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# Prévalence de l'obésité sarcopénique chez les patients atteints d'obésité suivis au CSO PACA Ouest et effet d'un traitement de 6 mois par Semaglutide en accès précoce

Margot Bouchet

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Faculté des sciences  
médicales et paramédicales  
Aix-Marseille Université

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Semaglutide en accès précoce**

# **T H E S E     A R T I C L E**

**Présentée et publiquement soutenue devant**

**LA FACULTÉ DES SCIENCES MEDICALES ET PARAMEDICALES**

**DE MARSEILLE**

**Le 20 Octobre 2023**

**Par Madame Margot BOUCHET**

**Née le 14 juin 1995 à Montpellier (34)**

**Pour obtenir le grade de Docteur en Médecine**

**D.E.S. d' ENDOCRINOLOGIE - DIABETOLOGIE - NUTRITION**

**Membres du Jury de la Thèse :**

**Madame le Professeur DUTOUR-MEYER Anne**

**Président**

**Madame le Professeur GABORIT Bénédicte**

**Directeur**

**Madame le Professeur COUDERC Anne Laure**

**Assesseur**

**Monsieur le Docteur LE GARF Sébastien**

**Assesseur**





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CASANOVA Ludovic (MCF Méd. Gén. Temps plein)

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ROUSSEAU-DURAND Raphaëlle (MCF associé Méd. Gén. À mi-temps)

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# **Prevalence of sarcopenic obesity in patients living with obesity followed in CSO PACA Ouest and effect of 6 months early access anti-obesity Semaglutide treatment**

## **INTRODUCTION**

Obesity is defined by the World Health Organization as excessive fat accumulation, represented by a body mass index (BMI)  $\geq 30$  kg/m<sup>2</sup>, that presents a risk to health. According to the latest estimations in 2016, the prevalence of obesity worldwide is around 13%, and has tripled since 1975 [1], [2]. Sarcopenia is defined by a loss of skeletal muscle mass and strength and is considered as a geriatric pathology. Its prevalence ranges from 5 to 50% depending on age [3]–[5]. Sarcopenic obesity (SO) is therefore defined as the combination of excess adiposity and low muscle mass and function [6]. SO should be considered as a unique clinical condition, different than obesity or sarcopenia alone [7].

The precise diagnostic criteria of SO are not clearly established to date. The absence of globally adopted common definition criteria for sarcopenic obesity impairs precise epidemiological assessment, proper patient identification, and, ultimately adapted management to limit related adverse complications. A group of international experts met in November 2022 to propose a consensus definition, screening and diagnostic method [7].

The SO prevalence is very variable and difficult to assess, depending on definition and diagnostic methods used as demonstrated by Batsis *et al.* who find a prevalence of SO that ranged from 4,4 to 84% in men, and from 3,6 to 94% in women [8], [9].

Prevalence also varies according to the population studied, particularly in terms of age.

Indeed, SO is more frequent and known in the elderly. With ageing, body composition changes. Lean mass decreases because of the reduction of physical activity and protein intake whereas fat mass increases with the reduction of total energy expenditure [9], [10].

SO is also due to sedentary lifestyle, adipose tissue ageing, insulin resistance, oxidative stress, lipotoxicity, mitochondrial dysfunction, chronic inflammation, proteostasis and unhealthy dietary habits [6], [9], [11]. Sarcopenic obesity is characterized by adipose tissue expansion and muscle loss that cause increased pro-inflammatory cytokine levels, increased leptin, reduced adiponectin, and intramyocellular lipid deposits [12].

Sedentary lifestyles and obesity are rising in the general population, particularly in the younger age groups [13]–[15]. The prevalence of SO is consequently tending to increase and to affect a younger population than expected. However, limited data and studies are available on body composition and muscle strength changes in young individuals with obesity and weight care [10], [16]. Furthermore, sarcopenia screening methods, such as SARC-F used in the ESPEN and EASO diagnostic procedure, are only validated in the elderly subjects [17], [18].

Moreover, SO can trigger many complications like disability, frailty, depression, osteoporosis, cancer and fractures, cardio-vascular (major cardiovascular events) and metabolic complications (such as type 2 diabetes), the latter increasing the risk of hospitalization and mortality. These complications occur more frequently than in the presence of sarcopenia or obesity alone [10], [19]. Therefore, it is important to identify specific risk factors for SO in young subjects to prevent its onset and identify patients at risk of complications as soon as possible.

Furthermore, obesity management is a public health concern, and one of its main objectives is weight loss. This weight loss involves a loss of fat mass, which is often associated also with a loss of muscle mass [16], [20]. In the absence of appropriate physical activity or diet, the loss of lean body mass reduces the basal metabolic rate, possibly creating a vicious circle leading to sarcopenic obesity [21], [22]. Indeed, the changes in white adipose tissue mass and the local infiltration of muscle tissue by lipid deposits leads to secretion of proinflammatory adipokines, thereby inhibiting protein synthesis and inducing protein catabolism, which exacerbate the decline in muscle mass. [23].

Dietary monitoring, adapted physical activity and drugs targeting appetite regulation are all part of our therapeutic arsenal in the management of obesity [24], [25].

Current anti-obesity medications include Semaglutide, a glucagon-like peptide 1 receptor agonist once-weekly subcutaneously administered. Semaglutide was approved for early access in France in July 2022, in adults with an initial body mass index (BMI)  $\geq 40$  kg/m<sup>2</sup> in the presence of at least one weight-related co-morbidity: treated hypertension, treated dyslipidaemia, established cardiovascular disease, or treated sleep apnoea syndrome [26]. The STEP program was developed to investigate the specific effect of Semaglutide on weight loss. The mean percentage of total weight loss after 68 weeks of treatment compared to baseline ranged from 10 – 16% but there were only few details on the change of body composition [27], [28]. Few real life studies have examined changes in body composition under glucagon-like peptide 1 receptor agonists, and the effect of these medications on lean body mass loss remains partly unknown [29], [30].

The primary objective of this real-life study was to assess the prevalence of sarcopenia in patients living with obesity (PLO) followed in a specialized obesity health care centre (Centre spécialisé d'obésité (CSO) PACA-Ouest) and who came for an ambulatory visit between August 2022 and July 2023. The secondary objectives were to determine the clinical and biological factors associated with SO in PLO, and to assess the effect of 6 months Semaglutide treatment on body composition (fat mass and lean body mass).

## **METHODS**

### **Study population**

In the first cross-sectional part, the aim was to establish the prevalence of SO in a monocentric cohort of PLO followed in the CSO PACA Ouest.

The inclusion criteria were:

- Age  $\geq$  18 years old
- PLO defined as BMI  $\geq$  30 kg/m<sup>2</sup>
- Ambulatory day hospitalization between the 30<sup>th</sup> of August 2022 and 30<sup>th</sup> of June 2023 at CSO PACA Ouest
- With body composition evaluation by dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA) the day of inclusion
- With skeletal muscle function analysis with hand-grip test or 5-time sit-to-stand test the day of inclusion

The exclusion criteria were:

- Pregnant women
- Monogenic forms of obesity
- Patient not affiliated to the social security system

The patients included were referred for complications of their obesity or for a pre- or post-bariatric surgery follow-up. Data were collected by interview, clinical examination and blood tests.

A subgroup of patients was eligible for Semaglutide treatment as an anti-obesity medication, according to early access indications (n=60). Patients were seen in day hospital or in consultation by an endocrinologist prior to the introduction of treatment. Baseline clinical and laboratory information were collected at the day of inclusion. These PLO were followed-up 6 months after the introduction of Semaglutide, in order to assess changes in bioclinical and body composition data.

The first study was conducted in accordance with the general ethical principles of the Declaration of Helsinki and the conformity of the study with European data privacy rules was approved by the local data protection officer (RGPD PADS reference QFK9YB).

Patients from the sub-study with treatment intervention were part of the SEMASEARCH study (NCT05897398) and temporary authorisation for use ATU cohort coordinated by FORCE (French Obesity Research Centre of Excellence) and HCL (Hospices Civils de Lyon).

