# Progress in the Impact of Nutritional Intervention on Improving Elderly T2DM Patients With Muscle Atrophy

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**Abstract.** With the aging of the population, the number of elderly patients with Type 2 diabetes mellitus (T2DM) is increasing, and it often coexists with sarcopenia due to common pathological and physiological basis. In recent years, the pathogenesis of the combination of the two has been continuously explored, diagnostic methods have been continuously improved, and treatment plans have been expanded in both drug and non drug aspects. This study aims to explore the effectiveness of different nutritional interventions on elderly T2DM patients with muscle atrophy through clinical trials and observational studies, providing scientific basis for clinical practice. While implementing nutritional interventions, this study will focus on patient safety, evaluate the potential side effects of intervention measures, and ensure that patients do not face additional health risks during the treatment process. By studying the effects of nutritional interventions, this study aims to provide guidance and recommendations for clinical doctors, as well as a basis for policy makers, to promote the management and treatment of sarcopenia in elderly T2DM patients, improve their quality of life, increase muscle mass and function, reduce complications, and enhance their self-care ability.

**Keywords:** Type 2 diabetes mellitus; elderly patients; nutritional intervention.

## 1. Introduction

Type 2 diabetes mellitus (T2DM) is a type of diabetes, and its incidence is higher than T1DM. Most patients are elderly. The main physiological feature of T2DM is overexpression of blood glucose levels. As the condition progresses, various complications may arise, with the most common affected organs currently including the eyes, kidneys, and feet. In recent years, researchers have begun to focus on the population of elderly people with T2DM combined with sarcopenia. The reason why T2DM is prone to sarcopenia may be related to metabolic abnormalities and improper nutrient intake in the patient's body. Nutritional intervention is the most basic and effective preventive measure for T2DM complicated with muscle atrophy. For elderly T2DM patients, especially those with muscular dystrophy, their intake should be personalized according to their nutritional status, physical activity level, and health condition. Due to the causal relationship between T2DM and sarcopenia, which often coexist as comorbidities in the same person, it increases the likelihood of adverse events such as physical weakness, falls, disability, decreased quality of life, and increased risk of death in the elderly. According to the Clinical Nutrition and Guidelines for Geriatric Medicine of the European Society of Clinical Nutrition and Metabolism, elderly people consume approximately 30 kcal. Kg of energy per day. For patients with T2DM and sarcopenia, to avoid malnutrition, nutritional interventions should focus on protein or amino acids, which can help improve both T2DM and sarcopenia simultaneously. Especially when combined with increased protein intake and exercise, it can maintain the balance of the body's abilities, increase muscle mass and strength. According to the data of the International diabetes Alliance, 537 million people worldwide will be diagnosed with diabetes in 2024. 90% of these patients have T2DM, and the vast majority of elderly people among them suffer from sarcopenia. It is urgent to solve the problem of sarcopenia in elderly patients with T2DM. While reducing the pain of patients, it can also provide a more thorough and effective health management plan for this patient group. The goal is to have more research in the future to open up new perspectives and methods for the treatment and care of T2DM with sarcopenia.

## 2. The Pathogenesis of T2DM Combined with Sarcopenia

### 2.1. Insulin Resistance

Insulin resistance is the core pathological feature of T2DM and one of the important mechanisms underlying the onset of sarcopenia. Research has confirmed that the expression level of PGC-1 α in skeletal muscle of T2DM patients decreases in the early stage, and the biological oxidative function of adipocytes and skeletal muscle cell mitochondria is significantly reduced [1,2]. The secretion of adiponectin by visceral adipocytes in T2DM patients is significantly reduced, and the decrease in adiponectin leads to a decrease in the activation level of the AMPK/PGC-1 pathway in skeletal muscle [3]. Due to PGC-1 α being the most important transcription factor controlling mitochondrial production, the reduction of adiponectin downregulates the level of PGC-1 α, which may be the main cause of mitochondrial dysfunction. In summary, insulin mediated PI3K/protein kinase B may be an upstream signaling pathway generated by mitochondria. Reduced insulin function inactivates the signaling pathway, leading to decreased glucose uptake and utilization in muscles, which in turn affects muscle protein synthesis and metabolism. The resulting IR has a direct impact on mitochondrial function, confirming that mitochondrial dysfunction may be a result of IR in some cases. [4]

