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ESPEN Endorsed Recommendation

Challenges of nutritional support in patients with diabetes: A position paper of the ESPEN special interest group

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S U M M A R Y

Malnutrition affects up to 30 % of the general population, and especially older people with polymorbid conditions. In parallel, the prevalence of diabetes increases with age affecting over 800 million adults worldwide. Healthcare providers are increasingly challenged to care for patients with diabetes that require nutritional support. To address this issue, the ESPEN Special Interest Group "Nutrition and Diabetes", aims to provide guidance for health care providers that treat these patients. This paper had three aims: 1) to summarise the guidelines and recommendations regarding nutritional support and diabetes or stress hyperglycaemia provided by scientific societies, 2) to review the associations of nutritional disorders with diabetes and its pharmacological treatments, and 3) to identify the challenges of optimal nutritional care for patients with diabetes and stress hyperglycaemia. To this end, we con-ducted a systematic search of guidelines and recommendations on nutritional support for patients with diabetes or stress hyperglycaemia, that have been published in English by national and international societies over the last 15 years. Our systematic search showed that published guidelines and recom-mendations rarely addressed the practical management of blood glucose control according to the

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modality of nutritional support. The literature on the association of malnutrition with diabetes and its pharmacological treatment is very limited. The identified challenges include the multidisciplinary and multiprofessional continuity of care between the hospital and ambulatory settings, the ideal pattern of hospital food, the choice of oral nutritional supplements, the adjustment of diabetes management to nutritional support, and diabetes technology to support nutritional care in these patients.

1. Introduction

The ESPEN guidelines on clinical nutrition terminology defines several types of nutritional disorders [1]: malnutrition/undernu-trition (also termed protein-energy wasting), overweight and obesity [2], sarcopenia [3] and frailty [4], and micronutrient ab-normalities [5] that may occur simultaneously. Malnutrition has been defined by an international expert consensus as the presence of at least one phenotypic criterion (low body mass index (BMI), unintentional weight loss, or low muscle mass) and one etiologic criterion (inflammatory state, or decreased food intake or assim-ilation) [6]. It occurs in up to 30 % of the population depending on the healthcare setting [7], with an especially high risk among older people with polymorbid conditions. Malnutrition can coexist with obesity [8], whose definition has recently been extended beyond the excess fat mass to include also medical, functional and psy-chological impairments [2,9]. The prevalence of obesity is increasing rapidly and affects nearly 20 % of adults in Europe [10].

The prevalence of diabetes has dramatically decreased to 828 million adults worldwide. It is predominantly associated with ageing and excessive fat mass, and affects over 20 % adults older than 60 years [10]. The large majority is type 2 diabetes (T2D), but the prevalence of type 1 diabetes (T1D) now reaches 9 million adults, of which 1.1 million are over 60 years [11]. Hyperglycaemia is very common in patients receiving nutritional support, even in those without a previous diagnosis of diabetes. In hospitalized patients, the prevalence of hyperglycaemia reaches 50 % in pa-tients receiving parenteral nutrition (PN) and 30 % in those receiving enteral nutrition (EN) [12]. The additional burden of malnutrition increases the risk of morbidity and mortality in pa-tients with diabetes [13,14] or stress hyperglycaemia [15,16], highlighting the importance of preventing and treating malnutri-tion in these patients. Furthermore, with the increase in life ex-pectancy, healthcare providers are expected to care for more and more patients who require the combination of nutritional support and diabetes care. However, there is currently limited consensus and guidance on the management of nutritional support in the context of diabetes care and/or stress hyperglycaemia.

Thus, we have created a multidisciplinary ESPEN special in-terest group (SIG) that first met during the ESPEN congress in September 2023. The aims of this SIG are to provide guidance and evidence for optimal nutritional support in patients suffering from diabetes and stress hyperglycaemia. Here, we 1) summarise the guidelines and recommendations regarding nutritional support and diabetes or stress hyperglycaemia provided by scientific so-cieties, 2) review the associations between nutritional disorders and diabetes, and 3) identify the challenges of nutritional support in patients with diabetes and stress hyperglycaemia. These pro-ceedings are intended to pave the way for in-depth consensus papers on the identified gaps of knowledge.

2. Current guidelines and recommendations

We systematically retrieved guidelines and recommendations on nutritional support in patients with diabetes and stress hyperglycaemia (oral nutritional supplements (ONS), EN and PN) that have been published in English over the last 15 years by na-tional and international societies. The dietary guidelines for gen-eral T2D management were excluded and the reader is referred elsewhere for these guidelines [17–19].

We used the following search strategy in PubMed: (("Nutritional Support"[Mesh] OR "tube feeding"[tiab] OR "enteral nutrition"[tiab] OR "enterc feeding"[tiab] OR "intravenous feeding"[tiab] OR "intravenous nutrition"[tiab] OR "nutrition* support"[tiab] OR "oral nutritional supplement*"[tiab] OR "parenteral nutrition"[tiab]) AND ("Diabetes Mellitus"[Mesh] OR "Hyperglycemia"[Mesh] OR "dia-betes"[tiab] OR "dysglycemia"[tiab] OR "glycemi*"[tiab] OR "hyper-glycemia"[tiab]) AND ("guideline*"[ti] OR "recommendation*"[ti] OR "practice*"[ti] OR "consensus"[ti] OR "standard*"[ti])) AND ((humans[Filter]) AND (2010:2025[pdat]) AND English[Filter])).