### **Primary endpoint Sarcopenic obesity assessment**

The diagnosis of sarcopenic obesity, was made according to the decision algorithm established by the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) shown in appendix 1 [6].

Firstly, patients skeletal muscle function and strength were collected by handgrip strength (HGS) and 5-time sit-to-stand test (5TSTS). To execute the hand grip test, the patient was seated with his arms closed to his body and his elbow bent at 90°. He squeezed the handle of the dynamometer (Camry®, EH 101) with maximum isometric effort for at least 5 seconds. This HGS was repeated three times for each hand. The highest value was recorded. The hand grip was considered pathological according to the manufacturer's standards for age and sex (*see appendix 2*).

To achieve the 5TSTS, the patient was instructed to stand up 5 times from a chair with his arms crossed over his chest to avoid using hands to stand. The test was timed and was considered pathological if the 5 stands were completed in more than 15 seconds [31].

Secondly, for all patients, analysis of body composition was collected by BIA (InBody France® IB570-2.0) or by DXA (GE Lunar iDXA, GE Healthcare). Body composition was defined as altered if the ratio of appendicular lean mass to weight (ALM/weight) in kg was below the lowest threshold defined in the literature in Caucasians by international experts as pathological [32]. The threshold value was set at <19.9% in women and < 25.7% in men. We chose a lower threshold than the one considered pathological in the elderly, given the young age of our population.

According to the consensus algorithm, SO was considered to be present if both alteration in skeletal muscle function and body composition were found, ie. the HGS AND/OR the 5TSTS was pathological AND the ALM/weight ratio was strictly below the threshold defined above.

### **Other parameters collected**

According to the ESPEN and EASO procedure, the SARC-F questionnaire can be used to screen for sarcopenia before proceeding to the diagnostic stage. The SARC-F is a questionnaire designed to screen for the presence of sarcopenia based on 5 questions (appendix 3). The questionnaire was defined as pathological if the result was  $\geq 4/10$ . We calculated the Sensitivity and Specificity of this questionnaire in our population.

In addition, one study reports that question 1 (on strength), question 2 (on assistance walking) and question 4 (on climbing stairs with difficulties) are the most sensitive for SO screening in the elderly [33]. These questions are graded on 2 points. We therefore calculated the sensitivity and specificity of these questions taken separately in our study population.



### **Clinical and biological data**

Weight history (duration of obesity and weight cycling), medical history, menopausal status, history of bariatric surgery, current antidepressant and psychotropic medications, current or past corticosteroid therapy, current vitamin D medications, current or past GLP1 analogue medication, waist circumference, systolic and diastolic blood pressure were systematically recorded.

Weight cycling, also known as yo-yo dieting, was defined as the repeated loss and gain of weight, resembling the up-down motion of a yo-yo more than 3 times since the beginning of the disease.

Particular attention was paid to patients eating habits, in particular the presence of eating disorders (compulsions, bulimia, binge eating disorder, night eating disorder, restriction) and protein consumption with a dedicated interview by the dietitian. Physical activity was assessed according to WHO recommendations for adults aged 18 to 64, i.e. at least 150 to 300 minutes per week of moderate-intensity endurance activity or at least 75 to 150 minutes of sustained-intensity endurance activity. Physical activity was therefore classified as "sedentary", "active below" or "active above" WHO recommendations [34]. We also collected the number of steps per day.

Lipid, glycaemic and hormonal profile were assessed by blood sampling and included fasting blood glucose and insulin levels, HbA1c, total, low-density lipoprotein, and high-density lipoprotein cholesterol, triglycerides, prealbumin, c-reactive protein (CRP), creatinine, urine albumin-to-creatinine ratio, testosterone and Insulin-like Growth Factor One (IGF-1).

In patients treated by Semaglutide, weight, waist circumference, blood pressure, physical activity, treatment compliance, side effects evaluation (diarrhoea, epigastralgia, nausea, vomiting) and blood sampling including lipid and glycaemic profile, CRP and creatinine were collected 6 months after introduction of treatment.

### **Statistical analysis**

First, a descriptive analysis of the entire sample was performed. Categorical variables are presented as proportions and numbers while quantitative variables are presented as mean  $\pm$  standard deviation. For each variable, the proportion of missing data was specified.

Then, qualitative variables were compared between the two groups (sarcopenic obesity yes/no) using Chi-square or Fisher's exact tests according to the conditions of application, and the quantitative variables were compared using Student's t-test or the Mann-Whitney U-test when the application conditions were not met. The variables associated with the diagnosis of SO were analysed using logistic regression. The results are expressed as odds ratios (OR) and their 95% confidence intervals (95% CI).

A multivariate model was then performed. The set of independent variables was selected based on a p-value  $< 0.05$  in the univariate analysis and on literature review of factors classically associated with SO. All the results of the logistic regression are presented with the OR and 95% CI. Finally, a Receiver

Operating Characteristic (ROC) curve was used to analyse sensitivity and specificity of SRAC-F questionnaire.

The significance threshold was set at 5%.

All the analyses were carried out using IBM SPSS® Statistics software version 20.

## RESULTS

### 1) Population

During August 2022 and July 2023, 161 PLO met the inclusion criteria and were included in study one. Twenty-eight patients were excluded for lack of a functional test on muscle strength or function and 4 patients were excluded due to the absence of a body composition analysis. All the excluded patients were eligible to Semaglutide treatment initiation. Finally, 129 patients were analysed for SO diagnosis assessment. Among the 129 PLO analysed, 28 patients met the inclusion criteria for early access to Semaglutide treatment and received the treatment. In the sub-group study, 6 months after the introduction of Semaglutide, only 7 patients were included. Twenty-one patients were excluded for lack of functional test or body composition analysis at 6 months (*Fig 1*).

The baseline characteristics are summarised in Table 1. Mean age was  $44.4 \pm 13.8$  years, with a majority of women (71.3% (W) and 28.7%(M)). Mean BMI was  $43.4 \pm 7.3$  kg/m<sup>2</sup> and 81 out of 129 patients (62.8%) had body weight cycling history. Mean duration of the disease was  $20.5 \pm 10.6$  years and 31% of the studied population had already undergone a bariatric surgery. Only 14.7% of patients were considered active according to OMS recommendations. Less than 8% of patients had a fracture history and 11.6% of patients had already received a corticosteroid therapy for at least one month.

