

Dietary Transition and Musculoskeletal Health in China: From Population-Level Patterns to Mechanism-Based Prevention

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Abstract. The national nutrition survey conducted in 2020 in China shows that despite the improvement in living standards, the musculoskeletal health of the older population is not improved. Instead, a diet that has been changed 30 years ago, switching whole grain, vegetables, and legumes for refined cereals and processed foods, and only marginally changing dairy and soy has resulted in a hidden burden of osteosarcopenia. Three mechanisms that converge from this dietary pattern to clinical disease were explored. Insulin resistance and advanced glycation end-product accumulation, brought about by repeated postprandial hyperglycemia, act to inhibit osteoblasts and to promote muscle protein breakdown. Chinese nutritionists refer to the lack of calcium, vitamin D and protein as "hidden hunger", and these deficiencies are known to keep NF- κ B signaling on, causing a degradation of both bone matrix and muscle satellite-cell pool. The loss of nutrient synergy then affects IGF-1/PI3K/Akt anabolic signaling, causing a combined loss in bone and muscle, instead of a stand-alone loss in muscle or bone. The message is, that musculoskeletal health in old age is not just about the diet at the adolescent age, but about cumulative diet from adolescence. In the article, a tiered approach to prevention is proposed, which includes achieving peak bone mass prior to 30 years with combined calcium-protein-vitamin D sufficiency, adopting the traditional Chinese nutrition model for older populations with anti-inflammatory changes, pulsed feeding, and resistance exercise, and optimizing nutrition delivery based on resource gaps in different settings.

Keywords: Dietary transition, Osteosarcopenia, Life-course nutrition, Refined carbohydrates, Tiered intervention

1. Introduction

China is rapidly becoming an old society with a growing life expectancy, which will be accompanied by a declining healthy life expectancy. The 2020 national survey revealed that the prevalence of osteoporosis is already one in five in adults older than 50 years and higher in women, as well as in adults over 65 years of age [1]. Meanwhile, low body weight and central obesity exist in the same population, and this is an indicator of increasing sarcopenic obesity. The overall global

osteosarcopenia prevalence stands at roughly one-fifth among the elderly. Risk determinants include female sex, advanced age, and prior fracture. However, these clinical trends are at odds with economic development. The Chinese diet has changed: plates that used to be filled with whole grains, vegetables and legumes are now filled with refined cereals and processed foods. Dairy and soy are still not part of the mainstream and less than 30% of older people achieve the recommended intake for protein and calcium [2].

At present, most studies have focused on single bone or muscle outcomes and individual nutrients as the exposure. Thus, the two above are avoided. First, cross-sectional designs do not cover all the nutrition-related damage that occurs over a person's life, such as the time from when peak bone mass is achieved in youth to subsequent years of muscle loss. Second, they have not been connected between general eating behaviour and the development of musculoskeletal diseases. Although ESPEN, the European Society for Clinical Nutrition and Metabolism, has issued life-course nutritional recommendations, they have failed to consider the special pattern of China's nutritional transition and have not defined the role of "hidden hunger" (simultaneous marginal deficiencies of calcium, vitamin D and protein) in osteosarcopenia [3].

Based on the 2020 Report on Nutrition and Chronic Diseases of Chinese Residents and in line with life course theory, this study explores the impact of dietary changes in China on musculoskeletal health for middle-aged and older adults through a structure-process-outcome pathway. Specifically, this paper wishes to determine whether macronutrient imbalance (high carbohydrate and low protein) and micronutrient dual deficiency independently and together contribute to musculoskeletal disorders, provide support for primary prevention through optimisation of adolescent bone reserves, and propose secondary prevention measures by modifying the diet of older adults. At the same time, this paper will also explore the way in which changes in diet promote musculoskeletal deterioration and identify prolonged inflammation as a potential link. Based on the above results, this study will put forward a tiered intervention plan for the Chinese food environment and offer context-specific references.

