Nutritional Health Considerations for Persons with Spinal Cord Injury

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Chronic spinal cord injury (SCI) often results in morbidity and mortality due to all-cause cardiovascular disease (CVD) and comorbid endocrine disorders. Several component risk factors for CVD, described as the *cardiometabolic syndrome* (CMS), are prevalent in SCI, with the individual risks of obesity and insulin resistance known to advance the disease prognosis to a greater extent than other established risks. Notably, adiposity and insulin resistance are attributed in large part to a commonly observed maladaptive dietary/nutritional profile. Although there are no evidence-based nutritional guidelines to address the CMS risk in SCI, contemporary treatment strategies advocate more comprehensive lifestyle management that includes sustained nutritional guidance as a necessary component for overall health management. This monograph describes factors in SCI that contribute to CMS risks, the current nutritional profile and its contribution to CMS risks, and effective treatment strategies including the adaptability of the Diabetes Prevention Program (DPP) to SCI. Establishing appropriate nutritional guidelines and recommendations will play an important role in addressing the CMS risks in SCI and preserving optimal long-term health.

Key words: cardiometabolic syndrome, nutrition, obesity, spinal cord injury, treatment strategies

Spinal cord injury (SCI) results in often permanent neurological deficits and high risk of accelerated morbidity and mortality¹ over the course of a lifetime. With advances in general medical practices,¹⁻⁴ there has been an increase in longevity with SCI^{5,6} and increased risk for developing chronically acquired diseases. In fact, several risk factors for all-cause cardiovascular disease (CVD) and comorbid endocrine disorders have become more widely identified, including component disorders of central obesity,⁷⁻¹¹ significant dyslipidemia,¹²⁻¹⁸ hypertension (depending on the extent and level of neurological injury), and insulin resistance^{4,19} (Table 1).^{20,21} The clustering of these risks – described as the *cardiometabolic syndrome* (CMS) – is also widely reported after SCI^{7,22-25} and is thought to worsen the CVD risk prognosis.²⁶ As such, CVD is a leading cause of mortality in chronic SCI.^{27,28}

Although each CMS constituent risk shares equal weight in determining the CMS diagnosis, the individual risks of obesity and insulin resistance are known to pose more substantial health hazards than the others.^{10,29-31} In fact, the

Table 1. Target levels for markers of cardiometabolic syndrome risk

Risk factor	Criterion (ATP III) ^a
Abdominal obesity	BMI ≥ 22 kg/m ² for persons with SCI ²⁰
Triglycerides	≥ 150 mg/dL
HDL-C	<40 mg/dL
Blood pressure	>140/90 mm Hg
Hyperglycemia	FPG ≥ 100 mg/dL
HOMA-IR	≥ 2.3 ²¹

Note: ATP III = Adult Treatment Panel III; BMI = body mass index; FPG = fasting plasma glucose; HDL-C = high-density lipoprotein cholesterol; HOMA-IR = homeostatic model assessment of insulin resistance; SCI = spinal cord injury.

^aNational Cholesterol Education Program and American Heart Association/National Heart, Lung, and Blood Institute.

health risk posed by obesity alone represents the sole chronic condition whose speed of growth and widespread dispersion resembles a pandemic of communicable diseases.^{32,33} A diagnosis of obesity is now considered an Americans with Disabilities Act (ADA)–qualified disability.³⁴ Although the development of CMS after SCI is complex,

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maladaptive dietary/nutritional status is foremost in its pathogenesis²²; this is a problem that presents in the early phases of SCI.^{10,35} This key factor impacts the metabolic milieu and contributes to CMS risks (discussed below).

Lifestyle modification, including changes in diet/nutrition, is a widely accepted recommendation for the treatment and prevention of CMS,^{10,35,36} and it targets the energy mismatch that leads to obesity. Although several studies in SCI have shown that exercise can reduce CMS risks of dyslipidemia and insulin resistance,³⁷⁻⁴² examination of the caloric deficit needed to reduce body fat suggests that exercise alone will not satisfy this need.^{22,43,44} The Academy of Nutrition and Dietetics has published nutrition practice guidelines that include the need for appropriate assessment and care for nutrition-related health concerns in SCI,⁴⁵ including nutritional intervention. Currently, there are no evidence-based nutritional guidelines to directly address the CMS risk in SCI, although the extent of risks and their unique impact on the population have led to a call for such recommendations (reviewed in refs. 22, 46, 47).

This monograph will discuss factors contributing to CMS risks in SCI and their effect on metabolic regulation, nutritional biochemistry, and feeding behavior. We describe the current understanding of the nutritional profile and its contribution to CMS risks, effective countermeasures for the pandemics of obesity, and barriers to optimal nutrition.