Figure 1: Flow chart of the study and the sub-group study

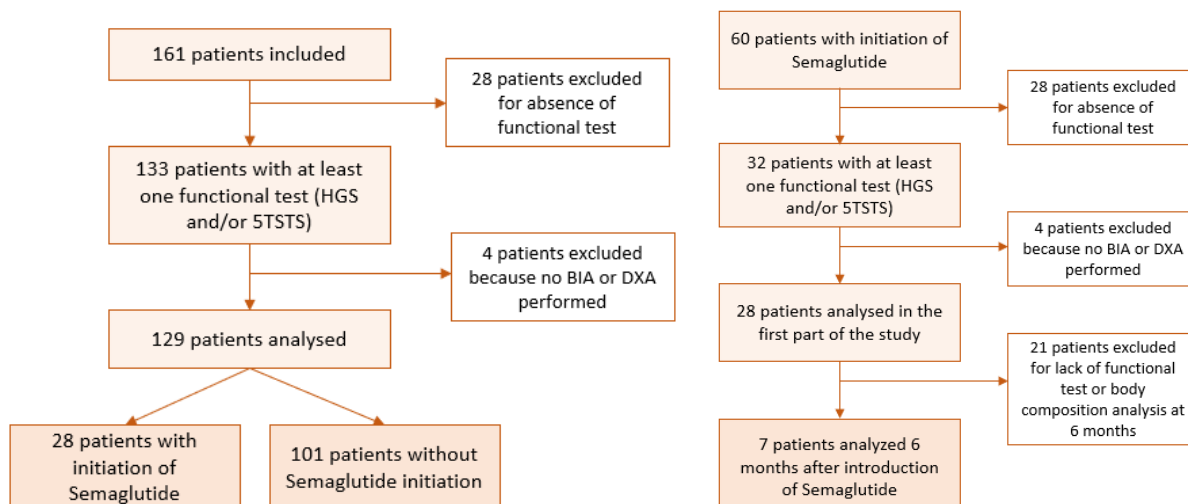


Table 1: Baseline Demographics and Clinical Characteristics

	Study population	Missing data (n)
Age	44.41 (13.8)	0
Women	92 (71.3)	0
Men	37 (28.7)	0
Body weight, kg	121.3 (21.8)	0
BMI, kg/m <sup>2</sup>	43.4 (7.3)	0
Waist circumference, cm	123.6 (17.4)	25
Duration of obesity, years	20.5 (10.6)	8
Body weight cycling	81 (62.8)	10
Bariatric surgery history	40 (31)	0
Eating disorder	25 (19.4)	1
Fracture history	10 (7.8)	0
Treated dyslipidemia	33 (25.6)	1
Treated hypertension	37 (28.7)	1
Treated sleep apnea syndrome	55 (42.6)	1
Established cardiovascular disease	4 (3.1)	0
Type 2 diabetes	23 (17.8)	0
Depression	24 (18.6)	0
Treated hypothyroidia	20 (15.5)	0
Menopausal status <sup>£</sup>	30 (23.3)	4
Physical activity, number of steps per day	6041 (3177)	81
Sedentary lifestyle	43 (33.4)	0
Active below recommendations <sup>[34]</sup>	67 (51.9)	0
Active above recommendations <sup>[34]</sup>	19 (14.7)	0
Proteins intake, g per day (%)	92.4 (15.2)	91
Analog GLP1 receptor treatment <sup>§</sup>	13 (10.1)	0
Corticosteroid treatment <sup>#</sup>	15 (11.6)	1
Systolic blood pressure, mmHg	124 (14.6)	32
Diastolic blood pressure, mmHg	68.4 (13.4)	32
Prealbumin, g/L	0.239 (0.05)	47
CRP, mg/l	7.7 (7.9)	27
Fasting blood glucose, g/l	0.98 (0.2)	18
Insulin level, mUI/L	18.7 (10.4)	44
HbA1c, %	5.8 (0.75)	24
Total cholesterol, g/L	1.79 (0.33)	12
LDL, g/L	1.10 (0.28)	11
HDL, g/l	0.45 (0.13)	11
Triglycerides, g/L	1.34 (0.74)	11
IGF1 deficiency <sup>+</sup>	13 (9.3)	74
Testosterone deficiency <sup>+</sup>	5 (3.9)	116
Creatinine, µmol/L	65.5 (17)	14
Urine albumin-to-creatinine ratio, mg/mmol	1.67 (2.59)	105

<sup>£</sup>Only in women

<sup>§</sup>Analog GLP1 receptor treatment: actual or past, different than Semaglutide

<sup>#</sup>Corticosteroid treatment: treatment more than 1 month, actual or in medical history

<sup>+</sup> Number of patients with a reduced rate according to laboratory standards for age

## 2) Primary objective: Sarcopenic obesity prevalence

Data for each stage of the diagnostic procedure are detailed in Table 2.

Thirty-nine patients out of 129 (30.2%) were identified as having impaired muscle function or strength. Body composition was analysed in 55% of subjects by DXA and 45% by BIA. An alteration in ALM/weight was found in 61 out of 129 patients (47.2%) and in 33 out of 92 women (35.8%) and 28 out of 37 men (75.6%).

In total, SO was identified in 24 patients (18.6%), 14 women and 10 men (15.2% and 27% respectively,  $p = 0.14$ ). ALM/weight was significantly lower ( $p = 0.03$ ) in sarcopenic patients (mean 17.3% in women and 23.3% in men) than in non-sarcopenic patients (mean 21.1 in women and 25.1% in men).

Among patients eligible for treatment with Semaglutide, 7 out of 28 patients (25%) had SO. Concerning patients without Semaglutide introduction, 17 out 101 patients had SO diagnostic (16.8%). This difference was not statistically different ( $p = 0.41$ ) (Fig 2a).

There was no statistical difference depending on the functional test realised (HGS or 5TSTS) or the method used for body composition (BIA or DXA) for the diagnosis of SO (Fig 2b).

Table 2: Results at each stage of the OS diagnostic procedure of ESPEN and EASO

<b>Altered skeletal muscle functional parameters (at least one pathological functional test)</b>	39 (30.2%)
<b>Pathological hand grip test</b>	20 (15.5)
<b>Pathological 5-time sit-to-stand test</b>	28 (21.7)
<b>Both functional test pathological</b>	9 (7)
<b>Method used to assess body composition : DXA – BIA, %</b>	55 – 45
<b>Fat mass, kg</b>	60.2 (11.1)
<b>Fat mass, %</b>	49.6 (7.2)
<b>Lean mass, kg</b>	55.9 (13.6)
	<b>In women</b> 52.6 (9.5)
	<b>In men</b> 63.4 (11.3)
<b>Lean mass, %</b>	46.4 (6.1)
	<b>In women</b> 45.6 (6.1)
	<b>In men</b> 48.3 (9.1)
<b>ALM/weight, %</b>	21.7 (3.5)
	<b>In women</b> 20.5 (3.1)
	<b>In men</b> 24.6 (2.6)
<b>Pathological ALM/weight</b>	61 (47.2)
	<b>In women</b> 33 (35.8)
	<b>In men</b> 28 (75.6)
<b>Diagnostic of sarcopenia</b>	24 (18.6)
	<b>In women</b> 14 (15.2)
	<b>In men</b> 10 (27)
	<b>Eligible patients for Semaglutide</b> 7 (25)
	<b>Patients without Semaglutide initiation</b> 17 (16.8)

Figure 2a: Primary outcome: prevalence of SO

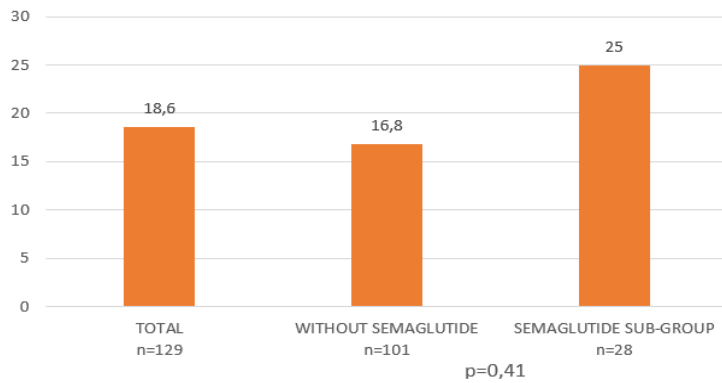
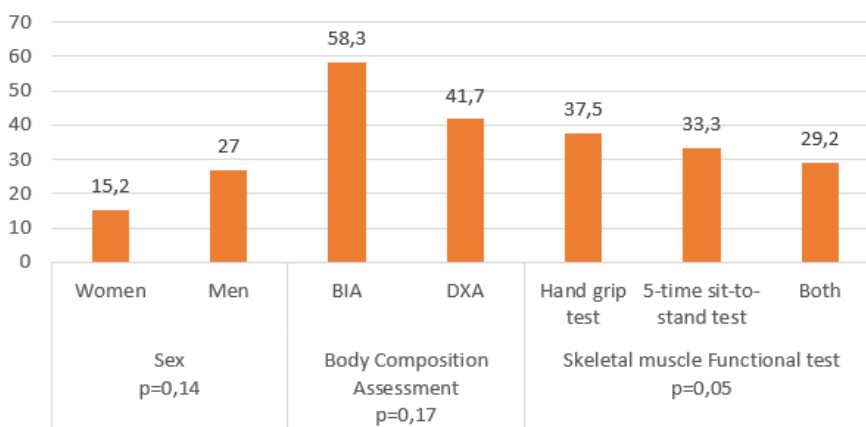


Figure 2b: Prevalence of SO depending on sex, functional test and body composition assessment



Difference between patients eligible for Semaglutide treatment and patients without Semaglutide treatment initiation are showed in Table 3.

