Energy expenditure and nutrient intake after spinal cord injury: a comprehensive review and practical recommendations

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Abstract

Many persons with spinal cord injury (SCI) have one or more preventable chronic diseases related to excessive energetic intake and poor eating patterns. Appropriate nutrient consumption relative to need becomes a concern despite authoritative dietary recommendations from around the world. These recommendations were developed for the non-disabled population and do not account for the injury-induced changes in body composition, hypometabolic rate, hormonal dysregulation and nutrition status after SCI. Because evidence-based dietary reference intake values for SCI do not exist, ensuring appropriate consumption of macronutrient and micronutrients for their energy requirements becomes a challenge. In this compressive review, we briefly evaluate aspects of energy balance and appetite control relative to SCI. We report on the evidence regarding energy expenditure, nutrient intake and their relationship after SCI. We compare these data with several established nutritional guidelines from American Heart Association, Australian Dietary Guidelines, Dietary Guidelines for Americans, Institute of Medicine Dietary Reference Intake, Public Health England Government Dietary Recommendations, WHO Healthy Diet and the Paralyzed Veterans of America (PVA) Clinical Practice Guidelines. We also provide practical assessment and nutritional recommendations to facilitate a healthy *dietary pattern* after SCI. Because of a lack of strong SCI research, there are currently limited dietary recommendations outside of the PVA guidelines that capture the unique nutrient needs after SCI. Future multicentre clinical trials are needed to develop comprehensive, evidence-based dietary reference values specific for persons with SCI across the care continuum that rely on accurate, individual assessment of energy need.

Key words: **Spinal cord injury: Energy expenditure: Energy intake: energetic intake: Macronutrients: Micronutrients: Carbohydrates: Protein: Fat: Alcohol: Fiber: Vitamins: Minerals: Nutrition: Diet**

A spinal cord injury (SCI) results from trauma to or disease of the spinal cord, often causing permanent neurological deficits and accelerated morbidity and mortality throughout the lifespan[1,2]. Depending on level and completeness of injury, SCI is associated with a range of co-morbidities that can limit functional independence, mobility and nutrient utilisation. These co-morbidities include motor paralysis, sensory loss, neurogenic restrictive and obstructive pulmonary disease, neurogenic bradycardia, neurogenic hypotension, sympathetic dysfunction, neurogenic adaptive myocardial atrophy, coronary artery disease, anabolic deficiency, spasticity, sarcopenia, heterotopic ossification, osteoporosis, upper extremity overuse, neurogenic obesity, cardiometabolic syndrome (CMS; including, dyslipidemia, hypertension and type 2 diabetes mellitus), pressure injuries, sexual dysfunction, and neurogenic bowel and neurogenic bladder[3].

In the acute phase of SCI, spinal shock often occurs in which all motor and sympathetic reflex activity is absent[4]. The patient is often mechanically ventilated in the acute phase, such that even muscles of respiration are inactive[5]. Basal/resting metabolism plummets as the body sheds unneeded paralysed muscle and bone, and nutrient needs are reduced[6]. Once weaned from a ventilator, the individual will at least be able to activate muscles of respiration which may marginally increase total daily energy expenditure (TDEE) as they contract. During this time, the body will continue to lose unused muscle and bone until a

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Abbreviations: ADG, Australian Dietary Guidelines; AHA, American Heart Association; CMS, cardiometabolic syndrome; DGA, Dietary Guidelines for American; IOM, Institute of Medicine; LOI, level of injury; PHE, Public Health England; PVA, Paralyzed Veterans of America; SCI, spinal cord injury; TEF, thermic effect of food digestion; TEPA, thermic effect of physical activity.

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homoeostasis is attained with minimal muscle protein reserve and bone mineral content reduced to fracture threshold[5,6]. The acute phase may last up to 12 months as the individual completes physical rehabilitation and reintegrates into the community with a new baseline functional level, and subsequent energy and nutrient 'setpoint', that falls well below previous baseline levels[5–8]. The new setpoint is rarely matched by a similar reduction in energetic intake[9]. Overeating relative to energetic need and poor dietary habits (e.g. overeating, consuming sugary drink, etc.) contribute to inadequate nutrition and chronic health problems in the population with SCI[10,11]. Therefore, ensuring the appropriate consumption of macronutrients and micronutrients relative to need becomes a challenge despite several dietary recommendations.

Authoritative guidelines provide evidence-based dietary recommendations. These guidelines are used to establish goals in planning healthy diets and lifestyles and provide the public information about nutritional science and a wholesome diet. The US Department of Agricultural (USDA) Dietary Guidelines for Americans (DGA)[12], Public Health England (PHE) Dietary Recommendations[13], Australian Dietary Guidelines (ADG)[14] and Institute of Medicine (IOM) Dietary Reference Intakes[15] are meant for use by healthy populations to meet nutritional needs and maintain an overall healthy diet and lifestyle. Both the WHO Healthy Diet[16] and the American Heart Association (AHA)[17,18] primarily focus on the prevention of obesity and chronic disease and, in the case of the AHA, reducing the risk of cardiovascular disease. Food and estimated average requirements, recommended dietary allowance, adequate intakes and tolerable upper-level intake (definitions are provided in Table 1) recommendations from these organisations are often harmonised. However, they differ in their methodologies, ratings of evidence, geographic location of the population of interest, references and ease of translation. A fundamental shortcoming of the guidelines is their translation to persons with life-changing injuries and/or those who have developed chronic health conditions that require special dietary modifications and considerations, such as those with a SCI. Persons with special needs are typically excluded from consideration when designing these guidelines.

The overall purpose of this narrative review is to (1) critically appraise dietary intake relative to energy needs after SCI and (2) compare the existing literature with authoritative dietary guidelines from several countries that target obesity and cardiometabolic risk reduction. We extend the findings from our published meta-analysis and systematic review[19] that determined greater energetic intake relative to energy expenditure and an imbalance in fibre and micronutrient intake compared with the DGA in chronic SCI. In the present paper, we specifically aim to review both the acute (< 1-year post-injury) and chronic (≥ 1-year post-injury) phases of a SCI, include a wider collection of dietary and energy literature in SCI, critically evaluate energy expenditure and dietary intake assessment methods, incorporate dietary guidelines outside the US, and provide practical assessment and nutritional recommendations to facilitate a healthy dietary pattern after SCI. We also highlight neurogenic obesity and cardiometabolic risk after SCI, explore the potential influence of SCI on the central and peripheral mechanisms regulating energy homoeostasis and provide direction for future research by comparing existing literature on persons with and without SCI.

Neurogenic obesity and cardiometabolic risk after spinal cord injury

The prevalence of neurogenic obesity[7,20] in adults with SCI ranges from 22 % to 97 %, compared with 42 % in the non-disabled population[5,11,21–29]. Neurogenic obesity results from the dysfunction of energy metabolism, physical deconditioning[30], a sedentary lifestyle[31], impaired fitness[32], sympathetic nervous system dysfunction[33,34], altered hormonal homoeostasis[5,35–40], changes in satiety[41] and loss of lean body mass after SCI (Table 2)[42–47]. The volume of marrow fat increases 36 % following the initial 12 weeks after the injury in part, because increases in fat mass are dissociated from obesity-related mechanical loading[48]. Following the SCI, bone loss is prompt[49], with bone mineral density at the knee and hip declining 2 to 4 % every month[49,50] and decreasing up to roughly 20 %[51,52] within the first year of the injury. Precipitous loss of skeletal muscle mass below the level of injury (LOI) is marked by decreased cross-sectional area of up to 48 % as immediate as 6 weeks after the injury[45]. Muscle atrophy from 30 to 60 % of total lean body mass has also been reported[53]. Significant gains in fat mass occurring 2 to 7 months post-SCI contribute to a pathological cardiometabolic profile observed in the chronic phase of the injury[54].

The accumulation of visceral fat is considered the principle mediator in the development of dyslipidemia, insulin resistance, hypertension, arteriosclerosis and CMS in the non-disabled population[55–57]. Similar risk factors are used to quantify CMS in persons with SCI (Table 2)[7,58–64]. Cirnigliaro *et al.*[65] reported that, when compared with a non-disabled group, persons with SCI had a 27 % increase in visceral fat volume for every centimetre

Table 1. Definition of terms relating to dietary reference intakes

Adequate intake (AI)	The recommended average daily intake is based on observed or experimentally determined approximations of estimates of nutrient intake by a group(s) of apparently healthy individuals that are *assumed* to be adequate. AI is used when an RDA cannot be determined.
Estimated average requirement (EAR)	The average daily nutrient intake level is estimated to meet the requirement of half the healthy individuals in a particular life stage and sex group.
Recommended Dietary Allowance (RDA)	The average daily dietary nutrient intake level sufficient to meet the nutrient requirement of nearly all healthy individuals in a particular life stage and sex group. Developed from EAR.
Upper limit (UL)	The highest average daily nutrient intake is expected to pose no adverse health risks to almost all persons in the general population. As intake exceeds the upper limit, the potential for adverse health risks may increase.

Adapted from US Department of Agricultural (USDA) Dietary Guidelines for Americans (DGA)[1] and Institute of Medicine (IOM) Dietary Reference Intakes[2].

Table 2. Factors contributing to neurogenic obesity and cardiometabolic syndrome after spinal cord injury (SCI)

Neurogenic obesity	Cardiometabolic syndrome
• Physical decondition	General health risks
• Reduction in lean body mass	• Age
• Obligatory sarcopenia	• Family history
• Mechanical unloading	• Sex
• Blunted anabolic hormones	• Hypertension
• Inactivity	• Hypercholesterolemia
• Limited range of motion	• Type 2 diabetes
• Decreased energy expenditure	• Smoking/tobacco use
• Decreased BMR/RMR	Cardiometabolic syndrome risk factors
• Altered satiety	• Abdominal obesity/visceral adiposity
• Excess energetic intake	• Insulin resistance/type 2 diabetes
• Impaired fitness	• Hypertension
• Genetic predisposition	• Hypertriacylglycerolaemia
	• Low HDL-cholesterol
	Non-traditional risk factors
	• Genetics
	• Prothrombic state
	• Proatherogenic state
	• Malnutrition
	• Excess energetic intake
	• Chronic, low-grade inflammation
	SCI-specific risk factors
	• Sympathetic nervous system dysfunction
	• Physical deconditioning
	• Neurogenic obesity and its causes

Adapted from Farkas and Gater[7], Gater *et al.*[5] and Nash *et al.*[8].

increase in waist circumference, a marker of central obesity, and a 20 % increase in visceral fat volume for every unit increase in body mass index (BMI). Nash *et al.*[66] identified that being overweight/obese was significantly associated with CMS diagnosis. In a sample of 477 veterans with SCI, Gater *et al.*[11] reported that 76·7 % were classified as obese when using an SCI-specific BMI cut-off of 22 kg/m²[67]. The authors also reported that 55·1 % had or were undergoing treatment for hypertension; approximately 50 % currently had or were previously diagnosed with type 2 diabetes mellitus; 69·7 % had or were under treatment for HDL-cholesterol < 40 mg/dl; and more than 57 % had CMS using modified International Diabetes Federation criteria[68]. More recently, Gater and colleagues[29] studied body composition using the gold standard four-compartment model and CMS in a sample of seventy-two participants with chronic motor complete SCI. The authors identified a mean BMI of 27·3 kg/m² corresponding to 42 % body fat and CMS was in 59·4 % of the sample[29]. These findings demonstrate the high prevalence of neurogenic obesity and cardiometabolic complications after SCI and the need for dietary countermeasures (Table 2).