Factors in SCI Contributing to CMS Risk

Autonomic dysfunction

In examining the problem of obesity and endocrine-related diseases in SCI, it is likely that maladaptive metabolism associated with nutrient intake, albeit a discernable explanation, does not describe a unified cause. Homeostatic energy balance and metabolic regulation requires gastrointestinal (GI) signal integration and complex bidirectional communication between autonomic and neuro-endocrine systems. Modulation of this homeostatic feedback via higher order neural components, including cognitive and reward mechanisms, highlights the expansive

biological processes involved. There is limited information regarding GI neuraxis dysfunction in SCI, although recent preclinical reports provide evidence of alterations in peripheral tissues and central neuro-endocrine brain regions involved in GI signal integration and metabolic regulation.^{46,47} Investigations examining autonomic dysregulation after SCI^{48,49} call into question whether autonomic imbalance, interrupted neurotransmission within the “gut-brain” axis, or neuroendocrine alterations following injury⁵⁰⁻⁵² are among inciting causes for obesity.

While interrupted neurotransmission between the central nervous system (CNS) and sensorimotor targets is an accepted outcome of SCI, damage may also disrupt transmission of the gut-brain axis.^{53,54} The GI tract is under extensive autonomic regulation including intrinsic (enteric) and extrinsic (sympathetic/parasympathetic) components. Interactions between enteric reflexes, reflexes involving sympathetic ganglia, and reflexes that directly pass from the gut through the CNS provide physiological feedback contributing to regulation of energy balance and metabolism.⁵⁵ The enteric nervous system (ENS) responds to nutrient intake, luminal chemistry, and mechanical distortion.⁵⁶⁻⁵⁹ Sympathetic communication via the lower thoracic vertebral column (T5-T12)⁶⁰ and prevertebral ganglia⁶¹ innervate portions of the GI tract including the stomach and small and large intestine.⁶² This innervation targets the ENS, endocrine, and secretory cells,^{62,63} regulating motility, fluid exchange, and gut blood volume⁶¹ in response to nutrient ingestion and absorption. Parasympathetic vagal afferents represent the first direct link in gut-brain signaling,⁶⁴ informing on the quality and quantity of ingested nutrients.⁶⁵ Given the importance of an integrated nervous system in nutrient cycling and feeding, it is reasonable to suspect that these functions are interrupted when the central and peripheral nervous systems are dissociated following SCI.

Little is known about functional changes in this system after SCI, including whether it becomes dysregulated by interruption of spinal neurotransmission or whether dysregulation influences feeding behaviors. However, it is clearly known that endocrine cell types secrete messenger

molecules in the GI tract,^{57,66} underlying both short- and long-term feeding behavior by transmitting nutrient-related gastric and intestinal signals.^{67,68} For example, acylated Ghrelin originating from the fundic mucosa of the stomach stimulates appetite^{69,70} via endocrine mechanisms that act on hypothalamic and brainstem centers.^{67,71} The intestinal peptide cholecystokinin (CCK) released from entero-endocrine cells responds to luminal lipid and protein^{67,71-73} and acts on the hypothalamus and brainstem,⁷⁴ having a suppressive effect on food intake.^{73,75,76} Glucagon-like peptide-1 (GLP-1) released primarily from the distal GI tract in response to glucose⁷⁷ exerts sustained satiety effects on brain centers,^{71,72,78,79} evidence for long-term appetite regulation. Protein YY (PYY) signals through gastric vagal afferents⁸⁰ in response to nutrient ingestion⁸¹ exert satiety effects thought to be conveyed via hypothalamic neural pathways.⁸² These peptides, and other noted satiation peptides released from the pancreas (insulin) and adipose tissue (leptin),⁸³ highlight the robust appetite regulation mechanisms that contribute to central integration of feeding behavior.

