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Review

How Sarcopenia, Muscle Mass, Strength, and Performance Relate to Non-Alcoholic Fatty Liver Disease: A Systematic Review

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Abstract: Background and aim: Metabolic dysfunction-associated steatotic liver disease (MASLD), previously known as non-alcoholic fatty liver disease (NAFLD) affects up to 25% of the population and causes significant morbidity and mortality. Sarcopenia, the loss of muscle strength and quantity/quality, shares multiple pathways with MASLD, pointing to their possible association. In this systematic review, we aimed to describe the association between low muscle mass and/or strength and/or performance and the presence, development, or severity of MASLD/NAFLD. Methods: A search was performed in PubMed, Web of Science, Scopus, and LILACS, on 16 October 2020, for relevant studies, using a comprehensive search query and following PRISMA guidelines for systematic review conduction and reporting. Results: The search yielded 1042 results, of which 42 full papers and 11 poster abstracts were included. NAFLD was determined by imaging (n = 27), non-invasive tests (n = 13), liver biopsy (n = 11), or transient elastography (n = 2), and its severity by liver biopsy (n = 10), non-invasive tests (n = 7), transient elastography (n = 7), or imaging (n = 4). Muscle mass was, in most cases, adjusted for weight (n = 25), body mass index (BMI; n = 13), or height (n = 10). Most studies showed an association of low muscle mass and low strength with NAFLD presence and severity. However, the association of muscle mass with NAFLD was present when muscle mass was adjusted for weight or BMI but not when it was adjusted for height. A meta-analysis was not possible due to high heterogeneity in the methods of assessment of muscle parameters and NAFLD. Conclusions: This systematic review suggests the need for standardization of methods in this discipline. The issuing of specific guidance regarding sarcopenia in NAFLD would be of importance.

Keywords: non-alcoholic fatty liver disease; sarcopenia; muscle mass; strength; systematic review

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1. Introduction

Accompanying the trend of rising rates of obesity and diabetes, metabolic dysfunction-associated steatotic liver disease (MASLD), previously known as non-alcoholic fatty liver disease (NAFLD), is establishing itself as the most prevalent chronic liver disease. Set to become the leading cause of liver transplantation and hepatocellular carcinoma, it is

estimated to affect 25% of the population [1]. The nomenclature of MASLD was introduced to provide an inclusive and affirmative term for steatosis in the presence of cardiometabolic risk factors, in replacement of the previous concept of NAFLD, based on the exclusion of other causes of steatotic liver disease [2]. In practice, the diagnosis of MASLD relies foremost on the detection of liver steatosis by imaging, most commonly abdominal ultrasound (US), but also computed tomography (CT) and magnetic resonance imaging (MRI); in clinic and research, US is the most frequently used, as it is inexpensive and does not require the use of radiation, even if it does suffer from inter and intra-observer variability [2]. In large population registries, it is acceptable to use hepatic steatosis algorithms, such as the fatty liver index (FLI) and the hepatic steatosis index (HSI) to determine a likelihood of the presence of MASLD [3].

MASLD exists in a spectrum, from simple steatosis to steatohepatitis and cirrhosis; while steatosis is common and "benign", progressive inflammation and fibrosis leads to increase negative liver-related outcomes [4]. Therefore, staging MASLD, particularly regarding the severity of fibrosis, is fundamental in its management [5]. Despite continuous advancements in the area, liver biopsy remains the gold standard for MASLD staging; however, in clinical practice, it has been sufficiently replaced by non-invasive tests [6]. Broadly, these non-invasive tests can be divided into clinical equations, such as the Fibrosis-4 Index (FIB-4) and the NAFLD fibrosis score (NFS), and image-based methods, most commonly vibration-controlled transient elastography (VCTE) [6]. While the latter requires specific equipment and expertise, it not only provides information on fibrosis, via liver stiffness measurement (LSM), but also on the degree of steatosis infiltration, via the controlled attenuated parameter (CAP) [6].

While both the prevalence of and the interest in MASLD keep rising, many questions remain unanswered regarding the pathophysiology of this disease [7]. The most popular theory of causality suggests that multiple different injuries ("hits"), either concomitantly or sequentially, lead to progressive activation of the immune system, lipid peroxidation, inflammation, fibrosis and architectural distortion of the liver [7]. The interplay of different organs via specific cytokines may modulate the onset and progression of MASLD [8].

The term sarcopenia has been informally used to describe loss of mass; however, most recent guidelines bring loss of muscle strength to the forefront, with low muscle quantity or quality confirming the diagnosis [9]. In clinical practice, sarcopenia may present as a "silent disease". While sarcopenia in the presence of low body weight may be easily recognizable, sarcopenia in obesity poses a clinical challenge. It is not enough to determine body weight and body mass, but vital to assess body composition. When possible, low muscle mass is best quantified by dual-energy X-ray absorptiometry (DXA) or by bioelectrical impedance analysis (BIA) [9]. DXA permits an accurate assessment of three-compartment mass, consisting of fat mass, non-bone lean mass, and bone mineral compartment via a whole body scan that uses a safe low dose radiation [10]. Since DXA is ordinarily used for the diagnosis of osteoporosis, it is widely available; it is also non-invasive and affordable, being recommend for use in both day-to-day practice and clinical research [9,10]. BIA leverages the contrast in electric conductivity of different body tissues to automatically calculate, via specific equations, the mass of fat and muscle in the body [11]. It uses no radiation and can be portable, offering additional benefits when compared to other validated methods of body composition assessment [11]. Imaging of a cross-section of muscle by magnetic resonance imaging (MRI) or computed tomography may also be used [11]. However, these methods are frequently more expensive and less accessible, being mostly reserved for the context of clinical research [9]. In the office, simple measurements of calf circumference may provide indirect information on muscle mass, and indexing to waist-circumference may give further insight into body composition and the presence of sarcopenic obesity [12,13]. However, these measurements have been less well researched and are best reserved for situations where the tools previously described are not available [9]. Muscle strength can be determined with the use of implements like the dynamometer to measure handgrip

strength (HGS), or by tests like the chair stand test (also called chair rise test) to indirectly assess lower-body (particularly quadriceps) strength [9].

