Interventional biological markers for sarcopenia and muscle frailty in Iraqi subjects

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Objective This study was carried out to define the changes of some biomarkers in sarcopinic and compared the results with non-sarcopinic subjects.
Methods Between the first of September 2016 to the end of March 2017, sarcopenic subjects (100 males and females) and non-sacopenic subjects (50 males and females) were included in this study.
Results Mean values of appendicular skeletal muscle mass (ASM), LBM and a1-ACA in control group were more than in sarcopenic group with a highly significant difference $P < 0.01$ between the values of ASM, LBM and a significant difference $P < 0.05$ between the values of a1-ACA. While 1-interleukin (IL-6), hs-CRP, and BMI mean values in sarcopenic group were more than in control group with a highly significant difference between their values they were, respectively. The mean values of ASM, LBM and a1-ACA in control group were more than in sarcopinic group, which is more in males than females and the values were inversely proportional to age. The mean values of IL-6, hs-CRP, and BMI in sarcopinic group were more than in control group, which is less in male than female, and the values were diretly proportional to the age except BMI in male, which is more than female.
Keywords sarcopenia, dsability, Iraq, physical performance

Introduction

In 1989, Irwin Rosenberg proposed the term sarcopenia (from Greek σάρξ *sarx*, "flesh" and πενία *penia*, loos" or 'poverty of flesh') it is mean the degenerative or progressive loss of skeletal muscle mass, strength and/or function (also called senile muscle atrophy) is an age-related loss of skeletal muscle mass and function.[1] Sarcopenia is either primary age-related sarcopenia with no other causes except ageing or secondary sarcopenia that is age related to activity (bed rest life style), disease (heart, liver, kidney, inflammatory diseases), nutrition (inadequate dietary intake, malabsorption, anorexia).[2]

Sarcopenia is a major cause of frailty. It has multiple causes including, diseases, decreased caloric intake, and poor blood flow to muscle, mitochondrial dysfunction, a decline in anabolic hormones, and an increase in proinflammatory cytokines.[3]

Sarcopenia's muscle wasting begins to appear in the fourth decade of life and accelerates after the age of 75 years, but it may also speed up as early as 65 or as late as 80. It is estimated that sarcopenia affects 30% of people over the age of 60 and more than 50% of those over the age of 80. Between the ages of 30 and 60, the average adult will gain 0.45 kg of weight and lose 0.225 kg of muscle yearly, a total gain of 13.5 kg of fat and a loss of 6.75 kg of muscle. After the age of 70, muscle loss accelerates to 15% per decade.[4]

There is a significant association between the inflammatory markers 1-interleukin (IL-6), CRP levels and loss of skeletal muscle. High concentrations of IL-6 correlate with movement disabilities, slower walking speed, and lower grip strength. Dehydroepiandrosterone (DHEA) inhibits IL-6 production. As DHEA levels drop with age, its inhibitory influence on IL-6 production becomes attenuated. Interestingly, elevated IL-6 production appears to play a role in anorexia (loss of appetite). Loss of appetite is a major concern in older adults as insufficient nutrient intake can contribute to muscle loss.[5] Consequently, IL-6 may mediate sarcopenia directly via its catabolic effects on muscle and indirectly as diminished appetite increasing the risk of malnutrition. Cortisol and IL-6 are released into the bloodstream as part of an inflammatory response. Levels of these agents change in sarcopenia, cortisol increasing along with IL-6.[6]

IL-6 promotes chronic inflammation and plays a large role in joint inflammation and in the hepatic production of hs-CRP and alpha 1-antichymotrypsin (α1-ACT).[7]

Interleukin (IL)-6

Interleukin-6 (IL-6) is a pro-inflammatory cytokine that also has an important role in immunity. Many types of cells, including macrophages, T cells, fibroblasts, and endothelial cells, produce IL-6 in response to stimuli such as bacteria, viruses, and other cytokines. Physical exercise produces a 10-fold increase in serum IL-6, mostly released from skeletal muscle and perhaps aimed at potentiating the insulin stimulation of glycogen synthesis in muscle cells.[8]