Developing research indicates that dysfunctional reward processing is related to overconsumption of food⁸⁴⁻⁸⁶; there is evidence of disparities in reward functions occurring between nondisabled lean and obese people.⁸⁷⁻⁹⁰ Neural activity generated in response to visual food stimuli is exaggerated in leptin-deficient humans⁹¹ (associated with severe early onset of obesity)⁹² and with allelic variants of dopamine D2 or μ -opioid receptor.^{87,90,93,94} Deficient mesolimbic/dopamine signaling in obesity is implicated in altered anticipation and overconsumption of food.⁹⁵ In these cases, a reward deficiency is suggested as a mechanism explaining increased food intake to compensate for reduced dopaminergic signaling.⁹⁶⁻⁹⁸ When comparing lean and obese individuals in the fasted state, obese individuals have increased neural response to visual food cues within the orbitofrontal cortex and amygdala.^{99,100} Obese individuals also demonstrate increased postprandial neural response to visual food cues compared to lean individuals in corticolimbic regions¹⁰¹ and the ventromedial prefrontal cortex (which receives input from the amygdala) and may also have a disordered

response to satiation in obese humans.¹⁰² A recent comprehensive study on food reward signaling supports both increased sensitivity to food cues while fasted and decreased inhibitory control postprandial in overweight individuals.¹⁰³ Collectively, these data suggest that brain reward circuitry is involved in the pathophysiology of obesity. Unfortunately, there is no evidence concerning food reward signals that drive hunger or satiety following SCI. It is, however, understood that SCI causes characteristic cortical remodeling, including sensorimotor atrophy and functional reorganization.¹⁰⁴⁻¹⁰⁹ The extent of these variances, or whether they affect neurocircuitry involved in reward and/or feeding integration, has not been investigated. Understanding these novel aspects of GI and neuroendocrine dysregulation imposed by SCI is necessary to better develop timely clinical interventions that address maladaptive feeding responses and behaviors.

Musculoskeletal effects on energy balance and metabolism after SCI

It is important to note that, irrespective of dietary/nutritional complications, SCI incites persistent metabolic abnormalities^{5,19,23,110,111} that contribute to long-term effects on body systems^{112,113} and energy balance. Physical deconditioning and limitations related to movement, musculoskeletal activity, and weight-bearing result in deleterious changes in body composition, typified by rapid and long-term declines in metabolically active muscle mass¹¹⁴⁻¹²² and bone.¹²³⁻¹²⁹ In particular, loss of sublesional muscle mass is characterized by decreased cross-sectional area up to 48% as early as 6 weeks post SCI,¹¹⁹ and atrophy ranges of 30% to 60% have been reported, depending on muscle type and duration and completeness of injury.¹²⁸ This atrophy is associated with a significant muscle fiber-type transformation from slow oxidative type I to fast glycolytic type II,^{114,118,130-134} although both type I and type II atrophy contribute to overall muscular decline.^{120,135-137} This impaired skeletal muscle oxidative phenotype is linked to insulin resistance, glucose intolerance, and progression of CMS.^{138,139} In addition, compared to able-bodied controls, individuals with SCI exhibit infiltration of intramuscular fat^{7,122,140,141} and have greater

visceral adipose tissue (VAT) cross-sectional area,^{7,9-11} predictive of dyslipidemia, impaired glucose tolerance, and insulin resistance.^{23,142,143} These changes in tissue composition after SCI generate an environment that supports CMS risks.

Nutritional Profile of the SCI Population

Dietary contributions to maladaptive energy balance and overweight/obesity in SCI

Energy imbalance is a central factor in the development of overweight/obesity. With SCI, measured resting metabolism is 14% to 27% lower than able-bodied controls,¹⁴⁴⁻¹⁴⁹ explicable by marked reduction in fat-free mass and altered sympathetic nervous system (SNS) activity, particularly with higher neurologic injury. Studies comparing resting energy expenditure (REE) between able-bodied and SCI populations report on average 10% lower energy requirements for adults with chronic SCI (although this difference is reduced when REE is normalized to body weight).^{144,149-151} Average REE for adults with chronic SCI have been reported to range from 1,392 to 1,855 kcal/d for men,^{144,149,150,152-156} and 1,042 to 1,290 kcal/d for women.^{144,152} These values likely better represent caloric needs of persons with SCI than those published for the general population. Data comparing REE and energy intake indicate a surplus of ~300 to 500 kcal per day in SCI,^{22,157-159} which sustained, leads to weight gain and CMS risk. Although a recent study reports that a subset of the study group had caloric intake below their basal energy expenditure,¹⁶⁰ the authors note, "It is possible that the observed reduction in caloric intake... is due to underreporting of caloric intake in the food diaries."^(p1651) This is a common problem and noted study limitation. In addition, several large-scale studies in SCI^{10,36,161} report caloric intakes that are more closely aligned with the excess indicated here, suggesting that dietary habits have not changed drastically in recent years. These data highlight that inadequate dietary restriction contributes to a maladaptive energy balance and obesity risk.