Physical activity has known beneficial influence on cardiometabolic risk factors and ensuing disease, such as cardiovascular disease and MASLD [14,15]. Sarcopenia was initially considered a geriatric syndrome but is increasingly being studied in relation with several chronic diseases, such as type 2 diabetes mellitus (T2DM) and insulin resistance, chronic pulmonary, heart or kidney diseases, cancer, and rheumatoid arthritis [9,16–19]. The coexistence of obesity and sarcopenia is of particular importance as their synergy can serve to aggravate cardiometabolic risk factors and associated diseases [20].

It has been proposed that MASLD and sarcopenia are connected, as they share common pathways in inflammation, insulin resistance and altered hormonal status (including, but not limited to, sex hormones and vitamin D) [21–27]. Sarcopenia has been found to be more frequent in patients with cirrhosis of NAFLD compared to alcoholic and viral etiology and early reports have shown association of low muscle mass and gamma-glutamyl transferase [28,29]. Patients with end-stage liver disease and sarcopenia have a shorter median survival time and are more likely to develop complications such as gastrointestinal bleeding, sepsis, and encephalopathy [30,31]. Moreover, sarcopenia negatively impacts the outcomes of hepatocellular carcinoma treatment and liver transplantation [30,32]. The skeletal muscle is an endocrine organ that secrets communicating proteins called myokines [33]. These specialized cytokines have been proven to have receptors in several organs, including the liver, which may explain the proposed associations between liver disease and sarcopenia [34]. In patient management, the link between muscle and liver health may serve as a diagnostic clue, while in research, it may open avenues of therapy for MASLD, a disease still waiting specific pharmacological treatment.

The aim of this study was to systematically review the present literature describing the association of sarcopenia, low muscle mass and low muscle function with the presence and severity of MASLD/NAFLD.

2. Methods

2.1. Protocol and Search Strategy

This review was registered in the International prospective register of systematic reviews (PROSPERO), ID CRD42020209051. The Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines were followed [35]. A literature search was performed, on 16 October 2020, in PubMed, Web of Science, Scopus and LILACS with the following query ("Non alcoholic Fatty Liver Disease" [Mesh] OR "NAFLD" OR "Nonalcoholic Fatty Liver Disease" OR (Fatty AND Liver* AND Nonalcoholic) OR (Nonalcoholic AND Steatohepatiti*) OR "NASH") AND (Sarcopenia [Mesh] OR Sarcopen* OR (Loss AND muscle) OR "low muscle mass"); in Scopus and LILACS Mesh, terms were not used. Given the timing of the review and the use of Mesh terms, the term NASLD was preferred instead of the newer MASLD. Included languages were English, Portuguese, Spanish and French, and the search was not restricted by date of publication. Furthermore, the list of references of pertinent articles were examined for relevant studies.

2.2. Study Selection and Eligibility Criteria

Studies were eligible if they were analytical studies and included information about the association between muscle mass/strength/performance and the presence/development/severity of NAFLD. No other restrictions were imposed, such as population group, sex, or age.

2.3. Data Extraction

After excluding duplicates, search results were analyzed by two separate researchers (JR and DMM) independently with the use of a reference manager (EndNote 20, Clarivate, Philadelphia, PA, USA, 2013). In a first stage, records were screened by title and abstract, and, in a second stage, the references' eligibility was assessed by full-text anal-

ysis. Disagreements were resolved by conference. Data were extracted by JR and confirmed by DMM in accordance to previously determined variables: author, year of publication, country of origin, type of study, population, sex and age distribution, low muscle mass/strength/performance definition, method of NAFLD diagnosis/severity assessment, association or risk measure and confounder adjustment. The Newcastle–Ottawa Scale was applied by JR and confirmed by DMM to assess the risk of bias for each included study.

3. Results

Our search yielded 1042 references and the included studies' bibliography review identified 1 additional study. In the end, 42 studies were included in the final selection. Eleven poster abstracts were also reviewed and are discussed separately.

Figure 1 shows the literature search and selection process. Tables 1 and 2 summarize the characteristics and findings of the studies, regarding studies analyzing muscle mass and muscle strength/performance, respectively. Table 3 describes the findings of the poster abstracts. (Supplementary Tables S1–S5 expand on the findings).