We found 63 articles after excluding those not reporting guidelines, recommendations or consensuses issued by national or international societies, and those focusing on critically ill (ICU) patients. When a society had published several guidelines, we used the most recent one. We finally included 11 papers. Table 1 summarizes the recommended glycaemic management in the context of nutritional support from the included articles.

Overall, the published guidelines and recommendations are mainly based on expert consensus. None of them provided advice for glycaemic management for patients taking ONS. They focus mainly on hyperglycaemia management and insulin dosage *per se* and generally recommend a target blood glucose concentration between 140 and 180 mg/dL (7.8–10.0 mmol/L) under artificial nutrition [20,21]. However, the practical management of blood glucose control according to the modalities of nutritional support (type, macronutrient content, timing, frequency, quantity and rate of administration) is rarely detailed.

3. Pathophysiology

Several studies suggest that hyperglycaemia during nutritional support, especially PN, is associated with increased morbidity and mortality [15,16], although this association may not be causal and just reflect, at least partly, the severity of the disease. Optimal glucose control plays a critical role in promoting tissue healing, immune function, and minimizing the risk of infection. In critically ill patients, maintaining glucose within a tight range can improve wound healing, reduce sepsis risk, and minimize the need for invasive interventions [22].

Hyperglycaemia arises from an imbalance between insulin production and action. Insulin resistance, commonly associated with excess adipose tissue, type 2 diabetes and stress hyper-glycaemia, is characterised by impaired insulin action leading to increased endogenous glucose production and reduced peripheral glucose uptake. The underlying mechanisms of insulin resistance include systemic and tissue-specific inflammation, adipose tissue dysfunction with increased efflux of fatty acids, and ectopic lipid accumulation in liver (hepatic steatosis) and skeletal muscle [23]. Inflammatory proteins and specific lipid species can interfere with insulin signalling and reduce glucose uptake [24]. In addition,

Table 1
Guidelines or recommendations of diabetes or hyperglycaemia management in patients receiving enteral (EN) or parenteral nutrition (PN) (alphabetic order).

Scientific Societies	References	General comments	Glycemic management under EN or PN	Glucose monitoring during EN and PN
American Association of Clinical Endocrinologists (AACE) & American Diabetes Association (ADA)	[107]		• EN: Basal insulin and correction insulin. Half of the patients require also intermediate-acting insulin • PN: Insulin therapy highly recommended	• Continuous EN or PN: every 4–6 h • Cyclic EN or PN: frequency individualized
American Association of Clinical Endocrinologists (AACE)	[108]	Patients with EN or PN at high risk of hyperglycaemia	–	Continuous EN or PN: Glucose monitoring every 4–6 h, or more frequently after sudden discontinuation of EN
AACE, Obesity Society, American Society for metabolic and bariatric surgery, Obesity Medicine Association, & American Society of Anesthesiologists	[109]	EN or PN may be required after bariatric surgery in patients at high nutritional risk, guided by clinical practice guidelines	–	
American Diabetes Association (ADA)	[110]	Risk of hypoglycaemia if interruption of oral, EN or PN Appearance of better glucose control with diabetes-specific EN	• EN a) Continuous EN: Basal: prior basal insulin or 5 IU intermediate acting insulin every 12 h or 10 IU long-acting insulin per day Nutritional: regular insulin every 6 h or rapid-acting insulin every 4 h, starting with 1 IU per 10–15 g carbohydrates b) Bolus EN: Basal: prior basal insulin subcutaneous or 5 IU intermediate acting insulin every 12 h or 10 IU long-acting insulin per day Nutritional: regular insulin or rapid-acting insulin before each feeding, starting with 1 IU per 10–15 g carbohydrates • PN: addition of regular insulin in PN solution, starting with 1 IU per 10 g carbohydrates	Under regular insulin: every 6 h Under rapid acting insulin: every 4 h
American Society for Parenteral and Enteral Nutrition (ASPEN)	[111]	In case of nutritional support: Target glycemia: 7.8–10 mmol/l Hypoglycaemia: <3.9 mmol/l	No recommendation for diabetes-specific EN formulae in hyperglycaemia	–
Diabetes UK	[112]		• EN: Use of Diabetes-specific formulae even if not used in routine in the UK	–
European Federation of Internal Medicine (EFIM)	[113]	In case of nutritional support: Target glycemia: 7.8–10 mmol/l	If EN or PN in people needing insulin, treatment with basal insulin (generally continuation of preadmission dose, with long-acting basal insulin being 20–40 % of total daily dose), prandial insulin if oral intakes and correction insulin. • EN: standard EN formulae acceptable a) Cyclic EN: Short-acting or rapid acting insulin. 1 IU every 6 h for every 10–15 g of b) Continuous EN: Regular insulin every 6 h.1 IU every 6 h for every 10–15 g of c) Bolus EN: Rapid-acting insulin before every bolus • PN: 1IU for every 10 g dextrose in PN solution + correction insulin subcutaneously. T1D: Long-acting insulin + rapid-acting depending on the modalities of nutritional support. Continuation of basal insulin if feeding discontinuation	Glucose monitoring at least every 6 h if EN or PN

