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# Sarcopenic Obesity in Cancer Patients: Focus on Pathogenesis

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ABSTRACT

**Introduction:** Sarcopenic obesity is an emerging problem in cancer patients. However, this is often difficult to diagnose without the measurement of body composition. Sarcopenic obesity is associated with increased mortality, chemo-toxicity, and other complications in cancer patients. Until now, there is scarce information about sarcopenic obesity in the cancer population.

**Method:** We identified 1955 articles related to sarcopenic obesity in adult cancer patients using PubMed, PubMed Central, and Cochrane Library databases from January 1, 1989, until January 1, 2020. Firstly, we screened the titles and abstracts which mentioned sarcopenic and obesity, especially in pathogenesis. 29 articles could proceed to the next step; then, they were screened for the full text. All steps were reviewed by two authors.

**Results:** At last, they were 9 articles included. Sarcopenic obesity is defined as the coexistence of sarcopenia and obesity, an increase in fat mass in the body. Sarcopenic obesity carried cumulative risks from each of the two individual body compositions. CT-scan offers the highest available precision in determining body composition parameters, especially in the cancer population. Multiple causes and interactions between hormonal changes, aging, disuse, neuronal, poor nutrition, physical inactivity, and low-grade inflammation played roles. Sarcopenic obesity is associated with chemotherapy toxicity. High protein intake should be initiated to ensure adequate protein intake. Resistance training is beneficial in improving muscle mass and strength by focusing on strength training, flexibility, and balance.

**Conclusions:** Sarcopenic obesity is an emerging problem but is often neglected. Further research needs to be conducted especially in explaining the pathogenesis of sarcopenic obesity. The combination of physical exercise and diet modification is the best management to improve sarcopenia obesity in cancer patients.

## INTRODUCTION

Sarcopenic obesity is a chronic condition characterized by the low skeletal muscle mass and function in the context of excess adiposity and correlates with a decline in physical strength and status. It is a major public health problem with increasing prevalence worldwide. Sarcopenia is a progressive and generalized skeletal muscle disorder that is associated with adverse outcomes (falls, fractures, physical disability, and mortality). The excess adiposity in a sarcopenic person leads to sarcopenic obesity and may exacerbate fat infiltration into the muscle, which further lowers physical function and increases the risk of mortality [1,2]. Thus, both sarcopenia and sarcopenic obesity play a role as an independent prognostic factor in cancer patients [3–5].

Body composition plays a significant role in oncology, referring to the amount and distribution of lean and adipose tissues in the human body. One of the best-known parameters in assessing human anthropometry is body mass index defined as weight/height [2]. Body mass index alone has limitations, especially in the settings of sarcopenic obesity where a patient loses muscle mass but gains adipose tissues. Body composition is an important feature because it affects the distribution of the chemotherapy drugs, thus, affecting efficacy, toxicity in chemotherapy patients, major postoperative complications, and outcomes in cancer patients [6–10].

In conditions related to autoimmune and cancer population, lean body mass is more likely to decrease while fat mass may be preserved or even increased [11]. This may mask the body weight because of the preserved fat mass. It is another thing to consider that fat may infiltrate into muscle and may cause low muscle quality and interfere with muscle performance. These findings are well documented in the elderly where intramuscular and visceral fat increases with aging and subcutaneous fat declines [12–14].

The current definition of sarcopenic obesity relies on the combination of obesity and sarcopenia. This makes the diagnosis of sarcopenic obesity is challenging when the definition of obesity and sarcopenia itself is not clear, leading to confusion and preventing inter-study comparisons [15]. In this review, we look further into current articles to evaluate the pathogenesis, diagnosis, impacts, and intervention of sarcopenic obesity in cancer patients.

