Clinical Nutrition 55 (2025) 208–218

Contents lists available at ScienceDirect

Clinical Nutrition

journal homepage: http://www.elsevier.com/locate/clnu

ESPEN Endorsed Recommendation

Sarcopenic diabetes is an under-recognized and unmet clinical priority. A call for action from the European Society for Clinical Nutrition and Metabolism and the Diabetes Nutrition Study Group☆

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Abbreviations: European Society for Clinical Nutrition and Metabolism, (ESPEN); Diabetes Nutrition Study Group, (DNSG); Type 1 Diabetes Mellitus, (T1DM); Type 2 Diabetes Mellitus, (T2DM); Adiposity-Based Chronic Disease, (ABCD); Incretin-Mimetic Drugs, (IMDs); Global Leadership Initiative on Malnutrition, (GLIM); Sarcopenic Obesity Global Leadership Initiative, (SOGLI); Global Leadership Initiative on Sarcopenia, (GLIS); European Working Group on Sarcopenia in Older People, (EWGSOP2); European Association for the Study of Obesity, (EASO); Adjusted Body Weight, (ABW).

☆ MDB-P and SK are Associate Editors of Clinical Nutrition. This paper was accepted after the normal peer review process that they were not part of.

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https://doi.org/10.1016/j.clnu.2025.10.007

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ARTICLE INFO

Article history:
Received 24 July 2025
Accepted 21 October 2025

Keywords:
Sarcopenia
Diabetes
Skeletal muscle
Obesity
Complications

SUMMARY

Diabetes mellitus is a systemic chronic disease with growing prevalence and potential multiorgan complications leading to clinical, social, and economic burdens. Nutritional and metabolic derangements are important components of both type 1 (T1DM) and type 2 diabetes (T2DM), but assessment of nutritional state, body composition and muscle function is commonly neglected. Likely reasons include high prevalence of overweight, obesity, or excess visceral fat in highly-prevalent T2DM, potentially diverting attention from undernutrition risk. Diabetes and adiposity are mechanistically related to sarcopenia, defined as reduction of skeletal muscle strength and mass, through complex muscle-catabolic derangements, conferring additional risk for negative outcomes. Awareness of diabetes-induced muscle abnormalities remains low among healthcare professionals, patients and policymakers, contributing to research, knowledge and practice gaps. Lifestyle recommendations and treatments centered on nutritional care and physical activity to preserve and improve muscle mass and function remain poorly implemented. The European Society for Clinical Nutrition and Metabolism (ESPEN) and the Diabetes Nutrition Study Group (DNSG), reference group for the European Association for the Study of Diabetes, recognize sarcopenic diabetes as a distinct clinical condition and priority for research and education, and call for action to enhance awareness, stimulate research and promote consensus on sarcopenic diabetes diagnostic criteria, prevention and management.

1. Introduction

The prevalence and incidence of both type 1 (T1DM) and type 2 diabetes mellitus (T2DM) have grown worldwide for decades [1,2]. According to the 2025 Diabetes Atlas from the International Diabetes Federation, 589 million people worldwide were diagnosed with diabetes, and approximately 40 % more were believed to be undiagnosed [3]. Although T1DM prevalence is also growing, T2DM prevalence accounts for ≈90 % of diabetes cases worldwide, due to its causal association with two major global risk factors: the rapidly growing prevalence of overweight and obesity, and enhanced life expectancy with growing aging population [4–7]. Indeed, overweight, obesity and other aspects of abnormal adiposity (namely, increased visceral-ectopic fat) affect ≈90 % of people living with T2DM [4–9]. According to recent pathophysiological models of dysglycemia-based chronic disease, the main driver of T1DM is autoimmunity and those of T2DM are abnormal adiposity, inflammation, insulin resistance, and β-cell dysfunction [10]. Due to dysmetabolic and inflammatory processes, not only do cardiovascular complications arise [11], but there may be subsequent changes in body composition [12].

ESPEN and DNSG recognize that the emerging association of diabetes and altered body composition, with particular regard to skeletal muscle loss with loss of muscle function, is a relevant but under-recognized clinical entity. This may be exacerbated by the presence of abnormal adiposity, but may also occur independently of obesity as a result of diabetes- and hyperglycemia-associated metabolic derangements. We will therefore describe here the mechanisms leading to loss of muscle mass and function in diabetes, with particular attention to T2DM, where metabolic derangements associated with abnormal adiposity and dysglycemia directly affect maintenance of body composition and nutritional homeostasis. Concomitant diabetes and sarcopenia have been indeed reported in various clinical settings, clearly showing the existence of sarcopenic diabetes. Most importantly, diabetes with concomitant low muscle mass and function are clearly associated with negative clinical outcomes, and we will describe estimated prevalence and clinical impact. However, also due to low awareness, definition and diagnosis of sarcopenic diabetes in clinical research has lacked consensus-based rigorousness, and its identification in clinical practice has been limited at best, with lack of effective treatment options. The paper will therefore provide a call to action on sarcopenic diabetes, including strategies to enhance awareness, detection, and treatment.

2. Mechanisms (Fig. 1)

The pathophysiological mechanisms linking sarcopenia and diabetes are multifactorial and synergistic. They involve changes in lifestyle, in body composition, metabolic alterations, inflammation, and iatrogenesis (i.e. medications).

