University of Rhode Island DigitalCommons@URI

**Open Access Master's Theses** 

2005

# Weight Loss and Health Outcome Differences Between African Americans and Caucasions Following Gastric Bypass Surgery

Wendy A. Anderson University of Rhode Island

Follow this and additional works at: https://digitalcommons.uri.edu/theses Terms of Use All rights reserved under copyright.

## **Recommended Citation**

Anderson, Wendy A., "Weight Loss and Health Outcome Differences Between African Americans and Caucasions Following Gastric Bypass Surgery" (2005). *Open Access Master's Theses.* Paper 1170. https://digitalcommons.uri.edu/theses/1170

This Thesis is brought to you by the University of Rhode Island. It has been accepted for inclusion in Open Access Master's Theses by an authorized administrator of DigitalCommons@URI. For more information, please contact digitalcommons-group@uri.edu. For permission to reuse copyrighted content, contact the author directly.

# WEIGHT LOSS AND HEALTH OUTCOME DIFFERENCES BETWEEN AFRICAN AMERICANS AND CAUCASIANS FOLLOWING

## GASTRIC BYPASS SURGERY

BY

WENDY A. ANDERSON

A THESIS SUBMITTED IN FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE IN NUTRITION AND FOOD SCIENCES

UNIVERSITY OF RHODE ISLAND

## MASTER OF SCIENCE THESIS

OF

## WENDY A. ANDERSON

APPROVED:

Thesis Committee:

Major Professor alduce

DEAN OF THE GRADUATE SCHOOL

UNIVERSITY OF RHODE ISLAND

## Abstract

**Background:** Data suggest that African Americans lose less weight and show fewer improvements in cardiovascular diseases such as blood pressure following gastric bypass surgery (GBP).

**Objective:** The purpose of this study was to describe differences in weight loss between Caucasian and African American patients following GBP and to identify if these differences are related to dietary intake. This study also sought to identify differences in blood pressure and serum lipid levels between African Americans and Caucasians after GBP.

Methods and Subjects: This was a retrospective database review of a sample of 84 adult patients, 24 African American and 60 Caucasian women and men between the ages of 33 and 53 years. All subjects had GBP surgery in 2001 at the Bariatric Surgery Program at Boston Medical Center (BMC) in Boston, MA and were followed for one year postoperatively. Patients were excluded if weight data were missing at baseline, three months and one year following GBP. A total of 9 African Americans and 41 Caucasians provided data at all three time points and were included in the study. Weight change was calculated using percent of initial body weight and change in body mass index (BMI). Differences in dietary intake were determined using dietary records completed by the patients or by patient dietary recalls collected by the dietitian. Blood pressure, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglyceride levels at baseline and one year were abstracted from the GBP database. **Results:** There were no differences in baseline characteristics between African Americans and Caucasians. Caucasians lost more weight (BMI reduced from  $56 \pm 12$  to  $34 \pm 8$  at one year after surgery with a percent weight loss of  $38\pm 8$ ) than African Americans (BMI reduced from  $54 \pm 8$  to  $40 \pm 11$  with a percent weight loss of  $26 \pm 10$ ) (p < .001). There were main effects for time and race on weight loss expressed as change in BMI and percent weight loss at one year following GBP (p < .001) as well as an interaction between and within race weight loss (p < .001). There were no differences between races for diet, blood pressure, or serum lipids. However, there were differences within race for each of these variables (p < .05).

**Conclusions:** These data demonstrate that GBP promotes significant weight loss among morbidly obese African Americans and Caucasians. African Americans lost less weight at one year after GBP; however, dietary parameters and cardiovascular risk indices were not significantly different between races. Prospective intervention studies should be conducted to help clarify the primary etiologies for differential weight loss between races following GBP. Identification of these causes will help delineate specific interventions that are sensitive to racial differences, and ultimately optimize the outcome of bariatric surgery in an ethnically diverse patient population.

## Acknowledgements

There are many individuals that have supported me during my journey as a graduate student. I want to thank my mother for her unconditional support as I strived to complete my Master of Science while working full-time. I truly appreciate all the time and effort provided to me by each of my committee members, including Dr. Marjorie Caldwell, Dr. Bryan Blissmer, and Dr. Deborah Riebe. I appreciate my colleagues at work for their support and encouragement along the way, including Dr. Armour Forse, Adrienne O'Brien, and especially Dr. Nawfal Istfan. I want to give special thanks to Dr. Geoffrey Greene for believing in me and providing me with his time and guidance.

## Table of Contents

	Page
Abstract	ii
Acknowledgements	iv
Table of Contents	V
List of Tables	vi
Introduction	1
Methods	3
Gastric Bypass Surgery (GBP) Program Protocol	3
Subjects	5
Measurements	5
Data Analysis and Statistics	6
Results	8
Subjects	8
Weight Loss	8
Dietary Intake	9
Cardiovascular Differences	10
Discussion	11
Literature Cited	
Appendices	25
A Literature Review	25
B Common Dietary Stages Following GBP	

## List of Tables

Table	Page
1: Baseline Characteristics of Subjects by Race	.21
2: Body Mass Indices and Percent Weight Loss 1 Year After GBP	.22
3. Energy and Protein Intake Before and 1 Year After GBP	23
4: Preoperative and 1 Year Postoperative Cholesterol and Blood Pressure Values	24

.

.

\*

### Introduction

The incidence of obesity in the United States (US) has reached epidemic proportions and is considered a significant public health threat. The National Health and Nutrition Examination Survey (NHANES) of 1999-2000 reported that 64.5% of adult Americans are overweight with 30% considered obese. Despite increased medical awareness of obesity and a multitude of intervention programs, the prevalence of obesity has increased more than 75% during the past 25 years (1) with a mortality rate of 300,000 deaths annually (2). Obesity predisposes individuals to multiple comorbid conditions such as cardiovascular disease (CVD) and type 2 diabetes. The incidence of diabetes has increased over the past ten years with 25% of this increase associated with obesity (3). Hypertension and dyslipidemia are also associated with obesity. The primary cause of mortality associated with elevated BMI is cardiovascular disease (4).

Although traditional diets are effective for weight loss between 2-3 kilograms, (5) intensive programs with long-term maintenance including diet, exercise, and behavioral change strategies can produce 5-10 % weight loss (6). This is generally ineffective for Class III obesity (BMI  $\geq$  40 kg/m<sup>2</sup>) with concurrent co-morbid conditions (7). The negative impact of Class III obesity on morbidity and mortality in the US cannot be ignored. GBP has been found to be effective for morbidly obese patients with subsequent improvements in cardiovascular and metabolic sequelae (7). The Roux-en-Y gastric bypass surgery (RYGBP) is now recognized as the gold

standard treatment for patients with Class III obesity with concurrent co-morbid conditions. The RYGBP promotes weight loss by limiting gastric volume and the rate of gastric emptying with resultant early satiety (8). This procedure has demonstrated sustainable weight loss with manageable complications both in the short- and long-term (9).

Studies have demonstrated significant improvements in cardiovascular risk factors and diabetes among Caucasian morbidly obese patients following GBP (10). Similar studies in other ethnic groups remain limited. This might be related to the less frequent use of bariatric surgery in minority populations (11,12). A small number of recent clinical studies suggest that morbidly obese African American women lose less weight and show less improvement in blood pressure than Caucasian morbidly obese women following GBP (9). Although there might be metabolic differences between morbidly obese African Americans and Caucasians, behavioral factors such as dietary intake might also account for differences in weight reduction (13). Clarification of these differences are important for optimizing the post-surgical treatment of morbidly obese African Americans. Due to these racial disparities, it is important that weight loss interventions be targeted toward ethnic needs.

We conducted the following study to compare weight loss between African American and Caucasian morbidly obese patients following GBP surgery in an urban obesity treatment referral center. We also examined differences in dietary intake and cardiovascular risk factors in this patient population before and after weight loss.

#### Methods

## Gastric Bypass Program Protocol

#### Screening

The bariatric surgery program at BMC was launched in 1999, and has since performed more than 600 operations. Patients are referred by their primary care physician to the Bariatric Surgery Program for screening by the bariatric surgeon, a physician board certified in nutrition, a psychologist, and the bariatric registered dietitian for surgical clearance. If cleared by all members of the team, preparation for surgery includes preoperative weight loss, preoperative and postoperative nutrition education, and diagnostic testing. Patients are instructed to follow a low fat, high fiber diet prior to surgery to help promote preoperative weight loss and healthy eating behaviors. Patients are scheduled for surgery approximately nine to 12 months following their initial appointment with the surgeon and must attend at least one gastric bypass support group meeting.

### Perioperative and Postoperative Phases

The hospital stay following surgery is approximately five to six days. The surgical procedure performed is a distal RYGB with a 100 to 150 cm Roux limb length and 30 mL pouch created in the proximal portion of the stomach. Patients are NPO for 48 hours following surgery with advancement to PO if the upper GI series presents as normal. Subsequently, patients are advanced to sips of water (Stage I) followed by a no concentrated sweet clear liquid diet (Stage II), and then to a no

concentrated sweet full liquid diet (Stage III), as tolerated. Patients are discharged on a Stage III diet and continue to follow this diet at home for three weeks. Patients are advised to begin taking chewable multivitamin/mineral and calcium supplements. **Postoperative follow-up visits are scheduled with both the surgeon and the dietitian** ten to 14 days following their hospital discharge. At this time, patients will be instructed to begin a high protein soft solid diet (Stage IV) at three weeks after surgery as tolerated. Patients remain on soft solids for approximately one month, and are then scheduled with the dietitian to advance to a low fat solid diet (Stage V), as tolerated. Patients continue on the low fat diet indefinitely with modifications made as needed at follow-up visits.

Dietary assessment tools, including the Exchanges for Weight Management (14) and dietary logs created by the bariatric dietitian, were provided to patients at their initial postoperative visit. The Exchanges for Weight Management provide patients with macronutrient analyses including the caloric density of their food and beverage intake. Patients were provided dietary logs to record energy, protein, and fluid intake. During each follow-up dietitian visit, food and beverage models were used as visual aids to better assess the reliability of portion sizes noted in patientreported dietary logs or recalls. The bariatric dietitian reviewed all dietary logs and recalls. Patients are advised to consume a minimum of 60 grams of protein from high biological value sources and two liters or more of sugar-free non-caffeinated fluids daily. Patients are encouraged to document energy intake if there is a decrease in the rate or degree of their weight loss. Postoperative follow-up visits with the surgeon are scheduled every four to six weeks for the first year or until patients are weight stable,

and yearly thereafter. Postoperative follow-up visits with the dietitian occur monthly for the first six months and every six months thereafter.

## Subjects

This retrospective study included African American and Caucasian patients who had GBP in 2001 at BMC located in Boston, Massachusetts. A total of 84 patients completed surgery during this period including 52% Caucasian women, 20% Caucasian men, 23% African American women, and 5% African American men between the ages of 31-55 years of age. Patients were excluded if baseline, three months or one year postoperative weight data were missing. A total of nine African Americans and 41 Caucasians provided complete data and were included in this study. There were no differences in age, gender, and proportion with dyslipidemia, hypertension, and diabetes mellitus between the 50 subjects providing complete data and the 34 subjects excluded from the study for incomplete data. The non-completers included 19 Caucasians and 15 African Americans. Those subjects presenting with incomplete weight data had a lower preoperative mean BMI (50  $\pm$  9) compared to those with complete data.

The University of Rhode Island and Boston Medical Center Institutional Review Board approved this study.

## Measurements

Body weight in kilograms was measured at baseline (preoperative visit), three months and one year after GBP. Body weight was measured using a Scale-Tronix

digital scale calibrated every 12 weeks. Height was measured in centimeters using an attached vertical rod.

The bariatric dietitian collected dietary records or dietary recalls from patients at baseline and each postoperative visit. Dietary protein and energy intake for each visit were calculated using the Exchange Lists for Weight Management (14) and Bowes' and Church's Food Values of Portions Commonly Used (15).

Blood pressure and lipid profiles in both ethnic groups were assessed at baseline and one year following GBP. Blood pressure was measured using a baumamometer Standby model (W.A. Baum CO, Inc.) in a postural position. A certified phlebotomist collected blood samples with subjects in a postural position. Blood was taken from an antecubital vein following a 12-hour fast. Low-density lipoproteins (LDL) were calculated using the Friedewald equation if triglycerides were below 400 mg/dL (16). Total cholesterol, high-density lipoproteins (HDL), and triglyceride levels were measured using the Ektachem DT II System. HDL cholesterol levels were then determined by the precipitation method (17).

#### Data Analysis and Statistics

Data were collected from the GBP database and analyzed using the SPSS software version 10.5. Means and standard deviations for all parameters were calculated. Weight and dietary intake were assessed at baseline, three months, and one year after GBP. Primary comparisons were changes in these variables compared between the African American and Caucasian groups at three months and one year following GBP using ANOVA for repeated measures with ethnicity as a between

subjects factor and time as a within subjects factor. This determined changes over time in the above variables as well as ethnicity main effects and ethnicity by time interactions. Weight data were expressed as percent of initial body weight and BMI. Baseline body weight was defined as subject's preoperative weight taken two weeks prior to surgery. Protein intake was expressed as percentage of total kilocalories and energy intake was expressed as kilocalories per kilogram of body weight. Weight and protein and energy intake were assessed at three months and one year after surgery. Changes in health parameters were assessed at baseline and one year after GBP by paired t-tests comparing changes within and between groups.

### Results

#### Subjects

Baseline anthropometric and demographic characteristics of participants providing complete weight data are summarized in Table 1. There were no differences between race in age, BMI, female/male ratio, and prevalence of dyslipidemia, **by**pertension, and diabetes. All subjects had at least one of the noted comorbidities with hypertension (47 %) being the most prevalent. The average patient seeking barjatric surgery was 43 years old with a BMI of 55 kg/m<sup>2</sup>.

Subjects providing incomplete weight data at three months and one year after GBP were not different by race in all measured variables, except for a lower baseline BMI, compared to those subjects providing complete weight data.

## Weight Loss

The observation period for this study was one year following GBP. The mean preoperative BMI for African Americans and Caucasians was  $54 \pm 8$  (N = 9) and  $56 \pm 12$  (N = 41), respectively. Both African Americans and Caucasians significantly reduced their BMI and percent body weight (p < .001). There was an overall effect of race on BMI and percent weight loss with Caucasians losing more weight than African Americans (P < .001). Post hoc analyses demonstrated no ethnic differences in BMI and percent weight loss between the races at baseline or three months following surgery but differences were observed at one year (p < .05) as shown in Table 2. On average, Caucasian patients lost six percent more weight at three months (NS) and 12 % more weight at one year (p < .001) after GBP than African Americans.

## Dietary Intake

As shown in Table 3, African Americans and Caucasians did not differ in dietary intake after GBP as measured by energy (kilocalories) per kilogram and percentage of energy as protein at three months and one year following surgery. The mean energy intake in kilocalories at baseline and one year after GBP was lower in Caucasians ( $2596 \pm 588$  and  $1123 \pm 349$ , respectively) than in African Americans  $(2700 \pm 722 \text{ and } 1368 \pm 349, \text{ respectively})$ . However, at three months after surgery, Caucasians consumed more kilocalories than African Americans (704  $\pm$  238 and 621  $\pm$ 127, respectively). African Americans had a 70 % and 33 % decrease in energy intake at three months and one year after GBP, respectively. Caucasians had a 67 % and 31 % decrease in energy intake at three months and one year, respectively. The mean protein intake in grams at baseline and one year after surgery was higher in African Americans  $(83 \pm 17 \text{ and } 61 \pm 12; \text{ respectively})$  than Caucasians  $(82 \pm 19 \text{ and } 59 \pm 12; \text{ respectively})$ respectively). There was no difference in protein inake between races at three months after surgery. However, Caucasians consumed more grams of protein than African Americans (Caucasians  $62 \pm 14$  and African Americans  $53 \pm 9$ ; p > .05). Both races consumed significantly less energy and more kilocalories as protein at three months (p <.001, p <.001, respectively) and one year (p <.001, p <.001, respectively) after surgery compared to their intake prior to surgery. Using ANOVA for repeated measures, main effects of race were not indicated for changes in energy per kilogram

and percentage of energy from protein one year after GBP. However, main effects of time on energy intake per kilogram and percentage of kilocalories as protein were significant for African Americans and Caucasians (p < .001).

