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Impact of Behavioral Genetic Traits on Weight Maintenance Success Following Medically Supervised Very Low Calorie Diet

Lori Elizabeth Arguello
California State University, Northern California Consortium Doctor of Nursing Practice

ABSTRACT
IMPACT OF BEHAVIORAL GENETIC TRAITS ON WEIGHT MAINTENANCE SUCCESS FOLLOWING MEDICALLY SUPERVISED VERY LOW CALORIE DIET

Obesity has reached worldwide epidemic proportions and is associated with the leading causes of death. A person's predisposition to obesity is strongly related to genetics and specific genes have been identified that influence weight control. The aim of this quantitative retrospective chart review is to identify the impact of behavioral genetics on weight loss maintenance following a medically supervised very low calorie diet. A total of 330 patient charts that met inclusion criteria were reviewed. Six behavioral genetic results were reviewed which included snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth along with patient weight at 3, 6, and 12 month post weight loss program. Dropout rates at 6 month and 12 months were also reviewed. Results indicated no association between the genetic behavioral results of hunger, satiety, food desire and sweet tooth with weight maintenance, however findings did indicate a relationship between the snacking and eating disinhibition gene results with weight maintenance success at certain time points. Interestingly, results indicated that patients who were at increased risk for snacking had lower dropout rates from the maintenance program compared to those that tested typical snacking behavior. Based on prior research and the results of this current study the author recommends referral to medical weight loss programs for patients that struggle with weight loss as well as early genetic testing during the weight loss program so that high risk patients can be identified early. Current study findings suggest there is a place for genetic testing in bariatric medicine, however more research is needed in order to better understands the extent of those benefits and the exact role genetic testing will play.

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by
Lori Elizabeth Arguello

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We, the undersigned, certify that the project of the following student meets the required standards of scholarship, format, and style of the university and the student's graduate degree program for the awarding of the master's degree.

Lori Elizabeth Arguello
Project Author

Deepika Goyal, PhD, FNP-C (Chair)
Nursing

Sharon Ball, MD
The Hernried Center

Shelly Rodgers, MSN, FNP-C
The Hernried Center

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Page

TABLE OF CONTENTS

Page

Page

LIST OF TABLES

Page

CHAPTER 1: INTRODUCTION

Purpose and Background

Normal body weight is most often defined by body mass index (BMI) with normal BMI ranging between 18.5 and 24.9 kg/m2. Obesity defined as a BMI ≥30 kg/m2 and morbid obesity is defined as a BMI ≥40 kg/m2. Furthermore, obesity has recently been reclassified as class I obesity (BMI of 30-34.9 kg/m2), class II obesity (BMI of 34.9-39.9 kg/m2), and class III obesity (BMI ≥40 kg/m2) (Centers for Disease Control and Prevention, 2015; National Heart, Lung, and Blood Institute, n.d.). Obesity is a complex disorder involving excessive body fat amount of that affects people of all ages and ethnicities at any stage of their lives. In the United States (U.S.) obesity has reached epidemic proportions and is associated with some of the leading causes of death (American Heart Association, 2014). Currently 78.6 million U. S. adults are obese, representing 34.9% of the adult population with similar rates noted between men and women (Centers for Disease Control and Prevention, 2015). The health, psychosocial, and economic consequences of obesity have serious negative implications for the well-being and health of the U.S. population.

Research suggests that medically supervised very low-calorie diets (VLCD) are more effective for weight loss compared to the usual care of advice, education, and non-medically supervised methods (Anderson, Konz, Frederich, & Wood, 2001). However, given high rates of obesity in the U.S, patients continue to struggle with maintaining weight loss long term (Anderson, Konz, Frederich, & Wood, 2001; Jensen et al., 2014). Long-term weight loss maintenance success has been defined as an intentional loss of at least 10% of initial body weight and maintaining that weight for at least 1 year (Kraschnewski, et al., 2010; Wing &

Hill, 2001; Wing & Phelan, 2005). Bariatric medicine, a medical specialty that treats overweight or obese individuals by offering support and treatment for weight and weight-related problems (Obesity Action Coalition, 2016), has begun to incorporate genetic testing as an alternative way to examine barriers to long-term weight loss maintenance. Genetic testing, most often completed from a saliva or blood sample, is transforming healthcare and gaining popularity in many areas of medicine and bariatrics (American Medical Association, AMA, 2015; O'Rahilly & Farooqi, 2008). Although still in early stages, genetic testing findings and education may be useful tools for successful long-term weight loss maintenance.

A person's predisposition to obesity is strongly related to genetics (O'Rahilly & Farooqi, 2008). Furthermore, specific genes have been identified that influence eating behaviors, impact food perception, and impact how food cravings (Doehring, Kirchof, & Lotsch, 2009; Eny, Corey, & El-Sohemy, 2008; Epstein, et al. 2007). Health care providers and patients can incorporate this valuable information to further understand how genetic testing together with lifestyle modification and behavior change can promote long-term weight loss maintenance. Providing patient education regarding genetic predisposition for certain eating behaviors and traits versus learned behaviors would be of great value. Therefore, the overall aim of this project is to identify whether certain genetic behavioral traits impact long term weight loss maintenance success. The goal of this quantitative retrospective chart review is to identify the impact of behavioral genetics on weight loss maintenance following a medically supervised very low calorie diet. The hypothesis is that the more behavioral genetic markers identified via Pathway Fit DNA testing individuals have, the less successful they will be with weight loss maintenance. There will be examination of the effect of certain behavioral genes have on weight loss maintenance success based on body

weight regain at 3 month, 6 month, and 12 months as well as dropout rates from the maintenance program.

Background of Each Behavioral Genetic Markers

Snacking Background

Snacking can be a healthy or unhealthy behavior depending on how it is done, the individual, and what foods are consumed (Zizza, 2014). Snacking behavior is linked to genetic markers with variants in receptor for leptin, which is a necessary hormone for the regulation of food intake. Individuals with the G/G genotype in a leptin receptor genetic marker have been shown to exhibit increased snacking behavior compared to individuals with the typical genotype. The association of the leptin genetic marker and snacking behavior has not been tested in men. (de Krom, et al., 2007).