2.1. Lifestyle

Unhealthy diet and sedentary lifestyle with low physical activity are primary drivers of positive energy balance leading to abnormal adiposity, fat accumulation and T2DM, while also leading to poor glycemic control in T1DM. An unhealthy diet, rich in energy, saturated fatty acids (e.g. palmitic acid) and high glycemic index foods, and poor in unsaturated fat, antioxidants and fiber, may also have a direct negative impact on skeletal muscle metabolism by promoting inflammation and oxidative stress, as well as disrupting mitochondrial function [13–16]. The amount and quality of dietary protein also play a role when intake is below recommended requirements [14–18]. Sedentary lifestyle per se is also associated with positive energy balance and skeletal muscle loss and dysfunction [19,20].

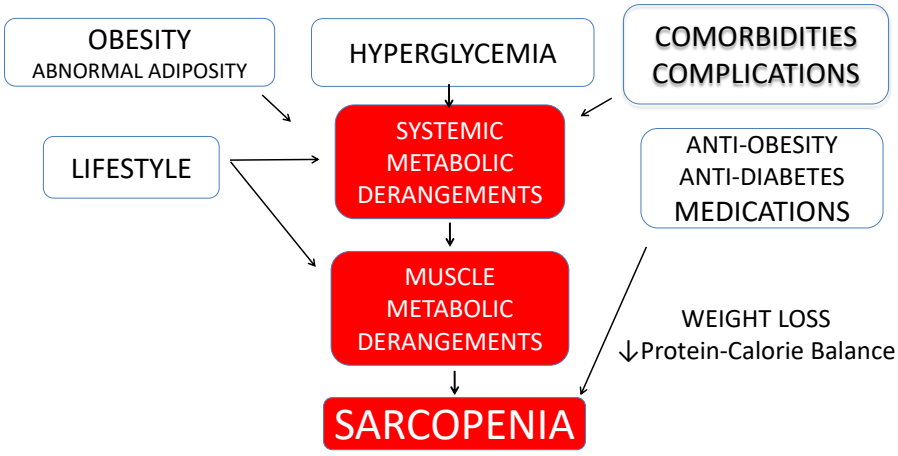

Fig. 1. Summary of mechanisms potentially contributing to sarcopenia in diabetes.

2.2. Excess and abnormal adiposity

Excess adipose tissue (with body mass indices in the overweight/obese ranges), abnormal fat distribution (with excess visceral and lean tissue deposition), and/or abnormal adipokine signatures as part of adiposity-based chronic disease (ABCD) are common in diabetes, particularly in T2DM and increasingly in T1DM [21–23]. In the presence of insufficient adipose tissue expandability and inadequate fat storage capacity, excess fat may cause adipocyte hypertrophy, tissue hypo-perfusion and mechanical/oxidative cellular stress; this is associated with pro-inflammatory adipokine patterns that can trigger systemic metabolic derangements [24]. Low adipocyte fat storage capacity may directly contribute to excess fat deposition in the visceral abdominal compartment [25] and in lean tissues such as liver and skeletal muscle, with further metabolic damage [9,24]. Thus, ABCD directly contributes to metabolic syndrome, T2DM, cardiovascular complications [9,21–23], and other physical and mental comorbidities [26]. Notably, these events may increase systemic inflammation and insulin resistance, impelling metabolic vicious cycles with further negative impact on muscle tissue.

2.3. Inflammation and insulin resistance

Unhealthy lifestyle and ABCD may lead to systemic low-grade inflammation, which is a major determinant of insulin resistance [9]. Both inflammation and insulin resistance contribute to β-cell defects and the onset of hyperglycemia, but they also negatively affect skeletal muscle protein turnover [22,27–29], particularly the mitochondrial fraction [30–32]. Muscle mitochondrial dynamics are indeed negatively modulated in obesity and diabetes, which may directly reduce energy production and ATP availability [33,34]. These combined alterations may negatively affect skeletal muscle mass, strength and endurance [35]. Additional obesity- and diabetes-associated muscle-catabolic changes include impaired regenerative capacity via reduced muscle progenitor cells [36,37], endothelial reticulum stress [38] and impaired muscle capillarization [39].

2.4. Hyperglycemia

The onset of hyperglycemia may independently enhance oxidative stress, inflammation, and muscle catabolic changes. Glycation of muscle proteins and accumulation of advanced glycation end products (AGE) may directly promote skeletal muscle protein loss and dysfunction [40], with altered myosin structure demonstrated in pre-clinical models [41]. Clinical studies have demonstrated associations among hyperglycemia, muscle catabolism, and low skeletal muscle mass and strength [42–44], and these findings are further confirmed in the presence of comorbidities which may present with distinct metabolic pofiles. In patients with heart failure, T2DM was associated with muscle mitochondrial dysfunction, fiber atrophy, and reduced capillary perfusion [45]. Isotopic turnover studies have shown a direct negative impact of hyperglycemia on whole-body protein breakdown in patients with diabetes and chronic kidney disease undergoing hemodialysis [46]. Consistent with the hypothesis that hyperglycemia and AGE are involved in sarcopenia and frailty [12], AGE levels were also directly associated with a frailty diagnosis in patients with T2DM receiving hemodialysis [47]. In patients with cancer, tight glucose control improved hyperglycemia-associated muscle catabolism [48]. Taken together, the above observations support a causal association between hyperglycemia and muscle derangements defining sarcopenia, with potential aggravation by comorbidities.