Central and peripheral mechanisms regulating energy homoeostasis and their implications in spinal cord injury

The central nervous system plays a vital role in modulating energy status, and the hypothalamus is the integrating, superordinate principal regulator of whole-body energy homoeostasis (Fig. 1). The arcuate nucleus within the hypothalamus plays a critical role in the regulation of feeding and metabolism. It integrates hormonal and nutritional signals from the peripheral circulation, as well as peripheral and central neuronal inputs, to generate a coordinated feedback response. The arcuate nucleus projects to second-order neurons in the paraventricular, dorsomedial, lateral and ventromedial nuclei of the hypothalamus. The second-order neurons further process the received information and project to multiple extrahypothalamic neurocircuits, leading to an integrated response that regulates energy intake and energy expenditure[69]. These centres jointly summate influences from various circulating substrates, hormones, neuropeptides and neurotransmitter signals that regulate food intake.

Gastrointestinal hormones also have a principal role in regulating central nervous system-dependent energy control. Ghrelin is mainly secreted from the stomach during a fasted state and stimulates body weight gain, adiposity and central feeding centres by activating neurons in the hypothalamus that stimulate food intake[69]. Various other hormones, such as peptide YY_{3-36}, cholecystokinin and glucagon-like peptide 1 are secreted from the small intestine upon the ingestion of foodstuff and exert appetite-suppressing effects in various brain regions, such as hypothalamic and brainstem nuclei, and by modulating vagal afferents, the peripheral elements of the brain–gut axis (Fig. 1)[70]. Gut peptides provide information on 'real-time' food consumption and modify electrical activity of the vagal afferent pathway by attaching to vagal receptors that extend into the digestive tract mucosa. These intestinal-derived signals are sent by the vagus nerve to the nucleus of the solitary tract, with further projection to hypothalamic regions (Fig. 1)[71].

Disruption of the central mechanisms modulating energy metabolism has been previously recognised as the aetiology of obesity in non-disabled persons[69,71]. Obesity and cardiometabolic disorders are frequently associated with diminished production or resistance to the production of central and peripheral regulators of energy homoeostasis, including food intake and energy expenditure[72]. Naznin *et al.*[73] and Waise *et al.*[74] reported that obesity-induced systemic inflammation spreads to the vagus nerve and subsequently the hypothalamus leading to the dysregulation of central and peripheral mechanisms governing satiety, energy regulation and fuel metabolism. With obesity and high-fat, energy-dense diets, increases in the concentration of saturated fatty acids from the periphery cross the blood–brain barrier and induce an inflammatory response on hypothalamic neurons[75]. Vinik *et al.*[76] reported that obesity and type 2 diabetes mellitus-induced neuropathies alter vagal nerve neurotransmission, preventing bidirectional crosstalk between the central nervous system and the gut.

Although supraspinal centres remain intact following SCI, several neurological and endometabolic factors are influenced by the disruption of the central nervous system. With an SCI, compromised afferent and efferent signals from central and peripheral locations lead to dysregulation of the intricate equilibrium of energy metabolism. Physiological cues that are present in persons with SCI and influence appetite, satiety/satiation and energy balance are disrupted, further contributing to an imbalance in energy homoeostasis[41]. Besecker *et al.*[77]
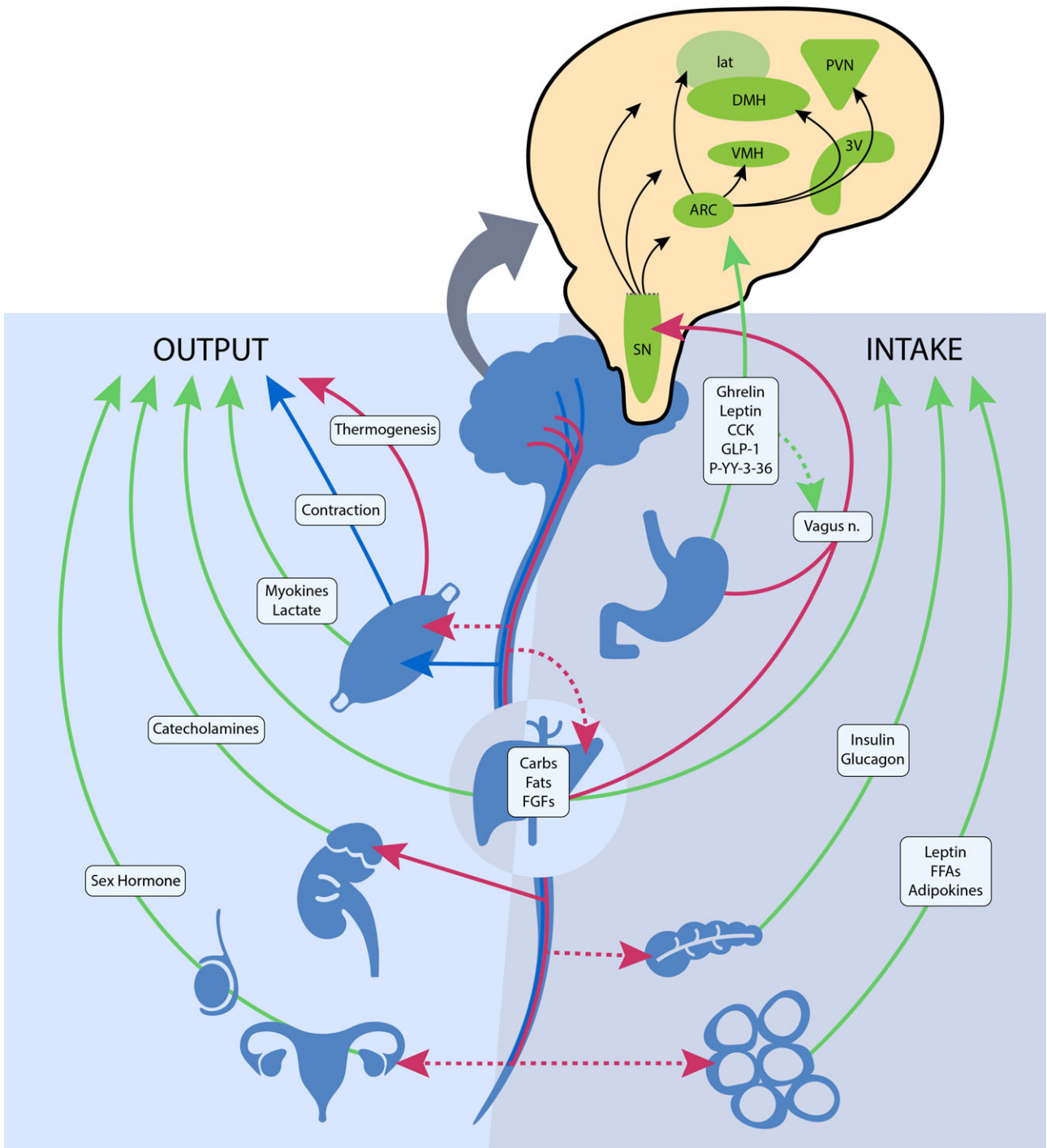
Fig. 1. The neuroendocrine components involved in the regulation of energy balance relevant to spinal cord injury. Organs and systemic signaling pathways are represented with green lines (circulating hormonal signals), red (voluntary neurological signals) and blue (autonomic neurological signals). The pop-out shows the action of these signals on regions in the hypothalamus and brainstem. Legend: 3V, third ventricle; ARC, arcuate nucleus; Carbs, carbohydrates, CCK, cholecystokinin; DMH, dorsomedial hypothalamic nucleus; FGF, fibroblast growth factors; FFA, free fatty acids; GLP-1, glucagon-like peptide 1; lat, lateral nucleus; n., nerve; PVN, paraventricular nucleus; P-YY-$_{3–36}$, peptide YY$_{3–36}$; SN, substantia nigra; and VMH, ventromedial hypothalamic nucleus. ——, Humoral; ——, Autonomic; ——, Voluntary

have proposed an SCI-induced gastric vagal afferent neuropathy as a cause for homoeostatic dysregulation of energy balance in experimental SCI in rats. The authors hypothesise the disruption of the reflex transmission of chemical feeding-related signals from the gastrointestinal tract to the CNS[77].

The central and hypothesised peripheral dysregulation of energy homoeostasis and a deterioration of body composition that results in physical deconditioning produce the 'perfect storm' for the onset of neurogenic obesity and cardiometabolic risk in persons with SCI.

Energy expenditure after spinal cord injury

Energy balance reflects a dynamic relationship between energy expenditure and energy intake. TDEE represents the number of energies burned over 24 h and is the sum of basal metabolic rate (BMR), the thermic effect of physical activity (TEPA) and the thermic effect of food digestion (TEF)[78].

In the non-disabled population, TEPA and TEF account for approximately 20 % and 8% of TDEE[78], whereas after SCI they account for about 5 % and 6 %, respectively[34,79]. To date, limited research has tested differences in the TEPA and TEF between persons with and without SCI. Monroe et al.[34], measured TEPA using a respiratory chamber. They observed significantly less TEPA in men with SCI compared with men without SCI[34]. This finding is likely because movement is restricted to the upper limbs. Consequently, the energy cost of exercise and activities of daily living is significantly lower in SCI compared with a non-disabled person[80]. Aksnes et al.[81] and Buchholz et al.[79]did not find differences in TEF between persons with chronic SCI and non-disabled controls, possibly because of a 2-h post-prandial testing window that only captured the parasympathetic-controlled obligatory phase of TEF. Alternatively, Monroe et al.[34] and Asahara and Yamasaki[82] reported significant differences between persons with and without SCI for a 24- and 3-hour test, respectively. The authors of both studies incorporated a longer testing duration, therefore, capturing both the obligatory and the sympathetic and skeletal muscle mass-mediated facultative phases of TEF[82]. Both TEF and TEPA present unique assessment challenges attributed to the scarcity of literature. Therefore, TEF and TEPA should remain an activeresearch focus given their influence on energy intake and TDEE.

BMR typically accounts for 60 to 70 % of TDEE in the non-disabled population[83], but in persons with SCI, it accounts for 70 to 80 %[84]. Fat-free mass, composed of bone, muscle and organs, contributes the most to BMR, and of fat-free mass, skeletal muscle mass accounts for 85 % of the variance. Attenuation of BMR following SCI originates from a significant reduction in metabolically active tissue[9], sympathetic nervous system dysfunction[85] and altered hormonal milieu[86].

Most SCI literature measures resting metabolic rate (RMR) or resting energy expenditure rather than the more precise measurement of BMR (Table 3). The available literature indicates that the mean measured BMR for persons with SCI ranges from 1022 to 1943 kcal/d, and mean measured RMR ranges from 959 to 2519 kcal/d (Table 3)[9,34,79–81,84,87–120]. Buchholz et al.[79] reported resting metabolism was significantly lower in persons with paraplegia (1472 kcal/d) compared with BMI-matched non-disabled controls (1677 kcal/d). In another study by Buchholz et al.[121], authors examined RMR in twenty-seven persons with SCI by injury completeness (complete: 1417 v. incomplete: 1480 kcal/d) and sex (men: 1555 v. women: 1245 kcal/d), only observing significant differences by sex. Gorgey et al.[110] recently reported non-significant differences in BMR by sex (men: 1421 v. women: 1367 kcal/d) and Farkas et al.[9] observed significant differences by LOI (tetraplegia: 1224 and paraplegia: 1517 kcal/d) in motor complete SCI. Collins et al.[80], however, did not report significant differences in RMR by LOI. These differences may stem from population demographics as

Farkas et al.[110] examined chronic motor complete SCI, whereas Collins et al.[80] included complete and incomplete SCI. Additionally, Farkas et al.[110] measured BMR and Collins et al.[80] assessed RMR in their respective studies.