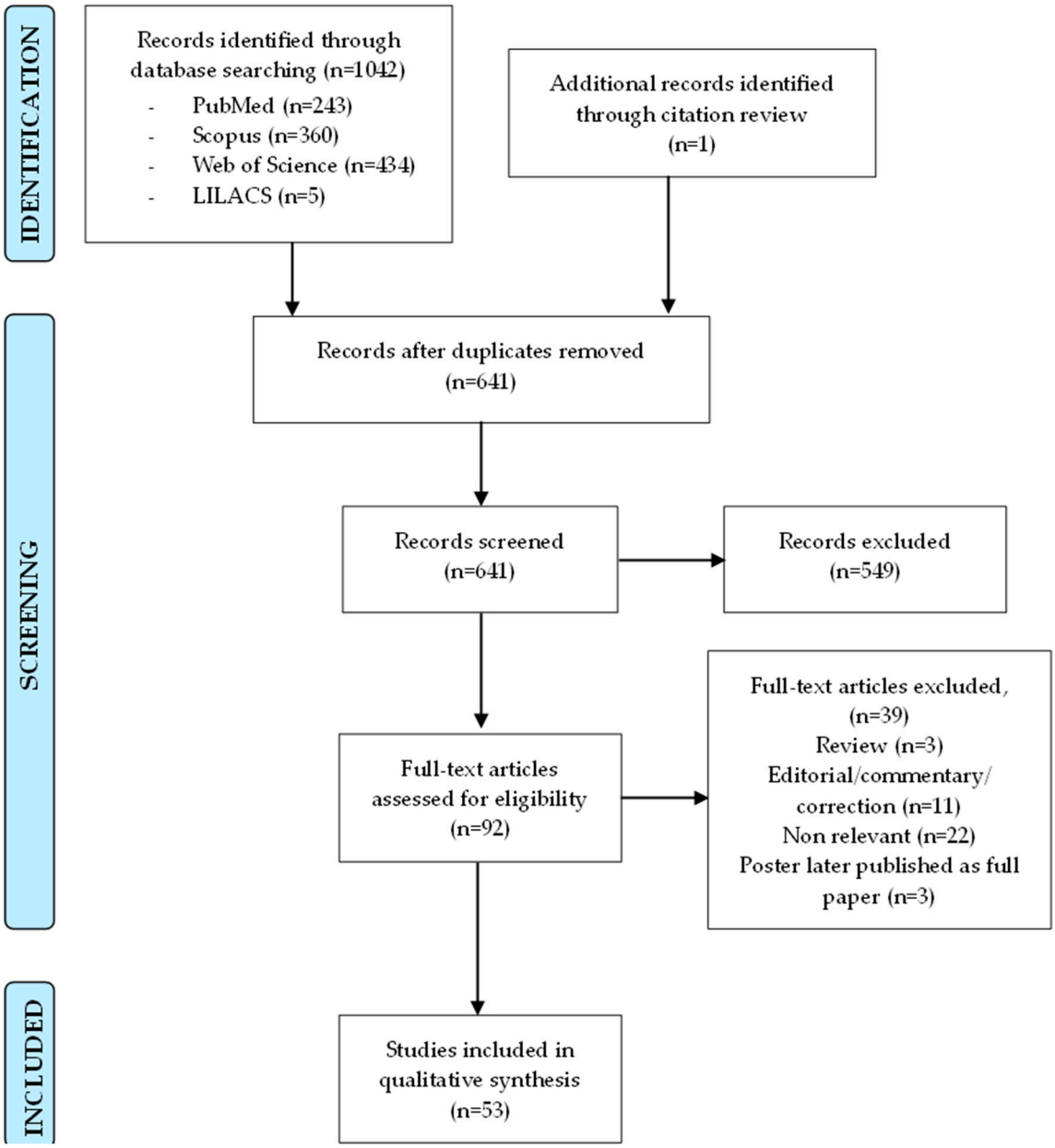

Figure 1. PRISMA 2009 flow diagram for study selection and inclusion.

The results for the Newcastle–Ottawa Scale are reported in Table 4. Most studies included were retrospective analysis of a selected sample within large surveys of the general population, created to assess overall health and nutritional status and not to respond to this specific research question. There is a possible bias of selection as samples within these surveys were frequently chosen according to the availability of the variables of interest. Adjustment for confounders was not performed or not reported for at least some exposure or outcome of interest in 19 (35.8%) papers and posters. There was seldom information on blinding of the researchers.

3.1. Full Papers

The methodology of the studies varied significantly. While liver biopsy is the gold standard for NAFLD diagnosis, it was only performed in five studies (11.9%) [36–40]. Most frequently, authors used imaging techniques [41–65], validated clinical equations [66–76], or controlled attenuation parameter in transient elastography [77]. To assess NAFLD severity, liver biopsy [36–40] and non-invasive test were equally employed [41,51,65,68,71]. However, when considering sample size, non-invasive tests were the most commonly employed.

Twelve studies (28.6%) analyzed data by sex categories [39,44–47,56,60,64,66,73,75,77], while three studies only looked at either a male [58,63] or female [52] population. An association between variables was found in men but not women in three studies [45,47,56] and women but not men in two [44,60]; in the first, measures of muscle were adjusted for weight or BMI, while in the latter, they were adjusted for height or fat mass.

3.2. Low Muscle Mass

Weight-adjusted measures of muscle mass were the most common [36,37,41–53,66–70,77], followed by body mass index (BMI)-adjusted [36,38,41,42,47,54–57,69,71] and height-adjusted measures [38,39,42–44,55,58,59]. When weight- or BMI-adjusted measures were used, there was consistently an inverse association between muscle mass and NAFLD presence or severity. This contrasted with studies with height-adjusted measures, which either showed a positive [42–44,55,59] or no association [39,44,58]. Seven studies used measurements adjusting for fat [38,39,60–62,67,76], and all found an inverse relationship with NAFLD, in at least part of the population analyzed.

Longitudinal studies were rare but suggested that low muscle mass precedes NAFLD and that variations in muscle mass affect the development and remission of this disease [38,39,43,46,62,69].

3.3. Low Muscle Strength

Muscle strength was primarily assessed by handgrip strength (HGS) [44,45,50,52,55,58,59,63,72–75], though elbow flexion strength (EFS) [52,64] and knee extension strength (KES) [45,52,55,64] were also used. Most studies showed an inverse relationship with NAFLD or NAFLD severity.