2.5. Diabetic complications

Diabetes-associated complications and comorbidities (e.g., diabetic cardiovascular disease, nephropathy, neuropathy, diabetic foot disease, and retinopathy) are also associated with higher prevalence of sarcopenia [49–54]. Neuropathy may directly contribute to skeletal muscle atrophy through altered neuromuscular junction and secondary reduction of physical activity [55–57]. Clinical associations have been accordingly reported between peripheral neuropathy and low mass of affected muscle groups [55–57], with longitudinal observations that directly support a causal link [56]. A common pathogenetic background for atherosclerosis and sarcopenic diabetes includes inflammation and insulin resistance, with confirmed clinical associations [49,50]. According to a systematic review, a strong association exists between peripheral vascular disease with lower limb ischemia and skeletal muscle metabolism and dysfunction [58].

2.6. Antiobesity and antidiabetic medications

People living with diabetes and ABCD are encouraged to undergo lifestyle modifications with dietetic intervention and physical exercise. When indicated, metabolic/bariatric procedures may be performed. In the last 15 years, incretin-mimetic drugs (IMDs)

have been introduced with favorable effects on body weight, glycemic control and overall cardiometabolic risk [59]. Second-generation IMDs such as semaglutide and tirzepatide have been approved for obesity treatment in may Countries, showing unprecedented effectiveness with weight loss above 15 % and 20 %, respectively, in people without diabetes [60,61]. Parallel lean mass loss is inevitable in the context of any weight loss treatment, and approximately one-fourth to one-third of lost weight appears to be attributable to lean tissue [62,63]. Interestingly, IMDs-induced weight loss in people with T2DM is reported to be less profound than in those without T2DM by approximately 50 %, for yet unclear reasons [64]. The growing use of highly effective IMDs suggests that, despite unprecedented potential for clinical benefits, there will be higher potential risk for sarcopenia. This implies that treatment with IMDs and potentially other anti-obesity medications requires keen attention to dietetic interventions including adequate dietary protein intake, and enhanced physical activity including strength training to minimize muscle loss [60,61]. These combined interventions will be particularly important in older and frail patients, and in those with comorbidities, where assessment of sarcopenia should be performed prior to weight loss, and the impact of treatment on body composition and muscle function should be regularly monitored. Research is also needed in order to evaluate the effect of IMDs on lean body mass, body composition, and muscle strength in real-world scenarios where medications may be discontinued leading to weight oscillations [65].

3. Epidemiology and clinical impact

Studies on the prevalence and clinical impact of sarcopenia in T2DM remain relatively scarce and suffer from heterogeneity in sarcopenic diabetes diagnostic criteria and cohorts studied. Available reports using heterogeneous definitions support a high prevalence of 28 % in T2DM, with 60 % increased risk compared to people without diabetes, reported in a systematic review of 20 studies with over 54.000 participants [66]. In this metanalysis, the presence of diabetic complications in 1800 patients was associated with a more than doubled risk of sarcopenia [66]. Another metanalysis investigating sarcopenic obesity [67] has reported a similar prevalence of 27 % in diabetes groups, with higher risk of adverse outcomes and several complications. A subsequent metanalysis has confirmed a comparable higher risk of sarcopenia in persons with T2DM (odds ratio of 1.55), mainly driven by reduced skeletal muscle strength [68]. In this paper, prevalence in individual studies was reported, confirming high variability ranging from 5 to 50 % [68]. Worse glycemic control has been also directly associated with an increased risk of sarcopenia [44]. The above figures support the concept that sarcopenia is a major, albeit neglected, diabetes complication [69]. Clinical, sex-related, diabetes-related and potentially regional or ethnic factors [70] may contribute to reported large variability, which is however also likely due, at least in part, to highly heterogeneous diagnostic approaches. Importantly, several studies have inaccurately defined sarcopenia as isolated low skeletal muscle mass, which is not consistent with currently accepted definitions of sarcopenia and sarcopenic obesity, that also include low muscle function [71–73].

3.1. Negative prognostic impact and bidirectional relationships

Similar to other clinical settings, sarcopenia is associated with poor clinical outcomes in people with diabetes. Sarcopenia in diabetes predicted longer hospital stays and 1-year mortality in older patients [74], and it was associated with higher overall mortality in the outpatient diabetes setting [75]. Sarcopenic diabetes may lead to frailty, disabilities and loss of autonomy, a still under-appreciated but increasingly recognized cluster of clinical features [76–78]. Sarcopenia has been also associated with, and may worsen prognosis in major chronic organ failures in diabetes, including heart failure, fibrotic liver disease and chronic obstructive pulmonary disease [79,80]. Finally, both diabetes and sarcopenia have been related to an increased risk of osteoporosis and fractures especially in ageing populations. The coexistence of osteoporosis and sarcopenia has been recently considered as a syndrome termed osteosarcopenia, where abnormal adiposity and inflammation may play an important etiologic role [81]. It should be also pointed out that, while diabetes is a strong risk factor for sarcopenia, sarcopenia is also a risk factor for diabetes and poor glycemic control, establishing a potential vicious cycle [82,83]. Low skeletal muscle mass is associated with reduced whole-body glucose utilization by muscle tissue. Bidirectional relationships also appear to exist between sarcopenia and major diabetic comorbidities and complications, including but not limited to cardiovascular disease and nephropathy [84–86]. However, mechanisms linking sarcopenia and complications, as well as their mutual mechanistic interactions, remain unclear. Based on the above observations, prevention and treatment of sarcopenia in affected or at-risk individuals has the potential to reduce the incidence of diabetes and potentially its complications.