### 2.2. Inflammatory Response

The level of inflammatory response in T2DM patients is elevated, and inflammatory factors such as IL-6 and TNF-α can inhibit muscle protein synthesis and promote muscle breakdown. IL-6 is a multifunctional cytokine secreted by macrophages, endothelial cells, lymphocytes, and adipocytes, playing a central role in the body's response to various inflammatory stimuli [5, 6]. Studies have shown that elevated IL-6 levels in long-term hemodialysis patients are associated with malnutrition and affect changes in muscle related indicators within 3 years [7]. TNF-α is produced by various types of cells such as macrophages, neutrophils, and monocytes. Under normal circumstances, it has anti-infective, anti-tumor, and tissue repair promoting effects. However, sustained release can cause tissue damage through changes in inflammatory state [8-9]. TNF-α plays a decisive role between adipose tissue and skeletal muscle, and with abnormal muscle development, it can gradually cause muscle strength and function to disappear [10]. IL-6 and TNF-α are related to the occurrence of muscle atrophy in T2DM patients. As the levels of serum IL-6 and TNF-α increase, the risk of muscle atrophy in T2DM patients gradually increases. Analyzing the reasons, it may be due to the elevated levels of serum IL-6 and TNF-α, which can cause metabolic imbalance in muscle tissue synthesis, increase protein breakdown and metabolism, promote skeletal muscle fiber degeneration and protein content reduction, and destroy the body's muscle and muscle strength tolerance to fatigue. [11]

### 2.3. Oxidative Stress

Oxidative stress damages muscle cells, leading to a decrease in muscle mass, a decrease in antioxidant capacity in T2DM patients, an increase in oxidative stress levels, and an exacerbation of sarcopenia. The characteristics of oxidative stress are an increase in reactive oxygen species (ROS)/reactive nitrogen content and a decrease in antioxidant capacity. T2DM patients are in a long-term chronic hyperglycemia state, with increased levels of ROS in the body. High ROS levels promote protein degradation and reduce synthesis, affecting muscle repair and potentially leading to the occurrence of sarcopenia [12]. Satellite cells are an important type of skeletal muscle stem cells, and oxidative stress can damage their differentiation and DNA, inhibiting the process of muscle fiber regeneration. Mitochondrial dysfunction is closely related to aging and diabetes, and excessive ROS production will cause progressive damage to mitochondrial DNA [13].

### 2.4. Neurological Level

The research results of Kalyani et al. [14] suggest that in the late stage of T2DM, diabetes peripheral neuropathy may accelerate the decline of muscle strength, and that neuromuscular

dysfunction is an important mechanism of myopenia. Therefore, it is proposed that the prevalence of myopenia in T2DM patients with chronic complications such as renal insufficiency and peripheral neuropathy may be higher than that in diabetes patients without complications. The longer the course of diabetes, the higher the prevalence of its chronic complications, and the risk of myopenia will also increase. [15]

