Dietetics After Spinal Cord Injury: Current Evidence and Future Perspectives

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Following spinal cord injury (SCI), individuals are at high risk for obesity and several chronic cardiometabolic disorders due to a deterioration in body composition, hypometabolic rate, and endometabolic dysregulation. Countermeasures to the consequences of an SCI include adopting a healthy diet that provides adequate nutrition to maintain good body habitus and cardiometabolic health. A proper diet for individuals with SCI should distribute carbohydrates, protein, and fat to optimize a lower energy intake requirement and should stress foods with low caloric yet high nutrient density. The purpose of this article is to present available evidence on how nutritional status after SCI should advance future research to further develop SCI-specific guidelines for total energy intake, as it relates to percent carbohydrates, protein, fat, and all vitamins and minerals, that take into consideration the adaptations after SCI. **Key words**: *caloric intake calories, carbohydrates, dietary intake, fat, macronutrients, micronutrients, nutrition, protein, spinal cord injury*

Introduction

Obesity, a worldwide public health concern, is a metabolic disorder characterized by the accumulation of adipose tissue.[1,2] Following a spinal cord injury (SCI), the changes in body composition and endometabolic milieu contribute to a significant decrease in total daily energy expenditure and often lead to an accumulation of adipose tissue, referred to as neurogenic obesity.[1-3] Neurogenic obesity is a known risk factor for type 2 diabetes mellitus,[4-7] dyslipidemia,[6,8-11] hypertension,[12,13] systemic inflammation,[1,14-17] and cardiometabolic syndrome (CMS).[6,15,17-22] The CMS reported in ~23% of the US adult population[23] is well below the prevalence in adults with SCI that ranges from 31% to 72%, depending on the number of possible risk factors included in the definition.[6,9,24] Regardless of the criteria used to identify CMS, addressing the component risk factors can be an effective approach to improving health. Although the component risk factors for CMS do not include physical deconditioning or excessive caloric intake, it is still considered a central factor in the development obesity and obesity-related comorbidities.[25-28]

The purpose of this article is to review the existing data on nutritional intake and status after SCI with a primary focus on persons with acute (<1 year since injury) and chronic (≥1 year since injury) SCI.[1] We describe current dietary intake profiles and our understanding of nutritional status as it relates to the advancement of future research.

Nutritional Status After SCI

Neurogenic obesity partly results from an imbalance between energy consumed and energy expended.[1] In a venerable work by Todhunter,[29] nutritional status was defined as a person's health as it is influenced by the intake and expenditure of nutrients. The Food and Agriculture Organization further defines nutritional status as "…the physiological state of an individual, which results from the relationship between nutrient intake and requirements and from the body's ability to digest, absorb and use these nutrients. The term malnutrition indicates a bad nutritional status."[30] According to the World Health Organization, malnutrition includes deficiencies, excesses, or imbalances in an individual's energy intake and/or nutrients.[31] Malnutrition is not only present with a cachexic state or condition, but it can coexist with obesity as undernutrition.[32] Worldwide, people are consuming food and drink that are more energy-dense, while engaging in sedentary activities and

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less exercise and physical activity. Nutritional deficiencies are multifactorial and common after SCI, such that nutritional status is dependent on several factors including dietary habits, food selection and availability, injury-induced changes, and comorbidities (e.g., hypertension, insulin resistance, etc.).

Dietary habits and food selection are influenced by environmental factors, including but not limited to transportation barriers to and from the grocery store, barriers within the stores (e.g., aisle width, shelving height and stability, access to refrigerated and frozen cases, etc.); difficulties preparing meals in kitchens constructed for the nondisabled population (e.g. counter, stovetop, refrigerator/freezer, and/or microwave heights); and meals prepared by caregivers, family, or friends (e.g., food provided as comfort). Culture (e.g., religion, ethnicity/race) and geographic location (e.g., available cuisine in local grocery stores and restaurants) are additional factors that may impact dietary habits and food selection. Healthy food choices are typically more expensive ($1.48/day) compared to eating an unhealthy diet,[33] harder to locate and access in grocery stores, and require preparation that individuals with SCI may have difficulty completing due to neurological impairment of the limbs, mobility restrictions, inaccessible kitchen appliances, and the need for assistance. The financial, access, and food preparation limitations may lead to a heavy reliance on store-bought, prepared, and/or precooked snacks and meals, some of which may have a high amount of simple sugars and saturated fat. Other factors, such as pain, education, psychosocial health, nutritional knowledge, socioeconomic status, and family and marital status, also influence dietary choices and therefore nutritional status.[34,35]