The hypercaloric diet^{22,157-159} described above begins as early as during the acute hospitalization after injury.¹⁰ Prevalence rates for overweight and

obesity in persons with chronic SCI are concerning, ranging from 55% to 68% of the population.²⁹ Notably, obesity in individuals with barriers to physical activity is far more difficult to reverse than obesity occurring in persons without disability,^{8,10} and an overweight body habitus contributes to overall CVD risk to a greater extent than any other single component hazard.^{10,29} Moreover, overweight/obesity is alarmingly comorbid with glucose intolerance and insulin resistance,^{23,24,162,163} both strongly associated with a pro-inflammatory phenotype that further accelerates atherogenesis.¹⁶⁴ This risk clustering confers the same health risk as a clinical diagnosis of diabetes.³⁰

Lipid intake and dyslipidemia

The majority of studies discussing nutritional/dietary habits report that persons with SCI consume levels of fat that approach or exceed the current recommended level^{10,36,142,157,159,161,165-167} outlined by the United States Department of Agriculture (USDA) Dietary Guidelines for Americans 2015, American Heart Association (AHA), and World Health Organization (WHO) (Table 2). An early multicenter study,¹⁶⁵ including our group, reports that fat intake accounted for 37.9% of calories, well above the WHO recommended level of 30% but typical of the American diet, and the ratio of polyunsaturated to saturated fat was approximately half the recommended levels. Separate reports^{36,161,167} indicate that the mean percentages of energy from total and saturated fat range from 36.2 to 39.0 and 11.2 to 12.6, respectively. Groah et al¹⁰ report that irrespective of sex and level of injury, saturated fat intake was higher than the maximum recommended and was as high as ~12% in male tetraplegics. They determined that the obesogenic diet of persons with SCI has not changed over a 15-year timeframe and is still excessive in total and saturated fat. Notably, this fact is not SCI-specific, and it represents an issue that extends to the general population.

High fat intake is commonly associated with weight gain; in particular, high saturated fat intake appears to negatively affect metabolic profiles and chronic disease outcomes.^{168,169} We^{4,25,170,171} and others^{12,13,19,110,172-177} have written

Table 2. Studies reporting macronutrient intake in individuals with SCI that deviates from current United States and global recommendations and associated cardiometabolic syndrome (CMS) risk

Macronutrient	Study	Sample, N (male, female)	Recommended (% of total caloric intake)		Associated CMS risk
Total lipid %			USDA (2015)/AHA (20%-35%)	WHO (<30%)	
			Intake status		
	Levine et al ⁶⁵	33 (24, 9)	Avg above recommendation for males	Avg above recommendation for males	i. Obesity
			Avg above recommendation for females ^a	Avg above recommendation for females	ii. Dyslipidemia
	Aquilani et al ¹⁵⁷	10 (m)	Avg within recommendation	Avg above recommendation	
	Moussavi et al ¹⁶⁷	189 (145, 44)	Avg above recommendation for males	Avg above recommendation for males	
			Avg above recommendation for females	Avg above recommendation for females	
	Tomey et al ³⁶	95 (m)	Avg above recommendation	Avg above recommendation	
	Groah et al ¹⁰	73 (61, 12)	Avg above recommendation for males	Avg above recommendation for males	
			Avg above recommendation for females	Avg above recommendation for females	
	Walters et al ¹⁹⁹	77 (61, 16)	Avg within recommendation for males	Avg at the cut-score limit for males	
			Avg within recommendation for females	Avg within recommendation for females	
Saturated fat %	Perret & Stoffel-Kurt ¹⁵⁹	Acute SCI: 12 (9, 3)	Avg within recommendation for acute SCI ^a	Avg above recommendation for acute SCI	
		Chronic SCI: 12 (9, 3)	Avg above recommendation for chronic SCI	Avg above recommendation for chronic SCI	
	Sabour et al ¹⁶¹	162 (131, 31)	Avg above recommendation for males	Avg above recommendation for males	
			Avg above recommendation for females	Avg above recommendation for females	
			Recommended (% of total caloric intake)		
			USDA (2015)/AHA and WHO (<10%)		
			Intake status		
	Levine et al ⁶⁵	33 (24, 9)	Avg above recommendation for males	Avg above recommendation for males	i. Obesity
			Avg above recommendation for females	Avg above recommendation for females	ii. Dyslipidemia
	Moussavi et al ¹⁶⁷	189 (145, 44)	Avg above recommendation for males	Avg above recommendation for males	
			Avg above recommendation for females	Avg above recommendation for females	
	Tomey et al ³⁶	95 (m)	Avg above recommendation	Avg above recommendation	
	Groah et al ¹⁰	73 (61, 12)	Avg above recommendation for males	Avg above recommendation for males	
			Avg above recommendation for females ^a	Avg above recommendation for females ^a	
	Sabour et al ¹⁶¹	162 (131, 31)	Avg at the cut-score limit for males	Avg at the cut-score limit for males	
			Avg above recommendation for females	Avg above recommendation for females	