## 3. Nutritional Intervention for the Treatment of T2DM Combined with Sarcopenia

### 3.1. Optimize the Intake of Essential Substances

Adequate protein intake is an important means of preventing and treating sarcopenia. According to research, amino acids play a key role in promoting muscle protein synthesis and regulating blood sugar, not only reducing fasting blood glucose and triglyceride levels, but also improving insulin response [16]. Upper arm muscle circumference, serum albumin, hemoglobin, and MNA scores are commonly used to assess nutritional status. The reduction of basic metabolic rate directly affects the intake of nutrients. The decline of these indicators suggests that diabetes patients have a higher risk of myopenia. Malnutrition can lead to a decrease in muscle protein synthesis in elderly people, directly resulting in a significant decline in muscle mass and strength. Additionally, patients with malnutrition are at a higher risk of blood sugar fluctuations and hypoglycemia [17-18]. In addition, low hemoglobin level will also lead to local hypoxia of skeletal muscle, damage muscle strength and function [19], further increasing the risk of diabetes with myopenia. Carbohydrates are the key factors for blood sugar control in patients with diabetes. Elderly T2DM patients with sarcopenia should choose carbohydrates with low glycemic index, such as whole grains, legumes, and vegetables, to reduce postprandial blood glucose fluctuations and lower the risk of complications. Dietary and nutritional adjustments combined with conventional hypoglycemic therapy can more effectively reduce patients' blood glucose levels. It is recommended that the carbohydrate intake of elderly T2DM patients with muscle atrophy be controlled at around 250 g/d, which can quickly decompose and provide energy in the body. This not only ensures the daily energy needs, but also reduces the risk of hypoglycemia. A high proportion of fat in the diet can lead to insulin resistance, and adjusting it to around 20% is recommended to consume more fiber rich foods to improve blood sugar levels [20]. Reduce saturated fatty acids and increase intake of monounsaturated and polyunsaturated fatty acids. Unsaturated fatty acids enhance insulin sensitivity to glucose and amino acids, promote protein synthesis, inhibit skeletal muscle atrophy, and exert anti-inflammatory effects by blocking the arachidonic acid metabolism pathway, which is crucial for protecting and improving skeletal muscle quality. A cross-sectional study found a negative correlation between sarcopenia and intake of unsaturated fatty acids in elderly T2DM patients.

### 3.2. Supplement Intake of Trace Elements

Calcium is not only a key factor in regulating and maintaining skeletal muscle, but also participates in glycolysis metabolism and mitochondrial energy metabolism processes. Vitamin D helps with the uptake and regulation of calcium in muscle cells, as it stimulates the proliferation and differentiation of skeletal muscles, improving muscle mass and strength. Vitamin D deficiency is associated with weakness and sarcopenia [21]. Supplementing vitamin D combined with protein intake or physical exercise can improve lower limb muscle mass and function in elderly patients with sarcopenia [22]. Therefore, it is recommended to increase dietary protein intake and exercise while supplementing vitamin D for elderly diabetes patients with myopenia. Moderate supplementation of these two nutrients can effectively prevent muscle atrophy and is beneficial for T2DM patients. It is recommended that patients consume 1000-1200mg of calcium daily [23]. Iron dependent cell death is a new form of cell death characterized by iron homeostasis imbalance and accumulation of reactive oxygen species (ROS). The pathological process involves depletion of glutathione (GSH), decreased

activity of glutathione peroxidase 4 (GPX4), lipid peroxidation, and mitochondrial dysfunction. Research has shown that serum iron, ferritin, and transferrin saturation are positively correlated with T2DM disease, while transferrin is negatively correlated with T2DM disease, suggesting that iron homeostasis imbalance and iron overload are important factors leading to the deterioration of T2DM disease [25].

## 4. Evaluation of the Effectiveness of Nutritional Intervention

### 4.1. Detection of Physiological Indicators

#### 4.1.1. Muscle mass change

The change in muscle mass is the key to evaluating the effectiveness of interventions for sarcopenia. By using techniques such as bioelectrical impedance analysis (BIA) or dual energy X-ray absorptiometry (DXA), muscle mass can be accurately measured. BIA is a non-invasive and fully validated measurement of body composition, with the advantages of fast execution speed, ease of bedside application, no radiation exposure, low cost, and simple operation. BIA measured the impedance of low-intensity, fixed frequency alternating current through the human body. Of course, there are also drawbacks, as its measurement can be affected by water content, such as interference with muscle mass measurement when in an edematous state, as well as environmental temperature, recent physical activity, excessive dietary intake before the examination, and the position of electrodes during the examination process, all of which can affect its accuracy [26]. DXA is a standard diagnostic method for human body composition at the molecular level, based on a "three part" model: fat mass, muscle mass, and mineral salt content [27]. DXA has the advantage of low radiation, with a whole-body scanning radiation dose of less than 1mrem, making it a safer choice for repeated measurements of body composition [22].