Mean weight, BMI and waist circumference were significantly higher in the group with Semaglutide treatment because of the indications for introducing treatment ( $p = 0.001$  and  $0.011$  respectively). Twenty-one out 28 patients (75%) had a BMI between 40 and 49.9  $\text{kg}/\text{m}^2$ , 6 patients (21.4%) between 50 and 59.9  $\text{kg}/\text{m}^2$  and only one (3%) up or equal to 60  $\text{kg}/\text{m}^2$ .

None had normal waist circumference values ( $< 80$  and  $< 94$  cm for women and men, respectively).

Patients treated with Semaglutide had a significantly longer history of obesity ( $24.4 \pm 10.8$  years) than patients in the untreated group ( $19.4 \pm 10.4$  years). They had also more treated hypertension ( $p = 0.03$ ) and treated sleep apnoea ( $p = 0.001$ ), as expected (Table 3).

Few patients had established cardiovascular disease (myocardial infarction, for example), given the young age of our population. There were no differences concerning the others obesity complications such as type 2 diabetes or dyslipidemia.

Sedentary lifestyle and proteins intake were similar in both groups. There were no statistical differences between the groups regarding biological data.

Table 3: Comparison between patients before initiation of Semaglutide treatment and non-treated patients

	Before Semaglutide treatment initiation (n = 28)	Without Semaglutide treatment initiation (n = 101)	P value
Age	47.9 (13.8)	43.4 (13.7)	0.126
Women	18 (64.3)	74 (73.3)	0.479
Men	10 (35.7)	27 (26.7)	
Body weight, kg	131.8 (21.4)	118,3 (21.4)	<b>0.003</b>
BMI, kg/m <sup>2</sup>	47,5 (6.9)	42,3 (7)	<b>0.001</b>
Waist circumference, cm	131.7 (15.3)	121,3 (17.3)	<b>0.011</b>
Duration of obesity, years	24,4 (10.8)	19,4 (10.4)	<b>0.028</b>
Body weight cycling	19 (67.8)	62 (61.4)	0.470
Bariatric surgery history	10 (35.7)	30 (29.7)	0.645
Eating disorder	8 (28.6)	17 (16.8)	0.185
Fracture history	4 (14.3)	6 (5.9)	0.222
Treated dyslipidemia	9 (32.4)	24 (23.7)	0.464
Treated hypertension	13 (46.4)	24 (24.8)	<b>0.03</b>
Treated sleep apnea syndrom	20 (71.4)	35 (34.6)	<b>0.001</b>
Established CVD	1 (3.5)	3 (2.9)	1
Type 2 diabetes	8 (28.6)	15 (14.8)	0.102
Depression	7 (25)	17 (16.8)	0.410
Treated hypothyroidia	6 (21.4)	14 (13.8)	0.377
Menopausal status <sup>£</sup>	11 (39.2)	19 (18.8)	<b>0.017</b>
Sedentary lifestyle	8 (28.6)	35 (34.7)	0.653
Active	20 (71.4)	66 (65.3)	
Proteins intake*, g (%)	138 (16.6)	92 (15.1)	0.285
Analog GLP1 receptor treatment <sup>§</sup>	4 (14.3)	9 (8.9)	0.477
Actual corticosteroid treatment	1 (3.7)	4 (3.9)	1
Past corticosteroid treatment	0	10 (9.9)	0.117
Systolic blood pressure, mmHg	128.7 (17.6)	123.9 (13.2)	0.150
Diastolic blood pressure, mmHg	72.9 (13.9)	70.4 (10.7)	0.863
Prealbumin, g/L	0.259 (0.042)	0.239 (0.048)	0.06
CRP, mg/l	6.6 (5.6)	7.7 (8.4)	0.581
Fasting blood glucose, g/l	1.09 (0.17)	0.95 (0.21)	0.162
Insulin level, mUI/L	20 (18.25)	17.9 (9.10)	0.796
HbA1c, %	6.02 (0.88)	5.73 (0.72)	0.205
Total cholesterol, g/L	1.9 (0.35)	1.77 (0.31)	0.124
LDL, g/L	1.13 (0.29)	1.08 (0.26)	0.534
HDL, g/l	0.47 (0.12)	0.45 (0.13)	0.436
Triglycerides, g/L	1.56 (0.74)	1.28 (0.71)	0.112
Creatinine	69.9 (18.5)	65.8 (14.2)	0.178
Urine albumin-to-creatinine ratio	1.71 (2)	11 (3.2)	0.943

CVD = cardiovascular disease

<sup>£</sup>Only in women

\*Protein intake: data for only 3 patients in the Semaglutide treatment group

<sup>§</sup>Analog GLP1 receptor treatment: actual or past, different than Semaglutide

### 3) Sarcopenic obesity associated factors

We analysed several factors that may be associated with SO.

In our population, 47.2 % of patients had an altered body composition with a decrease in ALM/weight, and 30.2% had an alteration in skeletal muscle function (pathological HGS or 5 time sit-to-stand test).

There was a significant correlation between HGS and lean body mass ( $r=0.32$ ,  $p < 0.001$ ).

There was no difference in the percentage of MM between sarcopenic and non-sarcopenic patients.

In univariate analysis, the only factors significantly associated with SO were BMI ( $p = 0.003$ , OR=1.03 CI [1.03-1.17]) and waist circumference ( $p = 0.03$ , OR =1.04 CI [1.003-1.07]).

Gender, menopausal status, duration of obesity, body-weight cycling, history of bariatric surgery and eating disorders were not significantly associated with SO.

There was also no significant association between physical activity or sedentary lifestyle or protein intake (in grams and %).

We also investigated if treatment with GLP1 analogues (other than Semaglutide) or high-dose corticosteroids treatment (current or past) could lead to increased SO. There was no significant association in our study.

Patients with cardiovascular disease ( $n=4$ ) had a significantly higher risk of developing SO ( $p = 0.02$ ) in univariate but not in multivariate analysis. In contrast, other weight-related co-morbidities (i.e. hypertension, dyslipidemia, sleep apnea syndrome, type 2 diabetes) were not significantly associated with SO in univariate analysis. There was also no association of SO with the biological parameters collected notably with low testosterone rates ( $p = 0.128$ ) and low IGF-1 rates ( $p = 0.352$ ).

In multivariate analysis including age, BMI, level of physical activity, body weight cycling and history of cardiovascular disease only BMI was independently associated with SO ( $p = 0.026$ , OR 1.08 CI [1.01-1.16]) (Table 4).

Table 4: Multivariate analysis

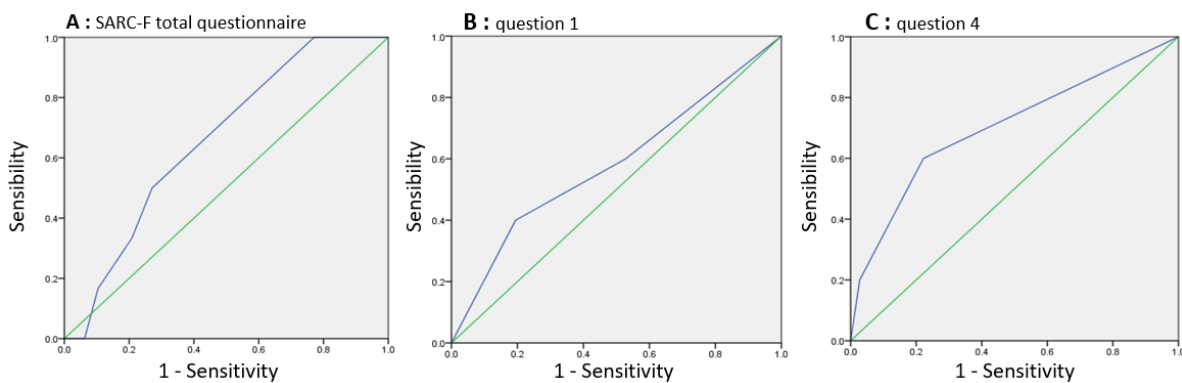
	Odds Ratio	95% Confidence Interval	p-value
Age	0.99	0.95 – 1.03	0.551
BMI at inclusion	1.08	1.01 – 1.16	0.026
Body weight cycling	0.43	0.15 – 1.22	0.112
CVD	8.8	0.64 – 122.8	0.104
Physical activity	0.77	0.25 – 2.36	0.651

CVD : cardiovascular disease

Finally, the SARC-F questionnaire was completed by only 54 patients out of 129. A pathological result (i.e. if the result was  $\geq 4/10$ ) was found in 12 patients (21.8%). Only 2 patients with pathological result had SO. In addition, 4 patients had SO, although their results were  $< 4$ .