Macronutrient	Study	Sample, N (male, female)	Recommended (% of total caloric intake)	Associated CMS risk
Fiber (g/100 kCal)				
Recommended (g/100 kCal of total caloric intake)				
USDA (2015)/AHA (28–33 g)				
Intake status				
	Levine et al ⁶⁵	33 (24, 9)	Avg below recommendation for males	i. Dyslipidemia
			Avg below recommendation for females	
	Tomey et al ³⁶	95 (m)	Avg below recommendation	
	Groah et al ¹⁰	73 (61, 12)	Avg below recommendation for males	
			Avg below recommendation for females	
	Walters et al ⁹⁹	77 (61, 16)	Avg below recommendation for males	
			Avg below recommendation for females	
	Perret & Stoffel-Kurt ¹⁵⁹	Acute SCI: 12 (9, 3)	Avg below recommendation for acute	
		Chronic SCI: 12 (9, 3)	Avg below recommendation for chronic	
	Sabour et al ¹⁶¹	162 (131, 31)	Avg below recommendation for males	
			Avg below recommendation for females	
Recommended (% of total caloric intake)				
USDA (2015)/AHA (<10%)				
Simple carbohydrate (%)				
Intake status				
	Sabour et al ¹⁶¹	162 (131, 31)	Avg above recommendation for males	i. Hyperglycemia ii. Insulin resistance
			Avg above recommendation for females	

Note: AHA = American Heart Association; Avg = average; USDA = US Department of Agriculture; WHO = World Health Organization.

*Original article reports it as within recommendation but is different from current USDA (2015)/AHA and/or WHO recommendation.

extensively about a common state of dyslipidemia after SCI and its cardioendocrine risks. There is evidence for a direct relationship between high fat intake and serum triglycerides in persons with SCI.¹⁶⁷ Several studies report depressed high-density lipoprotein cholesterol (HDL-C) with SCI compared to able-bodied controls.^{140,178} Saturated fats are potentially atherogenic compounds, especially in high-risk people.^{161,167,179,180} Recent attention has focused on their relationship to blood levels of pro-inflammatory cytokines¹⁸¹⁻¹⁸⁵ as a unified forerunner of CVD¹⁸⁶⁻¹⁸⁹ and predictor of atherogenesis and cardiovascular events.¹⁹⁰ This pro-inflammatory phenotype has been documented after SCI by several laboratories.^{24,191-198} In most cases, testing of persons with SCI reveals elevated C-reactive protein (CRP), IL-6, soluble vascular adhesion molecule (sVCAM)-1, and endothelin-1.¹⁹⁸ Notably, the levels of these inflammatory products after SCI surpass evidence-based cut-scores for elevated CVD risk (eg, CRP >3 mg/dL).

Carbohydrate intake and glucose regulation

A recent review²² reports that carbohydrate intake after SCI appears to fall within recommended ranges of 45% to 55% total calories.^{10,142,159,199} However, when comparing sex and level of injury (paraplegia vs tetraplegia), Groah et al¹⁰ report carbohydrate intakes (g/d) were higher than recommended for all but the female paraplegia group. It is of greater health concern that most persons with SCI (a) are below the recommended intakes of fruit and vegetable^{36,200} and (b) have excess intake of simple carbohydrates¹⁶¹ (**Table 2**); both of these factors are strongly associated with unfavorable metabolic profiles.^{36,161,201} In fact, prospective studies in the able-bodied population suggest that the intake of fruit and vegetables is inversely related to the risks for developing diabetes,²⁰² CMS,²⁰³ and CVD.²⁰⁴ Given these data, it is not surprising that fiber intake is low in SCI^{36,157,159} even among women.^{10,161,165,199} Levine et al¹⁶⁵ and Tomey et al³⁶ report an average fiber intake of ~17.5 g (**Table 2**); when evaluated per 1,000 kcal, it is substantially less (~7.5-10g). This is of particular relevance as fiber consumption has been related to levels

of cardioprotective HDL-C²⁰⁵⁻²⁰⁸ and combating heart disease.²⁰⁹ Notwithstanding, high fiber diets (20–30 g/day) may stimulate undesirable changes in bowel function – related to increased mean transit time – that differ from the nondisabled population; this may render high fiber diets impractical for persons with SCI.^{199,210,211} Future studies will need to determine the best ratio and sources for simple and complex carbohydrates that satisfy specific nutrient needs after SCI.