#### 4.1.2. Functional Improvement

Functional improvement includes the enhancement of daily activity abilities, such as walking, going up and down stairs, etc. Through functional tests, such as the Time Up Walk (TUG) test, patients sit on chairs of appropriate height with armrests and are required to stand up independently, reach a distance of 3m as quickly and safely as possible, and then return to sit down again. The tester records the entire process to evaluate the patient's ability to move and balance their lower limbs safely when going out alone. The less time it takes, the better the recovery of lower limb function [28]. This can better evaluate the impact of nutritional interventions on the functionality of elderly patients and guide further intervention measures.

#### 4.1.3. Complications Risk

Nutritional intervention is also an important aspect of assessing the reduction of complications risk. Due to the prolonged and difficult to cure nature of T2DM, the disease course is long, and long-term exposure to high blood sugar stimulation can lead to kidney disease, lung infections, cardiovascular and cerebrovascular diseases, etc., exacerbating the physical and mental pain of patients and making them unwilling to eat normally; In addition, various bodily functions gradually decline with age, and patients are affected by diseases and various comorbidities or complications, which prevent them from eating normally through the mouth, leading to nutritional risks [29]. So it is necessary to track the occurrence of complications in patients for a long time in order to better evaluate the effectiveness of nutritional interventions in preventing complications.

### 4.2. Blood Sugar Control and Prevention of Complications

At present, the main treatment methods for T2DM patients with muscle atrophy who do not meet blood glucose control standards in clinical practice are lifestyle intervention, strengthening blood glucose monitoring, health education and self-management intervention, medication treatment, etc. When T2DM patients with sarcopenia cannot achieve ideal blood glucose control goals after lifestyle

interventions, doctors will add oral medication or inject insulin to help regulate their blood glucose levels. The commonly used oral hypoglycemic drugs are metformin, dapagliflozin, etc. Dapagliflozin is a common sodium glucose transporter 2 (SGLT2) inhibitor in clinical practice, which plays a crucial role in regulating glucose metabolism in the body. The clinical efficacy of Dapagliflozin is similar to that of dipeptidyl peptidase inhibitors, which can control blood sugar while reducing high glucose toxicity in T2DM patients, thereby inhibiting insulin secretion in the body and delaying the progression of T2DM with muscle atrophy to a certain extent.

Taking dapagliflozin as adjuvant therapy can effectively improve the control of blood glucose and inflammatory reactions in T2DM patients with muscle atrophy who do not meet blood glucose control standards, ensuring better control of the patient's condition. The reason why dapagliflozin can improve blood glucose regulation may be that dapagliflozin is an alpha glucosidase inhibitor, which can block the digestion and absorption of carbohydrates by inhibiting the activity of alpha glucosidase in the intestine. This slows down the rate of blood sugar rise and helps control fluctuations in blood sugar levels; Dapagliflozin can stimulate pancreatic beta cells to secrete more insulin. And insulin is an important hormone that can promote the uptake and utilization of glucose by cells, thereby reducing blood sugar levels. In addition, dapagliflozin can enhance blood glucose regulation by improving insulin resistance status. Insulin resistance is a common metabolic abnormality in T2DM patients. Dapagliflozin can reduce the degree of cell resistance to insulin through certain mechanisms, enhance the effectiveness of insulin, and thereby improve blood glucose regulation. The application of dapagliflozin may improve the control effect of inflammatory reactions for the following reasons: Firstly, dapagliflozin has antioxidant properties that can reduce the production of intracellular free radicals and lipid oxidation processes, thereby reducing the degree of inflammatory response. Inflammatory reactions are often accompanied by oxidative stress, and by reducing oxidative stress, dapagliflozin can help reduce the severity of inflammatory reactions.