It is important to consider snacking behavior because people who have a strong preference for snack foods tend to gain weight compared to those that do not have a preference for snack foods (Nederkoorn, et al., 2010). Studies show that snacking has significantly increased over the decades across all age groups (Piernas & Popkin, 2010; Zizza, Siega-Riz, & Popkin, 2001).

Hunger Background

Most individuals know what hunger feels like but some individuals can experience hunger more often and more intensely. Predisposition to hunger can partially be explained by a variation in the Neuromedin B gene which is associated with increased feelings of hunger (Bouchard, et al., 2004). Feelings of increased hunger has been linked with weight gain and lower success rates with weight maintenance (Ludwig & Ebbeling, 2010; Pasman, Saris, Westerterp-Plantenga, & Biologie, 1999).

Satiety Background

Satiety describes the feeling of fullness after consumption of food. The Alpha-Ketoglutarate-Dependent Dioxygenase gene (FTO-rs9939609) is associated with difficulty feeling full. People who do not feel full after eating a meal tend to eat more which can lead to weight gain. A study by Wardle et al. (2008) revealed that the A/A genotype at the rs9939609 in the FTO gene was associated with difficulty feeling full in children. Although the participants in this study were children however, it does reveal preliminary data to support that the link between the Alpha-Ketoglutarate-Dependent Dioxygenase gene and fullness level.

Eating Disinhibition Background

Eating disinhibition is a tendency to overeat in the presence of appetizing foods or other disinhibiting triggers, such as emotional stress (Stunkard & Messick, 1988). Studies have shown that eating disinhibition is positively associated with weight (Hays, et al., 2002: Lindross, et al., 1997; Williamson et al., 1995). The Taste Receptor Type 2 Member 38 gene is associated with eating disinhibition in women, however there is currently not enough evidence to support the association in men (Dotson, Shaw, Mitchell, Munger, & Steinle, 2010). Eating disinhibition along with weight cycling, binge eating, increased hunger, and eating in response to negative emotions, is linked with weight regain (Elfhag, & Rössner, 2005).

Food Desire Background

The decision to eat, and to prefer particular foods, varies for different individuals and develops throughout life. Although it can be difficult to quantify feelings of hunger or preference for food a study by Epstein et al. (2007) found a method to test how much effort an individual is willing to exert to achieve access to food and with this method discovered a genetic component in food reinforcement. The findings of the study by Epstein, et al. (2007) revealed that the

T allele (increased) variant of the genetic marker Ankyrin Repeat and Kinase Domain Containing 1/Dopamine Receptor D2 genes were associated with greater food reinforcement compared to those with the C/C genotype (typical).

Sweet Tooth Background

Previous research has shown that people who have the C/T and T/T variants in the Solute Carrier Family 2 (Facilitated Glucose Transporter)/Member 2 gene (SLCA2A-rs5400) exhibit an increased likelihood to consume foods higher in sugar compared to those with the C/C genotype (Eny, et al., 2008). Studies have shown that decreasing consumption of certain foods, such as foods high in sugar, can be associated with successful weight loss maintenance (French, et al., 1994; Holden, et al., 1992; Mela, 2001).

Research Questions

1. What is the type and frequency of the following genetic markers (snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth) in a sample of patients who have participated in medical weight loss program? (please refer to table 1 for scientific genetic marker names).
2. Is there a relationship between the following genetic markers (snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth) and weight at 3, 6, and 12 months post treatment?
3. Which of the 6 genetic markers is more predictive of weight maintenance failure at 3, 6, and 12 months post treatment?
4. Which of the 6 genetic markers is more predictive of patient drop out at any time post treatment or at 3, 6, and 12 months?

Table 1. Scientific name of genetic marker

Common name	Scientific name
Snacking	Leptin Receptor gene
Hunger	Neuromedin B gene
Satiety	Alpha-Ketoglutarate-Dependent Dioxygenase gene
Eating disinhibition	Taste Receptor Type 2 Member 38 gene
Food desire	Ankyrin Repeat and Kinase Domain Containing 1/Dopamine Receptor
Sweet tooth	D2 genes 6.7%, Solute Carrier Family 2/Member 2 gene

Theoretical Framework

The self-determination theory (SDT) is a theory of motivation and personality that addresses issues of extrinsic and intrinsic motivation. Psychologists Edward Deci and Richard Ryan developed SDT in the mid 1980s. Extrinsic motivation, such as external rewards of money or prizes, can motivate people. SDT focuses more on intrinsic motivation, which are internal sources of

motivation such as a need to gain independence and knowledge. This theory of motivation suggests that people tend to be driven by a need to grow and gain self-actualization (Deci, & Ryan, 2008 & Ng., et al., 2012). According to the SDT, people need to feel competent, connection/readiness, and autonomous in order to achieve psychological growth. When people feel competent, independent, and ready, they become self-determined and able to be intrinsically motivated to pursue their goals. Ongoing social support is another important component of personal growth and well-being (Deci & Ryan, 2008). SDT suggests that offering people positive encouragement and feedback increases their intrinsic motivation (Deci, 1971). This theory has been applied to many fields of discipline, specifically sports, education, and healthcare (Ng, et al., 2012). The SDT can also be applied to obese adults.

The hypothesis is that the more behavioral genetic markers one has the less successful they will be with weight loss maintenance. Whether my hypothesis is correct or not, the SDT plays a significant role with patient weight loss maintenance success. A patient that tests negative for all behavioral traits will still need to contend with their learned behaviors and reversal of several years of bad habits. A patient that tests positive for one or several behavioral traits such as excessive snacking, sweet tooth, etc, will need to find ways to overcome their genetic predispositions. Whether the patient issue is genetically based or learned behavior, the SDT can be the key to success for weight loss maintenance.

The SDT emphasizes the importance of autonomy, social support and gaining knowledge in order for a person to fulfill their goals (Deci & Ryan, 2008). Providing obese patients with genetic testing and counseling translates into increased knowledge and increased feelings of social support by their medical provider, which results in increased independence and self-motivation. Ng., et al.,

(2012) conducted a meta-analysis, which examined the self-determination theory application to health contexts. The authors concluded that their findings supported the value of SDT as a conceptual framework to study motivational processes and to use as an aid to plan interventions for improved patient outcomes. Findings also indicated that promoting patient autonomy also promoted better mental and physical health. Providing patients with ongoing support, encouragement, and education will help ensure ongoing weight loss maintenance success. Regardless of what the upcoming DNP project retrospective data review reveals, the SDT can provide a valuable framework for assisting patients maintain their healthier weight long-term.