Sensitivity and specificity of SARC-F questionnaire in young subjects were therefore 33% and 79% respectively (Fig 3). We did not produce an ROC curve for question 2 (walking assistance), given the limited pathological data available in our young patients. For question 1 on muscle strength, in the presence of a positive response (i.e.  $\geq 1$ ), sensitivity was 60% and specificity 78% (Fig 3). Question 4 on stair-climbing difficulty had a sensitivity of 60% and a specificity of 47% in the presence of a positive response (Fig 3).

Fig 3 : ROC curves. A : Sarc-F total questionnaire, B : question 1, C : question 4



#### 4) Semaglutide treatment: sub-group analysis

All patients included in the sub-group analysis are described in Table 5.

Of the 7 patients included, one was sarcopenic at baseline and was no longer sarcopenic at 6 months after introduction of Semaglutide (patient 2 in table 3). In contrast, 1 patient who was not sarcopenic at baseline was sarcopenic at 6 months after introduction of Semaglutide (patient 1 in table 3).

Patient 2, had a BMI of 51.6 kg/m<sup>2</sup> at inclusion and a history of cardiovascular disease, both significantly associated with SO. At 6 months, he had lost 6.8 kg and normalized his HGS compared with inclusion without an increase in physical activity but his ALM/weight was similar (16.7% at baseline, 16.4% at 6 months).

Patient 1 had a BMI of 47.4 kg/m<sup>2</sup> at inclusion, a compulsive eating disorder and had a sedentary lifestyle. Duration of her obesity was 9 years, unlike the other patients, who presented an obesity history of at least 15 years. She was the only patient among the 7 to have a pathological SARC-F questionnaire. At inclusion, she already had a pathological 5TSTS. At 6 months, after a 3 kg loss, she still had a pathological 5TSTS and a decrease in ALM/weight.

Semaglutide treatment was not significantly associated with the diagnosis of SO after 6 months of treatment.



Table 5: Description of the 7 Semaglutide treated patients included in the subgroup analysis

Patient	1	2	3	4	5	6	7
Sex	Women	Women	Women	Women	Men	Women	Women
Age, years	59	37	37	54	55	41	53
BMI at inclusion, kg/m <sup>2</sup>	47.4	51.6	44.2	43	48.1	41.9	46
Weight at inclusion, kg	137	125.6	109	141.1	152.7	114	118.8
Waist circumference at inclusion, cm	133	118	104	137	149	115	138
Duration of obesity, years	9	29	17	15	43	21	28
Bariatric surgery history	No	GB and SG	Intragastric balloon	No	No	No	GB and SG
Eating disorder	Compulsion	BED	Boulimia	No	No	No	No
Physical activity	SLS	ABR	SLS	ABR	ABR	SLS	SLS
Comorbidity	HBP, OSAS, Depression	CVD	Dyslip*	HBP	OSAS, T2D	HBP, OSAS	Dyslip* HBP, OSAS, T2D
SARC-F score	5	NA	2	0	NA	2	1
Pathological HGS	No	Yes	No	No	NA	Yes	No
Pathological 5TSTS	Yes	No	No	No	No	No	No
Pathological ALM/weight	No	Yes	No	No	Yes	No	No
SO at inclusion	No	Yes	No	No	No	No	No
Weight loss at 6 months, kg	3	6.8	16	3.4	4.8	10.4	+ 2.2
Waist circumference loss at 6 months, cm	NA	7	10	NA	NA	NA	NA
Changes in physical activity	No	No	No	No	No	No	No
Pathological HGS at 6 months	No	No	No	NA	No	No	No
Pathological 5TSTS at 6 months	Yes	No	No	No	No	No	NA
Pathological ALM/weight at 6 months	Yes	Yes	No	No	No	No	No
SO at 6 months	Yes	No	No	No	No	No	No

GB = Gastric Banding SG = Sleeve Gastrectomy, BED = Binge Eating Disorder, SLS = sedentary lifestyle, ABR = active below recommendations, HBP = High Blood Pressure, OSAS = Obstructive Sleep Apnea Syndrome, T2D = Type 2 Diabetes, CVD = Cardiovascular Disease, NA = not available. \*Dyslipidemia

## DISCUSSION

In our real-life study, the prevalence of SO according to ESPEN and EASO diagnostic procedures was 18.6% in patients followed up in CSO, 15.2% in women, 27% in men. However, comparison of this prevalence with the literature is difficult given the variability of the methods used to diagnose SO and the pathological thresholds for appendicular lean mass.

In the literature review by Donini *et al.* conducted from 2008 to 2018, i.e. prior to the publication of the diagnostic procedure for SO, no study out of the 75 selected used the HGS and/or 5TSTS associated with ALM/weight [35]. Most studies defined SO by ALM/weight alone, without assessment of muscle function, or defined decreased muscle mass according to European Working Group on Sarcopenia in Older People (EWGSOP) and Asian Working Group for Sarcopenia (AWGS) definition, by appendicular muscle mass adjusted for height.

Since publication of the expert consensus diagnostic procedure, few studies have applied the method to different populations.

In a study conducted in 2022 by Vieira *et al.* in 186 patients with a mean age comparable to our population and operated by Roux-en-Y gastric bypass, we found a prevalence of SO of 23% after analysis of body function by DXA with diagnostic thresholds of ALM/weight of 23.47% in women and 28.27% in men, thus higher than those used in our study [36].

The study by Godziuk *et al.* used thresholds that are closer to our study, with a pathological ALM/weight cut-off of 19.43% in women and 25.7% in men. The prevalence of SO in their study was 8.6%, lower than in our study. However, their population differed from ours, with a lower mean BMI of 37 kg/m<sup>2</sup> in women and 34.3 kg/m<sup>2</sup> in men, and a higher median age of 64.9 years in women and 65.5 years in men, which may explain this discordance [37].

In another study published in 2021 on 108 volunteers with an average age of 43 and an average BMI of 42.2 kg/m<sup>2</sup>, Silva and Mudler used pathological thresholds similar to ours (19.43% in women and 25.72% in men) and found a total SO prevalence of 13%. [38]. This prevalence is approaching the prevalence in our population not treated with Semaglutide (i.e. 16.8%), which has an almost identical mean BMI.

Despite the choice of low pathological thresholds for diagnosing altered muscle mass, we have a higher prevalence of SO than in the literature. This may be explained by the high BMI in our study (mean 43.4 kg/m<sup>2</sup>), a parameter significantly associated with SO in multivariate analysis.

Indeed, it is now well known that the infiltration of skeletal muscle by ectopic fat is a phenomenon that increases with BMI and body fat. This myosteatosis promotes lipotoxicity and inflammation, and

induces dedifferentiation of progenitor cells expressing adipose tissue genes. Muscle regeneration potential is impaired, resulting in a vicious circle leading to SO [39], [40].

In addition, the average age of duration of the disease in our population was high 20.5 years, increasing the duration of lipotoxicity of fat mass on muscles.