## METHODS

### Search Strategies

In searching the articles, the researcher used several medical subject headings (MeSH) such as "sarcopenic", "muscle mass", "muscle strength", "obesity", "sarcopenic obesity", "cancer" in three electronic databases (PubMed, PubMed central, Cochrane library) from January 1, 1989, to January 1, 2020 (**Table 1**). The articles were included in this study if several inclusion criteria below were met: 1. Population included in the studies were cancer patients younger than 60 years old; 2. Interest: patients with sarcopenic obesity, either low muscle mass diagnosed using CT-scan, DEXA, or BIA and concomitant with BMI >25 kg/m$^2$; 3. Comparison or control: non-sarcopenic obesity; 4. Outcomes: the outcome of this study is to evaluate pathogenesis, diagnosis, impact, and intervention; 5. Study design: observational study, interventional study, and article review. Articles were excluded if any exclusion criteria were met: Population with hematological cancer.

### Data Extraction

Several details were extracted, including the author, the country of the study population, inclusion/exclusion criteria, and the type of cancer. The data were also extracted considering the definition of sarcopenia used (low muscle mass and or strength), methods used in defining sarcopenic obesity (CT-scan, DEXA, BIA), and study outcomes (e.g., pathogenesis, diagnosis, impact, and treatment).

Multiple articles were read and appraised based on the Newcastle-Ottawa Validity test. Further information about the characteristics of the studies can be seen in **Table 2**.

### Quality Assessment and Evidence

The Newcastle-Ottawa quality assessment of cohort trials was used to measure the risk of bias. Both reviewers assessed the methodological qualities and standards in reporting the outcomes of the studies. The quality of the evidence was graded according to the Agency for Healthcare Research and Quality (AHRQ). The level of evidence was presented as good, fair, and poor. The assessment of the studies can be seen in **Table 3**.

**Table 1.** Literature search startegy

| Database | Keyword | Result |
|---|---|---|
| PubMed | (("sarcopenia"[title/abstract] OR "muscle mass"[title/abstract] OR "muscle strength"[title/abstract]) AND ("obesity"[title/abstract] OR "sarcopenic obesity"[title/abstract]) AND "cancer"[title/abstract]) | 132 |
| Cochrane library | "sarcopenia" OR "muscle mass" OR "muscle strength" AND "obesity" OR "sarcopenic obesity" AND "cancer" | 35 |
| PubMed Central | (Sarcopenic[All Fields] AND ("obesity"[MeSH Terms] OR "obesity"[All Fields]) AND ("neoplasms"[MeSH Terms] OR "neoplasms"[All Fields] OR "cancer"[All Fields])) | 1788 |

**Table 2.** Newcastle-Ottawa quality assessment of cohort observational trials

| | Prado 2008 | Malietzis 2016 | Rier 2017 | Cushen 2017 | Tan 2009 | Rollins 2016 | Anandavadivelan 2016 | Palmela 2017 | Heidelberger 2017 |
|---|---|---|---|---|---|---|---|---|---|
| **Selection** | | | | | | | | | |
| Representative of the cohort? | * | * | * | * | * | * | * | * | * |
| Selection of the non-exposed | * | * | * | * | * | * | * | * | * |
| Ascertainment of exposure | * | * | * | * | * | * | * | * | * |
| **Demonstration that outcome was not present at start of study** | | | | | | | | | |
| Comparability | | | | | | | | | |
| Comparability on basis of design or analysis | | | | | | | | | |
| **Outcome** | | | | | | | | | |
| Assessment of outcome | * | * | * | * | * | * | * | * | * |
| Was follow-up long enough for outcomes to occur | * | * | * | * | * | * | * | * | * |
| Adequacy of follow-up of cohorts | * | * | * | * | * | * | * | * | * |
| **Results** | Fair | Fair | Fair | Fair | Fair | Fair | Fair | Fair | Fair |


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**Table 3.** Characteristics of the studies