RMR and BMR primarily differ in testing procedures, but both are non-invasively measured with indirect calorimetry using a metabolic cart[78]. The participant lies in a supine position in a dark room with minimal movement following at least an 8-h fast for RMR or a 12-h fast for BMR[122]. Because RMR is not at a basal state, it is usually higher than BMR for persons with and without SCI as only a short quiescent period is required (10 to 20 min[123]) prior to data acquisition[87]. Rather for BMR, the participant is awakened in the morning following an overnight stay, refrains from exercise, caffeine and alcohol for the previous 24 h, is free from emotional stress, and familiar with the apparatus[122,124]. Bauman et al.[87] examined BMR and RMR in pairs of monozygotic twins with and without SCI. The authors reported lower basal metabolism (SCI twin: 1387 and non-SCI twin: 1660 kcal/d) in both groups compared with RMR values (SCI twin: 1682 and non-SCI twin: 1854 kcal/d). Additionally, both values were significantly lower in SCI compared with individuals without the injury[87]. There is an approximate 20 % difference between BMR and RMR values in persons with SCI compared with an 11% difference in persons without SCI. Considering that resting/basal metabolism are the largest components of TDEE in persons with and without SCI and it is significantly influenced by fat-free mass, several studies have reported that RMR can be used as a strong predictor of energy intake[125–128]. When using RMR rather than BMR as a predictor of energetic intake in persons with SCI, dietary need can be overestimated by nearly 400 kcal/d (assuming approximately a 1900 kcal/d diet[19]). These data indicate BMR is a more sensitive indicator of energetic need, and less reliance should be placed on RMR in SCI research. However, TDEE remains superior as it accounts for the multiple components of daily energy expenditure.

According to published studies, during the acute phase of SCI, TDEE ranges from 2030 to 3344 kcal/d (Table 4)[89,93,94,97,101,102,129]. In chronic SCI, TDEE is from 1332 to 2834 kcal/d (Table 4)[9,34,94,98–100,121,130]. TDEE is reduced in persons with chronic SCI by as much as 54 % in persons with tetraplegia[130] and nearly 20 % in individuals with paraplegia[121]. TDEE can be assessed by measuring average daily energy expenditure using direct, or whole body, calorimetry, doubly labelled water, or mechanical ventilation. Of the SCI literature that assessed TDEE, only 33 % measured TDEE. Direct calorimetry measures the amount of heat produced while enclosed within a respiratory chamber and is the gold standard for measuring energy metabolism[78]. A test participant is completely enclosed in the chamber where there are no social interactions during the measurements and audiovisual contact with investigators[131]. In cross-sectional study designs, the participants spend a minimum of 24 h continuously, and up to a week (or more) in dietary intervention studies[131]. This method has several limitations, including the cost of highly specialised equipment, space to house the equipment, confinement of the participant and the need to exclude anything emitting heat other than the research subject. For persons with paralysis, and especially high injury levels, direct calorimetry is unrealistic because

Table 3. BMR/RMR in spinal cord injury literature

Author, year	Group(s)	n	Sex	Age (years) Mean	SD	LOI	AIS	TSI/range (years) Mean	SD	BMR/RMR (kcal/d) Mean	SD
Aksnes et al., 1993	Group A-meal	6	M	27	2	C6-C7	Frankel A	5	2	1321*,†	
	Group B-water	3	M	28	3	C6-C7	Frankel A	4	1	1218*,†	
Alexander et al., 1995	Para-PI	14	M	53	3	Para		20	3	1891	97*
	Para-no PI	24	M	50	3	Para	C/I	22	3	1780	62*
Aquilani et al., 2001	All	10	M	42	19	Para	A	≥ 0·2		1469	217*
Barco et al., 2014	All	11	M	32†		C1-C7	C/I	Acute		1943**,†	
Bauman et al., 2004	Twin with SCI	13	M/F	38†		C5-L2	C/I	15	9	1387	268**
Bauman et al., 2011	Testosterone Replacement	11	M	43	6	Para/tetra	C/I	13	10	1328	262*
	Control	11	M	35	9	Para/tetra	C/I	12	9	1319	112*
Bauman et al., 2015	Testosterone Treatment	13	M	44	6	Para/tetra	A-C	15	10	1283	246*
	Control	11	M	35	9	Para/tetra	A-C	12	9	1341	105*
Broad et al., 2020	Wheelchair rugby athletes	14	M	31	6	Para/tetra	I			1735	257*
Buccholz et al., 2003	Para	28	M/F	34	9	Para	C/I	11	10	1465	288*
	Controls	34		29	8					1677	233*
Chun et al., 2017	All	50	M/F	42	11	Para/tetra	A, B	12	7	1284	139**
	Para	23	M/F	42	12	Para	A, B	11	7	1250	147**
	Tetra	27	M/F	42	9	Tetra	A, B	13	8	1317	124**
Collins et al., 2010	Tetra	32	M/F	53	14	C5-C8	A-D	11	12	1411	315*
	Para	34	M/F	52	12	T1-L4	A-D	16	14	1433	233*
Cox et al., 1985	All	45	M/F	30†		Para/tetra		0·18	0·04	1324**,†	
Farkas et al., 2019	Tetra	28	M/F	43	11	C4-C8	A, B	16	11	1517	398**
	Para	13	M/F	46	10	T2-L1	A, B	13	12	1224	390**
Farkas et al., 2020	Mid-para	6	M/F	31	11	T6–T8	A, B	5	6	1491	241**
	Low-para	5	M	39	11	T10-L1	A, B	10	6	1693	329**
	Control	5	M/F	29	12					1647	233**
Farkas et al., 2021	Para	11	M/F	35	11	T5-L1	A, B	7	6	1583	289**
	Controls	6	M/F	29	12					1647	233**
Gorgey et al., 2010	All	10	M/F	33	7	C6-T11	A, B	11	7	1256	231*
Gorgey et al., 2011	All	2	M	53†		C4/5, T11	D	0·33	2	1227*	
Gorgey et al., 2012	Exercise + diet	5	M	36	9	C5-T10	A, B	16	9	1363	132*
	Diet	4	M	33	10	T4-T11	A, B	8	10	1793	397*
Gorgey et al., 2015	All	16	M	38	9	C5-T10	A, B			1494	34**
	Tetra	6	M	39	9	C5-C7	A, B			1411	10**
	Para	10	M	38	8	T3–10	A, B			1526	34**
Gorgey et al., 2016	Exercise	6	M	41	7	C5-T10	A, B	13	9	1470	173**
	Control	5	M	35	8	C5-T10	A, B	5	4	1147	403**
Gorgey et al., 2018	Male	8		38	9					1421	503**
	Female	8		39	13					1367	396**
Gorgey et al., 2019	Testosterone + exercise	11	M	37	12	C5-T11	A, B	10	9	1443	231*
	Testosterone only	11	M	35	8	C6-T11	A, B	7	6	1519	331*
Gorgey & Gater, 2011	All	32	M	36	9	C5-T11	A, B			1431	345*
	Tetra	11	M			C5-C7	A, B			1259	204*
	Para	25	M			T4-T11	A, B			1483	365*
Hayes et al., 2002	All	11	M/F	36	8	Para/tetra		> 3		1390	245*
Holmlund et al, 2018	Tetra-male	19	M	41	15	C5–C8	A, B	≥ 0		1195	207*
	Tetra-female	7	F	42	12	C5–C8	A, B	≥ 0		959	140*
	Para-male	28	M	45	12	T7–T12	A, B	≥ 0		1286	223*
	Para-female	10	F	39	11	T7–T12	A, B	≥ 0		1030	206*

Table 3. (*Continued*)

Author, year	Group(s)	n	Sex	Age (years) Mean	SD	LOI	AIS	TSI/range (years) Mean	SD	BMR/RMR (kcal/d) Mean	SD
Kearns et al., 1992	All	10	M/F	32	19	C4-T10	Frankel A	0·2†		1523	109*
Kolpek et al., 1989	All	7	M/F	34	5	C2-T3		0–0·05		1760	288*
Lee et al., 1985	Hypometabolic	6	M	44	15		C/I	16	9	1588	209*
	Normometabolic	5	M	41	5		C/I	19	14	1757	283*
	Hypermetabolic	6	M	48	16		C/I	19	12	1786	255*
Liu et al., 1996	Tetra-PI	16	M	40	3	Tetra		10	2	1775	296*
	Tetra-no PI	16	M	40	2	Tetra		15	3	1538	264*
	Controls	16	M	43	3			N/A		1847	268*
Monroe et al., 1998	SCI	10	M	36	8	C6-L3	Frankel A	9	2	1756	64*
	Controls	59	M	32	7					2212	317*
Nightingale et al., 2017	All	33		44	9					1481	32*
Nightingale & Gorgey, 2018	All	30	M/F	35	11	C5-L1	A, B	35	11	1499	162**
	Tetra	9								1467	178**
	Para	21								1497	148**
Pelly et al., 2017	All	7	M	31	7	T3-L5	C/I	10–15		1538	139*
Perret & Stoffel-Kurt, 2011	Acute	12	M/F	28	7	C4-T10	A, B	0·4	0·3	1414	327*
	Chronic	12	M/F	29	7	C5-T12	A, B	5	2	1304	232*
Rodriguez et al., 1997	All	12	M/F	32†		Para/Tetra	C/I	< 1		2519	693*
Sedlock & Laventure, 1990	All	4	M	28	2	T4-L1		7	3	1530	330*
Shea et al., 2018	All	25	M/F	44†		C4-C8	C/I	18†		1414**,† (M) /1104**,† (F)	
Spungen et al., 1993	All	12	M	42	3	Para		10	2	1854	70*
Sumrell et al., 2018	All	22	M	36	10	C5-T11	A, B	8	8	1137	280**
	Para	14	M	35	9	T4-T11	A, B	8	9	1216	278**
	Tetra	8	M	37	12	C5-C7	A, B	8	7	1022	240**
Tanhoffer et al., 2012	All	14	M/F	40	13	C4-T12	A, C	10	8	1432	228*
Tanhoffer et al., 2014	Sedentary group	8	M	39	12	C6-T12	A, C	90	6	1244	304*
	Exercise group	8	M	40	15	C6-T12	A, C	90	6	1200	234*
Yilmaz et al., 2007	AIS A	22	M	32	11	Tetra/para	A	3†		1433	488*
	AIS B	8	M	33	11	Tetra/para	B	3†		1170	394*
	Tetra	11	M	29	10	Tetra	C	3†		1129	300*
	Para	19	M	34	10	Para	C	3†		1499	508*
Yilmaz et al., 2007	≥ T6	13	M	29	9	Tetra/para	A, B	3	3	1407	586*
	≤ T7	7	M	37	14	Tetra/para	A, B	3	3	1504	204*

LOI, level of injury; AIS, American Spinal Injury Association Impairment Scale; TSI, time since injury; Para, paraplegia; PI, pressure injuries; C, complete; I, incomplete; Tetra, tetraplegia.
Blank spaces indicate data were not provided in the study; data are presented as mean ± standard deviation.
* RMR/resting energy expenditure was measured.
† Standard deviation not provided.
** BMR was measured.

