 (4.9)	NS	0.017*	NS
PUFA (g)	5 (4.3)	12 (3.2)	12 (3.2)	14 (2.7)	NS	NS	NS
MUFA (g)	12 (6.6)	21 (4.9)	23 (4.9)	33 (4.1)	0.034	NS	NS
Cholesterol (mg)	263 (70.6)	206 (52.6)	275 (52.6)	352 (43.8)	0.089	NS	NS
Sugar (g)	54 (19.3)	73 (14.4)	86 (14.4)	97 (11.9)	0.077	NS	NS
Thiamin (mg)	0.8 (1.23)	1.86 (0.91)	1.5 (0.91)	2.6 (0.76)	NS	NS	NS
Riboflavin (mg)	1.6 (1.33)	2.3 (0.99)	1.7(0.99)	3.4 (0.82)	NS	NS	NS
Vitamin C (mg)	313 (120.6)	98 (89.0)	84 (90.0)	181 (74.8)	NS	NS	NS
Vitamin A (IU)	2202 (584.6)	1160 (435.7)	561 (435.7)	808 (363.0)	0.074	NS	NS
Vitamin D (IU)	1 (1.8)	3 (1.3)	2 (1.3)	4 (1.1)	NS	NS	NS
Vitamin E (units)	16 (4.3)	7 (3.2)	8 (3.2)	9 (2.7)	NS	NS	NS

Folate (ng)	346 (133.4)	495 (99.4)	320 (99.4)	436.5 (82.7)	NS	NS	NS
Potassium (mg)	2509	3052	3136	3257	NS	NS	NS
	(568.0)	(423.4)	(423.3)	(352.3)			
Calcium (mg)	648 (169.1)	631 (126.0)	522 (126.0)	865.2	NS	NS	NS
				(104.9)			
Magnesium (mg)	340 (62.8)	400 (46.8)	340 (46.8)	361 (38.9)	NS	NS	NS
Iron (mg)	9 (3.2)	15 (2.4)	12 (2.4)	13 (2.0)	NS	NS	NS
Zinc (mg)	8 (3.0)	11 (2.3)	12 (2.3)	14 (1.9)	NS	NS	NS
Iodine (mg)	111 (54.2)	111 (40.4)	83 (40.4)	158 (33.6)	NS	NS	NS
Caffeine (mg)	139 (61.6)	135 (45.9)	97 (45.9)	154 (38.2)	NS	NS	NS

Data are mean (SEM) *p<0.05, # Two-way ANOVA, NS: not significant at 5% significance level

Table 15. Pearson's correlation between body composition and AUC subjective sensations by ethnicity and weight stability

			weig	ht stable			non- we	ight stability	
		Asiar	ns (n=9)	Caucasia	ans (n=12)	Asia	ns (n=9)	Caucas	sians (n=14)
		r*	p value	r*	p value	r*	p value	r*	p value
	AUC of hunger	0.036	NS	-0.163	NS	-0.612	0.080	-0.481	NS
	AUC of satiety	0.116	NS	-0.195	NS	-0.532	NS	0.648	0.012
Fat Mass	AUC for desire to eat	-0.167	NS	-0.040	NS	-0.468	NS	-0.354	NS
	AUC for amount of food to be consumed	0.014	NS	-0.193	NS	-0.479	NS	-0.437	NS
	AUC of hunger	0.595	NS	0.118	NS	-0.390	NS	-0.205	NS
	AUC of satiety	-0.865	0.006	0.157	NS	-0.203	NS	0.302	NS
Fat Free Mass	AUC for desire to eat	0.676	0.066	-0.011	NS	-0.44	NS	0.127	NS
	AUC for amount of food to be consumed	0.652	0.080	0.086	NS	-0.364	NS	-0.145	NS

^{*}Pearson's correlation coefficient, NS: not significant at 5% significance level.



Endothelial Function and Food Intake (EFFI)

Informed Consent Form

J	I,	 	 	

Hereby consent to be a volunteer for the above-mentioned study. I understand that as part of the study, I will allow myself to be screened for suitability as a volunteer.

I have read the Participant Information Sheet and,

I consent to the following:

- ✓ To attend the medical check up
- ✓ To be fasting for 8 hours prior to the appointment time and to only take glucose solution that consists of 75 ml glucose as my breakfast.
- ✓ To have my finger pricked for 4 times on 30 minutes, 1, 2 and 3 hour after I taken the solution at the appointment time.
- ✓ To have my body composition measured using the DEXA machine. I understand that this method involves a very small dose of radiation and that I will have to undergo 1 scan at the beginning of the appointment time.
- ✓ To complete physical activity questionnaire at the time of the study.
- ✓ To complete a 3-day food and drink diary after the study Day.
- ✓ To consume the lunch that is provided at the specified time.
- ✓ To attend all appointments as outlined in the protocol attached.

I fully understand all of the potential risks of these procedures as explained to me. I also understand that my participation is purely voluntary and that signing this form DOES NOT prevent me from withdrawing from the study at any time. I have been assured that confidentiality will be maintained at all stages throughout the study.

Signature:	Date:
Witness:	Date:

This study has been approved by the Curtin University Human Research Ethics Committee (Approval number HR 109/2010). If needed, verification of this approval can be obtained either in writing to the Curtin University Human Research Ethics Committee c/- Office of Research Development, Curtin University of Technology, GPO Box U1987, Perth 6845, or by telephoning 9266-2784.



Endothelial Function and Food Intake (EFFI)

Volunteer information Sheet

Synopsis:

As part of this program, participants will need:

- 1. To come to Curtin University on two occasions.
- 2. To attend a medical check-up.
- 3. To fast for at least 10 hours prior to the Study Day time and to undergo the glucose tolerance test.
- 4. To allow finger prick to be done on 4 occasions before and after drinking the glucose solution.
- 5. To have body composition measured using the DEXA machine.
- 6. To complete a Food Frequency Questionnaire prior to the trial day and to note physical activity diary.
- 7. To complete the hunger and satiety questionnaire and to record their subsequent 24-h food intake.

The participation to this program is on voluntary basis in which participants may opt out of this study at any time, without any fear of recrimination.

Personal and medical information collected for this study by the study doctor and the study team will be kept confidential.

Endothelial Function and Food Intake (EFFI)

Volunteer information Sheet

Information to Volunteers

Thank you for agreeing to participate in the above mentioned study. This document serves to assist you in understanding what is involved in this project. For this study to be a success, we need volunteers who are able to adhere to all parts of the study. Please read this document carefully before you agree to join the study. There is a consent form attached which you will be asked to sign on your first visit. Please be sure that you fully understand the nature of the study and feel free to query us on any aspect, anytime.

Background information

Insulin resistance is a common state among people who are overweight all around the world. Moreover people who have excess body weight are prone to eat more than required due to an inability to feel full after a meal. According to several studies, insulin resistance over the long term leads to endothelial dysfunction, abnormal lipid levels, and hypertension amongst other conditions. Along with factors such as aging, genetics, also sedentary life style may also contribute to insulin resistance.

Study aim

The aim is to study whether people who have excess body weight are at risk for diabetes by having abnormal blood glucose level status. Furthermore, we also would like to study hunger and satiety cues among the study population. The information is crucial in order to be able to plan nutritional advice for the betterment of health of Australians.

Study design

Visit 1- Experiment day (3-4 hours, Curtin School of Public Health and Building 404)

The *day prior to this visit* you will need to consume your last meal by 10:30 pm at the latest as you need to be fasting at least for 10-12 hours. You may drink as much of the provided water as you want; however after this time <u>please do not eat ANYTHING else</u>. This will ensure that you are in a truly fasting state for our measurements. To ensure accuracy of our measurements, it is *vital* to fulfil the following conditions:

- 1. A minimum of 8hours sleep.
- 2. No strenuous exercise for at least 1½days (36hours) prior to the visit.
- 3. Try to avoid too much activity on the morning of the test.

You will need to come to Curtin University by 8 am. All measurements on this day will be conducted at the Health Services Centre of Curtin University. At all times while you are at the health service, health care staff will be available for care and support at all times.

During this visit, we will be determining as your hunger and satiety level throughout the 2 hours of testing. It is important that you come to the laboratory as relaxed as possible and remain relaxed till the end of the testing. Please make sure that you wear comfortable clothing, with minimal metal objects, to facilitate the taking of blood samples, blood pressure measurements.

We will conduct the assessment at School of Public Health, at Building 400 room 209, Curtin University. We will measure your weight, height, waist and hip circumferences. You will then rest for ~30 minutes on a comfortable bed, before the commencement of any

measurement. We will do 4 times finger pricks: before the glucose tolerance test, 30 minutes, 1 hour and 2-hour after the given glucose drink.

You will be served water with 75 g glucose load (sugar) as breakfast. We will ask you to fill in the hunger and satiety questionnaire at 30 minutes, 1 hour, 1.5 hour and 2 hour after the glucose drink. Approximately 2.5 hours after drinking the glucose solution, you will be served an early lunch meal that you chosen previously. You also need to fill in a palatability questionnaire right after you finish your lunch.

We will also do your body scan (Dual X-ray Absorptiometry, DEXA). The DEXA will give us more detailed information about your body composition. The machine uses a small dose of X-rays to get an image of the body. The dose is much less than a chest x-ray and this technique is used on babies and adults alike. You will be required to lie on the scan table of the machine and to stay as motionless as possible while being scanned.

Your responsibility in this study:

If you agree to participate in this study, you should follow the advice given to you by the study team. The attendance day will be according to the agreement between you and us. We request you to return the 24-h food and drink diary by posting the reply paid envelope by the end of the following day.

Possible benefits from participating in the study:

At the end of the study, we will provide a complete report on blood glucose level and your response towards hunger /satiety. You will be given a copy of your body composition measured with the DEXA instrument. You will be informed of your personal results and when all the data have been analysed, a brief final outcome will be sent to your mailing address. If we detect any irregular results that need medical attention, we will share the result with you so that you can consult further with your GP.

Possible risks and side effects:

There are a very few risks and side effects that we foresee. The risk involved in blood collection process includes the possibility of having minor bruises and temporary pain around the area where the blood will be taken. This usually subsides within 24-48 hours.

Also, during the DEXA measurement, you will be exposed to a tiny amount of X-rays which is not deemed harmful to human health. However, we do not scan pregnant women or women who may possibly be pregnant. It is your duty to inform us if you are unsure. Should you have any issues after the trial please contact the Principal Investigator (A/Prof Mario Soares) as soon as possible, on +61-8-92663220 or by email on m.soares@curtin.edu.au.