Because of its relationship with adiposity, it has been hypothesized that IL-6 and other proinflammatory cytokines are the main causes of insulin resistance. Elevated serum IL-6 is positively associated with the markers of physical frailty such as low-walking speed, poor muscle strength. IL-6 may contribute to sarcopenia through different mechanisms, including a direct interference with insulin signal transduction and inhibition of the production and biological activity of insulin-like growth factor-1 (IGF-1).[9] Diet may affect IL-6 secretion both acutely and chronically. A high-fat meal, but not a high-carbohydrate meal, increases plasma levels of IL-6.[10] Circulating levels of polyunsaturated fatty acids,

especially total n-3 fatty acids, are independently associated with lower levels of proinflammatory markers, including IL-6. Moreover, frail study participants have higher levels of IL-6 than non-frail, age-matched individuals.[11]

C-reactive protein (CRP)

It is a glycoprotein produced by the liver and its level rises when there is inflammation through the body. Other names for CRP are high-sensitivity C-reactive protein (hs-CRP) and ultra-sensitive C-reactive protein (us-CRP).

A high level of CRP in the blood is a marker of any condition that causes: heart diseases, (lymphoma), diseases of the immune system, Crohn's (or Crohn), giant cell arteritis, rheumatoid arthritis, inflammatory bowel disease, osteomyelitis, burns, trauma and infections, such as pneumonia or tuberculosis.[12]

The serum hs-CRP levels were significantly increased by obesity and by sarcopenic obesity status. Therefore, inflammation may have an important role in the development of sarcopenic obesity. High body fat and low grip strength led to an increase in CRP levels. IL-6 plays a central role in the hepatic production of hs-CRP, α1-ACT.[13]

Inflammatory cytokines have been shown to prompt muscle wasting, ultimately stimulating protein catabolism and suppressing muscle synthesis. Sarcopenia seems to be associated with elevated serum CRP levels. Future longitudinal studies are needed to clarify this relationship.[14]

Alpha1-antichymotrypsin (α1-ACT)

Alpha-1-antichymotrypsin (α1-ACT), also called SERPINA3, is a member of the serine protease inhibitor (serpin) family of acute phase proteins. It inhibits a wide variety of proteases, and protects tissues from enzymes causing inflammatory cells especially neutrophil elastase and has a reference range in blood of 1.5–3.5 g/l, but the concentration rise many fold upon a cut inflammation in the absence or deficiency of α1-ACT, neutrophil elastase is free to break down elastin.[15]

Although α1-ACT is predominantly produced in the liver, it is also synthesized in the brain. Elevated levels of α1-ACT are found in the brain, serum and cerebrospinal fluid (CSF) of AD patients and high levels of α1-ACT in plasma is associated with cognitive decline in elderly subjects. This suggests that α1-ACT may serve as a biomarker for early diagnosis of Alzheimer disease.[16] Deficiency of this protein has been associated with liver diseases. Mutations have been identified in patients with Parkinson disease and chronic obstructive pulmonary disease. IL-6 promotes chronic inflammation and plays a large role in joint inflammation, contributes to the production of α1-ACT and together, the two inflammatory markers are associated with a two- to three-fold risk of reduced muscle strength in older adults. α1-ACT were associated with the loss of muscle strength or muscle mass (sarcopenia) in older persons.[17]

Subjects, Materials and Methods

Subjects

In this study, specimens were collected during the period from the first of September 2016 to the end of March 2017. The study was included 100 participated (sarcopenic) subjects (50 males and 50 females) age range ≥65–90 years and 50 participated subjects (not sarcopenic) as a control group aged between 40 and 65 (25 males and 25 females) from Baghdad teaching hospital. Subjects with any inflammatory disease (RA, SLF, etc), DM, thyroid disease, using steroid therapy were excluded in this study. The study group was divided into three groups depending on age (years) (≤65–69, 34 subjects – 17 males and 17 females), (70–79, 34 subjects – 17 males and 17 females) and (≥80 years, 32 subjects – 16 males and 16 females).

Blood Samples

Blood samples were collected in the morning following an overnight fasting. A quantity of 5 ml was taken from a peripheral vein and put in a gill tube without any anticoagulant. Blood in the tubes were allowed to clot for 30 min and centrifuged at 1500 rpm for 10 min. Each subject serum was immediately put in to three Eppendorf tubes and stored at −80°C freezer until analysis.