| Author, years, country | Subjects | Cancer sites | CT MRI DXA BIA | Sarcopenia criteria | Obesity criteria | Sarcopenic obesity % | NDQ | Outcome |
|---|---|---|---|---|---|---|---|---|
| Prado et al, 2008, Canada | Retro-spective data | Solid tumor of respiratory tract and Gastro-intestinal tract | CT | CT L3 SMI: men ≤52.4 $cm^2/m^2$; women ≤38.5 $cm^2/m^2$; cut-offs by optimal stratification | BMI ≥30 $kg/m^2$ | NA | Fair | Sarcopenic obesity was associated with poorer functional status compared with obese patients who did not have sarcopenia (p=0.009), and was an independent predictor of survival (hazard ratio [HR] 4.2 [95% CI 2.4–7.2], p<0.0001). |
| Malietzis et al, 2016, UK | Retro-spective data | Resectable colorectal cancer | CT | CT L3 SMI: men ≤52.4 $cm^2/m^2$; women ≤38.5 $cm^2/m^2$; cut-offs by optimal stratification | BMI ≥30 $kg/m^2$ | 9.9 | Fair | The presence of myosteatosis was associated with prolonged primary hospital stay (P=0·034), and myopenic obesity was related to higher 30-day morbidity (P=0·019) and mortality (P<0·001) rates. |
| Rier et al, 2017, Netherlands | Retro-spective data | Breast cancer | CT | SMI ≤41 $cm^2/m^2$ | BMI ≥27.5 $kg/m^2$ | 2.3 | Fair | Low muscle mass (LMM) and sarcopenic obesity were not associated with overall survival (OS) (median OS 19 vs. 18 months, p=0.845 for LMM and 20 vs. 18 months, p=0.481 for sarcopenic obesity |
| Cushen et al, 2017, Ireland | Retro-spective data | Prostate cancer | CT | CT L3 SMI: female <41 $cm^2/m^2$; men <43 (BMI <25 $kg/m^2$) and <53 (BMI ≥25) | BMI ≥27.5 $kg/m^2$ | 12.6 | Fair | No statistically significant differences were noted regarding the occurrence of DLT (p=0.827) and sarcopenic obesity was not predicative of DLT (p=0.511) |
| Tan et al, 2009, Canada | Retro-spective data | Prostate cancer | CT | CT L3 SMI: men ≤52.4 $cm^2/m^2$; women ≤38.5 $cm^2/m^2$; cut-offs by optimal stratification | BMI ≥25 $kg/m^2$ | 16.2 | Fair | Overweight/obese sarcopenia (hazard ratio, 2.07; 95% confidence interval, 1.23–3.50; P=0.006) were identified as independent predictors of survival |
| Rollins et al, 2016, UK | Retro-spective data | Pancreatic cancer | CT | CT L3 SMI: female <41 $cm^2/m^2$; men <43 (BMI <25 $kg/m^2$) and <53 (BMI ≥25) | BMI ≥25 $kg/m^2$ | 25.4 | Fair | Survival rates is lower in myosteatotic and sarcopenic (median 114 days) and those who were myosteatotic but not sarcopenic (median 131 days) were lower than in those who were sarcopenic but not myosteatotic (median survival 280.5 days) (p=0.003). |
| Ananda-vadivelan et al, 2016, Sweden | Retro-spective data | Esopha-geal cancer | CT | CT L3 SMI: men ≤52.4 $cm^2/m^2$; women ≤38.5 $cm^2/m^2$; cut-offs by optimal stratification | BMI ≥25 $kg/m^2$ | 14 | Fair | Sarcopenia combined with overweight/obesity (n=10) showed a significantly higher risk (OR=5.54; 95% CI 1.12–27.44) of toxicity compared to non-sarcopenic obese patients. |
| Palmela et al, 2017, Portugal | Retro-spective data | Gastric cancer | CT | CT L3 SMI: female <41 $cm^2/m^2$; men <43 (BMI <25 $kg/m^2$) and <53 (BMI ≥25) | BMI ≥25 $kg/m^2$ | 10.4 | Fair | Patients with sarcopenic obesity showed lower overall survival (median survival of 6 months [95% confidence interval {CI}=3.9–8.5] vs. 25 months [95% CI=20.2–38.2] |
| Heidel-berger et al, 2016, France | Retro-spective data | Mela-noma | CT | CT L3 SMI: men ≤52.4 $cm^2/m^2$; women ≤38.5 $cm^2/m^2$; cut-offs by optimal stratification | BMI ≥25 $kg/m^2$ | 19 | Fair | Patients who experienced early ALT, the mean BMI was higher (27.9 versus 24.7 $kg/m^2$; p=0.04). Among the 32 female patients, sarcopenic overweight patients had a 6.5-fold increased risk of ALT (50% versus 7.7%; p=0.01). |

Abbreviations: NOQ: Newcastle-Ottawa validity test; CT: computed tomography; MRI: Magnetic Resonance Imaging; BIA: Bio-Electrical Impedance analysis; DXA: Dual-energy X-ray Absorptiometry; BMI: Body mass Index, SMI: skeletal muscle index; NA: not applicable.