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Table 4. TDEE after SCI

Author, year	Group(s)	n	Sex	Age (years) Mean	SD	LOI	AIS	TSI/range (years) Mean	SD	TDEE (kcal) Mean	SD	Measured/ predicted	Measured/prediction method
Buchholz et al., 2003	Male	17	M	39	11	Para	C/I	10	8	2490	637	Predicted	Heart Rate Monitor
	Female	10	F	32	6		C/I	16	11	1870	607	Predicted	Heart Rate Monitor
	Complete	17	M/F	36	10		C			2072	505	Predicted	Heart Rate Monitor
	Incomplete	10	M/F	37	10		I			2582	852	Predicted	Heart Rate Monitor
Barco et al., 2014	All	11	M	32*		C1-C7	C/I	Acute		2425–2629	434–458	Measured	Ventilator
Cox et al., 1985	All	45	M/F	30*		Tetra/para	C/I	0·18	0·04	2030	41	Predicted	BMR × 1·2 (Long Method)
	All	45	M/F	30*				0·18	0·04	3164	61	Predicted	BMR × 1·75 (Rutten Method)
Desneves et al., 2019	All	20	M/F	43	20	C1-L5	A-D	0·05–0·21		2354	774	Measured	Doubly labeled water
Farkas et al., 2019	Tetra	13	M/F	46	10	C4-C8	A, B	13	12	1530.	640	Predicted	BMR × 1·2 (Long Method)
	Para	28		43	11	T2-L1		16	11	1851	405	Predicted	BMR × 1·2 (Long Method)
	Tetra	13		46	10	C4-C8		13	12	1774	388	Predicted	BMR × 1·15 (Farkas Method)
	Para	28		43	11	T2-L1		16	11	1467	614	Predicted	BMR × 1·15 (Farkas Method)
Farkas et al., 2020	Mid-para	6	M/F	31	11	T6–T8	A, B	5	6	1712	238	Predicted	BMR × 1·15 (Farkas Method)
	Low para	5	M	39	11	T10-L1	A, B	10	6	1949	456	Predicted	BMR × 1·15 (Farkas Method)
Mollinger et al., 1985	High tetra	14	M	35	8	C4-C6	C	6	5	1332	112	Predicted	Measure of Oxygen Consumption
	Low tetra	13		33	6	C6-C7	C	7	4	2108	523	Predicted	Measure of Oxygen Consumption
	High para	16		33	7	T1-T10	C	9	5	2611	620	Predicted	Measure of Oxygen Consumption
	Low para	5		33	9	T10-L2	C	4	3	2693	427	Predicted	Measure of Oxygen Consumption
Monroe et al., 1998	All	10		36	8	C6-L3	Frankel A	9	2	1870	73	Measured	Respiratory chamber
Rodriguez et al., 1997	All	12	M/F	32*		C3-T12	C/I	< 1		3344	431	Predicted	BMR × 1·2 × 1·6 (Long Method)
Rowan & Klazemi, 2020	All	16	M/F	43*		C4-C6		0·06		2784*		Predicted	66·5 + (13·7 × weight (kg)) + (5·003 × height (cm)) × (6·755 × age) × 1·2 × 1·1 (Harris-Benedict with Long, Trauma Method)
Shea et al., 2018	All	25	M/F	44*		C4-C8	C/I	18*		1703	416	Predicted	Collins et al., 2010; Ainsworth et al., 1993 Methods
Tanhoffer et al., 2012	All	14	M/F	40	10	C4-T12	A-C	10	8	2346	595	Measured	Doubly labeled water
	All	14								2031	362	Predicted	Heart rate Monitor
	All	14								2728	775	Predicted	Multi-sensor armband
Tanhoffer et al., 2015	All	8	M/F	42	13	C6-T12	A, C	9	6	2406	552	Measured	Doubly labeled water
	All	8	M/F	42	13	C6-T12	A, C	9	6	2834	648	Predicted	Multi-sensor armband
Wouda et al., 2018	High-intensity interval training	10	M/F	50	15	Tetra/para	D	0·19	0·08	2666	528	Predicted	Multi-sensor armband
	Moderate-intensity training	10		34	15		D	0·18	0·09	2736	603	Predicted	Multi-sensor armband
	Control	10		40	10		D	0. 2	0·07	2437	341	Predicted	Multi-sensor armband
Wouda et al., 2020	All	30	M/F	41	17	Tetra/para	D	0·19*		2632	509	Predicted	Multi-sensor armband

SCI, spinal cord injury; LOI, level of injury; AIS, American Spinal Injury Association Impairment Scale; TSI, time since injury; TDEE, total daily energy expenditure; Para, paraplegia; C, complete; I, incomplete; Tetra, tetraplegia; Blank spaces indicate data were not provided in the study; data are presented as mean ± standard deviation.
* Standard deviation are not provided.

of the need for caregiver assistance, power wheelchairs and/or assistive electronic devices. To date, Monroe and colleagues[34] are the only investigators to use direct calorimetry with a respiratory chamber in persons with SCI. The authors demonstrated a TDEE of 1870 kcal/d in chronic complete SCI compared with 2376 kcal in persons without SCI[34], a 24 % difference.

Less labour- and time-intensive methods to measure energy expenditure are with doubly labelled water or a metabolic cart during mechanical ventilation. Doubly labelled water is centred around the difference between the apparent turnover rates of the hydrogen and oxygen of body water as a function of carbon dioxide production. The procedure encompasses enriching a research participant with heavy oxygen and heavy hydrogen and then determining the difference in washout kinetics between the isotopes. The oxygen isotope is lost as water and as carbon dioxide due to exchange in the bicarbonate pools. The hydrogen isotope is lost only as water[132]. The strength of doubly labelled water is that it is a non-invasive and inconspicuous free-living evaluation of TDEE with no constraint or restriction for the participant. The total number of variables in the equations used to calculate TDEE from doubly labelled water is nine, plus two additional constants, from which the equation for isotope dilution spaces calculation includes five variables and one constant[131], thus, making the technique mathematically complex and prone to miscalculation. Double labelled water is infrequently used (Desneves *et al.*[94] and Tanhoffer *et al.*[99,100]) to measure TDEE after SCI. In 2012 and 2015, Tanhoffer *et al.*[99,100] reported a mean TDEE of 2346 and 2406 kcal/d, respectively, in chronic SCI, while Desneves *et al.*[94] reported 2354 kcal/d during the acute stage. These observed values far exceed the reported values by Monroe *et al.*[34] In the acute phase, mechanical ventilators provide a unique option to continuously measure respiratory gases with the addition of a metabolic monitor, similar to indirect calorimetry[133], as published by Barco *et al.*[93] Because nutritional risk is associated with ventilatory support after SCI and high levels of injury often need respiratory management[134], the use of metabolic monitoring with mechanical ventilators can provide an easy method to determine energetic need during the initial hospitalisation. However, it is likely because of the lack of availability of the specialised equipment, cost and trained personnel required to measure TDEE that most studies rely on predicting TDEE, a method prone to error[78]. Of the literature that assessed TDEE, 71 % estimated it through various methods (Table 4). Several of these methods are examined in the next section given their clinical use in defining energetic targets.

Energy (energetic) intake relative to energy expenditure after spinal cord injury

Energy intake reflects energetic gain by the ingestion of foodstuff of different energetic densities. Defining optimal nutrient intake and its management is challenging after SCI because practical guidelines for determining energy requirements for this niche population are limited. Energetic need is dependent on several factors after SCI, including the injury phase[117] and the physical activity/therapy within phase[130], the level[9,89] and completeness[130] of the injury, sex[135], body composition and its post-injury changes[85,103], presence of infection or pressure

injuries[120], and frequency and accuracy of reporting on the energetic intake used to assess nutrition[19,84]. Consequently, tremendous variability in energetic intake (Table 5) is reported across the literature, even though most studies report it is within or exceeds daily AHA, ADG, DGA and PHE recommendations (Table 6).

The IOM encourages establishing energetic intake using a sex-specific prediction equation, relying on age, height, weight and physical activity. While less precise than measuring energy requirements, the IOM is the only guideline that makes such a recommendation rather than providing an acceptable macronutrient distribution range. A limitation of IOM's equation is that it neglects to include resting or basal metabolism as the largest determinant of TDEE, and therefore energetic intake. TDEE can also be estimated using the product of BMR (or RMR) and the common activity correction factor of 1·2[9]. Several studies have predicted TDEE using 1·2 and other previously published activity, stress, injury, and/or trauma correction factors to determine energetic intake for persons with SCI[34,89,97,98,101,102,121,129].

In the acute phase of SCI, the literature indicates total energetic intake ranges from 755 to 2290 kcal/d (Table 5)[89,97,119,120,136–138]. Over the first 4 weeks of the SCI, this value increases by over 400 kcal/d[119] and likely results from the thermic effect of voluntary respiration (diaphragmatic and intercostal muscle activation) for those weaned from mechanical ventilators. The conversion from the catabolic state to declining energetic needs is not well researched, though RMR has been shown to decrease 10 weeks post-SCI[139]. In seminal work by Cox *et al.*[89], the authors reported that persons in the early rehabilitation phase of the injury require up to 54 % less energy content than would be predicted by most standard formulae. The authors further determined that in the rehabilitation phase, persons with tetraplegia need 22·7 kcal/kg/d and persons with paraplegia need 27·9 kcal/kg/d[89], guidelines that are still widely used today[140]. Of note, this calculation published by Cox *et al.*[89] was developed in fifteen persons with tetraplegia and five with paraplegia and mostly men (86 %) whom typically expend[110,121] and consume[136,141] more than women. The equations also do not account for the serial weight loss and weight regain that occurs during the early phase of the SCI and a drop in energy expenditure that persists through the rehabilitation phases of treatment.

When evaluating the energy intake and expenditure data, persons with acute SCI appear to be in a negative energy balance (TDEE: 2030 to 3344 kcal/d *v.* energetic intake: 755 to 2290 kcal/d). Distinct from other trauma conditions, persons with acute SCI do not demonstrate hypermetabolism following injury[119,129,142,143]. While a negative nitrogen balance does occur, this is *obligatory*. While the underlying mechanism contributing to a negative nitrogen balance following SCI remains poorly understood[142–145], efforts made to shift the obligatory loss of nitrogen by increasing energetic intake can lead to overfeeding. Confounding this matter is that several studies and registered dietitians use correction factors to increase energetic intake. Kaufman *et al.*[137] and Barco *et al.*[93] used an activity factor of 1·1. However, a stress factor of 1·2 to 1·75 is routinely used in in the literature[89,129,137,143]. Rodriguez *et al.*[129] examined the Harris-Benedict equation with an activity factor of 1·2 and an