Secondly, dapagliflozin may affect inflammatory responses by regulating the function of the immune system. This drug can inhibit the production of specific inflammatory factors, reduce the activity of inflammatory cells, and thus reduce the occurrence and progression of inflammatory reactions. Some literature suggests that this regulatory effect on immune function may help improve inflammation related disease states [30].

Finally, as a hypoglycemic drug, dapagliflozin can effectively lower blood sugar levels. High blood sugar levels can induce inflammatory reactions, and by controlling blood sugar levels, dapagliflozin can reduce the stimuli of inflammatory reactions in the body, thereby improving anti-inflammatory effects and promoting a better quality of life for patients.

In summary, the adjunctive treatment regimen of dapagliflozin can effectively improve blood glucose regulation and inflammatory response control in T2DM patients with muscle atrophy who do not meet blood glucose control standards, and help improve patient prognosis.

### 4.3. Quality of Life and Psychological Impact

#### 4.3.1. Exercise Guidance

After T2DM combined with sarcopenia, physical activity decreases and weight increases. Therefore, it is necessary to develop exercise prescriptions for patients and guide their implementation. Pocket information cards can be developed for patients, and patients can be guided to carry candy with them before exercise, with walking as the main form of exercise, and the amount of exercise should be based on slight sweating of the body, gradually and gradually, in order to achieve the true goal of auxiliary treatment. Through clinical observation of patients, it was found that there is a certain degree of improvement in their motor function, which can significantly improve their mood and sleep quality.

#### 4.3.2. Psychological Intervention

Inquire and talk about the life background and expectations of T2DM patients with sarcopenia, communicate with the patient's family members, receive their care and support, and guide the patient

to actively participate in activities within their abilities, strengthen connections with friends, eliminate negative emotions, and reduce loneliness; At the same time, relevant knowledge about diabetes and myopenia, diet control, exercise therapy, drug therapy, psychological support, and comfort will be explained to patients and their families, helping to relieve patients' negative emotions through methods such as relaxation training and music therapy. Correcting unhealthy lifestyle habits and behaviors, and cooperating with treatment with a positive attitude, have significant therapeutic effects on improving patients' symptoms.

#### 4.3.3. Social Support

Patients with T2DM and sarcopenia have a higher demand for medical services, occupy more medical resources, and incur higher medical expenses. It is difficult for patients to support the lifelong medical expenses on their own. Therefore, in addition to calling for support from family members, social organizations should also pay attention to patients with T2DM complicated with sarcopenia and solve practical problems for them. Relieve psychological burden. Encourage them to actively participate in governance.

## 5. Clinical Application Progress

T2DM has become a major non communicable disease affecting residents' quality of life and life expectancy due to its severe and complex acute and chronic complications, as well as its impact on multiple organ functions throughout the body. The overall prevalence rate of people aged 20 and above in China is about 9.7%, including 17.4% of people aged 45 and above, and 26.5% of people aged 80 and above. The impact of diabetes on China's medical and health system cannot be ignored. The damage of high blood sugar to large and micro blood vessels in the body has been widely recognized, and the interaction between skeletal muscle and glucose metabolism, as the main energy metabolism and exercise productivity organ in the whole body, has also received widespread attention from researchers. It was previously reported that the risk of myopenia in T2DM patients was 1.5~3 times higher than that in non-diabetes patients. The risk of new onset diabetes in patients over 75 years of age with myopenia is 1.5 times higher. The occurrence of sarcopenia increases the risk of disability such as falls and fractures in T2DM patients. Therefore, early identification of sarcopenia in T2DM patients and effective prevention and intervention are of great significance for improving their prognosis. Although sarcopenia has received widespread attention, there is still a lack of effective intervention measures, and its treatment mainly focuses on exercise prevention and improvement. Further clarification of the pathophysiological processes of T2DM and sarcopenia from a mechanistic perspective will be of great significance for their prevention and treatment.