Materials and Methods

For the diagnosis sarcopenic subjects, the study was carried out by assessing the following parameters:

Clinical diagnostic measurements

Physical Performance: Short Physical Performance Battery (SPPB)

The Short Physical Performance Battery (SPPB) has been used in this study to diagnose sarcopinic subjects because it is emerged as one of the most promising tools to measure physical performance status and evaluate functional capability in older adults. It's based on three timed tasks: standing balance, walking speed, and chair stand tests. The timed results of each subtest are rescaled according to predefined cut-points for obtaining a score ranging from 0 (worst performance) to 12 (best performance).[18]

Measuring the Skeletal Mass Index by Dual Energy X-ray Absorptiometry (DEXA)

Total and regional body composition was evaluated using dual energy X-ray absorptiometry (DEXA) technologies and all DEXA scans were ordered by a licensed physician in Baghdad Hospital. The muscle mass of the four limbs from a DXA scan summed as appendicular skeletal muscle mass (ASM) and defined a skeletal muscle mass index (SMI) as ASM/height^2 (kg/m^2) to adjust for the strong association between body height and ASM. The cut-off values for sarcopenia was (7.25 (kg/m^2) for men and 5.67 (kg/m^2) for women).[19]

Measures of total body composition included (total lean body mass, appendicular lean body mass body mass index, bone mass and whole body fat were obtained on a whole body scan).[20]

Biological Markers (in serum)

IL-6, hs-CRP and α1-ACT were measured in serum by using ELISA.

Results

Table 1 shows that the mean values of (ASM, LBM and α1-ACA) in control group were more than study group with a direct relation between α1-ACA and ASM, LBM, while for the other variables mean values in study group were more than control group with a highly significant differences $P < 0.01$ between the study group and the control group for all clinical variables except for (α1-ACA). There was a significant difference $P < 0.05$ between them, with indirect relation between (hs-CRP, IL-6, BMI) and (ASM, LBM).

The mean value of ASM for control group is more than subjects group with a highly significant difference between the two groups $P < 0.01$ as shown in Fig. 1.

The mean value of TLBM for control group is more than subjects group with a highly significant difference $P < 0.01$ between the two groups as shown in Fig. 2.

The mean value of α1-ACT for study group is more than control group with a significant $P < 0.05$ difference between the two groups as shown in Fig. 3.

The mean value of IL-6 for study group is more than control group with a highly significant difference $P < 0.01$ between the two groups as shown in Fig. 4.

The mean value of hs-CRP for study group is more than control group with a highly significant difference $P < 0.01$ between the two groups as shown in Fig. 5.

The mean value of body mass index BMI for study group is more than control group with a highly significant difference $P < 0.01$ between the two groups as shown in Fig. 6.

The results in Table 2 show that there were indirect relationship between ASM, LBM and (IL-6, hs-CRP, BMI) for all ages, and the mean values of (IL-6, hs-CRP, BMI) for all ages increase with increase the age, while there were a direct relationship between the mean values of (ASM, LBM) and (α1-ACT) for all ages, and the mean values of all ages decrease with increase the age with a highly significant difference ($P < 0.01$) between all the variables for all ages.

The results in Table 3 show that there was an indirect relationship between ASM, LBM and (IL-6, hs-CRP, BMI), while there was a direct relationship between ASM, LBM and (α1-ACT) with a highly significant difference $P < 0.01$ between the mean values of all the variables between males and females except α1-ACT. There were a significant difference $P < 0.05$ between the mean values.

Table 4 show the most effective variables limitation factor R^2 in sarcopenia, and they were, respectively, (ASM/height^2, TLBM, then α1-ACA) and all have a highly significant correlation $P < 0.01$ with each other.

Table 1. **Comparison (Mean ± SD) between study and control groups for the following variables**

Clinical variables	Group	No	Mean ± SD	*t*	*P*-value	C.S
ASM/High^2 (kg/m^2)	Control	50	8.038 ± 0.888	15.378	0.000	$P < 0.01$ (HS)
	Study	100	5.896 ± 0.759			
LBM (kg)	Control	50	31.584 ± 1.7284	34.891	0.000	$P < 0.01$ (HS)
	Study	100	21.584 ± .402			
α1ACT (ng/ml)	Control	50	28.302 ± 5.346	2.106	0.037	$P < 0.05$ (S)
	Study	100	37.427 ± 30.358			
IL-6 (ng/l)	Control	50	26.859 ± 7.403	18.255	0.000	$P < 0.01$ (HS)
	Study	100	72.274 ± 16.772			
hs-CRP (ng/ml)	Control	50	4.960 ± 1.610	3.929	0.000	$P < 0.01$ (HS)
	Study	100	6.652 ± 2.820			
BMI (kg/m^2)	Control	50	29.594 ± 2.882	16.648	0.000	$P < 0.05$ (S)
	Study	100	34.888 ± 4.254			