Table 5. Total energetic and macronutrient intake in SCI literature

Author, year	Group(s)	N	Sex	LOI	AIS	TSI/range (years) Mean	SD	Energetic intake (kcal/d) Mean	SD	Protein intake (kcal/d) Mean	SD	Fat intake (kcal/d) Mean	SD	Carbohydrate intake (Kcal/d) Mean	SD	Dietary collection method
Abilmona et al., 2018	All	22	M	C5-T11	A, B	8	8	1362	500	242	65	534	182	588	267	SR 5-d dietary recall
Allison et al., 2018	Diet intervention	12	M/F	C2-L3	A-D	13	11	1815	743	290	95	635	313	907	466	SR 7-d, 5-d dietary recall
Aquilani et al., 2001	All	10	M	Para	A	≥ 0·2*		755 + 344								Weight, type recorded
Beal et al., 2017	All	20	M	T3-L1	A, B			1448	484	255*		527*		667*		SR 3-d dietary recall
	High vitamin D	10	M	T3-L1	A, B	19	12	1683	609	303*		606*		816*		
	Low vitamin D	10	M	T3-L1	A, B	15	12	1212	358	206*		448*		519*		
Chen et al., 2006	All, baseline	13	M/F	Para/tetra	A-D	18*		1606	672							Dietary recall
Cox et al., 1985	All	45	M/F	Para/tetra		0·18	0·04	1774*								24-h RD-assessed dietary recall
Doubelt et al., 2015	All	34	M/F	Para/tetra	C/I					328	144					FFQ
Edwards et al., 2008	All	15	M/F	Para/tetra	C/I	≥ 1*		2090	652	320	22	699	54	1064	83	SR 3-d dietary recall
Farkas et al., 2019	Para	28	M/F	T2-L1	A, B	16	11	1516	548	273	79	523	102	709	114	SR 3-d dietary recall
	Tetra	13	M/F	C4-C8	A, B	13	12	1619	564	277	81	534	92	762	121	
Gorgey et al., 2012	Exercise + diet	5	M	C5-T10	A, B	16	9	1781	228	321	36	623	53	819	89	7-d food diaries
	Diet	4	M	T4-T11	A, B	8	10	1731	127	329	69	589	87	814	52	
Gorgey et al., 2015	All	16	M	C5-T10	A, B			1350	477							SR 5-d dietary recall
Gorgey et al., 2019	Testosterone + exercise	11	M	C5-T11	A, B	10	9	1532	547	291	69	567	107	659	138	SR 3-d dietary recall
	Testosterone only	11	M	C6-T11	A, B	7	6	1497	127	314	90	539	90	629	120	
Groah et al., 2009	Tetra-male	24						2012*		343*		733*		881*		SR 4-d food log
	Para-male	37						2088*		350*		744*		992*		
	Tetra-female	1						2685*		382*		945*		1408*		
	Para-female	11						1662*		301*		563*		805*		
Iyer et al., 2020	All	50	M/F	Para/tetra	C/I	0·1–0·4		1751	294	356	52	486	90	844	180	SR, RD-assessed 3-d dietary record
	Male	35	M	Para/tetra	C/I			1809	245	364	48	495	90	872	140	
	Female	15	F	Para/tetra	C/I			1648	372	332	52	459	83	780	240	
Kaufman et al., 1985	Male	8	M	C4-L2		0·03*		848	414							10-d calorie count
Kearns et al., 1992	All	10	M/F	C4-T10	Frankel A	0·2*		1909	43							RD-interviews, nursing records
Krempien & Barr, 2011	All	32						2003	517	352	104	567	189	1100	304	SR 3-d food diary
	Male	24						2028	528	352	88	576	207	1100	312	
	Female	8						1927	510	360	140	522	162	1088	308	
Laven et al., 1989	All	29	M/F	Para/tetra	Frankel A-C	< 0·08*		1494	879	232	132					Daily meal tray observation
	Para	16						1285	505	212	120					
	Tetra	13						1752	1163	260	148					
Lee et al., 1985	Hypometabolic	6	M		C/I	16	9	2116	415							24-h dietary record
	Normometabolic	5	M		C/I	19	14	2152	709							
	Hypermetabolic	6	M		C/I	19	12	2005	508							
Levine et al., 1992	Male	24			C/I			1682	429	276	83	603	215	816	324	7-d dietary record
	Female	9						1282	418	224	86	423	203	664	289	
	All	100						2601	2006	401	282	901	785	1308	1096	FFQ

Table 5. (*Continued*)

Author, year	Group(s)	N	Sex	LOI	AIS	TSI/range (years) Mean	SD	Energetic intake (kcal/d) Mean	SD	Protein intake (kcal/d) Mean	SD	Fat intake (kcal/d) Mean	SD	Carbohydrate intake (Kcal/d) Mean	SD	Dietary collection method
Lieberman et al., 2014																
Liu et al., 1996	Tetra-PI	16		Tetra		10	2	1603	604							Inpatient, Measured from food remaining on hospital tray for 3 d; Outpatient, 3-d dietary record
	Tetra-no PI	8		Tetra		15	3	1561	808							
Mollinger et al., 1985	High tetra	14	M	C4-C6	C	6	5	2209	894							Measured food remaining on tray for 3 d, 24-h dietary recall
	Low tetra	13	M	C6-C7	C	7	4	2213	698							
	High para	16	M	T1-T10	C	9	5	2384	742							
	Low para	5	M	T10-L2	C	4	3	2732	866							
Moussavi et al., 2001	All	189	M/F	Para/tetra	A-D	13	10					70	29			SR 3-d record
Nightingale et al., 2017	All	33						1742	72	306	13	592	30	787	38	SR 7-d dietary record, food weighing
Peiffer et al., 1981	Para	9	M/F	Para		≥ 0·3*		2446	251	488	92					24-h dietary recall
	Tetra	9	M	Tetra		≥ 0·3*		1795	447	288	84					
Perret & Stoffel-Kurt, 2011	All	12						1775	234	286	32	644	88	775	224	SR 7-d dietary record
Rowan & Klazemi, 2020	All	16	M/F	C4-C6		0·06*		2290								Inpatient, energy content recorded from all sources of feeding (enteral, intravenous)
	Ca only	36	M/F			9	8	1589	709			510	336	886	375	
Sabour et al., 2012	n-3 fatty acid, Ca+	39	M/F			14	26	2003	658			772·2	652	1001	367	SR 3-d dietary record
Sabour et al., 2012	All	162						2032	699			746	302	1077	437	FFQ
	Male	131						2078	724			746	284	1115	462	
	Female	31						1839	547			748	377	918	260	
	Complete	48						1967	726	259	93	704	291	1042	390	
	Incomplete	114						2060	688	257	100	764	307	1092	456	
	Tetra	94						2013	681	255	95	735	318	1071	423	
	Para	68						2060	727	261	102	762	281	1086	459	
Sabour et al., 2016	Males	83	M	Para/tetra	A-D	12	6	1826	553	316	96	588	178	922	279	3-d dietary record/recall
	Females	17	F	Para/tetra	A-D	15	9	1413	350	222	53	582	144	619	153	
Sabour et al., 2016	All	103	M/F	Para/tetra	A-D	≥ 1*		1756	542	312	100	544	154	900	288	24-h dietary recall
Sabour et al., 2016	All	157	M/F	Para	C/I	≥ 1*		1847	589	284	104	675	306	952	332	3-d dietary recall
Tomey et al., 2005	All	95						2265	745	329	127	835	364	1100	347	Modified FFQ to assess 7-d intake
Walters et al., 2009	Male	63						2096	420	335	67	629	126	1090	218	24-h dietary recall
	Female	14						1711	152	291	26	479	43	907	81	

SCI, spinal cord injury; LOI, level of injury; AIS, American Spinal Injury Association Impairment Scale; TSI, time since injury; SR, self-report; Para, paraplegia; Tetra, tetraplegia; C, complete; I, incomplete; PI, pressure injuries; FFQ, food frequency questionnaire; RD, registered dietitian.
Blank spaces indicate data were not provided in the study; data are presented as mean ± standard deviation.
* Standard deviation not provided.

Table 6. Comparison of authoritative, evidence-based non-spinal cord injury dietary guidelines

	American Heart Association[6,7]*		Australian Dietary Guidelines[8]	Dietary Guidelines for Americans[1]		IOM Dietary Reference Intakes[2]		PHE Government Dietary Recommendations[9,10]		WHO Healthy Diet[11]
Total energy	Female	Male	Female/Male	Female	Male	Female	Male	Female	Male	Female/male
Total energy (kcal/d)	1200–1500	1500–1800	2108–2259	1600–2000	2000–3000	Estimated energy requirement†	Estimated energy requirement‡	1840–2000	2294–2500	≥ 1200 with 500–600/d deficit or in balance with energy expenditure
Macronutrients: protein, carbohydrate and fat										
Protein (kcal) (RDA)	180–375	225–450	393–398	184	224	Varies based on total energy		180–186	213–222	Varies based on total energy
Protein (% kcal) (AMDR)	15 or 25		15–25	10–35	10–35	10–35	10–35	15	15	15
Carbohydrates (kcal) (RDA)	420–975	525–1170	1088–1108	520	520	Varies based on total energy		980–1068	1224–1332	Varies based on total energy
Carbohydrates (% kcal) (AMDR)	35, 45, 55 or 65		45–65	45–65	45–65	45–65	45–65	50	50	55–60
Dietary fibre (g)	25–30[12]		24–26	22–28	28–34	14 (21–38)§		30	30	
Total fat (kcal) (AMDR)	240–600	300–720	799–820	300–700	400–1050	Varies based on total energy		648–702	801–873	Varies based on total energy
Total fat (% kcal) (DGA)	20 or 40		20–35	20–35	20–35	20–35	20–35	35	35	≤ 20–30
Saturated fat (% kcal) (DGA)	5–6		< 10	< 10	< 10	As low as possible		11	11	< 10
MUFA	8 %		32–34 g	No standards set		No standards set		27–29 g	33–36 g	
n-6 PUFA/linoleic acid (AI)	2 %		13·4–13·9 g	11–12 g/d	14–17 g/d	5–10 %	5–10 %	13–14 g	17–18 g	
n-3 PUFA/linolenic acid (AI)				1·1 g/d	1·6 g/d	0·6–1·2 %	0·6–1·2 %			
Micronutrients: vitamins										
Choline (mg) (AI)				425	550	425	550			
Vitamin A (mg/d) (RDA)			1·2–1·3	700	900	700	900	600	700	
Vitamin B$_1$/thiamin (mg/d) (RDA)			1·74–1·83	1·1	1·2	1·1	1·2	0·7–0·8	0·9–1·0	
Vitamin B$_2$/riboflavin (mg/d) (RDA)			2·18–2·27	1·1	1·3	1·1	1·3	1·1	1·3	
Vitamin B$_3$/niacin (mg/d) (RDA)			45·5–45·9	14	16	14	16	12·1–13·2	15·1–16·5	
Vitamin B$_5$/pantothenic Acid (mg/d) (AI)						5	5			
Vitamin B$_6$ (mg/d) (RDA)				1·3–1·5	1·3–17	1·3–1·5	1·3–1·7	1·2	1·4	
Vitamin B$_7$/biotin (µg/d) (AI)						30	30			
Vitamin B$_9$/folate (µg/d) (RDA)			286–299	400	400	400	400	200	200	
Vitamin B$_{12}$ (µg/d) (RDA)				2·4	2·4	2·4	2·4	1·5	1·5	
Vitamin C (mg/d) (RDA)			130–142	75	90	75	90	40	40	
Vitamin D (µg/d)				15 (RDA)	15 (RDA)	5–15 (AI)	5–15 (AI)	10	10	
Vitamin E (mg/d) (RDA)				15	15	15	15			
Vitamin K (µg/d) (AI)				90	120	90	120			
Micronutrients: minerals										
Ca (mg/d)			888–945	1000–1200 (RDA)	1000–1200 (RDA)	1000–1200 (AI)	1000–1200 (AI)	700 (RDA)	700 (RDA)	
Cr (µg/d) (AI)						20–25	30–35			
Chloride (mg/d) (AI)						1800–2300	1800–2300	2500	2500	
Cu (µg/d) (RDA)				900	900	900	900	1·2	1·2	
Fluoride (mg/d) (AI)						3	4			

Table 6. (*Continued*)

	American Heart Association[6,7]*		Australian Dietary Guidelines[8]	Dietary Guidelines for Americans[1]		IOM Dietary Reference Intakes[2]		PHE Government Dietary Recommendations[9,10]		WHO Healthy Diet[11]
	Female	Male	Female/Male	Female	Male	Female	Male	Female	Male	Female/male
Total energy										
Iodine (µg/d)				150			150	140	140	
Fe (mg/d) (RDA)			15·0–15·6	8–18	8	8–18	8	8·7–14·8	8·7	
Mg (mg/d) (RDA)			353–366	310–320	400–420	310–320	400–420	270	300	
Mn (mg/d) (AI)				1·8	2·3	1·8	2·3			
Mo (µg/d) (AI)				45		45	45			
P (mg/d) (RDA)			1626–1673	700	700	700	700	550	550	
K (mg/d) (AI)			3495–3551	4700	4700	4700	4700	3500	3500	
Se (µg/d) (RDA)				55	55	55	55	60	75	
Na (mg/d) (UL)	≤ 2400 (≤ 1500 is better)			2300	2300	2300	2300	2500	2500	< 5000
Zn (mg/d) (RDA)			12·9–13·3	8	11	8	11	7	9·5	