(continued on next page)

Table 1 (*continued*)

Scientific Societies	References	General comments	Glycemic management under EN or PN	Glucose monitoring during EN and PN
Endocrine Society	[114]	Proposed research areas: • insulin therapies according to mode of EN delivery • non-insulin injectables and oral antihyperglycemic drugs in case of EN • CGM devices with closed-loop insulin pump devices in case of EN.	• EN: In non-critical ill patients fed by diabetes-specific EN formulae, NPH-based or basal bolus regimens In selected T2D patients with mild hyperglycaemia, DPP4i with correction insulin or scheduled insulin therapy adapted to mode of EN delivery (continuous, bolus, cyclic) • PN: Not addressed	–
European Society of Clinical Nutrition and Metabolism (ESPEN)	[98]	Hyperglycaemia risk: • Higher with PN than EN due to incretin effect • PN: Reduction in patients with obesity or diabetes if initial glucose administration < 2 g/kg/d	• EN: use of diabetes-specific EN formulae	–
Spanish Society of Endocrinology and Nutrition (SEEN) and Spanish Society of Enteral and Parenteral Nutrition (SENPE)	[97]	Post-pyloric feeding in case of gastroparesis Targets: Hb1Ac: <7 % Proposed research areas: • Regular updates on carbohydrate metabolism in patients under EN impact of simultaneous administration of other nutrients • Impact of feeding formulae according to etiology of hyperglycaemia, drugs and co-morbidities; • Impact of different insulin regimens	• EN: Use of diabetes-specific EN formulae with fibers in outpatients and inpatients, including mechanically ventilated patients Basal-bolus regimens: Basal insulin: NPH/8-12 h or Glargin/24 h or Detemir/12-24 h + prandial insulin: rapid acting insulin before each bolus or every 8 h in continuous feeding + correction insulin every 6–8 h according to monitoring • PN: not addressed	Glucose monitoring every 4–8 h according to monitoring
China International Exchange and Promotive Association for Medical and Health Care Chinese Nutrition Society, Chinese Diabetes Society, Chinese Society for parenteral and enteral nutrition, Chinese Medical Doctors association	[115]	Patients with diabetes have a higher incidence of nutritional risk/malnutrition Oral nutritional supplements preferred in patients with diabetic nephropathy and malnutrition	• EN: Preferred to PN for stress hyperglycaemia Diabetes-specific EN formulae in patients with stress hyperglycaemia rather than standard EN formulae to improve glycaemic control and reduce costs	–

oxidative stress, stress hormones, concomitant medication and surgical interventions contribute to insulin resistance and hyperglycaemia [25]. Finally, hyperglycaemia itself can negatively affect beta-cell function and induce oxidative stress, which might affect insulin production, secretion and peripheral action, although this mechanism is still debated [26].

Beyond glucose regulation, insulin is a critical anabolic regu-lator of protein and fat metabolism. Insulin promotes protein synthesis and reduces muscle protein breakdown [27]. This effect on net protein balance is particularly important in maintaining muscle mass, especially in periods of stress or illness where pro-tein catabolism is accelerated [27]. In addition, insulin facilitates triglyceride (TG) storage in adipose tissue and increases *de novo* lipogenesis. Insulin also plays a major role in non-esterified fatty acid (NEFA) metabolism in the postprandial state by inhibiting intracellular TG lipolysis and disrupting the TG lipolysis/NEFA re-esterification cycle in adipose tissue, thus increasing adipose tis-sue retention of meal-derived fatty acids [28]. Considering the anabolic roles of insulin is essential to ensure nutritional support is tailored for glucose control, protein synthesis and lipid metabolism.

Most metabolic processes follow circadian rhythms, which are orchestrated by the master clock in the hypothalamic suprachiasmatic nucleus, entrained by external light and environmental cues, and are then reported downstream at the organ level by the peripheral clock machinery, including the release of glucoregulatory hormones [29]. The interplay between circadian rhythms and insulin sensitivity can be observed in the diurnal patterns of blood glucose levels [30,31]. Disrupted circadian rhythms lead to impaired glucose control, via misaligned insulin secretion and sensitivity [30].

4. Nutritional disorders associated with diabetes

Nutritional risk and malnutrition occur frequently in patients with diabetes but are likely underestimated due to the presence of obesity [8]. A recent meta-analysis of over 18,000 patients (mainly in the hospital setting) found that the overall prevalence of malnutrition and of nutritional risk in patients with T2D was 33 % and 44 %, respectively, depending on the used diagnostic tool [32]. The factors most often associated with malnutrition in diabetes were a low BMI, older age, longer duration of diabetes, insulin treatment, severe diabetic foot ulcers, cardiovascular diseases (CVD), as well as low blood levels of albumin, haemoglobin, tri-glycerides, high-density lipoprotein (HDL)-cholesterol, and elevated C-reactive protein and glycosylated haemoglobin

(HbA1c). Interestingly, it has also been suggested that chronic malnutrition in underprivileged populations may be a trigger for the development of diabetes [33].