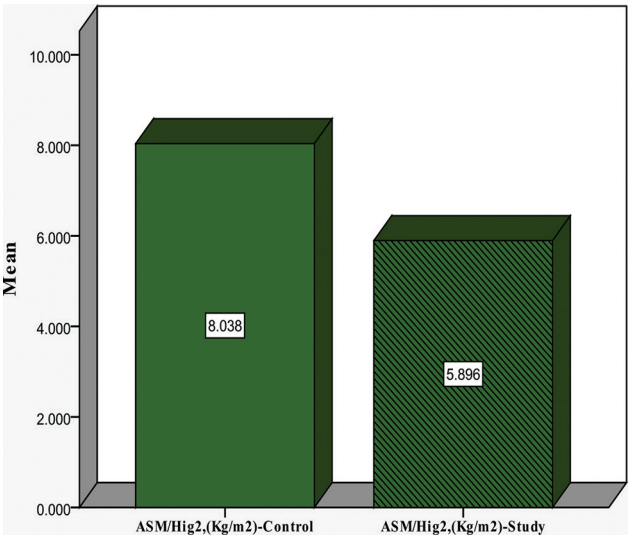

Fig. 1 **The mean values of appendicular skeletal muscle mass ASM/Hig^2, (Kg/m^2) in control & study groups.**

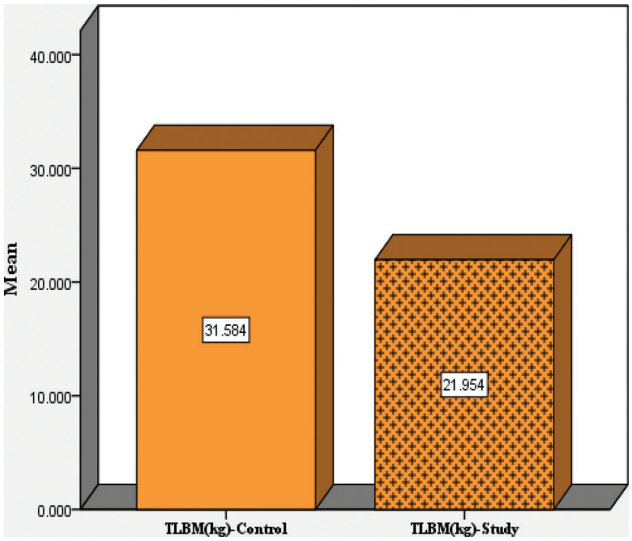

Fig. 2 **The mean of total lean body mass TLBM (kg) in control & study groups.**

Statistical analysis

Interferential Data Analysis

We used accept or reject statistical hypotheses as follows:

1. T-test was used to compare the means parameters between the groups.
2. Pearson correlation coefficient (r) was used to test the relation between two parameters.
3. *P* value.
   -If $P \leq 0.05$ significant
   -If $P \leq 0.01$ high significant
   -If $P \geq 0.05$ non-significant
4. R^2 the most effective variables limitation factor

Descriptive data analysis

1. Tables correlationship (Pearson's correlations).
2. Mean value, standard deviation.

Computer and programmers

All the statistical analyses were done by using Pentium-4 computer through the Statistical Package of Social Science (SPSS) program (Version -10) and excel application (2010) for figures.

Discussion

Scientists summed the muscle mass of the four limbs from a DXA scan as ASM and defined SMI as ASM/height^2 (kg/m^2). They define sarcopenia as a reduction in ASM/height^2, also coin with the term "sarcopenia." Total lean body mass has been a major focus of researchers used it for the past 25 years.[21] The motivating idea is that weakness, a hallmark of physical disability, is determined by skeletal muscle mass. Therefore, a logical strategy to prevent disability would be to slow or reverse age-related decreases in muscle mass and high levels of