Furthermore, in many patients in our study, there was an alteration in muscle mass (47.2%) without alteration in muscle strength. The EWGSOP defines this alteration in muscle mass alone as "presarcopenia" [3]. Presarcopenia is more frequently described than sarcopenia in young people [41], [42]. It is associated with an increased risk of osteoporosis and fractures, impaired quality of life and a worse prognosis for certain diseases such as cancer [43]–[46]. Moreover, according to Ruthes *et al.*, 12 months after bariatric surgery, presarcopenia was diagnosed in all patients included in the study. [47]. This suggests that special attention needs to be given to these presarcopenic patients, who are often candidates for bariatric surgery, notably by reinforcing support for physical activity. [48].

Indeed, increased physical activity and adequate protein intake are part of SO prevention. Physical activity should include both resistance and aerobic exercise, in order to obtain a negative energy balance, that facilitates weight loss, while limiting the loss of muscular strength [49]. Protein and physical activity have a key anabolic role in muscle protein synthesis. In addition, increased muscle fibres sensitizes insulin action and reduces inflammation, leading to a reduction in catabolism and ending the vicious circle of SO [9].

Unexpectedly, we found no significant association between physical activity and protein intake and SO in our study. This is probably explained by the large number of sedentary patients in our population, homogeneously distributed between the sarcopenic and non-sarcopenic groups, and missing data on protein intake, thus leading to a lack of power. In contrast, Eglseer *et al.* showed that PLO with low grip strength had a significantly lower protein intake than patients without SO [50]. In addition, no patient in the Semaglutide subgroup significantly increased his physical activity at 6 months.

Among the other risk factors we studied, body weight cycling is often described as a factor associated with SO. Indeed, when weight is regained, the increase in fat mass predominates over variations in lean mass [51]. In our study, body weight cycling tended to be associated with SO without being significant. However, in the literature, studies on the association between lean mass loss and body weight cycling are divergent. For example, Rossi *et al.* have shown that patients with a large number of body weight cycling have 6.2-times greater risk of developing SO [23]. On the other hand, Murphy *et al.* found no significant change in body composition, particularly lean mass, in the case of body weight cycling [52].

As mentioned above, weight loss can lead to SO. Semaglutide is a treatment known to cause weight loss in treated patients. It therefore seems legitimate to question the effect of Semaglutide on body composition. Few studies have investigated the evolution of body composition after initiation of treatment, but the results are discordant. McCrimmon *et al.* report a reduction in muscle mass, while Uchiyama *et al.* and Ozeki *et al.* find no change in lean body mass percentage, and Volpe *et al.* no change in muscle strength [29], [53]–[55].

In all studies, the authors found an increase in the proportion of relative skeletal muscle mass (i.e. compared with the proportion of fat mass). Ren *et al.* have the same observation after using Semaglutide on mice [56].

In our study, there was no increase in the prevalence of SO after 6 months of Semaglutide treatment but the small sample size limits the reliability of this finding and no conclusion can be made without additional follow-up. Only one patient out of the 7 who completed the 6-month follow-up developed SO. This patient already had impaired muscle strength at baseline, with a pathological 5TSTS. In addition, she had a pathological SARC-F questionnaire at inclusion (score = 5). With a loss of only 3kg at 6 months, her appendicular lean body mass was reduced, possibly because of a sedentary lifestyle and with a context of depression.

The SARC-F screening questionnaire has only been validated in subjects over 65 years. In our study, we found low sensitivity and specificity (33% and 79% respectively) for this questionnaire, which therefore does not appear to be a good screening questionnaire for sarcopenia in young subjects. This is probably due to the high prevalence of presarcopenia in our population. Indeed, in consideration of their young age, patients have a discrete impairment of muscular strength, which is not symptomatic, for example, no patient has fallen down the stairs in the last 12 months (Question 5).

Furthermore, in our study, gender was not significantly associated with SO, but the prevalence of SO was higher in men, as in the studies by Ji *et al.*, Lim *et al.* and Johson Stoklossa *et al.* [57]–[59]. This gender difference could be explained by sex hormones. Indeed, an association between sarcopenia and lower salivary testosterone levels was shown in the study by Diago *et al.* [60]. However, testosterone was not significantly associated with sarcopenia in our study, probably due to missing data.

Finally, we found only BMI as a clinical factor independently associated with SO, highlighting the need to test patients for skeletal muscle function in clinical practice.

Our study has some limitations. First, there is a selection bias in relation to the site of patient inclusion (tertiary care center). Indeed, patients followed in CSO generally present morbid and complicated obesity and require specialized support. The PLO included therefore had a higher BMI than patients in other studies, which may overestimate the prevalence of SO. In addition, due to the cross-sectional nature of the first part of our study, some data could not be found, notably concerning protein intake and biological parameters, which may lead to a lack of power.

However, this is a real-life study of a pathology under studied in young subjects. To our knowledge, this is the first study to evaluate the SARC-F questionnaire as a screening tool in young subjects. Finally, we studied the evolution of body composition after the introduction of Semaglutide, a subject not often discussed in the various studies on weight loss under this treatment.

In conclusion, the prevalence of SO in PLO with a mean age of less than 45 years and followed in CSO was 18.6%, with a higher prevalence in patients eligible for treatment with Semaglutide (prevalence 25%) and in men. BMI was the only risk factor independently associated with SO in multivariate analysis.

Moreover, at 6 months after introduction of Semaglutide, there was no increase in SO in a sample of 7 patients. Future longitudinal studies involving a larger number of subjects and a longer follow-up are needed to determine conclusively if this treatment is associated with changes in muscle mass and strength.

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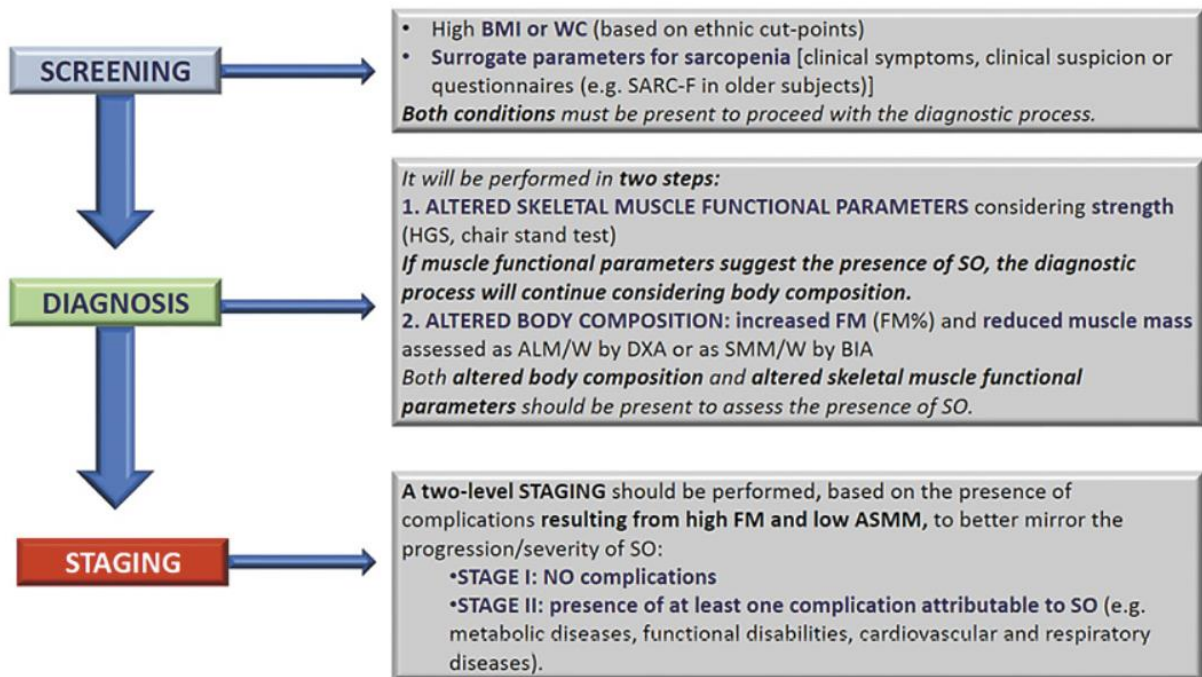
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## APPENDIX

### Appendix 1 : Diagnostic algorithm for the assessment of sarcopenic obesity according to ESPEN et EASO



*ALM/W*, appendicular lean mass adjusted to body weight; *ASMM*, absolute skeletal muscle mass; *BIA*, bioelectrical impedance analysis; *BMI*, body mass index; *DXA*, dual X-ray absorptiometry; *ESPEN*, European Society for Clinical Nutrition and Metabolism; *EASO*, European Association for the Study of Obesity; *FM*, fat mass; *HGS*, handgrip strength; *SMM/W*, total skeletal muscle mass adjusted by weight; *SO*, sarcopenic obesity; *WC*, waist circumference; *SARC-F*, strength, assistance with walking, rising from a chair, climbing stairs and falls.