In addition, the sublesional muscle atrophy with SCI significantly diminishes the primary storage site for carbohydrate, promoting increased circulating plasma glucose and risk of type 2 diabetes. As described above, physical deconditioning with SCI favors storage of glucose as fat, expansion of adipose tissue depots,¹⁴⁰ and infiltration of intramuscular fat.¹²² In fact, Elder et al²¹² found that intramuscular fat explained more than 70% of glucose intolerance following an oral glucose challenge, contributing to the increased prevalence of carbohydrate disorders in the SCI population compared to healthy controls.^{140,178}

Protein intake

In chronic SCI, several reports indicate that protein intake either meets or exceeds the current recommendations^{10,36} and is sufficient to maintain protein stores.¹⁴⁰ The considerable skeletal muscle atrophy during acute SCI is a significant predecessor to carbohydrate storage disorders²¹³ and metabolic abnormalities, as described above. In this regard, there is conjecture that greater acute-phase protein intake may be beneficial to offset these deleterious changes. However, earlier studies by Rodriguez et al^{111,214} report that protein intake (2.4 g/kg) significantly greater than that recommended (0.8-1.0 g/kg) was not able to alter this pathological consequence. The nutrition practice guidelines published by the Academy of Nutrition and Dietetics include basic protein recommendations for both acute and rehabilitation phases of SCI,⁴⁵ however the ratings are considered weak as evidence-based recommendations. Consequently, a more comprehensive analysis of the effects of early protein intake on chronic CMS risks will be necessary to better develop this understanding.

Nutrient deficiencies

Although research indicates that caloric intake of macronutrients is either adequate or in excess (except fiber), deficiencies are reported for several micronutrients in the SCI population and have been well summarized.^{22,215} Strong evidence affirms that vitamins A, B5, D, E, C, and biotin are underconsumed by persons with SCI and the essential minerals calcium, chloride, magnesium, and potassium are also reported below the daily recommended intake outlined by the USDA dietary guidelines.^{22,215} Each of these vitamin and mineral deficiencies are associated with either impaired glucose²¹⁶⁻²²² or lipid^{220,223,224} metabolism and, in the case of vitamin C²²⁵ and magnesium,²¹⁶ are implicated with existing atherosclerosis and diabetes (**Table 3**). Conversely, sodium (salt) intake by persons with SCI is much greater than the RDA (~1,500 mg) and generally exceeds the upper safe limit (2,300 mg)^{10,36,159,199} outlined by the AHA and other health organizations.^{226,227} These levels are established for the general population because of the positive relation of salt intake with high blood pressure,¹⁶⁶ a central CMS risk factor. Of relevance to SCI is the neurologic level of injury; persons with SCI at or above the spinal level (T6) of SNS outflow – resulting in functional sympathectomy – often suffer from hypotension.^{10,29,162,201,228-230} Therefore, in these individuals, there must be unique considerations when forming recommendations

for salt intake. Despite reports indicating that multivitamin/micronutrient supplementation is common in SCI,^{199,231,232} these unfavorable nutritional profiles persist and should be included as a part of nutritional recommendations.

Additional considerations

Taking a broader view of the complexities of SCI physiology, it is evident that optimal nutrition can play a key role in several outcomes beyond obesity and CMS risks. Nutritional support to meet nitrogen needs may reduce the deleterious effects of the catabolic, nitrogen-wasting process that begins in the acute and subacute phases in SCI (reviewed in ref. 235). As well, several preclinical studies have reported positive effects on functional outcome and histopathology with dietary restriction,²³³ creatine,^{234,235} and omega-3^{236,237} supplementation. Although promising, more comprehensive research is needed. The assessment of timing and appropriate composition of nutritional support to either enhance SCI recovery or offset secondary complications is necessary to establish appropriate dietary guidelines.

Dietary Countermeasures to CMS Risks in SCI

Current literature and recommendations

There are limited studies addressing an optimal diet and little advancement in defining dietary guidelines to combat CMS risks in SCI. Accepted recommendations are commonly in line with those for the general population (USDA 2015)²²⁶ (with exceptions for energy intake and sodium, as outlined above^{140,215}). Foremost, the assessment of energy balance is key to setting appropriate caloric targets for circumventing CMS risk (reviewed in ref. 217). To achieve caloric deficit, diets should reduce or eliminate primarily energy-dense food high in components associated with CMS risk, such as saturated fats, simple sugars, refined grains, and alcohol. Although USDA guidelines provide reference values for age and gender, these are understandably overestimated for persons with SCI given their lower metabolic requirements. Average REE values described above better represent