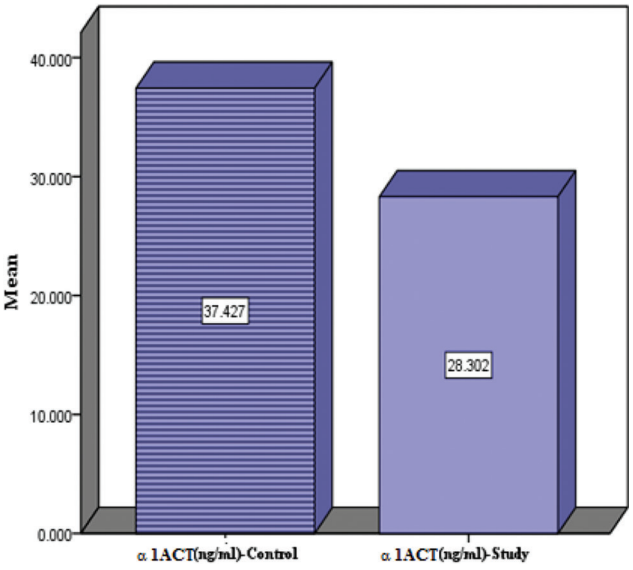

Fig. 3 **The mean of Alpha1-Antichymotrypsin α1-ACT (ng/ml) in control & study groups.**

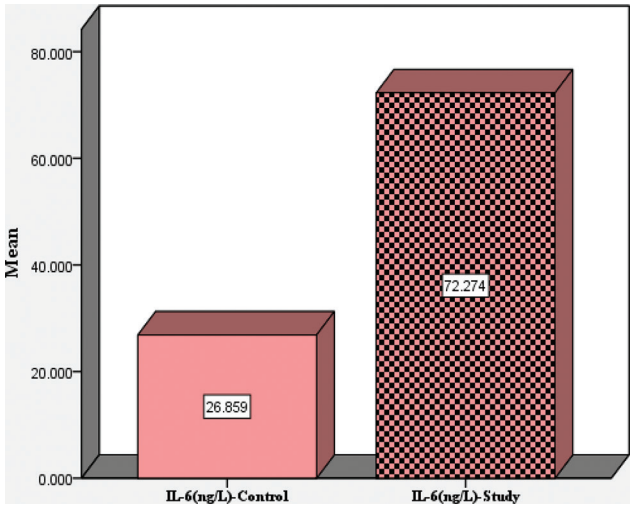

Fig. 4 **The mean of Interlukine-6 IL-6 (ng/L) in control & study groups.**

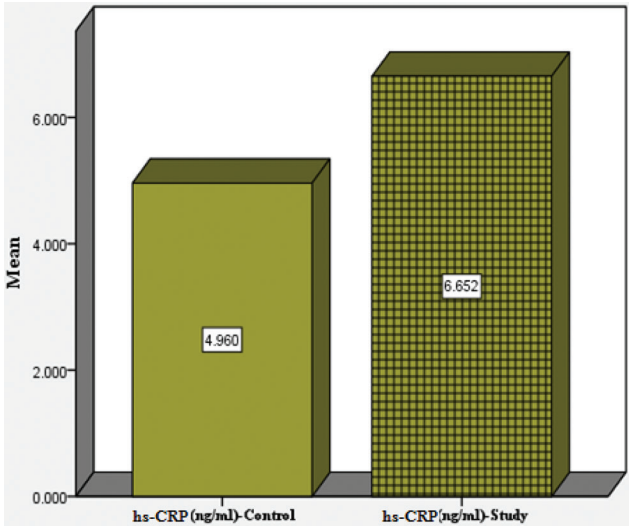

Fig. 5 **The mean of high sensitivity C-reactive protein hs-CRP (ng/ml) in control & study groups.**

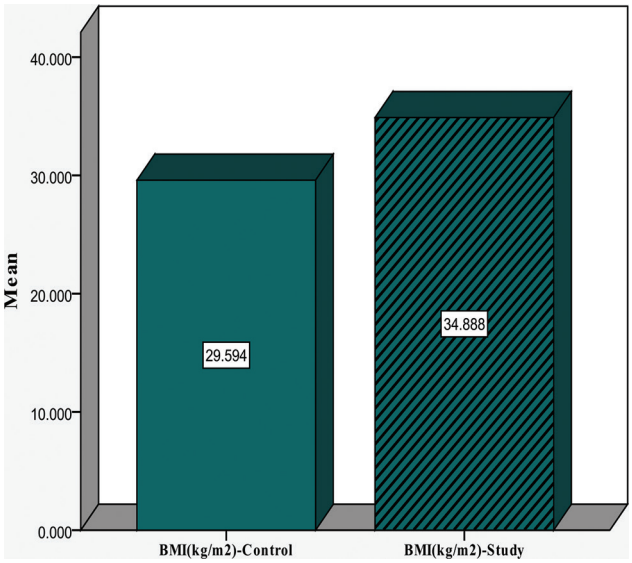

Fig. 6 **The mean of body mass index (BMI) (kg/m^2) in control and study groups.**