2. How nutritional imbalance damages musculoskeletal health: an attribution analysis

China has undergone different diet changes in the last 30 years at a rate unmatched by most populations anywhere else in the world. The traditional diet of whole grains, vegetables and legumes as a daily staple has been replaced by refined cereals, processed foods, and high fat animal products. The 2020 national survey records the musculoskeletal price of this change [1]. There is considerable inter-population variability in the prevalence of osteosarcopenia as reported in meta-analyses (ranging from 1.5% to 65.7%) and there is a strong positive correlation with scores on the Dietary Inflammatory Index (DII).

2.1. The carbohydrate problem

The new diet is largely made up of refined carbohydrates. They have a high glycemic index which causes the glucose to spike again and again. With time, the metabolic stress will have two effects: it will reduce the activity of osteoblasts and increase the release of cortisol; therefore, there will be more breakdown of muscle protein. Many large-scale studies have shown that people on a pro-inflammatory diet are more likely to develop sarcopenia, and they are about 50% more likely than those on an anti-inflammatory or neutral diet. A High-Inflammation Diet is associated with an elevated risk. The same applies to other groups in the study [4].

It should be pointed out that the problems of carbohydrates and proteins mentioned above are not separate; instead, they work together to increase metabolic stress. A high refined-carbohydrate diet can inhibit the formation of new bone and promote the degradation of muscle protein by causing postprandial hyperglycaemia; at the same time, the consumption of protein-rich foods will decrease, and micronutrient deficiencies will thus be exacerbated. Therefore, there is a deficiency of sufficient protein for anabolic processes, and at the same time, excessive carbohydrates lead to metabolic stress and further damage to the musculoskeletal system.

2.2. Protein and micronutrient gap

Changes in the diet have also been organised, and the amount of good-quality protein and micronutrients has decreased. Dairy and legumes have been absent from Chinese cuisine for a long time, and people are not getting enough calcium and protein in their diets now. Therefore, the reduction in IGF-1 (insulin-like growth factor-1) will decrease the speed of new bone and muscle formation [5]. According to the above research, an amount of 1.2 g/kg/day of protein is required to maintain muscle mass in older adults [6]. Pooled dose-response data further show that the lean-mass response is a saturation curve; for older adults, consuming about 1.2 grams of protein per kilogram of body weight each day appears to be sufficient to maintain muscle mass. It is 1.3 g/kg higher than the first. However, it does not provide additional benefits. Thus, the ideal range is between 1.2 and 1.5 g/kg [6]. Based on the above model, anabolism will occur most effectively within the 1.2-1.5g/kg/day range, and closer to the upper bound of this range, muscle-sparing effects are achieved [6].

2.3. The epidemiological signature

The above dietary patterns are shown in the population's health data. According to the 2020 National Survey in China, osteoporosis has been occurring more often among older adults, especially women. Sarcopenia is also age-relatedly acquired. And the two problems often go hand in hand; about half of the people with osteoporosis are also sarcopenic [1, 7]. Sarcopenia is also more pronounced in older age, and the two conditions frequently occur together; about half of the people with osteoporosis also show severe muscle loss [7]. At global level, patients with both conditions are 2.5 times more likely to fall, 3 times more likely to fracture and have significantly higher mortality. About one-half of patients with osteoporosis also suffer from sarcopenia. It is not just two diseases that occur together, it is two diseases that work together [7, 8].

2.4. Urban-rural divergence

The pattern is different from one setting to another. Urban older adults are at risk for high energy, low micronutrient density' diets, which have lots of energy but little nutrition. There is another limitation that rural older adults have: there are no nutrient dense foods available or affordable. Depleted protein intakes (<30% of recommendations) with high refined carbohydrate intakes create a non-linear increase in the risk for osteosarcopenia, demonstrating that these nutritional deficiencies do not add up but multiply.

3. Mechanisms at the molecular level of musculoskeletal stress caused by dietary structure transition

To appreciate the effects of the high-refined-carbohydrate and low-dairy-and-soy eating pattern on both bone and muscle, it is necessary to move beneath the surface of epidemiology to biology, where a shift from food quantity to food quality gives rise to a three-layered pathology (see Table 1).