Obesity has long been associated with the development of T2D and affects 80–90 % of people with T2D [34]. The prevalence of obesity in T1D is much lower than in T2D [35] and is estimated to be either similar [35] or lower [36] than in the general population, depending on the studied population.

The link of diabetes with sarcopenia [37,38] and micronutrient deficiency [5] has only been recently addressed. Proposed risk factors for sarcopenia in diabetes are older age, potentially sex, low BMI, poor glycaemic control, and diabetic complications (ne-phropathy, neuropathy, CVD, metabolic dysfunction-associated liver disease (MASLD), or cognitive impairment) [37]. Figure 1 shows potential associations between known diabetes complica-tions and nutritional disorders.

5. Nutritional management of common diabetic complications

The dietary guidelines for general T2D management are beyond the scope of this article [17–19]. We will focus here on common diabetic complications that may compromise nutritional status, as patients with these complications may benefit from nutritional support.

5.1. Diabetic nephropathy

Malnutrition is a major problem in patients with chronic kid-ney disease (CKD) and has multifactorial causes [39]. Diabetes is associated with the progression of CKD and all-cause mortality [40].

The ESPEN guidelines do not suggest specific nutritional man-agement strategies for patients with CKD due to diabetes, compared to other aetiologies of CKD [41,42]. Other societies provide specific recommendations for protein needs in adults with diabetes. The Kidney Disease Outcomes Quality Initiative (KDOQI) recommends a daily protein intake of 0.6–0.8 g/kg/day in patients with decreased estimated glomerular filtration rate (eGFR) who are not on dialysis, independently of the presence of diabetes. However, restricting dietary protein below 0.8 g/kg/day, alongside advised restrictions on intake of carbohydrates and fat, may significantly reduce caloric intake and increase the risk of malnutrition [43]. The Diabetes and Nutrition Study Group rec-ommends a protein intake of 10–15 % of total energy intake in moderate diabetic nephropathy (KDIGO stage 3a, i.e. eGFR 45–60 ml/min/1.73 m^2) [44]. Finally, the American Diabetes As-sociation recommends dietary protein intake of ~0.8 g/kg/day for patients with diabetes and non-dialysis-dependent CKD [45]. In contrast, for patients with diabetes on dialysis, higher levels of dietary protein intake should be considered because of higher risk of malnutrition and protein losses during dialysis [43]. Of note, a systematic Cochrane review showed that protein restriction has an unclear impact on mortality and kidney failure in patients with diabetes, with limited data regarding its impact on nutritional status [46].

5.2. Diabetic gastroparesis and autonomic dysfunction

Gastroparesis refers to delayed gastric emptying of solids without mechanical obstruction [47]. Long-standing diabetes can affect autonomic function which can lead to diabetic gastroparesis, especially in case of sustained poor glucose control. Patients may experience greater glycaemic variability with fast excursions from hypo-to hyperglycaemia, as absorption of carbohydrates is highly variable. Nausea and abdominal complaints associated with gas-troparesis can reduce food intake, increasing the risk of malnutrition.

For patients with mild gastroparesis, dietary modifications such as small, frequent, low-fat, low fibre containing meals and increased liquid nutrient intake are recommended to enhance gastric emptying and mitigate the risk of malnutrition [48]. Acidic and spicy foods should be avoided. Tolerance to liquids is better than solids, therefore homogenized food can be tried if gastric emptying of solid foods is delayed. In severe cases, enteral nutri-tion through naso-jejunal tube, jejunostomy or gastrostomy may