CHAPTER 2: LITERATURE REVIEW

Although there is an abundance of literature on obesity and weight loss, there is limited literature on weight loss maintenance as well as obesity related behavioral genetics. This section will discuss and summarize the beginning collection of relevant research pertaining to the upcoming Doctoral of Nursing Practice (DNP) project.

Conradt et al. (2009) conducted a survey research study that examined the effects of a medical consultation with genetic information on obese adults attitudes, goals, coping, body shame, and self-blame. Participants were randomly assigned to two standardized consultations, with and without genetic information about obesity, and a control group without any intervention. After a 6-month follow-up, 253 obese individuals of the intervention groups and 98 individuals of the control group had a complete dataset. Attitudes about weight loss goals, weight-related self-blame, coping, and body shame were assessed by questionnaire or interview. A medical staff assessed body weight and height at baseline. Attitudes about losing weight and satisfaction with weight loss were assessed with a set of interview questions. Self-blame concerning eating was assessed by the Shame and Guilt concerning Eating Scale. Coping behavior was assessed with the Coping Strategies Inventory–Short Form (CSI-S). Body shame associated with obesity was assessed by the shame subscale of the Weight- and Body-Related Shame and Guilt Scale. Results revealed that regardless of family predisposition, consultation focusing on genetic factors was potentially helpful for obese individuals. Only predisposed participants showed a decrease in self-blame about eating. Negative thoughts and feelings about current weight were identified as being able to predict future weight gain.

Eny, et al. (2008) used a repeated measures study using data collected from two populations. Data was collected for the first population from baseline data that came from the Canadian trial of Carbohydrates in Diabetes multicenter intervention study (n=100). Data was collected for the second population from the Toronto Nutrigenomics and Health Study, which was a cross-sectional examination of men and women between 20 and 29 years of age (n=720). The authors investigated whether the Thr110Ile polymorphism is associated with differences in the consumption of sugar. Measurements were repeated within a population and between two distinct populations using two different methods of dietary assessment. Population one consisted men and women between the ages of 42–75 years that were participants of the Canadian trial of Carbohydrates in Diabetes multicenter intervention study. Subjects included 127 men (n=60) and women (n=67) who had early Type 2 diabetes and did not require medications. Population two consisted of participants of the Toronto Nutrigenomics and Health Study, which was a cross-sectional examination of young men and women between 20 and 29 years of age recruited from the University of Toronto campus. Subjects included 720 men (*n* 224) and women (*n* 496). To access intake of food and beverage for the population a one-time 3-day food record (two sets) was used. For population two each participant completed a 196-item self-administered food frequency questionnaire (FFQ) to assess habitual food intake over the past month. Measurements included height, weight, and waist circumference and body mass index. Each participant had blood drawn after a 12-hour overnight fast to measure glucose and insulin. Deoxyribonucleic acid (DNA) was isolated from whole blood using the GenomicPrep Blood DNA Isolation kit. The Thr110Ile polymorphism was detected by using a TaqMan allelic discrimination assay. The results demonstrated that a genetic polymorphism of GLUT2 is associated with

differences in consumption of sugars both within and between two distinct populations, using two types of dietary assessment tools.

Konttinen et al. (2015) conducted a population-based cross-sectional study in which data was collected from two independent population-based Finnish cohorts (4632 adults aged 25–74 years and 1231 twin individuals aged 21–26 years). Genotyping of the DILGOM cohort was done at the Wellcome Trust Sanger Institute and the FIMM Technology Centre. The genetic predisposition to obesity was assessed by calculating a PRS using 90 of 97 BMI-associated loci. Participants' weight, height, and waist circumference were measured using standardized international protocols in both cohorts. Uncontrolled and emotional eating was assessed with the Three-Factor Eating Questionnaire in both cohorts. The results indicated that genetic predisposition to obesity may act partly through appetitive traits reflecting lack of control over eating or eating in response to negative emotions. The results were somewhat more consistent in the cohort of 25–74 year-old Finnish adults than in the 21–26 year-old Finnish twins. One of the strengths of the study was the use of two independent population-based cohorts with identical measurements on appetitive phenotypes and anthropometric traits. A limitation of the study was that it was a cross-sectional design and results need to be confirmed using a longitudinal approach.

Krom et al. (2007) examined the association between genetic variations in cck, leptin, and leptin receptor genes with specific human eating patterns. The sample was drawn from the Prospect-European Prospective Study into Cancer and Nutrition (EPIC) study, which consisted of 17,357 females aged 49–70 years between 1993 and 1997 living in Utrecht or the Netherlands. After exclusion, there were a total of 135 cases. There were 287 control subjects who were randomly selected from the total cohort. Detailed data on dietary habits, blood samples,

BMI, and eating habits and physical activity (both based on a validated questionnaire) were collected for all women. Using allele-specific polymerase chain reactions (PCRs), the authors tested several single nucleotide polymorphisms in the candidate genes and performed haplotype analysis. The participants were classified according to extreme snack behavior (*n* 60) and meal size (*n* 72). The genotype and allelic distributions were compared between the two selection groups and the random control sample (*n* 287). Four of the five tested CCK SNPs showed a specific association signal with extreme meal size but not with extreme snack behavior. One of eight SNPs of the leptin receptor and two of four SNPs of leptin were associated with extreme snack behavior but not with meal size. Obese carriers of common allelic variations in leptin or the leptin receptor gene had an increased risk of exhibiting extreme snacking behavior. Obese carriers of common allelic variations in CCK had an increased risk to eating larger portion sizes.

Savage et al. (2009) examined the effect of dieting, restraint, and disinhibition predicted weight change among 163 Non-Hispanic White women. Data, including subjective questionnaires and objective height and weight measurements, were collected 4 times across a 6-year period. Dietary restraint and disinhibition was assessed with the Eating Inventory questionnaire, which consists of 51 true-false items designed to tap 3 subscales of dietary restraint, dietary disinhibition, and susceptibility to hunger (Stunkard & Messick, 1985). For the purpose of this study, only restraint and disinhibition subscales were used. Results indicated increased levels of dietary restraint might be helpful in moderating weight by lessening the positive association between disinhibition and weight in dieting women. Effects of restraint, disinhibition, and dieting all must be examined in order to understand weight and weight change.