3.2. Sarcopenic diabetes, malnutrition and nutritional state

In recent years, low skeletal muscle mass has been increasingly recognized as a major diagnostic criterion for malnutrition, thereby underscoring the nutritional component of sarcopenia [87,88]. It should therefore not be surprising that malnutrition is also emerging as a relevant comorbidity in diabetes [89]. In a recent guidance paper, the Global Leadership Initiative on Malnutrition (GLIM) consortium has indicated T2DM as a potential state of chronic low-grade inflammation that could also fulfill an etiologic malnutrition diagnostic criterion [90]. Malnutrition and sarcopenia need to be considered separately, but it appears clinically reasonable to check patients with diabetes and malnutrition for sarcopenia, whereas patients with diabetes and sarcopenia should be thoroughly assessed for malnutrition and nutritional deficiencies. Malnutrition diagnosis is indeed increasingly common in diabetes both in hospitals and the community [91–93], and it is associated with poor outcomes and high resources utilization [91–93], making it also a major target for clinical management and research.

3.3. We need a call to action

The European Society for Clinical Nutrition and Metabolism (ESPEN) and the Diabetes Nutrition Study Group (DNSG) reference groups for the European Association for the Study of Diabetes, consider sarcopenic diabetes to be a major underappreciated phenotype and a diabetes complication-comorbidity. Therefore, ESPEN and DNSG issue this call for action to all relevant stakeholders (healthcare professionals, scientists, professional scientific societies, policymakers, industry, and patients) to promote sarcopenic diabetes awareness, diagnosis and treatment. While very limited high-quality evidence on optimal identification and treatment of patients with diabetes and sarcopenia is an inherent limitation, we also seek to provide a consensus approach to sarcopenic diabetes prevention, diagnosis and treatment strategies, based on available evidence and consensus in management of sarcopenia and diabetes, with or without obesity.

4. Clinical assessment

4.1. Clinical suspicion

ESPEN and DNSG maintain that prevention, limitation or treatment of sarcopenia should be a major clinical goal in patients with diabetes and the following conditions [73].

- signs or symptoms compatible with low skeletal muscle function and mass;
- at-risk conditions for low skeletal muscle function and mass such as older age, sedentary lifestyle, poor glycemic control, diabetic complications, comorbidities, and recent acute catabolic events;
- ongoing weight-management programs including intensive lifestyle changes, medications such as IMDs and-or metabolic/bariatric procedures.

4.2. Diagnosis

Diagnosis of sarcopenic diabetes has been elusive due to lack of consensus in both research and clinical fields [88], leading to high variability and difficult comparison of limited available findings. Algorithms are available for the diagnosis of sarcopenia regardless of adiposity status, generally focusing on geriatric populations [71], and the global leadership initiative on sarcopenia (GLIS) has recently introduced an international consensus-based conceptual definition [72]. In the context of T2DM, the high prevalence of obesity, overweight, and other forms of ABCD supports the use of a diagnostic approach based on anthropometric criteria. In 2022, a consensus-based diagnostic algorithm for sarcopenic obesity diagnosis was established under the auspices of ESPEN and the European Association for the Study of Obesity (EASO) [73]. The group explored dissemination and implementation of the algorithm as well as specific initiatives oriented to open research and clinical questions, in the framework of the Sarcopenic Obesity Global Leadership Initiative (SOGLI) [94]. The implementation of the SOGLI algorithm has demonstrated its effectiveness in identifying individuals with sarcopenic obesity and predicting adverse clinical outcomes [95]. In patient subgroups such as older adults living in the community, sarcopenic obesity prevalence ranged between 7 and 10 % [95,96].

ESPEN and the DNSG therefore support the following with respect to sarcopenic diabetes.

- The SOGLI algorithm should be used for a sarcopenic diabetes diagnosis in T2DM, when body mass index (BMI) or waist circumference meet ethnicity-specific thresholds [73].
- The 2018 European Working Group on Sarcopenia in Older People (EWGSOP2) algorithm should be used for a sarcopenic diabetes diagnosis in patients without obesity or excess visceral fat according to ethnicity- and sex-specific BMI and WC cut-offs [71].

ESPEN and DNSG also envision collaborations to develop a diabetes-oriented strategy to optimize sarcopenic diabetes identification. This could potentially involve inclusion of diabetes-specific parameters in relevant algorithms, such as those directing glycemic control and management of diabetic complications [97]. Modified parameters could allow stratification of clinical risk, and identification of higher risk for negative outcomes, thereby potentially allowing for further optimization of sarcopenic diabetes prevention and treatment strategies.