Table 3. Micronutrient deficiencies in SCI and associated metabolic abnormalities

Micronutrient	Associated metabolic abnormality
<i>Vitamin</i>	
A	Glucose metabolism
B5	Glucose and lipid metabolism
Biotin	Glucose metabolism
D	Glucose metabolism
E	Glucose metabolism
C	Lipid metabolism
<i>Mineral</i>	
Calcium	Lipid metabolism
Chloride	Glucose metabolism
Magnesium	Glucose and cholesterol metabolism
Potassium	Glucose and insulin metabolism

caloric needs and can be used as initial targets for caloric deficits. A few studies that adopt standard dietary recommendations report positive changes in lipid²³⁸ and metabolic²³⁹ profiles in chronic SCI, albeit limited in scope and of questionable value in altering disease trends.

Treatment strategies that include long-term compliance with diet and physical activity,^{240,241} blood pressure control,²⁴² correction of dyslipidemia,²⁴³ and glycemic control²⁴⁴ have shown promise in reversing CMS risks. Unfortunately, these changes are seldom long-lasting, and they rarely address so-called “hard end-points” that define disease. To address these limitations, it is generally accepted that effective countermeasures are enhanced when integrated as a multimodal strategy.²⁴⁵ One recent uncontrolled study²⁴⁶ in an SCI population reports weight loss and reduced body mass index (BMI) following 3 clinical consultations providing diet and exercise instruction. Also, Chen et al⁸ conducted a 12-week program of once-weekly education and exercise, followed by a 12-week diet prescribing specific caloric intake and emphasizing low-energy density foods. Findings show reduction of body mass by 2.9% and total fat mass by 2.2% although without a change in lean body mass. Study limitations included a predictable slowing (or reversal) of weight loss during an unsupervised postintervention phase, emphasizing the need for a more structured approach to compliance.

As such, more advanced recommendations incorporate a therapeutic lifestyle intervention (TLI) containing diet, exercise, patient education, and professional support, with refinement for SCI where applicable.^{35,215} The contemporary TLI strategy for SCI is modeled after the successes of the Diabetes Prevention Program (DPP),^{247,248} reporting both significant weight loss and a 58% decrease in the incidence of type 2 diabetes mellitus²⁴⁹ following TLI. The DPP target to achieve and maintain weight loss of 7% or more was based on epidemiological data correlating diabetes risk with increased levels of body weight and BMI.^{250,251} This proved to be achievable within the TLI framework, which was successful in maintaining a low diabetes rate onset as reported in a separately designed DPP outcome study (median follow-up of 5.7 years).²⁵²

Nutritional management

The dietary component of the DPP consisted of a 24-week energy-restricted Mediterranean-style diet. After considering baseline and physical activity energy expenditure,²⁵³ daily energy intake was reduced to achieve a caloric deficit of 500 to 1,000 kcal. This caloric reduction targeted a body mass reduction of .45 to .90 kg/wk and approximately 7% of baseline body mass after the 24-week intervention. Compared to low-fat diets, Mediterranean-style diets for duration of 3 months to 4 years result in significantly greater improvements in body weight,²⁵⁴⁻²⁵⁶ insulin sensitivity,^{254,257-259} CVD risk,^{254,255,259} endothelial function, and vascular inflammation²⁵⁵ in overweight/obese subjects at risk for or newly diagnosed with type 2 diabetes. A recent meta-analysis covering more than 1.5 million individuals followed over 3 to 18 years observed reduced overall mortality (9%), CVD mortality (9%), cancer mortality (6%), and incidence of Parkinson and Alzheimer diseases (13%).²⁶⁰ Of note, the Mediterranean-style diets are sometimes considered high in fat content and calories for sedentary populations, although the fats are generally monounsaturated and are not the more atherogenic saturated fats. This should be taken into advisement when ratifying and adapting principals for the needs in SCI. Also, because widely used predictors (eg, Harris-Benedict equation) may not be valid indicators for estimating caloric expenditure,²⁶¹ caloric intake should be matched to expenditure using metabolic calculations.²⁶²

Utilizing the DPP principals, we adapted the TLI to examine its mutability and efficacy in SCI (**Table 4**). We implemented a 6-month TLI incorporating a calorie-restrictive Mediterranean-style diet (1,200-2,000 kcal/d), 3-times weekly circuit resistance exercise,^{37,40,42} and 16 educational sessions with a lifestyle coach. Preliminary data include 7 men with chronic SCI who were obese and prediabetic by authoritative guidelines.³⁵ TLI lead to significant reduction in body mass, fasting plasma glucose, and glycated hemoglobin (%); these improvements reflected a change from prediabetic to low-risk/normal according to guidelines. In addition, we recently reported on a case series of participants with chronic paraplegia having