Important Information for Women Subjects:

X-Ray radiation is prohibited for pregnant women. Please make us aware if you are unsure about the possibility of a pregnancy, as this excludes you from our study.

Costs and payments of participating in the study:

There will be no charge to you for participating in this study. Assuming you come by your own vehicle, an appropriate parking place will be provided for you to facilitate your access to the related buildings. At the end of the study, you will be given a small token of appreciation for your attendance.

Voluntary participation:

You may opt out of this study at any time, without any fear of recrimination. Your decision to quit will not affect your relationship with us or your existing relationship with Curtin University, nor will it impinge on the level of any medical care you may be receiving outside of Curtin University. If you choose to stop taking part in this study, please inform the Principal Investigator or research scientist, as a courtesy.

Your participation in this study may be stopped by the Principal Investigator for any of the following reasons:

- If you do not follow the study instructions
- The study doctor decides it is in the best interest of your health and welfare to stop participating.

Also, you will be informed of new information that might affect your willingness to continue this study.

Confidentiality of study and medical records:

Personal and medical information collected for this study by the study doctor and the study team will be kept confidential. Data will be de-identified by assigning each subject a code. All data will be stored in a locked cabinet for a period of 5 years, in the supervisor's office. Any e-data will be password protected. Your records, to the extent of the applicable laws and regulations, will not be made publicly available. However, Curtin University of Technology Institutional Review Board for the study will be granted direct access to your original personal and medical records if they need to check that the study was done properly, keeping such information confidential. By signing the Informed Consent Form attached, you are authorizing such access to your personal and medical records by concerned people. Data collected and entered into the Case Report Forms are the property of Curtin University of Technology. We will ensure that when we publish our results, your identity will always remain confidential.

In Summary

To participate in this study you will need to be able to:

- ✓ Have finger prick for blood glucose and undergo DEXA scan.
- ✓ Complete and return Food Frequency Questionnaire and activity diary.
- ✓ Complete and return the Hunger and Satiety Questionnaires at the study day.
- ✓ Complete and return 24 h detailed food intake diary after the study day.

As a participant, you will benefit by the following:

- An opportunity to understand whether you are at higher risk to develop diabetes as compared to other people with excess weight.
- On completion of the entire program, you will receive a complete nutritional and blood glucose and body composition.

Further information:

If you have any further queries, please do not hesitate to contact:

Dr. Leilani Lestarina, MD, MSc, MPhil Student

e-mail: l.muharoi@postgrad.curtin.edu.au.

Principal Investigator:

A/Prof. Mario Soares, MBBS, PhD, RNutr

(08) 9266 3220 (Working hours)

Email: m.soares@curtin.edu.au

Associate Professor

Department of Nutrition, Dietetics & Food Science,

School of Public Health, Curtin University of Technology

GPO Box U1987, WA 6845

This study has been approved by the Curtin University Human Research Ethics

Committee (Approval number: HR 109/2010). The Committee is comprised of members

of the public, academics, lawyers, doctors and pastoral carers. Its main role is to protect

participants. If needed, verification of approval can be obtained either in writing to the

Curtin University Human Research Ethics Committee c/- Office of Research

Development, Curtin University of Technology, GPO Box U1987, Perth 6845, or by

telephoning 9266 2784 or by emailing hrec@curtin.edu.au.

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Appendix 4- Screening Questionnaire

Endothelial Function and Food Intake Program (EFFI)

February 2014

Thank you for your interest in the *EFFI* Program. Curtin University Human Research

Ethics Committee has given ethics approval for the conduct of this study. If you have any

ethical concerns regarding the study, (HR 109/2010).you may contact The Secretary, Human

Research Ethics Committee, Curtin University, GPO Box U1987, Perth, WA 6845;by phone

on +618 9266 2784; or by email hrec@curtin.edu.au

Purpose of study

You are being invited to participate in a study which aims to investigate whether the amount of glucose in the body is important to how we handle a meal (appetite sensations). At the end of the study, you will receive a report on your health condition especially in relation to your body composition (fat, muscle and bone mass), sensation of hunger and fullness.

Risks and benefits of participating in the study

This survey is voluntary and has no known risks associated with your participation. You may withdraw from the study at any time even after submission of this questionnaire. Once you are accepted in the study we will furnish you with a participant information sheet that will detail the requirements and objectives of this program. You will also be required to sign an informed consent form.

Screening Survey

In order for us to determine your suitability for our program we invite you to kindly fill this survey. The survey will take approximately 5 minutes.

Confidentiality

Any information that you provide us regarding your identity will be de-identified before being stored, to protect your privacy.

If you have any further questions or would like to have the information sheet/informed consent form prior to filling this survey please email Dr. Leilani Muhardi at l.muharoi@postgrad.curtin.edu.au

Endothelial Function and Food Intake Program (EFFI)

Screening Questionnaire

Please select the box (put an X) corresponding to your answer or complete the spaces as indicated below. If your response is YES to any question, you may provide further details in the space provided.

<i>1</i> .	Demog	raphics
	a)	First name:
	a)	Last name:
	b)	Address:
		Suburb:
		Postcode:
	c)	Telephone: (home)
	d)	(mobile)
	e)	Date of Birth: Age:
	f)	Reported Height (m):
	g)	Reported Weight (kg):
	h)	Menopause: Yes
	No	
	Not Ap	plicable

		i)	Country of Birth:				
		j)	Parents' country of birth:				
		k)	Duration lived in Australia:	(years).			
2.	Ar	re you cu	errently a smoker			Y N [
		If Yes	provide details (optional):				
3.	Do	-	ve more than 2 alcoholic drink provide details (optional):	es per day	Y	N 🗌	
4.			weight fluctuated by 2(or more pride details (optional):			N 🗌	
5.			fer from any of the following o	conditions?			
	a)	Diabete If Y	s 'es provide details			Y L N	Ш
		(optiona					
	b)		ood lipids Yes provide details (optional):			Y 🗌 N	
	c)		ood pressure 'es provide details (optional):			Y 🔲 N	
	d)	Kidney	Disease		Y	N 🗌	

	If Yes provide details (optional):			
e)	Infections requiring antibiotics If Yes provide details (optional):	Y	N 🗌	
f)	Heart/Chest pain If Yes provide details (optional):		Y 🔲 I	v [
g)	Stroke in the last 6 months If Yes provide details (optional):		Y 🗌 Y	v [
Are	you currently on any medications?			
a)	Hormone replacement therapy If Yes provide details (optional):	Y	N□	
b)	Steroids If Yes provide details (optional):	Y	N 🗌	
c)	Lipid lowering drugs		Y 🗌 1	N 🗌
	If Yes provide details (optional):			

	If Yes provide details (optional):			
e)	Vitamin Supplements		Y 🗌	N [
	If Yes provide details (optional):			
f)	Weight loss pills		Y 🗌	N [
	If Yes provide details (optional):			
g)	Any Other medicines		Y 🗌	N
	If Yes provide details (optional):			
	part of this program, we offer you a choice of a dinner and	d lunch meal.	. Please	
	part of this program, we offer you a choice of a dinner and oose at least <u>one</u> of the following as your preferred meal.	d lunch meal.	. Please	
	oose at least <u>one</u> of the following as your preferred meal.	d lunch meal.	. Please	
ch a	oose at least <u>one</u> of the following as your preferred meal.	d lunch meal.	. Please	
ch a	Pose at least one of the following as your preferred meal. Butter chicken & Rice (2-minute cooking)	d lunch meal.	. Please	
a) b)	Butter chicken & Rice (2-minute cooking) Honey Beef & Rice (2-minute cooking)	d lunch meal.	. Please	
a) b) c)	Butter chicken & Rice (2-minute cooking) Honey Beef & Rice (2-minute cooking) Beef black pepper & Rice (2-minute cooking) Tuscan Tomato Fusili (2-minute cooking)- Vegetarian	d lunch meal.	. Please	
a) b) c) d)	Butter chicken & Rice (2-minute cooking) Honey Beef & Rice (2-minute cooking) Beef black pepper & Rice (2-minute cooking) Tuscan Tomato Fusili (2-minute cooking)- Vegetarian	d lunch meal.	Y	N

9. Do you have any jood related duergies	Y	IN
If Yes please specify		

Appendix 5- Visual Analog Scale(Flint et al., 2000, Gregersen et al., 2008, Stubbs et al., 2000)

Visual Analog Scale

Endothelial Function and Food Intake Program (EFFI)

Date: _	
(dd/n	nm/yy)
	_ _ ′mm)
	Baseline
	30 min after meal (OGTT)
	60 min after meal (OGTT)
	90 min after meal (OGTT)
	120 min after meal (OGTT)
	10 min after Lunch

Instructions:

Please provide feedback on the following sensations by placing a vertical line "I" at any point along the scale for each questionnaires below.

1. How hungry do you feel right now?	
Not hungry at all	as hungry as I've felt
2. How full do you feel right now?	
Not full at all	very full
3. How strong is your desire to eat now?	
Very weak	very strong
4. How much food do you think you could eat right now?	
Nothing at all	a large amount

Appendix 6- Palatability Questionnaire (Stubbs et al., 2000)

Palatability Questionnaire

Endothelial Function and Food Intake Program (EFFI

Date: _	_
(dd/mm/yy	y)
Time: _	
(hh/mm)	
	After Breakfast
	After Lunch

Instructions:

Please provide feedback on the following sensations by placing a vertical line "I" at any point along the scale for each questionnaires below.

Too small	too much
- 00 2	iso much
2. How do you feel about the taste of the meal?	
	