4.3. Methods for muscle and body composition assessment

Handgrip strength by dynamometer or sit-to-stand tests are recommended for muscle functional assessment [73]. The SOGLI and EWGSOP2 recommend dual-energy X-ray absorptiometry as a clinical gold standard for body composition. Well standardized bioimpedance analysis is also supported as a simpler approach implementable at patient bedside or in the outpatient setting [71,73]. Anthropometry-based surrogate measures for muscle mass such as calf circumference have been deemed acceptable for a malnutrition diagnosis [88] but they may be difficult to implement in obesity. BMI-normalized calf circumference has been recently proposed as muscle surrogate, but its utilization requires further validation [98].

4.4. Nutritional state

In the presence of a sarcopenic diabetes diagnosis, a complete nutritional assessment including GLIM diagnosis of malnutrition is recommended [73,87]. Clinical assessment in patients with sarcopenic diabetes should include functional status, particularly in the presence of complications and comorbidities. Disabilities, loss of autonomy, and frailty should be investigated under at-risk conditions including sarcopenic diabetes and malnutrition, not only in older adults but in all at-risk patients.

5. Prevention and treatment (Fig. 2)

5.1. Healthy dietary patterns

Healthy dietary patterns play a key role in preserving nutritional and metabolic homoestasis through regulation of body weight, composition and inflammation. A strong body of evidence has demonstrated the positive impact on health of dietary patterns based on low-glycemic index carbohydrates, higher content of plant-based unsaturated fat, adequate fiber and whole-grain, and adequate micronutrients including anti-oxidants and anti-inflammatory components [99–102]. The Mediterranean diet has been highly investigated as a traditional pattern with beneficial impact on health [103–105]. Other dietary patterns (e.g. traditional Nordic or vegetarian) also demonstrated cardiometabolic benefits in randomized controlled trials and large prospective cohort studies [103,106]. Of note, such patterns should be considered in the context of durable lifestyle components, such as conviviality, eating time and sleep patterns [100,101]. The primary cardiovascular prevention PREDIMED trial [107] has demonstrated the beneficial effect of a Mediterranean diet in the prevention of cardiovascular disease, T2DM and other secondary outcomes, compared to a low-fat diet. Cardiovascular protection from a Mediterranean diet has also been reported in the CORDIOPREV study for secondary prevention of cardiovascular disease [108]. An interim analysis of the PREDIMED-Plus study, conducted in older patients with metabolic syndrome and approximately 25 % prevalence of T2DM reported an improvement in body composition with lower body fat and preserved lean mass after three years [109]. Intervention in the PREDIMED-Plus study consisted in an intensive energy-reduced Mediterranean diet and physical activity lifestyle program, compared to the control group only receiving general recommendations on Mediterranean dietary pattern adherence [109]. In addition to these healthy traditional dietary patterns, evidence-based, plant-forward therapeutic dietary patterns are recommended to meet therapeutic goals. These diets include the Portfolio diet (a dietary portfolio of cholesterol

lowering foods including nuts-seeds, plant protein, viscous soluble fibre, plant sterols and high monounsaturated fat plant oils) and the Dietary Approaches to Stop Hypertension (DASH) diet (blood pressure lowering dietary pattern emphasizing fruit, vegetables, fat-free or low-fat dairy, whole grains, nuts and legumes) [110–112]. These result in clinically meaningful reductions in Low-Density Lipoprotein-cholesterol and blood pressure, respectively, as well as improvements in other intermediate cardiometabolic risk factors [112–114], associated with lower incident diabetes and cardiovascular disease in large prospective cohort studies [112–114]. Overall, the above general provisions are notably aligned with recommendations from the DNSG for dietary management of diabetes [103] and they appear to have potential to improve muscle mass, muscle function, and body composition.

5.2. Protein

Provision of adequate high-quality dietary protein to preserve or enhance muscle protein anabolism is supported by sarcopenia consensus documents and guidelines [71,73,115]. This is considered to be at least 1 g/kg actual body weight per day for persons without obesity. However, only limited high-quality evidence is available, with little evidence for persons with diabetes and-or overweight/obesity [15]. Longitudinal observational studies have shown associations between protein intake > 1 g/kg·day and slower three-year age-related loss of skeletal muscle mass [116]. Longer follow-up in observational studies showed that these findings may be sex- and ethnicity specific, with only partial associations between protein intake and long-term changes in muscle mass and strength [116–119]. Some intervention studies have shown a beneficial impact of higher protein intake in people with sarcopenia [15]. Expert consensus documents recommend protein intake 1–1.2 g/kg·day or higher for people 70 years or older (1,2 to 1,5 g/kg·day in recent Nordic Nutrition recommendations) [120] to preserve skeletal muscle mass in healthy older adults, with adequate strength training [17,18]. Higher protein intakes are commonly recommended to preserve or recover lean and skeletal muscle mass for patients with malnutrition, and-or acute hypercatabolic conditions such as critical illness [121–125]. For people with sarcopenic obesity, consensus and position papers (e.g. the ones from ESPEN and EASO) have supported protein intakes above 1 g/kg adjusted body weight (ABW)·day (ABW = ideal body weight + 25 % excess body weight) [73,126]. Importantly, the DNSG guidelines recommend for weight-stable, normal-weight people with diabetes a protein intake between 10 and 20 % total energy under the age of 65 years with an estimated glomerular filtration rate >60 ml/min per 1.73 m^2 [103]. Higher intakes (15–20 % total energy) are recommended for those aged 70 years or older [103]. Considering caloric intakes of 1500–2000 cal/day for normal weight individuals, an equivalent of up to 75–100 g protein are envisioned, which may indeed reach 1–1.2 g/kg body weight·day. In the presence of diabetic nephropathy beyond stage 3a, protein intake should be limited to the lower recommended range of 10–15 % (<1 g/kg·day).