3.4. Low Physical Performance

Muscle performance was determined by gait speed in three studies and was not found to be associated with the presence [44] or severity [42] of NAFLD when taken in isolation but was when used for the definition of NAFLD [59].

3.5. Sarcopenia

Only three studies [42,44,59] defined sarcopenia as a compound of low muscle mass and low muscle strength and/or performance, with conflicting results probably attributed to different assessment methods.

3.6. Poster Abstracts

The posters found focused on associations between muscle mass and NAFLD and found the same variable-dependent associations as the full studies [78–88]. As with the full

papers, methods to determine the presence and severity of NAFLD and to quantify muscle mass varied greatly, with the use of liver biopsy [78,81,82,85–87], US [79,83], CAP [80], and non-invasive tests [84,88] for the first, and BIA [79,80,82,83,86], DXA [81,88], and CT [78,85–87] for the latter.

Table 1. Summary of characteristics and main results of studies analyzing muscle mass.

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Moon, JS (2013) [67]	FLI	-	BIA	SMM/weight SMM/VFA (continuous and Q4)	Negative correlation, decreased AOR for SMM/VFA
Hong, HC (2014) [48]	CT	-	DXA	SMM/weight (Q1)	Increased AOR
Issa, D (2014) [40]	LB	LB	CT	TPA	Lower TPA in NAFLD, and in NASH-cirrhosis vs. NASH.
Lee, YH (2015) [68]	HSI (>36), CNS (≥40), LFS (≥−0.640)	BARD (≥2), FIB-4 (≥2.67)	DXA	ASM/weight (<32.2% ♂, 25.5% ♀)	Increased AOR
Hashimoto, Y (2015) [77]	CAP (>237.8)	-	DXA	SMM/weight (continuous)	Decreased AOR
Kim, HY (2016) [66]	FLI (≥60)	-	DXA	ASM/weight (continuous)	Lower ASM/weight increased AOR
Lee, YH (2016) [71]	LFS ≥ −0.640 [b]	NFS (Q4), FIB-4 (≥2.67), Forns index (Q4)	DXA	ASM/BMI	Decreased AOR for NFS and FIB-4, NS for Forns index
Poggiogalle E, (2016) [76]	FLI	-	DXA	TrFM/ASM	Positive correlation
Koo, BK (2017) [36]	LB	LB, LSM (NASH and F ≥ 2)	BIA	ASM/weight (<29.0% ♂, <22.9% ♀) ASM/BMI (<0.789 ♂, <0.512 ♀)	Increased AOR
Osaka, T (2017) [53]	US [b]	LSM	BIA	SMM/weight	Inverse correlation. Decreased OR for F ≥ 2
Peng, TC (2017) [42]	US	US	BIA	SMM/weight (<37.0% ♂, <28% ♀) SMM/height^2 (<10.76 ♂, <6.75 ♀)	Increased AOR for SMM/weight and SMM/weight + gait speed, decreased AOR por SMM/height
Petta, S (2017) [37]	LB [b]	LB	BIA	ASM/weight (<37 ♂, <28 ♀)	Increase AOR for grade 3 steatosis, ballooning and fibrosis but not NASH

Table 1. *Cont.*

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Rachakonda, V (2017) [a] [43]	CT	-	DXA, CT	FFM, FFM/height^2, FFM/weight, MMA, MMA/height^2, MMA/weight	Higher FFM, FFM/height^2, MMA, and MMA/height^2. FFM/weight and MMA/weight NS. Resolved vs. persistent NAFLD: NS
Choe, EK (2018) [54]	US	-	CT	SMA/BMI (<8.37 ♂, 7.47 ♀)	Increased AOR
Choe, EK (2018) [65]	US	FIB-4	Physical examination	WCR (T3)	Increased AOR
Kim, G (2018) [a] [69]	HSI (>36.0; resolution of NAFLD <30)	-	BIA	ASM/weight ASM/BMI ΔASM/weight ΔASM/BMI	Decreased AHR for incident NAFLD and increased AHR for NAFLD resolution
Shida, T (2018) [61]	US and elevated ALT [b]	LSM (≥12), CAP (≥260)	BIA	SMM/VFA (Q1)	Increased AOR
Zhai, Y (2018) [59]	US	-	DXA	ASM/height^2	Low muscle mass and low muscle strength and performance (simultaneously) inversely correlated with NAFLD
Alferink, LJM (2019) [44]	US	LSM (≥8.0 kPa)	DXA	ASM/weight, ASM/height^2	In normal weight ♀: decreased AOR for ASM/weight and ASM/height^2 for NAFLD. In ♀decreased AOR for ASM/height^2 for LSM ≥ 8.0 kPa
Chen, VL (2019) [45]	CT	-	DXA	ASM/weight	Negative correlation in ♂, NS in ♀.
Chung, GE (2019) [49]	US	US	BIA	ASM/weight (<29% ♂, <22.9% ♀; and Q1)	Increased AOR
Debroy, P (2019) [58]	CT	-	DXA	ASM/height^2	ASM/height^2 NS
Gan, D (2019) [50]	US	-	DXA	ASM/weight (<28.64% ♂, <24.12% ♀)	Increased AOR
Hsing, J C(2019) [70]	FLI (≥60)	-	DXA	ASM/weight (≥29.1 ♂, ≥25.1 ♀)	Decreased AOR