Table 2. **Comparison between clinical variables (Mean ± SD) in study groups with age groups**

Clinical variables	Age groups years	No	Mean ± SD	*t*	*P*-value	C.S
	(≤65–69)	34	6.402 ± 0.8167			
ASM/High^2 Kg/m^2	(70–79)	34	5.961 ± 0.502	2.686	0.009	$P < 0.01$ (HS)
	(≥80)	32	5.288 ± 0.442	6.833	0.000	$P < 0.01$ (HS)
	(≤65–69)	34	24.301 ± 1.060			
TLBM (kg)	(70–79)	34	28.854 ± 1.680	21.892	0.000	$P < 0.01$ (HS)
	(≥80)	32	32.862 ± 2.266	2.686	0.000	$P < 0.01$ (HS)
	(≤65–69)	34	72.816 ± 18.071			
α 1ACT (ng/ml)	(70–79)	34	28.669 ± 15.923	10.688	0.000	$P < 0.01$ (HS)
	(≥80)	32	9.132 ± 6.443	18.833	0.000	$P < 0.01$ (HS)
	(≤65–69)	34	57.794 ± 8.741			
IL-6 (ng/L)	(70–79)	34	70.412 ± 9.700	5.635	0.000	$P < 0.01$ (HS)
	(≥80)	32	89.636 ± 13.110	11.674	0.000	$P < 0.01$ (HS)
	(≤65–69)	34	4.089 ± 0.980			
Hs-CRP (ng/ml)	(70–79)	34	6.401 ± 1.527	7.428	0.000	$P < 0.01$ (HS)
	(≥80)	32	9.641 ± 2.323	12.785	0.000	$P < 0.01$ (HS)
	(≤65–69)	34	32.862 ± 2.266			
BMI (kg/m^2)	(70–79)	34	34.301 ± 1.060	4.747	0.000	$P < 0.01$ (HS)
	(≥80)	32	36.854 ± 1.680	31.188	0.000	$P < 0.01$ (HS)

Table 3. **Comparison (Mean ± SD) between clinical variables in study group and gender**

Clinical variables	Gender	No	Mean ± SD	*t*	*P*-value	C.S
ASM/High^2 Kg/m^2	Male	50	6.361 ± 0.773	7.738	0.000	$P < 0.01$ (HS)
	Female	50	5.430 ± 0.354			
TLBM (kg/m^2)	Male	50	38.288 ± 4.040	6.237	0.000	$P < 0.01$ (HS)
	Female	50	33.619 ± 3.421			
α 1ACT (ng/ml)	Male	50	31.082 ± 27.439	2.127	0.036	$P < 0.05$ (S)
	Female	50	43.773 ± 32.047			
IL-6 (ng/L)	Male	50	67.922 ± 14.451	2.674	0.009	$P < 0.01$ (HS)
	Female	50	76.625 ± 17.911			
hs-CRP (ng/ml)	Male	50	5.872 ± 2.428	2.864	0.005	$P < 0.01$ (HS)
	Female	50	7.432 ± 2.989			
BMI (kg/m^2)	Male	50	30.165 ± 4.482	3.107	0.02	$P < 0.01$ (HS)
	Female	50	27.612 ± 3.699			

Table 4. **Relationship between variables for control and study groups with sarcopenia**

Clinical variables	R^2	F	*P*-value	C.S
ASM/height^2 (Kg/m^2)	98.8%	8124.313	0.000	$P < 0.01$ (HS)
TLBM (kg)	86.6%	638.770	0.000	$P < 0.01$ (HS)
α 1ACT (ng/ml)	34.6%	52.331	0.000	$P < 0.01$ (HS)

adiposity accelerate aging-related loss of lean mass. Lean body mass was the most important predictor of upper body strength, controlling for habitual physical activity and dietary protein intake.[22]