In conclusion, despite the lack of high-quality intervention studies, maintaining a protein intake above 1 g/kg·day (>1 g/kg ABW·day for persons with overweight or obesity) appears to be a reasonable recommendation for those with, or at risk for sarcopenic diabetes. If such intake is not reached through regular diet, addition of protein in powder or liquid form in foods and beverages or use of oral energy and protein supplementations or medical nutrition treatments should be considered.

5.3. Non-protein calories

The quality of dietary carbohydrates and fat is highly relevant for people with diabetes who often present with overweight-obesity and develop systemic inflammation and insulin resistance. We recommend adherence to the DNSG guidelines [103] with adequate amounts of low-glycemic index carbohydrates and plant-based unsaturated fat, as well as an adequate amount of fiber (35 g/day). Different healthy traditional (e.g. Mediterranean, Nordic, and vegetarian) and therapeutic (e.g. DASH and Portfolio) dietary patterns as well as transitional (flexitarian) patterns are also recommended to satisfy energy and nutritional requirements and meet therapeutic goals of diabetes management [104,105,110–114]. Energy intake and physical activity should be balanced and appropriate to individual requirements to ensure long-term maintenance of a healthy body weight [103]. On the other hand, a large number of people with diabetes who have overweight-obesity, particularly those with T2DM, should aim for weight loss by adopting an intensive lifestyle intervention involving an energy-reduced diet and increased physical activity [103].

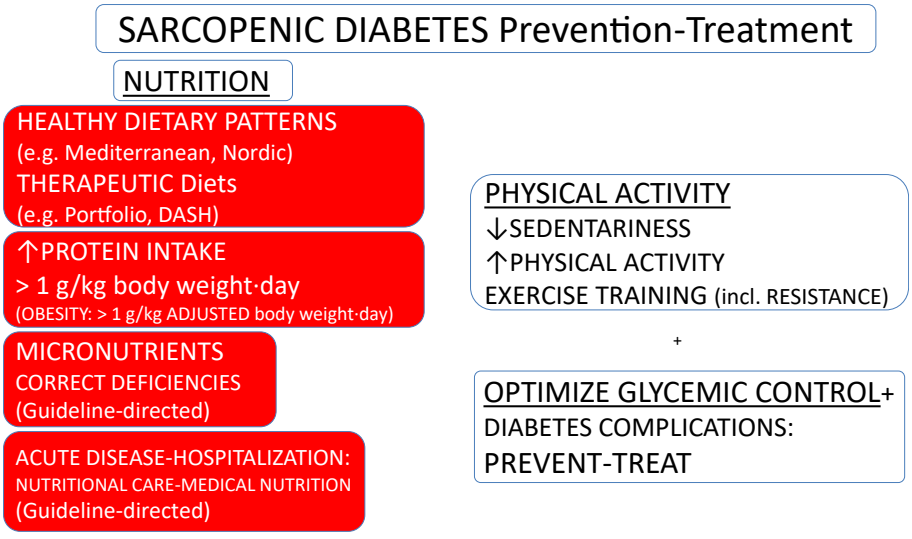

Fig. 2. Summary of proposed strategies for prevention and treatment of sarcopenia in diabetes.

5.4. Micronutrients

Provision of micronutrients should be guideline-directed and according to published daily-recommended intake. Patients with obesity and-or diabetes are at higher risk for micronutrient deficiencies, many of which contribute to the risk of sarcopenia [103,127]. Vitamin D deficiency is the most commonly described, and vitamin D supplements should only be administered in case of proven low circulating levels [127]. Nevertheless, potential adjustments and higher doses for vitamin D supplementation for people with diabetes and-or obesity should be investigated.

5.5. Physical activity

Appropriate and safe levels of physical activity and structured exercise programs should be routinely implemented in patients with diabetes, particularly in the presence of overweight-obesity [103,128]. In the presence of sarcopenia, strength-resistance training has been recommended as the first-line treatment in older adults [115,129]. In people with frailty and disabilities, physical rehabilitation should be individually implemented whenever possible according to patient status [115,129]. Reducing sedentariness should also be targeted independently of enhanced physical activity and exercise [130].