AMDR, Acceptable Macronutrient Distribution Range; DGA, Dietary Guidelines for American; AI, adequate intake; UL, tolerable upper intake level.
* Guidelines were established to prevent CVD and manage overweight and obese adults.
† Estimated energy requirement equation for males = 662 − (9·53 × age (years)) + PA × ((15·91 × weight (kg)) + (539·6 × height (m))), where PA is the physical activity coefficient.
‡ Estimated energy requirement equation for females = 354 − (6·91 × age (years)) + PA × ((9·36 × weight (kg)) + (726 × height (m))), where PA is the physical activity coefficient.
§ Values in parentheses are an example of the total g/d of total fibre calculated from 14 g/1000 kcal multiplied by the median energy intake (kcal/1000 kcal multiplied by the median energy intake (kcal/1000 kcal) from the Continuing Survey of Food Intakes by Individuals (1994–1996, 1998).

injury factor of 1·6 and identified that the equation overestimated energetic requirements in twelve persons with acute SCI. The same authors reported that establishing nutritional management upon serial indirect calorimetry measurements with higher stress and activity factors result in overfeeding[129]. Kearns et al.[119] compared measured RMR with the Harris-Benedict equation in five individuals with tetraplegia and reported that the use of the equation leads to an overfeeding by nearly 70 %. While the time frame of the investigation relative to SCI was not specified, the authors suggested administering 80 % of predicted energetic needs[119].

The 2008 Paralyzed Veterans of America (PVA) *Early Acute Management in Adults with Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professions* state 'Provide appropriate nutrition when resuscitation has been completed and there is no evidence of ongoing (spinal) shock or hypoperfusion'. The PVA recommendations do endorse the determination of energetic requirements for nutritional support using a 30-min energy expenditure measurement by indirect calorimetry. These guidelines for acute SCI do not define or provide a reference for what 'appropriate nutrition' entails, and hospitals and inpatient rehabilitation facilities do not use or often have access to metabolic carts, and insurance plans do not cover the cost of indirect calorimetry. Similarly, the Academy of Nutrition and Dietetics (AND) recommends the use of indirect calorimetry during the acute phase of SCI. They state 'actual energy needs are at least 10 % below predicted needs[140]'. But the AND recommends in the absence of indirect calorimetry to use the Harris-Benedict formula using admission weight and an injury factor of 1·1 and an activity factor of 1·2[140]. This type of prediction method overestimates TDEE and subsequently energetic intake in persons with SCI, thereby leading to overfeeding[129,143]. Persons with SCI remain in an obligatory negative nitrogen balance, but providing excess energy content should be avoided. Overfeeding carries unique complications, such as hyperglycaemia, hypercapnia, hypertriacylglycerolaemia, uremia and obesity[8,146].

With regard to chronic SCI, the PVA Consortium for Spinal Cord Medicine recently assembled an expert panel to compile the *Clinical Practice Guidelines on Identification and Management of Cardiometabolic Risk after SCI* (PVA guidelines; Table 7; Fig. 2)[10]. The inaugural guidelines recommended when establishing energetic targets, all persons with chronic SCI should undergo an energetic assessment using indirect calorimetry to estimate energy expenditure and assess energy needs[10]. Given indirect calorimetry is used to measure resting and basal metabolism, an important consideration is how to determine TDEE. In 2019, Farkas et al.[9] developed a novel SCI-specific correction factor of 1·15 to estimate TDEE from BMR (or RMR) using 2·7 ml of oxygen/kg of body weight/min[80], a MET (metabolic equivalent of task) for SCI. The SCI-specific TDEE prediction equation requires validation against the gold standard respiratory chamber but provides promise. It is a novel method to estimate TDEE, and to accurately determine energetic needs in chronic SCI.

Across the chronic SCI literature, energetic intake ranges from 1212 to 2732 kcal/d (Table 5)[9,34,84,86,92,108,109,115,117,130,135,141,147–157], seemingly appearing to be in energy balance when evaluating

Table 7. Practical dietary recommendations with example foods to consume and avoid for persons with SCI

Paralyzed Veterans of American (PVA) Dietary Criteria[5]	Examples of foods to consume	Examples of foods to avoid
Fruits	Apples, apricots, avocado, bananas, blueberries, cherries, clementines, cranberries, dates, dried fruit (unsweetened), figs, grapes/raisins, kiwi, mango, melon, nectarines, papaya, pears, pineapples, plums, pomegranates, prunes, raspberries, strawberries and tomatoes. *Frozen fruits are a good alternative when fresh fruit is not available.*	Fruit cups (with syrup), fruit juice, fruit snacks, jam and jelly. *Unsweetened cranberry juice may help reduce excess bacteria in the urinary tract to prevent urinary tract infections.*
Vegetables	Artichokes, asparagus, beets, broccoli, brussels sprouts, cabbage, carrots, cauliflower, maize, cucumber, eggplant, garlic, green beans, kale, mushrooms, onions, peas, peppers, pickles, potatoes, romaine, spinach, squash, sweet potatoes and turnips. *Frozen vegetables are good alternatives when fresh vegetables are not available.*	Canned vegetables (high in Na), French fries, ketchup, potato chips, onion rings, relish and sweet potato chips. *Avoid high-fat food preparations (e.g. frying/deep frying and use healthier methods such as pan-frying with olive oil, baking, broiling, braising, poaching, steaming and stewing.*
Poultry	Chicken, duck, eggs, egg whites and turkey	Deep-fried turkey, deviled eggs, fried chicken and fried duck
Fish	Cod, halibut, herring, lake trout, mackerel, mahi-mahi, rainbow trout, salmon, sardines, swordfish, tuna (albacore and canned light) and whitefish	Anchovies (cured/canned, high in Na), fried fish (all), fried shellfish (all) and shrimp (high in cholesterol)
Low-fat dairy products	Skim/1 % milk, low-fat cheese (e.g. Cheddar, mozzarella, goat, provolone, muenster, feta, swiss, etc.), low-fat cottage cheese and low-fat yogurt	Whole/2 % milk, cream cheese, half and half, creamer, and condensed milk, sour cream, heavy cream, heavy whipping cream and whipped cream
Whole grains	Brown rice, buckwheat, millet, oats, quinoa, spelt, wild rice, whole wheat bread and whole-grain pasta	Crackers (all), granola bars (high in sugar), muffins, processed cereals (all) and oatmeal packets with high sugar content and additives, white rice cakes, white bread, white pasta and white rice
Legumes	Black beans, black-eyed peas, chickpeas/garbanzo beans, fava (broad) beans, kidney beans, lentils, pinto beans, soyabeans, split peas, tofu and white beans	Baked beans (all)
Nuts (and seeds*)	*All unsalted:* almonds, brazil nuts, cashews, chestnuts, flaxseed, hazelnuts, peanuts, pecans, pumpkin seeds, sesame seeds, sunflower seeds and walnuts	Salted nuts, salted seeds and processed nut butters (e.g. processed peanut butter with extra sugar and additives, hazelnut chocolate spread, etc.)
Non-tropical vegetable oils	Rapeseed, maize, olive, peanut, safflower, soyabean and sunflower oils	Coconut, hydrogenated, (full, partial), palm kernel and palm oils. Processed salad dressings and oil-based products (e.g. BBQ sauce, mayonnaise and margarine)
Limit†		
Sweets	Dark chocolate (in small quantities), dried fruit (unsweetened) and popcorn (unsalted, no butter)	Cakes, candy, caramel, caramelised popcorn, cookies, croissants, donuts, ice cream, milk chocolate and pastries
Sugar-sweetened beverages	Carbonated water (flavoured and unflavoured) and splash of zero (0) calorie liquid water enhancer	Fruit punch, fruit-flavoured beverages, juice and soda (diet and regular)
Red meats	Lean cuts with ≤ 5 % fat, trim off fat before cooking‡ and pour off melted fat after cooking, use healthier cooking methods (e.g. bake, broil, stew, grill and roast). *Packages for lean cuts will usually say 'round', 'loin' or 'sirloin'*	*All processed meats:* bacon, beef jerky, cold cuts, deli slices, frankfurters, ham, hot dogs, pepperoni, salami and sausages
Na intake (≤ 2400 mg)§	*Consult nutrition facts on specific food items*	
Saturated fat (< 5–6 %)		

* The PVA guidelines do not mention seeds; however, the authors are including seeds in the nut category.
† Limit to special occasions (i.e., birthdays, weddings, holidays, etc.)
‡ For persons with limited upper extremity function, ask the butcher to trim the fat at the supermarket.
§ For persons with hypertension, although the authors recommend adopting ≤ 2400 mg of Na for all individuals regardless of hypertension status given the elevated consumption of Na-dense foods reported in the literature.

TDEE (1332 to 2728 kcal/d). At face value, an energy balance does not appear to be congruent with the reported high rates of obesity[8,9,11,29,60]. However, in a recent meta-analysis by Farkas *et al.*[19], the authors reported in a sample of 606 persons with chronic SCI, a pooled energetic intake of 1876 kcal/d and a pooled RMR of 1492 kcal/d. Estimating a TDEE of 1716 kcal/d (using RMR × 1·15[9]), there is a positive energy balance of over 150 kcal/d. This is further supported by the additional work by Farkas and colleagues[9]. The authors reported a greater energetic intake in persons with tetraplegia compared with paraplegia when adjusting energy intake by body weight, thereby accounting for body composition that is significantly different by injury level[9,107,158,159]. The authors concluded that their findings may explain why persons with tetraplegia had significantly great percentage body fat relative to paraplegia[9]. Collectively, these data provide support that persons with chronic SCI may overconsume relative to their need, and this may contribute to the high rates of neurogenic obesity. However, the findings are subject to how energetic intake was operationalised.
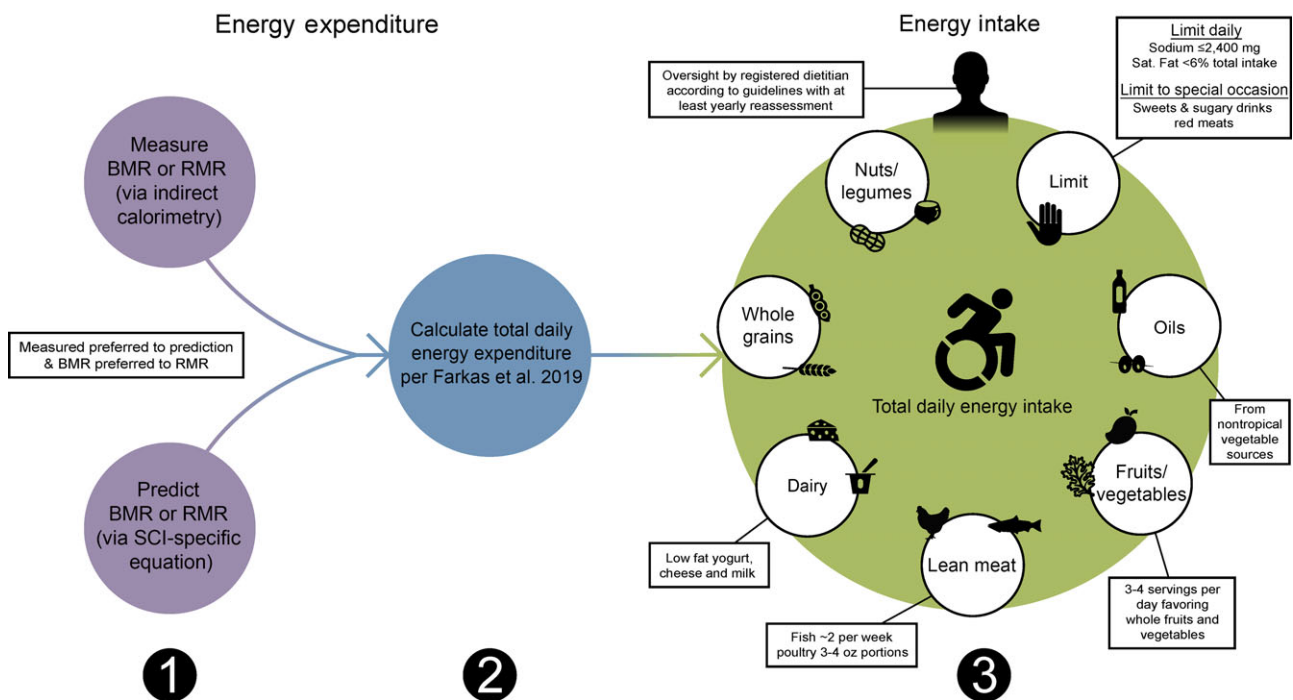
Fig. 2. Sequential dietary recommendations for persons with a spinal cord injury (SCI). First, BMR or RMR should be annually measured with indirect calorimetry or estimated using SCI-specific predictions equations (Nightingale and Gorgey[116], Chun et al.[105] or Buchholz et al.[79]) when indirect calorimetry is unavailable. Second, total daily energy expenditure should be estimated as the product of BMR or RMR and 1·15 for persons with SCI using the Farkas et al.[9] equation. Third, a registered dietician should oversee a healthy dietary pattern following the *Clinical Practice Guidelines on Identification and Management of Cardiometabolic Risk after SCI* and additional recommendations provided in this review[10].