## RESULTS

### Literature Search Results

We identified 1955 studies evaluating sarcopenia obesity in cancer patients using PubMed, PubMed Central, and Cochrane Library databases. We removed 7 duplicate articles in our findings. The titles and abstracts of the articles were screened, and 1.919 studies were excluded due to full-text unavailability or the articles not related to both sarcopenia and obesity. 20 studies were excluded because of the wrong assessment of sarcopenia or obesity and obscured report of the outcome. At last, there were 9 articles included in this review. Further information can be seen in **Figure 1**.

### Definition of Sarcopenic Obesity

Multiple definitions of sarcopenia have been proposed, each of which leads to different perspectives. The lack of an accurate definition leads to confusion and difficulties among physicians in diagnosing or assessing patients with suspected sarcopenia. Several anthropometric measurements are being conducted such as mid-arm and calf circumference [4,15,16].

Sarcopenic obesity is defined as the coexistence of sarcopenia and obesity with the latter defined as an increase in fat mass in the body [4]. Sarcopenia normally occurs with aging where a decline of muscle mass is associated with age-related changes in body composition [15]. Combined with the increasing prevalence of obesity, sarcopenia leads to an increased sarcopenic obesity prevalence [17].

According to European Working Group for the study of sarcopenia 2 (EWGSOP2), sarcopenia is defined as a syndrome characterized by decreased muscle mass and function (strength or performance), which increases the risk of disability, poor quality of life, and mortality. As a better predictor than muscle mass, muscle function is used to assess the severity of the sarcopenia [2]. The operational definition of sarcopenia can be seen in **Table 4** [2].

Obesity is defined as unhealthy excess body fat that increases the risk of medical illness and mortality [18]. However, no consensus defines obesity cut-off points. The American Association of Clinical Endocrinologists recommends the use of WHO threshold in diagnosing obesity (men >25% body fat and women >35% body fat) and BMI ≥30 kg/m2 and waist circumference (men >102 cm and women >88 cm) as a surrogate in diagnosing obesity [19]. The current definition of sarcopenic obesity relies on the combination of obesity and sarcopenia. This makes the diagnosis of sarcopenic obesity is challenging when the definition of obesity and sarcopenia itself is not clear, leading to confusion and preventing inter-study comparisons [15].

**Table 4.** Operational definition of sarcopenia [2]

| |
|---|
| Diagnosis based on criteria 1 (probable) and confirmed when criteria 2 are met and considered severe if all of the criterion are all met. |
| Low muscle strength |
| Low muscle quantity or quality |
| Low physical performances |