6. Management of obesity in patients with sarcopenic diabetes

Weight loss should be sought in persons with diabetes and overweight/obesity [103,128]. Unfortunately, it is well established that obesity management strategies (i.e. dietary interventions, physical activity, medications, and metabolic/bariatric procedures) lead to parallel reductions in both fat and lean body mass [60,61,131,132]. Typically, the loss of body fat is substantially larger than the loss of muscle (by a 2:1 to 3:1 ratio) across different treatment modalities, including semaglutide and tirzepatide [60,61]. This commonly results in improved body composition and enhanced percent lean body mass, improving the sarcopenic obesity phenotype when muscle mass is normalized by body weight [73]. On the other hand, in patients at higher risk for muscle mass loss, such as older adults or those with more severe pro-inflammatory and catabolic comorbidities, changes in body composition could be more unfavorable, even leading to new onset or worsening of sarcopenic obesity and higher risk of weight regain. Treatment with semaglutide and tirzepatide leads to unprecedented >15–20 % weight loss, with potential for significant absolute amounts of lost muscle mass, although their impact on total weight in persons with T2DM is reported to be less profound [64]. The impact of these newer agents on body composition in diabetes is less studied, and potential interactions between body composition changes and important clinical variables such as age, glycemic control or diabetes complications need to be clarified.

In general, the effects of weight loss on sarcopenia are not straightforward. In fact, not taking into account initial body composition, age, and-or pre-existing or concomitant catabolic conditions, the balance between fat and muscle loss during weight loss programs may lead either to improved overall body composition with a relative increase in percent lean mass, or to worsened sarcopenia. In addition, a positive impact of fat loss on muscle quality and strength, likely through reduced muscle fat deposition and improved energy metabolism, is common following supervised weight loss; thus, potential changes in muscle function should be also taken into consideration [133]. Therefore, in patients with or at risk for sarcopenic diabetes, the clinical risk-benefit balance of relative losses of body fat and muscle need to be assessed and monitored. In patients at higher risk or in the presence of sarcopenia, obesity management should not be contraindicated, but it should be monitored and accompanied by all available strategies to minimize loss of skeletal muscle mass and function.

7. Management of T2DM in patients with sarcopenic diabetes

Beyond management of excess or abnormal adiposity, specific issues in diabetes care regarding sarcopenia may need to be considered. Optimization of glucose control is a high priority in patients with established or high risk of sarcopenic diabetes, given the proven negative impact of hyperglycemia on muscle mass and function [44]. However, diabetes medications are not muscle-neutral however not muscle-neutral and may either reduce or enhance the risk of sarcopenic diabetes [134,135]. Although muscle parameters have not been included among hard outcomes of large randomized controlled studies, a potential negative impact on muscle has been reported for sulfonylureas and glinides [134,135]. Muscle-neutrality with potential rationale and reports of positive muscle impact has been suggested for metformin [134,135]. For drugs inducing relevant weight loss such as newer IMDs, inevitable loss of lean mass has been confirmed, in parallel to large fat mass reduction [60,61]. Potential beneficial impact on body composition and muscle functional parameters in diabetes warrants additional investigation [60,61]. Sodium-glucose transport protein-2 (SGLT2) inhibitors also induce less pronounced weight loss by increasing urinary glucose output and have therefore potential to induce muscle loss (134,135). Studies on their impact on body composition are largely missing, and body composition and muscle function should be monitored in patients with diabetes undergoing SGLT-2 treatment with higher risk for sarcopenia. If clinically appropriate, medications with reported muscle benefits should be given priority in individuals with established or higher risk for sarcopenic diabetes. Comprehensive management strategies should include special attention to complications that may directly or indirectly enhance the risk of sarcopenic diabetes, including but not limited to neuropathy and peripheral vascular disease. Successful management may enhance patient mobility and physical activity with potential to prevent, limit or delay muscle loss and dysfunction. Prevention of osteoporosis and falls is a key component of care to prevent fracture and trauma with related immobility and muscle deterioration, particularly in patients with visual impairment due to retinopathy and impaired balance due to neuropathy [136].

Accelerated muscle loss occurs with hypercatabolic conditions or prolonged underfeeding, requiring sustained protein and calorie administration and medical nutrition [137]. Care must be taken under these conditions to avoid exacerbating hyperglycemia [137,138]. Higher insulin doses may be needed but they may increase the risk for hypoglycemia and glucose variability [137–139]. Diabetes-specific nutrition formulas have lower total carbohydrate content and glycemic index, higher fiber content, and higher unsaturated fat than standard/enteral nutrition formulae [103,137,138]. Diabetes-specific nutrition formulas, particularly when used together with continuous glucose monitoring, may help blunt prandial glycemic excursions and reduce overall variability [137,140]. Therefore, nutritional care may exert salutary effects on muscle loss, sarcopenic diabetes, and malnutrition under acute catabolic and chronic at-risk conditions.

8. Comment on type 1 diabetes and sarcopenia

Most available evidence on sarcopenic diabetes has been collected in T2DM, which is closely associated with obesity and

ABCD as key determinants of muscle derangements and sarcopenia. However, a special mention of T1DM is also needed. Uncontrolled T1DM is characterized by catabolic changes in muscle, primarily due to insulin deficiency and its catabolic impact on protein turnover, with resulting muscle loss [141]. At variance with T2DM, muscle catabolism is normalized in patients with T1DM following adequate insulin replacement therapy [141]. Regardless of glycemic control levels, diabetes complications may also negatively affect muscle mass and function in T1DM. Importantly, epidemiological shifts at population level, with growing prevalence of obesity in children and young adults ("double diabetes"), enhance the prevalence of previously described metabolic derangements with negative impact on skeletal muscle mass and function in T1DM [142]. For these reasons, particularly in the presence of longer disease duration, prevention of obesity, diabetes complications, and sarcopenia needs to be prioritized in patients with T1DM, and skeletal muscle mass and function need to be assessed in patients at higher risk.

