A Descriptive Study of Body Composition Abnormalities and Health Risks in Patients with Obesity

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A DESCRIPTIVE STUDY OF BODY COMPOSITION ABNORMALITIES AND HEALTH RISKS IN PATIENTS WITH OBESITY

By

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LIST OF ABBREVIATIONS

ASM: Appendicular Skeletal Muscle
BIA: Bioelectrical Impedance Analysis
BMI: Body Mass Index
BCM: Body Cell Mass
CRP: C-Reactive Protein
CT: Computerized Tomography
CV: Coefficient of Variance
DXA: Dual Energy X-Ray Absorptiometry
FFM: Fat-Free Mass
FFMI: Fat-Free Mass Index
FM: Fat Mass
FMI: Fat Mass Index
GH: Growth Hormone
HR: Hazard Ratio
IGF-1: Insulin-Like Growth Factor 1
IL: Interleukin
LBM: Lean Body Mass
LST: Lean Soft Tissue
MRI: Magnetic Resonance Imaging
NHANES: National Health and Nutrition Examination Survey
REE: Resting Energy Expenditure
SMI: Skeletal Muscle Index
**TNF-α:** Tumor Necrosis Factor

**WHO:** World Health Organization
DEFINITION OF TERMS

Adipose Tissue: A connective tissue formed by adipocytes, collagenous and elastic fibers, fibroblasts and capillaries. There are four types of adipose tissue: subcutaneous, visceral, interstitial and yellow marrow.

Appendicular Skeletal Muscle Mass (ASM) Index: The sum of skeletal muscle mass from arms and legs adjusted by height in meters squared (kg/m²).

Body Cell Mass (BCM): Body composition compartment consisting of all intracellular components, including intracellular water.

Fat-Free Mass (FFM): The sum of lean tissues of the body including bone mineral content, therefore it includes: total body water, total body protein, carbohydrate, non-fat lipid, soft tissue minerals and bone mineral content.

Lean Body Mass (LBM) or Lean Soft Tissue (LST): the sum of lean compartments of the body (excluding bone mineral content), it includes: total body water, total body protein, carbohydrate, non-fat lipid and soft tissue minerals.
ABSTRACT

**Background:** Body composition abnormalities are independent predictors of health outcomes in a variety of disease states. The simultaneous condition of low muscle mass and high fat mass, termed sarcopenic obesity, is an abnormal body composition phenotype associated with metabolic abnormalities and comorbidities.

**Objectives:** The purpose of this study was to investigate the overall body composition variability among obese patients, and to compare health characteristics between sarcopenic obese and non-sarcopenic obese patients.

**Methods:** In this retrospective, chart review study, patients (≥18 years old) seeking weight loss treatment at a local center in Tallahassee, FL and with available baseline bioelectrical impedance analysis (BIA) data were included in this study. The ratio between fat mass index [FMI, defined as fat mass (kg)/height (m)²] and fat free mass index [FFMI, defined as fat free mass (kg)/height (m)²] were used to define sarcopenic obesity. Medical records were further reviewed for information on metabolic profile and health status.

**Results:** Ninety-one obese patients with a mean age of 57 ± 11 years were included in this study. Body mass index (BMI) ranged from 31.6 to 68.7 kg/m². Eighty-one percent were morbidly obese. The FMI/FFMI ratio was variable ranging from 0.35 to 2.46 kg/m², independent of body weight. A gender-specific FMI/FFMI ratio above the median was used to depict the sarcopenic obesity phenotype. This corresponded to: FMI/FFMI ≥ 1.05 kg/m² in women and FMI/FFMI ≥ 0.78 kg/m² in men. As expected, men presented with higher body weight, height, waist circumference and FFMI, compared to women. On the contrary, women presented with higher percent body fat and FMI/FFMI ratio. No gender differences were observed for body mass index (BMI), fat mass and FMI. Plasma albumin concentration was lower in sarcopenic obese patients compared to non-sarcopenic obese patients (P=0.032). Sarcopenic obese patients reported a higher prevalence of low back pain compared to their counterparts. In fact, sarcopenic obesity was the strongest predictor of low back pain, with an odds ratio of 2.3 (95% CI=1.01-5.41, P=0.048). Similarly, the prevalence of alcoholism and sexual dysfunction were significantly greater among sarcopenic obese patients compared to non-sarcopenic obese patients (P=0.026 and P=0.030, respectively).
Conclusion: A wide variability in body composition was observed in this cohort of patients, illustrating how the proportions of fat to fat-free tissues may differ among patients with similar BMI. We suggest the use of the FMI/FFMI ratio as a potential approach for the assessment of sarcopenic obesity in patients with severe obesity. Using this approach, patients with a sarcopenic obesity phenotype presented with higher risk of certain metabolic abnormalities and comorbidities.
CHAPTER ONE
INTRODUCTION

1.1 Background

Body composition refers to the amounts/proportions of fat and lean tissues in the body. New in vivo technologies to assess human body composition have rapidly developed in the past decades, such as bioelectrical impedance analysis (BIA), dual X-ray absorptiometry (DXA), and computerized-tomography (CT) image analysis. These techniques have allowed for the identification of abnormal body composition phenotypes, which are associated with increasing health risks, such as physical frailty (1), functional limitations (2) and mortality (3). Abnormal body composition phenotypes include sarcopenia (deficiency of muscle mass and/or strength), obesity (excess adipose tissue) and sarcopenic obesity, which is defined as being sarcopenic and obese simultaneously (4).

Of particular interest is sarcopenic obesity, which is difficult to detect since BMI (body mass index = body weight/ height$^2$) only provides an assessment of the patient’s overall body weight rather than the composition of that body weight (i.e. body composition) (5). Although obesity is commonly assessed body weight and BMI, more sophisticated tools are needed to assess the presence of sarcopenia. Sarcopenia is a common age-related disease, which is characterized by gradual loss of muscle mass and decline of muscle function (6-8). One of the most commonly accepted definitions of sarcopenia is a level of skeletal muscle mass lower than 2 standard deviations (SD) below the mean of a young reference population (9). The National Institutes of Health and the World Health Organization (WHO) defines obesity as having a BMI of 30 kg/m$^2$ or higher (10).

The prevalence of sarcopenic obesity has been reported to range from 2% among elderly individuals aged 60 to 69 years (4) to up to 90% among women aged 18 to 87 years (11). Nonetheless, the prevalence of sarcopenic obesity has not been well characterized for several reasons such as different definitions, different populations and reference groups being studied, and various measurements of health outcomes. However, the prevalence of sarcopenic obesity is rising because our population has continued to get older and more obese (12).
At present, the pathogenesis of sarcopenic obesity is not clearly understood. Possible factors may include a combination of aging, hormone changes, pro-inflammatory factors and reduced dietary intake, especially protein intake (13). Roubenoff (14) has proposed a schema, identifying possible cyclic metabolic behaviors: the accumulation of adipose tissue leads to loss of muscle mass by increasing circulating pro-inflammatory cytokines and insulin resistance; and in turn, muscle loss reinforces fat gain by impairing the ability of physical activity.

Various published studies suggest that sarcopenic obesity is the worst-case scenario for health risks and has been associated with poorer functional status and health outcomes compared with sarcopenia or obesity alone (9, 15). Sarcopenic obesity has been associated with physical disability (16, 17), functional impairment (18), increased cardiovascular disease risk (19), and reduced survival (5).

1.2 Purpose of Study

The purpose of the present study was two-fold: to investigate the overall body composition variability among obese patients seeking treatment at the Tallahassee Memorial Bariatric Health Center (TMBC) and to compare the demographic and clinical characteristics between sarcopenic obese and non-sarcopenic obese patients.

1.3 Research Hypotheses

1.3.1 Hypothesis 1

The distribution of FMI/FFMI ratio will present with a degree of variability greater than two folds.

1.3.2 Hypothesis 2

Compared to non-sarcopenic obese patients, sarcopenic obese patients:
a) will present with higher rates of metabolic abnormalities and co-morbidities, as assessed by the TMBC New Patient Questionnaire:
   - higher prevalence of metabolic syndrome.
- lower levels of albumin, creatinine, total protein, vitamin D, TSH, T3, T4, AST and ALT.
- higher prevalence of co-morbidities (acid reflux, arthritis, asthma, alcoholism, cancer, diabetes, diminished hygiene, fatigue, gout, heart disease, joint pain, nausea, osteoporosis, sexual dysfunction, shortness of breath, sleep apnea, thyroid disease, and weakness).
- higher prevalence of immobility and low back pain.

b) will report lower physical activity readiness and levels:
- at least one impediment to becoming physically active on the PAR-Q & YOU Questionnaire.
- lower frequency of physical activities as reported by the Exercise History and Attitude Questionnaire.
CHAPTER TWO
LITERATURE REVIEW

2.1 Definition of Sarcopenia

The concept of sarcopenia was originally proposed by Rosenberg in 1989 (8). Sarcopenia was characterized by an involuntary age-related decline in lean body mass (8). More recently, the European Working Group on Sarcopenia in Older People suggested including reduced muscle strength in the definition of sarcopenia rather than considering low muscle mass alone (6). To date, most studies of sarcopenia were conducted among elderly populations. Considering the increased lifespan and progressive muscle atrophy with aging, a remarkable number of studies have been focusing on the prevalence of this syndrome and its associations with health. However, there has not been a standard diagnostic criterion to define sarcopenia due to different populations, reference groups, and measurement methods being used (6).

The most commonly used definition of sarcopenia was developed by Baumgartner et al. (9) based on data from the New Mexico Elderly Health Survey, conducted from 1993 to 1995. The investigators defined sarcopenic individuals as those having a height-adjusted appendicular skeletal muscle mass (ASM, the sum of lean tissues from arms and legs assessed by DXA) lower than 2SD below the mean of young reference adults (9). Yet, this definition has been criticized for not taking body fat into consideration, which leads to an underestimation of sarcopenia among obese subjects (20). In order to control for body fat, Newman et al. (20) used ASM adjusted for height and body fat mass to define sarcopenia. To estimate the prevalence of sarcopenia, Janssen et al. (18) developed a similar approach based on BIA-derived skeletal muscle mass. Muscle mass was then divided by body weight and expressed as a percentage of skeletal muscle index (SMI= skeletal muscle/body weight × 100). Two levels of sarcopenia were identified: Class I (if SMI was between one and two SD below the young reference mean value) and Class II (if SMI was lower than 2SD below the young reference mean value). Class I and class II sarcopenia were identified as follows: men 37% to 31%, and less than 31%; women 28% to 22%, and less than 22% (18). Later on, Janssen and colleagues identified two levels of sarcopenia cutoff values in older individuals based on the association between sarcopenia and physical disability risk. The height-normalized skeletal muscle mass (SM/height$^2$) cutoffs within
5.76 to 6.75 kg/m\(^2\) or within 8.51 to 10.75 kg/m\(^2\) were selected to define moderate risk physical disability in women and men respectively, whereas cutoffs lower than 5.75 kg/m\(^2\) or 8.50 kg/m\(^2\) were selected to define high risk physical disability in women and men respectively (17).

### 2.1.1 Prevalence of Sarcopenia

The variation in diagnostic criteria leads to different prevalence rates of this disordered body composition phenotype (21). Nevertheless, there is consensus that the occurrence of sarcopenia is increasing with age and usually associated with reduced muscle strength (6). Baumgartner et al. (9) reported that more than 50% elderly individuals (≥80 years) were sarcopenic in the New Mexico Aging Process Study. Janssen et al. (18) analyzed a population-based data set from the Third National Health and Nutrition Examination Survey (NHANES III) and reported a prevalence of sarcopenia of approximately 59% in women and 47% in men, aged 60 to 69 years. Additionally, prevalence rates increased from the 3\(^{\text{rd}}\) to the 6\(^{\text{th}}\) decade of life. More recently, the prevalence of sarcopenia has also been reported among different ethnicities. Masanes and colleagues examined a cohort of healthy community-dwelling elderly in Spain and reported sarcopenia prevalence rates of 33% in women and 10% in men, aged 70 to 80 years (22). Using data from communities in Taipei, China, Chien and colleagues reported sarcopenia prevalence rates of 18.6% and 23.6% in elderly women and men respectively (23). The prevalence of sarcopenia was estimated at 23.21% among an elderly women cohort (≥60 years) in Brazil (24). Although in most cases sarcopenia has been defined as a decline in muscle mass for epidemiologic purposes, reduction in muscle strength and muscle efficiency (muscle strength per unit of muscle mass) have also been characteristic features of sarcopenia (25).

### 2.1.2 Mechanisms of Sarcopenia

A number of underlying mechanisms have been proposed to cause this loss of muscle mass/strength, including changes in muscle structure and composition, metabolism and lifestyle factors. Muscle contributes approximately 40% of the total body mass and 75% of the body’s cell mass (BCM, body composition compartment consisting of all intracellular components, including intracellular water), which is the metabolic active compartment of the body (26).
Muscle changes associated with aging include a disproportionate loss of fast myosin heavy-chain (MHC) and actin, which are key contractile proteins (25-27). The ability to synthesize muscle contractile proteins is directly associated with muscle strength (28). Progressive loss of motor neurons, which stimulates muscle contraction; and losses of motor neurons from the spinal cord may play a role in the development of sarcopenia (29). Oxidative stress and molecular inflammation also play important roles in age-related muscle protein breakdown, modulating transcription factors and kinases, and contributing to sarcopenia (30).

Furthermore, several studies have confirmed an age-related decline in muscle fiber size and number, especially pointing towards fast-twitch type II fibers which are predictive of muscle strength and power (31, 32). “The motor unit remodeling” can also be interpreted as a process that the surviving neurons adopt muscle fibers as a compensation for dropout of motor neurons (33). Considering motor unit remodeling, older adults were reported to have larger motor units than young adults; nevertheless, the remodeled motor units are less efficient in stimulating fiber function and in extreme cases can cause tremor or weakness (34). In addition to changes of muscle mass and structure, alterations in muscle composition also contribute to the loss of muscular strength. With the use of CT, an increase in connective tissue and fat tissue within the muscle has been observed among sarcopenic subjects (35).