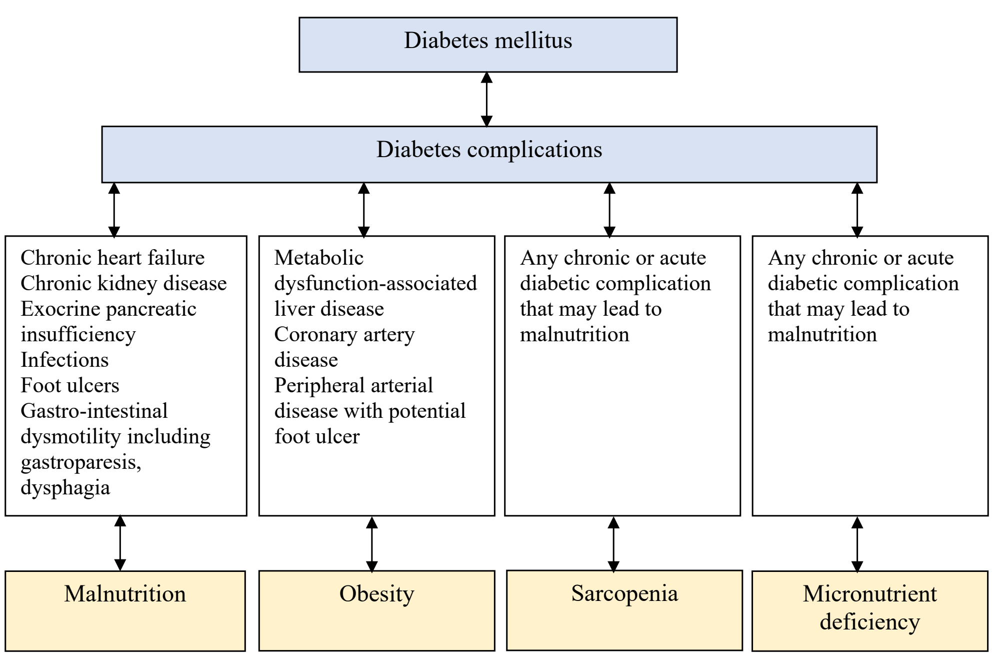

Figure 1. Association of diabetes and diabetic complications with nutritional disorders.

be required [48]. Gastroparesis is an adverse effect of GLP-1 receptor agonists, which can increase symptoms in patients with autonomic dysfunction [49].

5.3. Diabetes in pancreatic diseases

Also termed ‘pancreatic diabetes’, ‘pancreatogenic diabetes’ or ‘type 3c diabetes’, diabetes can develop in the context of diseases or interventions affecting the pancreas (e.g. pancreatic surgery, pancreatic cancer, acute or chronic pancreatitis, cystic fibrosis, haemochromatosis) [50] and carries a higher risk of malnutrition than other types of diabetes [51]. Pancreatic diabetes is often associated with exocrine pancreatic insufficiency, which compromises macronutrient absorption even when treated with enzyme replacement therapy [52]. Although pancreatic diabetes accounts for 5–10 % of diabetes cases, it is frequently misclassified as type 2 diabetes [53], leading to inappropriate dietary recommendations [34].

Pancreatic enzyme replacement therapy should be administered during meals and snacks to improve digestion and absorption of fat and protein [54]. The ESPEN guidelines recognise the presence of diabetes as a main cause of malnutrition in chronic pancreatitis but do not provide any specific dietary guidance [54].

5.4. Diabetic foot

Compromised nutritional status is very common in patients with diabetic foot ulcers. Observational studies indicate that the prevalence of malnutrition is 60 % [55], while the prevalence of nutritional risk is 85 % [56]. Poor nutritional status has been associated with the severity of infection and increases the risk of amputation [55,56]. However, the benefits of nutritional interventions are yet uncertain [57,58].

5.5. Diabetes and liver disease

T2D in the context of obesity is associated with MASLD and liver cirrhosis. The prevalence of T2D in liver cirrhosis is between 27 and 51 % and depends on the underlying cause [59]. Malnutrition occurs in 20–60 % of patients with liver cirrhosis justifying nutritional support [60]. In patients with liver cirrhosis, protein intake should be increased to 1.2–1.5 g/kg/day and total energy intake to 30–35 kcal/kg/day [61]. Patients are recommended to consume at least 6 meals per day including a late evening snack, thus reducing fasting periods as much as possible [62]. This may be challenging in patients treated with bolus regimens of insulin, leading to either hyperglycaemia or insufficient calorie intake. When nutritional needs are not met by oral intake, EN is favoured over PN. In hepatic encephalopathy due to gastrointestinal bleedings or infections, protein intake should not be reduced. Other causes of hepatic encephalopathy might benefit from a restriction in animal protein [62].

6. Association between nutritional state and hyperglycemia

6.1. Association of weight loss and hyperglycaemia

In case of hyperglycaemia, weight loss can be explained by glycosuria, which is responsible for calorie loss through the urine, in addition to the impairment of skeletal muscle metabolism by reduced insulin action [27]. Glucosuria occurs when the renal reabsorption threshold is reached, usually at a glycaemia $\geq$180 mg/dL (10 mmol/L), and can be substantial with caloric losses up to 300 kcal per day in uncontrolled T2D [63]. The resulting negative energy balance can trigger hyperphagia in some but not all patients. In older or polymorbid patients, the classic symptoms of polyuria, polydipsia and compensatory hyperphagia might be dampened, and the reduced perception of thirst increases the risk of dehydration. Urinary incontinence, nocturnal polyuria and dehydration can be clinical signs of hyperglycaemia. Therefore, weight loss warrants assessment of glycaemia.

6.2. Association of body composition and diabetes

The relationship between obesity and the development of T2D is described above [64]. In addition to excess fat, unfavourable body composition with low muscle mass and function, (sarcopenia), has also been associated with the development of T2D. Muscle plays a key role in glucose uptake via GLUT-4 transporters [65].