# Gardiovascular Risk Differences

Differences in blood pressure and lipid values between African Americans and Caucasians are detailed in Table 4. There were significant improvements in blood pressure and all lipid serum indices within both races one year following surgery (p < .01) indicating decreased cardiovascular risks. There were no differences between races in diastolic and systolic blood pressure at baseline and one year following GBP. Similarly, there were no differences between races in serum total cholesterol, HDL cholesterol, calculated LDL cholesterol, and triglycerides at baseline and one year following surgery. ANOVA for repeated measures did not show main effects of race for blood pressure (systolic and diastolic) and serum lipids (total cholesterol, HDL cholesterol, LDL cholesterol, and triglycerides).

## Discussion

This study showed significant weight loss at one year following GBP among the entire sample. There were differences in weight loss between races with African Americans losing 12% less weight compared to Caucasians at one year after GBP; African Americans and Caucasians lost a mean total of 44 and 61 kilograms at one year after surgery, respectively. These results are similar to those found by Sugerman and Latner et. al. (18,19). The etiology for this difference is still unclear; however, it has been suggested that genetic, environmental, behavioral, psychosocial, cultural, and metabolic differences contribute to this disparity (20).

This study observed a significant decrease in energy intake at three months and one year within each race following GBP. This is expected following GBP due to the reduced gastric pouch capacity (21, 22). Our findings of similar energy intake between races at each observed time point following GBP suggest the possibility of metabolic differences between races. Other studies have found that African American women appear to adapt to energy deficits more efficiently than Caucasian women; they require fewer calories for weight maintenance and have a reduced rate of weight loss on hypocaloric diets compared to Caucasian women (23-25). Carpenter et. al. found lower basal metabolic rates and activity energy expenditure (AEE) in African Americans (26). Weisner et. al. found that AEE was significantly different between races with African Americans having a lower AEE following weight loss. Caucasians showed greater improvements in VO2 max, physical activity, and energy expenditure (EE) compared to their African American counterparts in response to diet and exercise

weight loss interventions (27). These findings have been shown in other studies (28, 29) suggesting decreased weight loss efficiency among African American women compared to Caucasian women. Our study did not evaluate differences in physical exercise and energy expenditure. Future intervention studies exploring the behavioral and metabolic effects on weight loss are needed to further understand the influences of ethnicity on weight reduction following bariatric surgery.

Results from this study showed a significant decrease in energy intake with an increase in percentage of kilocalories from protein within each race at three months and one year after GBP. Moize and colleagues also observed these postoperative dietary changes. Due to the reduced capacity of the gastric pouch energy intake significantly decreases, especially during the first six months to a year following GBP. High biological value protein sources are encouraged to promote postoperative healing and to preserve and restore lean body tissue (30). Modular protein supplements were recommended if daily protein intake was less than 60 grams daily, especially during the first six months following surgery. Six out of the nine African Americans consumed less than 60 grams of protein at three months after surgery, potentially resulting in decreased visceral protein stores and wound healing. Despite the greater weight loss among Caucasians, we found no ethnic differences in energy and percentage of calories from protein at each observed time point. This was also observed in other studies (31, 32) and might be due to under-reporting of energy intake among African Africans as shown by Lovejoy et. al. (33). Latner and colleagues found that African Americans and Caucasians lost 62 % and 80 % of excess BMI following GBP, respectively, but African Americans consumed overall

fewer kilocalories than Caucasians with no differences found in exercise frequency (19). The utility of self-reported dietary intake to assess energy and macronutrient intake might have lead to a 20-50 % error of actual intake (34), which was a major limitation in most of these studies. Future research can improve the accuracy of actual dietary intake by use of doubly labeled water methods (29, 35) or by designing more controlled dietary interventions.

This study examined ethnic differences in cardiovascular disease risk factors following GBP. Presently, there are limited data comparing the effect of weight loss on cardiovascular risk between African Americans and Caucasians following bariatric surgery (35). Our results did not show differences between race in serum lipid profiles at baseline and one year following GBP. However, we did find a greater percentage of African Americans compared to Caucasians with dyslipidemia at baseline (44 % and 35 %, respectively). Finally, HDL levels were greater at baseline and one year following surgery but were not statistically different between races. The small African American sample size might have affected the reduced ability of this study to find statistically significant differences, but the effect of size for race was low suggesting that a larger sample size might not have found differences. Irrespective of BMI, data have shown that African Americans as compared to Caucasians have higher HDL levels with a subsequent lower risk for CVD (36, 37).

Our findings suggest no difference in cardiovascular risk factors between races. Residori and colleagues reported that African Americans had the highest incidence of hypertension compared to Caucasians and other ethnic groups independent of BMI (20). That study did not examine subjects having undergone

GBP. Sugerman et. al. found that hypertension improved in most subjects with weight loss at one to two years following GBP. However, African Americans responded less favorably to improvements in hypertension following GBP induced weight loss as compared to Caucasians (35). There were several limitations of our study. First, the small sample of African Americans studied compared to Caucasians. We might have observed the same results as Sugerman et. al. with a larger African American sample size. Secondly, the effect of medications that subjects might have been taking indicated by their medical condition was not monitored in this study.

In conclusion, our data confirm that morbidly obese African Americans tend to lose less weight following gastric bypass surgery in comparison to Caucasians. These differences in weight loss appear to be related to lower energy expenditure and not dietary intake. However, despite smaller weight losses among the African Americans, improvements in cardiovascular risk factors are similar between the two races.

#### Literature Cited

- Flegal KM, Carroll MD, Ogen CL, Johnson CL. Prevalence and trends in overweight among US adults, 1999-2000. JAMA. 2002; 288 (14): 1723-1727.
- Allison DB, Fontaine KR, Manson JE, Stevens J, Vanltalle TB. Annual deaths attributable to obesity in the United States. JAMA. 1999; 282:1530-1538.
- Harris ML, Flegal KM, Cowie CC, Eberthardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: The Third National Health and Nutrition Examination Survey, 1988-94. *Diabetes Care*. 1998; 21:518-524.
- Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. *Int J Obes Relat Metab Disord*. 1996; 20:63-75.
- Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. JAMA. 2005; 293(1): 43-53.
- Expert Panel. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Bethesda, MD: National Institute of Health, National Heart Lung, and Blood Institute, U.S. Department of Health and Human Services, Public Health Service, 1998.
- O'Connell TL. An overview of obesity and weight loss surgery. *Clinical Diabetes*. 2004; 22(3): 115-120.

- Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J: Outcomes after laparoscopic Roux-En-Y gastric bypass for morbid obesity. *Ann Surg.* 2000; 232:515-529.
- Sugerman HJ, Wolf LG, Sica DA, Clore JN. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg.* 2003; 237(6): 751-756.
- Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjostrom CD, Sullivan M, Wedel H. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004; 351(26): 2683-2693.
- Choban P, Lu B, Flancbaum L. Insurance decision about obesity surgery: a new type of randomization? Obes Surg. 2000; 10(6):553-6.
- Livingston EH, Ko CY. Socioeconomic characteristics in the population eligible for obesity surgery. Surgery. 2004; 135(3): 288-96.
- Price RA, Reed DR, Guido NJ. Resemblance for body mass index in families of obese African American and European American women. *Obes Res.* 2000; 8:360-366.
- Exchange Lists for Weight Management. American Diabetes and Dietetic Associations, revised 1995.
- Pennington J.A.T. Bowes and Church's Food Values of Portion Commonly used.1989, 15<sup>th</sup> edition.

- 16. Friedewald WT, Levy RI, Frederickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma without use of an ultracentrifuge. *Clin Chem.* 1972; 18:499-502.
- 17. Gidez LI, Miller GO, Burstein M. Separation and quantification of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. *J Lipid Res.* 1982; 23:1206-1223.
- Sugerman HJ. Bariatric surgery for severe obesity. J Assoc Acad Minor Phys. 2001; 12(3): 129-136.
- Latner JD, Wetzler S, Goodman ER, Glinski J. Gastric bypass in a lowincome, inner-city population: eating disturbances and weight loss. *Obes Res.* 2004; 12(60): 956-961.
- Residori L, Garcia-Lorda P, Flancbaum L, Pi-Sunyer X, Laferrere B.
   Prevalence of co-morbidities in obese patients before bariatric surgery: effect of race. *Obes Surg.* 2003; 13: 333-340.
- 21. Coughlin K, Bell RM, Bivins BA, Wrobel S, Griffen WO Jr. Preoperative and postoperative assessment of nutrient intakes in patients who have undergone gastric bypass surgery. *Arch Surg.* 1983;188 (7):813-816.
- 22. Kenler HA, Brolin RE, Cody RP. Changes in eating behavior after horizontal gastroplasty and Roux-en-Y gastric bypass. *Am J Clin Nutr.* 1990; 52:87-92.
- 23. Chitwood LF, Brown SP, Lundy MJ, Dupper MA. Metabolic propensity toward obesity in black vs white females: responses during rest, exercise and recovery. *Int J Obes.* 1996; 20: 455-462.

- 24. Geissler CA, Aldouri MSH. Racial differences in the energy cost of standardized activities. Ann Nutr Metab. 1985; 29: 40-47.
- 25. Weisner RL, Hunter GR, Zuckerman PA, Redden DT, Darnell BE, Larson DE, Newcomer BR, Goran MI. Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr.* 2000; 71:1138-46.
- 26. Carpenter WH, Fonong T, Toth MJ, Ades PA, Calles-Escandon J, Walston JD, Poehlman ET. Total daily energy expenditure in free-living older African-Americans and Caucasians. Am J Physiol. 1998; 274(1 Pt 1): E96-101.
- 27. Weinsier RL, Hunter GR, Schutz Y, Zuckerman PA, Darnell BE. Physical activity in free-living, overweight white and black women: divergent responses by race to diet-induced weight loss. *Am J Clin Nutr.* 2002; 76(4): 736-42.
- 28. Washburn RA, Kline G, Lackland DT, Wheeler FC. Leisure time physical activity: are there black/white differences? *Prev Med.* 1992; 21:127–35.
- 29. Foster GD, Wadden T A, Swain RM, Anderson DA, Vogt RA. Changes in resting energy expenditure after weight loss in obese African American and white women. *Am J Clin Nutr.* 1999:69:13-17.
- 30. Moize V, Geliebte A, Gluck ME, Yahav E, Lorence M, Colarusso T, Drake V, Flancbaum L. Obese patients have inadequate protein intake related to protein intolerance up to 1 year following Roux-en-Y gastric bypass. *Obes Surg.* 2003; 13:23-28.

- 31. Kumanyika SK, Obarzanek E, Stevens VJ, Hebert PR, Whelton PK. Weightloss experience of black and white participants in NHLBI-sponsored clinical trials. Am J Clin Nutr. 1991; 53(6 Suppl): 1631S-1638S.
- 32. Sugerman HJ, Londrey GL, Kellum JM, Wolf L, Liszka T, Engle KM,
  Birkenhauer R, Starkey JV. Weight loss after vertical banded gastroplasty and
  Roux-en-Y gastric bypass for morbid obesity with selective versus random
  assignment. Am J Surg. 1989; 157:93-102.
- 33. Lovejoy JC, Smith SR, Rood, JC. Comparison of regional fat distribution and health risk factors in middle-aged white and African American women: the Healthy Transitions Study. Obes Res. 2001; 9:10-16.
- 34. Black AE, Prentice AM, Goldberg GR, Jebb SA, Bingham SA, Livingstone MB, Coward WA. Measurements of total energy expenditure provide insights into the validity of dietary measurements of dietary intake. J Am Diet Assoc. 1993; 93: 572-9.
- 35. Schoeller DA. Measurement of energy expenditure in free-living humans by using doubly labeled water. *J Nutr.* 1988; 118:1278–89.
- 36. Nelson TL, Hunt KJ, Rosamond WD, Ammerman AS, Keyserling TC, Mokdad AH, Will JC. Obesity and associated coronary heart disease risk factors in a population of low income African American and White women: The North Carolina WISEWOMAN Project. *Prevention Medicine*. 2002; 35: 1-6.

37. Racette SB, Horowitz JF, Mittendorfer B,Klein S. Racial differences in lipid metabolism in women with abdominal obesity. *Am J Physiol Regulatory Integrative Comp Physiol.* 2000; 279:R944-R950.

- Byellipidemie
- AND DESCRIPTION OF THE OWNER OF T
- Department of the
- and set of a

# Table 1

# Baseline Characteristics of Subjects by Race

	African Americans	Caucasians
N	9	41
Age (Mean±SD)	43±10	43±10
BMI (Mean±SD)	54±8	56±12
Female/Male %	78/22	73/28
Dyslipidemia %	44	35
Hypertension %	44	50
Diabetes Mellitus %	44	25

N = subjects with weight data at baseline, 3 months and 1 year BMI = body mass index; calculated as  $kg/m^2$ 

#### Table 2

## Body Mass Indices and Percent of Weight Loss Up to 1 Year Following GBP

	Afri	can Americans	Caucasi	ans
	BMI <sup>1*</sup> kg/m2	% intial body weight <sup>2*</sup> ( % weight loss )	BMI <sup>1*</sup> kg/m2	% initial body weight <sup>2*</sup> (% weight loss)
N	9	9	41	41
Preop	54±8	100 (0)	56±12	100 (0)
Post 3 months	50±9	91.4±7.4 (-8.7±7)	48±11	85.4±3.8 (-15.2±5.5)
Post 1 year	<sup>ab</sup> 40±11	73.9±9.6 <sup>ab</sup> (-26±9.6)	<sup>ab</sup> 34±8	61.7±7.9 <sup>ab</sup> (-38±7.9)

- All data are means  $\pm$  SD; N = subjects without missing data
- BMI = body mass index; calculated as  $kg/m^2$

Weight loss = % of initial weight

\*F (df hypothesis, df error), p by repeated measures

1. 130 (2, 47), p = 0.000 for time; 7.6 (2, 47), p = 0.001 for race x time

2. 280 (2, 47), p = 0.000 for time; 12.3 (2, 47), p = 0.000 for race x time

#### Table 3

Energy and Protein Intake Before and Up to 1 Year Post GBP.

	African	Americans	Caucasians		
	kilocalories/kilogram <sup>1*</sup>	% kilocalories from protein <sup>2*</sup>	Kilocalories/kilogram <sup>1*</sup>	% kilocalories from protein <sup>2*</sup>	
N	9	9	41	41	
Preop	16±5	13±2	17±4	13±2	
Post 3 months	*5±2	**33±8	*6±2	**37±12	
Post 1 year	*12±4	**23±7	*12±4 **23±9		

All data are means  $\pm$  SD; N = subjects without missing data

\*F (df hypothesis, df error), p by repeated measures

- 1. 126(2, 45), p = 0.000 for time; 1.530(2, 45), p = 0.230 for race x time
- 2. 67 (2, 45), p = 0.000 for time; 0.424 (2, 45), p = 0.657 for race x time

	African Americans		Caucasians			
	Preop	Post 1 year	Mean Change(95%CI) <sup>(1)</sup>	Preop	Post 1 year	Mean change 95%(CI) <sup>(1)</sup>
тс	220±19 (7)	181±33 (7)	-35(-10, -61) *	199±33 (40)	179±35 (40)	-21(-10, -32)*
HDL	56±17 (6)	66±21 (6)	10(25, 5)**	48±10 (33)	54±16 (33)	6(10, 2)**
LDL	132±7 (5)	107±37 (5)	-25(-29, -79)**	120±32 (35)	103±31 (35)	-17(-6, -28)**
TG	130±57 (7)	80±24 (7)	-50(-4, -96)**	159±55 (37)	109±53 (37)	-50(-26, -74)**
Sys	136±18 (9)	117±15 (9)	-19(-3, -35)*	135±23 (41)	123±20 (41)	-12(-6, -18)*
Dias	82±7 (9)	70± 8 (9)	-12(-4, -19)*	84±12 (41)	73±11 (41)	-11(-7, -15)*

Preoperative and 1 Year Postoperative Cholesterol and Blood Pressure Values

All data are means  $\pm$  SD; N = subjects without missing data;

TC = total cholesterol; HDL = high density lipoproteins; LDL = low density lipoproteins; TG = triglycerides

Sys = systolic blood pressure; Dias = diastolic blood pressure

(1) Mean Change = negative markers indicate decreases

\*P < .001; \*\*P < .01 within race

## Appendices

## Appendix A Literature Review

## Introduction

The incidence of obesity in the United States (US) has reached epidemic proportions and is considered a significant public health threat. The National Health and Nutrition Examination Survey (NHANES) of 1999-2000 reported that 64.5% of adult Americans are overweight with 30% considered obese. Despite the increased medical awareness of obesity and a multitude of diet and exercise intervention programs, obesity prevalence has increased by more than 75% during the past 25 years (Flegal Km, Carroll MD, Ogen CL, Johnson Cl, 2002) with a mortality rate of 300,000 deaths yearly (Allison DB, Fontaine KR, Manson JE, Stevens J, Vanltalle TB, 1999).