There is evidence that endocrine function deterioration with aging is associated with the loss of muscle mass and quality. Many hormones are involved in the regulation of protein metabolism throughout lifetime, such as testosterone, estrogen, growth hormone (GH) and insulin-like growth factor-1 (IGF-1). As people age, testosterone production reduces, speeding up the loss of muscle mass (36). As an anabolic hormone, testosterone treatments have been associated with increased muscle mass, even though the effects on muscle strength are contradictory (37). IGF-1 and GH have an anabolic effect on muscle protein synthesis (38). Notably, circulating IGF-1 level is correlated with MHC synthesis rate (28). Therefore, the decrease of these hormones with aging contributes to muscle loss or lack of maintenance (39). The increase of inflammatory cytokines among elderly individuals is another complicating factor that impetuses muscle atrophy. A high level of interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-a (TNF-a) is correlated with increased muscle catabolism (13).

Malnutrition is also a crucial risk factor in the development of sarcopenia in elderly populations. Reduced energy intake leads to muscle atrophy with reduced muscle contraction.
and metabolism (40). A lack of adequate amino acids intake will inhibit protein synthesis affecting muscle anabolism and muscle strength increments (41). It is well established that Vitamin D deficiency is also associated with low muscle mass and strength (40). Physical inactivity plays an important role in the decline of muscle quantity and quality (33). In addition, increased physical activity has multifactorial benefits on muscle function and performance. Exercise training exerts a direct effect on several signaling pathways which improves anabolic activities (42). It is suggested that exercise training is associated with improvements in insulin sensitivity and increased lipid oxidation (43). Resistant training has been demonstrated to increase muscle strength and size in older adults and aerobic exercise may also has an anabolic effect on skeletal muscle (44).

2.1.3 Health Consequences of Sarcopenia

Sarcopenia was reported to be associated with impaired physical function, physical disability, low quality of life and increased mortality rate in numerous studies. In 1998, Baumgartner et al. investigated the health problems associated with sarcopenia. In this study, sarcopenia was an independent predictor of self-reported physical disability, after controlling for age, ethnicity, income, comorbidity, physical activity, alcohol intake and smoking (9). Visser et al. (45) conducted a prospective study to examine whether low muscle mass and strength were predictive of incident mobility limitation in persons aged 70 to 79 years. After 2.5 years follow up, males and females in the lowest quintile of mid-thigh muscle cross-sectional area were respectively 90% and 68% more likely to develop mobility limitations compared to the highest muscle size quintile. In addition, the risk of developing mobility limitations in the lowest quintile of muscle strength was 2.64 and 2.15 times greater for males and females, respectively (45). Along with these findings, Janssen (46) investigated the influence of sarcopenia on the development of physical disability among elderly individuals at both cross sectional and longitudinal levels. Muscle mass was categorized into normal, moderate sarcopenia, and severe sarcopenia in this study. At baseline, 71.5% of males had moderate sarcopenia and 15.8% had severe sarcopenia, while the prevalence of moderate and severe sarcopenia were of 44.1% and 9.3% among females respectively. Those in the severe sarcopenia groups were found to have the greatest likelihood of disability with stronger associations observed in males compared to
females (46). However, when compared to the longitudinal analysis, the influence of sarcopenia on disability was considerably stronger in the cross-sectional analysis (46). Sarcopenia has also been associated with increased risk of overall death. Recently, Landi et al. (1) analyzed the relationship between sarcopenia and mortality using data from the Aging and Longevity Study, a prospective cohort study conducted among people over 80 years old. Sarcopenic patients had a greater risk of death for all causes than non-sarcopenic subjects (HR =2.32, CI =1.01 to 5.43) (1).

In addition to clinical problems, sarcopenia has also been associated with increased public health expenditures. The estimated healthcare cost attributable to sarcopenia in the United State in 2000 was $18.5 billion, which represented about 1.5% of total healthcare expenditures for that year (47).

2.2 Definition of Obesity

According to the WHO, BMI can be categorized into five strata of height-adjusted body weights: ≤18.5 kg/m² for underweight, 18.5-24.9 kg/m² normal weight, 25.0-29.9 kg/m² overweight, ≥30.0 kg/m² class I obesity, 35.0-39.9 kg/m² class II obesity, and ≥40.0 kg/m² morbid obesity (10). Therefore, obesity is defined as BMI equal or greater than 30 kg/m². Body fat percentage has also been used to define obesity and several studies have reported a high correlation between BMI and percent body fat (48, 49). “Healthy” ranges of percent body fat are approximately 12% to 20% for men and 20% to 30% for women (50). The WHO defines obesity as having percent body fat greater than 25% in males or 35% in females. These values were established based on a BMI value of 30 kg/m² (51). Adipose tissue distribution also has a great impact on health outcomes. Particularly, abdominal or upper-body obesity has been associated with a number of metabolic abnormalities and diseases, such as increased blood pressure, and insulin resistance (52, 53).

2.2.1 Prevalence of Obesity

The prevalence of obesity has been dramatically rising in the United State in the last three decades. According to NHANES data, the prevalence of obesity showed a large increase between NHANES II and NHANES III (NHANES I, 14.1%; NHANES II, 14.5% and NHANES III 22.5%) (54). Approximately 36% of American adults were obese in 2009 to 2010, and the
prevalence significantly increased with age among women but not men (55). Likewise, a high prevalence of obesity is observed among elderly individuals (aged 60 or older) (56). More than one-third of older adults aged 65 and over were obese in 2007–2010 (57). By using values greater than the median percent body fat for each sex (> 27% in men and > 38% in women), Baumgartner et al. (4) reported a prevalence of obesity of 43.5% among elderly individuals (≥ 60 years old).

2.2.2 Mechanisms of Obesity

The etiology of obesity is multifactorial. Several factors have contributed to this syndrome, such as demographic, social economic status, lifestyle and biological factors. Among them, metabolic factors, diet and physical inactivity are believed to play a major role in the development of obesity (58). However, ultimately, excess adipose tissue is caused by an imbalance between energy intake and energy expenditure (59).

Both genetic and environmental factors are involved in the pathophysiology of excess body fat storage. Heredity accounts for 30% to 40% of the variations in BMI while environment contributes 60% to 70% (53). People with “obese genes” are only inherited with a tendency to be obese but not destined to be obese. Evidence comes from a research conducted among obese individuals, in collaboration with their family members, indicating that genes explained 25% to 40% of the individual differences in BMI (60). However, genetic predisposition itself cannot explain why the tendency of obesity is more prevalent today than it was 30 years ago. Lifestyle changes including adaptation to increased energy-dense food intake and sedentariness, and lack of drive for physical activity are all thought to interact with genes and contribute to the dramatic increase in the prevalence of obesity in the past two decades (61). Nevertheless, the influence of energy intake on obesity rates seems paradoxical. Evidence from the U.S. Department of Agriculture National Wide Food Consumption Survey demonstrated a decrease in average fat and energy intake in both men and women, regardless of the increase in obesity rate between the years of 1977 and 1988 (62). Schoeller and Buchholz hypothesized that this paradox may be explained by the increased emphasis on carbohydrate intake which might not have a metabolic advantage (63).
In addition to the consumption of extra calories, reduced physical activity, and other lifestyle related misbehaviors, social economic status and cultural diversity are factors leading to positive energy balance. Several studies have demonstrated that low incomes, low levels of education and poverty are highly associated with obesity (64, 65). However, the correlation between social economic status and obesity varies among different ethnicities and gender (63).

Overweight and obesity are strongly related with higher circulating levels of inflammatory markers, such as C-reactive protein (CRP), IL-6 or TNF-a (56). Low-grade inflammation has been recognized as a potential mechanism in increasing adipose tissue storage (66). Alteration in endocrine function also affect fat storage, and several hormones are believed to play a role, including ghrelin, leptin, growth hormone, androgens, and IGF-1(67, 68). In addition, hypothyroidism and hypercortisolism are potential contributors to obesity (69).

2.2.3 Health Consequences of Obesity

The economic costs for obesity are incremental. Additionally, the health care costs associated with obesity include direct medical costs and indirect costs, such as medical care, surgery, absenteeism and decreasing productivity (70, 71).

Obesity is closely associated to several metabolic abnormalities, such as insulin resistance, hyperinsulinemia, and glucose intolerance; and the associations were independent of genetic factors (72). The metabolic changes can further develop into a cascade of syndromes, including diabetes, dyslipidemia, and even heart failure (53). In a 12-year prospective study, adipocyte fatty acid binding protein, which is one of the most abundant circulating adipokines, was reported to be predictive of the development of cardiovascular disease (73).

Excess body fat and high BMI influence the overall quality of life of obese individuals. Based on the NHANES III study conducted from 1988 to 1994, elderly individuals (aged 70 and older) in the highest quintile for body fat had higher likelihood to report functional limitations and the risk was higher for women (74).
2.3 Definition of Sarcopenic Obesity

Sarcopenic obesity is a simultaneous occurrence of low skeletal muscle mass (6) and excess adipose tissue (52). Like sarcopenia and obesity, the definition of sarcopenic obesity remains debatable.

Using DXA measurements, Baumgartner et al. (16) defined sarcopenic obese individuals as those who presented with an ASM index below two SD and a percent body fat above the 60th percentile of an age-matched population. Accordingly, males with sarcopenic obesity had an ASM index lower than 7.26 kg/m$^2$ and a body fat percentage above 28%; females with sarcopenic obesity had an ASM index lower than 5.45 kg/m$^2$ and a body fat percentage above 40% (16). Chung et al. developed a modified method to define sarcopenic obesity among an Asian population by using ASM/Weight (ASM/Wt) and BMI. Using a cross-sectional survey of elderly individuals aged 60 years or older, these researchers classified patients as sarcopenic obese if their ASM/Wt was lower than one SD below the mean of young healthy reference adults and their BMI was equal or greater than 25 kg/m$^2$ (75). Using prediction equations developed by previous studies (9, 76), Davison and colleagues defined sarcopenic obese as a body fat percentage in the upper two quintiles and a level of muscle mass in the lower two quintiles (74). There were also attempts to define sarcopenic obesity based on fat-free mass (FFM) and fat mass (FM). Schutz et al. (77) defined reference values of FFMI (FFM/height$^2$) and FMI (FM/height$^2$) for sarcopenic obesity. Additionally, residuals of the regression equation of FFM relative to FM have also been used as cutoffs and shown to be associated with reduced muscle strength and reduced aerobic fitness among postmenopausal women (78).

2.3.1 Prevalence of Sarcopenic Obesity

In the combined New Mexico Elder Health Survey and New Mexico Aging Process Study, the prevalence of sarcopenic obesity increased from 2% in individuals aged 60 - 69 years to up to 10% in individuals above 80 years of age (4). In a cross-sectional study of 1526 women and 1391 men participants from NHANES III, Davison et al. (74) reported the prevalence of sarcopenic obesity to be 7.4% in women and 9.6% in men aged 70 years and older. Zoico et al. (79) reported a prevalence of sarcopenic obesity of 12.4% in a sample of 167 healthy women aged 67-78 years old.
2.3.2 Etiology of Sarcopenic Obesity

Sarcopenic obesity is the confluence of two epidemics: aging and obesity (12). Roubenoff (14) has contextualized sarcopenic obesity as a cyclical pattern. The increase in adipocytes induces elevated secretion of leptin, tumor necrosis factors (TNFs) and other pro-inflammatory factors. These metabolic changes interfere with muscle protein metabolism and insulin sensitivity. Additionally, altered amino acid metabolism and impaired insulin sensitivity further promote fat mass gain. The loss of LBM is also related with the loss of the metabolically active BCM, which results in reduced resting energy expenditure. Ultimately, the cumulative result of these metabolic changes leads to fat gain. Concurrently, muscle loss affects physical activity level, which is also a contributor to fat mass accumulation, which in turn, reinforces muscle loss (14). Therefore, this cyclical pattern of multifactorial processes, eventually lead to muscle loss and fat gain. Excess adipose tissue causes increased production of certain hormone-like cytokines, particularly leptin, TNF-α as well as IL-6, promoting protein degradation and insulin resistance, enforcing muscle loss and fat gain (16). There is also evidence that altered endocrinal functions, such as age-related hormone changes, are involved in the development of sarcopenic obesity, including alterations of insulin, GH, IGF-1, corticosteroids and testosterone. It has been well established that insulin acts as an inhibitor in muscle protein breakdown and promotes skeletal muscle protein synthesis (80). Excess weight gain impairs the anabolic action of insulin, leading to a decrease in muscle synthesis (41). GH and IGF-1 are anabolic factors involved in protein anabolism and activation of satellite cell proliferation and differentiation (41). Waters et al. investigated the relationships between hormonal alterations and body composition among elderly individuals. Forty-five healthy participants were divided into normal lean, sarcopenic, obese, and sarcopenic-obese groups. A decreased secretion of GH was reported in obese and sarcopenic-obese individuals and this decrease was unresponsive to glucocorticoid suppression in those with sarcopenic obesity, indicating a distinguished regulatory mechanism of GH secretion in these individuals. In addition, no differences in cortisol concentrations were found among all groups, although leptin levels were higher in obese and sarcopenic-obese groups compared to the other two groups (81).