Very bad	very pleasant
Very bad 3. What do you think about the smell of the meal?	very pleasant

Appendix 7- Food intake form during lunch

Endothelial Function and Food Intake Program (EFFI

|--|

Subject no : Date

Items	Unit	Amount prepared (gram)	Amount consumed (gram)	Energy (kcal)	Protein (g)	Fat (g)	Sodium (mg)
Tomato			, ,		107	107	
Cucumber							
Main Menu							
Potato chips/ thin crackers							
Juice							
Fruits							
KitKat							
Total							

24-h Food record diary (Block et al., 1990) with permission from Fred Hutchinson Cancer Center

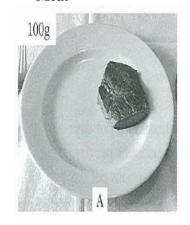
Endothelial Function and Food Intake Program (EFFI

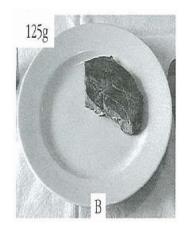
Part 1 – Effects of insulin sensitivity on subjective satiety, hunger and food intake

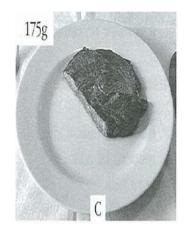
Name:
Subject No: _
Subject Initials: _ _
Date of Clinical Experiment: _ _ _ (dd/mm/yy)
Lunch Time: _ _ (hh/mm)

 $\underline{\textbf{Sample serving size}}$ Please refer to the pictures when you are doing for 24 hour food record it will help you estimate your portion size

Meat







Cereal and Soup









Cooked Vegetables (Green beans, carrot, potato, corn, peas, broccoli)



Pasta, Rice, Noodles



Salad







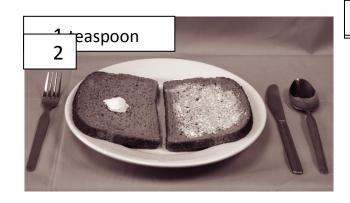




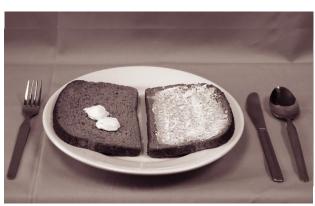


Spread such (E.g. Butter, Mayonnaise or peanut butter)

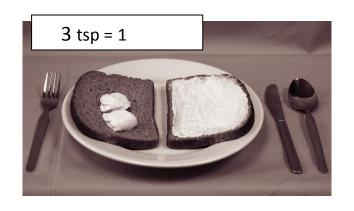
Margarine













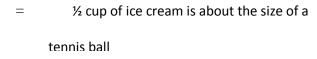




40 grams of cheese is about the size of 4







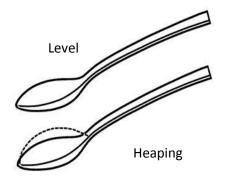


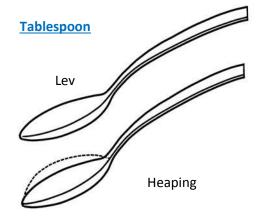


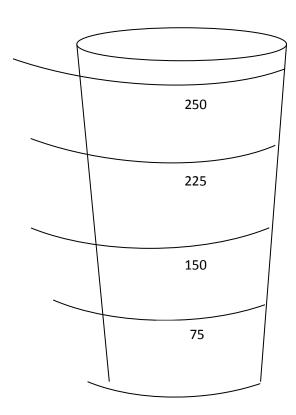
1 cup of mashed potatoes or b

about the size of a fist

Teaspoons (tsp)







24 hour food record

General Instructions

- Please eat as you usually eat.
- Record everything you eat and drink, including snacks.
- Complete the *every* column for each meal or snack.
- Please write clearly.

How to Record Each Food

• Describe each food and beverage in detail, as best you can.

Please refer to the pictures when you are doing for 24 hour food record it will help you estimate your portion size

For example

Tim	Meal	Food and beverage description	Quantity consumed	
e				
7.3	Breakfas	Toast (white bread)	2 slices	
0 am	t	Cheese	1 slices (~20g)	
		Butter	¼ teaspoon	
10.	Morning	Fruit yoghurt	250 g tub	
30	tea (or			
	snack)			
12.	Lunch	Burger (chicken, lettuce, tomato,	1 burger bun	
30		mayo)	~50g chicken (no	
			skin)	
			2 slices tomato	
			2 leaves lettuce	
			1 teaspoon mayo	

EFFI 2010	Subject N°: _	Subject initials:
-----------	-----------------	-------------------

Ti	Meal	Food and beverage description	Quantity consumed
me		description	
			1

Ti	Meal	Food and beverage	Quantity consumed
me		Food and beverage description	

Appendix 9- Total Food Emotio	onal Questionnaire (TEFQ-Short version)

Total Food Emotional Questionnaire (Short version) (Karlsson et al., 2000)

Endothelial Function and Food Intake Program (EFFI)

Part 1 – Effects of insulin sensitivity on subjective satiety, hunger and food intake

Instructions

For question number 1-5: please tick the answers that mostly reflect your situation.

Pai	rt A.
1. I	deliberately take small helping as means of controlling my weight.
	Definitely True
	Mostly True
	Mostly False
	Definitely False
2. I	consciously hold back at meals in order not to gain weight.
	Definitely True
Ш	Mostly True
	Mostly False
	Definitely False
3. I	do not eat some foods because they make me fat.
	Definitely True
	Mostly True
П	Mostly False

	Definitely False	
4.]	How frequently do you avoid 'stocking up' on tempting foods?	
	Almost never	
	Seldom	
	Usually	
	Almost always	
5.]	How likely are you to consciously eat less than what you want	
	Unlikely	
	Slightly likely	
	Moderately	
	Very likely	
6. (On a scale of 1 to 8, where 1 means no restraint in eating (eating whatever you wan	nt,
whenev	ver you want it) and 8 means total restraints (constantly limiting food intake and no	ever
'giving	in'), what number would you give your self	

For	questions	helow	nlesse	tick the	answers	that	mostly	reflect	vour	situation	
T. OI	questions	DCIOW	picasc	uck uit	answeis	шаі	mosuv	ICHECL	vou	Situation	io.

Part. B.

	When I smell a sizzling steak or a juicy piece of meat, I find it very difficult to keep
from ea	ating, even if I have just finished a meal
	Definitely True
	Deliberately True
	Mostly True
	Mostly False
	Definitely False
2. 8	Sometimes when I start eating, I just can't seem to stop.
	Definitely True
	Deliberately True
	Mostly True
	Mostly False
	Definitely False

3. Being with someone who is eating often makes me hungry enough to eat also.

	Definitely True
	Deliberately True
	Mostly True
	Mostly False
	Definitely False
4.	When I see a real delicacy, I often get so hungry that I have to eat right away
	Definitely True
	Mostly True
	Mostly False
	Definitely False
5.	I also get so hungry that my stomach often seems like a bottomless pit.
	Definitely True
	Mostly True
	Mostly False
	Definitely False

6. I a	am always hungry so it is hard for me to stop eating before I finish the food on my
plate.	
	Definitely True
	Mostly True
	Mostly False
	Definitely False
7. I a	am always hungry enough to eat at any time.
	Definitely True
	Mostly True
	Mostly False
	Definitely False
8. H	ow often do you feel hungry?
	Only at meal times
	Sometimes between meals
	Often between meals
	Almost always

9. Do you go on eating binges though you are not hungry?			
	Never		
	Rarely		
	Sometimes		
	At least once a week		
	part C.		
1.	When I feel anxious, I find myself eating		
	Definitely True		
	Mostly True		
	Mostly False		
	Definitely False		
2.	When I feel blue, I often overeat.		
	Definitely True		
	Mostly True		
	Mostly False		
	Definitely False		

3. When I feel lonely, I console myself by eating.			
	Definitely True		
	Deliberately True		
	Mostly True		
	Mostly False		
П	Definitely False		

Appendix 10- Physical Activity Dairy

Physical Activity Dairy (Baecke et al., 1982)

Endothelial Function and Food Intake Program (EFFI)

Part 1- Effects of insulin sensitivity on subjective satiety, hunger and food intake

Subject Screening No: _	_
Initials:	
(First 2 letters of Last/First n	ames)
Date: _ _ _	ı
Date. _ _ _	.l
(dd/mm/yy)	
Time:	
(hh/mm)	
Occupation:	

Please put an X on the boxes below.

1.	At work, I sit					
	☐ Never					
	Seldom					
	Sometimes					
	oft	en				
	alv	vays				
2.	. At work, I stand					
	☐ Ne	ver				
	\square Se	ldom				
	Sometimes					
	oft	en				
	always					
3.	At wor	k, I walk				
		Never				
		Seldom				
		Sometimes				
		often				
		always				
4.	At wor	k, I lift heavy loads				
		Never				
		Seldom				
		Sometimes				
		often				
		always				

5.	After w	orking I am tired
		Never
		Seldom
		Sometimes
		often
		always
6.	At worl	x I sweat
		Never
		Seldom
		Sometimes
		often
		always
7.	Do you	play sport?
		If yes:
		- Which sport do you play most frequently?
		- How many hours per week ?
		- How many months a year ?
8.	During	leisure time, I sweat
		Never
		Seldom
		Sometimes
		often
		always

9. During leisure time, I play sport			
	Never		
	Seldom		
	Sometimes		
	often		
	always		
10. During	g leisure time, I watch TV		
	Never		
	Seldom		
	Sometimes		
	often		
	always		
11. During	g leisure time, I walk		
	Never		
	Seldom		
	Sometimes		
	often		
	always		
12. During leisure time, I cycle			
	Never		
	Seldom		
	Sometimes		
	often		
	always		