Table 1. *Cont.*

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Kang, MK (2019) [41]	US [b]	NFS, FIB-4	BIA	ASM/weight (<29 in ♂, <22.9 in ♀) ASM/BMI (<0.789 in ♂, <0.512 in ♀)	Increased AOR
Lee, MJ (2019) [a] [46]	US	-	BIA	ΔASM, ΔASM/weight (T3)	Increased AOR for ΔAMS. Higher loss of ASM/weight in NAFLD
Mizuno, N (2019) [a] [39]	LB [b]	LB, ΔALT (decrease)	BIA	SMM/height^2, SMM/FM	Baseline: lower SMM/FM for NASH than simple steatosis, SMM/height^2 NS, fibrosis NS. Follow-up: SMM/FM with increased AOR for decrease in ALT
Oshida, N (2019) [55]	US	-	BIA	ASM/BMI, ASM/height^2	Lower ASM/BMI and higher ASM/height^2 in NAFLD
Seko, Y (2019) [a] [38]	LB [b]	LB, ΔALT (decrease > 30%)	BIA	ASM/BMI, ASM/FM, ASM/height^2	Baseline: higher ASM/BMI and ASM/FM in F < 2 and NAS < 6, NS for ASMI/height^2. Follow-up: ΔASM/FM increased AOR for ALT decreased, ΔASM/BMI NS
Seo, DH (2019) [47]	US	US	BIA	ASM/weight (<29.0% ♂, <22.9% ♀) ASM/BMI (<0.789 ♂, <0.512 ♀)	Increased AOR in ♂, NS in ♀. Higher proportion of moderate-to-severe NALFD in low ASM/weight
Shida, T [a] [62]	US [b]	LSM, CAP	BIA	ΔSMM/VFA	Decreased CAP in improved SMM/VFA, ΔLSM NS
Su, X (2019) [60]	US	-	BIA	ASM/VFA (T1)	♂increased OR (not AOR), ♀increased AOR
Wijarnpreecha, K (2019) [51]	US	NFS (>0.676 or >0.12 if ≥65 y)	BIA	SMM/weight (<37.0% in ♂, <28.0% in ♀)	Increased AOR
Zhang, Y (2019) [52]	^{1}H MRS	-	DXA	ASM/weight SMM/weight (continuous)	Negative correlation for all

Table 1. *Cont.*

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Hyun Kim, K (2020) [57]	US	CAP, LSM	BIA	ASM/BMI (<0.789 ♂, <0.521 ♀)	Higher prevalence of low ASM/BMI in NAFLD vs. CHB. Higher LSM in low ASM/BMI. CAP NS
Tanaka, M (2020) [56]	US	-	CT	SMA/BMI	Decreased AOR in ♂, NS in ♀

[a] longitudinal, [b] for population definition only; ♂—male, ♀—female, [1]H-MRS—single-voxel proton magnetic resonance spectroscopy, AHR—adjusted hazard ratio, ALT—alanine aminotransferase, AOR—adjusted odds ratio, ASM—appendicular skeletal muscle mass (kg), BIA—bioelectrical impedance analysis, BMI—body mass index (kg/m^2), CAP—controlled attenuation parameter, CHB—chronic hepatitis B, CNS—comprehensive NAFLD score, CT—computed tomography, DXA—dual-energy X-ray absorptiometry, FFM—fat-free mass (kg), FIB-4—fibrosis-4 index, FLI—Fatty Liver Index, FM—fat mass (kg), HSI—Hepatic Steatosis Index, KES—knee extension strength, LB—liver biopsy, LFS—liver fat score, LSM—liver stiffness measurement, MMA—midthigh muscle area (cm^2), NAFLD—non-alcoholic fatty liver disease, NAS—NAFLD activity score, NASH—non-alcoholic steatohepatitis, NFS—NAFLD fibrosis score, NS—non-significant, OR—odds ratio, Q1—lowest quartile, Q4—highest quartile, SD—standard deviation, SMA—skeletal muscle area, SMM—skeletal muscle mass, T1—lowest tercile, T3—highest tercile, TPA—total psoas muscle area (cm^2), TrFM—truncal fat mass (kg), US—ultrasound, VFA—visceral fat area (cm^2), WCR—waist-to-calf ratio.

Table 2. Summary of characteristics and main results of studies analyzing muscle strength and/or performance.

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Strength and/or Performance	Main Results
Peng, TC (2017) [42]	US	US	Gait speed (<0.8 m/s)	Increased AOR for SMM/weight + gait speed, NS AOR (but increased OR) for gait speed
Lee, K (2018) [72]	HSI (>36.0)	-	HGS/BMI (1SD decrease, Q1)	Increased AOR
Zhai, Y (2018) [59]	US	-	HGS (<26 ♂, <18 ♀), Gait speed (<0.8 m/s)	Low muscle mass and low muscle strength and performance (simultaneously) inversely correlated with NAFLD
Alferink, LJM (2019) [44]	US	LSM (≥8.0 kPa)	HGS, Gait speed	In normal weight ♀: lower HGS in NAFLD. NS for gait speed
Chen, VL (2019) [45]	CT	-	HGS, KES	Lower HGS in NAFLD in ♂, NS in ♀. KES NS
Cruz, JF (2019) [64]	US	US	EFS/BMI, KES/BMI	Inverse relationship with NAFLD. Lower EFS/BMI and KES/BMI in grade 3 steatosis