Appendix 2: table of hand-grip standards by gender and age group according to the manufacturer

AGE	MALE			FEMALE		
	Weak	Normal	Strong	Weak	Normal	Strong
10-11	<12.6	12.6-22.4	>22.4	<11.8	11.8-21.6	>21.6
12-13	<19.4	19.4-31.2	>31.2	<14.6	14.6-24.4	>24.4
14-15	<28.5	28.5-44.3	>44.3	<15.5	15.5-27.3	>27.3
16-17	<32.6	32.6-52.4	>52.4	<17.2	17.2-29.0	>29.0
18-19	<35.7	35.7-55.5	>55.5	<19.2	19.2-31.0	>31.0
20-24	<36.8	36.8-56.6	>56.6	<21.5	21.5-35.3	>35.3
25-29	<37.7	37.7-57.5	>57.5	<25.6	25.6-41.4	>41.4
30-34	<36.0	36.0-55.8	>55.8	<21.5	21.5-35.3	>35.3
35-39	<35.8	35.8-55.6	>55.6	<20.3	20.3-34.1	>34.1
40-44	<35.5	35.5-55.3	>55.3	<18.9	18.9-32.7	>32.7
45-49	<34.7	34.7-54.5	>54.5	<18.6	18.6-32.4	>32.4
50-54	<32.9	32.9-50.7	>50.7	<18.1	18.1-31.9	>31.9
55-59	<30.7	30.7-48.5	>48.5	<17.7	17.7-31.5	>31.5
60-64	<30.2	30.2-48.0	>48.0	<17.2	17.2-31.0	>31.0
65-69	<28.2	28.2-44.0	>44.0	<15.4	15.4-27.2	>27.2
70-99	<21.3	21.3-35.1	>35.1	<14.7	14.7-24.5	>24.5

Appendix 3 : SARC-F questionnaire (french version)

Composante	Question	Score
<b>Force</b>	Avez-vous des difficultés pour lever et transporter 4,5 kg ?	Aucune = 0 Un peu =1 Beaucoup ou incapable = 2
<b>Troubles de la marche</b>	Avez-vous des difficultés pour traverser une pièce ?	Aucune = 0 Un peu =1 Beaucoup ou incapable = 2
<b>Lever d'une chaise</b>	Avez-vous des difficultés pour vous lever d'une chaise?	Aucune = 0 Un peu =1 Beaucoup, avec aide ou incapable = 2
<b>Montée des escaliers</b>	Avez-vous des difficultés pour monter 10 marches ?	Aucune = 0 Un peu =1 Beaucoup, avec aide ou incapable = 2
<b>Chutes</b>	Combien de fois êtes-vous tombé dans les 12 derniers mois ?	Pas de chute = 0 1 à 3 chutes = 1 ≥ 4 chutes=2

*Appendix 4: Cut off values for Sarcopenic Obesity diagnosis, extract from ESPEN and EASO procedures. Donini et al.*

Body composition					
<b>SMM/W (BIA or DXA)</b>	CLASS I of Sarcopenia (1-2 SD): 31.5-37% for M 22.1-27.6% for F; CLASS II of Sarcopenia (< 2 SD): <31.5% for M <22.1% for F	Class I: SMM/W within -1 to -2 SD of young adult values Class II: SMM/W -2 SD of young adult values	Mixed ethnicity, M and F, 18-39y	6414	[61]
	CLASS I of Sarcopenia (1-2 SD): 42.9-38.2% for M 35.6-32.2% for F; CLASS II of Sarcopenia (< 2 SD): <38.2% for M <32.2% for F	Class I: SMM/W within -1 to -2 SD of young adult values Class II: SMM/W -2 SD of young adult values.	Asian, M and F, ≥40y (RG 18-40y)	309 (273 RG)	[57]
	CLASS I of Sarcopenia (1-2 SD): 27-23% for F CLASS II of Sarcopenia (< 2 SD): <23% for F	Class I: SMM/W within -1 to -2 SD of young adult values Class II: SMM/W -2 SD of young adult values	Caucasian, F, 20-50y (RG)	120 (RG)	[54]
<b>ALM/W (DXA)</b>	<29.9% for M <25.1% for F	1 SD below the sex specific mean for young adults	Asian, M and F, mean age 28.4 ± 3.1 and 26.3 ± 2.6	70 (RG)	[62]
	<30.1% M <21.2% F	1 SD below the mean of a young population RG	Asian, M and F, ≥ 40y (RG 20-39y)	10118 (5944 RG)	[63]
	<30.65% for M <23.9% for F	1 SD below the mean of a healthy young RG	Asian, M and F, ≥ 65y (RG 20-39y )	3483 (4192 RG)	[64]
	< 25.7% for M < 19.4% for F	2 SD below the mean of a healthy young RG	Mixed ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, "other"), M and F, ≥ 60y (RG 18-59y)	4984 (10877 RG)	[65]
	<30.3% for M <23.8% for F	1 SD below the mean of a healthy young RG	Asian, M and F, ≥ 20y (RG 20-39y)	11521 (4987 RG)	[66]
	< 32.5% for M < 25.7% for F	1 SD below the mean of a healthy young RG	Asian, M and F, ≥ 60y (RG 20-39y)	2943 (2781 RG)	[67]
	< 29.53% for M < 23.2% for F	2 SD below the mean of a healthy young RG	Asian, M and F, ≥60y (RG 20-39y)	2221 (2269 RG)	[68]
	<31.3% for M <24.76% for F	1 SD below the mean of a healthy young RG	Asian, M and F, ≥40y (RG 20-39y)	3320	[69]
	<32.2% for M <25.6% for F	Class I: within -1 to -2 SD of the healthy young adult values Class II: 2 SD below the mean of the healthy young adult values	Asian, M and F, ≥20y (RG 20-39y)	10485 (2513 RG)	[70]
	<29.5% for M < 23.2% for F	2 SD below the mean of a healthy young RG	Asian, M and F, ≥50y (RG 20-40y)	3169 (2392 RG)	[71]
	< 26.8% for M < 21% for F	2 SD below the mean of the young RG	Asian, M and F, ≥50y (20-40y RG)	2893 (2113 RG)	[72]
	< 32.2 for M < 25.5% for F	2 SD below the mean of the young RG	Asian, M and F, ≥20y (RG 20-30y)	15132 (2200 RG)	[73]
	< 44% for M <52 % for F	2 SD below the mean of the young RG	Asian, M and F, ≥60y (RG 20-39y)	1433 (1746 RG)	[74]
< 28.27% for M < 23.47% for F	2 SD below the mean of the young RG	Caucasian, M and F, 18-65y (RG 20-39y)	727 (222 RG)	[75]	

**Legend:** 6MWT 6 minutes walking test, **ALM** appendicular lean mass, **AUC** area under the curve, **BIA**, bioelectrical impedance analyses, **BMI** body mass index, **CART** Classification and Regression Tree model, **CPF** Composite Physical Function, **DXA**, dual-energy X-ray absorptiometry, **FM** fat mass, **HGS** hand grip strength, **mPPT** modified physical performance test, **RFM** relative fat mass, **RG** reference group, **ROC** Receiver operating characteristic, **SD** standard deviation, **SMM** skeletal muscle mass, **TMSE** Thai mental state examination, **W** weight, **WC** waist circumference,

## Abstract

**Introduction:** Sarcopenic obesity (SO) is defined as the combination of excess adiposity and low muscle mass and function. Recently, ESPEN and EASO proposed a consensus screening and diagnostic procedures of SO. The aim of our study was to assess the prevalence of SO in patients living with obesity (PLO) followed in a specialized obesity health care centre (*centre spécialisé en obésité*, CSO). The secondary objectives were to determine the clinical and biological factors associated with SO and to evaluate the effect of 6 months Semaglutide treatment on body composition.