3.1. Metabolic disruption

It is not neutral fuel and it is refined carbohydrates. They constantly test the insulin signalling system, which increases metabolic stress over and above the physiological slowing down that occurs with ageing. Chronic hyperinsulinemia caused by a high glucose load reduces osteoblast glucose uptake and utilisation, and favours bone resorption through an increase in the RANKL/OPG ratio. These advanced glycation end-products (AGEs) bind to the receptor RAGE on bone collagen and thus weaken bones. In muscle, they turn on the protein-degrading system, and thus muscles are lost more rapidly [7]. Given the diet of the Chinese people, too much consumption of refined staples such as rice and noodles in a high-carbohydrate diet can cause metabolic disorders. In Western countries, the main source of added sugars in processed snacks for added-sugar consumption is relatively small; however, for Chinese adults, most of their refined carbohydrate intake comes from polished white rice and wheat-based noodles that are eaten daily. This type of diet has kept bone and muscle tissue in a state of continuous postprandial high blood sugar for a long time, thus increasing the formation of AGEs and insulin resistance in a particular way under the East Asian nutritional system.

3.2. Inflammatory activation

Chronic marginal deficiency of calcium, vitamin D and protein is more than the lack of building blocks for tissues. It induces the NF- κ B pathway activation together with related inflammatory cascades which result in release of IL-6 and TNF- α cytokines. These mediators directly cause osteoblasts to be suppressed and to affect the functioning of the muscle satellite cells, causing a gradual depletion of musculoskeletal reserves over decades. At the molecular level, inflammatory factors inhibit Wnt/ β -catenin signaling, which is an essential axis for bone formation, and thus, unbalance bone remodeling [7]. But here, the protein source is important: animal-derived proteins offer superior anabolic stimulus owing to their complete essential amino acid profile, whereas plant-based options require strategic combining to achieve equivalent myofibrillar protein synthesis (e. g. legumes) [5, 6].

Table 1. Three converging pathways from dietary transition to musculoskeletal damage

Driving Level	Dietary Characteristics	Molecular/Physiological Mechanisms	Musculoskeletal Health Outcomes
First level: Metabolic disorders	High refined carbohydrates, high-GI diet	Recurrent postprandial hyperglycemia $\rightarrow$ insulin resistance $\rightarrow$ elevated RANKL/OPG; AGE accumulation disrupts collagen cross-linking	Suppressed osteoblasts, accelerated bone resorption; increased muscle protein degradation
Second level: Inflammatory activation	Chronic calcium–vitamin D–protein deficit	NF- κ B activation $\rightarrow$ IL-6, TNF- α release; Wnt/ β -catenin blockade	Imbalanced bone remodeling; impaired muscle satellite cell function

Table 1. (continued)

Third level: Synthetic blockade	Loss of nutrient synergy	Reduced IGF-1 + vitamin D deficiency → PI3K/Akt disruption	Simultaneous decline in bone density and muscle mass
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3.3. Disturbances of bone metabolic pathway signaling

The final failure mode is synergistic failure. Protein deficiency decreases the secretion of IGF-1 and vitamin D deficiency disrupts the uptake of calcium in the gut and the regulation of calcium within muscle cells. These deficits combine to cause a breakdown in the IGF-1/PI3K/Akt anabolic signaling pathway. Thus, supplementation of single nutrients that does not increase the overall diet pattern has usually not been able to stop the fact that bone density and muscle mass often goes down in parallel [5].

Under a life-course perspective, musculoskeletal disorders in the elderly are the crystallization of the nutritional debts that have been accumulated: insufficient peak bone mass in adolescence, progressive loss of muscle mass in adulthood and transformation of the early "hidden hunger" into a common condition in later life.

4. Tiered intervention strategy

Based on the above attribution analysis and understanding of how mechanisms are important, a tiered approach to interventions based on life stage and social circumstances is suggested. To shift from root cause mitigation in early life to functional maintenance in old age, and delivery adapted to the urban/rural realities.