Table 2. *Cont.*

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Strength and/or Performance	Main Results
Debroy, P (2019) [58]	CT	-	HGS/weight (<25th percentile, 25–50th percentile)	Low HGS/weight increased AOR.
Gan, D (2019) [50]	US	-	HGS/weight (<51.26% ♂, <35.38% ♀)	Increased AOR
Kim, B-J (2019) [73]	HSI (per unit increase)	-	HGS (♂<28.9, ♀<16.8)	Increased AOR
Oshida, N (2019) [55]	US	-	HGS, KES	Lower KES (in <60 y) in NAFLD. KES in >60 y and HGS NS
Zhang, Y (2019) [52]	^{1}H MRS	-	HGS/weight, KES/weight, EFS/weight	Negative correlation for all except for EFS/weight (NS)
Hao, L (2020) [63]	US	-	HGS/weight	Decreased AOR
Kang, S (2020) [74]	HSI (>36.0)	-	HGS/BMI (Q1)	Increased AOR
Park, SH (2020) [75]	LFS (>−0.640)	FIB-4, NFS	HGS/BMI (Q4)	Decreased AOR for NAFLD. HGS/BMI quartiles showed inverse relationships with FIB-4 and NFS score quartiles

♂—male, ♀—female, ^{1}H-MRS—single-voxel proton magnetic resonance spectroscopy, AOR—adjusted odds ratio, BMI—body mass index (kg/m^{2}), CT—computed tomography, EFS—elbow flexor strength, FIB-4—fibrosis-4 index, HGS—handgrip strength (kg), HSI—Hepatic Steatosis Index, KES—knee extension strength, LFS—liver fat score, LSM—liver stiffness measurement, NAFLD—non-alcoholic fatty liver disease, NFS—NAFLD fibrosis score, NS—non-significant, OR—odds ratio, Q1—lowest quartile, Q4—highest quartile, SD—standard deviation, SMM—skeletal muscle mass, US—ultrasound.

Table 3. Summary of characteristics and main results of posters.

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Tsien, C (2012) [78]	LB	LB	CT	TPA	Lower TPA in NASH and NASH cirrhosis than controls or steatosis. Fibrosis and lobular inflammation inversely correlated with TPA.
Choi, YJ (2013) [79]	US	-	BIA	SMM/weight (Q1)	Increased AOR
Yamaguchi, A (2015) [80]	CAP [a]	LSM (≥9.0)	BIA	SMM/weight	Lower SMM/weight
Joo, SK (2016) [81]	LB [a]	LB	DXA	ASM/weight	Decreasing ASM/weight with increasing fibrosis
Kim, W (2016) [82]	LB	LB	BIA	ASM/weight (Q1)	Lower ASM/weight in NAFLD. Increased AOR for NASH

Table 3. *Cont.*

First Author (Year)	Diagnosis of NAFLD	Assessment of NAFLD Severity	Assessment of Muscle Mass		Main Results
			Method	Parameter	
Shen, H (2016) [83]	US	-	BIA	SMM/height^2 ($\leq$10.75% ♂, $\leq$6.75 ♀)	Decreased OR, NS AOR
Kallwitz, ER (2017) [84]	FLI, NFLS	NFS	NR	ASM/BMI	Increased AOR for every 1SD decrease
Kapuria, D (2018) [85]	LB [a]	LB	CT	TPA/height^2	Higher TPA/height^2 in advanced steatosis, AOR NS. Fibrosis and NASH NS
Kwanten, WJ (2018) [86]	LB	LB	BIA, CT	Muscle mass [b] / weight (<2SD below reference)	Low muscle mass more prevalent in NAFLD, and in $\geq$F2 vs. <F2 vs. NAFL
Yerragorla, P (2018) [87]	LB	-	CT	SMA	Lower SMA in NAFLD
Gerber, L (2019) [88]	US-FLI	-	DXA	ASM/BMI (<0.789 ♂, <0.512 ♀)	Higher prevalence of low ASM/BMI in NAFLD

[a] for population definition only, [b] not specified if ASM or SMM,; ♂—male, ♀—female, AOR—adjusted odds ratio, ASM—appendicular skeletal muscle mass (kg), BIA— bioelectrical impedance analysis, BMI—body mass index (kg/m^2), CAP—controlled attenuation parameter, CT—computed tomography, DXA—dual-energy X-ray absorptiometry, FLI—Fatty Liver Index, LB—liver biopsy, LSM—liver stiffness measurement, NAFL—non-alcoholic fatty liver, NAFLD—non-alcoholic fatty liver disease, NASH—non-alcoholic steatohepatitis, NFLS—NAFLD Liver Fat Score, NFS—NAFLD fibrosis score, NR—not reported, NS—non-significant, OR—odds ratio, Q1—lowest quartile, SD—standard deviation, SMA—skeletal muscle area, SMM—skeletal muscle mass, TPA—total psoas muscle area (cm^2), US—ultrasound, US-FLI—U.S. Fatty liver index.

Table 4. Modified Newcastle–Ottawa Scale for assessment of bias.

Longitudinal Studies								
	Selection				**Comparability**	**Outcome**		
First Author (Year)	**Representativeness of the Exposed Cohort [a]**	**Selection of the Non-Exposed Cohort [b]**	**Ascertainment of Exposure [c]**	**Demonstration That Outcome of Interest Was Not Present at Start of Study [d]**	**Comparability of Cohorts [e]**	**Assessment of Outcome [f]**	**Was Follow-Up Long Enough for Outcomes to Occur [g]**	**Adequacy of Follow Up of Cohorts [h]**
Rachakonda, V (2017) [43]	*	*	*	*	0	*	*	0
Kim, G (2018) [69]	*	*	*	*	**	*	*	*
Lee, MJ (2019) [46]	*	*	*	*	**	*	*	*
Mizuno, N (2019) [39]	*	*	*	*	**	*	*	*
Seko, Y (2019) [38]	*	*	*	*	**	*	*	*
Shida, T (2019) [62]	*	*	*	*	0	*	*	*
Cross-Sectional Studies								
	Selection				**Comparability**	**Outcome**		
Authors	**Representativeness of the Sample [i]**	**Sample Size [j]**	**Non-Respondents [k]**	**Ascertainment of the Exposure [l]**	**Comparability of Subjects in Different Outcome Groups [m]**	**Assessment of Outcome [n]**		**Statistical Test [o]**
Tsien, C (2012) [78]	0	0	0	**	0	*		0
Choi, YJ (2013) [79]	*	*	0	**	**	*		*
Moon, JS (2013) [67]	*	*	0	**	**	*		*
Hong, HC (2014) [48]	*	*	0	**	**	*		*
Issa, D (2014) [40]	0	0	0	**	0	*		0