#### I. Obesity

## A. Classification of Obesity and Comorbidities

According to the World Health Organization (WHO) obesity is a disease that affects all ages, both genders, and all ethnic groups. WHO classifies weight status based on BMI, which is defined as weight in kilograms divided by height in meters squared (WHO, 2000). Obesity is defined as a BMI of  $\geq$ 30 kg/m<sup>2</sup> and morbid obesity as a BMI of  $\geq$ 40 kg/m<sup>2</sup> with the risks for morbidity and mortality being greatest at a BMI of  $\geq$ 40 kg/m<sup>2</sup> (WHO, 2000). The diagnosis of diabetes has increased during the past ten years with 25% of this increase associated with obesity (Harris ML, Flegal KM, Cowie CC, Eberthardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD 1998). Hypertension, dyslipidemia, and metabolic syndrome are highly associated with obesity. The primary causes of mortality associated with elevated BMI are from rdiovascular diseases (Troiano RP, Frongillo EA, Sobal J, Levitsky DA, 1996).

## B. Etiology

Obesity is a condition influenced by genetic, psychosocial, metabolic, cultural, and behavioral factors (NIH, NHLBI, 1998). Although genetic predisposition plays a role in the expression of obesity, genetic factors cannot explain the exponential rise in obesity over the past two decades. Behavioral and environmental factors are the most probable causes for the expression of genes associated with obesity (Mun EC, Blackburn GL, Mathews JB, 2001). Some of these factors include reduced physical activity and the availability of high calorie foods resulting in positive energy balance (Harnack LJ, Jeffery RW, Boutelle KN, 2000; Flegal Km, Carroll MD, Ogen CL, Johnson Cl, 2002). Energy intake and output are difficult to quantify, therefore it is important to further examine all causal factors involved such as cultural, **pcioeconomic**, psychological, and metabolic influences (Flegal Km, Carroll MD, Ogen CL, Johnson Cl, 2002).

## i. Genetics

Body adiposity is influenced by the interaction of genotype and environment. Data suggests that at least 40% of body mass is genetically determined (Bouchard C, Perusse L, 1993). This has been examined in twin, adopted, and family studies Lefebvre PJ, Scheen AJ, 2001). Based on a series of twin studies, Bouchard
suggested that 25-40% of body weight regulation might be related to genetic factors
Bouchard C, 1994; Bouchard C, Tremblay A, Despres JP, Nadeau A, Dussault J,
Moorjani S, Pinault S, Fournier G, 1990).

## ii. Cultural/Socioeconmic

Data suggest that an inverse relationship exists between obesity and cioeconomic status (Sobal J, Stunkard AJ, 1989). The reasons for this association might be due to childhood experiences such as parental neglect (Lissau I, Sorensen TIA, 1994) indigent residence (Lissau I, Sorensen TIA, 1994), and social isolation (Kinra S, Nelder RP, Leweendon GJ, 2000). In addition, a relationship between ocioeconomic hierarchy and BMI has been observed. Data has shown that people of higher socioeconomic status have a lower prevalence of obesity in part due to their heightened interest in health and fitness (Wardl J, Griffith J, 2001). Other factors involved include the higher financial expense to eat healthy including foods such as fresh fruits and vegetables. Data suggest that people of lower socioeconomic status tend to purchase economical foods, which are most often higher in fat and calories **Potentially leading to weight gain (Drewnowski A**, 2003).

Cultural and ethnic factors have been associated with obesity. Minority groups have had a higher increase in obesity as compared to Caucasians with the exception of Asians (Drewnowski A, 2004). African American women have the highest prevalence of obesity compared to other minority groups (Flegal Km, Carroll MD, Ogen CL, Johnson Cl, 2002). The reasons for this disparity are still unclear; however, evidence

points to physical inactivity (Weisner RL, Hunter GR, Heini AF, Goran MI, Sell SM, 1998) as a primary factor as well as lifestyle, environment, genetics, race, and energy metabolism (Lovejoy JC, Champagne CM, Smith SR, de Jonge L, Xie H, 2001).

# iii. Psychological

According to the NHANES, depression is associated with an increased prevalence of obesity in the US (DiPietro L, Anda RF, Williamson DF, Stunkard AJ, 1992). The psychological influences on the onset of obesity include both mental health status and the use of psychotropic medications. These medications block the receptors for acurotransmitters associated with weight regulation, such as serotonin, resulting in potential weight gain (Devlin MJ, Yanovski SZ, Wilson GT, 2000). In addition, people with depression often rely on eating to improve their mood resulting in weight gain (Wurtman RJ, Wurtman JJ, 1998).

## iv. Behavioral and Environmental (Diet and Physical Activity)

Positive energy balance is the primary cause for increased adiposity (NIH, NHLBI, 1998). Energy intake has increased over the past two decades most likely from the increased consumption of meals away from the home, the availability of high calorie snack foods and convenience foods, and an increase in portion sizes (Young LR, Nestle M, 2002). These foods include high fat foods, added sugar in snacks (Zizza C, Siega-Riz AM, Popkin BM, 2001), sugared beverages (Harnack L, Stang J, Story M, 1999), and fast foods (French SA, Story M, Jeffery RW, 2001). WHO suggests that obesity epidemic (WHO, 2003). This was supported in a secondary analysis of the CARDIA Study. Subjects who ate fast foods more than twice a week over 10 years gained an extra 4.5 kilograms of body weight compared to those subjects consuming fast foods less than one time per week (Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS, 2005).

Studies have shown a negative relationship between BMI and physical activity Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH, 1996). A sedentary lifestyle promotes weight gain by decreasing energy expenditure. Data suggest that inadequate physical activity is strongly associated with weight gain in both genders. This was shown in a study lasting a decade and included adult subjects between the ages of 25 and 74 years. Subjects who were inactive were more likely to gain between three and four times more weight than the active subjects (Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers TJ, 1993). The sedentary behavior most likely to promote weight gain is television viewing. The Nurses' Health Study identified a 23% increase in obesity with frequent television watching (Hu FB, Li TY, Colditz GA, Willett WC, 2003).

## C. Non-Surgical Obesity Interventions

#### i. Diet

Non-surgical interventions for the treatment of obesity include four modalities: diet, physical exercise, behavior modification, and medications (Mun EC, Blackburn GL, Matthews JB, 2001). Dietary interventions include moderate calorie restriction and very low calorie diets (VLCDs). VLCDs consist of an energy intake of 200-800 kilocalories daily (Bray G, 1998). Individuals presenting with a BMI greater than 30 kg/m<sup>2</sup> and have failed other weight loss approaches might be prescribed a VLCD under close medical supervision. Although this approach promotes rapid initial weight loss, long-term efficacy is lacking (Wadden TA, Foster GD, Letizia KA, 1994). Moderate calorie restriction is more balanced in terms of nutrient content and consists of a caloric deficit of 500-1000 kilocalories daily, promoting a weight loss of approximately ½-1 kilogram weekly.

A recent study by Dansinger and colleagues compared four commercial diets (Weight Watchers, Ornish, Atkins, and Zone) on dietary compliance along with weight loss outcomes and CVD risk factor improvements. The 160 participants in this study were randomized to one of the above commercial diets. They were advised to follow these diets per dietary protocol for two months at which time participants were able to adhere to the diets per self-selection. All measured outcomes at one year were self-reported by participants. Those participants that completed the study had more weight loss and improvements in CVD risk factors compared to those that did not complete the study. All of the diets significantly improved participants' cholesterol ratio; however, there were no differences in blood pressure and blood glucose at one year. Participants with greater dietary compliance had more weight loss and improvement in CVD risk factors. Among the four diets, total mean weight loss was at one year was 2.1 to 3.3 kilograms indicating modest weight loss (Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ).

# ii. Exercise

Physical activity and exercise are important elements in successful weight loss Derventions. Some studies have reported limited or no weight loss implementing physical activity and exercise without adjunct dietary therapy (Garrow JS, Summerbell CD, 1995). However, interventions combining diet and exercise have been effective. In regards to the health benefits from exercise, several studies have reported beneficial health effects such as improvement of diabetes and cardiovascular disease (Tremblay A, Despres JP, Maheux J, Pouliot MC, Nadeau A, Moorjani S, Lupien PJ, Bouchard C, 1991; Ekelund LG, Haskell WL, Johnson JL, Whaley FS, Criqui MH, Sheps DS. 1988).

#### iii. Behavior Therapy

Behavior therapy seeks to identify the barriers preventing adherence to dietary and physical activity interventions important for the promotion of weight reduction and weight maintenance. This therapy has shown to be effective in conjunction with diet therapy. In fact, long-term follow-up of patients has indicated weight regain without behavior therapy (Wadden TA, Foster GD, Letizia KA, 1994). The **Identification**, Evaluation and Treatment of Overweight and Obesity in Adults NIH **Expert** Panel recommends that weight reduction and weight maintenance programs **consist** of both dietary and behavior therapy for the promotion and maintenance of **weight** loss (NIH, 1998). iv. Marmacotherapy

Pharmacotherapy for the promotion of weight loss includes two primary classes based on their mode of action: appetite suppression and initiation of thermogenesis and intestinal fat malabsorption or both. (Bray GA, 1993). Currently, there are several weight loss medications in use: Orilstat, an inhibitor of pancreatic lipase promoting malabsorption of dietary trigylcerides (Hauptman JB, Jeunet FS, Hartman D, 1992); Sibutramine, an anorexiant (Rolls BJ, Shide DJ, Thorwart ML, Ulbrecht JS, 1998); and Phentermine, an anorexiant (Bray GA, 1993). These medications are most effective when prescribed in adjunct with diet therapy, behavior therapy, and exercise (Wadden TA, Berkowitz RI, Sarwer DB, Prus-Wisniewski R, Steinberg C, 2001). The efficacy of these medications is variable, most often promoting a weight loss up to ten percent based on pre-treatment weight with an expected regain of at least some of the weight lost. However, some studies suggest that there is less weight regain in those subjects on pharmacotherapy for weight reduction (Sjostrom L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP, Krempf M, 1998; James WP, 1995; Finer N, Bloom SR, Frost GS, Banks LM, Griffiths J, 2000). Pharmacotherapy has not promoted significant weight loss for the morbidly obese person, and therefore is not considered an efficacious treatment for those with a BMI > 40 kg/m<sup>2</sup>.

# D. Racial Disparities in Obesity and Health

There are clear ethnic differences in the prevalence of obesity. More than twothirds of African American women are considered overweight or obese which is 1.4

times greater than Caucasian women (Linquist CH, Gower BA, Goran MI, 2000; Weisner RL, Hunter GR, Zuckerman PA, Redden DT, Darnell BE, Larson DE, Newcomer BR, Goran MI, 2000). The prevalence of obesity as reported by the NHANES 1999-2000 increased by 11.5 % and 7.2 % among African American and Caucasian women, respectively, compared to NHANES III (Flegal Km, Carroll MD, Ogen CL, Johnson Cl, 2002).

## i. Comorbidities

Obesity has been shown to be a major risk factor in the development of several comorbidities including CVD, diabetes, and certain cancers (Linquist CH, Gower BA, Goran MI, 2000). Obesity in African Americans is associated with diabetes and **Expertension**. African Americans have double the risk of developing CVD and type 2 diabetes compared to Caucasians (Harris MI, 1990). Complications associated with type 2 diabetes; such as retinopathy, nephropathy, neuropathy, and lower extremity amputations are greater in African Americans than Caucasians (Harris MI, 2001). Data suggest that pre- and postmenopausal African American women are more insulin resistant than their Caucasian counterparts indicating an increased propensity to developing type 2 diabetes (Ryan AS, Nicklas BJ, Beman DM, 2002). Gower et. al. found that both premenopausal obese African American and Caucasian women showed improvements in HDL cholesterol, total cholesterol, and LDL cholesterol with weight loss (Gower BA, Weisner RL, Jordan JM, Hunter GR, Desmond R, 2002). The North Carolina WISEWOMAN project showed that obese African American women had higher body mass indices but did not show significant differences in the

evalence of hypertension compared to their obese Caucasian counterparts. However,
con-obese African American women had a significantly higher prevalence of
ipertension than non-obese Caucasian women (Nelson TL, Hunt KJ, Rosamond WD,
immerman AS, Keyserling TC, Mokdad AH, Will JC, 2002). Weight differences
between African Americans and Caucasians appear in childhood, which might
contribute to the increased risk for diabetes and CVD seen in African Americans
(Straus RS, Pollack HA, 2000; Freedman DS, Srinivasan SR, Valdez RA, Williamson
DF, Berenson GS, 1997). Due to these racial disparities, it is important to target
weight loss and the prevention of dsease interventions toward ethnic needs.

#### ii. Metabolism

Ethnic differences in EE have been observed in several studies. Data suggest that African Americans have a lower resting energy expenditure (REE) compared to their Caucasian counterparts (Sharp TA, Bell ML, Grunwald GK, Schmitz KH, Sidney S, Lewis CE, Tolan K, Hill JO, 2002). Gannon et. al. reviewed 15 studies examining the effects of energy expenditure parameters on weight loss comparing African Americans and Caucasians. Twelve studies found lower EE in African Americans using indirect calorimetry and doubly labeled water methods. Two of the five remaining studies found that African Americans have lower total daily energy expenditure (TDEE) as a result of decreased physical activity energy expenditure (PAEE) (Gannon B, DiPietro L, Poehlman ET, 2000). Hunter et. al. suggested that a **Possible** reason for this lower REE in African Americans might be from the decreased **Example** active trunk (organ) tissue found in African Americans compared to **Example** (Hunter GR, Weisner RL, Darnell BE, Zuckerman PA, Goran MI, 2000).

Data suggest that muscle tissue type might have an effect on weight loss with Type I fibers being more metabolically active compared to type IIb muscle fibers (Tanner CJ, Barakat HA, Lynis Dohm G, Pories WJ, MacDonald KG, Cunningham PRG, Swanson MS, Houmard JA, 2002). Tanner et.al. compared muscle fiber type and weight loss outcomes between African American and Caucasian women. Subjects were grouped and compared by ethnic background and BMI. Results showed that obese African American women had a significantly higher number of type IIb muscle fibers compared to their Caucasian counterparts. Interestingly, this study found a positive relationship between the amount of type 1 muscle fibers and the degree of weight loss in the obese groups (Tanner CJ, Barakat HA, Lynis Dohm G, Pories WJ, MacDonald KG, Cunningham PRG, Swanson MS, Houmard JA, 2002).

Lovejoy et. al. examined the disparities in EE and dietary intake between premenopausal middle-aged African American and Caucasian women participating in the Parent, Healthly Transition Study. Dietary data was collected from food journals completed by subjects for four days and analyzed using nutrient analysis computer software. Using direct calorimetry, sleep energy expenditure (SEE) was lower in African American women compared to Caucasian women. Intake of protein was penificantly lower in African American women than in their Caucasian counterparts. Nowever, no ethnic differences were found in fat and carbohydrate intake (Lovejoy JC, Champagne CM, Smith SR, de Jong L, Xie H, 2001). The above findings suggest

that ethnic differences in EE and protein intake might have an impact on the revalence of obesity in African American women.