Table 1 shows the comparison (Mean ± SD) between study (sarcopinic) and control groups. There were a highly significant difference $P \leq 0.001$ between the values of (ASM, LBM) for the elderly sarcopinic participants and control participants. This result was in agreement with other results, which show that aging is associated with a decline in lean body mass and an increase in adiposity in sarcopenia subjects and decreased ASM/ht^2 and LBM, should be the most suitable index for skeletal muscle mass measurements.[23]

The reason for decrease (ASM, LBM) with aging and with sarcopenia: Muscle mass loss was caused by reduced numbers of muscle fibers, motor units and decline of muscle fiber size. If muscle fibers decrease a critical minimal size, apoptosis begins. Other causes of apoptosis with aging process are

denervation and loss of neurons.[24] With aging muscle metabolism, synthesis of muscle protein, muscle repair capacities decreases and increases the risk of muscle damage.[25] With aging, there were a decline of anabolic hormones, (testosterone, dehydroepiandrosterone, growth hormone, and insulin-like growth factor-I). In men, andropause takes place in this period. The menopause of women begins between 45th and 55th life year. The decline of hormonal leads to decreasing muscle mass and strength.[25]

Besides the loss of anabolic factors such as neural growth factors, growth hormone, androgens and estrogens, and physical inactivity, an increase of catabolic factors such as inflammatory cytokines could contribute to muscle mass and strength loss. Especially interleukin-1β, tumor necrosis factor (TNF)-α, and interleukin-6 support a decrease in muscle mass.[24,25]

The decrease in physical activity with aging process is the key factor in the development of strength and muscle mass loss. Physical inactivity leads to muscle atrophy. Loss of appetite is an additive problem in older adults as insufficient nutrient intake that can also contribute to muscle loss.[26]

Also, the same table shows that the mean values of IL-6 and hs-CRP were increased in sarcopinic subjects compared with control while α 1ACT decrease. There were highly significant difference $P \leq 0.01$ between the mean values of (hs-CRP) and (IL-6) for the elderly sarcopinic participants and control participants while there were a significant difference $P \leq 0.05$ between the mean values of α 1ACT for the elderly sarcopinic participants and control participants. High C-reactive protein and IL-6 are negatively associated with ASM and LBM. That is in agreement with other study, which suggest that higher levels of IL-6 and hs-CRP increase the risk of muscle strength loss, whereas higher levels of α1-ACT decrease the risk of muscle strength loss in older men and women. This is because during inflammation, muscle tissue might be protected from breakdown by high levels of α 1ACT but inflammation persons with high levels of both α 1ACT and IL-6 seem to have an increased risk of muscle strength loss, suggesting that IL-6 is able to suppress or undo the protective role of α1-ACT in muscles.[27]

A clear inverse association was found in a study among ASM and LBM and the inflammatory markers (the serum IL-6 and CRP) in older non-sarcopenic men and women aged 60–84.[28] Also in agreement with other study but in non-sarcopinic subjects were free of chronic diseases. The results showed elevation in the level of C-reactive protein (CRP) which negatively affects skeletal muscle mass in elderly subjects, and have high values of serum hs-CRP seem to be related to reduced protein synthesis and increased protein catabolism.[29]

The explanation that aging is associated with increased free radical formation and circulatory changes that exacerbate inflammatory processes. This inflammatory cascade may include an increase in levels of pro-inflammatory cytokines, such as interleukin (IL)-6 which play a central role in the hepatic production of hs-CRP, α1-ACT[30] and because IL-6 ordinarily an important component of muscle hypertrophy, but an inhibitor of muscle recovery at elevated its concentrations. IL-6 promotes chronic inflammation and plays a large role in joint.[31]

Also age-associated decline in estrogen and testosterone are related to increases in levels of the pro-inflammatory cytokines IL-6 and NFα, which may accelerate the loss of muscle mass during sarcopenia.[32] Increase in visceral fat may lead to the secretion of pro-inflammatory cytokines that may promote a catabolic effect on muscles, as well as insulin resistance. C-reactive protein and interleukin-6 (IL-6) are positively associated with total fat mass and negatively associated with appendicular lean mass. Consequently, body fat may play a role in sarcopenia by influencing hormones and cytokines that affect muscle mass. When obese patients undergo weight loss, CRP and IL-6 are significantly reduced.[33]