Table 4. *Cont.*

Cross-Sectional Studies							
	Selection				Comparability	Outcome	
Authors	Representativeness of the Sample [i]	Sample Size [j]	Non-Respondents [k]	Ascertainment of the Exposure [l]	Comparability of Subjects in Different Outcome Groups [m]	Assessment of Outcome [n]	Statistical Test [o]
Lee, YH (2015) [68]	*	*/0 [P]	0	**	**/0 [P]	*	*
Yamaguchi, A (2015) [80]	0	0	0	**	0	*	0
Hashimoto, Y (2015) [77]	*	*	0	**	**	*	*
Joo, SK (2016) [81]	*	0	0	**	0	*	0
Kim, HY (2016) [66]	*	*	0	**	**	*	*
Kim, W (2016) [82]	*	*	0	**	**/0 [P]	*	*/0 [P]
Lee, YH (2016) [71]	*	*	0	**	**	*	*
Poggiogalle E (2016) [76]	*	*	0	*	**	*	*
Shen, H (2016) [83]	*	0	0	**	0	*	*
Kallwitz, ER (2017) [84]	*	0	0	**	0	*	0
Koo, BK (2017) [36]	*	0	0	**	**	*	*
Osaka, T (2017) [53]	*	0	0	**	**	*	*
Peng, TC (2017) [42]	*	*	0	**	**	*	*
Petta, S (2017) [37]	*	*	0	**	**/* [P]	*	*
Choe, EK (2018a) [54]	*	*	0	**	**	*	*
Choe, EK (2018b) [65]	*	*	0	*	**	*	*
Kapuria, D (2018) [85]	*	0	0	**	**	*	0
Kwanten, WJ (2018) [86]	*	*	0	**	0	*	*
Lee, K (2018) [72]	*	*	0	**	**	*	*
Shida, T (2018) [61]	*	*	0	*	**	*	*
Yerragorla, P (2018) [87]	*	0	0	**	0	*	0
Zhai, Y (2018) [59]	*	*	0	**	**	*	*
Alferink, LJM (2019) [44]	*	*	0	**	**	*	*/0 [P]
Chen, VL (2019) [45]	*	*	0	**	**/0 [P]	*	*/0 [P]
Chung, GE (2019) [49]	*	*	0	**	**	*	*
Cruz, JF (2019) [64]	*	*	0	**	**/0 [P]	*	0
Debroy, P (2019) [58]	*	0	0	**	**/0 [P]	*	*/0 [P]
Gan, D (2019) [50]	*	*	0	**	**	*	*
Gerber, L (2019) [88]	*	*	0	**	**	*	0
Hsing, JC (2019) [70]	*	*	0	**	**	*	*
Kang, MK (2019) [41]	*	*	0	**	**	*	*
Kim, B-J (2019) [73]	*	*	0	**	**	*	*
Oshida, N (2019) [55]	*	0	0	**	0	*	0
Seo, DH (2019) [47]	*	*	0	**	**/0 [P]	*	*
Su, X (2019) [60]	*	*	0	**	**	*	*
Wijarnpreecha, K (2019) [51]	*	*	0	**	**	*	*
Zhang, Y (2019) [52]	*	*	0	**	*	*	*
Hao, L (2020) [63]	*	*	0	**	*	*	*
Hyun Kim, K (2020) [57]	*	*	0	**	0	*	0
Kang, S (2020) [74]	*	0	0	**	**	*	*
Park, SH (2020) [75]	*	*	0	**	**/0 [P]	*	*/0 [P]
Tanaka, M (2020) [56]	*	*	0	**	**	*	*

Point (*) if: [a]—truly or somewhat representative, [b]—drawn from the same population as the exposed cohort, [c]—validated method, [d]—yes, [e]—study controls for age (another point for any additional factor), [f]—validated method, [g]—follow-up was ≥12 months, [h]—lost to follow-up <20% or description provided of those lost, [i]—truly or somewhat representative of target population, [j]—justified and satisfactory or adequately powered to detect a difference (>10 events per variable in multivariable analysis, [k]—response rate ≥ 60% and comparability between respondents and non-respondents characteristics is established, [l]—adequate method (another point if validated/recommended), [m]—controls for age, another point for other factors, [n]—validated method, [o]—clearly described and appropriate, either odds ratio with 95% interval confidence or *p*-value, or correlation coefficient and *p*-value, [P]—depending on outcome/exposure analyzed.

4. Discussion

In this systematic review, we intended to describe the association between muscle mass, strength and performance and the presence and severity of NAFLD (now MASLD). Most studies found an association between low muscle mass and the presence and/or severity of NAFLD when these measures were adjusted for weight or BMI but not for height. There is ongoing debate as to which adjustment is optimal in differing situations [89]. This is of particular importance since these tools were initially designed to assess sarcopenia in the elderly and frail. In the presence of overweight or obesity, the use of weight or BMI indexing is likely more informative of body composition, while also allowing for the inclusion of patients in a larger range of body sizes. Height indexing can mask sarcopenia in overweight individuals and may underplay the interaction of muscle and fat tissues.