13. How many minutes do you walk/cycle per day from work, school and shopping?

REFERENCES

- World Health Organization. Waist circumference and waist-hip ratio : report of a WHO expert consultation, Geneva, 8-11 December 2008.
- ABDULLAH, A. R., HASAN, H. A. & RAIGANGAR, V. L. 2009. Analysis of the relationship of leptin, high-sensitivity C-reactive protein, adiponectin, insulin, and uric acid to metabolic syndrome in lean, overweight, and obese young females. *Metab Syndr Relat Disord*, 7, 17-22.
- ABS 2013. 4338.0 Profiles of Health, Australia, 2011-13. Australian Bureau of Statistics.
- ADA 2010. Standards of medical care in diabetes-2010. Diabetes Care, 33 Suppl 1, S11-61.
- AL RIFAI, M., SILVERMAN, M. G., NASIR, K., BUDOFF, M. J., BLANKSTEIN, R., SZKLO, M., KATZ, R., BLUMENTHAL, R. S. & BLAHA, M. J. 2015. The association of nonalcoholic fatty liver disease, obesity, and metabolic syndrome, with systemic inflammation and subclinical atherosclerosis: the Multi-Ethnic Study of Atherosclerosis (MESA). *Atherosclerosis*, 239, 629-33.
- ALMIRON-ROIG, E., CHEN, Y. & DREWNOWSKI, A. 2003. Liquid calories and the failure of satiety: how good is the evidence? *Obes Rev*, 4, 201-12.
- ANDERSON, G. H. & WOODEND, D. 2003. Consumption of sugars and the regulation of short-term satiety and food intake. *Am J Clin Nutr*, 78, 843S-849S.
- ANDREWS, J. M., DORAN, S., HEBBARD, G. S., RASSIAS, G., SUN, W. M. & HOROWITZ, M. 1998a. Effect of glucose supplementation on appetite and the pyloric motor response to intraduodenal glucose and lipid. *Am J Physiol*, 274, G645-52
- ANDREWS, J. M., RAYNER, C. K., DORAN, S., HEBBARD, G. S. & HOROWITZ, M. 1998b. Physiological changes in blood glucose affect appetite and pyloric motility during intraduodenal lipid infusion. *Am J Physiol*, 275, G797-804.
- ASTELL-BURT, T., FENG, X., CROTEAU, K. & KOLT, G. S. 2013a. Influence of neighbourhood ethnic density, diet and physical activity on ethnic differences in weight status: A study of 214,807 adults in Australia. *Social Science & Medicine*, 93, 70-77.
- ASTELL-BURT, T., FENG, X., CROTEAU, K. & KOLT, G. S. 2013b. Influence of neighbourhood ethnic density, diet and physical activity on ethnic differences in weight status: a study of 214,807 adults in Australia. *Soc Sci Med*, 93, 70-7.
- AUSTIN, J. & MARKS, D. 2009. Hormonal regulators of appetite. *Int J Pediatr Endocrinol*, 2009, 141753.
- BAECKE, J. A., BUREMA, J. & FRIJTERS, J. E. 1982. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr*, 36, 936-42.
- BANDINI, L. G., SCHOELLER, D. A., CYR, H. N. & DIETZ, W. H. 1990. Validity of reported energy intake in obese and nonobese adolescents. *Am J Clin Nutr*, 52, 421-5.
- BATTERHAM, R. L., FFYTCHE, D. H., ROSENTHAL, J. M., ZELAYA, F. O., BARKER, G. J., WITHERS, D. J. & WILLIAMS, S. C. 2007. PYY modulation of cortical and hypothalamic brain areas predicts feeding behaviour in humans. *Nature*, 450, 106-9.
- BAVDEKAR, A., YAJNIK, C. S., FALL, C. H., BAPAT, S., PANDIT, A. N., DESHPANDE, V., BHAVE, S., KELLINGRAY, S. D. & JOGLEKAR, C. 1999. Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? *Diabetes*, 48, 2422-9.
- BELFIORE, F., IANNELLO, S. & VOLPICELLI, G. 1998. Insulin sensitivity indices calculated from basal and OGTT-induced insulin, glucose, and FFA levels. *Mol Genet Metab*, 63, 134-41.

- BELLISLE, F., DREWNOWSKI, A., ANDERSON, G. H., WESTERTERP-PLANTENGA, M. & MARTIN, C. K. 2012. Sweetness, satiation, and satiety. *J Nutr*, 142, 1149S-54S
- BENEDICT, C., AXELSSON, T., SODERBERG, S., LARSSON, A., INGELSSON, E., LIND, L. & SCHIOTH, H. B. 2014. Fat mass and obesity-associated gene (FTO) is linked to higher plasma levels of the hunger hormone ghrelin and lower serum levels of the satiety hormone leptin in older adults. *Diabetes*, 63, 3955-9.
- BHOPAL, R. 2006. Race and ethnicity: responsible use from epidemiological and public health perspectives. *J Law Med Ethics*, 34, 500-7, 479.
- BIRO, G., HULSHOF, K. F., OVESEN, L., AMORIM CRUZ, J. A. & GROUP, E. 2002. Selection of methodology to assess food intake. *Eur J Clin Nutr*, 56 Suppl 2, S25-32.
- BISOGNI, C. A., FALK, L. W., MADORE, E., BLAKE, C. E., JASTRAN, M., SOBAL, J. & DEVINE, C. M. 2007. Dimensions of everyday eating and drinking episodes. *Appetite*, 48, 218-31.
- BLOCK, G. 1982. A review of validations of dietary assessment methods. *Am J Epidemiol*, 115, 492-505.
- BLOCK, G., WOODS, M., POTOSKY, A. & CLIFFORD, C. 1990. Validation of a self-administered diet history questionnaire using multiple diet records. *J Clin Epidemiol*, 43, 1327-35.
- BLUNDELL, J. E. & GILLETT, A. 2001. Control of food intake in the obese. *Obes Res*, 9 Suppl 4, 263S-270S.
- BLUNDELL, J. E., LAWTON, C. L., COTTON, J. R. & MACDIARMID, J. I. 1996. Control of human appetite: implications for the intake of dietary fat. *Annu Rev Nutr*, 16, 285-319.
- BORAI, A., LIVINGSTONE, C., ZARIF, H., MEHTA, S., KHOLEIF, M., ABDELAAL, M., AL-GHAMDI, H. & FERNS, G. 2009. A comparative study of insulin resistance for Saudi and Caucasian subjects across a range of glycaemic categories. *Diabetes & Metabolic Syndrome: Clinical Research & Reviews*, 3, 204-210.
- BOUGOULIA, M., TZOTZAS, T., EFTHYMIOU, H., KOLIAKOS, G., KONSTANTINIDIS, T., TRIANTOS, A. & KRASSAS, G. E. 1999. Leptin concentrations during oral glucose tolerance test (OGTT) in obese and normal weight women. *Int J Obes Relat Metab Disord*, 23, 625-8.
- BRENNAN, I. M., LUSCOMBE-MARSH, N. D., SEIMON, R. V., OTTO, B., HOROWITZ, M., WISHART, J. M. & FEINLE-BISSET, C. 2012. Effects of fat, protein, and carbohydrate and protein load on appetite, plasma cholecystokinin, peptide YY, and ghrelin, and energy intake in lean and obese men. *Am J Physiol Gastrointest Liver Physiol*, 303, G129-40.
- BROWNELL, K. D. & RODIN, J. 1994. Medical, metabolic, and psychological effects of weight cycling. *Arch Intern Med*, 154, 1325-30.
- BUTTET, S. & DOLAR, V. 2014. Toward a quantitative theory of food consumption choices and body weight. *Econ Hum Biol*.
- CAKIR, M., SARI, R., TOSUN, O. & KARAYALCIN, U. 2005. Leptin Response to Oral Glucose Tolerance Test in Obese and Nonobese Premenopausal Women. *Endocrine Research*, 31, 1-8.
- CANCELLO, R. & CLEMENT, K. 2006. Is obesity an inflammatory illness? Role of low-grade inflammation and macrophage infiltration in human white adipose tissue. *BJOG*, 113, 1141-7.
- CASTELLANOS, E. H., CHARBONEAU, E., DIETRICH, M. S., PARK, S., BRADLEY, B. P., MOGG, K. & COWAN, R. L. 2009. Obese adults have visual attention bias for food cue images: evidence for altered reward system function. *Int J Obes (Lond)*, 33, 1063-73.
- CATON, S. J., BALL, M., AHERN, A. & HETHERINGTON, M. M. 2004. Dose-dependent effects of alcohol on appetite and food intake. *Physiol Behav*, 81, 51-8.

- CHAPMAN, I. M., GOBLE, E. A., WITTERT, G. A., MORLEY, J. E. & HOROWITZ, M. 1998. Effect of intravenous glucose and euglycemic insulin infusions on short-term appetite and food intake. *Am J Physiol*, 274, R596-603.
- CHENG, G., HILBIG, A., DROSSARD, C., ALEXY, U. & KERSTING, M. 2013. Relative validity of a 3 d estimated food record in German toddlers. *Public Health Nutr*, 16, 645-52.
- CHIU, K. C., COHAN, P., LEE, N. P. & CHUANG, L. M. 2000. Insulin sensitivity differs among ethnic groups with a compensatory response in beta-cell function. *Diabetes Care*, 23, 1353-8.
- CLARKSTON, W. K., PANTANO, M. M., MORLEY, J. E., HOROWITZ, M., LITTLEFIELD, J. M. & BURTON, F. R. 1997. Evidence for the anorexia of aging: gastrointestinal transit and hunger in healthy elderly vs. young adults. *Am J Physiol*, 272, R243-8.
- CONSIDINE, R. V., SINHA, M. K., HEIMAN, M. L., KRIAUCIUNAS, A., STEPHENS, T. W., NYCE, M. R., OHANNESIAN, J. P., MARCO, C. C., MCKEE, L. J., BAUER, T. L. & ET AL. 1996. Serum immunoreactive-leptin concentrations in normal-weight and obese humans. *N Engl J Med*, 334, 292-5.
- CONUS, F., RABASA-LHORET, R. & PERONNET, F. 2007. Characteristics of metabolically obese normal-weight (MONW) subjects. *Appl Physiol Nutr Metab*, 32, 4-12.
- CONWAY, J. M., INGWERSEN, L. A., VINYARD, B. T. & MOSHFEGH, A. J. 2003. Effectiveness of the US Department of Agriculture 5-step multiple-pass method in assessing food intake in obese and nonobese women. *Am J Clin Nutr*, 77, 1171-8.
- DAGOGO-JACK, S., FANELLI, C., PARAMORE, D., BROTHERS, J. & LANDT, M. 1996. Plasma leptin and insulin relationships in obese and nonobese humans. *Diabetes*, 45, 695-8.
- DE GRAAF, C. 1993. The validity of appetite ratings. Appetite, 21, 156-60.
- DE GRAAF, C., BLOM, W. A., SMEETS, P. A., STAFLEU, A. & HENDRIKS, H. F. 2004. Biomarkers of satiation and satiety. *Am J Clin Nutr*, 79, 946-61.
- DE GRAAF, C., DE JONG, L. S. & LAMBERS, A. C. 1999. Palatability affects satiation but not satiety. *Physiol Behav*, 66, 681-8.
- DE LAUZON, B., ROMON, M., DESCHAMPS, V., LAFAY, L., BORYS, J. M., KARLSSON, J., DUCIMETIERE, P., CHARLES, M. A. & FLEURBAIX LAVENTIE VILLE SANTE STUDY, G. 2004. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. *J Nutr*, 134, 2372-80.
- DE WIT, J. B., STOK, F. M., SMOLENSKI, D. J., DE RIDDER, D. D., DE VET, E., GASPAR, T., JOHNSON, F., NUREEVA, L. & LUSZCZYNSKA, A. 2014. Food Culture in the Home Environment: Family Meal Practices and Values Can Support Healthy Eating and Self-Regulation in Young People in Four European Countries. *Appl Psychol Health Well Being*.
- DICKINSON, S., COLAGIURI, S., FARAMUS, E., PETOCZ, P. & BRAND-MILLER, J. C. 2002. Postprandial hyperglycemia and insulin sensitivity differ among lean young adults of different ethnicities. *J Nutr*, 132, 2574-9.
- DOBBELSTEYN, C. J., JOFFRES, M. R., MACLEAN, D. R. & FLOWERDEW, G. 2001. A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health Surveys. *Int J Obes Relat Metab Disord*, 25, 652-61.
- DONINI, L. M., POGGIOGALLE, E., PIREDDA, M., PINTO, A., BARBAGALLO, M., CUCINOTTA, D. & SERGI, G. 2013. Anorexia and eating patterns in the elderly. *PLoS One*, 8, e63539.
- DONINI, L. M., SAVINA, C. & CANNELLA, C. 2003. Eating habits and appetite control in the elderly: the anorexia of aging. *Int Psychogeriatr*, 15, 73-87.