Summary of Literature Review Findings

Genetic counseling for obesity management is becoming more popular and patients that receive genetic counseling appear to have a decrease in self-blame about eating, which has been noted as an important component to increasing self-esteem and motivation (Conradt et al., 2009). Not only can obesity itself be linked to certain genomic allele but behaviors that lead to obesity such as excessive consumption of sugar, overeating, and eating in response to negative emotions can also be linked to certain genes (Eny et al., 2008; Konttinen et al., 2015; Krom, 2007). People who exhibit increased levels of restraint with their eating habits tend to be able to moderate their weight better than people who show less restraint (Savage, Hoffman, & Birch, 2009).

Although there is research on behavioral genes being associated with certain unhealthy eating behaviors that can lead to obesity, no research to date has examined how specific genes affect a weight loss maintenance success. Further research is needed on comparing the effects of different behavioral traits on weight maintenance success. There is also a gap in the literature in regards to the comparing patient outcomes with weight maintenance success in terms of whether their unhealthy eating behaviors are genetic or a learned behavior.

CHAPTER 3: METHODS

Design

A quantitative retrospective design was used for this electronic medical chart review.

Setting

The setting for this study included three of the Hernried Medical Weight Loss Centers in the Sacramento area.

Subjects

All electronic medical records (EMR) of patients meeting the following criteria was included in this study: Adults over 18 years of age, successful completion of the medically supervised very low calorie diet (VLCD) with a least a 10% drop in initial body weight, Pathway DNA Fit test results, and at least 3-month attendance in post weight loss maintenance program which consists of weekly group meetings and body weight assessment. Only EMR of patients who have a signed consent form for the Pathway DNA Fit testing, which provides consent for their results to be used for research purposes, was included. No data was collected until IRB approval was obtained. EMR of patients was not excluded based on gender or race. A de-identified dataset was created using patient EMR and the Pathway Genomics Portal.

Data Collection Procedure

The study examined the impact of behavioral genetics on weight loss maintenance success, which required a retroactive data review of patient body weights and behavioral genetic results. Since 2012 the weight loss clinic has offered patients Pathway Fit DNA testing once they are in the maintenance program. Completion of the genetic testing is completely voluntary by the patients and they do sign a consent form that indicates whether they opt in or opt out for

the use of their sample for research purposes. The Pathway Fit DNA testing examines specific genetic markers. The test is obtained through saliva or blood and evaluates over 75 genetic markers to provide the patient with individualized information regarding diet, exercise, addictive behaviors, and weight-related health conditions. Patient demographic data was collected via electronic medical records (See table 2). Subject genetic data, which was already available, was be collected from all maintenance patients from October 2012 until July 2015 and this data was extracted via Pathway Genomics Portal Review. Since this is a retrospective data review no interventions were performed.

Six components of genetic data (See table 2) that was included were eating DNA behavior traits such as snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth. The following genes that are used by the Pathway Fit DNA are the Leptin Receptor gene (LEPR-rs2025804) for snacking, the Neuromedin B gene (NMB-rs1051168) for hunger, the Alpha-Ketoglutarate-Dependent Dioxygenase gene (FTO-rs9939609) for satiety, the Taste Receptor Type 2 Member 38 gene (TAS2R38-rs1726866) for eating disinhibition, the Ankyrin Repeat and Kinase Domain Containing 1/Dopamine Receptor D2 genes (ANKK1/DRD2-rs1800497) for food desire, and the Solute Carrier Family 2 (Facilitated Glucose Transporter), Member 2 gene (SLCA2A-rs5400) for sweet tooth (The Hernried Center, 2011). Along with the genetic data patient information such as dropout rates and weight regain at three month, six month, and twelve months was also obtained.

Certain physical and mental comorbidities are associated with obesity and can have an impact on weight loss and weight loss maintenance success, therefore, additional data from the electronic medical record was collected which include: Hemoglobin A1C results, Epworth Sleepiness Scale Results, Depression, Anxiety,

and Stress Scales (DASS) questionnaire results, and Questionnaire of Eating and Weight Patterns (QWEP) results. History of type II diabetes, hypertension, hyperlipidemia, and sleep apnea was also obtained.

Table 2. Data Extraction Plan

Characteristic	Specific Data that was Extracted
Demographic Data	Age, gender, marital status, occupation, and race-ethnicity.
Other Participant Characteristics	1. Wellbeing (Depression, Anxiety, and Stress Scale, Lovibond & Lovibond, 1995) 2. Daytime Sleepiness (Epworth Sleepiness Scale, Johns, 1991) 3. Eating disorders (Questionnaire of Eating and Weight Patterns, Spitzer, et al., 1991) 4. Comorbid conditions (Diabetes Mellitus, HbA1C, hypertension, hyperlipidemia, sleep apnea, polycystic ovarian syndrome)
Genetic Markers	1. Snacking - Leptin Receptor gene (LEPR-rs2025804)

	2. Hunger - Neuromedin B gene (NMB-rs1051168) 3. Satiety Alpha-Ketoglutarate-Dependent Dioxygenase gene (FTO-rs9939609) 4. Eating disinhibition Taste Receptor Type 2 Member 38 gene (TAS2R38-rs1726866) 5. Food desire Ankyrin Repeat and Kinase Domain Containing 1/Dopamine Receptor D2 genes (ANKK1/DRD2-rs1800497) 6. Sweet tooth- Solute Carrier Family 2 (Facilitated Glucose Transporter)/Member 2 gene (SLCA2A-rs5400) Solute Carrier Family 2 (Facilitated Glucose Transporter), Member 2 gene (SLCA2A-rs5400)
Weight (Lbs)	Baseline, 3, 6, and 12 months post treatment

Drop Out Rate	Assessed from patient attendance in the weight maintenance program at 6 and 12 months.

CHAPTER 4: RESULTS

Descriptive Results for Total N

A total of 768 charts were reviewed. Of the charts reviewed, 43% (n=330) of the weight loss patients met the inclusion criteria. Many charts were excluded due to reasons such as age or not losing at least 10% of their initial body weight (please refer to table 3). Participant characteristics of age, gender, race/ethnicity, marital status and payee type were analyzed using descriptive statistics using SPSS software version 23.