Foster et.al. compared the effects of weight loss in obese African American (BMI  $36.8 \pm 4.4$ ) and Caucasian ( $36.1 \pm 5.1$ ) women. Subjects included non-smoking women without significant medical or psychiatric disorders. Prior to the weight loss intervention, REE was measured using indirect calorimetry and was shown to be pignificantly lower in African American women than in Caucasian women. After following equivalent dietary plans, African Americans lost 2.5% less weight than Caucasian women after the weight loss intervention suggesting that obese African American women had greater reductions in REE than Caucasian women have increased efficiency in conserving body weight compared to obese Caucasian women resulting from a greater decrease in REE (Foster GD, Wadden TA, Swain RM, Anderson DA, Vogt RA, 1999).

II. Gastric Bypass Surgery (GBP) for the Treatment of Extreme Obesity

The Roux-en-Y gastric bypass procedure (RYGBP) was first introduced for the treatment of morbid obesity in the late 1960's. Edward Mason, MD developed this procedure based on the significant weight loss outcomes from patients following partial gastrectomy surgeries (Mason EE, 1992). The RYGBP is now recognized as the gold standard treatment for many patients with Class III obesity, especially those with serious medical co-morbid conditions. Approximately 109,000 weight loss **surgeries** are performed annually in the US (Klein S, Burke LE, Bray, George, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH, 2004). This procedure has demonstrated term (Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J, 2000).

The RYGBP is achieved by creating approximately a 30 mL pouch in the proximal stomach. The gastric pouch is divided from the remaining distal stomach by staples. The transit of food passes from the pouch through a tight outlet or stoma into the jejunum while bypassing the duodenum. The small pouch and narrow outlet result in restricted calorie intake. Weight loss results primarily from the small pouch capacity leading to early satiety and decreased energy intake (Sonnanstine TE, Kim JJ, Shikora SA, 2004).

Bariatric surgeries have proven to be efficacious for long-term weight loss, with the RYGBP yielding the greatest benefit with fewer complications (NIH Conference Development Conference Panel, 1991). These surgeries predispose patients to maldigestion and malabsorption due to the anatomical changes of the stomach and small bowel, and the potential for inappropriate dietary and eating behaviors. It is critical that patients fully comprehend the dietary and eating behavior restrictions following bariatric surgery necessitating comprehensive preoperative nutrition education and behavior modification counseling.

The postoperative care of bariatric surgery patients should include a **Pultidisciplinary** approach. The dietitian plays a primary role in this team approach, in the prevention and treatment of disordered eating behaviors and nutritional **deficiencies**. Patients with preoperative active eating disorders are not considered **candidates** for bariatric surgery, unless they undergo treatment with documented **resolution** of this behavior prior to surgery. However, disordered eating behavior

might develop postoperatively, requiring judicious monitoring by the bariatric team (Betsy Lehman Center Expert Panel, 2004). Nutrition support following bariatric surgery aims to promote adequate nutrition and hydration to ensure homeostatic support of bodily functions, promote wound healing, and spare lean body mass. The set-bariatric surgery diet should be advanced in stages to achieve adequate nutrition support and food tolerance (Elliot K, 2003). The following review will delineate the primary nutritional considerations following bariatric surgeries including short- and long-term medical nutrition therapy.

## A. Energy and Macronutrient Needs

#### i. Energy

Insufficient data is available to provide evidence-based recommendations for daily calorie, protein, carbohydrate, and fat intake following bariatric surgery. Caloric intake during the first few months postoperatively might be fewer than 1000 kilocalories daily with a moderate increase during the first year (Moize V, Geliebter A, Gluck ME, Yahav E, Lorence E, Colarusso T, Drake V, Flancbaum L, 2003; Kenler HA, Brolin RE, Cody RP, 1990; Coughlin K, Bell RM, Bivins BA, Wrobel S, Griffen WO Jr, 1983; Betsy Lehman Center Expert Panel, 2004). As dietary intake increases after surgery, energy provided varies with the volume of food and fluid that is tolerated. Daily caloric intake usually approaches 400 calories for the first four weeks postoperatively. Intake can be expected to increase to 800-1000 kilocalories daily by the end of the third postoperative month, with weight loss being most rapid during this time period (Betsy Lehman Center Expert Panel, 2004). Greater than 25% of the total daily calories should come from high quality protein (60-80 grams daily). Fat intake should be approximately 25-30 % of the total daily kilocalories. As carbohydrate is protein sparing, approximately 40-50 % of the remaining energy should be supplied as carbohydrate with at least 100 grams per day being recommended for the prevention of ketosis (Betsy Lehman Center Expert Panel, 2004).

#### ii. Protein

Protein-calorie malnutrition is uncommon in patients who have had gastric restrictive surgeries such as the RYGBP and vertical banded gastroplasty (VBG) unless an anatomical problem such as stomal stenosis is present leading to poor dietary intake (Bryne KT, 2001). Adequate protein intake is encouraged postoperatively for improved healing and to maintain adequate visceral protein stores as well as to decrease loss of lean body mass. Due to the small capacity of the gastric pouch following restrictive surgeries, an increased intolerance to meats might occur. Modular and liquid protein supplements might be necessary to promote adequate daily protein intake. The recommendations for protein intake as indicated by the Betsy Lehman Center Weight Loss Surgery Expert Panel are 1-1.5 g/kg ideal body weight (IBW) daily (Betsy Lehman Center Expert Panel, 2004). Some programs use adjusted body weight (ABW) rather than IBW to estimate macronutrient needs. High biological value protein sources such as meats (i.e. chicken, fish) and dairy (i.e. milk, cheese) are encouraged to ensure intake of all essential amino acids for the promotion of wound healing.

As patients become more weight stable, adequate protein intake continues to be acouraged for the preservation and restoration of lean body mass and increased satiety. As is widely accepted, adequate energy intake is required for positive nitrogen balance. It has been noted that protein intake and absorption are compromised due to gastric restriction and intestinal bypass. Decreased availability of pepsin, rennin, and hydrochloric acid might inhibit optimal protein digestion with subsequent intolerances (Moize V, Geliebter A, Gluck ME, Yahav E, Lorence M, Colarusso T, Drake V, Flancbaum L, 2003).

Protein malnutrition, particularly the hypoalbuminemic form, is of greatest concern following highly malabsorptive surgeries. Postoperative protein requirements have been estimated at 90 grams per day from high biological value sources (Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A, 1998).

#### iii. **Car**bohydrate

Carbohydrate intake is increased gradually postoperatively. Patients should be educated on the types and amounts of carbohydrate to consume using resources such as the ADA Exchange System (ADA, 1995). Due to limited evidence-based data on macronutrient needs following bariatric surgery, recommendations are usually based on the Dietary Reference Intakes (DRIs) of 2002 (IOM, 2002) and modified to individual need. A minimum of 100 grams of carbohydrate per day is recommended to prevent ketosis following the second postoperative month (Betsy Lehman Center Expert Panel, 2004). Nutrition education focusing on reading food labels is useful to help patients avoid foods containing large amounts of sugar. Consumption of such foods might lead to the rapid gastric emptying, diarrhea and gastrointestinal discomfort associated with dumping syndrome, discussed in greater detail later in this review. Patients should choose foods with less than seven grams of sugar per 100-kilocalorie serving. Furthermore, patients should be counseled on avoiding foods containing elevated levels of sugar alcohols due to their laxative effect (Betsy Lehman Center Expert Panel, 2004).

#### iv. Fat

The recommendations for fat intake usually change following most bariatric surgeries. Significant decreases in dietary intake usually occur up to one year **postoperatively** as a result of gastric restriction, potential food intolerances, and anatomic anomalies. After the first year, fat and carbohydrate intake usually increase while protein intake decreases, as patients begin to tolerate a greater variety of foods. Those patients who maintain higher protein intake and lower fat intake tend to consume fewer calories rendering a better weight loss. This suggests that weight loss efficiency following bariatric surgery might be, in part, associated with a decreased percentage of total energy from fat. Based on these results, dietary recommendations following bariatric surgery should not only focus on total energy intake but also on the distribution of calories from protein, carbohydrate, and fat. (Coughlin K, Bell RM, Bivins BA, Wrobel S, Griffen WO Jr., 1983).

Literature suggests that no more than 25% of total calories should come from fat for a healthy diet following bariatric surgery to promote better weight loss and weight maintenance (Mallory G, 1992). Small amounts of fat (10 grams) at each meal should be encouraged which will assist with maintaining gallbladder emptying and minimizing the risk of cholelithiasis, especially during rapid weight loss (Betsy Lehman Center Expert Panel, 2004). This is also recommended in patients who have had a cholecystectomy, to limit symptoms associated with fat malabsorption.

In order to provide a more balanced diet, adequate carbohydrate and fat are recommended with an emphasis on protein intake (Kenler HA, Brolin RE, Cody RP, 1990). However, insufficient data is available to provide specific evidence-based recommendations for energy and macronutrient intake. It is prudent that future research focus on the long-term nutritional needs following bariatric surgeries to help prevent weight recidivism and nutritional deficiencies.

#### **B.** Micronutrient Considerations

Micronutrient deficiencies might occur in all weight loss surgery patients regardless of procedure. This is due to reduction in overall food intake and decreased consumption of specific foods or classes of foods such as dairy and red meat. Because of the limited quantity of food consumed, nutrition support is focused on maximizing the quality of these foods (Collene AL, Hertzler SH, 2003). Bariatric surgeries, especially malabsorptive procedures, compromise micronutrient absorption as a result of the relatively low-acid environment of the gastric pouch, impaired binding to carrier proteins, insufficient capacity to liberate food-bound micronutrients, and decreased

bsorptive surfaces of the small bowel. For these reasons, weight loss surgery patients are at risk for several deficiencies including vitamin B<sub>12</sub>, folic acid, vitamin D and other fat-soluble vitamins, calcium, and iron (Elliot K, 2003; Betsy Lehman Center Expert Panel, 2004). All preoperative bariatric patients should undergo assessment of icronutrient status with repletion of any deficiencies. Since obesity is generally associated with normal or supranormal bone mineral density, routine preoperative bone density assessment does not appear to be needed; however, assessment might be advisable for post-menopausal women or others at high risk for osteoporosis. The following pages will discuss the primary micronutrient deficiencies with guidelines for prevention and treatment. (Betsy Lehman Center Expert Panel, 2004).

#### i. Iron

Iron is a trace mineral that is involved in oxidation and reduction activities involved in cellular respiration. Iron plays a primary role in red blood cell function, myoglobin activity, and the utilization of multiple heme and nonheme enzymes (Mahan K, Escott-Stump S, 2000). The average adult stores approximately one to three grams of iron with a depletion of about one milligram daily, primarily through the shedding of cells from the skin and lining of the intestinal mucosa. However, **thenstruating** women lose an average of two milligrams daily, increasing their risk for **the**-deficiency anemia (Betsy Lehman Center Expert Panel, 2004).

Bariatric surgeries that alter the gut physiology and bypass portions of the small intestine compromise digestion and absorption of iron leading to possible iron ficiency. Iron absorption occurs in the duodenum and proximal jejunum, which are

bypassed in the RYGBP and other malabsorptive procedures. Iron requires gastric acid, primarily hydrochloric acid of the stomach, for conversion to its soluble form. Following gastric restrictive intestinal bypass surgeries, both the acid environment of the stomach and available mucosal surface of the small intestine are altered increasing the risk for iron deficiency with or without anemia (Kushner R, 2000; Elliot K, 2003). Due to the small size of the gastric pouch and decreased acid production, many patients have difficulty tolerating meats containing heme iron, the most bioavailable form (Moize V, Geliebter A, Gluck ME, Yahav E, Lorence M, Colarusso T, Drake V, Flancbaum L., 2003)

Iron and ferritin should be measured and deficiencies corrected pre- and stoperatively for the prevention of iron deficiency and subsequent anemias. Serum iron levels tend to decline within the first year after surgery. Supplementation is recommended with a downward trend in serum iron levels to prevent the onset of irondeficiency anemia (Brolin RE, Gorman RC, Milgrim LM, Kenler HA, 1991). Due to declining iron storage over the long-term, lifelong monitoring is recommended. Patients presenting with iron deficiency should be instructed to take oral iron supplementation within a range of 180-220 milligrams of elemental iron daily for repletion of iron stores (Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LB, Kenler HA, Cody RP, 1998). Menstruating women should receive 40-65 milligrams of elemental iron prophylactically after gastric restrictive and intestinal bypass surgery. Iron sources such as ferrous sulfate, gluconate, or fumarate are usually recommended daily along with approximately 500 milligrams of vitamin C (Kushner R, 2000). Vitamin C enhances iron absorption due to its role in the conversion from

the ferric to the absorbable ferrous form. Iron supplements containing vitamin C are ideal for patients to prevent having to take additional supplements (Betsy Lehman Center Expert Panel, 2004).

## ii. Vitamin B12

The primary function of vitamin B12 is to promote normal metabolism of cells, primarily from the nervous system, gastrointestinal tract, and bone marrow (Mahan K, Escott-Stump S, 2000). Vitamin B12 deficiency is common following gastric restrictive intestinal bypass surgery.

Contributing factors to vitamin B12 deficiency include food restriction and reduced gastric hydrochloric acid, pepsin, and pancreatic enzymes leading to impaired release of protein-bound vitamin B12 from foods. Vitamin B12 deficiency might be, in part, a result of impaired secretion of intrinsic factor, impaired binding capacity of intrinsic factor with vitamin B12, and bacterial overgrowth as a result of reduced motility of the bypassed small intestine (Collene AL, Hertzler SH, 2003). The onset of this deficiency is usually after the first postoperative year because of enterohepatic recycling of vitamin B12 and the release of hepatic stores (Collene AL, Hertzler SH, 2003). Serum vitamin B12 levels will deplete rapidly without daily multivitamin supplementation and adequate dietary intake of vitamin B12. Patients should be counseled on increasing dietary sources of vitamin B12 such as dairy, meat, and eggs. pupplementation with crystalline vitamin B<sub>12</sub> found in multivitamins might prevent ficiency in some patients, and should be prescribed for all postoperative patients not identified as deficient. Vitamin B12 supplementation should be initiated in patients

presenting with suboptimal serum vitamin B12 levels. Oral vitamin B12 plementation of at least 350 micrograms per day is usually adequate; however, with severe deficiency, intramuscular vitamin B12 is recommended (Elliot K, 2003; Betsy Lehman Center Expert Panel, 2004).

#### iii. Folate

Folate, a B vitamin, is a coenzyme involved in the metabolism of nucleotides and amino acids (Mahan K, Escott-Stump S, 2000), and plays a primary role in the prevention of megaloblastic anemia. The jejunum is the primary site of folate absorption; however, some absorption occurs throughout the small bowel (Hunt SM, Groff JG, 1990). Approximately 20% of patients develop folate deficiency following gastric restrictive intestinal bypass surgery. Bacterial production of folate in the proximal large bowel might compensate for some of the decreased folate absorption (Collene AL, Hertzler SH, 2003; Kumpf VJ, 1996). Intake of high folate-containing food sources such as fortified grains and dark green leafy vegetables should be encouraged, when these foods are reintroduced into the diet. Prevention of deficiency can usually be achieved with daily multivitamin supplementation containing 200-400 micrograms of folate (Betsy Lehman Center Expert Panel, 2004).

#### iv. Thiamin

Thiamin deficiency is rare but has been reported following gastric restrictive intestinal bypass surgery (Deitel M, Cowan G, 2000). Thiamin plays an important role in energy transformation and neural function. Primary absorption occurs in the Jejunum, but thiamin can also be absorbed throughout the small intestine. Excessive vomiting, malnutrition and alcoholism are contributors to thiamin deficiency, otentially resulting in cranial nerve palsy, peripheral neuropathies, and the ocephalopathy associated with Wernicke-Korsakoff syndrome (Mahan K, Escott-Stump S, 2000). As with folate, deficiency of this B vitamin is most often prevented by daily multivitamin supplementation. Oral thiamin supplementation of oproximately ten milligrams daily is usually effective in correcting mild deficiency; however, severe deficiencies require intravenous infusion of 100 milligrams for seven to 14 days followed by ten milligrams daily until symptoms resolve (Kushner R, 2000). All patients should be monitored for thiamin deficiency, as patients without reoperative risk factors or concurrent alcohol abuse are at risk for deficiencies.