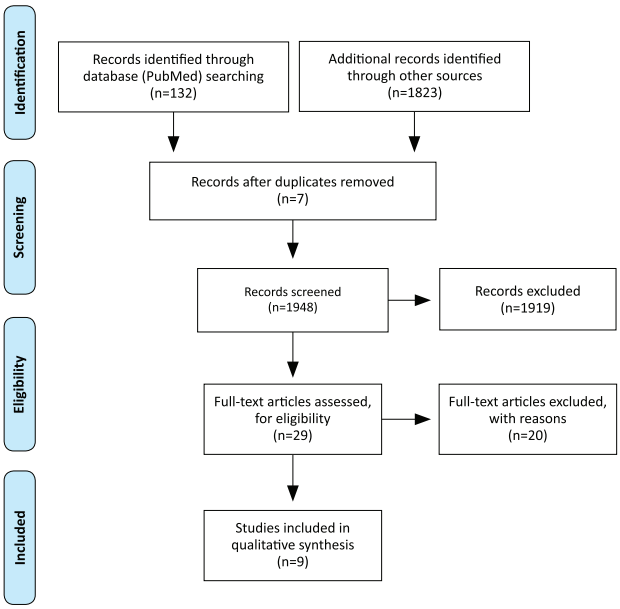


**Figure 1.** PRISMA literature searched flow diagram

### Prevalence of Sarcopenic Obesity in Cancer Patients

The prevalence of sarcopenic obesity is increasing with age. It also influences physical, metabolic, and cardiovascular functions and is a major concern to nutritionists, geriatricians, and public health officers [20]. Sarcopenic obesity may carry cumulative risks from each of the two individual body compositions. Excess obesity may increase cardiovascular risk, and loss of muscle mass in older individuals may be significantly associated with extended hospital stays which contribute to infectious and non-infectious complications and overall mortality [21–25]. Sarcopenic obesity has a prevalence of 9% in the population with advanced solid tumors (range 2.3–14.6%). It also has a prevalence of 24.7% (range 5.9–39.2%) in the population with a body mass index of more than 30 kg/m2. The lowest prevalence of sarcopenic obesity is found in the early-stage disease and the highest in the locally advanced or metastatic disease [26–30].

## Diagnosing Sarcopenia Obesity

Several anthropometric measures can be done to determine body composition. The most reliable instruments to measure the whole-body and fat-free mass are either Dual-energy X-ray absorptiometry (DXA) or computed tomography (CT) scan on the 3rd lumbar vertebra [31]. CT-scan images provide details on specific muscles, adipose tissues, and organs, not visualized on DXA, thus providing great significance in precision to quantify specific tissues and to predict whole-body composition [31]. CT-scan also offers the highest available precision and specificity in determining muscle mass, fat mass, and distribution. Besides, CT-scan is freely available at the medical record and often used for the diagnostic and evaluative procedure. BIA provides a convenient and cost-effective measurement method by measuring based on the disparate electrical conductivity of fat and lead tissues, commonly described as height adjusted in $kg/m^2$ [17].

## Mechanism of Sarcopenic Obesity

Loss of muscle mass occurs due to multiple causes and interactions between hormonal changes, aging, disuse, neuronal, poor nutrition, physical inactivity, and low-grade inflammation. This results in a decrease in oxidative capacities, mitochondrial volumes, protein synthesis, proteolysis, neuromuscular integrity disruption, and alteration in the muscle fat content which leads to a decrease in lean mass [15,32–35].

## Age

Both cancer and sarcopenic obesity prevalence are increasing with age [36]. Most cancers are diagnosed in the elderly while, with aging, there will be changes in fat distribution with the distribution preferentially on ectopic areas and visceral organs rather than abdominal subcutaneous tissues. These changes are exacerbated by physical inactivity, hormonal changes including responsiveness to thyroid hormone, and leptin resistance. Intramuscular fat deposition is influenced by the degree of inflammation, mitochondrial dysfunction, and insulin resistance [37,38]. Decreased lean mass and increased fat mass increase the risk of chemotherapy toxicity. However, the exact mechanism between chemotherapy toxicity and body composition remains unclear [4,39]. Further information can be seen in **Figure 2**.

Body metabolism rates undergo several changes to protect the body from the reduction of body weight from aging. This includes reduced energy expenditure due to physical inactivity, alteration in the thermogenic effect of food and muscle, and resting metabolic rate. Reduced muscle mass and adaptive change of metabolism combined with a sedentary lifestyle cause alteration in energy expenditure. These changes contribute to a gradual increase in body fat [15].

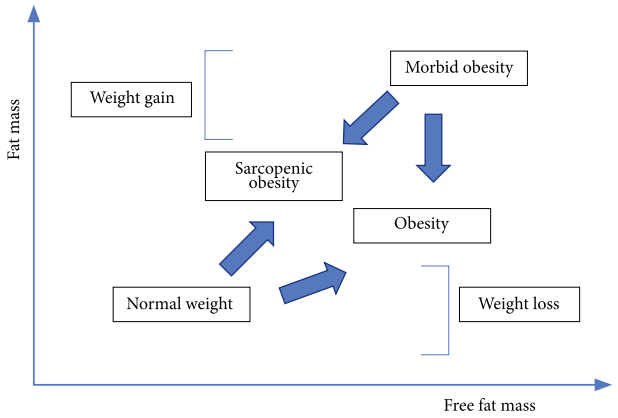


**Figure 2.** Hypothetical metabolic scenarios of sarcopenic obesity from two different trajectories [11]

## Sex Hormones

Sex hormones also contributes to fat composition in the human body. In women, the visceral area is affected by body weight gain and increased fat mass especially after menopause. Estrogen hormone plays a significant role in modulating inflammation in skeletal muscle through satellite cell activation and reducing fat deposition and muscle loss [40,41].