The majority of the sample were female, self-identified as Caucasian, with a mean age of 54.6 years with a standard deviation of 12, married, and had Sutter Select Health HMO care coverage (please refer to table 4).

Research Question Results

1. What is the type and frequency of the following genetic markers (snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth) in a sample of patients who have participated in medical weight loss program?

There were 5 possible results for the genetic marker testing: less likely, typical, increased, more likely, difficulty feeling full. The frequencies for each category were tabulated, and the percentage of patients falling into each category were calculated.

All 330 patient's results were accounted for snacking, satiety, eating disinhibition, and sweet tooth genes. Hunger and Food desire gene results each had one missing patient with a total of 329. For the snacking gene 288 patients tested "typical" and 42 tested "increased." For the hunger gene 316 patients tested "typical" and 53 tested "increased." For satiety gene 277 patients tested "typical" and 53 tested "difficulty feeling full." For the eating disinhibition gene 76 patients tested "less likely" and 254 tested "more likely." For the food desire gene 195 patients tested "typical" and 134 tested "increased." For the sweet tooth gene 258 patients tested "typical" and 72 tested "increased" (please refer to table 5).

2. Is there a relationship between the following genetic markers (snacking, hunger, satiety, eating disinhibition, food desire, and sweet tooth) and weight at 3, 6, and 12 months post treatment?

For each genetic marker, the mean weight at 3, 6, and 12 months post treatment for the 5 gene result groups were compared using mixed effects analysis of variance (ANOVA). In this analysis, group was a between-subjects independent variable (IV) and time was a repeated measures IV. Weight was the dependent variable (DV). The analysis tests whether weight differed between the 5 groups (main effect of group), whether weight changed over time (main effect of time), and whether weight followed a different trend over time for the two groups (interaction between group and time). A separate ANOVA was performed for each genetic marker, resulting in 6 ANOVAs.

There was a change in weight over time across all groups. The change in weight overtime was the same regardless of the genetic result. The Tukey Post Hoc test revealed an increase in weight over all three time points for all subjects. There was no significant difference in weight at any time point for any of the genetic results. There was no interaction between time and the genetic results for any of the genetic results (please refer to table 6).

3. Which of the 6 genetic marker results (5 result possibilities) is more predictive of weight maintenance failure at 3, 6, and 12 months post treatment?

The criteria for weight maintenance success was determined by the current literature, and patients were categorized into either weight maintenance failure or success. Chi-square test of independence was used to determine whether weight maintenance failure is related to genetic result, for each gene. The two categorical variables were genetic result (5 possibilities), and weight maintenance outcome (success vs. failure). A separate analysis was done for each gene and for each time point (3 months, 6 months, and 12 months).

For the snacking gene at 3-month time point, 87.9% (n=282) classified as successful with weight maintenance and 12.1% (n=5) classified as failure in the patients that tested "typical." For the patients that tested "increased" at the 3-month time point, 92.7% (n=39) classified as successful with weight maintenance and 7.1% (n=3) classified as "failure." At the 3-month time point the patients that tested "typical" for the snacking gene were significantly more successful with weight maintenance compared to the patients that tested "increased" for the snacking gene (p=0.034). For the 6-month and 12-month time points no statistically significant relationships were noted.

For the hunger gene at the 3, 6, and 12-month time points no statistically significant relationship between gene result and weight maintenance outcome were noted.

For the satiety gene at the 3, 6, and 12-month time points no statistically significant relationships between gene result and weight maintenance outcome were noted.

For the eating disinhibition gene at 6-month time point there were 100% classified as successful with weight maintenance and 0% classified as failure in the patients that tested "less likely." For the patients that tested "more likely" at the 6-month time point there were 92.7% classified as successful with weight maintenance and 7.3% classified as "failure." At the 6-month time point the patients that tested "less likely" for the eating disinhibition gene were more successful with weight maintenance compared to the patients that tested "more likely" for the eating disinhibition gene. This was statistically significant at 0.05. For the 3-month and 12-month time points there were no statistically significant relationships.

For the food desire gene at the 3, 6, and 12-month time points no statistically significant relationships between gene result and weight maintenance outcome were noted.

For the sweet tooth gene at the 3, 6, and 12-month time points no statistically significant relationships between gene result and weight maintenance outcome were noted (please refer to table 7).

4. Which of the 6 genetic marker results (5 possible results) is more predictive of patient drop out at any time post treatment or at 3, 6, and 12 months?

Chi-square test of independence was used to determine whether drop out is related to genetic result. The two categorical variables were genetic result and drop out (dropped out vs. did not drop out). A separate analysis was done for each gene and for each time point (6 months and 12 months).

The snacking gene labeled as "increased" was associated with lower dropout rates at the 6-month and 12-month time point compared to those labels as "typical" for the snacking gene. This was statistically significant with a p value of .008 at 6-month and 0.025 at 12-months. All the other genetic results including hunger, satiety, eating disinhibition, food desire, and sweet tooth were not related to drop out rate at either time point (please refer to table 9).

Table 3. Description of all charts reviewed and reasons for exclusion (N= 768)

Description	%, n
Met inclusion criteria	43%, n=330
Did not attend maintenance program for at least 3 months	33.1%, n=245
Quick 20 patient	11.6%, n=89
Never started weight loss program	7.5%, n=59
Completed weight loss program within the last 3 months of data collection or a current weight loss patient	2.9%, n=23
No Pathway Genetic results located or unable to locate patient in electronic medical record	1.7%, n=13
Did not lose at least 10% of initial body weight	0.1%, n=8
Under the age of 18	0.1%, n=1

Table 4. Sample demographics of weight loss patients (n = 330)

Characteristic	%, n
Age Range (years)	26 ± 85
Age Mean (sd)	54.6 (12)
Gender	
Female	79.4%, n=262
Male	20.6%, n=68
Race or ethnicity	
Caucasian	87.6%, n=289
Hispanic or Latino	6.7%, n=22
Asian	3.9%, n=13
African American	1.8%, n=6
Marital Status	
Married	70.3%, n=232
Single	14.5%, n=48
Divorced	8.2%, n=27
Other*	7.0%, n=14
Payee Type	
Sutter Select HMO**	42.1%, n=139
PPO***	32.1%, n=106
Medicare	13.6%, n=45
Private Pay	11.8%, n=39
CHAMPVA****	0.3%, n=1