With advancing age, serum concentration of pro-inflammatory cytokines, such as TNF-α, IL-1β, IL-6, elevates as a result of the imbalance between catabolic and anabolic hormones (82).
The relationship between higher cytokine (IL-6 and TNF-α) levels and low muscle strength was evident among elderly populations (83). In addition, obesity promotes inflammation status. IL-1β is produced mainly by macrophages, endothelial cells and astrocytes. The pro-inflammatory role of IL-1β is played through the activation of T-helper cells, the production of prostaglandin E2, and self-induction effect. TNF-α, produced by macrophages and adipocytes, is a contributor to cachexia syndrome, cardiovascular disease and osteoporosis. It works closely with IL-1β in the regulation of IL-6 production. IL-6, released mainly from macrophages, myocytes, and T lymphocytes, stimulates a pro-inflammatory process in the body, leading to muscle loss and bone catabolism (84). As such, TNF-α, IL-1β, and IL-6 have been indicated as pathogenic factors contributing to an imbalance between protein synthesis and breakdown (38). Cesari et al. (85) evaluated the relation between body composition and biomarkers of inflammation, reporting that both CRP and IL-6 had a strong and positive relation with total body fat mass. However, the considered biomarkers were inversely correlated with ASM after adjustment of fat mass, indicating that pro-inflammatory cytokines are major contributors in the vicious cycle between fat gain and muscle loss.

2.3.3 Health Consequences of Sarcopenic Obesity

The health outcomes of sarcopenic obesity involve the impact of both sarcopenia and obesity. Evidence suggests that sarcopenic obese individuals have a greater risk of interrelated health outcomes compared to those who have the onset of either body composition type alone. Sarcopenic obesity has been associated with several clinical outcomes, such as impaired physical function and quality of life (4, 86). By analysis of data from the New Mexico Aging Process Study and the New Mexico Elder Health Survey, Baumgartner et al. (9) reported that sarcopenic obesity was the strongest predictor of risk of falls and physical disabilities. The associations remained significant after controlling for age, ethnic status, smoking and comorbidity. In an 8-year follow up study, Baumgartner et al. (16) evaluated changes in self-reported disability, assessed by the Instrumental Activities of Daily Living (IADL) questionnaire among 451 community-dwelling elderly individuals. Sarcopenic obese individuals presented with a 2.5 to 3.0 times higher likelihood to report IADL disability compared to other body composition phenotypes (nonsarcopenic-obese, sarcopenic-nonobese or nonsarcopenic-nonobese) individuals.
Additionally, sarcopenic obese individuals presented with the greatest drop in IADL scores during the 8-year follow-up (16). Although sarcopenic obesity was not directly assessed, Sternfeld et al. (2) reported negative associations between fat mass and physical performance (assessed by walking speed and grip strength) and positive associations between LBM and muscle quality (assessed by grip strength). Sarcopenic obesity has also been associated with knee osteoarthritis. In a large cross-sectional study, the prevalence of knee osteoarthritis was higher in sarcopenic obese individuals compared to those with sarcopenia or obesity along (87). Sarcopenic obesity has also been associated with poor health outcomes in clinical populations. In end-stage renal disease patients, Honda et al. (88) showed that the prevalence of cardiovascular disease and diabetes were higher among those with sarcopenic obese. Sarcopenic obesity has also been predictive of shorter survival in patients with solid tumors of respiratory and gastrointestinal tracts (5).

2.4 Body Composition Methodologies

As described previously, body composition abnormalities are associated with several clinical implications; therefore, body composition assessment allows for the identification of chronic under or over nutrition, and the evaluation of the effectiveness of nutritional intervention on body composition changes (89). As discussed in Chapter One, conventional techniques, such as body weight and BMI are incapable to detect body composition abnormalities in that they cannot offer information on the distribution of different compartments and its predictive prognostic value (90). For example, at the same BMI, females have more adiposity than males and elderly individuals tend to have lower amounts of LBM compared to adults (91).

Several body composition techniques have been developed in the past few decades including: BIA, DXA, magnetic resonance imaging (MRI), and air displacement phthysmography (BOD POD®). However, DXA, MRI and BOD POD® measurements are cost-sensitive and require highly trained operators. Compared to these techniques, BIA has relative advantages; it is low-cost, portable, non-invasive, and convenient to operate (92). For these merits, BIA has been largely used in field and epidemiologic studies (93).

Bioelectrical impedance analysis measurement is based on the principle that alternating electric current passes through different body part at different rates. Impedance is defined as the frequency-dependent opposition of a conductor to the alternating electric current (94). It is
derived from resistance (R) and reactance (Xc), representing the pure opposition of the conductor and the dielectric component of impedance respectivly (94). Since the alternating current flows through the body tissues at different rates, the current is partitioned into different body components. BIA measures the conductivity of water content in human bodies which is the electrolyte-rich fluid (94). FFM is an electrolyte-rich tissue and muscle mass is the major component of FFM. Therefore, most current passes through muscle mass; whereas adipose tissue, bone and organ restrain the flow of current and rarely allow it to pass through (76). In summary, FFM is negatively associated with impedance and non-lean mass plays a minor role in influencing impedance. The principle of BIA measurement is that different body components of a conductor have different oppositions to the alternating current.

Bioelectrical impedance analysis is a commonly used technique in the assessment of sarcopenic obesity. A summary of studies investigating the prevalence and clinical impact of sarcopenic obesity (assessed by BIA) is presented in Table 2.1.

<table>
<thead>
<tr>
<th>Author, Year</th>
<th>Purposes</th>
<th>Indexes used to define SO</th>
<th>Major results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sternfeld et al., 2002 (2)</td>
<td>To relate physical function (self-reported and measured) with body composition and to investigate the influence of lean to fat mass ratio (absolute vs. relative) on physical performance and functional limitation</td>
<td>Relative measure of body composition, the lean-to-fat ratio, defined by dividing lean by fat mass</td>
<td>Total FM was inversely related to physical performance and functioning. Compared to lower muscle mass, excess FM was more predictive of poor physical performance, functional limitation, and subsequent disability and mortality. Muscle strength was associated with lean mass but the magnitude of the association decreased as FM increased.</td>
</tr>
<tr>
<td>Davison et al., 2002 (74)</td>
<td>To investigate the associations between body composition and functional limitations</td>
<td>Body fat% from Baumgartner’s predictive equation. Muscle mass from Janssen’s predictive equation. Upper two quintiles for fat and lower two quintiles for relative muscle mass</td>
<td>Prevalence of SO was 7.3%. High% FM and BMI associated with functional limitations with women but with a less clear pattern in men. SO was not associated with greater likelihood of functional limitations.</td>
</tr>
<tr>
<td>Author, Year</td>
<td>Purposes</td>
<td>Indexes used to define SO</td>
<td>Major results</td>
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<tr>
<td>Stenholm <em>et al.</em>, 2008 (86)</td>
<td>To study the relationship of OB with walking limitation and to examine the role CRP and handgrip strength in that association</td>
<td>OB: % FM and WC: sex-specific quartiles BMI: WHO cut-offs. Muscle strength: sex-specific quartiles</td>
<td>Persons with SO had higher prevalence of walking limitation compared with those with OB or SC. %FM, CRP and handgrip strength were associated with walking limitation.</td>
</tr>
<tr>
<td>Monteiro <em>et al.</em>, 2010 (95)</td>
<td>To establish a reference dataset for temporal parameters during walking and to explore the effect of OB and SO on the same parameters</td>
<td>Skeletal muscle mass index from Janssen et al. (17). OB: BMI ≥ 25.5 kg/m²</td>
<td>The ratio between visceral fat and thigh muscle area was significantly increased in subjects with metabolic syndrome.</td>
</tr>
<tr>
<td>Siervo <em>et al.</em>, 2011 (11)</td>
<td>To assess the prevalence of SC or SO and to determine the change in prevalence using different adiposity indexes</td>
<td>SC: skeletal muscle index Janssen’s method (17) OB: BMI ≥ 30kg/m² ; WC&gt; 88cm; FM% ≥ 35%; FM index ≥ 9.5kg/m²</td>
<td>Prevalence of SO ranged from 0 to 67% in those younger than 60 years of age and from 49 to 90% in those≥60 years.</td>
</tr>
<tr>
<td>Visser <em>et al.</em>, 2012 (96)</td>
<td>To assess the impact of SO and FM and FFM independently on adverse outcome and its relation with muscle function in patients undergoing cardiac surgery</td>
<td>SC: defined as FFM index. 14.5 kg/m² for women, and 15.7 kg/m² for men OB: FM index. 11.8 kg/m2 for women, and 8.3 kg/m2 for men.</td>
<td>Prevalence of SO was 2.1%. Low FFM was associated with postoperative infections while FM was not. SO was associated with lower muscle function.</td>
</tr>
<tr>
<td>Lu <em>et al.</em>, 2012 (97)</td>
<td>To explore the relationship between SO and metabolic syndrome</td>
<td>SC was defined by the percentage of total skeletal mass (total skeletal muscle mass/weight ×100). Cutoffs were established at &lt;37% in men and &lt; 27.6% in women. OB: BMI ≥ 25.5 kg/m²</td>
<td>SO is a major independent risk factor for metabolic syndrome with a 12-fold higher risk compared to those without SC and obesity.</td>
</tr>
<tr>
<td>Barbat-Artigas <em>et al.</em>, 2012 (98)</td>
<td>To test whether low muscle mass is associated with better muscle quality in obese individuals</td>
<td>SC: skeletal muscle mass index by Janssen et al. (17). OB: FM ≥ 40%. Muscle quality: handgrip strength</td>
<td>Muscle quality increased as skeletal muscle and skeletal muscle index decreased. Muscle quality was also related to functional capacity.</td>
</tr>
</tbody>
</table>
Numerous studies have developed predictive equations for estimating LBM/muscle mass from BIA measurements. These BIA-derived equations have been useful in estimating the prevalence of sarcopenic obesity (74, 86), public health costs (76) and clinical outcomes (100). However, the accuracy and validity of this technique is still under discussion. Several factors have been identified that might influence the accuracy of BIA measurement and should be controlled in clinical settings.

Food and beverage consumed prior to BIA measurement might affect impedance. Slinde et al. (101) reported a decrease of impedance lasted two to four hours after ingestion of a standard meal. Nevertheless, no significant change in body composition measurement after fluid consumption has been reported (102). In order to control for any variations, the recommended standard technique is an overnight fasting before impedance measurement (103).

Recent exercise is another factor contributing to the variation in BIA measurement. The hypothesized mechanisms are increased cardiac output, blood flow to skeletal muscle, heat dissipation and dehydration after exercise (104).

Variations in body composition is extremely variable by ethnicity, ethnic-specific predictive equations should be used for accurate BIA measurements. As the majority of BIA predictive equations have been developed on Caucasian populations. Biological differences, such as height, weight, fat distribution, and body density lead to major differences in body composition (92). Therefore, predictive equations should be validated for specific populations. In addition, medical conditions (edema), individual differences (menopause), posture of the subject and environment factors (temperature) may also play a role in altering body composition assessed by BIA measurement.
CHAPTER THREE
MATERIALS AND METHODS

3.1 Study Population

This study was based on data obtained from patients receiving treatment at the Tallahassee Memorial Bariatric Center and has been approved by The Florida State University Human Subjects Committee and the Institutional Review Board at Tallahassee Memorial HealthCare, Inc.

From 2011 onwards, BIA measurements were incorporated as part of patient’s initial assessment (first consultation) and were included in their medical records. Caucasian patients with available BIA measurements, aged ≥ 18 years and with a BMI ≥ 30 kg/m² were eligible to participate in this study. Ineligible patients included those with severe diseases such as stroke, class II heart failure, inflammatory digestive disease, lipodystrophy, and those who were on oral steroid therapy.

3.2 Data Collection

As part of the TMBC standard of practice for newly admitted patients, a thorough health examination was conducted which includes information on demographics, BIA body composition measurements, weight change history, medical/surgical histories, psychiatric disorders, laboratory results, and medications. Additional information on physical function [Physical Activity Readiness (PAR-Q & YOU) Questionnaire] and physical activity (Exercise and Attitude Questionnaire) were also available. From the available information, relevant data was selected and presented in Appendix A. This data collection form was converted to Microsoft Access (Microsoft Office 2010). A subset of information included in the data collection form (Appendix B) was used to answer the specific hypothesis discussed in this Thesis, as described below.
3.2.1 Demographic Characteristics

Marital status was divided into three categories: single, married/partner, or divorced/widow/widower. Educational level was categorized as grade school, high school, college or post-graduate. Self-reported income was used as proxy for socioeconomic status. Annual income was classified into five categories: 0 – 30,000, 30,000 – 50,000, 50,000 – 75,000, 75,000 – 100,000, and >100,000.

3.2.2 Anthropometrics / Body Composition Assessments

Body weight, FFM, FM, %BF, and TBW were the variables collected using a Tanita Body Composition Analyzer, Model: TBF -310 (Tanita Corporation of America, Inc., Arlington Heights, IL, USA). Measurements involving weight were rounded to the nearest 0.1 pound. Height was measured to the nearest 0.1 inch by Accustat Ross Stadiometer. Body mass index was calculated as the ratio of weight (kg)/height squared (m$^2$) and classified according to WHO categories (Class I Obesity, 30.0-34.9 kg/m$^2$; Class II Obesity, 35.0-39.9 kg/m$^2$; Class III Obesity, ≥40 kg/m$^2$). Waist circumference (WC) was measured at a graduation length of 1/8 inch at the narrowest point between the lower border of the rib cage and the iliac crest by SECA 201 Ergonomic Circumference Measuring Tape.

In view of the lack of a consensus definition on sarcopenic obesity (99), the fact that our population was relatively younger (age ≥ 18 years) than elderly populations in which sarcopenic obesity has been commonly studied, and presented with a high prevalence of extreme cases of obesity (81.3% morbidly obese), the ratio of FMI/FFMI was used to categorize our cohort into those with sarcopenia and those without sarcopenia. As proposed by Prado et al. (99), weight gain normally occurs alongside a variable rate of accretion of FFM which can potentially give origin to either an obese (normal FFM accretion) or to a sarcopenic obese (low FFM accretion) phenotype. Since the proposed ratio includes both FMI and FFMI, we are able to account for proportional differences in these body composition compartments.