4.1. Primary prevention: optimizing bone reserve before age 30

The critical period of time is before the closure of the critical window (the period of peak bone mass, approximately age 30). The goal is NOT just calcium, but rather a synergistic mix of calcium, protein and vitamin D, with ample calcium during adolescence known to significantly decrease adult fracture risk. Interventions at this stage should focus on dietary intake - increase dairy intake, increase soy products, increase vitamin D rich foods (fortified foods and egg yolk). Longitudinal studies have shown that adequate calcium, plus weight bearing exercise, during adolescence have a significant effect on peak bone mass and that this effect lasts into old age. The "high calcium, high protein" should be used as a benchmark for school nutrition programs and for family dietary recommendations to help minimize disease risk at the source.

4.2. Secondary prevention: dietary modification (age appropriate) of older people

At old age the metabolic environment and inflammatory baseline already have changed. The intervention objective shifts from "reserve" to "no loss and retain function". The emphasis should be on anti-inflammatory dietary changes: some substitution of red meat by fermented soy protein, such as sufu or natto, to provide high quality protein, probiotics and anti-inflammatory metabolites of fermentation; small frequent protein meals that mirror the pulsatile nature of muscle protein synthesis in older adults; and, under medical supervision, the inclusion of vitamin D and omega 3 to reduce inflammation.

Nutrition alone, however, cannot reverse sarcopenia. Nutritional intervention is a key component of the 2022 Asian Working Group for Sarcopenia (AWGS) expert agreement, which highlights that it needs to work hand-in-hand with resistance exercise to overcome anabolic resistance [9]. Dose response meta-analyses explain that protein alone is not enough. It is resistance training coupled with protein that gives rise to the benefits of protein. Studies show that when older adults do both—eat more protein and lift weights—they gain muscle strength. The benefit goes up with more protein, but only to a point. After that, extra protein does not add much [6]. A recent Chinese review also points out that osteoporosis and sarcopenia should be treated together, not separately. Nutrition and resistance exercise need to go hand in hand, and this combined approach should become standard care to lower the risk of falls and fractures [10]. A high-protein supplementation (1.2–1.5 g/kg/day) and resistance training should thus be the main approach to musculoskeletal health in older adults.

4.3. Flexible urban-rural adaptation

There is no one size fits all solution. Urban older adults are at risk of "high energy, low micronutrient density" – a focus is on decreasing refined carbohydrate proportion (e. g. partial replacement of refined grains with whole-grain alternatives) and structure imbalance. The other challenge for older adults in rural areas is the shortage of, and inability to afford, good protein. On this level, focus should be placed on small-scale household production of fermented soy products and policy subsidies need to be implemented in order to ensure access to fresh milk and eggs. The differentiated approach provides for interventions that are feasible and cost-effective in a variety of socioeconomic contexts.

5. Conclusion

Based on the nationally representative ecological data, this study proposes that musculoskeletal health issues of Chinese middle-aged and senior populations essentially bear the imprint of prolonged dietary shifts. By coupling macronutrient imbalance, micronutrient "dual deficit" and molecular inflammatory mechanisms, this reveals the triple pathways through which a "high refined carbohydrates, low dairy and legumes" dietary pattern drives musculoskeletal comorbidities. The bottom line is, that any pharmacological and/or supplemental interventions attempted later in life will not stop progression, unless the late-life dietary architecture is altered.

There are a number of directions that warrant future pursuit. First, China requires longitudinal cohort studies combining biomarker information (serum vitamin D, inflammatory cytokines) to explicitly quantify the whole "diet-blood-tissue" chain, which would help to address the limitations of causality that cross-sectional studies introduce. Second, it is clear that the digitalisation of the nutritional intervention is unavoidable: AI-powered analysis of each individual's diet image, coupled with body composition wearable monitoring, could allow for accurate prediction and personalised management of musculoskeletal risk. In conclusion, prevention policies should be shifted upstream to early life, prevention activities should be integrated in basic public health service indicators, and a "healthy kitchen" initiative should be launched to shift the traditional culture of Chinese food, by using cross-sectoral working in the sectors of agriculture, education and health. China must base the musculoskeletal health on the life-course pathway for dietary change in order to reduce the musculoskeletal crisis accompanying the ageing transition.

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