*Other includes: Separated, Widowed, Domestic Partner
**HMO=Health Maintenance Organization
***PPO=Preferred Provider Organization
****CHAMPVA=The Civilian Health and Medical Program of the Department of Veterans Affair

Table 5. Frequencies of genes and results (n=330)

Gene	Frequency/%
Snacking	
Typical	288 (87.3)
Increased	42 (12.7)
Hunger*	
Typical	316 (95.8)
Increased	13 (3.9)
Satiety	
Typical	277 (83.9)
Difficulty Feeling Full	53 (16.1)
Eating Disinhibition	
Less Likely	76 (23)
More Likely	254 (77)
Food Desire*	
Typical	195 (59.1)
Increased	134 (40.6)
Sweet Tooth	
Typical	258 (78.2)
Increased	72 (21.8)

*n=329

Table 6. Genetic markers and weights at various time points

Gene	Weight (*SD*) 3 months	Weight (*SD*) 6 months	Weight (*SD*) 12 months
Snacking			
Typical	165.3(28.0)	168.6 (29.0)	176.2(30.2)
Increased	154.3(24.0)	159.8(25.2)	164.9(25.0)
Hunger			
Typical	163.1 (28.0)	166.7(28.3)	174.1(30.0)
Increased	174.3(19.2)	180.0(20.4)	182.3(23.7)
Satiety			
Typical	165.3(29.0)	169.4(29.2)	176.9(29.8)
Difficulty Feeling	155.8(19.4)	158.0(21.4)	163.7(26.6)
Full			
Food Desire			
Typical	164.3(26.3)	167.0(26.0)	173.6(26.9)
Increased	162.5(30.1)	167.9(32.1)	176.0(33.9)
Sweet Tooth			
Typical	162.3(26.0)	165.7(25.3)	172.3(27.3)
Increased	167.1(32.2)	171.5(35.4)	180.6(35.5)
Eating Disinhibition			

Less Likely	164.9(21.5)	170.1(24.5)	177.2(29.6)
More Likely	163.1(29.3)	166.3(29.2)	173.5(29.6)

Table 7. Genetic marker results and time points

Gene	3M Success/Failure (n) %	6M Success/Failure (n) %	12M Success/Failure (n) %	P Value 3M/6M/12M
Snacking				
Typical	(282/5) 98.3%/1.7%	(172/10) 94.5%/5.5%	(77/5) 93.9%/6.1%	**0.034/0.896/0.981
Increased	(39/3) 92.7%/7.1%	(31/2) 93.9%/6.1%	(15/1) 93.8%/6.3%	
Hunger				
Typical	(309/6) 98.1%/1.9%	(194/10) 95.1%/4.9%	(88/6) 93.6%/6.4%	0.157/0.476/0.602
Increased	(12/1) 92.3%/7.7%	(9/1) 90%/10%	(4/0) 100%/0%	
Satiety				
Typical	(270/6) 97.8%/2.2%	(171/8) 95.5%/4.5%	(73/6) 92.4%/7.6%	0.489/0.113/0.215
Difficulty Feeling Full	(51/2) 96.2%/3.2%	(32/4) 88.9%/11.1%	(19/0) 100/0%	
Food Desire				
Typical	(191/3) 98.5%/1.5%	123/5 (96.1%/3.9%)	(56/4) 93.3%/6.7%	0.207/0.187/0.802
Increased	(129/5) 96.3%/3.7%	79/7 (91.9%/8.1%)	(35/2) 94.6%/5.4%	
Sweet Tooth				
Typical	(252/5)98.1%/1/9%	(155/8) 95.1%/4.9%)	(68/5) 93.2%/6.8%	0.279/0.446/0.608
Increased	(69/3) 95.8%/4.2%	(48/3) 92.3%/7.7%	(24/1) 96%/4%	
Eating Disinhibition				
Less Likely	(71/4) 98.7%/1.3%	(50/0) 100%/0%	(23/1) 95.8%/4.2%	0.482/**0.05/0.646
More Likely	(247/7) 97.2%/2.8%	(153/12) 92.7%/7.3%	(69/5) 93.2%/6.8%	

*M=month
**P value significant at less than 0.05

Table 8. Drop Out Results and Time Points

Gene	6M Drop Out No/Yes (n)%	12M Drop Out No/Yes (n)%	Unable to determine due to recent date (n)%	P Value 6M/12M
Snacking				
Typical	(176/75) 70.1%/29.9%	(85/158) 35%/65%	(0)0%	**0.008/**0.025
Increased	(31/6) 81.6%/15.8%	(16/21)41.2%/55.3%	(1)2.6%	
Hunger				
Typical	(196/80) 70.8%/28.9%	(96/172) 35.7%/63.9%	(1)0.4%	0.348/0.792
Increased	(10/1) 90.9%/9.1%	(5/6) 45.5%/54.5%	(0)0%	
Satiety				
Typical	(172/66) 72%/27.6%	(82/149) 35.3%/64.2%	(1)0.4%	0.854/0.818
Difficulty Feeling Full	(35/15) 70%/30%	(19/30) 38.8%/61.2%	(0)0%	
Food Desire	(124/48) 71.7%/27.7%	(59/107) 35.3%/64.1%	(1)0.6%	0.709/0.706
Typical	(82/33) 71.3%/28.7%	(41/72) 36.3%/63.7%	(0)0%	
Increased				
Sweet Tooth				
Typical	(156/67)69.6%/29.9%	(75/142) 34.4%/65.1%	(1) 0.4%	0.350/0.537
Increased	(51/14) 78.5%/21.5%	(26/37) 41.3%/58.7%	(0)0%	
Eating Disinhibition				
Less Likely	(45/20) 69.2%/30.8%	(24/40) 37.5%/62.5%	(0)0%	0.747/0.831
More Likely	(162/61) 72.3%/27.2%	(77/139) 35.5%/64.1%	(1)0.4%	

*M=month
**P value significant at less than 0.0

CHAPTER 5: CONCLUSION

Discussion

This study is the first to present findings regarding how specific genes affect weight loss maintenance success. The following sections will discuss study results regarding each gene type.