#### vi. Calcium

Calcium is a mineral essential for the health of bones and teeth, clotting of blood, hormone secretion and nerve function, and cardiac and skeletal muscle contraction (Hunt SM, Groff JG, 1990). Calcium absorption and utilization is affected by many factors. Reduced exposure to sunlight and diets high in oxalates found in some leafy green vegetables might result in compromised calcium absorption. Diets high in fat, protein, alcohol, and high doses of vitamin A decrease the bioavailability of dietary calcium. Caffeine and sodium, in large quantities, might increase urinary excretion of calcium (Charles P, 2003).

Patients undergoing weight loss surgeries are at increased risk for developing calcium deficiencies, potentially leading to metabolic bone disease. Contributing

factors include: decreased consumption of foods containing calcium and vitamin D; bypassing the duodenum and proximal jejunum, the primary sites for calcium absorption via active transport; and malabsorption of vitamin D (Elliot, 2003).

Most obese patients have a decreased risk for osteoporosis; however, bone mineral density tends to lessen with weight loss, and is usually proportional to the rate and degree of weight loss and dietary intake of calcium (Goode LR, Brolin RE, Chowdhury HA, Shapses SE, 2004; Brano G, Rodin DA, Pazianas M, Nussey SS, 1999). Gastric secretions are more acidic prior to surgery, facilitating optimal calcium absorption. Following gastric restrictive surgery, the acidic environment lessens due to the decreased volume of the gastric pouch, resulting in impaired calcium absorption. Calcium absorption can occur throughout the bowel via passive diffusion; however, the amount of dietary calcium must increase above the recommended allowances to achieve adequate absorption (Kushner R, 2000).

Calcium deficiency is more difficult to detect than vitamin and iron deficiencies. Serum levels are not indicative of calcium status, as multiple mechanisms, such as bone demineralization, exist to maintain serum calcium (Collene, AL, Hertzler, SH, 2003 ; Kushner, R, 2000). Serum calcium is bound to albumin and is found free-floating as ionized calcium. Calcium levels might be falsely depleted if serum protein levels are low. It is prudent to obtain ionized calcium levels in these **pircumstances** (Kushner R, 2000).

Markers for metabolic bone disease have been observed as early as three months after surgery, and include increased alkaline phosphatase levels, reduced serum calcium levels, and decreased 25 (OH) vitamin D levels (Coates PS, Fernstrom

JD, Fernstrom MH, Schauer PR, Greenspan SL, 2004). Hypocalcemia occurs more frequently with procedures inducing a greater degree of malabsorption. Serum calcium, phosphorus, alkaline phosphatase and 25 (OH) vitamin D levels should be periodically monitored postoperatively. Long-term consequences of altered calcium metabolism have not been well defined (Coates PS, Fernstrom JD, Fernstrom MH, Schauer PR, Greenspan SL, 2004). More research is needed to establish conclusive evidence with regard to bone health.

As a result of the potential for metabolic bone disease, the literature suggests lifelong daily supplementation of 1200-1500 milligrams of calcium citrate with vitamin D in divided doses, as calcium citrate is the most soluble form in the absence of gastric acid production (Kushner R, 2000). Supplemental calcium absorption is most often increased when taken with meals. However, as parathyroid hormonestimulated bone resorption is decreased nocturnally, calcium supplements may be taken at bedtime without regard to food (Charles P, 2003). Calcium and iron tend to compete for absorption, and therefore should be taken at least one hour apart.

#### vi. Vitamin D

Vitamin D is essential for the regulation of normal serum calcium levels and bone health. It is rapidly absorbed in the duodenum, but a greater amount is absorbed in the ileum with the aid of pancreatic and biliary secretions. Due to the bypassing of the duodenum and upper jejunum in gastric restrictive intestinal bypass surgeries, these secretions have less contact with food leading to malabsorption of vitamin D (Hunt, SM, Groff JG, 1990; Collene, AL, Hertzler SH, 2003). This is not seen in

restrictive bariatric surgeries. However, due to the decreased volume capacity of the gastric pouch and variable tolerances to milk-based products, dietary intake is usually not adequate. Compliance with taking multivitamins and calcium with vitamin D, providing approximately 800 International Units (IUs) protects against this deficiency (Dietel M, Cowan G, 2000). Supplementation for vitamin D deficiency ranges from 10,000-50,000 IUs weekly, tapering as indicated by laboratory indices. In highly malabsorptive surgeries, vitamin D supplementation of 400,000 IUs intramuscularly per month is recommended (Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A, 1998).

#### C. Hydration

Hydration guidelines are not well documented in the literature for bariatric surgeries. Hydration requirements are most often estimated by energy expenditure, age, and body weight. As an estimated 25-50% of excess weight is metabolically active tissue, it is prudent to use ABW when calculating daily fluid requirements as follows: (ABW = [actual + (actual-ideal)/4 (or 2 for 50%)]). Generally, an estimate of 50% is used for those patients presenting with a BMI greater than 50 kg/m<sup>2</sup> (The Science and Practice of Nutrition Support: A Case-Based Core Curriculum, 2001; Marotta RB, 1991).

Dehydration is commonly seen after gastric restrictive intestinal bypass surgeries. Obese patients require greater amounts of fluid than their non-obese counterparts to maintain normal fluid balance and homeostasis (Kim JJ, Tarnoff ME, Shikora SA, 2003). It is recommended that patients drink no more than eight fluid ounces of water per hour, at least during the first two months following bariatric surgery due to increased swelling of the gastric pouch. While on soft solid food, patients should wait approximately 30 minutes before and after eating three ounces or one cup of solid food to consume fluids. After beginning a low fat diet consisting of added fruits, vegetables, and starches, patients should wait approximately one hour after meals to drink fluids. Following this fluid intake schedule helps to prevent dumping syndrome and vomiting. Non-compliance usually leads to subsequent grazing between meals due to increased hunger (Elliot K, 2003) with decreased weight loss. It is imperative that clinicians promote and monitor for adequate hydration.

Following gastric restrictive procedures, consumption of carbonated beverages tend to result in gastric sequelae (Elliot K, 2003) such as cramping, bloating, nausea, and vomiting. It is recommended that patients abstain from the consumption of carbonated beverages, especially during the first six postoperative months. Caffeinated beverages such as coffee and tea should be avoided in the short-term following most procedures, especially restrictive procedures, due to the potential for increased gastric acid secretion with resulting gastroesophageal reflux (Boekema PJ, Samsom M, Smout AJ, 1999). Caffeine also induces increased urine output , which can increase risk for dehydration, particularly during rapid weight loss. Caffeinated beverages in moderation may be resumed if hydration is adequate, and the risk for dehydration minimal, as determined by laboratory indices and symptomatology (Betsy Lehman Center Expert Panel, 2004).

D. Dietary Advancement and Eating Behaviors Following Bariatric Surgery

The importance of thorough nutrition education for bariatric surgery patients cannot be overstated. Evidence-based guidelines for dietary advancement have not been established for all of the bariatric procedures currently in use. Recommendations for dietary advancement following bariatric surgery have been compiled based on out-gastrectomy nutritional guidelines. Close monitoring of dietary advancement and tolerance by a bariatric dietitian is recommended to avoid potential complications (Betsy Lehman Center Expert Panel, 2004).

Short-term postoperative nutritional considerations include adequate intake of protein and fluid and the prevention of vomiting and dumping syndrome. Dumping syndrome is characterized by rapid emptying of food into the jejunum increasing the osmolarity of intestinal contents with a subsequent influx of fluid into the small intestine. It is most common in the early postoperative period and becomes less prominent after the first postoperative year (Collene AL, Hertzler SH, 2003). There is great individual variability in the degree and severity of symptoms. This syndrome is most often precipitated by ingestion of food and beverages with high sugar content, and is prominent in gastric restrictive malabsorptive procedures such as the RYGBP. To avoid symptoms, patients should be instructed to avoid juices, soda, and other high Rgar-containing beverages (Collene AL, Hertzler SH, 2003; Elliot K, 2003). Sensitive individuals should avoid large doses of sugar alcohols; less than five grams per serving can be used as a guideline to avoid a laxative effect (Betsy Lehman Center Expert Panel, 2004).

Several stages of dumping syndrome have been observed. The first stage occurs within ten to 20 minutes after eating and usually involves abdominal bloating, Chycardia, diaphoresis, flushing, and lightheadedness. The intermediate stage occurs proximately 20 to more than 60 minutes following a meal and presents as abdominal bloating, cramping, flatulence, and diarrhea. Finally, late stage dumping occurs oneto-three hours following a meal and is more often a consequence of reactive or alimentary hypoglycemia. Symptoms of this stage most often include diaphoresis, nervousness, lethargy, shakiness, decreased concentration, and increased hunger (Mahan K, Escott-Stump S, 2000). Following gastric restrictive intestinal bypass surgeries, food and liquid intake is minimized due to the small size of the gastric pouch. Immediately postoperatively, the pouch has a capacity of approximately two ounces, but can increase anywhere from two and a half to nine ounces over time (Flanagan L, 1996). Due to the small pouch size, it is important for the patient and dietitian to focus on incorporating high-quality, nutrient-dense foods. Fluid and protein goals must be established in achievable, realistic amounts based on the reduced gastric capacity and early satiety of the bariatric surgery patient.

The bariatric surgery diet progresses in a series of stages beginning with liquids and progressively increases in texture. There is great variation among **programs** in the rate of diet advancement due, in large part, to individual differences in tolerance and readiness to advance (Elliot K, 2003; Marcasan W, 2004). The standard **Postoperative** diet consists of five to six stages, progressing over a two-month period.

In the immediate postoperative period, patients consume no food or fluid by mouth for approximately 24 hours, and are supported with intravenous solutions of

dextrose, saline or lactated ringers to provide adequate hydration. It is recommended that this therapy be continued until patients tolerate a full liquid diet for a minimum of 24 hours. Amino acid/saline intravenous solutions are sometimes provided; however, there is no literature indicating this therapy will result in preservation of visceral protein given the small amount of calories provided and the short duration of the therapy (Betsy Lehman Center Expert Panel, 2004).

Stage I of the diet is initiated approximately 24 hours after surgery, and consists of 30 mL per hour of water for a 24-hour period. If tolerated, patients are advanced to Stage II, consisting of no-added sugar clear liquids for another 24 hours. No-added sugar full liquids are provided in Stage III, for another 24 hours. In the absence of complications, patients are normally discharged after 24 hours on a Stage III diet. Postoperative outpatient follow up visits are scheduled with both the surgeon and the dietitian. At this time patients are instructed to begin the Stage IV high-protein soft-solid diet three weeks after surgery, as tolerated. Patients remain on the Stage IV diet for approximately one month. Advancement to the Stage V low-fat solid diet occurs, when the patient demonstrates tolerance to soft solids. Patients continue on the low-fat diet indefinitely with modifications made as needed (Betsy Lehman Center Expert Panel, 2004).

Although not well documented in the scientific literature, there appear to be common food intolerances. Typically, patients have difficulty tolerating milk, red meat, dry or tough poultry or pork, bread or doughy starches, and vegetables high in insoluble fiber (Collene AL, Hertzler SH, 2003). Over time, tolerance to these foods often improves. Tolerance to diet advancement is facilitated by eating slowly, chewing

foods thoroughly, avoiding tough-textured meats, and drinking a minimum of 30 minutes before or after meals. Postoperative vomiting is often subsequent to happropriate eating behaviors. However, vomiting can also be sign of an anatomical problem such as an anastomatic stricture or stenosis, which should be ruled out if vomiting persists. Intractable vomiting can potentially lead to serious nutritional sequalae such as protein energy malnutrition (PEM) and thiamin deficiency. PEM is more likely to occur in highly malabsorptive procedures such as biliopancreatic diversion (BPD) (Marcason W, 2004; Knol, JA, 1994). Appendix B illustrates a series of basic recommendations for each stage of the diet following most bariatric surgeries.

There is a need for long-term studies to determine the macro- and micronutrient requirements for patients following bariatric surgery for weight loss. Current recommendations are based on requirements for weight loss, and specific complications that arise from the various similar surgical procedures as highlighted earlier in this review. Dietitians play an important role as a member of the medical team to help prevent nutrient deficiencies, and maximize nutritional intake for improved tolerance while preserving the desired rate of weight loss. Familiarity with potential complications of each procedure can help the dietitian and patient prevent and, if necessary, treat any feeding difficulties.

#### E. Restrictive Surgeries: An Overview

General nutrition recommendations for restrictive surgeries are largely mechanical in nature. Patients are encouraged to consume small amounts of food, with some variability depending on the size of the pouch created. Typically, one cup of

food is well tolerated at each sitting. To prevent macro- and micronutrient efficiencies, small, frequent meals containing nutrient-dense foods are recommended. Patients are encouraged to chew food thoroughly, until the bolus is nearly liquefied, prior to swallowing. This will aid in digestion, and help to allow the food to pass through the small outlet from the stomach into the small intestine. Generally, micronutrient deficiencies are uncommon, as absorptive capacity is unchanged. If patients are unable to consume adequate calories to provide the recommended daily intake of micronutrients supplementation should be considered (MacDonald KG Jr, 2001).

Nausea, vomiting, bloating, and reflux are some of the complications that might result from inadequate chewing, and consuming portions of food that are too large for the pouch size created. These complications might compromise the integrity of the staple line, and contribute to band erosion in gastric band procedures (Sonnanstine TE, Kim JJ, Shikora SA, 2004). Record keeping is useful to help patients identify types and quantities of food, which might cause discomfort, distress, or intolerance. As with any bariatric surgery, patients are encouraged to avoid highcalorie liquids and concentrated sweets, which might slow the rate of weight loss, or even result in weight gain and displace nutrient-dense foods (Kenler HA, Brolin RE, Cody RP, 1990).

Postoperative onset of disordered eating can occur as a result of induced vomiting to relieve discomfort after overeating. Binge eating behavior can decrease the integrity of the gastric pouch, and increase the volume capacity, which allows for increased calorie ingestion with decreased weight loss efficiency. To avoid

discomfort, patients tend to consume soft, high-calorie foods and liquids such as ice cream, creamed soups, and juices. Increased gastric volume can lead to outlet obstruction, symptoms of which include vomiting and gastroesophageal reflux MacDonald KG Jr, 2001).

# F. Combination Surgeries: An Overview

## i Biliopancreatic Diversion (BPD) with and without Duodenal Switch

BPD combines partial gastrectomy with intestinal bypass, with the greatest impact of weight loss resulting from malabsorption. While the weight loss is greater than that seen in RYGBP, the surgery carries an increased risk of long-term nutritional complications. It has been postulated that modification of the length of the bypassed intestinal limb might allow for desired weight loss, while minimizing malabsorptive complications (Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A, 1998).

For up to one year postoperatively, dietary recommendations are similar to those described earlier in this review. Patients may return to preoperative eating habits within one year after BPD surgery without discomfort or impact on weight loss, due to the effect of intestinal adaptation (Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A, 1998). Fewer side effects have been demonstrated with the duodenal switch procedure, although the incidence of the most troublesome side effects of abdominal bloating and malodorous stool was relatively unchanged. The degree of micronutrient deficiency appears to be **decreased** with the duodenal switch approach, but it is recommended that patients be

monitored for these deficiencies (Marceau P, Hould FS, Simard S, Lebel S, Bourque RA, Potvin M, Biron S, 1998).

# G. Special Considerations

## i. Nutrition Support

Nutrition support guidelines following gastric restrictive intestinal bypass surgeries generally are the same as for the postoperative gastrointestinal surgery patient. Enteral nutrition may be utilized if a gastrostomy feeding tube was placed during the bariatric procedure. If enteral access is not available, and the patient is expected to be NPO more than seven days, total parenteral nutrition (TPN) may be initiated. Contraindications to TPN are usually the same as for gastrointestinal surgery patients in the presence of critical illness and sepsis. Bariatric surgery patients might be at risk for refeeding syndrome if they have been NPO or on clear liquids for as few as 48 hours prior to initiation of nutrition support. Judicious monitoring of electrolytes and fluid balance is recommended while advancing nutrition support. Guidelines for advancement have been established by the American Society for Parenteral and Enteral Nutrition (ASPEN) and others, and are appropriate for the bariatric surgery patient as well (The Science and Practice of Nutrition Support, 2001).