Figure 3.1 illustrates the concept of the FMI/FFMI ratio. Because of our relatively small sample size, we dichotomized the FMI/FFMI ratio using the gender-specific median. Therefore,
FMI/FFMI ratios below the gender-specific median were classified as non-sarcopenic obese and ratios above median were classified as sarcopenic obese.

![Figure 3.1](image)

**Figure 3.1.** Scheme of the relationship between fat mass index (FMI, depicted by larger grey circles) and fat-free mass index (FFMI, black circles) portraying different body composition types. Adapted from: Prado et al. *Anti Canc Agents Med Chem.* Ahead of print (105)

### 3.2.3 Metabolic Profile

Current smoking status was categorized as ‘No’, ‘Yes-occasionally’ and ‘Yes-everyday’. Alcohol consumption was categorized into ‘occasional’, ‘every day’ and ‘none’. Blood pressure was measured in the right arm by large adult blood pressure cuff and two-tube bladder (Welch Allyn Diagnostics). Both systolic and diastolic blood pressure readings were recorded. Biochemical parameters were obtained in several local laboratories in the Tallahassee region within three months of BIA measurement collection. However, not all patients had blood tests on file and only laboratorial tests taken within three months of BIA measurement were collected. When available, data on fasting plasma glucose, lipid profile (total cholesterol, triglyceride, and high-density lipoprotein (HDL)-cholesterol levels), liver and kidney functions, and vitamin D were collected for this study.

Laboratorial and anthropometric results were further explored to investigate the presence of metabolic syndrome as per the United States National Cholesterol Education Program Adult Treatment Panel III Guidelines (106). Metabolic syndrome was defined by the presence of at least three of the following risk factors: central obesity (WC ≥ 88 cm or 35 inches for women and ≥102 cm or 40 inches for men); systolic blood pressure ≥ 130 mmHg and/or diastolic blood pressure ≥ 85 mmHg; fasting plasma glucose level ≥ 100 mg/dL; triglyceride levels ≥ 150 mg/dL; and low HDL-cholesterols (< 40 mg/dL for men and < 50 mg/dL for women).
3.2.4 Health Status: Comorbidities, Functional Outcomes, and Physical Activities

Medical and surgical history information was collected to further investigate the occurrence of certain conditions, including arthritis, asthma, gout, osteoporosis, cancer, heart disease and sleep apnea. Information on comorbidity problems was also collected and those related to body composition were selected: acid reflux, alcoholism, diminished hygiene, fatigue, immobility, joint pain, low back pain, nausea, shortness of breath, sexual dysfunction, thyroid disease and weakness. The PAR-Q & YOU questionnaire, adopted from American College of Sports Medicine Standards and Guidelines for Health and Fitness Facilities (107), was used for a pre-evaluation of patients’ physical activity readiness. Seven questions were asked with answer options of either ‘Yes’ or ‘No’ to each: 1) “Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?”; 2) “Do you feel pain in your chest when you do physical activity?”; 3) “In the past month, have you had chest pain when you were not doing physical activity?”; 4) “Do you lose your balance because of dizziness or do you ever lose consciousness?”; 5) “Do you have a bone or joint problem (for ex: back, knee or hip) that could be made worse by a change in your physical activity?”; 6) “Is your doctor currently prescribing drugs (for ex: water pills) for your blood pressure of heart condition?”; 7) “Do you know of any other reason why you should not do physical activity?”.

Patients were further divided into two groups: ‘Yes’ to one or more of the above questions and ‘No’ to all questions. These variables were further dichotomized into ‘No’ to depict no problems and ‘Yes’ for the presence of at least 1/7 items and a comparison between sarcopenic obese and non-sarcopenic obese patients was conducted. Relevant data were selected from the Exercise and Attitude Questionnaire to investigate patients’ physical activity levels; questionnaire items explored variables associated with daily activities: “How many hours do you spend watching TV per day?”; “How many hours do you spend using your computer?”; “How much of your work day is spent at a desk?”; “How much of your work day is spent walking around?”; and “How much of your day is spent standing in one spot?”; “At least once/week do you participate in regular activity like brisk walking, jogging, biking, swimming, etc. long enough to work up a sweat? (‘Yes’ or ‘No’)?”.
3.3 Statistical Analysis

In this descriptive, retrospective study, the total number of charts reviewed was estimated to provide a small margin of error and a tight confidence interval. Although this is an ongoing study, our current sample size of 91 patients provides us an estimated margin of error of $0.1 = 0.98/\sqrt{91}$. Such a small margin of error gives a tight confidence interval and high power.

Continuous data are presented as mean ± standard deviation (SD) and range. The Pearson’s correlation coefficient was used to measure the strength of association between continuous variables. Assumptions concerning normal distribution of residuals and constant SD were checked. Comparisons between sarcopenic obese and non-sarcopenic obese groups were assessed by independent samples t-Test or Mann-Whitney independent samples test for continuous variables, as appropriate. Categorical variables are presented as percentages and compared using Chi-square ($\chi^2$) test or Fisher’s exact test, as appropriate.

Binary logistic regression was used to obtain the odds ratio (OR) and 95% CIs for outcome prediction, with multivariate analysis pursued for variables in the model reaching a level of significance of $\leq 0.100$. All tests were two-sided and statistical significance was reported at the $P \leq 0.05$ level. All analyses were conducted using SPSS Statistics (version 21.0, IBM Corporation, Armonk, NY).
CHAPTER FOUR
RESULTS

4.1 Demographic Characteristics

To date, data on 132 patients have been collected as part of this ongoing study. Three individuals were overweight (BMI <30 kg/m$^2$) and 38 were non-Caucasians and were therefore not included in this analysis. Therefore, 91 individuals (67 women and 24 men) were included.

No differences were observed for marital status, education level, self-reported household income, and tobacco/drug use between sarcopenic obese and non-sarcopenic obese patients. Overall, 14.8% of the patients were single, 59.3% were married or had a partner, and 25.9% were divorced or widows/widowers. Education levels were as follows: 1.3% grade school, 35.4% high school, 44.3% college and 19.0% post-graduate. Approximately 24.0% of the patients had an annual household income lower than $30,000, 36.1% between $50,000 and $75,000, 12.5% between $75,000 and $100,000, and 5.6% had an income above $100,000. In regards to drug and tobacco use, 2 patients (out of 82 with available data) reported history of drug use; 3.7% reported occasional tobacco use and 4.9% reported daily tobacco use.

4.2 Anthropometric and Body Composition Characteristics

Table 4.1 shows the characteristics of study participants. The majority of patients were women (~74%) with a mean age of 57 ± 11 (range 27 – 79 years). Men presented with higher body weight, height, WC, FFM, FFMI and TBW compared to women. On the contrary, women presented with higher %BF and FMI/FFMI ratio. No gender differences were found for BMI, FM and FMI. The FMI/FFM ratio was extremely variable ranging from 0.35 to 2.46 kg/m$^2$. This variability was independent of body weight, Figure 4.1. As exemplified in this figure, a person with approximately 113 kg may have a FMI/FFM ratio anywhere between 0.54 and 1.23 kg/m$^2$.

Fat mass index was inversely correlated with age (r=-0.26, $P=0.014$) while no significant association was observed between age and FFMI or FMI/FFMI ratio (r=-0.02, $P=0.848$ and r=-0.19, $P=0.077$, respectively) Figure 4.2 A, B, and C.
4.2.1 Sarcopenic Obesity

The median FMI/FFMI ratio was 1.05 kg/m\(^2\) for women and 0.78 kg/m\(^2\) for men, therefore, sarcopenic obesity was defined as a FMI/FFMI \(\geq 1.05\) kg/m\(^2\) or \(\geq 0.78\) kg/m\(^2\) for women and men, respectively. Therefore, a total of 46 sarcopenic obese patients (74% women) were identified, Table 4.2.

No age differences were observed among sarcopenic obese and non-sarcopenic obese patients, although sarcopenic obese women tended to be older compared to their counterparts \((P=0.077)\), Table 4.2. The distribution of FFMI, FMI and %BF between sarcopenic obese and non-sarcopenic obese patients for men and women is shown in Figure 4.3 A, B and C. In both genders, body weight, %BF, FM, FMI and FMI/FFMI ratio were significantly greater in the sarcopenic obese group, Table 4.2. Although FFM and FFMI were higher in non-sarcopenic obese men compared to sarcopenic obese men, no differences were observed among women, Table 4.2. Sarcopenic obese women had significantly greater BMI compared to non-sarcopenic obese women, with no differences observed in men, Table 4.2.

No differences were found among BMI categories (obese classes I, II and III) by gender \((P=0.196,\) data not shown) but the prevalence of sarcopenic obesity was significantly higher in morbidly obese patients. The prevalence of sarcopenic obesity among BMI categories was 0%, 33.3% and 56.8% for classes I, II and III obesity respectively, compared to 100.0%, 66.7% and 43.2% for non-sarcopenic obesity \((P=0.022)\).

In order to investigate the prevalence of sarcopenic obesity among women receiving or not receiving HRT, women were divided by age \((< 50\) or \(\geq 50\) years). Among women \(\geq 50\) years \((n = 55)\), 18.2% reported taking HRT. A greater proportion of women taking HRT were non-sarcopenic obese although this difference was not statistically significant \((80.0\% \) vs. 20.0% sarcopenic obese, \(P=0.078)\).

4.3 Metabolic Profile by Body Composition Phenotype

The overall metabolic characteristics of sarcopenic obese and non-sarcopenic obese patients are presented in Table 4.3. Not all patients had laboratorial tests on file (sample size is depicted with data presentation in Table 4.3). With the exception of albumin levels, no differences were observed between sarcopenic obese and non-sarcopenic obese groups for any of
the assessed metabolic parameters. Albumin plasma concentration was significantly lower in sarcopenic obese patients compared to non-sarcopenic obese patients \((P=0.035)\). Although not statistically significant, the prevalence of metabolic syndrome was greater among sarcopenic obese patients. Approximately 63.0% of the sarcopenic obese patients and 44.4% of the non-sarcopenic obese patients reported metabolic syndrome \((P=0.075)\), Table 4.3. All patients presented with central obesity.

Only 2 patients were taking thyroid medication (levothyroxine). The prevalence of thyroid diseases was not different between sarcopenic obese and non-sarcopenic obese patients \((P=0.080)\), Table 4.3.

4.4 Health Status: Comorbidities, Functional Outcomes, and Physical Activities by Body Composition Phenotype

As presented in Table 4.3, all patients reporting alcohol abuse were sarcopenic obese \((P=0.026)\). Compared to non-sarcopenic obese patients, the prevalence of sexual dysfunction was also significantly greater among sarcopenic obese patients versus non-sarcopenic obese patients \((17.4\% \text{ vs. } 2.2\%, \ P=0.030)\). Although not statistically significant, the prevalence of asthma was higher among sarcopenic obese individuals compared to their counterparts \((23.9\% \text{ versus } 8.9\%, \text{ respectively, } \ P=0.088)\). Likewise, the prevalence of nausea was higher in sarcopenic obese individuals, but only with a trend towards significance \((P=0.059)\).

In regards to functional outcomes, the prevalence of mobility problems tended to be higher among sarcopenic obese patients compared to their counterparts \((17.4\% \text{ vs. } 4.4\% \text{ respectively, } \ P=0.090)\), Table 4.3. A greater proportion of sarcopenic obese patients presented with low back pain compared to non-sarcopenic obese patients \((60.9\% \text{ vs. } 40.0\% \text{ respectively, } \ P=0.046)\).

In order to further explore the association between sarcopenic obesity and low back pain, a univariate model was used including body composition variables, as well as variables known to predict low back pain (psychological distress/ depression, age, and gender) \((108)\), Table 4.4. A multivariate analysis was not pursed for all body composition variables due to multicollinearity \((2)\), Table 4.4. Sarcopenic obesity was the strongest predictor of lower back pain; patients presenting with sarcopenic obesity were 2.3 times more likely to present with lower back pain compared to non-sarcopenic obesity patients, Table 4.4.
Selected daily activities from the Exercise and Attitude Questionnaire are shown in Table 4.5. No significant differences were observed between sarcopenic obese and non-sarcopenic obese groups for any of the reported activities. Overall, 23.8% of patients (n=80) reported being involved in regular physical activity at least once/week; the mean FMI/FFMI ratio was not different between patients participating (0.95 kg/m$^2$) or not (0.99 kg/m$^2$) in regular physical activity ($P=0.124$, data not shown). No differences of physical activity readiness assessed by PAR-Q & YOU Questionnaire were observed between sarcopenic obese and non-sarcopenic obese patients, Table 4.5.

Table 4.1. Overall characteristics of study patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Women (N = 67)</th>
<th>Men (N = 24)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total (N = 91)</td>
<td>Mean ± SD (Range)</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>57.0 ± 11.0</td>
<td>56.6 ± 11.9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>128.3 ± 24.1</td>
<td>122.2 ± 21.0</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>166.1 ± 8.9</td>
<td>162.5 ± 6.9</td>
</tr>
<tr>
<td>WC (cm)(23M)$^b$</td>
<td>134.7 ± 16.0</td>
<td>132.3 ± 15.7</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>46.4 ± 7.6</td>
<td>46.2 ± 7.3</td>
</tr>
<tr>
<td>BF (%)</td>
<td>49.1 ± 7.1</td>
<td>51.0 ± 4.3</td>
</tr>
<tr>
<td>FM (kg)</td>
<td>63.4 ± 17.2</td>
<td>62.9 ± 14.5</td>
</tr>
<tr>
<td>FMI (kg/m$^2$)</td>
<td>23.0 ± 6.0</td>
<td>23.8 ± 5.0</td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>64.5 ± 13.6</td>
<td>59.3 ± 8.6</td>
</tr>
<tr>
<td>FFMI (kg/m$^2$)</td>
<td>23.3 ± 3.9</td>
<td>22.5 ± 3.2</td>
</tr>
<tr>
<td>Variables</td>
<td>Total (N = 91)</td>
<td>Women (N = 67)</td>
</tr>
<tr>
<td>---------------</td>
<td>---------------</td>
<td>----------------</td>
</tr>
<tr>
<td>FMI/FFMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>1.00 ± 0.29 (0.35-2.46)</td>
<td>1.06 ± 0.18 (0.73-1.43)</td>
</tr>
<tr>
<td>TBW (kg)</td>
<td>47.5 ± 10.0 (32.0-83.4)</td>
<td>43.4 ± 6.3 (32.0-70.2)</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD and range.