Neurogenic obesity is associated with significant sarcopenia, especially in the lower extremities.[36-38] Substantial muscle atrophy, especially that involving muscle contractile proteins, depletes the available protein resources needed for healing pressure injuries.[39] Poor nutrition status is an established risk factor for pressure injuries that have an SCI-specific global pooled prevalence of over 30% and an incidence of 2.2 person-years.[40-42] While total body protein needs may appear diminished in SCI, pressure injuries rapidly deplete the limited protein reserves as the body tries to heal the wound, generating a rapid transition to protein malnutrition. Prealbumin (also known as transthyretin) and albumin have historically been used as markers of protein nutrition and nutritional status, respectively, but fell into disfavor in 2012 when the Academy of Nutrition and Dietetics and the American Society for Parenteral and Enteral Nutrition discouraged their use as "sole" indicators of undernutrition due to their susceptibility to systemic inflammation.[43] Rather, this consensus panel on adult malnutrition recommended the identification of ≥2 of the following six characteristics for a malnutrition diagnosis: insufficient caloric intake, weight loss, loss of muscle mass, loss of subcutaneous tissue, localized or generalized fluid accumulation that may sometimes mask weight loss, and diminished functional status as measured by handgrip strength.[43] Nutrition-Focused Physical Exams are also encouraged and performed by a dietitian to assess for specific characteristics for malnutrition, such as loss of muscle/fat mass and edema. More recent international guidelines from Global Leadership Initiative on Malnutrition (GLIM) have adopted a combined approach that incorporates at least one phenotypic (nonvolitional weight loss, low body mass index, or reduced muscle mass) and one etiologic indicator (reduced food intake, assimilation or disease burden, or inflammatory condition).[44] Determination of reduced muscle mass in persons with SCI is best accomplished with dual-energy x-ray absorptiometry to yield fat-free mass; an index of fat-free mass <17 kg/m^2 for men or <15 kg/m^2 for women is considered "reduced muscle mass."[44] The GLIM has revitalized the utilization of prealbumin as a contributing element to monitor undernutrition in conjunction with C-reactive protein <15 mg/dL; above that level, prealbumin is not interpretable.[45-47] Prealbumin as a sole nutritional marker for malnutrition remains controversial; however, recent evidence suggests it can complement other parameters such as clinical history and anthropometrics to assess and monitor undernutrition.[48] The loss of metabolically active tissue below the level of injury, alterations in endometabolic physiology, environmental and physical barriers, and psychosocial factors further contribute to the suboptimal dietetics, poor body habitus, and CMS risk in persons with SCI.[49] Future research is needed to validate appropriate parameters of protein health established for nondisabled persons in individuals with SCI.

Dietary restriction is widely regarded to be a universal mechanism for prolonging lifespan, reducing obesity, and improving cardiometabolic health in several populations.[50] However, dietary restriction can result in the imbalance of macronutrients, thereby contributing to an already malnourished state after an SCI. To understand the implications of dietary restriction in persons with SCI, we must first assess their overall caloric intake and distinguish the percent total calories derived from carbohydrate, protein, and fat.

Total energy intake

The available literature indicates that energy intake in persons with SCI ranges from 1250 to 2112 kcal/day,[25,27,51-62] although this is contingent on the frequency and reporting accuracy of the dietary recall questionnaires used to asses caloric intake.[28,63] Several studies have reported that persons with SCI consume similar or fewer calories than the nondisabled population (1800 to 2600 kcal/day).[25,57,59,64] Nightingale et al.[60] presented evidence that the average energy intake in persons with chronic motor complete paraplegia was 1742 kcal/day. Groah et al.[25] and Sabour et al.[58] reported low energy intake for community-dwelling individuals with chronic tetraplegia compared to paraplegia. Barboriak et al.[62] noted similar findings among individuals with acute injuries. Farkas et al.[27] recently reported differences in caloric intake by level of injury where persons with chronic motor complete tetraplegia consumed a significantly greater amount of calories than persons with chronic motor complete paraplegia. The differences in the aforementioned studies may be because Farkas et al.[27] adjusted caloric intake to bodyweight, thereby accounting for body composition, which is significantly different by level of injury.[27,37,65,66]

Evidence comparing metabolic rate and total caloric intake demonstrates an excess of 300 to 500 kcal/day in persons with chronic SCI.[27,28,67] Farkas et al.[28] reported that pooled resting metabolic rate and total energy intake were 1492 kcal/day and 1876 kcal/day, respectively. Although the difference in energy need and consumption may at first seem insignificant, when sustained it will ultimately lead to the accumulation of body adipose tissue, resulting in adiposity and an increased risk of dyslipidemia, impaired glucose tolerance, insulin resistance, hypertension, and systemic inflammation.[1,3] Conversely, a reduction of approximately 500 kcal/day will result in gradual fat loss of approximately 1 pound/week that over time amounts to a significant loss of body fat (weight).