### ii. Pregnancy

Pregnancy following weight loss surgery is considered high risk and should be postponed for at least one year or until patients have reached a stable weight and nutritional status. Pre-pregnancy assessment and planning is prudent to identify and ameliorate micronutrient deficiencies prior to pregnancy. Prenatal vitamins are

preparation for pregnancy, and should continue throughout pregnancy (Wittgrove A, 1998; Preziosi P, 1997).

Iron requirements for women are greater in pregnancy and vary with each trimester. During the first trimester, iron needs are reduced as a result of amenorrhea. However, during the second trimester, as the plasma volume and red blood cell mass increases close to 50% and 40%, respectively, iron needs also increase. Bariatric surgery resulting in decreased intake of iron-containing foods due to intolerance and altered digestion and absorption require prudent monitoring during pregnancy to prevent the onset of iron-deficiency anemia. If patients present with iron deficiency with or without anemia, iron supplementation should be initiated. Ferrous fumerate is often tolerated and absorbed better by most patients following surgery (Wittgrove A, 1998).

To prevent the onset of iron deficiency, women should exhibit iron stores of more than 300 milligrams prior to pregnancy. Iron supplementation of 30 milligrams daily during the second trimester is recommended. However, 60 milligrams is recommended for women presenting with a pre-pregnancy iron deficiency. Iron supplements should be taken between meals to decrease gastric irritation and to increase absorption (Bothwell TH, 2000).

## H. Efficacy of Gastric Bypass Surgery

The RYGBP results in a weight loss of 65-80% of excess body weight at 1-1.5 years and 50-60% at five years after surgery. Given the efficacy of weight loss,

bariatric surgeries including the RYGBP are considered the most effective interventions for morbid obesity. Outcomes of the gastric bypass procedure not only include effective weight loss but also improve obesity comorbidities (Stocker DJ, 2003).

### i. Weight Reduction

The negative impact of obesity on the morbidity and mortality rates in the US cannot be ignored. Conservative approaches to promote weight loss have shown to be ineffective. Many traditional diets have promoted weight loss and subsequent recidivism. GBP improves both weight loss rates and degree of weight loss along with subsequent improvements in cardiovascular and metabolic sequelae (Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J, 2000). In a small number of clinical studies, it was reported that morbidly obese African American women lose weight less efficiently and show less improvement in blood pressure than their Caucasian counterparts following GBP (Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J, 2000).

GBP has been shown to promote a 66% loss of excess weight in patients one to two years following surgery, 60% at five years, and 50% at ten years (Sugerman HJ, 2001). Sugerman et.al. showed that African American patients lost less weight compared to Caucasian patients following the laparoscopic adjustable silicone gastric banding. The African American patients lost  $11.5\% \pm 18\%$  of their excess weight in contrast to Caucasians losing  $44\% \pm 26\%$  at two years following GBP (DeMaria EJ, Sugerman HJ, Meador JG, Doty JM, Kellum JM, Wolfe L, Szucs RA, Turner M, 2001). These racial disparities are important to acknowledge for targeting

promote better weight loss and long-term weight maintenance.

## ii. Comorbidities

Sugerman et.al. examined the relationship between diabetes and hypertension in a cohort of severely obese patients prior to and following GBP. The effects of race, age, gender, percent of excess weight loss, and the relationship of comorbidities such as obstructive sleep apnea and venous stasis were observed. The results suggested that the length of time with obesity was positively correlated with the incidences of diabetes and hypertension. The patients with a better resolution of hypertension and diabetes proved to have a greater percentage of excess weight loss and tended to be younger and Caucasian. African American patients did not lose weight as efficiently as Caucasians, 58 kg  $\pm$  16 and 69 kg  $\pm$  18, respectively (Sugerman HJ, Wolf LG, Sica DA, Clore JN, 2003). These ethnic differences are important to address to better promote weight loss and health outcomes following weight loss surgery, particularly among African Americans.

Studies have demonstrated significant improvements in cardiovascular risk factors and diabetes among Caucasian morbidly obese patients following GBP (Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjostrom CD, Sullivan M, Wedel H, 2004). Similar studies in other ethnic groups remain limited. This might be related to the less frequent use of bariatric surgery in minority populations (Choban P, Lu B, Flancbaum L, 2000; Livingston EH, Ko CY, 2004). Although there might be metabolic differences

between morbidly obese African Americans and Caucasians, behavioral factors such as dietary intake might also account for differences in weight reduction between African Americans and Caucasians (Foster GD, Wadden TA, Swain RM, Anderson DA, Vogt RA, 1999). Clarification of these differences is important for optimizing the post-surgical treatment of morbidly obese African Americans.

#### Conclusion

Presently there is limited data examining the differences in weight loss, dietary intake, and cardiovascular risk factors between African Americans and Caucasians following GBP. This research project looked at these differences and did find that African Americans lost less weight compared with Caucasians following GBP. Dietary intake appears to be non-contributory to these weight loss differences. However, despite smaller weight losses among African Americans, cardiovascular improvements were similar between each race. Further research is essential to better improve postoperative GBP outcomes among different racial groups.

#### Literature Cited

Allison DB, Fontaine KR, Manson JE, Stevens J, Vanltalle TB. Annual deaths attributable to obesity in the United States. JAMA. 1999; 282:1530-1538.

Betsy Lehman Center for Patient Safety and Medical Error Reduction-Expert Panel on Weight Loss Surgery (2004), retrieved on January 5, 2005, from. http://www.mass.gov/dph/betsylehman/index.htm.

Boekema PJ, Samsom M, Smout AJ. Coffee and gastrointestinal function: facts and fiction. A review. *Scand J Gastroenterol Suppl.* 1999; 230:35-9.

Bouchard C. Etiology of obesity: genetic factors. Arch Latinoam Nutr. 1992 Sep; 42 (3 Suppl): 127S-130S.

Bouchard C, Perusse L. Genetics of obesity. Annu Rev Nutr. 1993; 13:337-54.

Bouchard C, Tremblay A, Despres JP, Nadeau A, Dussault J, Moorjani S, Pinault S, Fournier G. The response to long-term overfeeding in identical twins. *N Engl J Med.* 1990.24; 322(21): 1477-82.

Brano G, Rodin DA, Pazianas, M, Nussey, SS. Reduced bone mineral density after surgical treatment for obesity. *Int J Obes Relat Metab Disord*. 1999; 23:361-5.

Bray GA. Use and abuse of appetite-suppressant drugs in the treatment of obesity. Ann Intern Med. 1993; 119:707-713.

Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LB, Kenler HA, Cody RP. Prophylactic iron supplementation after Roux-en Y gastric bypass: a prospective, double blind, randomized study. *Arch Surg.* 1998; 133(7): 740-744.

Brolin, RE, Gorman, RC, Milgrim, LM, Kenler, HA. Multivitamin prophylaxis in prevention of post-gastric bypass vitamin and mineral deficiencies. *Int J Obes*. 1991; 15:661-7.

Byrne KT. Complications of surgery for obesity. Surg Clin North Am. 2001; 81(5): 1181-93.

Charles P. Calcium absorption and calcium bioavailability. *J Internal Med.* 2003; 231:161-168.

Chitwood LF, Brown SP, Lundy MJ, Dupper MA. Metabolic propensity toward obesity in black vs white females: responses during rest, exercise and recovery. Int J Obes. 1996; 20: 455-462.

Choban P, Lu B, Flancbaum L. Insurance decision about obesity surgery: a new type of randomization? *Obes Surg.* 2000; 10(6): 553-6.

Coates PS, Fernstrom JD, Fernstrom MH, Schauer PR, Greenspan SL. Gastric bypass surgery for morbid obesity leads to an increase in bone turnover and a decrease in bone mass. *J Clin Endocrinol Metab.* 2004; 89: 1061-1065

Collene AL, Hertzler SH. Metabolic Outcomes of Gastric Bypass. Nutr in Clin Prac. 2003; 18:136-140.

Coughlin K, Bell RM, Bivins BA, Wrobel S, Griffen WO Jr. Preoperative and postoperative assessment of nutrient intakes in patients who have undergone gastric bypass surgery. *Arch Surg.* 1983; 188 (7): 813-816.

Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA*. 2005; 293(1): 43-53.

Deitel M, Cowan G S.M. Surgery for the morbidly obese patient. Lippincott Williams, and Wilken; 2000.

DeMaria EJ, Sugerman HJ, Meador JG, Doty JM, Kellum JM, Wolfe L, Szucs RA, Turner MA. High failure rate after laparoscopic adjustable silicone gastric banding for treatment of morbid obesity. *Ann Surg.* 2001; 233(6): 809-18.

Devlin MJ, Yanovski SZ, Wilson GT.Obesity: what mental health professionals need to know. *Am J Psychiatry*. 2000; 157(6): 854-66.

DiPietro L, Anda RF, Williamson DF, Stunkard AJ. Depressive symptoms and weight change in a national cohort of adults. *Int J Obes Relat Metab Disord*. 1992; 16(10): 745-53.

Drewnowski A. Obesity and the food environment: dietary energy density and diet costs. *Am J Prev Med*.2004.Oct; 27(3 Suppl): 154-62.

Drewnowski A. The role of energy density. Lipids. 2003;38(2):109-15.

Ekelund LG, Haskell WL, Johnson JL, Whaley FS, Criqui MH, Sheps DS. Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men. The Lipid Research Clinics Mortality Follow-up Study. *N Engl J Med.* 1988; 319(21): 1379-84.

Elliot K. Nutritional considerations after bariatric surgery. Critical Care Nursing Quarterly. 2003; 26(2): 133-8.

Exchange Lists for Weight Management. American Dietetic And Diabetes Associations, revised 1995.

Expert Panel. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Bethesda, MD: National Institute of Health, National Heart Lung, and Blood Institute, U.S. Department of Health and Human Services, Public Health Service, 1998.

Finer N, Bloom SR, Frost GS, Banks LM, Griffiths J. Sibutramine is effective for weight loss and diabetic control in obesity with type 2 diabetes: a randomised, doubleblind, placebo-controlled study. *Diabetes Obes Metab.* 2000; 2(2): 105-12.

Flanagan L. Measurement of Functional Pouch Volume following the Gastric Bypass Procedure. *Obes Surg*.1996; 6:38-43.

Flegal KM, Carroll MD, Ogen CL, Johnson CL. Prevalence and trends in overweight among US adults, 1999-2000. JAMA. 2002; 288 (14): 1723-1727.

French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. *Annu Rev Public Health*. 2001; 22:309-35.

Gannon, B, DiPietro L, and Poehlman, ET. Do African Americans have lower energy expenditure than Caucasians? *International Journal of Obesity*. 2000; 24:4-13.

Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. *Eur J Clin Nutr.* 1995; 49(1): 1-10.

Goode, LR, Brolin, RE, Chowdhury, HA, Shapses, SE. Bone and gastric bypass surgery: effects of dietary calcium and vitamin D. *Obes Res.* 2004; 12 (1): 40-47.

Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986-1990. *Arch Pediatr Adolesc Med.* 1996; 150(4): 356-62.

Gower, BA, Weinsier, RL, Jordan, JM, Hunter GR, and Desmond R. Effects of weight loss on changes in insulin sensitivity and lipid concentrations in premenopausal African American and white women. *Am J Clin Nutr.* 2002; 76:923-927.

Harnack LJ, Jeffery RW, Boutelle KN. Temporal trends in energy intake in the United States: an ecologic perspective. *Am J Clin Nutr.* 2000; 71(6): 1478-84.

Harris MI. Noninsulin-dependent diabetes mellitus in black and white Americans. *Diabetes Metab Rev.* 1990; 6:71-90.

Harris MI. Racial and ethnic differences in health care access and health outcomes for adults with type 2 diabetes. *Diabetes Care*. 2001; (3): 454-9.

Harris ML, Flegal KM, Cowie CC, Eberthardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: The Third National Health and Nutrition Examination Survey, 1988-94. *Diabetes Care*. 1998; 21:518-524.

Hauptman JB, Jeunet FS, Hartmann D. Initial studies in humans with the novel gastrointestinal lipase inhibitor Ro 18-0647 (tetrahydrolipstatin). *Am J Clin Nutr.* 1992; 55:309S-313S.

Hu FB, Li TY, Colditz GA, Willett WC. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA*. 2003; 9; 289(14): 1785-91.

Hunt, SM, Groff, JG. Advanced Nutrition and Human Metabolism. West Publishing Company; 1990

Hunter, GR, Weisnier, RL, Darnell, BE, Zuckerman, PA, Goran, MI. Racial differences in energy expenditure and aerobic fitness in premenopausal women. *Am J Clin Nutr.* 2000; 71:500-506.

James WP. A public health approach to the problem of obesity. Int J obes Rel Metab Disord. 1995; 19(suppl 3): S37-S45.

Kenler HA, Brolin RE, Cody RP. Changes in eating behavior after horizontal gastroplasty and Roux-en-Y gastric bypass. *Am J Clin Nutr*. 1990; 52:87-92.

Kim JJ, Tarnoff ME, Shikora SA. Surgical treatment for extreme obesity: Evolution of a rapidly growing field. *Nutr Clin Pract.* 2003; 18:109-123.

Kinra S, Nelder RP, Lewendon GJ. Deprivation and childhood obesity: a cross sectional study of 20,973 children in Plymouth, United Kingdom. *J Epidemiol Community Health.* 2000. 54(6): 456-60.

Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2004; 110(18): 2952-67.

Knol JA. Management of the problem patient after bariatric surgery. *Gastroenterol Clin Noth Am.* 1994; 23(2): 345-69.

Kumpf, VJ. Parenteral iron supplementation. Nutr Clin Pract. 1996; 11:139-146.

Kushner, R. Managing obese patient after bariatric surgery: A case report of severe malnutrition and review of the literature. *JPEN*. 2000 Mar-Apr; 24(2): 126-132.

Latner JD, Wetzler S, Goodman ER, Glinski J. Gastric bypass in a low-income, innercity population: eating disturbances and weight loss. *Obes Res.* 2004; 12(60): 956-961.

Lefebvre PJ, Scheen AJ. Obesity: causes and new treatments. *Exp Clin Endocrinol Diabetes*. 2001: 109(2): S215-S224.

Linquist, CH, Gower, BA, and Goran, MI. Role of dietary factors in ethnic differences in early risk of cardiovascular disease and type 2 diabetes. *Am J Clin Nutr.* 2000; 71:725-732.

Lissau I, Sorensen T. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet.* 1994; 343(8893):324-7.

Livingston EH, Ko CY. Socioeconomic characteristics in the population eligible for obesity surgery. Surgery. 2004; 135(3): 288-96.

Lovejoy JC, Champagne CM, Smith SR, de Jonge L, Xie H. Ethnic differences in dietary intakes, physical activity, and energy expenditure in middle-aged, premenopausal women: the Healthy Transitions Study. *Am J Clin Nutr.* 2001; 74(1): 90-5.

MacDonald Jr. KG. Bariatric Surgery: A Review. General Surgery News. 2001; 1-12.

Mahan, K, Escott-Stump, S. Krause's Food, Nutrition, & Diet Therapy. Ed L. WB Saunders Company A Harcourt Health Sciences Company. 2000 (10).

Mallory, G. Maximum nutrition, minimum calories. Obes Surg. 1992; 2:375-378.

Marcason W. What are the dietary guidelines following bariatric surgery? Journal of the American Dietetic Association. 2004; 104(3): 487-488.

Marceau P, Hould FS, Simard S, Lebel S, Bourque RA, Potvin M, Biron S. Biliopancreatic Diversion with Duodenal Switch. *World J. Surg.* 1998; 22:947-954.