Table 4. Components of therapeutic lifestyle intervention for CMS risk in spinal cord injury

Component	Key points
Dietary	<i>Specific evidence for SCI is lacking. Recommendations should follow general guidelines EXCEPT for:</i>
	BMI targets
	Energy requirement estimates
	<i>Energy balance must be assessed for appropriately targeted caloric deficit:</i>
	Energy intake: Dietary analysis
	Total energy expenditure: BMR/REE; PA/Exercise; TEE
	<i>Establish caloric deficit:</i>
	Reduce energy intake
	Reduce calorie-dense foods (ie, saturated fats)
	<i>Diet recommendations: Mediterranean-style and general guidelines:</i>
Exercise	Increase whole grain, fruit, and vegetable intake
	Emphasis on mono-unsaturated fat sources
	Reduce saturated fats, cholesterol, and simple sugars
	<i>Malnutrition considerations:</i>
	Macronutrient overconsumption:
	Fats, cholesterol
	Simple sugars
	Micronutrient underconsumption:
	Vitamins: A, B5, Biotin, D, E, C
	Minerals: Ca ²⁺ , Cl ⁻ , Mg ²⁺ , K ⁺
Behavior	<i>DPP prescribes at least 150 min/wk of moderate-intensity physical activity:</i>
	700 kcal activity target
	Achievable and beneficial in preventing diabetes
	<i>Circuit resistance training (CRT) shown to improve CMS risk in SCI^{31,34,36} and accepted as guideline by the APTA:</i>
	Satisfies DPP recommended weekly activity and caloric expenditure
	Satisfies WHO physical activity recommendations for adults (18-64 years)
	RHR/RPE taken before start
	2-4 min warm up: Arm cycle-ergometry set at low resistance (aerobic component)
	Consists of 6 resistive exercises:
	Bicep curl, lat pulldown, chest fly, posterior row, triceps extension, shoulder press
Behavior	Paired training design:
	Two resistance maneuvers
	Interposed aerobic exercise
	Complete circuit is repeated 3 times (1 training session)
	CRT was done 3 days/week for 24 weeks
Behavior	<i>Structured behavioral modification therapy adapted from the DPP 16-session protocol including education, problem-solving skills training, and cognitive restructuring:</i>
	Education/instruction on diet and exercise components and role in reducing CMS risk
	Self-monitoring of body weight, caloric intake, and exercise level
	Understanding psychosocial barriers to diet and exercise goals and developing cognitive strategies to overcome them

Note: APTA = American Physical Therapy Association; BMI = body mass index; BMR/REE = basal metabolic rate/resting energy expenditure; CMS = cardiometabolic syndrome; DPP = Diabetes Prevention Program; RHR/RPE = resting heart rate/rate of perceived exertion; TEE = total energy expenditure; WHO = World Health Organization.

major risk factors for CMS.²⁶³ TLI resulted in body mass reduction that exceeded the DPP criterion (7%), and demonstrated improvements in insulin resistance and lipid profile, particularly HDL-C and triglycerides; these risk factors are distinct threats in SCI.^{12-14,16,19,264} These preliminary data provide evidence that TLI can be effectively adapted for SCI.

Barriers

An important aspect of CMS prevention guidelines as outlined by the ACC/AHA²⁶⁵ is the “use of behavioral strategies to facilitate adherence”^(p37) to weight management recommendations. Recent reports have focused on barriers to behavioral strategies in SCI,^{22,215,266} addressing physical and environmental challenges, and inadequacies in education and psychosocial support as relevant contributors to imprudent lifestyle choices. As we better understand these challenges, it is important to integrate directed behavior change that can

positively influence nutrition and diet to reduce CMS complications in SCI.

Summary and Conclusions

Health risks associated with obesity and cardioendocrine disease are pervasive in people with chronic SCI. Lockstep programs of lifestyle intervention that incorporate sustained nutritional guidance, moderate-intensity exercise conditioning, and structured behavioral retraining represent the best approach to weight control and disease prevention. Nutrition is a key lifestyle factor and component of a successful TLI for disease management. In the future, accepted nutritional guidelines to countermeasure CMS risks could be adopted for CMS risk prevention during the early phases after injury. Nutrition as a preventative measure for disease risk is often overlooked and may be of greater utility in a population where reversing disease risks is difficult.

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