Data from the UK Biobank show that low handgrip strength, which measures muscle function, is associated with a higher incidence of T2D independent of confounding factors including age, adiposity and lifestyle (median follow-up 5.3 years) [66]. The lowest quintile of absolute handgrip strength was associated with a 50 % higher risk of T2D in men and 25 % higher risk in women compared with people in the highest quintile [66]. Sarcopenia is also associated with increased risk of CVD, which might occur earlier than in those without sarcopenia. T2D with sarcopenia had CVD incidence rates equivalent to those without sarcopenia who were 14.5 years older [67]. Therefore, sarcopenia screening and prevention may be useful to prevent the cardiovascular complications in patients with T2D [67].

Sarcopenia and T2D share multiple pathophysiological mechanisms, and this relationship may be bidirectional [68]. Common changes in muscle include a shift in myocyte composition, a reduced ability for muscle regeneration and a higher myosteatosis (fat infiltration within and between myocytes) [68]. Both conditions are linked to an imbalance in myokines, chronic low-grade inflammation and increased levels of oxidative stress [68]. Additional influencing factors related to sarcopenia in patients with T2D include advanced age, lower BMI, higher Hb1Ac, and the presence of diabetic nephropathy, diabetic neuropathy and MASLD [37].

Obesity combined with sarcopenia confers a greater risk of T2D than either condition alone [69]. A recent meta-analysis reported a prevalence of 27 % of sarcopenic obesity in patients with any type of diabetes, and an association with serious adverse outcomes including decreased eGFR, proteinuria, cognitive decline, and insulin resistance [70].

T1D has also been associated with sarcopenia and loss of muscle strength, termed dynapenia. In two cross-sectional studies including 39 and 62 patients with T1D, sarcopenia was present in 8 % [71,72] and dynapenia in 23 % [71]. Furthermore, a recent meta-analysis reporting on 15 studies in T1D subjects found a prevalence of sarcopenia ranging from 8 to 42.9 % [73]. Older age and lower BMI are the main determinants of muscle decline in these patients, while there is conflicting data regarding to impact of diabetes duration, glucose control, dietary and physical exercise behaviors.

6.3. Association between diabetes treatment and nutritional state

The impact of pharmacological diabetes treatments on body weight, HbA1c and hypoglycaemia is summarized in Table 2 (data from the German Diabetes Society, the German Society for Internal Medicine and the American Diabetes Association) [17,74,75]. The guidelines of these societies acknowledge the existence of sarcopenic obesity, i.e. the coexistence of excessive fat mass and low muscle mass and function. They highlight the risk of weight loss

Table 2
Impact of diabetes pharmacological treatments on body weight, HbA1c and hypoglycaemia.

Drug class	Effect on weight	Effect on HbA1c	Hypoglycaemia
Metformin	↔↓	↓↓	↔
SGLT-2 inhibitors	↓	↓↓	↔
GLP-1 receptor agonists	↓↓	↓↓	↔
DDP-4 inhibitors	↔	↓	↔
PPAR-gamma agonists	↑	↓	↔
Sulfonylurea	↑	↓↓	↑↑
Fast and long-acting Insulin	↑↑	↓↓	↑↑

Adapted from [17,74,75].
Abbreviations: HbA1c, glycated haemoglobin; SGLT-2, sodium-glucose cotransporter 2; GLP-1, glucagon-like peptide; DPP-4, dipeptyl peptidase; PPAR-gamma, Peroxisome Proliferator-Activated Receptor-gamma.

and advise against the use of GLP-1 receptor agonists and GLP-1/ GIP dual receptor agonists in frail patients. Frailty is a state of age-related increased vulnerability often defined by the Fried's criteria that include low handgrip strength, exhaustion, low walking speed, low physical activity and involuntary weight loss [4].

While some diabetes treatments may reduce body weight, their impact on muscle mass and function is less clear. A systematic review reported that metformin, GLP-1 receptor agonists and SGLT2 inhibitors may contribute to muscle loss by decreasing energy intake or increasing glucosuria and recognized the need for large trials to confirm the impact of these drugs on the develop-ment of sarcopenia [76]. GLP-1 receptor agonists when used for weight loss either alone or in combination with other incretins have been associated with loss of fat-free mass in randomized controlled trials [77–80] and meta-analyses [81,82]. Therefore, their use has raised concerns in patients with low muscle mass, such as those at nutritional risk or with malnutrition, sarcopenia or frailty. There is some evidence that GLP-1 receptor agonists do not compromise muscle function [83] and may even improve handgrip strength [84]. Thus, the impact of GLP-1 receptor ago-nists and their derived drugs on the whole spectrum of sarcopenia, i.e. muscle mass, but also function and strength, requires more detailed studies. Sulfonylurea may also contribute to muscle at-rophy, although they are promoting weight gain, but the mecha-nisms are unclear [76,85] and their use is now decreasing. Insulin treatment appears to decrease muscle protein breakdown but stimulates muscle protein synthesis only in case of increased de-livery of amino acids [27] and may preserve muscle mass in pa-tients with T2D [86]. Deciphering the impact of different diabetes drug classes also requires better understanding of the impact of diabetes on muscle mass. Diabetes might lead to muscle mass loss by an increased ratio of protein degradation over synthesis, as reported in other chronic diseases [87].