- DOUCET, E., ST-PIERRE, S., ALMERAS, N. & TREMBLAY, A. 2003. Relation between appetite ratings before and after a standard meal and estimates of daily energy intake in obese and reduced obese individuals. *Appetite*, 40, 137-43.
- DRAPEAU, V., BLUNDELL, J., THERRIEN, F., LAWTON, C., RICHARD, D. & TREMBLAY, A. 2005. Appetite sensations as a marker of overall intake. *Br J Nutr*, 93, 273-80.
- DUNSTAN, D. W., ZIMMET, P. Z., WELBORN, T. A., DE COURTEN, M. P., CAMERON, A. J., SICREE, R. A., DWYER, T., COLAGIURI, S., JOLLEY, D., KNUIMAN, M., ATKINS, R. & SHAW, J. E. 2002. The rising prevalence of diabetes and impaired glucose tolerance: the Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care*, 25, 829-34.
- ELLO-MARTIN, J. A., LEDIKWE, J. H. & ROLLS, B. J. 2005. The influence of food portion size and energy density on energy intake: implications for weight management. *Am J Clin Nutr*, 82, 236S-241S.
- EXPERT COMMITTEE ON THE, D. & CLASSIFICATION OF DIABETES, M. 2003. Report of the expert committee on the diagnosis and classification of diabetes mellitus. *Diabetes Care*, 26 Suppl 1, S5-20.
- FERNANDEZ-GARCIA, J. C., MURRI, M., COIN-ARAGUEZ, L., ALCAIDE, J., EL BEKAY, R. & TINAHONES, F. J. 2013. GLP-1 and peptide YY secretory response after fat load is impaired by insulin resistance, impaired fasting glucose and type 2 diabetes in morbidly obese subjects. *Clin Endocrinol (Oxf)*.
- FLINT, A., GREGERSEN, N. T., GLUUD, L. L., MOLLER, B. K., RABEN, A., TETENS, I., VERDICH, C. & ASTRUP, A. 2007. Associations between postprandial insulin and blood glucose responses, appetite sensations and energy intake in normal weight and overweight individuals: a meta-analysis of test meal studies. *Br J Nutr*, 98, 17-25
- FLINT, A., MOLLER, B. K., RABEN, A., SLOTH, B., PEDERSEN, D., TETENS, I., HOLST, J. J. & ASTRUP, A. 2006. Glycemic and insulinemic responses as determinants of appetite in humans. *Am J Clin Nutr*, 84, 1365-73.
- FLINT, A., RABEN, A., BLUNDELL, J. E. & ASTRUP, A. 2000. Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *Int J Obes Relat Metab Disord*, 24, 38-48.
- FLOWERS, E., MOLINA, C., MATHUR, A. & REAVEN, G. M. 2013. Adiposity and cardiovascular risk clustering in South Asians. *Metab Syndr Relat Disord*, 11, 434-40
- FRIEDMAN, M. I. & TORDOFF, M. G. 1986. Fatty acid oxidation and glucose utilization interact to control food intake in rats. *Am J Physiol*, 251, R840-5.
- GALGANI, J. & RAVUSSIN, E. 2008. Energy metabolism, fuel selection and body weight regulation. *Int J Obes (Lond)*, 32 Suppl 7, S109-19.
- GEORGE, L., BACHA, F., LEE, S., TFAYLI, H., ANDREATTA, E. & ARSLANIAN, S. 2011. Surrogate estimates of insulin sensitivity in obese youth along the spectrum of glucose tolerance from normal to prediabetes to diabetes. *J Clin Endocrinol Metab*, 96, 2136-45.
- GERSOVITZ, M., MADDEN, J. P. & SMICIKLAS-WRIGHT, H. 1978. Validity of the 24-hr. dietary recall and seven-day record for group comparisons. *J Am Diet Assoc*, 73, 48-55.
- GERSTEIN, H. C., SANTAGUIDA, P., RAINA, P., MORRISON, K. M., BALION, C., HUNT, D., YAZDI, H. & BOOKER, L. 2007a. Annual incidence and relative risk of diabetes in people with various categories of dysglycemia: A systematic overview and meta-analysis of prospective studies. *Diabetes Research and Clinical Practice*, 78, 305-312.
- GERSTEIN, H. C., SANTAGUIDA, P., RAINA, P., MORRISON, K. M., BALION, C., HUNT, D., YAZDI, H. & BOOKER, L. 2007b. Annual incidence and relative risk

- of diabetes in people with various categories of dysglycemia: a systematic overview and meta-analysis of prospective studies. *Diabetes Res Clin Pract*, 78, 305-12.
- GIBNEY, M. J. & WOLEVER, T. M. 1997. Periodicity of eating and human health: present perspective and future directions. *Br J Nutr*, 77 Suppl 1, S3-5.
- GIELKENS, H. A., VERKIJK, M., LAM, W. F., LAMERS, C. B. & MASCLEE, A. A. 1998. Effects of hyperglycemia and hyperinsulinemia on satiety in humans. *Metabolism*, 47, 321-4.
- GREGERSEN, N. T., FLINT, A., BITZ, C., BLUNDELL, J. E., RABEN, A. & ASTRUP, A. 2008. Reproducibility and power of ad libitum energy intake assessed by repeated single meals. *Am J Clin Nutr*, 87, 1277-81.
- HAUCK, K., HOLLINGSWORTH, B. & MORGAN, L. 2011. BMI differences in 1st and 2nd generation immigrants of Asian and European origin to Australia. *Health & Place*, 17, 78-85.
- HEALTH, O. W. 2008. Waist circumference and waist-hip ratio: report of a WHO expert consultation, Geneva, 8-11 December 2008.
- HEDEN, T. D., LIU, Y., SIMS, L., KEARNEY, M. L., WHALEY-CONNELL, A. T., CHOCKALINGAM, A., DELLSPERGER, K. C., FAIRCHILD, T. J. & KANALEY, J. A. 2013. Liquid meal composition, postprandial satiety hormones, and perceived appetite and satiety in obese women during acute caloric restriction. *Eur J Endocrinol*, 168, 593-600.
- HENRIKSEN, J. E., ALFORD, F., HANDBERG, A., VAAG, A., WARD, G. M., KALFAS, A. & BECK-NIELSEN, H. 1994. Increased glucose effectiveness in normoglycemic but insulin-resistant relatives of patients with non-insulin-dependent diabetes mellitus. A novel compensatory mechanism. *J Clin Invest*, 94, 1196-204.
- HENRY, C. J., LIGHTOWLER, H. J., NEWENS, K., SUDHA, V., RADHIKA, G., SATHYA, R. M. & MOHAN, V. 2008. Glycaemic index of common foods tested in the UK and India. *Br J Nutr*, 99, 840-5.
- HILL, J. O. 2006. Understanding and addressing the epidemic of obesity: an energy balance perspective. *Endocr Rev*, 27, 750-61.
- HOLT, S. H. & MILLER, J. B. 1995. Increased insulin responses to ingested foods are associated with lessened satiety. *Appetite*, 24, 43-54.
- HORNER, K. M., BYRNE, N. M. & KING, N. A. 2014. Reproducibility of subjective appetite ratings and ad libitum test meal energy intake in overweight and obese males. *Appetite*, 81, 116-22.
- HUSSAIN-GAMBLES, M. 2004. South Asian patients' views and experiences of clinical trial participation. *Fam Pract*, 21, 636-42.
- ILLNER, A. K., NOTHLINGS, U., WAGNER, K., WARD, H. & BOEING, H. 2010. The assessment of individual usual food intake in large-scale prospective studies. *Ann Nutr Metab*, 56, 99-105.
- INDULEKHA, K., ANJANA, R. M., SURENDAR, J. & MOHAN, V. 2011. Association of visceral and subcutaneous fat with glucose intolerance, insulin resistance, adipocytokines and inflammatory markers in Asian Indians (CURES-113). *Clin Biochem*, 44, 281-7.
- JACOBSEN, D. J., BAILEY, B. W., LECHEMINANT, J. D., HILL, J. O., MAYO, M. S. & DONNELLY, J. E. 2005. A comparison of three methods of analyzing post-exercise oxygen consumption. *Int J Sports Med*, 26, 34-8.
- JAGANNATHAN, R., NANDITHA, A., SUNDARAM, S., SIMON, M., SHETTY, A. S., SNEHALATHA, C., JOHNSTON, D. G. & RAMACHANDRAN, A. 2014. Screening among male industrial workers in India shows high prevalence of impaired glucose tolerance, undetected diabetes and cardiovascular risk clustering. J Assoc Physicians India, 62, 312-5.
- JONES, L. V., JONES, K. M., HENSMAN, C., BERTUCH, R., MCGEE, T. L. & DIXON, J. B. 2013. Solid versus liquid-satiety study in well-adjusted lap-band patients. *Obes Surg*, 23, 1266-72.