As Table 1 shows that in sarcopenic subjects, BMI was more than in control subjects with a highly significant difference between them. This is in agreement with a study, which got the same results that sarcopenia is common in adults over the age of 65 years and increases with age. BMI is a strong predictor of skeletal muscle mass in women and men.[34] The increase in body weight and fatness are probably due to progressive decline in total energy expenditure stemming from decreased physical activity and reduced basal metabolic rate.[35] Body fat level is often associated with insulin resistance. When combined with a great amount of amino acids in the blood, insulin brings on muscle protein synthesis. Alone, insulin inhibits excessive muscle protein breakdown and counters the catabolic effects of cortisol. Insulin resistance adversely affects those processes.[36]

The results in Table 2 show the relation between different variables and ages of sarcopinic subjects. First ASM and TLBM for the three ages, there were a highly significant difference between the three ages and the values decreases with increasing age. That is similar to other research which investigated the relationship between aging and ASM, TLBM, BMI and sarcopenia[37] but no any research found the relationship between the three sarcopinic ages like this research. They made only comparison between one old age (sarcopinic) with the same age non-sarcopinic. The explanation for why ASM, TLBM were decreased with aging as mentioned previously in Refs. 24–26 and for BMI.[35,36]

The results in Table 2 show that the relation between mean values of IL-6 and hCRP are increasing with aging in sarcopinic subjects with a highly significant difference between the three ages. While mean values of α1-ACT decrease with aging with a highly significant difference between values of study age groups, the value of α1-ACT decreases because of its reverse relation with IL-6 and hsCRP that was in agreement with Ref. 38. The results in this research are in agreement with other study which show IL-6 and hc-CRP increase with age but the difference those researchers have taken subjects who suffered from aging-related disability with poorer cognitive and/or functional performance, a higher risk of mortality and made the comparison between ages the researchers did not take sarcopinic subjects with different ages.[38] No other study did comparison between sacopinic subjects and ages like this. For α1-ACT, none of the research did any work about the relation between mean values of α1-ACT and ages for sacopinic subjects. Another reason for increase plasma level of IL-6, the plasma levels of (DHEA) and its sulfated form (DHEAS) decline ~80% between the ages of 25 and 75 year. (DHEA), inhibits IL-6 production. As DHEA levels drop with age, its inhibitory influence on IL-6 production becomes attenuated in Ref. 39. The other explanations for this results are same as in Ref. 24–27 for its relation with LBM and ASM and for the other reasons.[30,33]

As Table 2 shows that in sarcopinic subjects, BMI was increasing with age with a highly significant difference between the groups. With the aging process, lean muscle mass is changed into fatty muscle mass by an infiltration of fat into muscle. That is in agreement with a study which found that BIM increase with increasing age in sarcopinic subjects.[40] The reasons are explained in Refs. 35, 36.

Table 3 shows that the mean values of ASM, TLBM is higher in men than women with a highly significant difference between the two values. That finding was in agreement with other research which found the same result.[41] This is because muscle mass is lost at a rate of 4–6% per decade starting at age 40 years in women and age 60 years in men. The greatest decline in both men and women occurs with inactivity, acute illness and after the age of 70 years at which time the mean loss of muscle mass has been measured as 1% per year. At all ages, females appear to be more vulnerable to loss of lean tissue than males.[42] The other explanations for this results same as in Refs. 24, 27.

Table 3 shows that the values of IL-6 and hs-CRP increase with increasing LBM and ASM while α 1ACT decrease with increasing them.[27] The values of IL-6 and hs-CRP in women more than men with a highly significant difference between the mean values of male and female while the mean values of male and female for α 1ACT in men more than women with a significant difference between male and female. No study mentioned the difference between male and female for IL-6 and α 1ACT but for hs-CRP there is a study in agreement with the present study, and it revealed same results.[43] The explanation for increase IL-6 and hs-CRP with age in male and female were in Refs. 30 and 33.

Table 3 Shows that BMI for men indicate that they are obese and women highly over weight this means men have BMI more than women because there is indirect correlation between BMI and ASM, LBM with a highly significant difference between the mean value of male and female. That is in agreement with Ref. 42. The effect of aging and obesity may create an ideal environment for skeletal muscle catabolism, and decline in physical function. Advancing age and obesity contribute to the development of sarcopenic obesity. More reasons mentioned previously in Ref. 35 and 36. Table 4 shows that most effective factors in sarcopenia are ASM and LBM. Scientists defined sarcopenia as reduction in ASM/height^2 and TLBM.[21,22]

Conflict of Interest

None.

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