7. Challenges of nutritional care in diabetes

Nutritional care in patients with diabetes needs to be person-alised with targeted glycaemic control, weight management, preservation of muscle mass and function, and prevention and treatment of comorbidities. Below, we highlight challenges that healthcare professionals may encounter when addressing nutri-tional care in treating patients with diabetes.

7.1. Multidisciplinary and interprofessional collaborative care to improve patient outcomes

Since 20–25 % of hospital inpatients have T2D or T1D at admission [88], a multidisciplinary diabetes team should be available in every hospital [89]. In addition, diabetes treatment should be coordinated with nutritional support to have synergistic rather than antagonistic treatment effects. A high prevalence of nutrition-induced hyperglycaemia likely suggests insufficient multidisciplinary efforts.

Over the past decade, several drugs (e.g. SGLT2 inhibitors, GLP-1 receptor agonists, GLP-1/GIP dual agonists) and devices (e.g. continuous glucose monitoring (CGM), automated-insulin delivery (AID) and smart pen systems) have increased therapeutic options to achieve target glycaemic goals with no or minimal risk of hypoglycaemia (Table 2). In addition, novel non-insulin agents have consistently demonstrated therapeutic benefits beyond glucose control, such as cardiovascular, renal and metabolic health [90]. The use of these therapeutic options requires multidisci-plinary and multiprofessional collaborations to optimize treat-ment compliance and health benefits.

The lack of continuity of care between hospital and ambulatory settings for patients with diabetes and nutritional support might put the patient at risk of rehospitalisation. For example, if diabetes medications during hospital stay are not adjusted to nutritional support, there is an increased risk of hypo- or hyperglycaemia after discharge. Also, the loss of body weight and muscle mass during hospital stay might require additional adjustments of nutritional support and diabetes drugs before discharge. Therefore, discharge medication and follow-up is required in combined diabetes and nutritional care [91].

7.2. Standard hospital vs. diabetes-specific meals

The inconsistent content in carbohydrates and fibres between meals from one day to the next leads to glucose variability and difficulties in diabetes management. Glucose control should not be a pretext to reduce caloric and carbohydrate intake but diabetes therapy should be adjusted instead, to avoid increasing the risk of malnutrition [92]. Diabetes-specific diets with a lower carbohy-drate content are no longer common in hospitals. If standard hospital diet is high in proteins, patients with diabetes do not need any further increase in protein intake. The ESPEN guidelines for hospital nutrition defines no specific need for patients with dia-betes [93].

7.3. Choice of oral nutritional supplement (ONS)

ONS are sometimes withheld for fear of hyperglycaemia, which is inadequate because hyperglycaemia can be managed with the appropriate treatment and nutritional counselling. The impor-tance of diabetes-specific ONS with regards to fibre content, gly-caemic index/ load and effects on glucose excursion remains unclear. While most ONS do not contain sucrose, they sometimes have added non-caloric sweeteners or non-hydrolysed starches and modified maltodextrins. Patients with diabetes on insulin treatment need close glucose monitoring and insulin dose ad-justments when ONS therapy is started.

Current diabetes-specific ONS have decreased carbohydrate content which is usually replaced by a higher content of monounsaturated fatty acids and medium chain triglycerides (MCT). All diabetes-specific ONS contain soluble fibres to decrease glycaemic index. They could improve postprandial glycaemic control [94–96]. In addition, ONS without fibres could be desirable for some underlying gastrointestinal disease [97,98]. More high-quality studies should be performed to unveil potential specific beneficial effects of diabetes-specific ONS in selected clinical settings and conditions.

7.4. Diabetes treatment during EN and PN

Use of EN or PN in patients with diabetes or stress hyperglycaemia needs anticipatory actions for glycaemic control.

Intermittent administration of EN requires adjustment of glucose lowering drugs, usually insulin as a basal-bolus regimen. Glycaemic control in continuous EN administration is usually achieved with long-acting insulin but EN interruptions carry the risk for hypoglycaemia. Intermediate acting insulin, as Neutral Protamine Hagedorn (HPN), is indicated in cyclic EN, particularly if administered over 12 h.

For PN, adding short-acting insulin to PN bags is considered safe when this practice is strictly regulated [99] and prevents hypoglycaemia when PN is interrupted. The amount of added insulin to PN bags should be titrated according to glucose load and readings and adjusted daily. The initial insulin dosage can be estimated by the glucose content of the parenteral solution [100]. Short-acting insulin in PN bags is equally effective as 50 % of insulin needs as short-acting insulin in PN bags plus 50 % as long-acting insulin subcutaneously [101], but with a higher rate of non-severe hypoglycaemia. Another study reported a lower rate of hyperglycaemia with short-acting insulin in PN bags vs. long-acting insulin subcutaneously alone [102].