In the pathogenesis of sarcopenia, the main pathophysiological processes are changes in muscle fiber types and functional decline. Skeletal muscle mainly consists of type I muscle fibers (slow twitch fibers) and type II muscle fibers (fast twitch fibers), each accounting for an average of 50%. When stimulated, the contraction power produced by type II fibers is 3 to 5 times that of type I fibers, and they play an important role in the generation and maintenance of muscle strength. As age increases, muscle fibers transition from type II to type I, leading to a decline in muscle function. Previous studies have suggested that sarcopenia is the result of a combination of factors such as changes in hormone levels, mitochondrial dysfunction, neuromuscular dysfunction, and malnutrition, leading to a decrease in skeletal muscle mass and reduced motor function such as contraction. As age increases, the levels and sensitivity of a series of hormones such as sex hormones, growth hormone, and insulin-like growth factor-1 in the body decrease, leading to changes in skeletal muscle strength. Due to the changing immune regulatory ability of the elderly population with age, the levels of circulating inflammatory factors also change accordingly. Research has found that inflammatory factors participate in the balance regulation between skeletal muscle protein synthesis and breakdown through multiple molecular pathways. In addition, research shows that diabetes patients are in a low level of noninfectious inflammatory state for a long time. Inflammatory factors such as tumor necrosis factor and C-reactive protein are elevated, and they participate in a series of pathological processes

such as insulin resistance. As an aging related disease, chronic inflammation plays an important role in the common pathogenesis of sarcopenia and T2DM. The NLRP3 inflammasome complex, composed of NOD like receptor family nucleotides binding to NLRP3, ASC, and caspase-1, can cleave intracellular IL-1β precursor at position 116 aspartic acid, forming activated mature IL-1β and participating in various immune inflammatory responses. The study found that the activation of IL-1β mediated by NLRP3 inflammasome is involved in the inflammatory lesions of insulin resistance.

The counts of neutrophils, lymphocytes, and platelets in routine laboratory analysis reflect the degree of inflammation in the body, while serum albumin reflects the nutritional status of the body. Recently defined new inflammatory complex indicators, including NLR, PLR, SII, PNI, It can also reflect the nutritional status of patients. Many studies have shown that NLR, PLR, SII and PNI have diagnostic, therapeutic and predictive roles in many diseases, such as diabetes, cancer and myopenia.

## 6. Conclusion

Through in-depth research in the previous section, it can be seen that nutritional intervention has a very positive impact on the development of sarcopenia in elderly T2DM patients. Reasonable supplementation of essential and trace elements, such as high-quality proteins like whey protein, can promote muscle protein synthesis, increase muscle mass and strength. Meanwhile, sufficient energy intake can maintain normal metabolism and muscle function in the body, while nutrients such as vitamin D and calcium also play an important synergistic role in improving musculoskeletal and insulin resistance health.

However, there are still many challenges and shortcomings at present. The nutritional intervention plan has not yet achieved precise personalization, and the nutritional needs differences between individuals have not been fully met. Further exploration is needed to optimize intervention formulas for the complex interaction mechanisms between nutrients. In the future, research should develop towards precision, utilizing advanced technologies such as genetic testing and metabolomics to develop personalized nutrition plans based on the individual patient's genetic background and metabolic characteristics. Further explore the potential of new nutrients or functional foods in improving muscle strength, insulin sensitivity, and other aspects, conduct multi center, large sample, long-term follow-up clinical studies to verify the long-term effectiveness and safety of nutritional interventions, provide more scientific and effective strategies for the prevention and treatment of sarcopenia in elderly T2DM patients, and improve the quality of life and health level of the elderly.

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