While dietary recalls, dietary diaries/records/logs and food frequency questionnaire (FFQ) have demonstrated a strong agreement amongst themselves, research has identified systematic misreporting errors for all of the self-reported dietary instruments[160]. Most of the literature in both acute and chronic SCI describe using dietary assessments without indicating self-report or whether a registered dietitian administered or reviewed the instrument (Table 5). In fact, 40 % of the investigators specify the use of self-report dietary assessment techniques, whereas 11 % and 9 % indicated measuring the food consumed off the plate or if a registered dietitian performed the assessment, respectively (the remaining studies provided insufficient detail to clearly determine assessment methods). It is well established that dietary assessment methods underreport true energetic consumption in persons without SCI[160,161], and a similar phenomenon is likely present in the population with SCI[9,84]. Moreover, self-report after SCI becomes a challenge, especially with higher levels of injury. Persons with tetraplegia may have difficulty writing down, especially in detail, intake data and may limit, and in some cases omit, what they ate or drank on their assessment instruments. Portion size, food preparation and cooking details may be omitted and are details that can greatly influence the energy content of food. Family members or caregiver(s) may introduce a source of error by recording their food. Accordingly, it is likely that, as with the population without SCI[161], energetic intake is being underreported. Future large-scale studies with more stringent dietary assessment and testing methods are needed to examine the energetic need relative energy expenditure after SCI.

Macronutrient intake

Macronutrients are dietary constituents that provide energy. They include protein, carbohydrates, fats and alcohol. Although alcohol is considered a macronutrient and provides energy, it is not needed for survival.

Protein

Proteins (4 kcal/g) are considered the most abundant macronutrient; they are composed of amino acids, of which nine are essential and cannot be synthesised by the body but must be acquired in the diet. The quality of dietary protein is characterised by the protein's digestibility and its amino acid profile in relation to requirements as determined by repair, maintenance and growth[12]. Several studies report protein ingestion in persons with SCI is within or exceeds recommended daily values for the population without SCI (Tables 5 and 6)[19,108,115,117,135,136,150–154,162–167]. Approximately 15 to 19 % of the total daily energy intake came from protein for persons with SCI[108,115,117,147,148,151,164,165]. In persons with chronic SCI, Farkas et al.[19] reported consumption of 319 kcal/d of dietary protein surpassed the DGA recommendation of 184 to 224 kcal/d, representing 17 % of their total daily intake, even though fat-free (protein) body mass is markedly reduced. The value reported by Farkas et al.[19] also exceeded the PHE guidelines on protein consumption and was below recommendations by ADG (Table 6). For most individuals with or without SCI, it is not uncommon to meet or exceed total protein recommendations. The sources of dietary protein largely remain unknown.

Silveira *et al.*[150] identified seafood consumption was low in persons with SCI, although Lieberman *et al.*[164] reported more meat and fish/seafood were consumed compared with non-disabled controls. These conflicting findings may result from the location (Houston, TX[150] *v.* Charlotte, NC[164]) and/or the race/ethnicity (racially/ethnically diverse[150] *v.* Black/White[164]) of the participants and requires additional research.

Many individuals with SCI are not meeting the recommendations for specific amino acids[12,19]. Sabour *et al.*[154] reported that lysine, leucine, valine and isoleucine were the major constituents of total protein intake in persons with SCI, while arginine, alanine and aspartic acid had the lowest daily intake. Groah *et al.*[135] demonstrated that amino acid intake after SCI approached, or met, DGA dietary recommendations except for lysine, leucine, threonine, methionine and cysteine. The same authors noted that men with paraplegia consumed a greater amount of every amino acid compared with men with tetraplegia[135]. This evidence suggests that while protein consumption remains high among persons with SCI, some may still be missing key essential amino acids that can result in malnutrition and health consequences. Moreover, these essential amino acids are necessary for vital functions such as protein synthesis and tissue repair, which is particularly important in this population group that is prone to pressure injuries.

Pressure injuries after SCI precipitously deplete the limited protein stores as the body attempts to heal the wound, generating a rapid transition to malnutrition. This occurs in the presence of already markedly diminished protein reserves (i.e.. skeletal muscle mass). The AND recommends that in the presence of a pressure injury for persons with SCI, albumin and prealbumin laboratory values should be measured[140]. Prealbumin (also known as transthyretin) and albumin have traditionally been utilised as biomarkers of protein nutrition and nutritional status, respectively. A 2012 consensus statement from the AND and the American Society for Parenteral and Enteral Nutrition discouraged the use of prealbumin and albumin as 'sole' indicators of undernutrition due to their susceptibility to systemic inflammation[168]. In their place, the panel recommended the identification of two or more of the following six characteristics for a malnutrition diagnosis: insufficient energetic intake, weight loss, loss of muscle mass, loss of subcutaneous tissue, localised or generalised fluid accumulation that may sometimes mask weight loss, and diminished functional status as measured by handgrip strength[168]. For persons with SCI, lower extremity fluid accumulation is common and due to paralysis, handgrip strength cannot be measured in tetraplegia, thus potentially limiting their utility. The Global Leadership Initiative on Malnutrition recently published guidelines that favoured reinstituting the application of prealbumin as a contributing element to monitor undernutrition in conjunction with C-reactive protein under 15 mg/dl (denoting asymptomatic infection), as prealbumin levels above that are uninterpretable[169–171]. This is supported by recent evidence that suggests prealbumin can supplement other markers such as anthropometrics and clinical history to assess and monitor undernutrition[172]. In a retrospective chart review of 170 SCI patients with pressure injuries, Lussi *et al.*[173] reported 15·3 % and 34 % of the patients only had pathologic laboratory values of prealbumin and albumin, respectively. Poor protein blood

levels, however, were observed in 41 % of the patients[173], suggesting protein blood levels may be a promising measure to assess protein health and pressure injury risk after SCI. Because the use of prealbumin and albumin remain controversial, laboratory examinations, nutritional assessments and anthropometric measures are collectively needed to detect, correct, and treat pressure injuries and protein nutritional deficits after SCI.

Carbohydrate

Carbohydrates (4 kcal/g) are organic compounds in the form of sugars, starches and fibres. The energy source is rich in simple or complex carbohydrates[12]. Simple carbohydrates are largely consumed by persons with SCI[136,153], while added sugars surpass consumption by non-disabled individuals and the DGA, PHE, IOM, and ADG recommendations[150]. Moreover, simple carbohydrates and added sugars (i.e., processed foods) have a high glycaemic index, meaning consumption of these foods cause a rapid hyperglycemia and insulin release that is difficult to control in persons with SCI and prediabetes and type 2 diabetes mellitus.

Several studies identified that about half of the energy content consumed by persons with SCI were from carbohydrates (Table 5)[108,136,147,150–152,164,165], whereas Nightingale *et al.*[115] identified 44 % of the daily energy content came from carbohydrates, respectively. Perret and Stoffel-Kurt[117] observed that persons with acute SCI consume a greater percentage of carbohydrates compared with persons with chronic SCI[117]. Iyer *et al.*[136] and Sabour *et al.*[153] reported higher consumption of carbohydrates in men compared with women with SCI. The latter authors also identified that time since injury, education, and sex were significant predictors for carbohydrate intake in persons with SCI[153]. Farkas *et al.*[19] calculated the average carbohydrate intake for persons with long-standing SCI as 969 kcal/d, a value that exceeds the DGA of 520 kcal/d. This equates to over 50 % of ingested energy content coming from carbohydrates[19]. The DGA, IOM and ADG recommend that carbohydrates make up 45 to 65 % of an individual's total daily energy content when consuming a 2000 kcal/d diet. Although, on average, persons with SCI are consuming less than 2000 kcal/d according to Farkas *et al.*[19], suggesting 45 to 65 % of the total daily energy content coming from carbohydrates should be reduced for persons with SCI.

Carbohydrate consumption should come from complex carbohydrates with a low glycaemic index, such as whole grains. It is well established that whole grains inherently control blood sugar and increase micronutrients, satiation/satiety and fibre consumption[174]. For persons with SCI, data supporting whole-grain consumption are limited. Silveira *et al.*[150] reported that whole grains made up 15 % of the total grains in the diets of individuals with SCI compared with 19 % in non-disabled controls. Similarly, Lieberman *et al.*[164] reported significantly lower daily servings of whole grains in SCI compared (1·20) with non-disabled controls (2·44); however, refined grains did not significantly differ (SCI: 5·42 ± 3·45 *v.* controls: 6·44 ± 6·45), likely to a large variance in the control group. The same authors also showed that 9 % of persons with SCI adhered to consuming ≥ 3

ounces of whole grains compared with 21 % of age- and sex-matched non-disabled controlled[164]. The factors contributing to reduced whole-grain consumption in persons with SCI warrant further investigation because of their cardioprotective effects against the risk of heart disease, type 2 diabetes mellitus and obesity, co-morbidities with a high occurrence in the SCI population.

Fat

Fats (9 kcal/g) are a type of lipid and a dense source of energy. Fats are combinations of SFA and unsaturated fats, such as MUFA, PUFA, n-3PUFA, n-6PUFA and trans-fatty acids[12]. Fats serve various functions throughout the body, including vitamin transport, organ insulation, maintenance of body temperature, formation of the lipid bilayer of a cell and energy storage.