Testosterone is well known in promoting muscle-cell regeneration through satellite cell activation and increasing muscle protein synthesis by increasing amino acids utilization by muscle cells to develop and increase androgen receptor expression [42–44]. In men, testosterone levels tend to decrease by approximately 1% every year, which contributes to muscle mass loss and inappropriate fat distribution in the body [45,46]. These changes are associated with reduced lean mass, increased level of visceral fat, and obesity.

## Inflammation

The inflammatory pathway also plays a significant role in the alteration of body composition. Obesity is well known for causing increased inflammatory state by activating macrophages, mast cells, and T lymphocytes to induce secretion of tumor necrosis factor (TNF), leptin, and growth hormone (GH) [47–49]. All such secretory changes lead to insulin resistance which is further increased by muscle cells catabolism, promoting gain in fat mass and loss of muscle mass [47,50]. Frailty incidence is increased because of the increased level of leptin which blunts the anabolic effect of insulin and reduces the level of sex hormone [51]. Leptin upregulates the proinflammatory cytokines IL-6 and TNF and reduces the levels of IGF-1 [52]. Further, the elevated level of TNF inhibits adiponectin which acts to counter the effect of leptin [53]. Obesity also induces leptin resistance and causes reduced muscle fatty oxidation and ectopic fat deposition [54,55]. Inflammation has become an

important hallmark of cancer, as cancer itself is a chronic inflammation [56]. Higher systemic inflammation state is associated with higher metastatic risk and mortality. Several clinical markers, such as neutrophil to lymphocyte ratio (NLR), correlates with other systemic inflammation cytokines and implicates systemic catabolism on various cancers [57]. Systemic inflammation and several cytokines such as tumor necrosis factor and interleukin 6 (IL-6) promote insulin resistance and protein degradation and decrease muscle synthesis. Several mechanisms on systemic inflammation causing insulin resistance and protein degradation have been proposed, one of which is caused by the activation of the ubiquitin-proteasome proteolysis pathway leading to muscle wasting and degradation leading to exacerbation of insulin resistance [57].

## Myocellular Mechanism

Myocellular also plays an important role in reduced muscle strength in sarcopenic patients. Reduced perfusion, low availability of postprandial amino acids, reduced digestive capacity caused by sequestration of amino acids in splanchnic circulation may cause impaired skeletal metabolism owing to the process of aging [58]. As the results of these processes, reduction of muscle mass and strength, reduction of motor neurons, collagen deposition, fat cells infiltration in muscle cells, and fiber necrosis might occur in sarcopenic patients. Deposition of intramyocellular lipids may cause lipo-toxicity to muscle cells and disturbance in muscle-cell regenerative capabilities, impairment in oxidative capabilities, and insulin resistance caused by impairment in mitochondrial fatty acid oxidation [1,59–62]. Also, elevated concentration of fatty acids may cause the deposition of fat cells in the liver, heart, pancreas, and skeletal muscle [15]. Muscle cells tend to store fat more than glucose as proposed by one study which compares healthy men and women aged 18–28 years exposed to 30 days of leg disuse which results in muscle loss but an increase in intramyocellular lipid deposition [63–65]. The intramyocellular fat deposition may also impair muscle cell regeneration as pro-inflammatory cytokines, and hormones are released by lipid cells that interfere with the regenerative capabilities of muscle cells by satellite mesenchymal cells, exacerbating sarcopenia in obese patients [15,66–70]. All the mechanisms of sarcopenic obesity are highlighted in **Figure 3**.