<sup>a</sup>Independent samples t-test, women vs. men.

<sup>b</sup>N that differ from the whole group are shown.

WC = Waist Circumference; BMI = Body Mass Index; BF = Body Fat; FM = Fat Mass; FMI = Fat Mass Index; FFM = Fat Free Mass; FFMI = Fat Free Mass Index; TBW = Total Body Water.
Table 4.2. Comparison of body composition characteristics between sarcopenic obese and non-sarcopenic patients by gender

<table>
<thead>
<tr>
<th>Variables</th>
<th>Sarcopenic Obese (N = 34)</th>
<th>Non-sarcopenic Obese (N = 33)</th>
<th>P-value&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Sarcopenic Obese (N = 12)</th>
<th>Non-sarcopenic Obese (N = 12)</th>
<th>P-value&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>54.1 ± 11.8</td>
<td>59.2 ± 11.5</td>
<td>0.077</td>
<td>56.1 ± 7.6</td>
<td>60.0 ± 8.8</td>
<td>0.255</td>
</tr>
<tr>
<td>(27.0-73.0)</td>
<td>(28.0-79.0)</td>
<td></td>
<td></td>
<td>(35.0-67.0)</td>
<td>(41.0-73.0)</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>132.7 ± 17.9</td>
<td>111.3 ± 18.3</td>
<td>&lt;0.0001</td>
<td>156.2 ± 27.1</td>
<td>134.5 ± 16.3</td>
<td>0.029</td>
</tr>
<tr>
<td>(109.1-169.6)</td>
<td>(78.3-181.6)</td>
<td></td>
<td></td>
<td>(118.7-191.6)</td>
<td>(114.0-162.3)</td>
<td></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>163.5 ± 7.2</td>
<td>161.5 ± 6.6</td>
<td>0.239</td>
<td>176.6 ± 7.1</td>
<td>175.7 ± 4.3</td>
<td>0.719</td>
</tr>
<tr>
<td>(152.4-182.9)</td>
<td>(148.6-180.3)</td>
<td></td>
<td></td>
<td>(166.7-190.5)</td>
<td>(168.9-181.6)</td>
<td></td>
</tr>
<tr>
<td>WC (cm)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>140.3 ± 11.7</td>
<td>124.1 ± 15.3</td>
<td>&lt;0.0001</td>
<td>143.8 ± 18.0</td>
<td>139.5 ± 11.0</td>
<td>0.497</td>
</tr>
<tr>
<td>(112.4 – 160.0)</td>
<td>(97.8 – 167.6)</td>
<td></td>
<td></td>
<td>(119.4 – 170.2 )</td>
<td>(124.5 – 153.0)</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>49.6 ± 5.4</td>
<td>42.8 ± 7.4</td>
<td>&lt;0.0001</td>
<td>50.4 ± 10.2</td>
<td>43.5 ± 4.8</td>
<td>0.143</td>
</tr>
<tr>
<td>(39.4-58.8)</td>
<td>(31.6-68.7)</td>
<td></td>
<td></td>
<td>(36.0-65.6)</td>
<td>(36.6-49.2)</td>
<td></td>
</tr>
<tr>
<td>BF (%)</td>
<td>54.6 ± 2.3</td>
<td>47.4 ± 2.4</td>
<td>&lt;0.0001</td>
<td>50.7 ± 9.0</td>
<td>43.5 ± 4.8</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>(51.3-58.8)</td>
<td>(42.0-51.1)</td>
<td></td>
<td></td>
<td>(37.4-71.0)</td>
<td>(25.6-43.6)</td>
<td></td>
</tr>
<tr>
<td>FM (kg)</td>
<td>72.7 ± 11.5</td>
<td>52.8 ± 9.4</td>
<td>&lt;0.0001</td>
<td>80.1 ± 23.3</td>
<td>49.9 ± 11.2</td>
<td>0.001</td>
</tr>
<tr>
<td>(57.1-95.0)</td>
<td>(34.1-85.7)</td>
<td></td>
<td></td>
<td>(51.1-113.9)</td>
<td>(29.7-63.6)</td>
<td></td>
</tr>
<tr>
<td>FMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>27.1 ± 3.6</td>
<td>20.3 ± 3.6</td>
<td>&lt;0.0001</td>
<td>25.9 ± 8.1</td>
<td>16.2 ± 3.8</td>
<td>0.002</td>
</tr>
<tr>
<td>(20.5-32.8)</td>
<td>(13.7-32.4)</td>
<td></td>
<td></td>
<td>(14.9-36.7)</td>
<td>(9.4-21.0)</td>
<td></td>
</tr>
<tr>
<td>FFM (kg)</td>
<td>60.0 ± 7.4</td>
<td>58.5 ± 9.7</td>
<td>0.366</td>
<td>73.4 ± 15.8</td>
<td>84.5 ± 11.4</td>
<td>0.045</td>
</tr>
<tr>
<td>(50.2-77.0)</td>
<td>(43.8-95.9)</td>
<td></td>
<td></td>
<td>(46.4-100.5)</td>
<td>(69.1-113.9)</td>
<td></td>
</tr>
<tr>
<td>FFMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>22.4 ± 2.2</td>
<td>22.5 ± 4.0</td>
<td>0.711</td>
<td>23.7 ± 5.7</td>
<td>27.3 ± 2.8</td>
<td>0.052</td>
</tr>
<tr>
<td>(18.4-28.5)</td>
<td>(16.4-36.3)</td>
<td></td>
<td></td>
<td>(14.5-34.1)</td>
<td>(22.8-34.5)</td>
<td></td>
</tr>
<tr>
<td>FMI/FFMI</td>
<td>1.21 ± 0.11</td>
<td>0.90 ± 0.08</td>
<td>&lt;0.0001</td>
<td>1.14 ± 0.48</td>
<td>0.60 ± 0.15</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>(1.05-1.43)</td>
<td>(0.73-1.04)</td>
<td></td>
<td></td>
<td>(0.78-2.46)</td>
<td>(0.35-0.77)</td>
<td></td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD, range.
<sup>a</sup>Independent samples t-test or Mann-Whitney independent samples test.
WC = Waist Circumference; BMI = Body Mass Index; BF = Body Fat; FM = Fat Mass; FMI = Fat Mass Index; FFM = Fat Free Mass; FFMI = Fat Free Mass Index; TBW = Total Body Water.
<table>
<thead>
<tr>
<th>Variables</th>
<th>Sarcopeic Obese (N = 46) Mean ± SD</th>
<th>Non-sarcopenic Obese (N = 45) Mean ± SD</th>
<th>P-value&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Metabolic Variables</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic BP (mmHg)(44SO,43OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>133.7 ± 17.0</td>
<td>135.5 ± 16.1</td>
<td>0.452</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)(44SO,43OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>76.7 ± 11.1</td>
<td>79.2 ± 17.2</td>
<td>0.652</td>
</tr>
<tr>
<td>Total Cholesterol (mg/dl)(30SO,26OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>187.0 ± 45.4</td>
<td>178.0 ± 36.6</td>
<td>0.628</td>
</tr>
<tr>
<td>LDL (mg/dl)(29SO,25OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>105.1 ± 27.9</td>
<td>98.2 ± 33.8</td>
<td>0.362</td>
</tr>
<tr>
<td>HDL (mg/dl)(29SO,26OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>53.4 ± 22.3</td>
<td>48.2 ± 13.8</td>
<td>0.637</td>
</tr>
<tr>
<td>Fasting Glucose (mg/dl)(32SO,27OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>111.2 ± 27.0</td>
<td>105.3 ± 32.4</td>
<td>0.152</td>
</tr>
<tr>
<td>Triglycerides (mg/dl)(29SO,26OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>153.0 ± 79.0</td>
<td>170.0 ± 104.5</td>
<td>0.637</td>
</tr>
<tr>
<td>Albumin (g/dl)(33SO,26OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>4.0 ± 0.3</td>
<td>4.2 ± 0.3</td>
<td>0.035</td>
</tr>
<tr>
<td>Creatinine (mg/dl)(34SO,30OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>0.9 ± 0.50</td>
<td>0.9 ± 0.29</td>
<td>0.244</td>
</tr>
<tr>
<td>Total Protein (g/dl)(32SO,26OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>7.0 ± 0.55</td>
<td>6.9 ± 0.3</td>
<td>0.637</td>
</tr>
<tr>
<td>Vitamin D (ng/ml)(21SO,21OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>29.1 ± 12.6</td>
<td>34.2 ± 13.4</td>
<td>0.170</td>
</tr>
<tr>
<td>TSH (mIU/L)(23SO,21OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>2.3 ± 1.7</td>
<td>1.8 ± 1.3</td>
<td>0.452</td>
</tr>
<tr>
<td>T3 (pg/ml)(11SO,6OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>5.1 ± 7.0</td>
<td>7.7 ± 11.4</td>
<td>0.404</td>
</tr>
<tr>
<td>T4 (ng/dl)(18SO,21OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.9 ± 2.2</td>
<td>1.7 ± 1.8</td>
<td>0.530</td>
</tr>
<tr>
<td>AST (U/L)(32SO,30OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>22.8 ± 9.8</td>
<td>21.7 ± 5.4</td>
<td>0.657</td>
</tr>
<tr>
<td>ALT (U/L)(31SO,28OO)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>27.2 ± 18.7</td>
<td>25.2 ± 10.3</td>
<td>0.903</td>
</tr>
<tr>
<td><strong>Comorbidities / Medical Symptoms</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MetS&lt;sup&gt;c&lt;/sup&gt; (29SO,22O)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>29 (63.0%)</td>
<td>20 (44.4%)</td>
<td>0.075</td>
</tr>
<tr>
<td>Acid Reflux</td>
<td>21 (45.7%)</td>
<td>14 (31.1%)</td>
<td>0.154</td>
</tr>
<tr>
<td>Arthritis</td>
<td>26 (56.5%)</td>
<td>23 (51.1%)</td>
<td>0.605</td>
</tr>
<tr>
<td>Asthma</td>
<td>11 (23.9%)</td>
<td>4 (8.9%)</td>
<td>0.088</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>6 (13.0%)</td>
<td>0 (0.0%)</td>
<td>0.026</td>
</tr>
<tr>
<td>Cancer</td>
<td>4 (8.7%)</td>
<td>9 (20.0%)</td>
<td>0.145</td>
</tr>
<tr>
<td>Diabetes</td>
<td>12 (26.1%)</td>
<td>17 (37.8%)</td>
<td>0.231</td>
</tr>
<tr>
<td>Diminished Hygiene</td>
<td>4 (8.7%)</td>
<td>1 (2.2%)</td>
<td>0.361</td>
</tr>
<tr>
<td>Fatigue</td>
<td>23 (50.0%)</td>
<td>19 (42.2%)</td>
<td>0.457</td>
</tr>
<tr>
<td>Gout</td>
<td>7 (15.2%)</td>
<td>2 (4.4%)</td>
<td>0.158</td>
</tr>
<tr>
<td>Heart Disease</td>
<td>2 (4.3%)</td>
<td>3 (6.7%)</td>
<td>0.677</td>
</tr>
<tr>
<td>Joint Pain</td>
<td>31 (67.4%)</td>
<td>27 (60.0%)</td>
<td>0.463</td>
</tr>
<tr>
<td>Nausea</td>
<td>7 (15.2%)</td>
<td>1 (2.2%)</td>
<td>0.059</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>3 (6.5%)</td>
<td>3 (6.7%)</td>
<td>1.000</td>
</tr>
<tr>
<td>Sexual Dysfunction</td>
<td>8 (17.4%)</td>
<td>1 (2.2%)</td>
<td>0.030</td>
</tr>
</tbody>
</table>
Table 4.3. - Continued

<table>
<thead>
<tr>
<th>Variables</th>
<th>Sarcopenic Obese (N = 46)</th>
<th>Non-sarcopenic Obese (N = 45)</th>
<th>P-value&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Shortness of Breath</td>
<td>17 (37.0%)</td>
<td>18 (40.0%)</td>
<td>0.765</td>
</tr>
<tr>
<td>Sleep Apnea</td>
<td>23 (50.0%)</td>
<td>25 (55.6%)</td>
<td>0.596</td>
</tr>
<tr>
<td>Thyroid Disease</td>
<td>8 (17.4%)</td>
<td>15 (33.3%)</td>
<td>0.080</td>
</tr>
<tr>
<td>Weakness</td>
<td>11 (23.9%)</td>
<td>8 (17.8%)</td>
<td>0.472</td>
</tr>
</tbody>
</table>

**Functional Outcomes**

<table>
<thead>
<tr>
<th></th>
<th>Sarcopenic Obese (N = 46)</th>
<th>Non-sarcopenic Obese (N = 45)</th>
<th>P-value&lt;sup&gt;a&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immobility</td>
<td>8 (17.4%)</td>
<td>2 (4.4%)</td>
<td>0.090</td>
</tr>
<tr>
<td>Low Back Pain</td>
<td>28 (60.9%)</td>
<td>18 (40.0%)</td>
<td>0.046</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD or number (percentage).
<sup>a</sup>Mann-Whitney independent sample test for continuous variables and Fisher’s exact test or Chi-Square test for categorical variables.
<sup>b</sup>N that differ from the whole group are shown.
<sup>c</sup>MetS (prevalence was reported as having three or more of the following conditions: WC ≥ 88 cm or 35 inches for women and ≥102 cm or 40 inches for men; systolic blood pressure ≥ 130 mmHg or diastolic blood pressure ≥ 85 mmHg; fasting plasma glucose level ≥ 100 mg/dL; triglyceride levels ≥ 150 mg/dL; and HDL-cholesterols < 40 mg/dL for men and < 50 mg/dL for women).