**Methods:** We included PLO with a BMI  $\geq 30$  kg/m<sup>2</sup>. The diagnosis of SO, was made according to the 2-steps ESPEN EASO algorithm. Skeletal muscle function and strength were collected by handgrip strength (HGS) and 5-time sit-to-stand test (5TSTS) and analysis of body composition was collected by dual-energy X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA). SO was defined as present in our study population if HGS AND/OR the 5TSTS was pathological, AND the appendicular lean mass/weight ratio was strictly below 19.9% in women and 25.7% in men. A sub-group analysis was conducted on the evolution of SO in patients after 6 months anti-obesity Semaglutide treatment.

**Results:** 129 patients with a mean age of  $44.4 \pm 13.8$  years and a mean BMI of  $43.4 \pm 7.3$  kg/m<sup>2</sup> were included. The prevalence of SO was 18.6% and the prevalence in patients eligible for treatment with Semaglutide was higher (25%). There was no statistical difference regarding the functional test realised, or the method used for body composition (DXA or BIA) in terms of SO diagnosis. Only BMI was independently associated with SO ( $p = 0.026$ , OR 1.08 CI95% [1.01-1.16]). Seven patients were included in the sub-group analysis and only one developed a SO at 6 months. Semaglutide treatment was not significantly associated with the diagnosis of SO after 6 months of treatment., but this result must be taken with caution given the small sample size and the absence of long-term follow-up.

**Discussion:** In our real-life study, the prevalence of SO according to ESPEN and EASO algorithm was very high 18.6% in young patients followed up in CSO. We found only BMI as a clinical factor independently associated with SO, highlighting the need to test patients for skeletal muscle function in clinical practice. At 6 months Semaglutide treatment we found no increase in SO in a sample of 7 patients, but this needs larger cohort and better consensus on pathological thresholds in young patients. These interesting data have been collected in other CSO and will be included in the SEMASEARCH national multicentric study.

## Résumé

**Introduction :** L'obésité sarcopénique (OS) est définie comme la combinaison d'un excès d'adiposité et d'une diminution de la masse et de la fonction musculaires. Récemment, l'ESPEN et l'EASO ont proposé un consensus sur les procédures de dépistage et de diagnostic de l'OS. L'objectif de notre étude était d'évaluer la prévalence de l'OS chez des patients vivant avec une obésité (PVO) suivis dans un centre spécialisé en obésité (CSO). Les objectifs secondaires étaient de déterminer les facteurs cliniques et biologiques associés à l'OS et d'évaluer l'effet de 6 mois de traitement par Semaglutide sur la composition corporelle.

**Méthodes :** Nous avons inclus les PVO ayant un IMC  $\geq 30$  kg/m<sup>2</sup>. Le diagnostic d'OS a été établi selon l'algorithme de l'ESPEN et de l'EASO en deux étapes. La fonction et la force des muscles squelettiques ont été mesurées par handgrip (HG) et par test du lever de chaise (5 times sit-to-stand test, 5TSTS), et l'analyse de la composition corporelle a été réalisée par absorptiométrie à rayons X (DXA) ou par impédancemétrie bioélectrique (BIA). L'OS a été définie comme présente dans la population de l'étude si le HG ET/OU le 5TSTS étaient pathologiques ET si le rapport masse maigre appendiculaire/poids était strictement inférieur à 19,9 % chez les femmes et 25,7 % chez les hommes. Une analyse de sous-groupe a été réalisée sur l'évolution de l'OS chez les patients après 6 mois de traitement anti-obésité par Semaglutide.

**Résultats :** 129 patients avec un âge moyen de  $44,4 \pm 13,8$  ans et un IMC moyen de  $43,4 \pm 7,3$  kg/m<sup>2</sup> ont été inclus. La prévalence de l'OS était de 18,6 % et la prévalence chez les patients éligibles au traitement par Semaglutide était plus élevée (25 %). Il n'y avait pas de différence statistique concernant le test fonctionnel réalisé (HG ou 5TSTS) ou la méthode utilisée pour la composition corporelle (DXA ou BIA) en termes de diagnostic de l'OS. Seul l'IMC était indépendamment associé à l'OS ( $p = 0,026$ , OR 1,08 CI95% [1,01-1,16]). Sept patients ont été inclus dans l'analyse de sous-groupe et un seul a développé une OS à 6 mois. Le traitement par Semaglutide n'était pas significativement associé au diagnostic d'OS après 6 mois de traitement, mais ce résultat doit être pris avec précaution compte tenu de la petite taille de l'échantillon et de l'absence de suivi à long terme.

**Discussion :** Dans notre étude en vie réelle, la prévalence de l'OS selon l'algorithme de l'ESPEN et de l'EASO était très élevée (18,6 %) chez les jeunes patients suivis au CSO. Nous n'avons trouvé que l'IMC comme facteur clinique indépendamment associé à l'OS, soulignant la nécessité de tester la fonction musculaire squelettique des patients dans la pratique clinique. Après 6 mois de traitement par Semaglutide, nous n'avons pas trouvé d'augmentation de l'OS dans un échantillon de 7 patients, mais cela nécessite une plus grande cohorte et un meilleur consensus sur les seuils pathologiques chez les jeunes patients. Ces données intéressantes ont été recueillies dans d'autres CSO et seront incluses dans l'étude multicentrique nationale SEMASEARCH.

## SERMENT D'HIPPOCRATE

**Au moment d'être admis(e) à exercer la médecine, je promets et je jure d'être fidèle aux lois de l'honneur et de la probité.**

Mon premier souci sera de rétablir, de préserver ou de promouvoir la santé dans tous ses éléments, physiques et mentaux, individuels et sociaux.

Je respecterai toutes les personnes, leur autonomie et leur volonté, sans **aucune discrimination selon leur état ou leurs convictions**. J'interviendrai pour les protéger si elles sont affaiblies, vulnérables ou menacées dans leur intégrité ou leur dignité. Même sous la contrainte, je ne ferai pas **usage de mes connaissances contre les lois de l'humanité**.

**J'informerai les patients des décisions envisagées, de leurs raisons et de leurs conséquences.**

Je ne tromperai **jamais leur confiance** et **n'exploiterai pas le pouvoir hérité** des circonstances pour forcer les consciences.

**Je donnerai mes soins à l'indigent et à quiconque me les demandera**. Je ne me laisserai pas influencer par la soif du gain ou la recherche de la gloire.

**Admis(e) dans l'intimité des personnes, je tairai les secrets qui me seront confiés**. **Reçu(e) à l'intérieur des maisons, je respecterai les secrets des foyers** et ma conduite ne servira pas à corrompre les mœurs.

Je ferai tout pour soulager les souffrances. Je ne prolongerai pas abusivement les agonies. Je ne provoquerai jamais la mort délibérément.

**Je préserverai l'indépendance nécessaire à l'accomplissement de ma mission**. Je n'entreprendrai rien qui dépasse mes compétences. Je les entretiendrai et les perfectionnerai pour assurer au mieux les services qui me seront demandés.

**J'apporterai mon aide à mes confrères ainsi qu'à leurs familles dans l'adversité.**

**Que les hommes et mes confrères m'accordent leur estime si je suis fidèle à mes promesses ; que je sois déshonoré(e) et méprisé(e) si j'y manque.**