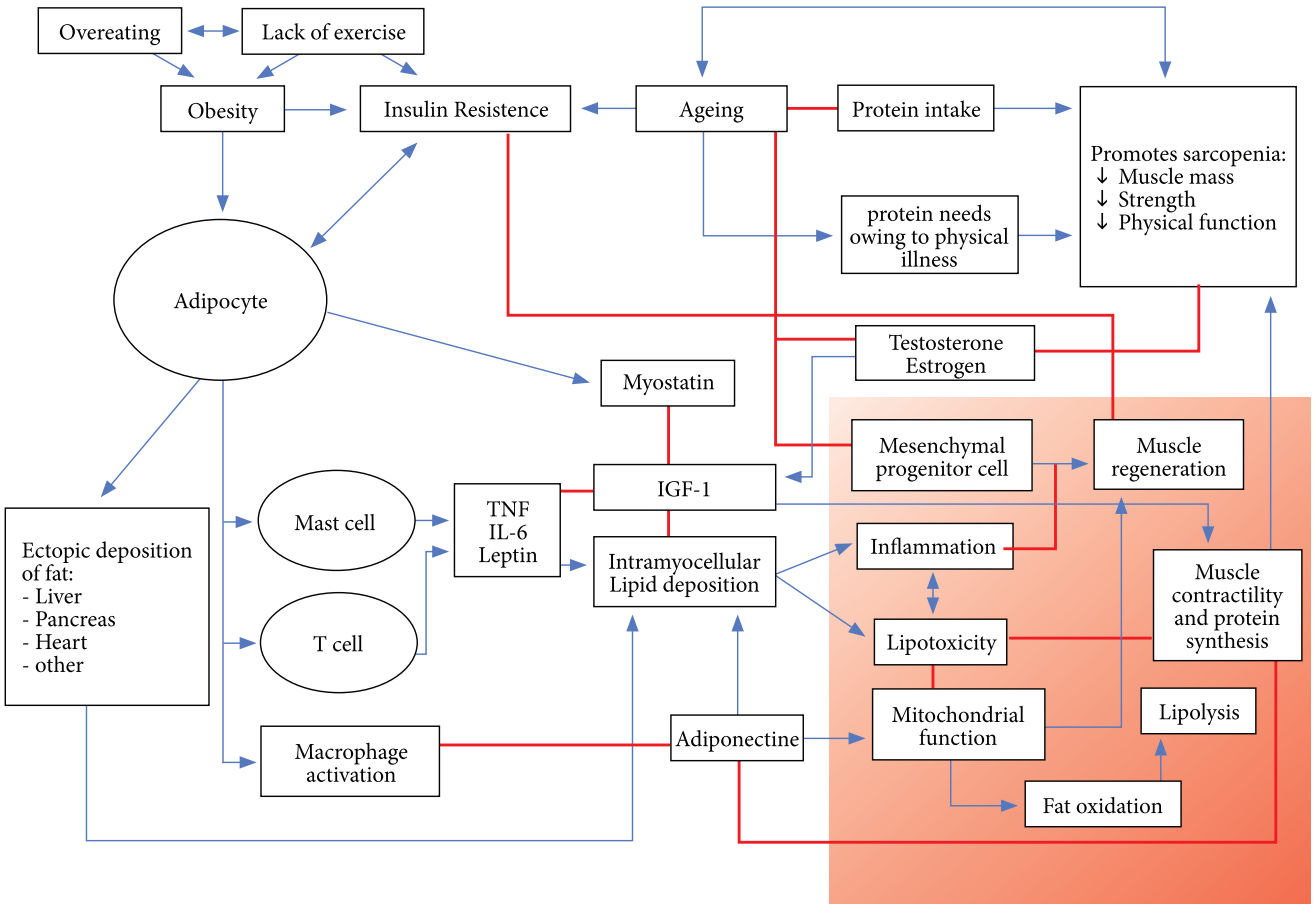


**Figure 3.** Summary of sarcopenic obesity mechanism [15]

## Impacts of Sarcopenic Obesity in Cancer Patients

Cancer is one of the leading causes of death, and multimodality treatments are needed to manage patients with cancer. In search of reliable prognostic factors, many physicians focus on tumor-specific factors (TNM/UICC) rather than taking the patient's specific conditions as their major concern [71]. Body mass is defined as the proportion and distribution of the bone, lean, and fat tissues in the human body that is not easily reflected by the weight-based metrics measurement, especially in cancer patients whose muscle mass does not strongly correlate with BMI or BSA [17].