BP = Blood Pressure; MetS = Metabolic Syndrome.

Table 4.4. Odds ratio and 95% CIs for the univariate effect of variables on low back pain

<table>
<thead>
<tr>
<th>Coefficient (SE)</th>
<th>Odds Ratio (95%CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sarcopenic Obese&lt;sup&gt;1&lt;/sup&gt;</td>
<td>0.85 (0.43)</td>
<td>2.3 (1.01-5.41)</td>
</tr>
<tr>
<td>FMI (kg/m&lt;sup&gt;2&lt;/sup&gt;)</td>
<td>0.11 (0.04)</td>
<td>1.1 (1.03 – 1.20)</td>
</tr>
<tr>
<td>FFMI (kg/m2)</td>
<td>0.04 (0.06)</td>
<td>1.0 (0.93 – 1.16)</td>
</tr>
<tr>
<td>BMI (kg/m2)</td>
<td>0.07 (0.03)</td>
<td>1.1 (1.01-1.15)</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>-0.09 (0.01)</td>
<td>0.1 (0.95-1.03)</td>
</tr>
<tr>
<td>Gender&lt;sup&gt;2&lt;/sup&gt;</td>
<td>0.30(0.48)</td>
<td>1.0 (0.41-2.62)</td>
</tr>
</tbody>
</table>

<sup>1</sup>Versus non-sarcopenic obese patients (defined as the gender-specific median FMI/FFMI). <sup>2</sup>Versus men
Table 4.5. Comparison of daily activities between sarcopenic obese and non-sarcopenic obese groups

<table>
<thead>
<tr>
<th>Activities</th>
<th>Sarcopenic Obese (N = 46)</th>
<th>Non-sarcopenic Obese (N = 45)</th>
<th>P-valuea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Watching TV (hours/day) (39SO,3900)b</td>
<td>3.0 ± 2.5</td>
<td>2.9 ± 1.9</td>
<td>0.705</td>
</tr>
<tr>
<td>Computer Use (hours/day) (28SO,3900)b</td>
<td>4.1 ± 3.6</td>
<td>3.8 ± 3.2</td>
<td>0.914</td>
</tr>
<tr>
<td>Work At Desk (hours/day) (36SO,4000)b</td>
<td>5.2 ± 3.4</td>
<td>3.8 ± 3.6</td>
<td>0.132</td>
</tr>
<tr>
<td>Walking Around (hours/day) (36SO,3700)b</td>
<td>1.5 ± 2.0</td>
<td>2.1 ± 2.2</td>
<td>0.107</td>
</tr>
<tr>
<td>Standing One Spot (hours/day) (33SO,3700)b</td>
<td>0.6 ± 1.3</td>
<td>0.7 ± 1.2</td>
<td>0.500</td>
</tr>
<tr>
<td>PAR-Q &amp; YOUc (40SO,380)b</td>
<td>40 (87.0%)</td>
<td>45 (84.4%)</td>
<td>0.732</td>
</tr>
</tbody>
</table>

Data are expressed as mean ± SD or percentage.

aMann-Whitney independent samples test for continuous variables and Chi-square test for categorical variables.
bN that differ from the whole group are shown.
cPrevalence reported as having at least one out of seven PAR-Q & YOU questions checked as ‘Yes’.

Figure 4.1. Relationship between body weight (kg) and Fat mass index/Fat-free mass index ratio (kg/m²)
Ratio ranges from 0.54 to 1.23 kg/m² for a person at ~113kg. FMI = Fat Mass Index; FFMI = Fat Free Mass Index
Figure 4.2. Relationship between age (years) and A) Fat mass index (FMI, kg/m$^2$); B) Fat mass index/Fat-free mass index ratio (FMI/FFMI ratio, kg/m$^2$); C) Fat-free mass index (FFMI, kg/m$^2$)
Figure 4.3. A) Fat-free mass index (FFMI, kg/m$^2$); B) Fat mass index (FMI, kg/m$^2$) and C) Percent body fat (%BF) distribution of sarcopenic obese and non-sarcopenic obese patients by gender.
CHAPTER FIVE

DISCUSSION

5.1 Review of Hypotheses and Conclusions

**Hypothesis 1**: The distribution of FMI/FFMI ratio will present with a degree of variability greater than two folds (Chapter One).

**Hypothesis 1** was accepted as the distribution of FMI/FFMI ratio ranged from 0.35 kg/m² to 2.46 kg/m² in the patient participants.

**Hypothesis 2**: Compared to non-sarcopenic obese patients, sarcopenic obese patients will:

a) present with higher rates of metabolic abnormalities and comorbidities, as assessed by the TMBC New Patient Questionnaire (Chapter One);

b) report lower physical activity readiness and levels, as assessed by the PAR&Q Questionnaire and Exercise History and Attitude Questionnaire (Chapter One).

**Hypothesis 2a** was partially accepted as a lower plasma albumin concentration ($P=0.035$) and a higher prevalence of low back pain (60.9%), alcoholism (13.0%) and sexual dysfunction (17.4%) were reported among sarcopenic obese patients, compared to non-sarcopenic obese patients. No other differences were observed for the other variables collected between the two body composition groups. **Hypothesis 2b** was rejected because no differences on physical activities were observed between sarcopenic obese and non-sarcopenic obese patients.

5.2 Summary

The major findings of this thesis demonstrated a wide variability in body composition as assessed by the FMI/FFMI ratio in this cohort of obese patients illustrating how variable the proportions of fat to fat-free tissues may differ even in patients with similar BMI. Furthermore, sarcopenic obesity was associated with higher risk of certain metabolic abnormalities and comorbidities. The findings reported here highlight the potential use of the FMI/FFMI ratio as an index for the assessment of abnormal body composition phenotypes in patients with severe obesity.
5.3 Discussion of Results

As reported in Chapter One, this study sought to describe the variability in body composition, as well as the relationship between abnormal body composition (defined as a high FMI/FFMI ratio) and overall health of obese patients. Overall health was defined using metabolic, functional and physical activity characteristics of the study population.

5.3.1 The FMI/FFM Ratio

In this study, we used the FMI/FFMI ratio to categorize patients into sarcopenic obese or non-sarcopenic obese, which is an index that considers the potential inter-relationship between these body composition compartments, rather than the absolute amount of each component (99). As expected, the ratio was strongly correlated with FM, %BF and FFM, as well as BMI. Fat mass was significantly higher in the sarcopenic obese group, compared to the non-sarcopenic obese group in both genders; however, a difference in FFMI was only observed among men (lower in the sarcopenic obese group). The inconsistency in FFM findings between genders may be reflective of differences in adiposity, since women had a significantly higher %BF than men. Moreover, the results of a study on a large cohort of healthy elderly individuals reported lower rates of skeletal muscle depletion in women, compared to men (FFM was 1.0 kg/decade lower in men and 0.4 kg/decade lower in women aged 60 years and older) (109). Therefore, FM contributed the most to the FMI/FFMI ratio difference observed between the two body composition groups in women and the concept of FMI/FFMI ratio may be particularly useful for clinical evaluation of sarcopenic obesity in men.

Compared to body composition indexes of separate FM or FFM measurements (and its derivatives), the FMI/FFMI ratio may be a more reliable prognostic index for the effect of body composition on health (99). Using data from a convenient clinical sample of 560 obese women (18 – 80 years old) with a BMI between 30 and 40 kg/m², Prado et al. (99) reported an age-related curvilinear relationship and a progressive narrowing of FM/FFM ratio with increased age, suggesting that the ratio accurately depict body composition changes expected with aging (4). In a similar approach, Sternfeld et al. (2) reported that a higher lean-to-fat ratio was related to faster walking speed and less likelihood of functional limitation among elderly individuals (≥ 55
years). The association of adiposity to muscle ratio and physical limitation was also demonstrated in a large community-dwelling elderly adult population (>65 years) (110). The above results suggested that the use of the lean-to-fat ratio considers the joint effects of lean mass (a protective factor) and fat mass (a risk factor), and hence was a better predictor of functional outcomes (2).

### 5.3.2 Gender- and Age-related Variations in Body Composition

The results of the present study indicate that both gender and age have an impact on the variability of body composition components. Although BMI was not different by gender, men presented with more FFM while women had a higher %BF, again confirming the variability in body composition independent of BMI. These gender differences in body composition were expected, as men have greater amounts of FFM than women, and women have greater %BF than men at any age (39). In a cohort of morbidly obese patients, Lafortuna et al. (111) reported that a unit increase in body weight was accounted for by an almost equal increase in FM and FFM in men, whereas female bodily weight gain consisted of nearly triple the amount of FM compared to FFM.

In this present study, we showed that FMI decreased with age. Previous studies have described contradictory results. A longitudinal cohort study of elderly individuals, reported a significant increase in FM with increasing age in men (112). Many cross-sectional studies indicate that FM increases with age in both men and women. Kyle et al. (109) have shown that FM increased with age in both genders in a large cohort of individuals aged 18 to 60 years and decreased from 60 to 94 years of age. Zhu et al. (113) reported a curvilinear pattern between %BF and age, that %BF reached a peak at the age of 50-59 in blacks and 60-90 in whites and decreased afterwards. Similar results were reported by Mott et al., (114) a deceleration in the rate of increase in fat mass with age was observed in both genders among almost all ethnicities (Asians, Blacks, and Whites), with exception of Puerto Rican men. The discrepancy between our observations and previous studies may be due to differences in the population studied, study design (cross-sectional versus longitudinal) and body composition measurements used. Moreover, our study sample size was relatively small which might also confound the correlation between age and FM. Although a negative relationship was observed
between FMI/FFM ratio and age, this difference was only trending towards significance ($P=0.077$), and likely driven due to differences in FM.

It is of interest that in the present study, age was not strongly associated with a decrease in FFMI, even though FMI decreased with aging. Although a parallel change in both compartments is expected, specific direction of FMI and FFMI changes remains controversial (115). Additionally, the high prevalence of morbidly obese patients in our study (81.3%) may help explain these conflicting results. We can speculate that in morbidly obese patients, FM decreases with age but not enough to induce significant losses of FFM, suggestive that FFM or even a slight increase in FFM is needed to support the heavy load of excess adiposity.

### 5.3.3 Metabolic Abnormalities

Although the prevalence of metabolic syndrome was not statistically higher in sarcopenic obese patients (63.0%) compared to non-sarcopenic obese (44.4%) patients, a trend towards significance was observed ($P=0.075$). The association between sarcopenic obesity and metabolic syndrome has been contradictory. Barbat-Artigas et al. (98) reported that sarcopenic obese postmenopausal women were metabolically healthy. Baumgartner et al. (16) showed that the prevalence of metabolic syndrome was highest in the group of non-sarcopenic obese subjects among healthy elderly individuals. In contrast, Kim et al. (116) used ASM to visceral fat area ratio (muscle/fat ratio) in a large cohort of healthy Korean adults to examine the association between sarcopenic obesity and metabolic syndrome. The authors reported that subjects with metabolic syndrome had significantly lower muscle/fat ratio (116). It has also been suggested that the increased prevalence of cardio-vascular diseases among sarcopenic obese individuals would be predicted by a high prevalence of metabolic syndrome among these individuals (117). Unfortunately, the precise relationship between FFM and FM and the incidence of metabolic complications is unclear (118).

In the present study, plasma albumin concentrations were lower for sarcopenic obese patients. Lower albumin concentration has been linked as a risk factor for muscle catabolism (119). Likewise, a recent study in elderly individuals showed that low albumin concentration was significantly associated with mortality and frailty ($P<0.001$) (120). Hemelrijck et al. (121) showed a combination of CRP, albumin, gamma-glutamyl transferase, and HDL as predictive
factors for mortality in individuals aged 50 years or over. Although information on mortality and frailty was not available in this study, our finding sheds light on the use of clinical biomarkers that recognize people potentially at risk for sarcopenic obesity, which could in turn be associated with these outcomes.

5.3.4 Comorbidities / Functional Limitations / Physical Activities

In this study, all alcohol-dependent subjects were sarcopenic obese. Previous studies have demonstrated that protein degradation rate exceeded the rate of protein synthesis among alcohol abusers, leading to impaired muscle function (alcoholic myopathy) (122). In a review study of the pathology of alcoholic myopathy, muscle atrophy caused by alcohol abuse might be selective of losing Type II fibers (123). Although the precise mechanism underlying the effect of alcohol intake on muscle decline is uncertain, potential factors have been proposed. Fernandez-Sola et al. (124) reported that apoptosis was present in the skeletal muscle of high-dose alcohol consumers suggesting apoptosis might be involved in the pathogenesis of skeletal myopathy among alcohol abusers by impairing cellular structures. Animal studies showed vitamin D deficiency could play a role in alcoholic muscle fiber atrophy (125). The association of low vitamin D level with muscle atrophy has been documented in human studies even though few studies reported this association among alcoholic abusers (126). Likewise, no differences in vitamin D levels were observed between alcoholic abusers and non-alcoholic patients (data not shown) or on vitamin D levels between sarcopenic obese and non-sarcopenic obese patients in our study.

Alcohol abuse has also been associated with a lower body weight and FM because of alcoholism-induced malnutrition though whether malnutrition is caused by alcohol itself or alcohol-related organ dysfunction remains unknown (127). Addolorato et al. (128) showed a low %BF and constant LBM in patients suffering from alcohol abuse and suggested that as alcohol cannot be utilized as an energy source, fat oxidation and water content of FFM both increase. This is contradictory to our results likely due to the lack of information on the duration and dosage of alcohol consumed by our patients since the authors suggested that three months are required to restore nutritional status of alcoholics (128).