Marotta RB, Floch MH. Diet and Nutrition in Ulcer Disease. *Med Clin North Am.* 1991 Jul; 75(4): 967-79.

Mason EE. Gastric surgery for morbid obesity. Surg Clin North Am. 1992; 72(2): 501-13.

Moize V, Geliebte A, Gluck ME, Yahav E, Lorence M, Colarusso T, Drake V, Flancbaum L. Obese patients have inadequate protein intake related to protein intolerance up to 1 year following Roux-en-Y gastric bypass. *Obes Surg.* 2003; 13:23-28.

Mun EC, Blackburn GL, Matthews JB. Current Status of Medical and Surgical Therapy for Obesity. *Gastroenterology* 2001;120:669-681.

National Academy of Sciences' Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids, 2002

Nelson TL, Hunt KJ, Rosamond, WD, Ammerman, AS, Keyserling, TC, Mokdad, AH, and Will, JC. Obesity and associated coronary heart disease risk factors in a population of low –income African –American and White women: The North Carolina WISEWOMAN Project. *Prevention Medicine*. 2002; 35:1-6.

NIH Conference Development Conference Panel: Gastrointestinal surgery for severe obesity. Ann Intern Med 115:956-961, 1991.

O'Connell TL. An overview of obesity and weight loss surgery. *Clinical Diabetes*. 2004; 22(3): 115-120.

Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study):15-year prospective analysis. *Lancet.* 2005; 365(9453): 36-42.

Preziosi P. Effect of iron supplementation on the iron status of pregnant women: consequence for newborns. *J Clin Nutr*. 1997; 66:1178-82.

Rolls BJ, Shide DJ, Thorwart ML, Ulbrecht JS. Sibutramine reduces foods intake in humans. *Am J Clin Nutr.* 1998; 6:1-11.

Ryan, AS, Nicklas, BJ, Berman, DM. Racial differences in insulin resistance and midthigh fat deposition in postmenopausal women. *Obes Res.* 2002; 10: 336-344.

Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J: Outcomes after laparoscopic Roux-En-Y gastric bypass for morbid obesity. *Ann Surg.* 2000; 232:515-529.

Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A.Scopinaro, N. Biliopancreatic Diversion. *World* J. Surgery. 1998; 22:936-946.

Sharp, TA, Bell, ML, Grunwald, GK, Schmitz, KH, Sidney S, Lewis, CE, Tolan, K, Hill, JO. Differences in resting metabolic rate between white and African-American young adults. *Obes Res.* 2002; 10:726-732.

Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjostrom CD, Sullivan M, Wedel H. Lifestyle,

diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004; 351(26): 2683-2693.

Sjostrom L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP, Krempf M. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *Lancet*. 1998; 352(9123): 167-72.

Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull*. 1989; 105(2): 260-75.

Sonnanstine TE, Kim JJ, Shikora SA. Surgical Options in the Treatment of Morbid Obesity: An Overview. *Support Line*. 2004; 26 (5): 3-17. Stocker DJ. Management of the bariatric surgery patient. *Endocrinol Metab Clin North Am*. 2003; 32(2): 437-57.

Strauss RS, Pollack, HA. Epidemic increase in childhood overweight, 1986-1998. JAMA. 2000; 286:2845-2848.

Sugerman HJ. Bariatric surgery for severe obesity. J Assoc Acad Minor Phys. 2001; 12(3): 129-136.

Sugerman HJ, Wolf LG, Sica DA, Clore JN. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg.* 2003; 237(6): 751-756.

Tanner, CJ, Barakat, HA, Lynis Dohm, G, Pories, WJ, MacDonald, KG, Cunningham, PRG, Swanson, MS, and Houmard, JA. Muscle fiber type is associated with obesity and weight loss. *Am J Physiol Endocrinol Metab.* 2002; 282:E1191-E1196.

The Science and Practice of Nutrition Support: A Case-Based Core Curriculum. Kendall/Hunt Publishing Company 2001.

Tremblay A, Despres JP, Maheux J, Pouliot MC, Nadeau A, Moorjani S, Lupien PJ, Bouchard C. Normalization of the metabolic profile in obese women by exercise and a low fat diet. *Med Sci Sports Exerc.* 1991; 23(12): 1326-31.

Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. *Int J Obes Relat Metab Disord*. 1996; 20:63-75.

Wadden TA, Berkowitz RI, Sarwer DB, Prus-Wisniewski R, Steinberg C. Benefits of lifestyle modification in the pharmacologic treatment of obesity: a randomized trial. *Arch Intern Med.* 2001; 161(2): 218-27.

Wadden, TA, Foster GD, Letizia KA. One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. *J Consult Clin Psychol*. 1994; 62:165-171.

Wardl J, Griffith J. Socioeconomic status and weight control practices in British adults. *Epidemiol Community Health.* 2001; 55(3): 185-90.

Washburn RA, Kline G, Lackland DT, Wheeler FC. Leisure time physical activity: are there black/white differences? *Prev Med.* 1992; 21:127–35.

Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity.*Am J Med.* 1998.105(2):145-50.

Weinsier RL, Hunter GR, Schutz Y, Zuckerman PA, Darnell BE. Physical activity in free-living, overweight white and black women: divergent responses by race to diet-induced weight loss. *Am J Clin Nutr*. 2002; 76(4): 736-42.

Weisner RL, Hunter GR, Zuckerman PA, Redden DT, Darnell BE, Larson DE, Newcomer BR, Goran MI. Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr.* 2000; 71:1138-46.

Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. *Int J Obes Relat Metab Disord*. 1993; 17(5): 279-86.

Wittgrove, A. Pregnancy following gastric bypass for morbid obesity. *Obes Surg.* 1998; 461-464.

World Health Organization: Obesity:preventing and managing the global epidemic: report of a WHO consultation. *World Health Org Tech Rep Ser.894.* 2000.

World Health Organization: Diet, nutrition and the prevention of chronic diseases Report of the joint WHO/FAO expert consultation. *World Health Org Tech Rep Ser* 916. 2003.

Wurtman RJ, Wurtman JJ. Brain serotonin, carbohydrate-craving, obesity and depression. Obes Res. 1995; 3 Suppl 4:477S-480S.

Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health*. 2002; 92(2): 246-9.

Zizza C, Siega-Riz AM, Popkin BM.Significant increase in young adults' snacking between 1977-1978 and 1994-1996 represents a cause for concern! *Prev Med.* 2001; 32(4): 303-10.

### Appendix B

# **Common Dietary Stages Following Bariatric Surgery**

# Stage I (does not include fluid provided by intravenous infusion)

Food Type: Water Duration: 0-1 day Begin: Postoperative day 1 End: Postoperative day 1 Amount: 1-2 oz/hr Fluid Goal: 14-32 oz/d (based on 14-16 waking hours) Protein Goal: 0 g/d

#### Stage II

Food Type: Clear liquids Non-caloric, decaffeinated, and noncarbonated beverages Duration: 1-2 days Begin: Postoperative day 1-2 End: Postoperative day 2-3 Amount: 2-3 oz/hr Fluid Goal: 32-48 oz/d (based on 14-16 waking hours) Protein Goal: 0 g/d

## **Other Suggestions:**

Decaffeinated, noncarbonated, sugar-free beverages

#### Stage III

Food Type: Full liquids
Liquids containing high biological value protein
Nonfat or low-fat milk, low-fat creamed soups, blended and
strained sugar-free pudding and custard, blended low-fat cottage
and ricotta cheese
Some protocols recommend adding additional protein to these drinks
through powdered skim milk or powdered egg white (approximately 1
Tbsp per serving)
Duration: 14-21 days/2-3 weeks
Begin: Postoperative day 2-4
End: Postoperative day 16-25/week 2.5-3.5
Amount: 3-6 servings of 4-8 oz/d
Fluid Goal: 48-72 oz/d, or based on estimated needs

(Cont) Protein Goal: 60-80 g/d, or based on estimated needs

# Other Suggestions:

Sip all liquids slowly, limit to approximately 8 oz/hr

## Stage IV

Food Type: Pureed/ground/diced foods

Generally foods allowed include only meat, poultry, fish, tofu and dairy products

Starches: fruits and vegetables are typically excluded from this stage although soft fruits and vegetables have been included in some protocols

Duration: 21-42 days/3-6 weeks

Begin: Postoperative day 17-26/week 2.5-3.5

End: Postoperative day 38-68/week 5.5-10

Amount: 2-3 oz of pureed/ground/diced food taken 3-5 times/d

Fluid Goal: 48-72 oz/d, or based on estimated needs

0-2 8-oz servings of full liquids (Drinks containing high biological value protein)

Protein Goal: 60-80 g/d, or based on estimated needs

### **Other Suggestions:**

Ground beef and shellfish might be difficult to tolerate Eat slowly: 10 minutes/oz Chew food thoroughly Drink fluids at least 1 hour after meals

### Stage V

Food Type: Small meals/snacks, consisting of low-fat solids Duration: Ongoing Begin: Postoperative day 38-68/week 4.5-10 End: Ongoing Amount: Typically based on the American Diabetes Association food exchange list. Number of servings tolerated is widely variable by individual.

## (Cont) <u>SAMPLE MEAL PLAN (recommendations might vary based on individual</u> <u>needs)</u>

Starch: 3-7 exchanges Fruit (soft): 2-3 exchanges Vegetable: 2-4 exchanges Milk: 1 exchange Meat: 6-9 exchanges

Fat: 1-5 exchange Fluid Goal: 48-72 oz/d, or based on estimated needs Protein Goal: 60-80 g/d, or based on estimated needs

#### **Other Suggestions:**

Make protein the priority food

Allow a minimum of 10 minutes per ounce of food, or 30 minutes per 3 ounces

Often hard starches such as toast and crackers are better tolerated than plain bread

Peel fresh vegetables and fruit when beginning to add in these foods

### Problematic foods typically include the following:

Red meat, ground beef, dry meats, shellfish, cores, hard skins (such as from an apple), seeds, membranes (particularly from thick-skinned fruits and vegetables), popcorn kernels, nuts, seeds, rice, and pasta

## (Above dietary stages adapted from the Betsy Lehman Center Expert Panel on Weight Loss Surgery Report)

#### Bibliography

- Allison DB, Fontaine KR, Manson JE, Stevens J, Vanltalle TB. Annual deaths attributable to obesity in the United States. JAMA. 1999; 282:1530-1538.
- Betsy Lehman Center for Patient Safety and Medical Error Reduction-Expert Panel on Weight Loss Surgery (2004), retrieved January 5, 2005, from http://www.mass.gov/dph/betsylehman/index.htm.
- Black AE, Prentice AM, Goldberg GR, Jebb SA, Bingham SA, Livingstone MB, Coward WA. Measurements of total energy expenditure provide insights into the validity of dietary measurements of dietary intake. *J Am Diet Assoc.* 1993; 93: 572-9.
- Boekema PJ, Samsom M, Smout AJ. Coffee and gastrointestinal function: facts and fiction. A review. *Scand J Gastroenterol Suppl.* 1999; 230:35-9.
- Bouchard C. Etiology of obesity: genetic factors. Arch Latinoam Nutr. 1992 Sep; 42(3 Suppl):127S-130S.
- Bouchard C, Perusse L. Genetics of obesity. Annu Rev Nutr. 1993; 13:337-54.
- Bouchard C, Tremblay A, Despres JP, Nadeau A, Dussault J, Moorjani S, Pinault S, Fournier G. The response to long-term overfeeding in identical twins. N Engl J Med. 1990.24; 322(21): 1477-82.
- Brano G, Rodin DA, Pazianas, M, Nussey, SS. Reduced bone mineral density after surgical treatment for obesity. *Int J Obes Relat Metab Disord*. 1999; 23:361-5.
- Bray GA. Use and abuse of appetite-suppressant drugs in the treatment of obesity. Ann Intern Med. 1993;119:707-713.
- Brolin RE, Gorman JH, Gorman RC, Petschenik AJ, Bradley LB, Kenler HA, Cody RP. Prophylactic iron supplementation after Roux-en Y gastric bypass: a prospective, double blind, randomized study. *Arch Surg.* 1998; 133(7): 740-744.
- Brolin, RE, Gorman, RC, Milgrim, LM, Kenler, HA. Multivitamin prophylaxis in prevention of post-gastric bypass vitamin and mineral deficiencies. Int J Obes. 1991; 15:661-7.
- Byrne KT. Complications of surgery for obesity. Surg Clin North Am. 2001; 81(5): 1181-93.

- Carpenter WH, Fonong T, Toth MJ, Ades PA, Calles-Escandon J, Walston JD, Poehlman ET. Total daily energy expenditure in free-living older African-Americans and Caucasians. *Am J Physiol*. 1998; 274(1 Pt 1): E96-101.
- Charles P. Calcium absorption and calcium bioavailability. J Internal Med. 2003; 231:161-168.
- Chitwood LF, Brown SP, Lundy MJ, Dupper MA. Metabolic propensity toward obesity in black vs white females: responses during rest, exercise and recovery. *Int J Obes.* 1996; 20: 455-462.
- Choban P, Lu B, Flancbaum L. Insurance decision about obesity surgery: a new type of randomization? *Obes Surg.* 2000; 10(6): 553-6.
- Coates PS, Fernstrom JD, Fernstrom MH, Schauer PR, Greenspan SL. Gastric bypass surgery for morbid obesity leads to an increase in bone turnover and a decrease in bone mass. *J Clin Endocrinol Metab.* 2004; 89: 1061-1065
- Collene AL, Hertzler SH. Metabolic Outcomes of Gastric Bypass. Nutr in Clin Prac. 2003; 18:136-140.
- Coughlin K, Bell RM, Bivins BA, Wrobel S, Griffen WO Jr. Preoperative and postoperative assessment of nutrient intakes in patients who have undergone gastric bypass surgery. *Arch Surg.* 1983; 188 (7): 813-816.
- Dansinger ML, Gleason JA, Griffith JL, Selker HP, Schaefer EJ. Comparison of the Atkins, Ornish, Weight Watchers, and Zone diets for weight loss and heart disease risk reduction: a randomized trial. *JAMA*. 2005; 293(1):43-53.
- Deitel M, Cowan G S.M. Surgery for the morbidly obese patient. Lippincott Williams, and Wilken; 2000.
- DeMaria EJ, Sugerman HJ, Meador JG, Doty JM, Kellum JM, Wolfe L, Szucs RA, Turner MA. High failure rate after laparoscopic adjustable silicone gastric banding for treatment of morbid obesity. *Ann Surg.* 2001; 233(6): 809-18.
- Devlin MJ, Yanovski SZ, Wilson GT.Obesity: what mental health professionals need to know. Am J Psychiatry. 2000; 157(6): 854-66.
- DiPietro L, Anda RF, Williamson DF, Stunkard AJ. Depressive symptoms and weight change in a national cohort of adults. *Int J Obes Relat Metab Disord*. 1992; 16(10):745-53.
- Drewnowski A. Obesity and the food environment: dietary energy density and diet costs. Am J Prev Med.2004.Oct; 27(3 Suppl): 154-62.

Drewnowski A. The role of energy density. Lipids. 2003; 38(2): 109-15.