- JOWITT, L. M., LU, L. W. & RUSH, E. C. 2014. Migrant Asian Indians in New Zealand; prediction of metabolic syndrome using body weights and measures. *Asia Pac J Clin Nutr*, 23, 385-93.
- KAJIOKA, T., TSUZUKU, S., SHIMOKATA, H. & SATO, Y. 2002. Effects of intentional weight cycling on non-obese young women. *Metabolism*, 51, 149-54.
- KANAUCHI, M., KANAUCHI, K., KIMURA, K., INOUE, T. & SAITO, Y. 2006. Utility of elevated 2-hour postload plasma glucose as an alternative to elevated fasting glucose as a criterion for the metabolic syndrome. *Metabolism*, 55, 1323-6.
- KARLSSON, J., PERSSON, L. O., SJOSTROM, L. & SULLIVAN, M. 2000. Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *Int J Obes Relat Metab Disord*, 24, 1715-25.
- KELEMEN, L. E. 2007. GI Epidemiology: nutritional epidemiology. *Aliment Pharmacol Ther*, 25, 401-7.
- KERSHAW, E. E. & FLIER, J. S. 2004. Adipose tissue as an endocrine organ. *J Clin Endocrinol Metab*, 89, 2548-56.
- KISSILEFF, H. R. 1984. Satiating efficiency and a strategy for conducting food loading experiments. *Neurosci Biobehav Rev*, 8, 129-35.
- KNUDSEN, V. K., GILLE, M. B., NIELSEN, T. H., CHRISTENSEN, T., FAGT, S. & BILTOFT-JENSEN, A. 2011. Relative validity of the pre-coded food diary used in the Danish National Survey of Diet and Physical Activity. *Public Health Nutr*, 14, 2110-6.
- KOBAYASHI, S., MURAKAMI, K., SASAKI, S., OKUBO, H., HIROTA, N., NOTSU, A., FUKUI, M. & DATE, C. 2011. Comparison of relative validity of food group intakes estimated by comprehensive and brief-type self-administered diet history questionnaires against 16 d dietary records in Japanese adults. *Public Health Nutr*, 14, 1200-11.
- KROTKIEWSKI, M., BJORNTORP, P., SJOSTROM, L. & SMITH, U. 1983. Impact of obesity on metabolism in men and women. Importance of regional adipose tissue distribution. *J Clin Invest*, 72, 1150-62.
- KRUGER, R., SHULTZ, S. P., MCNAUGHTON, S. A., RUSSELL, A. P., FIRESTONE, R. T., GEORGE, L., BECK, K. L., CONLON, C. A., VON HURST, P. R., BREIER, B., JAYASINGHE, S. N., O'BRIEN, W. J., JONES, B. & STONEHOUSE, W. 2015. Predictors and risks of body fat profiles in young New Zealand European, Maori and Pacific women: study protocol for the women's EXPLORE study. *Springerplus*, 4, 128.
- LAGERPUSCH, M., BOSY-WESTPHAL, A., KEHDEN, B., PETERS, A. & MULLER, M. J. 2012. Effects of brief perturbations in energy balance on indices of glucose homeostasis in healthy lean men. *Int J Obes (Lond)*, 36, 1094-101.
- LAKSHMI, S., METCALF, B., JOGLEKAR, C., YAJNIK, C. S., FALL, C. H. & WILKIN, T. J. 2012. Differences in body composition and metabolic status between white U.K. and Asian Indian children (EarlyBird 24 and the Pune Maternal Nutrition Study). *Pediatr Obes*, 7, 347-54.
- LASSALE, C., GUILBERT, C., KEOGH, J., SYRETTE, J., LANGE, K. & COX, D. N. 2009. Estimating food intakes in Australia: validation of the Commonwealth Scientific and Industrial Research Organisation (CSIRO) food frequency questionnaire against weighed dietary intakes. *J Hum Nutr Diet*, 22, 559-66.
- LEAN, M. E., HAN, T. S. & DEURENBERG, P. 1996. Predicting body composition by densitometry from simple anthropometric measurements. *Am J Clin Nutr*, 63, 4-14.
- LICHTMAN, S. W., PISARSKA, K., BERMAN, E. R., PESTONE, M., DOWLING, H., OFFENBACHER, E., WEISEL, H., HESHKA, S., MATTHEWS, D. E. & HEYMSFIELD, S. B. 1992. Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *N Engl J Med*, 327, 1893-8.

- LINDROOS, A. K., LISSNER, L. & SJOSTROM, L. 1999. Does degree of obesity influence the validity of reported energy and protein intake? Results from the SOS Dietary Questionnaire. Swedish Obese Subjects. *Eur J Clin Nutr*, 53, 375-8.
- LINDSTROM, M. & SUNDQUIST, K. 2005. The impact of country of birth and time in Sweden on overweight and obesity: a population-based study. *Scand J Public Health*, 33, 276-84.
- LU, J., BI, Y., WANG, T., WANG, W., MU, Y., ZHAO, J., LIU, C., CHEN, L., SHI, L., LI, Q., WAN, Q., WU, S., QIN, G., YANG, T., YAN, L., LIU, Y., WANG, G., LUO, Z., TANG, X., CHEN, G., HUO, Y., GAO, Z., SU, Q., YE, Z., WANG, Y., DENG, H., YU, X., SHEN, F., CHEN, L., ZHAO, L., DAI, M., XU, M., XU, Y., CHEN, Y., LAI, S. & NING, G. 2014. The relationship between insulin-sensitive obesity and cardiovascular diseases in a Chinese population: results of the REACTION study. *Int J Cardiol*, 172, 388-94.
- LUBREE, H. G., REGE, S. S., BHAT, D. S., RAUT, K. N., PANCHNADIKAR, A., JOGLEKAR, C. V., YAJNIK, C. S., SHETTY, P. & YUDKIN, J. 2002. Body fat and cardiovascular risk factors in Indian men in three geographical locations. *Food Nutr Bull*, 23, 146-9.
- MARI, A., PACINI, G., MURPHY, E., LUDVIK, B. & NOLAN, J. J. 2001. A model-based method for assessing insulin sensitivity from the oral glucose tolerance test. *Diabetes Care*, 24, 539-48.
- MARINOU, K., HODSON, L., VASAN, S. K., FIELDING, B. A., BANERJEE, R., BRISMAR, K., KOUTSILIERIS, M., CLARK, A., NEVILLE, M. J. & KARPE, F. 2014. Structural and functional properties of deep abdominal subcutaneous adipose tissue explain its association with insulin resistance and cardiovascular risk in men. *Diabetes Care*, 37, 821-9.
- MARTINEZ, J. A. 2000. Body-weight regulation: causes of obesity. *Proc Nutr Soc*, 59, 337-45
- MATSUDA, M. & DEFRONZO, R. A. 1999. Insulin sensitivity indices obtained from oral glucose tolerance testing: comparison with the euglycemic insulin clamp. *Diabetes Care*, 22, 1462-70.
- MATTHEWS, D. R., HOSKER, J. P., RUDENSKI, A. S., NAYLOR, B. A., TREACHER, D. F. & TURNER, R. C. 1985. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*, 28, 412-9.
- MAYER-DAVIS, E. J., MONACO, J. H., HOEN, H. M., CARMICHAEL, S., VITOLINS, M. Z., REWERS, M. J., HAFFNER, S. M., AYAD, M. F., BERGMAN, R. N. & KARTER, A. J. 1997. Dietary fat and insulin sensitivity in a triethnic population: the role of obesity. The Insulin Resistance Atherosclerosis Study (IRAS). *Am J Clin Nutr*, 65, 79-87.
- MAYER, J. 1955. Regulation of energy intake and the body weight: the glucostatic theory and the lipostatic hypothesis. *Ann N Y Acad Sci*, 63, 15-43.
- MELANSON, K. J., WESTERTERP-PLANTENGA, M. S., SARIS, W. H., SMITH, F. J. & CAMPFIELD, L. A. 1999. Blood glucose patterns and appetite in time-blinded humans: carbohydrate versus fat. *Am J Physiol*, 277, R337-45.
- MOHIUDDIN, S. M. & HILLEMAN, D. E. 1993. Gender and racial bias in clinical pharmacology trials. *Ann Pharmacother*, 27, 972-3.
- MONTANI, J. P., SCHUTZ, Y. & DULLOO, A. G. 2015. Dieting and weight cycling as risk factors for cardiometabolic diseases: who is really at risk? *Obes Rev*, 16 Suppl 1, 7-18.
- MURAKAMI, K., SHIMBO, M. & FUKINO, Y. 2005. Comparison of energy intakes estimated by weighed dietary record and diet history questionnaire with total energy expenditure measured by accelerometer in young Japanese women. *J Nutr Sci Vitaminol (Tokyo)*, 51, 58-67.

- MYERS, R. J., KLESGES, R. C., ECK, L. H., HANSON, C. L. & KLEM, M. L. 1988. Accuracy of self-reports of food intake in obese and normal-weight individuals: effects of obesity on self-reports of dietary intake in adult females. *Am J Clin Nutr*, 48, 1248-51.
- NAZARE, J. A., SMITH, J. D., BOREL, A. L., HAFFNER, S. M., BALKAU, B., ROSS, R., MASSIEN, C., ALMERAS, N. & DESPRES, J. P. 2012. Ethnic influences on the relations between abdominal subcutaneous and visceral adiposity, liver fat, and cardiometabolic risk profile: the International Study of Prediction of Intra-Abdominal Adiposity and Its Relationship With Cardiometabolic Risk/Intra-Abdominal Adiposity. *Am J Clin Nutr*, 96, 714-26.
- NI MHURCHU, C., PARAG, V., NAKAMURA, M., PATEL, A., RODGERS, A. & LAM, T. H. 2006. Body mass index and risk of diabetes mellitus in the Asia-Pacific region. *Asia Pac J Clin Nutr*, 15, 127-33.
- NIJS, I. M., MURIS, P., EUSER, A. S. & FRANKEN, I. H. 2010. Differences in attention to food and food intake between overweight/obese and normal-weight females under conditions of hunger and satiety. *Appetite*, 54, 243-54.
- NTUK, U. E., GILL, J. M., MACKAY, D. F., SATTAR, N. & PELL, J. P. 2014. Ethnic-specific obesity cutoffs for diabetes risk: cross-sectional study of 490,288 UK biobank participants. *Diabetes Care*, 37, 2500-7.
- OGLE, G. D., ALLEN, J. R., HUMPHRIES, I. R., LU, P. W., BRIODY, J. N., MORLEY, K., HOWMAN-GILES, R. & COWELL, C. T. 1995. Body-composition assessment by dual-energy x-ray absorptiometry in subjects aged 4-26 y. *Am J Clin Nutr*, 61, 746-53.
- OSBORN, R. L., FORYS, K. L., PSOTA, T. L. & SBROCCO, T. 2011. Yo-yo dieting in African American women: weight cycling and health. *Ethn Dis*, 21, 274-80.
- OSTLUND, R. E., JR., STATEN, M., KOHRT, W. M., SCHULTZ, J. & MALLEY, M. 1990. The ratio of waist-to-hip circumference, plasma insulin level, and glucose intolerance as independent predictors of the HDL2 cholesterol level in older adults. *N Engl J Med*, 322, 229-34.
- PARKER, B. A., LUDHER, A. K., LOON, T. K., HOROWITZ, M. & CHAPMAN, I. M. 2004. Relationships of ratings of appetite to food intake in healthy older men and women. *Appetite*, 43, 227-33.
- PENEAU, S., MEKHMOUKH, A., CHAPELOT, D., DALIX, A. M., AIRINEI, G., HERCBERG, S. & BELLISLE, F. 2009. Influence of environmental factors on food intake and choice of beverage during meals in teenagers: a laboratory study. *Br J Nutr*, 102, 1854-9.
- PIETILAINEN, K. H., KORKEILA, M., BOGL, L. H., WESTERTERP, K. R., YKI-JARVINEN, H., KAPRIO, J. & RISSANEN, A. 2010. Inaccuracies in food and physical activity diaries of obese subjects: complementary evidence from doubly labeled water and co-twin assessments. *Int J Obes (Lond)*, 34, 437-45.
- PING-DELFOS, W. C. & SOARES, M. 2011. Diet induced thermogenesis, fat oxidation and food intake following sequential meals: influence of calcium and vitamin D. *Clin Nutr*, 30, 376-83.
- PRATT-PHILLIPS, S. E., GEOR, R. J. & MCCUTCHEON, L. J. 2015. Comparison among the euglycemic-hyperinsulinemic clamp, insulin-modified frequently sampled intravenous glucose tolerance test, and oral glucose tolerance test for assessment of insulin sensitivity in healthy Standardbreds. *Am J Vet Res*, 76, 84-91.
- PRUESSNER, J. C., KIRSCHBAUM, C., MEINLSCHMID, G. & HELLHAMMER, D. H. 2003. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, 28, 916-31.
- RASMUSSEN-TORVIK, L. J., WASSEL, C. L., DING, J., CARR, J., CUSHMAN, M., JENNY, N. & ALLISON, M. A. 2012. Associations of body mass index and insulin resistance with leptin, adiponectin, and the leptin-to-adiponectin ratio across ethnic