Sarcopenic obesity alone has a greater prevalence of metabolic syndrome and cardiovascular outcome and has a negative impact on overall survival, dose-limiting toxicity, physical disability, and higher major postoperative complication in cancer patients [20,72]. The alteration in body composition also makes an impaired response to chemotherapy and radiation therapy in a variety of cancers, making treatments more difficult in these patients. In a retrospective study of pancreatic cancer patients, sarcopenic obesity is associated with lower overall survival and recurrence-free survival [7,17,73–78]

Multiple studies of different cancer patients conclude sarcopenic obesity as an independent risk factor on clinical outcomes and overall survival after surgical resection [28,79]. Sarcopenic obesity is also seen as an independent risk factor of mortality in lung, prostate, and GI cancer patients with the locally advanced or metastatic disease [11,80–83]. Some authors suggest several mechanisms contributing to these adverse outcomes including impaired immune response and chronic inflammation associated with sarcopenia and obesity that lead patients to the vulnerability of operative stress causing higher rates of surgical site infection and compromising wound healing mechanism and recovery [84–88].

Sarcopenic obesity is also hypothesized to be associated with chemotherapy toxicity caused by a combination of high absolute doses with a reduced volume of distribution and impaired metabolism and clearance of a very depleted lean body mass [10,11,17]. Decreased lean mass and increased fat mass causing the risk of chemotherapy toxicity increase as demonstrated in breast cancer patients undergoing chemotherapy. However, the exact mechanism between chemotherapy toxicity and body composition remains unclear [39].

Several chemotherapies have been proposed to induce sarcopenic obesity in cancer patients undergoing chemotherapy treatment. In lung cancer patients receiving cisplatin as chemotherapy, there is a reduction in muscle mass and an increase in fat mass accumulation in viscera. Weight gain during chemotherapy is sometimes misinterpreted as the signs of regaining health, but the change in body composition is often neglected [89,90].

## Treatment

Until now, no medications have been proven to be effective in treating this condition. The key management in treating sarcopenic obesity relies on preserving muscle mass and function by lifestyle modification including the combination of physical activity and dietary changes. Physical exercise may induce anabolism. Thus, protein intake before physical exercise or spreading protein intake throughout the day may provide beneficial effects [15].

## Diet

Acute caloric restriction may induce catabolism and proteolysis which lead to a decline in muscle mass. However, chronic caloric restriction may show opposite effects by increasing muscle protein synthesis and promoting muscle growth. High protein intake should be initiated to ensure adequate protein intake to counter the effect of weight loss-induced sarcopenia in individuals participating in a weight loss program. Higher protein intake for about 1.5 g/kg/day is recommended due to high metabolic rates in cancer patients with careful monitoring in renal function. Consumption of high essential amino acids (EAA), found in beef, fish, and peanut, is recommended [91,92].

## Physical Exercise

Physical exercise is recommended in older adults with a sum of 150 minutes per week consisting of moderate to vigorous aerobic exercise training with a minimum of two sessions of resistance training. Resistance training is beneficial in improving muscle mass and strength by focusing on strength training, flexibility, and balance. High-intensity resistance training combined with a short resting interval improves body composition in the elderly [93]. The combination of both diet and physical exercise also shows improvement in the adipose marker and reduced level of leptin, C-reactive protein, and IL-6 [94].

# CONCLUSION

Sarcopenic obesity is a condition where declining muscle mass and function coexist with high-fat mass which leads to lower lean mass. Sarcopenic obesity is related to higher rates of chemotherapy toxicities and mortality in cancer patients which physicians need to pay more attention to. However, this condition is commonly missed because the evaluation of body composition is not a routine procedure. The diagnosis of sarcopenic obesity requires a body composition measurement which is best demonstrated by using CT-scan. Physical exercise in combination with dietary modification is the best strategy to manage sarcopenic obesity in cancer patients.


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## DECLARATIONS

### Competing interest

The authors declare no competing interest in this study.

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