In our study, the prevalence of sexual dysfunction was significantly different between sarcopenic obese and non-sarcopenic obese patients ($P=0.030$). Seidman et al. (129) suggested
that testosterone deficiency was most consistently associated with sexual dysfunction and could be reversed by testosterone replacement therapy in aging men. In a study attempting to determine the relationship between testosterone, inflammation and symptom burden among male cancer patients, testosterone levels were predictive of erectile function in these patients and testosterone measures were potential markers of increased symptom burden, including sexual dysfunction (130). It is intriguing that grip strength was also predicted by testosterone levels, in addition, testosterone level was predicted by C-reactive protein levels, suggesting low testosterone level and high inflammatory cytokines might affect muscle mass and sexual function differently (130). Our finding is consistent with a recent report of lower muscle mass and higher fat mass in male patients with late-onset hypogonadism (131) and is also consistent with a previous report of the concurrent high prevalence of sexual dysfunction (64.1%) and muscle/joint problems (86.3%) in postmenopausal women (132). Unfortunately, the mechanisms involving sexual dysfunction and sarcopenic obesity are unknown, but we hypothesize that the high prevalence of sexual dysfunction among our sarcopenic obese patients could be related to hormone deficiency and increased inflammation.

Low back pain emerged as a significant condition associate with sarcopenic obesity. The prevalence of low back pain is estimated at 15% to 20% among American adults and 50% to 80% experience at least one episode of low back pain during lifetime (133). People with low back pain often experience a vicious cycle of pain and disuse, suffering from impaired physical function and reduced quality of life caused by little strength, endurance and flexibility in their lower back (134). In addition, low back pain accounts for one-third of total compensation costs and leads to approximately 40% of absenteeism from work (134). The present study asserts that FMI/FFMI ratio was a better predictor of back pain compared to conventional anthropometric parameters. Sarcopenic obese patients presented with a greater prevalence of low back pain compared to their counterparts. To the best of our knowledge, no previous studies have examined the relationship between the FMI/FFMI ratio and low back pain and most studies have relied on anthropometric measures reporting conflicting findings. A systematic literature review reported that body weight or BMI were not strongly associated with low back pain (135). In contrast, Urquhart et al. (136) reported BMI was related to higher levels of back pain intensity. Furthermore, by using dual radiograph absorptiometry, the researchers also reported that individuals with high total body and lower limb fat mass were at higher risk for suffering from
low back pain, independent of LBM (136). On the contrary, a study sample from the Health, Aging and Body Composition (Health ABC) study suggested that the onset of low back pain may be caused by poor trunk muscle composition, assessed by CT (137). Additionally, Monteiro et al. (95) reported a pressure increase in different foot areas among sarcopenic obese postmenopausal women, suggesting that high levels of FM loaded on the lower extremity placed the patients at higher risk of functional impairment. Other possible mechanism includes an increased fat load per muscle unit, or a pro-inflammatory milieu leading to muscle atrophy and eventually low back pain (138).

No differences were observed in self-reported physical activities levels between sarcopenic obese and non-sarcopenic obese patients. Previous studies have reported conflicting results. Two studies concluded that sarcopenia and sarcopenic obesity were not associated with reduced physical capacity when compared to non-sarcopenic obese or normal groups in older men and women (74, 139). On the contrary, Baumgartner et al. (16) showed that low muscle mass was predictive of decreased daily living activities and that sarcopenic obese individuals were 2.6 times more likely to develop physical incapacity than other groups (sarcopenic, obese or normal individuals) in a longitudinal cohort (16). Additionally, the authors reported that sarcopenic obesity was associated with a lower physical capacity compared to other body composition phenotypes (4). Unfortunately, we were unable to use validated and comparable measures of physical activity and were therefore unable to make meaningful assumptions and comparisons with our results.

5.4 Limitations and Future Research

The use of the gender-specific median of FMI/FFMI ratio to classify sarcopenic obese and non-sarcopenic obese patients was a limitation. Patients above the ratio median are not necessarily sarcopenic obese as this may be a fairly inclusive rather than exclusive cutpoint. Unfortunately, we were unable to explore other cutpoints such as quantiles of FMI/FMMI due to our relatively small sample size. It is likely that using a less inclusive cutpoint would significantly affect the association of the FMI/FFM ratio with the variables hereby studied, nonetheless, we were still able to report interesting results such as the association of a higher FMI/FFMI ratio (depicting a sarcopenic obese phenotype) with low back pain. Previous studies have also demonstrated the potential use of body composition ratios. Auyeung et al. reported that
adiposity to muscle ratio was predictive of physical limitation in a large cohort of older adults (110). Sternfeld et al. (24) showed a higher lean mass/fat mass ratio was associated with faster walking speed and less limitation. Likewise, the ratio of FFM/ body surface area has been suggested to be a better index than BMI alone in clinical oncology (24).

As mentioned above, the use of the median as a cutpoint may underestimate the occurrence and significance of sarcopenic obesity, especially in women who presented with a less variable FFM than men. Furthermore, the use of the FMI/FFMI ratio does not account for differences in muscle quality, which may be a more sensitive marker of sarcopenic obesity (140). Another limitation of our study included the retrospective, cross-sectional design and the relatively small sample size, with a smaller proportion of men. In addition, the health status information was self-reported, introducing the problem of interpretation/definition of each individual comorbidity, as well as mis-reporting the severity of the health problem (e.g. exaggeration or under-reporting of physical function and physical activity). All these factors may have affected the significant of sarcopenic obesity as a predictor of health in this patient population. Likewise, no cause-effect relationships between the FMI/FFMI ratio and health outcomes can be inferred.

We were also not able to collect potential confounder variables such as dietary intake and menopausal status, which may substantially impact body composition (141). Last but not least, patients were not fasting for BIA measurements and as mentioned in Chapter Two, food and beverage consumed prior to BIA measurement might affect impedance but to which extent remains unclear (92). Likewise, the use of BIA as a two compartment method (FM and FFM) is also limited as the FFM compartment includes both LBM and bone mineral mass.

Our study is however the first to investigate the prevalence of abnormal body composition among a relative young cohort of morbidly obese individuals. As this is an ongoing study, we anticipate that further associations will emerge as our sample size increases.

Future studies should prioritize establishing a consensus definition and diagnostic criteria for sarcopenic obesity and its related impact on metabolic abnormalities and health outcomes. The use of state-of-the-art tools to assess body composition in morbidly obese patients (e.g. DXA) is also recommended for a more in depth investigation of the biological validity of sarcopenic obesity. The further understanding of this condition should ultimately lead to appropriate strategies to prevent/ treat sarcopenic obesity.
APPENDIX A
APPROVALS

Office of the Vice President For Research
Human Subjects Committee
P O Box 3062742
Tallahassee, Florida 32306-2742
(850) 644-8673 • FAX (850) 644-4392

APPROVAL MEMORANDUM (for change in research protocol)

Date: 12/03/2012

From: Thomas L. Jacobson, Chair

Re: Use of Human subjects in Research
Project entitled: Prevalence and Characteristics of Sarcopenic Obesity in a Bariatric Population

The application that you submitted to this office in regard to the requested change/amendment to your research protocol for the above-referenced project has been reviewed and approved.

Please be reminded that if the project has not been completed by 08/12/2013, you must request renewed approval for continuation of the project.

By copy of this memorandum, the chairman of your department and/or your major professor is reminded that he/she is responsible for being informed concerning research projects involving human subjects in the department, and should review protocols as often as needed to insure that the project is being conducted in compliance with our institution and with DHHS regulations.

This institution has an Assurance on file with the Office for Human Research Protection. The Assurance Number is IRB00000446.

Cc:
HSC NO. 2012 9569
1. Project Title and Identification

1.1 Project Title

Prevalence and Characteristics of Sarcopenic Obesity in a Bariatric Population

Project is: Thesis

1.2 Principal Investigator (PI)

Name (Last name, First name MI): Prado, Carla M. M.

Highest Earned Degree: Doctorate

Mailing Address: 412 Sandels Building, 120 Convocation Way POBox 3061493 Campus Code: 1493

Phone Number: 6451522

Fax:

University Department: NUTRITION FOOD AND EXERCISE SCIENCES

Email: cprado@fsu.edu

The training and education completed in the protection of human subjects or human subjects records: NIH

Occupational Position: Faculty

1.3 Co-Investigators/Research Staff

Name (Last name, First name MI): Ormsbee, Michael ; Co-Investigator

Highest Earned Degree: Doctorate

Mailing Address: 430 Sandels Building Tallahassee, FL

Phone Number: (850) 644-4793

Fax: (850) 645-5000

University Department: NUTRITION FOOD AND EXERCISE SCIENCES

Email: Mormsbee@fsu.edu

The training and education completed in the protection of human subjects or human subjects records: FSU Training Module

Occupational Position: Faculty

Name (Last name, First name MI): 

Highest Earned Degree:
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November 13, 2012

Carla Prado  
PO 3081493  
Campus Code 1493  
Tallahassee, FL 32306-149  

Dear Dr. Prado:

Your study IRB # 2012-40 Titled: "Prevalence and Characteristics of Sarcopenic Obesity in a Bariatric Population: a Retrospective Study Disease Patients" met the criteria for review using the expedited review guidelines. - Joel Kramer, MD, Vice-Chairperson, Institutional Review Board (IRB) at Tallahassee Memorial HealthCare, Inc. (TMH) reviewed the study, the supporting documentation and approved the study on November 6, 2012, 2012 for one year. The expiration date of this approval is November 5, 2013.

IRB # 2012-40:

"Prevalence and Characteristics of Sarcopenic Obesity in a Bariatric Population: a Retrospective Study"

Principal Investigator: Carla Prado

Co-principal Investigators:

Michael Ormsbee, Jeong -Su Kim, Angelina Cain

Informed Consent: NA - retrospective chart review


Material Reviewed and Approved: Sarcopenic Obesity in Bariatric Patients data collection tool

Reporting Requirements:

- Report to the IRB any planned change in the study or informed consent and do not implement any change without receiving prior approval, except to eliminate immediate hazard;

FWA #00001868  
Tallahassee Memorial HealthCare, Inc. Institutional Review Board is organized and operates according to ICH-GCP standards and applicable laws and regulations.
- Report to the IRB any unanticipated problems involving risks to subjects;
- Report to the IRB any new information on the project that adversely influences the risk/benefit ratio;
- Report to the IRB any serious or unexpected adverse events per IRB guidelines;
- Report to the IRB any major protocol violations within ten days. Minor protocol deviations may be reported at the time of the Study Progress Report (Application for Renewal). Maintain a log throughout the year and establish a plan of correction to minimize the deviations.
- Report to the IRB when the study is terminated or completed and submit a summary of the study findings.

Please request approval for advertising copy, recruitment flyers, publications, that appear in any medium prior to use.

**Supplemental Reporting Requirements:** None

**Expiration Date:** November 5, 2013

**Continuation Review Requirements:**
At the time of renewal please check the Office of Research/IRB intranet site to ensure that you have the most current edition of the IRB Forms. The investigator must submit a completed Study Progress Report Application and supporting documentation packet to the Office of Research/IRB one month prior to the approval expiration date. Please note the expiration date to ensure timely review and processing of the study file prior to the study’s approval expiring. If you have any questions about the forms or submitting them, please contact the Office of Research/IRB, at (850) 431-5676.

As the principal investigator you are responsible for ensuring compliance with the study protocol, the applicable IRB at TMH Guidelines and Code of Federal Regulations set forth by the Department of Health and Human Services. The IRB Guidelines and forms required to comply with reporting requirements are available on the TMH Intranet.

Sincerely,

Cynthia Blair
Administrative Liaison/IRB

cc: Paula Fortunas, TMH Foundation (w/o attachments)
APPENDIX B
DATA COLLECTION FORM

Sarcopenic Obesity in Bariatric Patients

TMH Chart Number: [ ] FSU STUDY ID: [ ]

Date of Baseline Visit (initial consultation) ___/___/_______
mm   dd    yyyy

Date of BIA Scan ___/___/_______
mm   dd    yyyy

Demographics

Date of Birth ___/___/_______   Age______   Sex:  [ ] Male  [ ] Female
mm   dd    yyyy

Ethnicity_________

Household Income

[ ] 0 - 30,000   [ ] 30,000 – 50,000   [ ] 50,000 – 75,000

[ ] 75,000 – 10,000   [ ] >100,000

Current Highest Level of Education

[ ] Grade School   [ ] High School   [ ] College

[ ] Post-graduate

Body Composition Measurements

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Waist circumference = _______ inches

**Weight History**

Weight gain:  Gradual ☐ Rapid ☐

Onset: ☐ Childhood ☐ Puberty ☐ Pregnancy ☐ Adult Menopausal

Associated with life changes: ☐ College ☐ Career ☐ Marriage

☐ Divorce ☐ Death ☐ Stress

Weight loss attempts: ☐ Yes ☐ No

Eating Disorder: ☐ Yes ☐ No

**Physical/Lifestyle assessment**

**Blood Pressure** _____/____ mmHg

syst.  diast.

**Medical and Surgical History**

☐ Acid Reflux/Heartburn ☐ Diabetes ☐ Kidney Disease

☐ Anemia ☐ DVT ☐ Liver Disease

☐ Anxiety ☐ Gout ☐ Lung Disease

☐ Asthma ☐ Glaucoma ☐ Osteoporosis
[ ] Arthritis  [ ] Gallbladder Disorder  [ ] Polycystic Ovaries
[ ] Cancer (Type__)  [ ] Heart Attack  [ ] Pulmonary Embolism
[ ] Carotid Disease  [ ] Heart Disease  [ ] Rheumatic/Scarlet Fever
[ ] Chest Pain  [ ] Heart Murmur  [ ] Sleep Apnea
[ ] Clotting Disorder  [ ] High Blood Pressure  [ ] Stroke
[ ] COPD  [ ] High Cholesterol  [ ] Thyroid Disease
[ ] Depression  [ ] Irregular Menstrual Cycle  [ ] Vascular Disease

Other:_________________________________________________________

Surgery:_____________ Location:___________ Date:_________

Surgery:_____________ Location:___________ Date:_________

Surgery:_____________ Location:___________ Date:_________

Surgery:_____________ Location:___________ Date:_________

Surgery:_____________ Location:___________ Date:_________

Surgery:_____________ Location:___________ Date:_________

Psychiatric History

[ ] Anxiety  [ ] Alcoholism  [ ] Anorexia
[ ] Depression  [ ] Drug Addiction  [ ] Binge Eating
[ ] Bipolar Disorder  [ ] Schizophrenia  [ ] Bulimia
[ ] Panic Attacks  [ ] Nervous Breakdown  [ ] Stress

Review of Systems: Please check any problems you have had over the last month.