7.5. Diabetes technology challenges

Novel diabetes technology advances have remarkably changed diabetes care over the past 10 years. Improvement of reliability, accuracy, and ease of use have led to a rapid acceleration in the numbers of people using diabetes technology, such as CGM, AID and smart pen systems. In the context of nutritional support, these systems enable better alignment of insulin dosing with meal-induced glycaemic excursions. Several studies have highlighted the benefits of using CGM for timely identification of dysglycaemia and various clinical trials demonstrated that the deployment of AID systems significantly improves glucose control without increasing the risk of hypoglycaemia [103–105]. However, several barriers remain for the actual implementation and use of these technologies in inpatient care, ranging from interoperability and infrastructure issues to uncertainties regarding costs, revenues and data privacy.

8. Summary and goals of the special interest group

The ESPEN SIG on nutrition and diabetes recognises the need for practical guidance on diabetes management in patients requiring nutritional support. This guidance could be improved with the development of computer-based tools, flowcharts, and practical advice. Glycaemic management in the context of nutritional support needs to be adapted to the patient's socio-economic characteristics, clinical setting (ambulatory vs. hospitalised), type of diabetes (type 1, type 2, pancreatic, and other forms), and comorbidities (e.g. pancreatic diseases, cystic fibrosis, use of glucocorticoids and other glucose-increasing medications, major upper GI surgery etc). Practical guidelines should clarify the procedure of antidiabetic treatment adaptation (type of medication, frequency and dose) to actual nutritional state (malnutrition, at nutritional risk, sarcopenia/frailty, obesity, sarcopenic obesity), and to the introduction or tapering of nutritional support according to its modalities (route, composition, length of administration). The group also recognises the importance of more clinical studies on the impact of diabetes-specific nutritional formula on glycaemia and other outcomes and of the addition value of CGM monitoring [106].

Regarding education in the field of nutritional support and diabetes, the aim would be to develop educational material for patients and specialized and non-specialized health care professionals, and to promote pre- and postgraduate education and continuous training. Online and in-person courses should be encouraged and accessible to a large range of audience (healthcare professionals, patients, politicians, industry).

Many unanswered questions remain and require further research with the aim of providing sound scientific evidence and exceed expert opinions.

9. Conclusion

This position paper lays the groundwork for improving knowledge and guidance in the field of clinical nutrition in patients with diabetes and stress hyperglycaemia. ESPEN is a multinational and multiprofessional society with leadership in the field of clinical nutrition and has established collaborations with other clinical nutrition and non-clinical nutrition societies. This ESPEN SIG is therefore well positioned to develop practice guidelines on glycaemic management during nutritional support and to initiate research projects to improve the nutritional care in patients with diabetes and stress hyperglycaemia.

The high prevalence of diabetes and malnutrition and other nutrition-related disorders, e.g. obesity, sarcopenia, micronutrients deficiencies in patients with diabetes, highlights the need for guidance on nutritional care of patients with diabetes in all clinical settings, and to fill the gaps identified in this review. This implies evidence-based guidelines for composition of nutritional support formulas and optimization of glycaemic control during nutritional support integrating diabetes care technologies (CGM, AID, etc.) and artificial intelligence-based algorithms, and better understanding of the effects of diabetes drugs on nutritional state, body composition, and muscle mass and function.

Data sharing

There is no original data to share.

Credit author statement

L. Genton: conceptualization, methodology, writing-original draft, project administration.

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T.H. Collet: conceptualization, methodology, writing-original draft.

No one eligible for authorship has been excluded from the list of authors.

Declaration of generative AI and AI-assisted technologies in the writing process

No generative AI or AI assisted technology was used in the writing process.

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Conflict of interest

LG received grants from Nestlé Health Science, Foundation Nutrition 2000Plus, Geneva Cancer League; consulting fees from Nestlé Health Science, Fresenius Kabi, Lilly; honoraria for lectures, presentations from Abbott; support for attending meetings and/or travel from Nestlé Health Science.

MLS received support for attending meetings and/or travel by Nutrisense and FELANPE. He is President of the Spanish Alianza Masnutridos.

MBP received consulting fees from Nutricia, Fresenius Kabi, Abbott; honoraria for lectures, presentations from Nutricia, Fresenius Kabi, Abbott, Nutrition, Vegenat Healthcare, Nestlé Health Science, Novo Nordisk, Lilly, Boehringer; support for attending meetings and/or travel from Fresenius Kabi, Lilly.

LB received grants from the Swiss National Foundation, Innosuisse, Novonordisk; consulting fees from Novonordisk, Ypsomed, Eli Lilly; honoraria for lectures, presentations from Dexcom, Eli Lilly; support for meeting from EASD. She is member of the EASD guideline panel, EASD teaching and education committee, president of the Swiss Association for the Study of Obesity and Metabolism (ASEMO), board member of the Swiss Society of Endocrinology and Diabetes, extended board member of the Swiss Society of Clinical Nutrition and Metabolism, Member of the Scientific Senate of the Swiss Academy of Clinical Sciences (SAMW); received material from Dexcom, Ypsomed.

RB received honoraria for lectures, presentations from Eli Lilly, Novo Nordisk; participated to advisory boards of Pfizer, Boehringer.

CB received honoraria for lectures, presentations from Fresenius Kabi; support for attending meetings and/or travel from Fresenius Kabi, Baxter, Sanofi.

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