- groups: the Multi-Ethnic Study of Atherosclerosis (MESA). *Ann Epidemiol*, 22, 705-9.
- REAVEN, G. M. 1988. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes*, 37, 1595-607.
- RIEDY, C. A., CHAVEZ, M., FIGLEWICZ, D. P. & WOODS, S. C. 1995. Central insulin enhances sensitivity to cholecystokinin. *Physiol Behav*, 58, 755-60.
- ROBERSON, N. L. 1994. Clinical trial participation. Viewpoints from racial/ethnic groups. *Cancer*, 74, 2687-91.
- ROLLS, B. J. 1994. Appetite and satiety in the elderly. *Nutr Rev*, 52, S9-10.
- ROLLS, B. J. 1995. Carbohydrates, fats, and satiety. Am J Clin Nutr, 61, 960S-967S.
- ROLLS, B. J., ANDERSEN, A. E., MORAN, T. H., MCNELIS, A. L., BAIER, H. C. & FEDOROFF, I. C. 1992. Food intake, hunger, and satiety after preloads in women with eating disorders. *Am J Clin Nutr*, 55, 1093-103.
- ROLLS, B. J., DIMEO, K. A. & SHIDE, D. J. 1995. Age-related impairments in the regulation of food intake. *Am J Clin Nutr*, 62, 923-31.
- ROLLS, B. J., FEDOROFF, I. C. & GUTHRIE, J. F. 1991a. Gender differences in eating behavior and body weight regulation. *Health Psychol*, 10, 133-42.
- ROLLS, B. J. & HAMMER, V. A. 1995. Fat, carbohydrate, and the regulation of energy intake. *Am J Clin Nutr*, 62, 1086S-1095S.
- ROLLS, B. J., KIM-HARRIS, S., FISCHMAN, M. W., FOLTIN, R. W., MORAN, T. H. & STONER, S. A. 1994. Satiety after preloads with different amounts of fat and carbohydrate: implications for obesity. *Am J Clin Nutr*, 60, 476-87.
- ROLLS, B. J., KIM, S., MCNELIS, A. L., FISCHMAN, M. W., FOLTIN, R. W. & MORAN, T. H. 1991b. Time course of effects of preloads high in fat or carbohydrate on food intake and hunger ratings in humans. *Am J Physiol*, 260, R756-63.
- ROSHANIA, R., NARAYAN, K. M. & OZA-FRANK, R. 2008. Age at arrival and risk of obesity among US immigrants. *Obesity (Silver Spring)*, 16, 2669-75.
- ROSS, R., ARU, J., FREEMAN, J., HUDSON, R. & JANSSEN, I. 2002. Abdominal adiposity and insulin resistance in obese men. *Am J Physiol Endocrinol Metab*, 282, E657-63.
- ROSS, R., FORTIER, L. & HUDSON, R. 1996. Separate associations between visceral and subcutaneous adipose tissue distribution, insulin and glucose levels in obese women. *Diabetes Care*, 19, 1404-11.
- SANTIAGO-TORRES, M., ADAMS, A. K., CARREL, A. L., LAROWE, T. L. & SCHOELLER, D. A. 2014. Home food availability, parental dietary intake, and familial eating habits influence the diet quality of urban Hispanic children. *Child Obes*, 10, 408-15.
- SCHMID, R., SCHUSDZIARRA, V., SCHULTE-FROHLINDE, E., MAIER, V. & CLASSEN, M. 1989. Effect of CCK on insulin, glucagon, and pancreatic polypeptide levels in humans. *Pancreas*, 4, 653-61.
- SCHMIEGELOW, M. D., HEDLIN, H., MACKEY, R. H., MARTIN, L. W., VITOLINS, M. Z., STEFANICK, M. L., PEREZ, M. V., ALLISON, M. & HLATKY, M. A. 2015. Race and ethnicity, obesity, metabolic health, and risk of cardiovascular disease in postmenopausal women. *J Am Heart Assoc*, 4.
- SCHRIEKS, I. C., STAFLEU, A., GRIFFIOEN-ROOSE, S., DE GRAAF, C., WITKAMP, R. F., BOERRIGTER-RIJNEVELD, R. & HENDRIKS, H. F. 2015. Moderate alcohol consumption stimulates food intake and food reward of savoury foods. *Appetite*, 89, 77-83.
- SCHWARTZ, G. J. 2010. Brainstem integrative function in the central nervous system control of food intake. *Forum Nutr*, 63, 141-51.
- SCHWARTZ, M. W. 2006. Central nervous system regulation of food intake. *Obesity (Silver Spring)*, 14 Suppl 1, 1S-8S.

- SCHWARTZ, M. W., SEELEY, R. J., CAMPFIELD, L. A., BURN, P. & BASKIN, D. G. 1996. Identification of targets of leptin action in rat hypothalamus. *J Clin Invest*, 98, 1101-6.
- SCHWARTZ, M. W., WOODS, S. C., PORTE, D., JR., SEELEY, R. J. & BASKIN, D. G. 2000. Central nervous system control of food intake. *Nature*, 404, 661-71.
- SEELEY, R. J., VAN DIJK, G., CAMPFIELD, L. A., SMITH, F. J., BURN, P., NELLIGAN, J. A., BELL, S. M., BASKIN, D. G., WOODS, S. C. & SCHWARTZ, M. W. 1996. Intraventricular leptin reduces food intake and body weight of lean rats but not obese Zucker rats. *Horm Metab Res*, 28, 664-8.
- SEKIKAWA, A., EGUCHI, H., IGARASHI, K., TOMINAGA, M., ABE, T., FUKUYAMA, H. & KATO, T. 1999. Waist to hip ratio, body mass index, and glucose intolerance from Funagata population-based diabetes survey in Japan. *Tohoku J Exp Med*, 189, 11-20.
- SHAH, A., HERNANDEZ, A., MATHUR, D., BUDOFF, M. J. & KANAYA, A. M. 2012. Adipokines and body fat composition in South Asians: results of the Metabolic Syndrome and Atherosclerosis in South Asians Living in America (MASALA) study. *Int J Obes (Lond)*, 36, 810-6.
- SHAW, J. E., SICREE, R. A. & ZIMMET, P. Z. 2010. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract*, 87, 4-14.
- SHEN, W., PUNYANITYA, M., CHEN, J., GALLAGHER, D., ALBU, J., PI-SUNYER, X., LEWIS, C. E., GRUNFELD, C., HESHKA, S. & HEYMSFIELD, S. B. 2006. Waist circumference correlates with metabolic syndrome indicators better than percentage fat. *Obesity (Silver Spring)*, 14, 727-36.
- SHIDE, D. J., CABALLERO, B., REIDELBERGER, R. & ROLLS, B. J. 1995. Accurate energy compensation for intragastric and oral nutrients in lean males. *Am J Clin Nutr*, 61, 754-64.
- SIBBALD, C., SETO, W., TAYLOR, T., SAUNDERS, E. F., DOYLE, J. & DUPUIS, L. L. 2008. Determination of area under the whole blood concentration versus time curve after first intravenous cyclosporine dose in children undergoing hematopoietic stem cell transplant: limited sampling strategies. *Ther Drug Monit*, 30, 434-8.
- SKRHA, J., HILGERTOVA, J., JAROLIMKOVA, M., KUNESOVA, M. & HILL, M. 2010. Meal test for glucose-dependent insulinotropic peptide (GIP) in obese and type 2 diabetic patients. *Physiol Res*, 59, 749-55.
- SLEDDERING, M. A., BAKKER, L. E., JANSSEN, L. G., MEINDERS, A. E. & JAZET, I. M. 2013. Higher insulin and glucagon-like peptide-1 (GLP-1) levels in healthy, young South Asians as compared to Caucasians during an oral glucose tolerance test. *Metabolism*.
- SPARROW, D., BORKAN, G. A., GERZOF, S. G., WISNIEWSKI, C. & SILBERT, C. K. 1986. Relationship of fat distribution to glucose tolerance. Results of computed tomography in male participants of the Normative Aging Study. *Diabetes*, 35, 411-5.
- SPEAKMAN, J. R., STUBBS, R. J. & MERCER, J. G. 2002. Does body mass play a role in the regulation of food intake? *Proc Nutr Soc*, 61, 473-87.
- SPEECHLY, D. P. & BUFFENSTEIN, R. 2000. Appetite dysfunction in obese males: evidence for role of hyperinsulinaemia in passive overconsumption with a high fat diet. *Eur J Clin Nutr*, 54, 225-33.
- STRAIN, G. W., HERSHCOPF, R. J. & ZUMOFF, B. 1992. Food intake of very obese persons: quantitative and qualitative aspects. *J Am Diet Assoc*, 92, 199-203.
- STRAZNICKY, N. E., LAMBERT, G. W., MCGRANE, M. T., MASUO, K., DAWOOD, T., NESTEL, P. J., EIKELIS, N., SCHLAICH, M. P., ESLER, M. D., SOCRATOUS, F., CHOPRA, R. & LAMBERT, E. A. 2009. Weight loss may reverse blunted sympathetic neural responsiveness to glucose ingestion in obese subjects with metabolic syndrome. *Diabetes*, 58, 1126-32.
- STUBBS, R. J., HUGHES, D. A., JOHNSTONE, A. M., ROWLEY, E., REID, C., ELIA, M., STRATTON, R., DELARGY, H., KING, N. & BLUNDELL, J. E. 2000. The