Overall, there was an association of low muscle strength and NAFLD, regardless of the method of strength measurement. To the best of our knowledge, our review is the first to analyze this association. In most recent guidelines, strength has been considered the defining feature of sarcopenia [9]. This has come from evidence that low strength is the muscle parameter most often associated with adverse patient outcomes and, as such, the most useful in clinical practice [9]. In fact, sarcopenia can be considered "muscle failure", which may be incorporated in the multisystemic view of NAFLD. Several mechanisms have been proposed for this association. As with liver in NAFLD, in overfed states, the muscle tissue may be infiltrated with fat, a condition called myosteatosis [90]. Myosteatosis leads to mechanical and biochemical dysfunctional muscle, with decreased strength. Lipids have been associated with a reduction in mitochondrial activity in myocytes, fundamentally altering muscle function and metabolism [90]. Intramuscular adipose tissue and lipid peroxidation has been linked to insulin resistance, a risk factor for NAFLD [23,90]. In clinical practice, a simple test of handgrip strength may provide and indirect measure of muscle and overall (including liver) health.

Most studies in this review adjusted for insulin resistance, either as T2DM, levels of fasting glucose, glycated hemoglobin, homeostasis model of insulin resistance, insulin sensitivity index or short insulin tolerance test. Sarcopenia has long been associated both with T2DM and metabolic syndrome [21,22]. As stated above, myosteatosis is a known factor for insulin resistance [23,90].

Vitamin D was only considered in four studies. Levels are lower and deficiency is more common in patients with NAFLD [24]. Vitamin D is also intimately connected to sarcopenia, with receptors being expressed in skeletal muscle cells and mediating genomic and non-genomic effects that translate into reduced muscle performance with low levels of this hormone [25].

Analysis by sex was only performed in a minority of studies; however, most other utilized sex-specific cut-offs for low muscle mass or strength, which limited this bias. Men have a higher percentage of lean muscle mass than women. The age-related decrease in sex hormones, particularly of testosterone, contributes to the loss of muscle mass and function [26]. The prevalence of NAFLD is higher in men that women, and, in women, is higher after menopause [27].

Only 15 of the 53 studies were from non-Asian populations; this is important as extrapolating these results might be biased by several factors. NAFLD is a more recent phenomenon in Asia than in Europe and North America [91]. Body fat distribution is different in Asians: abdominal deposition of fat is more common and total body fat is several percentage points higher for the same BMI compared with other ethnicities [92]. As the etiology of NAFLD is still not completely understood, other genetic and cultural factors might also be at play. Nevertheless, studies in western populations have also shown the same tendency of associations. It is important for clinicians to be aware of these specific ethnic differences in body composition and their implications in practice, to have a critical view of evidence, and to adapt it to their specific population.

Seven studies used the Korea National Health and Nutrition Examination Survey (KNHANES), varying the years of inclusion and variables used, but with overlap of data;

the same is true for the United States equivalent, the National Health and Nutrition Examination Survey (NHANES). This may have created a bias given the similar methodology and duplication of used data. In addition, some of these data were collected over 30 years ago and may not reflect current trends of lifestyle habits, obesity, and body composition. It is important that research in this field is ongoing to increase its applicability to patients today. With globalization, both dietary patterns and physical activity around the world have started to converge to that of Western nations, which may diminish ethnic differences in body composition in the future.

An important limitation of this review was the high heterogeneity of definition of variables of interest, providing only two or less studies for each assessment method and outcome definition with extractable information, and precluding a meta-analysis. This was particularly noticeable in methods of assessment of muscle mass. The methods used were almost always in accordance with recommendations but were widely diverse. While the authors understand that methodology can be limited by local availability, further standardization would allow better comparability between studies and a more robust body of evidence. In clinical practice, maintaining a standardized diagnosis and stratification of patients would aid in the application of research to individuals and would allow more accurate follow-up. While language restrictions were established in the selection of studies, none were excluded for this reason only.

Our review is not the first to address the relationship between muscle mass and NAFLD. However, we were alone in including poster abstracts, which composed 21% of studies included. The exclusion of such a significant number of studies may pose a selection bias. Overall, studies that represented posters abstracts tended to be smaller and to use biopsy for NAFLD diagnosis and staging, as opposed to the studies more frequently published which were large cohort studies that relied on equations or other non-invasive methods.

While there are a significant number of studies that point to an association of low muscle mass and low muscle strength with the presence and severity of NAFLD, the high heterogeneity of methods of assessment of these variables is a hindrance for the progression of this field. More recent studies outside of this review have shown similar associations of muscle parameters and NAFLD/MASLD [93–97]. Unfortunately, heterogeneity of methodology persists, even as more authors follow guidelines on the topic.

Beyond the observation of associations between sarcopenia and MASLD, the pathophysiological bases and consequences of these associations need to be examined to determine their implication in clinical practice. The authors suggest that specific guidance be issued regarding sarcopenia in MASLD.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/sci6040059/s1, Table S1: Characteristics and results of studies assessing the association of muscle mass and the presence of NAFLD; Table S2: Characteristics and results of studies assessing the association of muscle strength and/or performance and the presence of NAFLD; Table S3: Characteristics and results of studies assessing the association of muscle mass and severity of NAFLD (ordered by variable of assesment of muscle mass, and sample size); Table S4: Characteristics and results of studies assessing the association of muscle strength and/or performance and severity of NAFLD; Table S5: Characteristics and results of studies assessing the association of sarcopenia and presence or severity of NAFLD.

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