Macronutrient intake: Carbohydrate, protein, and fat

Carbohydrate intake

Several studies indicate that the consumption of fruits and vegetables is below the recommended intake for persons with SCI[51,68-70]; in their place, simple carbohydrates are frequently consumed.[58] Perret and Stoffel-Kurt,[71] Walters et al.,[57] and Edwards et al.[64] reported that carbohydrates made up a large percentage (44% to 53%) of the total energy intake in the SCI population. Groah et al.[25] demonstrated that men and women with paraplegia and men with tetraplegia consumed a greater amount of carbohydrates than the Centers for Disease Control and Prevention recommended daily value. Sabour et al.[58] identified that time since injury, education, and sex were significant predictors for carbohydrate intake in persons with SCI.[58] A recent meta-analysis reported that total carbohydrate ingestion in the chronic SCI population exceeded US Department of Agriculture (USDA) 2015-2020 dietary guidelines of 520 kcal/day by over 400 kcal/day.[28] Data from Farkas et al.[28] suggest that over 50% of total calories consumed by persons with chronic SCI may come from carbohydrates. The current USDA guidelines recommend that carbohydrates make up 45% to 65% of an individual's total daily calories when consuming 2000 kcal per day. Within the USDA guidelines, the recommendations do not factor in the anatomical and physiological changes that occur as a result of neurological injury and must be interpreted with caution for persons with SCI. Alternatively, Gorgey et al.[53] identified a standard diet protocol where dietary carbohydrates comprised 45% of the total daily calories. The authors suggested that this dietary composition may help maintain body composition and cardiometabolic profiles in persons with chronic SCI. This warrants further investigation.

With regard to fiber intake, several studies have identified that fiber intake in persons with SCI is low, independent of sex and injury characteristics.[25,26,28,57,58,71,72] Of note, Levine et al.[26] reported that dietary fiber in males with SCI was a *third less* than the average intake in the nondisabled population. Farkas et al.[28] quantified fiber intake in the population with chronic SCI as 17 g/day, a value that is below USDA guidelines (22 to 34 g/day), although above the Academy of Nutrition and Dietetics Evidence Analysis Library (ANDEAL) recommendations of 15 g/day.[73] ANDEAL also recommends increasing fiber consumption up to 30 g/day as tolerated following SCI; however, they declare that this is based on weak, conditional evidence.[73] Diets high in dietary fibers (>20 g/day) can create unfavorable changes in neurogenic bowel that do not occur in the nondisabled population, such as fecal impaction, chronic constipation, ununiform stool, and long transit time.[73,74]

Protein intake

With regard to protein, ingestion of this macronutrient for persons with SCI has been shown to be within, or exceed, the nondisabled recommended daily values.[25,26,28,51,53,57,58,60,69-71,75-77] Farkas et al.,[27] Gorgey et al.,[53] and Nightingale et al.[60] reported that 17% to 19% of the total daily energy intake came from protein for persons with SCI. Farkas et al.[27] demonstrated consumption of protein was 251 kcal/day (62.7 g/day) for persons with paraplegia and 286 kcal/day (71.5 g/day) for persons with tetraplegia; this represented 18% and 17% of their total daily caloric intake, respectively. In another study by Farkas et al.,[28] the authors determined protein intake for individuals with chronic SCI surpassed the USDA guidelines of 184 to 224 kcal/day and represented 17% of their total daily intake. According to ANDEAL recommendations, persons with SCI in the rehabilitation (acute and subacute) phase or community living (chronic) phase of their injury, need 0.8 g to 1.0 g of protein/kg of bodyweight per day for maintenance of protein status in the absence of pressure ulcers or infection.[73] Rodriguez et al.[78,79] reported that ingestion of 2 to 2.4 g of protein/kg of bodyweight per day was not enough to achieve a positive nitrogen balance (intake of nitrogen through protein consumption is greater than its excretion) following the acute SCI period. The authors noted that nitrogen balance was not altered, and 11 out of 12 persons with SCI surpassed the recommended allowance of 2g of protein/kg of ideal bodyweight per day.[79,80]