9. A call for action

ESPEN and DNSG assert that sarcopenic diabetes is a relevant clinical and research priority. Research priorities include high-quality studies on optimal treatment strategies, including but not limited to: optimal protein intake to prevent and treat sarcopenic diabetes; optimal calorie-protein ratios for different populations; prevention of micronutrient deficiencies; potential nutraceutical approaches; and optimal exercise training regimens. These approaches should also be investigated in patients with T2DM undergoing weight loss for the management of overweight or obesity, particularly in those treated with IMDs, as more information is needed on their impact on body composition and potential interactions with glycemic control, complications, and comorbidities. Knowledge gaps on sarcopenic diabetes need to be addressed among healthcare professionals, by promoting awareness and best available education. Sarcopenic diabetes diagnosis needs to be routinely implemented in clinical practice, with routine muscle functional assessment (e.g. handgrip strength) and routine body composition measurement. ESPEN and DNSG support the implementation of the SOGLI diagnostic algorithm in patients with obesity and ABCD. Optimization of the SOGLI algorithm components for implementation in diabetes should also be considered. Finally, the best possible dietary and behavioral management should be directed toward patients with established sarcopenic diabetes or those at risk for it.

ESPEN and DNSG commit to coordinated actions to promote research and to increase awareness initiatives, including workshops, congress sessions and educational activities. We are convinced that achievement of these goals has strong potential to reduce the burden of morbidity and mortality in the increasing population of people with diabetes and sarcopenia.

Author contributions

RB conceptualized the paper and wrote the original draft; JIM contributed to critical draft revision and structure, reviewing and editing; JLS, LG, CWCK, MLS contributed to critical draft revision, reviewing and editing; MDB-P, YB, LC, TC, A-MA, CD, NED, SMS, SK, JS-S, AG, CC, HK, LMD, US, GR contributed to draft revision, reviewing and editing. No one eligible for authorship has been excluded from the list of authors.

AI statement

AI or AI generative technologies were not used in the preparation of the manuscript.

Funding statement

No funding was received for the preparation of the current manuscript.

Conflict of Interest

RB received speaker fees from Novo-Nordisk and Ely-Lilly; JLS received consulting fees from Perkins Coie LLP, Tate & Lyle, Brightseed, Ingredion, Almond Board of California; and speaker fees from Dairy Farmers of Canada, FoodMinds LLC, Nestlé, Abbott, General Mills, Nutrition Communications, International Food Information Council (IFIC), Calorie Control Council, International Sweeteners Association (ISA), Arab Beverages Association, Collaborative CME and Research Network (CCRN), Phynova; and other support from California Walnut Commission, Peanut Institute, Nutrartis, Dairy Farmers of Canada, Danone; LG received grant funding from Nestle Health Science, Foundation Nutrition2000Plus; and speaker fees from Fresenius Kabi and Lilly; CWCK received grant funding from Advanced Food Materials Network, Agriculture and Agri-Foods Canada (AAFC); consulting fees from Lantmannen; travel support from International Nut and Dried Fruit Council, McCormick Science Institute; MDB-P received consulting fees from Abbott Nutrition, Fresenius Kabi, Danone Nutricia; and speaker fees from Abbott Nutrition, Fresenius Kabi, Nestle Healthcare, Danone Nutricia; YB received grant support from Novo Nordisk, Ely-Lilly, Nestlé Health Science; LC received grant support from Protein Industries Canada, United Soybean Board, Alberta Pulse Growers; speaker fees from Arkansas Children's Hospital, Plant-Based Health Professionals UK; and travel support from World Sugar Research Organization; TC received speaker fees from Fresenius-Kabi, Danone-Nutricia; expert testimony fees from Nestle; travel support from Fresenius Kabi; A-MA received consluting fees from Novo-Nordisk Foundation; CD received travel support from Numil Hellas, Mavrogenis Empowering Health; NED received grant support from Abbott Nutrition; speaker fees from Abbott Nutrition; SMS received grant support from VectivBio, Nestlé Health Science, Dr. Falk, Sanofi; consulting fees from Baxter, Nestlé Health Science, Pfizer, Takeda; speaker fees from Amgen, Fresenius-Kabi, MSD, Nestlé Health Science, Nutricia, Thermo Fisher, Ely-Lilly, EverPharma; SK received speaker fees from Baxter, B-Braun, Nestle, Nutricia, Fresenius-Kabi; JS-S received grant support from International Nut and Dried Fruit Foundation; consulting fees from Spain Institute Danone Advisory Board; travel support from Nut and Dried Fruit Foundation; advisory board memberships in Scientific Committee of Danone Institute International; MLS received travel support from Nutricia and Abbott; JIM received speaker fees from Abbott Nutrition, Merck; advisory board fees from Abbott Nutrition, Twin Health; stock ownership for Twin Health; AG, CC, HK, LMD, US, GR have no conflicts of interest to declare.

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