[ ] Chest Pain  [ ] Frequent Urination  [ ] Headache
[ ] Leg Pain/Swelling  [ ] Increased Hunger  [ ] Dry Skin
[ ] Varicose Veins  [ ] Increased Thirst  [ ] Weakness
[ ] Acid reflux  [ ] Numbness or Tingling  [ ] Joint Pain
[ ] Abdominal Pain  [ ] Incontinence  [ ] Hemorrhoids
[ ] Nausea  [ ] Sexual Dysfunction  [ ] Hair loss
[ ] Vomiting  [ ] Snoring  [ ] Fatigue
[ ] Hernia  [ ] Fever  [ ] Low Back pain
Shortness of Breath  Cough  Irregular Heartbeats
Arm Pain  Chills  Brittle Hair/Nails
Cold Intolerance  Constipation  Dumping Syndrome
Heat Intolerance  Diarrhea  Immobility
Menstrual Changes  Dizziness  Diminished Hygiene
Infertility  Blurred Vision  Nose Bleeds

Social History

☐ Single  ☐ Divorced  ☐ Married

☐ Partner  ☐ Widow/Widower
Tobacco  ☐ Yes  ☐ No  How often? ____________
Drugs  ☐ Yes  ☐ No  How often? ____________
Alcohol  ☐ Yes  ☐ No  How often? ____________

Gynecologic History (For women only)

History of:  Gestational Diabetes  ☐ No  ☐ Yes
Menstrual Periods:  Hysterectomy  ☐ No  ☐ Yes
Hormone replacement  ☐ No  ☐ Yes

Laboratory Results

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</thead>
<tbody>
<tr>
<td>Cholesterol, Total</td>
<td></td>
<td>mg/dL</td>
<td>125-200 mg/dL</td>
</tr>
<tr>
<td>HDL- Cholesterol</td>
<td></td>
<td>mg/dL</td>
<td>&gt; OR=46 mg/dL</td>
</tr>
<tr>
<td>Triglycerides</td>
<td></td>
<td>mg/dL</td>
<td>&lt;150 mg/dL</td>
</tr>
<tr>
<td>LDL-Cholesterol</td>
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<td>mg/dL</td>
<td>&lt;130 mg/dl(calc)</td>
</tr>
<tr>
<td>Chol/HDL C Ratio</td>
<td></td>
<td>&lt; OR=5.0 (calc)</td>
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</tr>
<tr>
<td>Non-HDL Cholesterol</td>
<td></td>
<td>mg/dL</td>
<td>mg/dl(calc)</td>
</tr>
<tr>
<td>Glucose</td>
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<td>mg/dL</td>
<td>65-99 mg/dL</td>
</tr>
<tr>
<td>Urea Nitrogen (BUN)</td>
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<td>mg/dL</td>
<td>7-25 mg/dL</td>
</tr>
<tr>
<td>Creatinine</td>
<td></td>
<td>mg/dL</td>
<td>0.50-1.05 mg/dL</td>
</tr>
<tr>
<td>eGFR Non-AFR. American</td>
<td></td>
<td>ML/min/1.73m²</td>
<td>&gt; OR=60</td>
</tr>
<tr>
<td>eGFR AFR. American</td>
<td></td>
<td>ML/min/1.73m²</td>
<td>&gt; OR=60</td>
</tr>
<tr>
<td>Test</td>
<td>Unit</td>
<td>Range</td>
<td></td>
</tr>
<tr>
<td>-------------------------------------</td>
<td>------------------</td>
<td>----------------</td>
<td></td>
</tr>
<tr>
<td><strong>BUN/Creatinine Ratio</strong></td>
<td>mi/min/1.73m²</td>
<td>6-22 (calc)</td>
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</tr>
<tr>
<td>Sodium</td>
<td>nmol/L</td>
<td>135-146</td>
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</tr>
<tr>
<td>Potassium</td>
<td>nmol/L</td>
<td>3.5-5.3</td>
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</tr>
<tr>
<td>Chloride</td>
<td>nmol/L</td>
<td>98-110</td>
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<tr>
<td>Carbon Dioxide</td>
<td>nmol/L</td>
<td>21-33</td>
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<tr>
<td>Calcium</td>
<td>mg/dL</td>
<td>8.6-10.4</td>
<td></td>
</tr>
<tr>
<td>Protein, Total</td>
<td>g/dL</td>
<td>6.2-8.3</td>
<td></td>
</tr>
<tr>
<td>Albumin</td>
<td>g/dL</td>
<td>3.6-5.1</td>
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</tr>
<tr>
<td>Globulin</td>
<td>g/dL</td>
<td>2.2-3.9</td>
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<tr>
<td>Albumin/Globulin Ratio</td>
<td>(calc)</td>
<td>1.0-2.1 (calc)</td>
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</tr>
<tr>
<td>Bilirubin, Total</td>
<td>mg/dL</td>
<td>0.2-1.2</td>
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<tr>
<td>Alkaline Phosphatase</td>
<td>U/L</td>
<td>33-130</td>
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</tr>
<tr>
<td>AST</td>
<td>U/L</td>
<td>10-35</td>
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</tr>
<tr>
<td>ALT</td>
<td>U/L</td>
<td>6-40</td>
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</tr>
<tr>
<td>Vitamin D, 25-OH, Total</td>
<td>ng/mL</td>
<td>30-100</td>
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<tr>
<td>Vitamin D, 25-OH, D3</td>
<td>ng/mL</td>
<td>&gt; OR=20 Years</td>
<td></td>
</tr>
<tr>
<td>Vitamin D, 25-OH, D2</td>
<td>ng/mL</td>
<td>0.40-4.50 mIU/L</td>
<td></td>
</tr>
<tr>
<td>TSH</td>
<td>mIU/L</td>
<td>&gt; OR=20 Years</td>
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<tr>
<td>T4, Free</td>
<td>ng/dL</td>
<td>0.8-1.8</td>
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</tr>
<tr>
<td>T3, Free</td>
<td>pg/mL</td>
<td>2.3-4.2</td>
<td></td>
</tr>
<tr>
<td>White Blood Cell Count</td>
<td>Thousand/uL</td>
<td>3.8-10.8</td>
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<tr>
<td>Red Blood Cell Count</td>
<td>Million/uL</td>
<td>3.80-5.10</td>
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</tr>
<tr>
<td>Hemoglobin</td>
<td>g/dL</td>
<td>11.7-15.5</td>
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</tr>
<tr>
<td>Hematocrit</td>
<td>%</td>
<td>35.0-45.0%</td>
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</tr>
<tr>
<td>MCV</td>
<td>fL</td>
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<td>MCH</td>
<td>pg</td>
<td>27.0-33.0</td>
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<tr>
<td>MCHC</td>
<td>g/dL</td>
<td>32.0-36.0</td>
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</tr>
<tr>
<td>RDW</td>
<td>%</td>
<td>11.0-15.0%</td>
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</tr>
<tr>
<td>Platelet Count</td>
<td>Thousand/uL</td>
<td>140-400</td>
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## PATIENT MEDICATION LIST

<table>
<thead>
<tr>
<th>Medication name</th>
<th>Dose</th>
<th>Taken by:</th>
<th>Frequency (times per day)</th>
<th>Medication used for…</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td><strong>By mouth</strong></td>
<td><strong>Injection</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>By mouth</strong></td>
<td><strong>Injection</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
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</tr>
<tr>
<td></td>
<td></td>
<td><strong>By mouth</strong></td>
<td><strong>Injection</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
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</tr>
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<td></td>
<td><strong>By mouth</strong></td>
<td><strong>Injection</strong></td>
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<td><strong>_____</strong></td>
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<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
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</tr>
<tr>
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<td><strong>_____</strong></td>
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<td><strong>_____</strong></td>
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<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
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</tr>
<tr>
<td></td>
<td></td>
<td><strong>By mouth</strong></td>
<td><strong>Injection</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Inhaled</strong></td>
<td><strong>_____</strong></td>
<td></td>
</tr>
</tbody>
</table>

52
Tallahassee Memorial Bariatric Center
Bariatric Nutrition Initial Assessment Form

Weight History
How many years have you been overweight? _____
How many times have you tried to lose weight? ____
What is your maximum adult weight (non-pregnant) and when? ______________
When did you start to gain excess weight and why? ______________

PAR-Q & YOU

☐ YES ☐ NO 1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

☐ YES ☐ NO 2. Do you feel pain in your chest when you do physical activity?

☐ YES ☐ NO 3. In the past month, have you had chest pain when you were not doing physical activity?

☐ YES ☐ NO 4. Do you lose your balance because of dizziness or do you ever lose consciousness?

☐ YES ☐ NO 5. Do you have a bone or joint problem (for ex: back, knee, or hip) that could be made worse by a change in your physical activity?

☐ YES ☐ NO 6. Is your doctor currently prescribing drugs (for ex: water pills) for your blood pressure or heart condition?

☐ YES ☐ NO 7. Do you know of any other reason why you should not do physical activity?
Exercise History and Attitude Questionnaire

1. Please rate your exercise level on a scale of 1 to 5 (1= easy, 5= very strenuous) at each age.

   Age: 15-20 ____ 21-30 _____ 31-40 _____ 41-50 ____ 51-60 ____ 61+ _____

2. Were you a high school and/or college athlete?
   □ YES  □ NO  If yes, please explain: ___________

3. Rate yourself on a scale of 1 to 5 (1= least and 5= most)
   - Athletic Ability
   - Competition
   - Cardiovascular Capacity
   - Muscular Capacity
   - Flexibility Capacity

4. When you start an exercise program
   □ I stick with it until I accomplish my goal.
   □ I stick with it most of the time.
   □ I’m good for a month and then miss a month and then back on again repeatedly.
   □ I usually don’t stick with it very long and then quit.

5. How much time are you willing to devote to an exercise program?
   _____ minutes per day _____ days per week

6. Do you currently do cardiovascular exercise?
   Type(s): ___________ ___________ minutes per day _____ days per week

7. Rate your perception of exertion during your cardiovascular exercise.
   □ Light  □ Fairly Light  □ Somewhat Hard  □ Hard

8. How long have you been exercising regularly?
   _____ months _____ years

9. What other exercise, sport or active recreational activities have you participated in?
   In the past 6 months? _____
   In the past 5 years? _____

10. Can you exercise during your work day?  □ Yes  □ No

11. What types of exercise interest you?
    □ Walking  □ Cycling  □ Stair Climbing  □ Jogging  □ Group Exercise  □ Yoga/Pilates
    □ Elliptical  □ Swimming  □ Strength Training  □ Racquet Sports  □ Rock Climbing
Other______________
12. What do you want exercise to do for you? ______________________________
13. Rate each goal separately: 1 Not Important  2 Somewhat Important  3 Extremely Important
   a. Improve cardiovascular fitness ___
   b. Lose weight ___
   c. Lose body fat ___
   d. Reshape my body ___
   e. Improve performance for sports or other activity___
   f. Improve my ability to cope with stress___
   g. Improve flexibility____
   h. Increase strength___
   i. Improve balance___
   j. Increase energy level___
   k. Feel better___
   l. Prevent/treat a medical condition___
14. How many pounds would you like to lose? ____ pounds
15. What is your usual pace of walking?
   a. _____casual or strolling(less than 2 mph)
   b. _____average or normal (2-3mph)
   c. _____fairly brisk (3-4 mph)
   d. _____brisk or striding (>4mph)
16. How many flights of stairs do you climb each day? _____flights/day
17. How many hours do you spend watching TV per day? ____hours/day
18. How many hours do you spend using your computer? ____hours/day
19. How much of your work day is spent at a desk? ___hours/day
20. How much of your work day is spent waking around? ___hours/day
21. How much of your day is spent standing in one spot? ____hours/day
22. At least once/week do you participate in regular activity like brisk walking, jogging, biking, swimming, etc. long enough to work up a sweat?
   □ No   □ Yes   How many times/week? ________    Activity________

Completed by: _________________
Date: ____/____/_______
   mm   dd   yyyy
REFERENCES


adipose tissue is associated with metabolic disorders in morbidly obese patients. Obes Surg 2010;20:77-83.


139. Bouchard DR, Dionne IJ, Brochu M. Sarcopenic obesity and physical capacity in older men and women: data from the Nutrition as a Determinant of Successful Aging (NuAge)-the Quebec longitudinal Study. Obesity (Silver Spring) 2009;17:2082-2088.


BIOGRAPHICAL SKETCH

JINGJIE XIAO

Jingjie was born in Gongyi, Henan, China to Jianli Xiao and Xianfeng Yang. She attended high school at No. 2 High School in Gongyi. Jingjie majored in Food Quality and Safety in Shandong Agricultural University for a bachelor’s degree in 2007. After she graduated from college, she continued to pursue a master’s degree at the Florida State University in 2011; and Jingjie switched majors to Nutrition Sciences. During her master’s study, Jingjie received China linkage Scholarship for 2012-2013 term and she was also awarded the Pao-Sen Chi Scholarship in 2012. During 2012-2013, Jingjie completed her master’s course work and started working on her thesis project. She volunteered in the Youth Program in 2011 and later on participated in the Understanding Nutrition Now (SUNN) as a peer health educator. Jingjie has been dedicated with doing research and will continue to pursue a PhD degree under the guidance of Dr. Carla Prado after her graduation.