- Ekelund LG, Haskell WL, Johnson JL, Whaley FS, Criqui MH, Sheps DS. Physical fitness as a predictor of cardiovascular mortality in asymptomatic North American men. The Lipid Research Clinics Mortality Follow-up Study. N Engl J Med. 1988; 319(21): 1379-84.
- Elliot K. Nutritional considerations after bariatric surgery. Critical Care Nursing Quarterly. 2003; 26(2): 133-8.
- Exchange Lists for Weight Management. American Diabetes and Dietetic Associations, revised 1995.
- Expert Panel. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Bethesda, MD: National Institute of Health, National Heart Lung, and Blood Institute, U.S. Department of Health and Human Services, Public Health Service, 1998.
- Finer N, Bloom SR, Frost GS, Banks LM, Griffiths J. Sibutramine is effective for weight loss and diabetic control in obesity with type 2 diabetes: a randomised, double-blind, placebo-controlled study. *Diabetes Obes Metab.* 2000; 2(2): 105-12.
- Flanagan L. Measurement of Functional Pouch Volume following the Gastric Bypass Procedure. *Obesity Surgery*. 1996; 6:38-43.
- Flegal KM, Carroll MD, Ogen CL, Johnson CL. Prevalence and trends in overweight among US adults, 1999-2000. *JAMA*. 2002; 288 (14): 1723-1727.
- Foster GD, Wadden T A, Swain RM, Anderson DA, Vogt RA. Changes in resting energy expenditure after weight loss in obese African American and white women. *Am J Clin Nutr.* 1999:69:13-17.
- Freedman DS, Srinivasan SR, Valdez RA, Williamson DF Berenson GS. Secular increases in relative weight and adiposity among children over two decades: the Bogalusa Heart Study. *Pediatrics*. 1997; 99: 420-426.
- French SA, Story M, Jeffery RW. Environmental influences on eating and physical activity. Annu Rev Public Health. 2001; 22:309-35.
- Friedewald WT, Levy RI, Frederickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of an ultracentrifuge. *Clin Chem.* 1972; 18:499-502.
- Gannon, B, DiPietro L, and Poehlman, ET. Do African Americans have lower energy expenditure than Caucasians? International Journal of Obesity. 2000; 24:4-13.

- Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. *Eur J Clin Nutr.* 1995; 49(1): 1-10.
- Geissler CA, Aldouri MSH. Racial differences in the energy cost of standardized activities. Ann. Nutr. Metab. 1985; 29: 40-47.
- Gidez LI, Miller GO, Burstein M. Separation and quantification of subclasses of human plasma high density lipoproteins by a simple precipitation procedure. J Lipid Res. 1982; 23:1206-1223.
- Goode, LR, Brolin, RE, Chowdhury, HA, Shapses, SE. Bone and gastric bypass surgery: effects of dietary calcium and vitamin D. Obes Res. 2004; 12 (1): 40-47.

Gortmaker SL, Must A, Sobol AM, Peterson K, Colditz GA, Dietz WH. Television viewing as a cause of increasing obesity among children in the United States, 1986-1990. *Arch Pediatr Adolesc Med.* 1996; 150(4): 356-62.

- Gower, BA, Weinsier, RL, Jordan, JM, Hunter GR, and Desmond R. Effects of weight loss on changes in insulin sensitivity and lipid concentrations in premenopausal African American and white women. *Am J Clin Nutr.* 2002; 76:923-927.
- Harnack LJ, Jeffery RW, Boutelle KN. Temporal trends in energy intake in the United States: an ecologic perspective. Am J Clin Nutr. 2000; 71(6): 1478-84.
- Harris MI. Noninsulin-dependent diabetes mellitus in black and white Americans. *Diabetes Metab Rev.* 1990; 6:71-90.
- Harris MI. Racial and ethnic differences in health care access and health outcomes for adults with type 2 diabetes. *Diabetes Care.* 2001; (3): 454-9.
- Harris ML, Flegal KM, Cowie CC, Eberthardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in US adults: The Third National Health and Nutrition Examination Survey, 1988-94. *Diabetes Care*. 1998; 21:518-524.
- Hauptman JB, Jeunet FS, Hartmann D. Initial studies in humans with the novel gastrointestinal lipase inhibitor Ro 18-0647 (tetrahydrolipstatin). *Am J Clin Nutr.* 1992; 55:309S-313S.
- Hu FB, Li TY, Colditz GA, Willett WC. Television watching and other sedentary behaviors in relation to risk of obesity and type 2 diabetes mellitus in women. *JAMA*. 2003; 9; 289(14): 1785-91.
- Hunt, SM, Groff, JG. Advanced Nutrition and Human Metabolism. West Publishing Company; 1990

- Hunter, GR, Weisnier, RL, Darnell, BE, Zuckerman, PA, Goran, MI. Racial differences in energy expenditure and aerobic fitness in premenopausal women. *Am J Clin Nutr.* 2000; 71:500-506.
- James WP. A public health approach to the problem of obesity. Int J obes Rel Metab Disord. 1995; 19(suppl 3): S37-S45.
- Kenler HA, Brolin RE, Cody RP. Changes in eating behavior after horizontal gastroplasty and Roux-en-Y gastric bypass. Am J Clin Nutr. 1990; 52:87-92.
- Kim JJ, Tarnoff ME, Shikora SA. Surgical treatment for extreme obesity: Evolution of a rapidly growing field. *Nutr Clin Pract.* 2003; 18:109-123.
- Kinra S, Nelder RP, Lewendon GJ. Deprivation and childhood obesity: a cross sectional study of 20,973 children in Plymouth, United Kingdom. *J Epidemiol Community Health.* 2000. 54(6): 456-60.
- Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism. *Circulation*. 2004; 110(18): 2952-67.
- Knol JA. Management of the problem patient after bariatric surgery. Gastroenterol Clin North Am. 1994; 23(2): 345-69.
- Kumanyika SK, Obarzanek E, Stevens VJ, Hebert PR, Whelton PK. Weight-loss experience of black and white participants in NHLBI-sponsored clinical trials. *Am J Clin Nutr.* 1991 Jun; 53(6 Suppl): 1631S-1638S.
- Kumpf, VJ. Parenteral iron supplementation. Nutr Clin Pract. 1996;11:139-146.
- Kushner, R. Managing obese patient after bariatric surgery: A case report of severe malnutrition and review of the literature. JPEN. 2000 Mar-Apr; 24(2): 126-132.
- Latner JD, Wetzler S, Goodman ER, Glinski J. Gastric bypass in a low-income, innercity population: eating disturbances and weight loss. *Obes Res.* 2004; 12(60): 956-961.
- Lefebvre PJ, Scheen AJ. Obesity: causes and new treatments. *Exp Clin Endocrinol Diabetes*. 2001: 109(2): S215-S224.
- Linquist, CH, Gower, BA, and Goran, MI. Role of dietary factors in ethnic differences in early risk of cardiovascular disease and type 2 diabetes. *Am J Clin Nutr.* 2000; 71:725-732.

- Lissau I, Sorensen T. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet.* 1994; 343(8893): 324-7.
- Livingston EH, Ko CY. Socioeconomic characteristics in the population eligible for obesity surgery. Surgery. 2004; 135(3): 288-96.
- Lovejoy JC, Champagne CM, Smith SR, de Jonge L, Xie H. Ethnic differences in dietary intakes, physical activity, and energy expenditure in middle-aged, premenopausal women: the Healthy Transitions Study. *Am J Clin Nutr.* 2001; 74(1): 90-5.
- Lovejoy JC, Smith SR, Rood, JC. Comparison of regional fat distribution and health risk factors in middle-aged white and African American women: the Healthy Transitions Study. *Obes Res.* 2001; 9:10-16.
- MacDonald Jr. KG. Bariatric Surgery: A Review. General Surgery News. 2001;1-12.
- Mahan, K, Escott-Stump, S. *Krause's Food, Nutrition, & Diet Therapy.* Ed L. WB Saunders Company A Harcourt Health Sciences Company. 2000 (10).
- Mallory, G. Maximum nutrition, minimum calories. Obes Surg. 1992; 2:375-378.
- Marcason W. What are the dietary guidelines following bariatric surgery? Journal of the American Dietetic Association. 2004; 104(3):487-488.
- Marceau P, Hould FS, Simard S, Lebel S, Bourque RA, Potvin M, Biron S. Biliopancreatic Diversion with Duodenal Switch. World J. Surg. 1998; 22:947-954.
- Marotta RB, Floch MH. Diet and Nutrition in Ulcer Disease. *Med Clin North Am.* 1991 Jul; 75(4): 967-79.

Mason EE. Gastric surgery for morbid obesity. *Surg Clin North Am.* 1992; 72(2): 501-13.

- Moize V, Geliebte A, Gluck ME, Yahav E, Lorence M, Colarusso T, Drake V, Flancbaum L. Obese patients have inadequate protein intake related to protein intolerance up to 1 year following Roux-en-Y gastric bypass. *Obes Surg.* 2003; 13:23-28.
- Mun EC, Blackburn GL, Matthews JB. Current Status of Medical and Surgical Therapy for Obesity. *Gastroenterology* 2001;120:669-681.
- Nelson TL, Hunt KJ, Rosamond, WD, Ammerman, AS, Keyserling, TC, Mokdad, AH, and Will, JC. Obesity and associated coronary heart disease risk factors in a

population of low –income African –American and White women: The North Carolina WISEWOMAN Project. *Prevention Medicine*. 2002; 35:1-6.

- National Academy of Sciences' Institute of Medicine. Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids, 2002.
- NIH Conference Development Conference Panel: Gastrointestinal surgery for severe obesity. Ann Intern Med 115:956-961, 1991.
- O'Connell TL. An overview of obesity and weight loss surgery. *Clinical Diabetes*. 2004; 22(3): 115-120.
- Pennington J.A.T. Bowes and Church's Food Values of Portion Commonly used.1989, 15<sup>th</sup> edition.
- Pereira MA, Kartashov AI, Ebbeling CB, Van Horn L, Slattery ML, Jacobs DR Jr, Ludwig DS. Fast-food habits, weight gain, and insulin resistance (the CARDIA study):15-year prospective analysis. *Lancet*. 2005; 365(9453):36-42.
- Preziosi P. Effect of iron supplementation on the iron status of pregnant women: consequence for newborns. *J Clin Nutr*. 1997; 66:1178-82.
- Price RA, Reed DR, Guido NJ. Resemblance for body mass index in families of obese African American and European American women. *Obes Res.* 2000; 8:360-366.
- Racette SB, Horowitz JF, Mittendorfer B,Klein S. Racial differences in lipid metabolism in women with abdominal obesity. *Am J Physiol Regulatory Integrative Comp Physiol*. 2000; 279:R944-R950.
- Residori L, Garcia-Lorda P, Flancbaum L, Pi-Sunyer X, Laferrere B. Prevalence of co-morbidities in obese patients before bariatric surgery: effect of race. *Obes Surg.* 2003; 13: 333-340.
- Rolls BJ, Shide DJ, Thorwart ML, Ulbrecht JS. Sibutramine reduces foods intake in humans. *Am J Clin Nutr*.1998; 6:1-11.
- Ryan, AS, Nicklas, BJ, Berman, DM. Racial differences in insulin resistance and midthigh fat deposition in postmenopausal women. *Obes Res.* 2002; 10: 336-344.
- Schauer PR, Ikramuddin S, Gourash W, Ramanathan R, Luketich J: Outcomes after laparoscopic Roux-En-Y gastric bypass for morbid obesity. *Ann Surg.* 2000; 232:515-529.
- Schoeller DA. Measurement of energy expenditure in free-living humans by using doubly labeled water. *J Nutr.* 1988; 118:1278–89.

- Scopinaro N, Adami GF, Marinari GM, Gianetta E, Traverso E, Friedman D, Camerini G, Baschieri G, Simonelli A.Scopinaro, N. Biliopancreatic Diversion. *World J. Surgery*. 1998; 22:936-946.
- Sharp, TA, Bell, ML, Grunwald, GK, Schmitz, KH, Sidney S, Lewis, CE, Tolan, K, Hill, JO. Differences in resting metabolic rate between white and African-American young adults. *Obes Res.* 2002; 10:726-732.
- Sjostrom L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren S, Larsson B, Narbro K, Sjostrom CD, Sullivan M, Wedel H. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004; 351(26): 2683-2693.
- Sjostrom L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP, Krempf M. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *Lancet.* 1998; 352(9123): 167-72.
- Sobal J, Stunkard AJ, 1989 Socioeconomic status and obesity: a review of the literature. *Psychol Bull*. 1989; 105(2): 260-75.
- Sonnanstine TE, Kim JJ, Shikora SA. Surgical Options in the Treatment of Morbid Obesity: An Overview. *Support Line*. 2004; 26 (5): 3-17.
- Stocker DJ. Management of the bariatric surgery patient. Endocrinol Metab Clin North Am. 2003; 32(2): 437-57.
- Strauss RS, Pollack, HA. Epidemic increase in childhood overweight, 1986-1998. JAMA. 2000; 286:2845-2848.
- Sugerman HJ. Bariatric surgery for severe obesity. *J Assoc Acad Minor Phys.* 2001; 12(3): 129-136.
- Sugerman HJ, Londrey GL, Kellum JM, Wolf L, Liszka T, Engle KM, Birkenhauer R, Starkey JV. Weight loss after vertical banded gastroplasty and Roux-en-Y gastric bypass for morbid obesity with selective versus random assignment. Am J Surg. 1989; 157:93-102.
- Sugerman HJ, Wolf LG, Sica DA, Clore JN. Diabetes and hypertension in severe obesity and effects of gastric bypass-induced weight loss. *Ann Surg.* 2003; 237(6): 751-756.
- Tanner, CJ, Barakat, HA, Lynis Dohm, G, Pories, WJ, MacDonald, KG, Cunningham, PRG, Swanson, MS, and Houmard, JA. Muscle fiber type is associated with

obesity and weight loss. *Am J Physiol Endocrinol Metab.* 2002; 282:E1191-E1196.

- The Science and Practice of Nutrition Support: A Case-Based Core Curriculum. Kendall/Hunt Publishing Company 2001.
- Tremblay A, Despres JP, Maheux J, Pouliot MC, Nadeau A, Moorjani S, Lupien PJ, Bouchard C. Normalization of the metabolic profile in obese women by exercise and a low fat diet. *Med Sci Sports Exerc.* 1991; 23(12): 1326-31.
- Troiano RP, Frongillo EA Jr, Sobal J, Levitsky DA. The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. *Int J Obes Relat Metab Disord*. 1996; 20:63-75.
- Wadden TA, Berkowitz RI, Sarwer DB, Prus-Wisniewski R, Steinberg C. Benefits of lifestyle modification in the pharmacologic treatment of obesity: a randomized trial. Arch Intern Med. 2001; 161(2): 218-27.
- Wadden, TA, Foster GD, Letizia KA. One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. *J Consult Clin Psychol*. 1994; 62:165-171.
- Wardl J, Griffith J. Socioeconomic status and weight control practices in British adults. *Epidemiol Community Health.* 2001; 55(3):185-90.
- Washburn RA, Kline G, Lackland DT, Wheeler FC. Leisure time physical activity: are there black/white differences? *Prev Med.* 1992; 21:127–35.
- Weinsier RL, Hunter GR, Heini AF, Goran MI, Sell SM. The etiology of obesity: relative contribution of metabolic factors, diet, and physical activity. *Am J Med.* 1998.105(2):145-50.
- Weinsier RL, Hunter GR, Schutz Y, Zuckerman PA, Darnell BE. Physical activity in free-living, overweight white and black women: divergent responses by race to diet-induced weight loss. *Am J Clin Nutr*. 2002; 76(4): 736-42.
- Weisner RL, Hunter GR, Zuckerman PA, Redden DT, Darnell BE, Larson DE, Newcomer BR, Goran MI. Energy expenditure and free-living physical activity in black and white women: comparison before and after weight loss. *Am J Clin Nutr.* 2000; 71:1138-46.
- Williamson DF, Madans J, Anda RF, Kleinman JC, Kahn HS, Byers T. Recreational physical activity and ten-year weight change in a US national cohort. *Int J Obes Relat Metab Disord*. 1993; 17(5): 279-86.
- Wittgrove, A. Pregnancy following gastric bypass for morbid obesity. *Obes Surg.* 1998; 461-464.

- World Health Organization: Obesity:preventing and managing the global epidemic: report of a WHO consultation. *World Health Org Tech Rep Ser.894.* 2000.
- World Health Organization: Diet, nutrition and the prevention of chronic diseases Report of the joint WHO/FAO expert consultation. *World Health Org Tech Rep Ser 916.* 2003.
- Wurtman RJ, Wurtman JJ. Brain serotonin, carbohydrate-craving, obesity and depression. Obes Res. 1995; 3 Suppl 4:477S-480S.
- Young LR, Nestle M. The contribution of expanding portion sizes to the US obesity epidemic. *Am J Public Health*. 2002; 92(2): 246-9..
- Zizza C, Siega-Riz AM, Popkin BM.Significant increase in young adults' snacking between 1977-1978 and 1994-1996 represents a cause for concern! *Prev Med.* 2001; 32(4): 303-10.