- use of visual analogue scales to assess motivation to eat in human subjects: a review of their reliability and validity with an evaluation of new hand-held computerized systems for temporal tracking of appetite ratings. *Br J Nutr*, 84, 405-15.
- STULTS-KOLEHMAINEN, M. A., STANFORTH, P. R., BARTHOLOMEW, J. B., LU, T., ABOLT, C. J. & SINHA, R. 2013. DXA estimates of fat in abdominal, trunk and hip regions varies by ethnicity in men. *Nutr Diabetes*, 3, e64.
- STUNKARD, A. J. & MESSICK, S. 1985. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *J Psychosom Res*, 29, 71-83.
- STUNKARD, A. J. & WADDEN, T. A. 1990. Restrained eating and human obesity. *Nutr Rev*, 48, 78-86; discussion 114-31.
- SUNDQUIST, J. & WINKLEBY, M. 2000. Country of birth, acculturation status and abdominal obesity in a national sample of Mexican-American women and men. *Int J Epidemiol*, 29, 470-7.
- TAMAM, S., BELLISSIMO, N., PATEL, B. P., THOMAS, S. G. & ANDERSON, G. H. 2012. Overweight and obese boys reduce food intake in response to a glucose drink but fail to increase intake in response to exercise of short duration. *Appl Physiol Nutr Metab*, 37, 520-9.
- TAN, C. E., MA, S., WAI, D., CHEW, S. K. & TAI, E. S. 2004. Can we apply the National Cholesterol Education Program Adult Treatment Panel definition of the metabolic syndrome to Asians? *Diabetes Care*, 27, 1182-6.
- THOMAS, C., NIGHTINGALE, C. M., DONIN, A. S., RUDNICKA, A. R., OWEN, C. G., COOK, D. G. & WHINCUP, P. H. 2012. Ethnic and socioeconomic influences on childhood blood pressure: the Child Heart and Health Study in England. *J Hypertens*, 30, 2090-7.
- TOLONEN, H., KOPONEN, P., NASKA, A., MANNISTO, S., BRODA, G., PALOSAARI, T. & KUULASMAA, K. 2015. Challenges in standardization of blood pressure measurement at the population level. *BMC Med Res Methodol*, 15, 33.
- TORDOFF, M. G. & FRIEDMAN, M. I. 1986. Hepatic portal glucose infusions decrease food intake and increase food preference. *Am J Physiol*, 251, R192-6.
- UNWIN, N., HARLAND, J., WHITE, M., BHOPAL, R., WINOCOUR, P., STEPHENSON, P., WATSON, W., TURNER, C. & ALBERTI, K. G. 1997. Body mass index, waist circumference, waist-hip ratio, and glucose intolerance in Chinese and Europid adults in Newcastle, UK. *J Epidemiol Community Health*, 51, 160-6.
- VAN DER PLOEG, G. E., WITHERS, R. T. & LAFORGIA, J. 2003. Percent body fat via DEXA: comparison with a four-compartment model. *J Appl Physiol* (1985), 94, 499-506
- VANDERWEELE, D. A. 1994. Insulin is a prandial satiety hormone. *Physiol Behav*, 56, 619-22.
- VENDITTI, E. M., WING, R. R., JAKICIC, J. M., BUTLER, B. A. & MARCUS, M. D. 1996. Weight cycling, psychological health, and binge eating in obese women. *J Consult Clin Psychol*, 64, 400-5.
- VENN, B. S., WILLIAMS, S. M. & MANN, J. I. 2010. Comparison of postprandial glycaemia in Asians and Caucasians. *Diabet Med*, 27, 1205-8.
- VERDICH, C., TOUBRO, S., BUEMANN, B., LYSGARD MADSEN, J., JUUL HOLST, J. & ASTRUP, A. 2001. The role of postprandial releases of insulin and incretin hormones in meal-induced satiety--effect of obesity and weight reduction. *Int J Obes Relat Metab Disord*, 25, 1206-14.
- VIARDOT, A., HEILBRONN, L. K., HERZOG, H., GREGERSEN, S. & CAMPBELL, L. V. 2008. Abnormal postprandial PYY response in insulin sensitive nondiabetic subjects with a strong family history of type 2 diabetes. *Int J Obes (Lond)*, 32, 943-8.
- VRANG, N., MADSEN, A. N., TANG-CHRISTENSEN, M., HANSEN, G. & LARSEN, P. J. 2006. PYY(3-36) reduces food intake and body weight and improves insulin sensitivity in rodent models of diet-induced obesity. *Am J Physiol Regul Integr Comp Physiol*, 291, R367-75.

- WADDEN, T. A., FOSTER, G. D., STUNKARD, A. J. & CONILL, A. M. 1996. Effects of weight cycling on the resting energy expenditure and body composition of obese women. *Int J Eat Disord*, 19, 5-12.
- WANG, W. B., WANG, F. D., XU, B., ZHU, J. F., SHEN, W., XIAO, X. R. & JIANG, Q. W. 2006. [A cost-effectiveness study on a case-finding program of tuberculosis through screening those suspects with chronic cough symptoms in the rich rural areas]. *Zhonghua Liu Xing Bing Xue Za Zhi*, 27, 857-60.
- WASIM, H., AL-DAGHRI, N. M., CHETTY, R., MCTERNAN, P. G., BARNETT, A. H. & KUMAR, S. 2006. Relationship of serum adiponectin and resistin to glucose intolerance and fat topography in South-Asians. *Cardiovasc Diabetol*, 5, 10.
- WEBER, J. L., REID, P. M., GREAVES, K. A., DELANY, J. P., STANFORD, V. A., GOING, S. B., HOWELL, W. H. & HOUTKOOPER, L. B. 2001. Validity of self-reported energy intake in lean and obese young women, using two nutrient databases, compared with total energy expenditure assessed by doubly labeled water. *Eur J Clin Nutr*, 55, 940-50.
- WELLE, S. L., THOMPSON, D. A., CAMPBELL, R. G. & LILAVIVATHANA, U. 1980. Increased hunger and thirst during glucoprivation in humans. *Physiol Behav*, 25, 397-403.
- WHITING, D. R., GUARIGUATA, L., WEIL, C. & SHAW, J. 2011. IDF diabetes atlas: global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Res Clin Pract*, 94, 311-21.
- WILLIAMS, J. E., WELLS, J. C., WILSON, C. M., HAROUN, D., LUCAS, A. & FEWTRELL, M. S. 2006. Evaluation of Lunar Prodigy dual-energy X-ray absorptiometry for assessing body composition in healthy persons and patients by comparison with the criterion 4-component model. *Am J Clin Nutr*, 83, 1047-54.
- WOLEVER, T. M. 2004. Effect of blood sampling schedule and method of calculating the area under the curve on validity and precision of glycaemic index values. *Br J Nutr*, 91, 295-301.
- WOO, R., KISSILEFF, H. R. & PI-SUNYER, F. X. 1984. Elevated postprandial insulin levels do not induce satiety in normal-weight humans. *Am J Physiol*, 247, R745-9.
- WOODS, S. C. 1991. The eating paradox: how we tolerate food. *Psychol Rev*, 98, 488-505.
- WOODS, S. C. 2004. Gastrointestinal satiety signals I. An overview of gastrointestinal signals that influence food intake. *Am J Physiol Gastrointest Liver Physiol*, 286, G7-13
- WOODS, S. C. & PORTE, D., JR. 1983. The role of insulin as a satiety factor in the central nervous system. *Adv Metab Disord*, 10, 457-68.
- WULAN, S. N., WESTERTERP, K. R. & PLASQUI, G. 2010. Ethnic differences in body composition and the associated metabolic profile: a comparative study between Asians and Caucasians. *Maturitas*, 65, 315-9.
- WURTMAN, J. J. 1988. The anorexia of aging: a problem not restricted to calorie intake. *Neurobiol Aging*, 9, 22-3.
- YAJNIK, C. S., FALL, C. H., COYAJI, K. J., HIRVE, S. S., RAO, S., BARKER, D. J., JOGLEKAR, C. & KELLINGRAY, S. 2003. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int J Obes Relat Metab Disord*, 27, 173-80.
- YAJNIK, C. S., KATRE, P. A., JOSHI, S. M., KUMARAN, K., BHAT, D. S., LUBREE, H. G., MEMANE, N., KINARE, A. S., PANDIT, A. N., BHAVE, S. A., BAVDEKAR, A. & FALL, C. H. 2015. Higher glucose, insulin and insulin resistance (HOMA-IR) in childhood predict adverse cardiovascular risk in early adulthood: the Pune Children's Study. *Diabetologia*.
- YATSUYA, H., TAMAKOSHI, K., YOSHIDA, T., HORI, Y., ZHANG, H., ISHIKAWA, M., ZHU, S., KONDO, T. & TOYOSHIMA, H. 2003. Association between weight fluctuation and fasting insulin concentration in Japanese men. *Int J Obes Relat Metab Disord*, 27, 478-83.

- YUAN, T., ZHAO, W. G., SUN, Q., FU, Y., DONG, Y. Y., DONG, Y. X., YANG, G. H. & WANG, H. 2010. Association between four adipokines and insulin sensitivity in patients with obesity, type 1 or type 2 diabetes mellitus, and in the general Chinese population. *Chin Med J (Engl)*, 123, 2018-22.
- ZHU, Y., HSU, W. H. & HOLLIS, J. H. 2013. The effect of food form on satiety. *Int J Food Sci Nutr*, 64, 385-91.