Snacking

Our findings indicate patients testing as "typical snacking behavior" were more successful with weight maintenance up to the first 3 month post medically supervised VLCD appointment. Previous research indicates that people who prefer snack foods tend to gain weight compared to those that do not have a preference for snack foods (Nederkoorn, et al., 2010) so it is not surprising that patients who tested for an increased tendency for snacking to regain more weight compared to those who tested typical. This finding was only significant at the 3-month time point which may indicate that patients may revert back to old habits of excessive snacking behavior shortly after reaching weight loss goal. One could conclude that patients who have the genetic predisposition for increased snacking behavior return back to snacking behavior sooner (within the first 3-month post weight loss) compared to those that test typical. The number of patients in the maintenance program decreased at each time point so perhaps if there was not a decline in the sample size at months 6 (n=215) and 12 (n=98) there may have continued to be a statistically significant relationship between patients who tested typical for the snacking gene and weight maintenance success.

One unexpected finding of this study was the association between the increased snacking gene result and lower dropout rates at the 6-month and 12-month time point. A possible reason for this finding is that these patients were

aware of their own unhealthy snacking behavior so they felt more motivated to attend the maintenance program for a longer period of time. All the other genetic results including hunger, satiety, eating disinhibition, food desire, and sweet tooth were not related to drop out rate at either time point. There is no prior research on the association between the snacking gene and weight maintenance success or weight maintenance program drop out.

Hunger

There was no relationship between patients who tested positive for increased feelings of hunger and weight maintenance success or dropout rates. A possible reason for these results could be because patients must contend with the feelings of hunger once they are no longer following a very low calorie ketogenic diet which could result in weight regain and higher maintenance program dropout rates. Ketogenic diets are known to reduce appetite and also improve affect (Boden, Sargrad, Homko, Mozzoli, & Stein, 2005; Leibel, Rosenbaum, & Hirsch, 1995; Nickols-Richardson, Coleman, Volpe, & Hosig, 2005) so once patients complete a very low calorie ketogenic diet program they are at risk for feelings of hunger as well as a negative change in affect. This would be true whether a patient has a genetic predisposition to increased hunger or a patient with a typical level of susceptibility to hunger. There is no prior research on the association between the hunger gene and weight maintenance success or weight maintenance program drop out.

Satiety

In this present study, there was no statistically significant relationships between the satiety gene result and weight maintenance outcome or dropout rate. It is well established that protein is more satiating than the ingestion of carbohydrate or fat and that even a modest increase in protein, along with the

reduction of the other macronutrients, can promote satiety and facilitate weight loss through reduced energy consumption (Astrup, 2005; Westerterp-Plantenga, Rolland, Wilson, Westerterp, & Biologie, 1999; Westman, Yancy, Edman, Tomlin, & Perkins, 2002; Yancy, Olsen, Guyton, Bakst, & Westman, 2004). One potential reason for these results could be that once patients complete the very low calorie diet, which is high in protein and low in other macronutrients, they begin to eat foods that are less satiating. This can result in increased hunger and weight regain. Based on the current study the genetic results for satiety did not show an association with weight maintenance results. However, our findings are important given the lack of research regarding association between the satiety gene and weight maintenance success or weight maintenance program drop out.

Eating Disinhibition

Out of all the behavioral genetic traits the eating disinhibition gene was the most prevalent obesity related result with 254 out of 330 patients testing more likely to exhibit eating disinhibition. In this present study at the 6-month time point the patients that tested "less likely" for the eating disinhibition gene were more successful with weight maintenance compared to the patients that tested "more likely" for the snacking gene, however for the 3-month and 12-month time points there were no statistically significant relationships. Ongoing behavioral support and self-monitoring is linked with greater success with weight loss and weight maintenance (Elfhag, & Rössner, 2005; Wing & Hill, 2001). The patients that complete the weight maintenance program are required to come in for group classes weekly, weigh in at least once a month, and they are also required to self-monitor their dietary intake.

There seems to be a relationship between patients who test less likely for eating disinhibition and successful weight maintenance. One hypothesis for the

lack of statistical significance at the 3-month time point is that the novelty of achieving major weight loss is highly motivating to most patients (despite their genetic propensity for disinhibited eating) which could translate into more restrained eating patterns during the immediate 90 days post weight loss program. The number of patients in the maintenance program decreased at each time point so perhaps if there was not a decline in the sample size at 12 months there would have continued to be a statistically significant relationship between patients who tested less likely for the eating disinhibition gene and weight maintenance success. There is no prior research on the association between the eating disinhibition gene and weight maintenance success or weight maintenance program drop out.

Food Desire

Based on the results of the current study there does not appear to be any relationship between gene result and weight maintenance outcome. One could possibly conclude from this is that the effect of either learned behavior of increased food desire or a genetic predisposition to increased food desire have the same weight maintenance outcome. Another potential reason for these results is that overweight and obese individuals tend to prefer and select energy-dense foods, which can contribute to weight gain and failure to maintain weight loss (Mela, 2001). Once these patients complete the diet program they could be at risk for returning back to prior habits of eating higher energy dense foods which can lead to weight regain. There is no prior research on the association between the food desire gene and weight maintenance success or weight maintenance program drop out.

Sweet Tooth

In this current study there was no relationship between the sweet tooth gene and weight maintenance outcome. One hypothesis that could explain these

findings is that perhaps foods high in sugar are not the main culprit for weight gain or weight regain. Several research studies have linked foods high in fat with weight gain. Prior research has shown that consuming foods high in fat such as french fries, red meats, and dairy products has a positive association with weight loss maintenance (French, et al., 1994; Holden, et al., 1992). One could possibly conclude from this is that the effect of either learned behavior of increased desire for foods high in sugar or a genetic predisposition to this preference have the same effect of weight maintenance outcome. There is no prior research on the association between the sweet tooth gene and weight maintenance success or weight maintenance program drop out.

Limitations

Although this study addressed all of the original research questions, results should be interpreted with caution given several limitations. The following sections will review limitations involving the subject characteristics, data collection, outside factors, patient dropout, and prior research of behavioral genetics.