Fat intake

Studies have demonstrated that individuals with SCI ingest levels of dietary fat that approach or surpass recommended guidelines.[25-28,51,58,64,70-72,81,82] Several studies have reported that fat intake accounted for 34% to 40% of the ingested calories in persons with SCI, above the recommended amount of 30% but characteristic of a typical American diet.[26,51,53,60,70] Data from Farkas et al.[28] indicated that fat intake of persons with chronic SCI was 663 kcal/day, which was within the USDA recommended ranges of 400 to 875 kcal of fat/day. However, the review paper did not account for the USDA sex- and age-specific ranges for fat intake because of limited power. Saturated fat intake in persons with SCI is close to the limit or exceeds the recommended daily amount.[25,26,58,70,82] When comparing level of injury and sex, Groah et al.[25] reported saturated fat intake was greatest for a male tetraplegia group (~11.9%), followed by males with paraplegia (10.9%), females with paraplegia (9.9%), and one woman with tetraplegia (9.6%).[25] Multiple studies indicated that saturated fat consumption was higher in persons with SCI than the maximum of 10%, 7%, and 10% recommended by the National Cholesterol Education Program, American Heart Association, and USDA, respectively.[25,70,82] The recent Paralyzed Veterans of America Clinical Practice Guidelines on Identification and Management of Cardiometabolic Risk after SCI (PVA Guidelines)[18] further limit the recommended saturated fat intake for persons with SCI to 5% to 6% of total caloric intake due to body composition alterations after SCI and the mismatch in caloric intake and expenditure. The PVA Guidelines' limitation of saturated fat is justified given the significant reduction in energy expenditure and energy need after SCI.[2,28]

Alcohol intake

Available literature indicates that ingestion of alcohol is high among individuals with a new

SCI,[83] tetraplegia,[84] traumatic SCI,[85] and chronic SCI,[83,86] but many studies measuring it report low consumption.[25,28,60] Nightingale et al.[60] reported that 3% of the daily caloric intake came from alcohol in men with paraplegia. Groah et al.[25] reported that mean alcohol consumption was under 10 g/day (70 kcal/day) among persons with SCI but differed by sex (men: 6.43 g/day vs women: 2.24 g/day). Stigma can be associated with the consumption of alcohol and may result in participants underreporting the true amount. Additional research is needed to assess alcohol consumption in persons with SCI.

Future Perspectives

To date, much of the nutritional research following SCI is primarily concerned with the "status" of individual macronutrients, with proponents of carbohydrate, fat, and protein each taking center stage in the attempt to understand epidemic rates of obesity and CMS and dietary solutions for their etiology, treatment, and prevention. There is, however, an increasing amount of evidence that, rather than macronutrients acting individually, it is their interactive effects (their balance) that are more important for health and comorbidity prevention.[50,87,88] Research has emerged that the balance of protein to nonprotein (e.g., carbohydrates and fat) energy is significantly important in nutritional status, influencing total caloric intake, body habitus, gut microbes, function of the immune system, and obesity and CMS risk.[88] A change in the nutritional environment that dilutes dietary protein with carbohydrates and fat promotes overconsumption and fat storage, enhancing the risk for potential weight gain.[89,90] Targeting the ratio of protein to fat and protein to carbohydrate represents a novel dietary modification for many persons with SCI that warrants investigation to ameliorate rates of obesity and CMS.

Compared to their individual effects, combined diet and exercise is best to mitigate the adverse effects of neurogenic obesity and cardiometabolic dysfunction after SCI.[53] However, barriers limit exercise and physical activity in persons with SCI.[91] Given the barriers to exercise, research focused on dietary intervention may provide a large-scale "cure" to the cardiometabolic secondary complications that occur after SCI. Currently, limited data are available from randomized controlled trials assessing the effects of different dietary interventions, such as the Mediterranean or DASH (Dietary Approaches to Stop Hypertension) diets, on post-SCI physique and health.[18]

Conclusion

In summary, unhealthy diets and poor nutrition are among the top risk factors for obesity and diet-related comorbidities (e.g., type 2 diabetes mellitus, cardiovascular disease).[31] SCI is associated with alterations in body composition, endometabolic complications, and malnutrition that are also cause for severe comorbidities. Although authoritative guidelines for nondisabled individuals provide dietary recommendations, they are likely inappropriate (i.e., underestimated or overestimated) for individuals with SCI because of reduced metabolic requirements,[28,59,92-94] gut dysmotility,[95,96] and sympathetic nervous system dysfunction.[59,97] The PVA Guidelines are currently the strongest evidence-based dietary guidelines for individuals with SCI. Because several factors (injury characteristics, demographic parameters, accessibility, etc.) influence nutritional status after SCI, an emphasis should be placed on an individualized approach to nutrition assessment, diagnosis, intervention, monitoring, and evaluation. Future research should begin to explore the interactive effects of macronutrients rather than the absolute consumption of each macronutrient (i.e., nutrient intake) and how dietary intervention alone can improve rates of obesity and CMS in persons with SCI.

Conflicts of Interest

The authors declare no conflicts of interest.

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