Participants

The sample was primarily made up of self-selected. middle aged, married, Caucasian females, with health coverage limiting generalization of study findings to other patient populations. Future research should include single males or people from diverse race/ethnic and socioeconomic groups. Although this sample may not reflect the US population at large, our sample is in agreement to previous research indicating that Caucasian females are more likely to diet and be concerned with weight compared to males or other races/ethnicities (Davy, Benes, & Driskell, 2006; George & Johnson, 2001; Page & Fox, 1998).

Another limitation includes the use of self-report weight as well as measured weight during clinic visits. The data in this study was collected between October 2012 - July 2015. Patients were self-reporting their weight until 2014 with patients only more recently being weighed by a medical assistant in the clinic setting. Previous research indicates female patients often underreport weights obtained at home impacting the validity of the results (Hsiao, et al., 2014; Nawaz, Chan, Abdulrahman, Larson, & Katz, 2001). However self-reported weight is still considered a simple, cost effective method for tracking weight especially when using an algorithm adjusted for variables that are predictive for misreporting (Nyholm, 2007).

Although the original sample size was large (n=330) there was patient drop out at each time point during the maintenance program which yields a lower power and not be an accurate representation of results. Even though there was patient drop out at each time point the sample size only got as low as 98 patients which is still a reasonable amount. Studies have shown that if a patient is able to maintain their weight loss for 2-5 years, the chances of longer-term success greatly increase (Wing & Hill, 2001). There is a great need for further research on methods to keep patients in weight maintenance program.

Behavioral genetics

The Pathway DNA Fit test only tests for DNA markers and does not test on any epigenetics. This is considered a limitation because epigenetics alter gene activity without changing the DNA sequence and can significantly impact eating behaviors (Weinhold, 2006). Based on the data available to me I will be unable to determine whether an eating behavior is due to epigenetics or a learned behavior.

The prior research on the behavioral genes is lacking and have limitations. The research on the snacking gene and eating disinhibition gene has never been studied in men (den Krom, et al., 2007; Dotson, et al. 2010). The study regarding the hunger gene being only based on a questionnaire (Bouchard, et al, 2004). The research that has linked the Alpha-Ketoglutarate-Dependent Dioxygenase gene (FTO-rs9939609) to satiety was only studied in children (Wardle, 2008). Pathways Genomics use these genetic markers based on limited studies to determine if patients have certain behavioral genetic results that can lead to unhealthy eating behaviors, however the limitations of prior studies are clearly discussed in the result packets that patients receive. There is lack of prior research on this topic, however, when breaking new ground, there will be many gaps in the knowledge base that need to be addressed in future research. This current study should serve as a beginning foundation for research involving behavioral genetics and weight maintenance.

Clinical Implications

Despite growing recognition of the issue, the obesity epidemic continues in the United States, and obesity rates are increasing around the world (American Heart Association, 2014). Assisting patients with weight-loss maintenance remains a formidable challenge for health care professionals and it is imperative for providers to emphasize the favorable health effects that result from losing and maintaining at least a moderate 10% weight loss (Klein, et al., 2004). Medically supervised very low calorie diets combined with behavioral counseling is a very effective approach to weight loss compared to traditional weight loss approaches (Anderson, Konz, Frederich, & Wood, 2001). Medically supervised very low calorie diets have been linked to greater long-term weight maintenance success

compared to hypoenergetic balanced diets (Anderson, Konz, Frederich, & Wood, 2001). The use of medically supervised ketogenic diets seems to be a promising weight loss solution for many patients. Although most patients are able to lose a significant amount of weight with medically supervised diet programs, many of them struggle with keeping the weight off long-term (Anderson, Konz, Frederich, & Wood, 2001; Jensen et al., 2014). Research has indicated that the key to weight loss maintenance success is a multidisciplinary approach with both medical and behavioral supervision (Perri, 1998; Perri & Others, 1989). The author recommends that medical providers should encourage obese patients that struggle with weight loss to enroll in medically supervised diet program as well as a follow up weight maintenance program.

The overall aim of this study was to identify whether certain genetic behavioral traits impact long term weight loss maintenance success. Based on the results of this study there does not seem to be an association with the genetic behavioral results of hunger, satiety, food desire and sweet tooth with weight maintenance. There was an increase in weight over all three time points for all subjects regardless of the genetic result. Even though research indicates patients are more successful at maintaining weight loss in a weight maintenance program, this current study shows that even patients in a weight maintenance program are also at risk for weight regain. Health care providers should continue to encourage patient to complete weight maintenance programs, despite what method the patient used to lose the weight, to help ensure continued support, accountability, and better chances of keeping the weight off. Based on the results of this study one could conclude that all patients are at risk for weight regain, despite the method utilized for weight loss, participation in a weight management program, and behavioral genetic test results. It is important for health care providers to warn

patients about the risk of weight regain, especially within the first 12 months post weight loss. Patients must also be given hope and understand that weight maintenance can get easier overtime and that if they are able to maintain their weight loss for 2-5 years, the chances of long-term success increases (Wing & Hill, 2001). Based on the prior evidence on weight maintenance the author recommends medical providers strongly encourage weight loss patients to complete at least 12 to 18 months of a weight maintenance program to help increase the chances of long-term success.

There were some findings that suggest a relationship between the genetic behavioral results of increased snacking and more likely for eating disinhibition and weight maintenance failure. Based on these findings health care providers should consider early additional interventions for these high-risk patients such as pharmacotherapy or additional behavioral counseling.

Genetic testing is becoming a more common practice in medicine and has also proven to be beneficial in bariatric medicine (American Medical Association, AMA, 2015; O'Rahilly & Farooqi, 2008). Obesity and the behaviors that can lead to obesity are linked to specific genes and health care providers should be utilizing this valuable information to better patient care and weight loss management (Doehring, Kirchof, & Lotsch, 2009; Eny, Corey, & El-Sohemy, 2008; Epstein, et al. 2007; O'Rahilly & Farooqi, 2008). The author recommends that genetic testing be offered early in weight loss programs since the results can take several weeks to receive and so there is a better chance for early intervention for high risk patients. Genetic testing can provide opportunities for patient education and counseling which could improve patient weight management outcomes. Current study findings suggest there is a place for genetic testing in bariatric medicine,

specifically in weight maintenance, however more research is needed in order to better understand the extent of those benefits and the exact role genetic testing will play in weight loss and weight maintenance management.

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