An abundance of evidence indicates that individuals with SCI ingest amounts of dietary fat that are within or surpass DGA, PHE, ADG and IOM recommendations[9,12,19,117,120,135,136,141,150,151,153–155,162,165,175,176], indicating after carbohydrate consumption, a substantial number of energy content are derived from dietary fat (Table 6). Approximately 34 % to 40 % of daily energy comes from fat in persons with chronic SCI (Table 5)[108,115,147,148,150,151,162,164,165,176] where the upper range is characteristic of a typical US diet[162]. In the meta-analysis by Farkas et al.[19], the authors reported that fat intake made up 35 % (663 kcal/d) of the total energetic intake for persons with chronic SCI. The authors noted that fat intake was within the DGA; however, the analysis did not account for the DGA sex- and age-specific ranges for fat intake because of limited power[19]. Therefore, these findings should be interpreted with caution under the notion that age and sex were not considered.

High-fat diets often induce greater food intake and weight gain[177], and high saturated fat consumption negatively influences cardiometabolic health and chronic disease risk[178]. Intake of saturated fat in persons with SCI is close to the limit or exceeds the recommended daily amount of < 10 to 11 % of total energy content according to DGA, IOM, WHO, PHE and ADG (Table 6)[135,136,147,149,150,153,155,162,165]. Tomey et al.[165], Moussavi et al.[176] and Groah et al.[135] reported that saturated fat intake in persons with SCI is higher than the recommended maximum of 10 % of total daily energy content by the USDA's Food Guide Pyramid, 10 % by the National Cholesterol Education Program and 7 % by the AHA, respectively. The DGA, WHO, PHE and ADG recommend limiting saturated fat to < 10 % to 11 % of daily energetic intake, AHA to 7 %, and IOM to as low as possible (Table 6). The PVA guidelines[10] limit saturated fat to 5 to 6 % of total energetic intake. Persons with SCI exceed recommended values of saturated fat, despite an overall reduced energetic intake[150]. The total energy requirements after SCI are less than a non-disabled individual's, and dietary consumption of saturated fats should mimic the reduced energetic intake, by limiting foods discussed in Table 7.

The DGA, AHA, WHO and ADG recommend replacing saturated fats with unsaturated fatty acids as they provide a cardioprotective effect (Table 6)[12]. n-3 and n-6 PUFA are essential in the diet because they cannot be synthesised by humans. Allison et al.[147] reported MUFA consumption did not change following an anti-inflammatory diet, while n-3 and n-6 fatty acids increased and decreased, respectively. Farkas et al.[19] showed greater consumption of MUFA and PUFA in persons with chronic tetraplegia compared with paraplegia; however, this was not a significant finding. Sabour et al.[153] examined dietary fats by injury completeness and noted persons with incomplete injuries consumed more MUFA than those with a complete SCI. According to Silveira et al.[150], MUFA were within healthy ranges, while Groah et al.[135] reported that men and women with paraplegia had lower than the DGA recommended adequate intake of n-6 linoleic acid. According to Sabour et al.[155] linoleic acid consumption exceeded recommended values in persons with SCI. Iyer et al.[136] and Groah et al.[135] also observed that women with SCI exceeded or approached the recommended intake of n-3 linolenic acid, while men with SCI had lower than the recommended intake[135]. The latter finding mirrors the results reported by Sabour et al.[155] for men and women with SCI. Silveira and colleagues[150] noted that linoleic and linolenic acids were within normal ranges according to IOM recommendations. These data indicate additional research is needed on MUFA and PUFA and their health-promoting influence after SCI.

Alcohol

Consumed alcohol (7 kcal/g) is known as ethanol and is not a vital macronutrient[12]. Ethanol is passively absorbed in the digestive system and metabolised mainly in the liver, although some are also metabolised in the stomach[179]. Allison et al.[147] and Nightingale et al.[115] reported that 1·4 % and 3 % of the daily energetic intake came from alcohol in persons with SCI, respectively. Groah and colleagues[135] showed that mean alcohol consumption was overall low (< 10 g/d) among persons with SCI but greater for men than for women with SCI (6·43 v. 2·24 g/d). Another study reported persons with SCI did not consume any alcohol at home[136]. Contrary to these data, other studies report high alcohol consumption in persons with SCI[180–183]. Study participants with SCI are likely to underreport their true alcohol intake on dietary recalls/logs given the stigma that is often related to alcohol consumption and its effects on body weight and physical and mental health[182]. Asking if individuals are current drinkers (i.e. how much they drank in the last month) rather than how much they drink may be a better indicator of alcohol intake in this population.

Fruits and vegetables

Five studies reveal the consumption of fruits and vegetables among individuals with SCI is below the recommended intake according to DGA, ADG, PHE and IOM guidelines[136,150,164,165,184]. These data coincide with the reduced consumption of fruits and vegetables in the population without SCI[12–14]. Silveria et al.[150] recently revealed that consumption of fruits and vegetables were not only below DGA recommended values, but below the persons without SCI. Lieberman et al.[164] and Tomey[165] demonstrated similar results. Knight et al.[184] observed that fruit and vegetable consumption was greatest among persons with SCI with a high activity level of about 30 min/d compared with lower activity levels. However,

the authors did not differentiate between fruit and vegetable intake[184]. Interestingly, 80% of the studies that reported fruit and vegetable intake reported vegetable intake was greater than fruit intake[150,164,165]. This finding is intriguing given many fruits contain a high amount of simple carbohydrates (i.e. monosaccharides and disaccharides), and as reported by the present review, persons with SCI primarily consume simple carbohydrates *v.* complex carbohydrates[153].

Fibre

Fibre (about 1·5–2·5 kcal/g) consists of soluble and insoluble complex carbohydrates and lignin that are intrinsic to, and intact in, plants, such as whole grains, vegetables and fruits. Soluble fibre undergoes bacterial degradation in the large intestine to generate volatile free fatty acids that are then absorbed and used as energy[16]. Several studies have identified fibre intake in persons with SCI is low independent of sex and injury characteristics[19,117,120,135,147,149,150,152,153,162,165,175]. Iyer *et al.*[136] was the only study to report a high fibre consumption of 30 to 33 g/d, while all other studies report an average intake of 12 to 22 g/d[147,153,162,165,175]. Levine *et al.*[162] showed that dietary fibre intake in men with SCI was a third less than the average intake in the non-disabled population. Farkas *et al.*[19] quantified fibre intake in the population with chronic SCI as 17 g/d, which is below the recommendations of the DGA, PHE, ADG, IOM and AHA (Table 6). Low intakes of dietary fibre are likely due in part to the low intake of vegetables and fruits, and potentially whole grains.

Conversely, high fibre diets after SCI may cause negative consequences on neurogenic bowel and bladder conditions. Diets high in fibre (> 20 g/d) may instigate unfavourable changes in bowel function and bowel care programmes that do not occur in the non-disabled population[140]. Cameron *et al.*[185] reported that high dietary fibre before a bowel movement does not have the same effect on bowel function in motor complete SCI as in non-disabled individuals. Furthermore, fibre consumption that is too high without commensurate fluid intake can lead to constipation with an already decreased bowel motility[41]. The excess fluid intake that is required with high fibre diets may also require additional urethral cathing or lead to bowel/bladder accidents. Consequently, the effects on bowel and bladder care can make high dietary fibre diet recommendations inappropriate for individuals living with SCI[186]. Therefore, it is important to develop SCI guidelines on fibre that account for their bowel and bladder programmes (e.g., timing), fluid intake, and their reduced energetic needs relative to an individual without an SCI.

Micronutrients

Micronutrients include vitamins and minerals that are required for cellular communication, water and nutrient transport, the structural integrity of bones, wound healing, and acid–base balance[5]. Several micronutrient intakes are within inadequate ranges in persons with SCI according to the recommended guidelines established by the DGA, IOM, PHE and ADG (Table 6)[19,117,135,152,162,164–166]. Although others have reported below recommended intake values of vitamins A, B_5, B_7, C, D

and E in individuals with chronic SCI[117,135,152,163,165,175,187], as well as below-recommended intake in the minerals Ca, Mg and K[117,135,152,162,163,175,187]. In the meta-analysis by Farkas *et al.*[19], the authors reported below recommended intakes for vitamins A, B_5, B_7, B_9, D and E, and the minerals K and Ca in persons with chronic SCI according to the DGA report. The authors also found excess intake of vitamins B_1, B_2, B_3, B_{12} and K, and the minerals Cu, P, Zn and Na according to recommendations[19]. Na is one of the most widely studied micronutrients after SCI with an average consumption ranging from 2402 to 4300 mg[117,135,150,152,162,165,166]. PVA guidelines recommend Na consumption ≤ 2400 mg/d for all persons with SCI and hypertension. We argue that the high consumption of Na and the prevalence of hypertension in the population[8,11,29] necessitate the need to implement a Na intake ≤ 2400 mg/d for *all* with SCI.

Three studies have evaluated vitamin and mineral supplementation after SCI. Opperman *et al.*[188] reported that nutritional supplementation was common in individuals with long-standing SCI, but no common characteristics (e.g., sex, LOI, age, education, etc.) distinguished users from non-users. According to the authors, 71 % of the sample reported using supplements at least once, with approximately 51 % being classified as consistent supplement users at least twice across the three time points assessed in the study. Both Opperman *et al.*[188] and Walters *et al.*[152] reported that participants with SCI consumed a micronutrient supplement in the form of Ca, a multivitamin, or vitamin D. The latter authors also observed vitamin C supplementation in their participants[152]. Similarly, Wong and colleagues[189] reported that the three most *prescribed* supplements for persons with SCI were multivitamins, vitamins B and vitamin D at an SCI centre. The same authors noted that micronutrient supplementation was significantly associated with age, nutrition risk and serum albumin concentration[189]. Ca and vitamin D supplementation are important for bone health given the high prevalence of osteopenia and osteoporosis in persons with SCI[190]. Furthermore, vitamins B and C deficiencies are linked to anaemia and impaired wound healing[191], both of which are reported at high rates after SCI[192,193]. Multivitamin and mineral supplementation can correct deficiencies, but if there are no deficiencies, they can potentially be harmful. Persons with SCI should be prescribed vitamin and mineral supplements only if specific deficiencies have been detected or to prevent them (such as vitamin D and Ca for bone density), minimising toxicity risk. Health care professionals should place a greater emphasis on following a healthy dietary pattern (described below) to naturally consume vitamins and minerals rather than relying on supplements that may not completely correct deficiencies and carry some risk (e.g., toxicity).

Dietary recommendations after spinal cord injury

Persons with SCI are instructed to adopt a healthy diet[84,194]. But what is a healthy diet for this population? Evidence-based guidelines to ameliorate the risks of obesity and CMS did not exist for the SCI population until recently. The PVA guidelines are the first comprehensive publication to provide data-driven

recommendations on healthy eating for individuals with SCI. When considering the PVA guidelines, one should note the small sample sizes in SCI literature relative to non-disabled research in diet and nutritional status. This inherent delimitation of clinical research targeting a niche patient population restricts the depth of conclusions that can be drawn from a truly evidence-based approach. Therefore, the inaugural iteration of the PVA guideline recommendations corresponds to the several current recommendations for identifying and managing CMS in the non-disabled population. However, the guidelines also factor in the alterations in the body composition and the unique endometabolic physiology that accompany SCI.

The PVA guidelines recommend energetic assessment utilising indirect calorimetry to determine energy expenditure and assess energy needs to implement a heart-healthy nutrition plan focusing on vegetables, fruits, poultry, fish, low-fat dairy products, whole grains, legumes, nuts and non-tropical vegetable oils, while limiting sweets, sugar-sweetened beverages and red meats (Table 7; Fig. 2). The PVA report also limits dietary saturated fat to 5 % to 6 % of the total energetic intake and limits daily Na intake to ≤ 2400 mg for individuals with hypertension (Table 7; Fig. 2)[10].

A reduced emphasis should be placed on limiting macronutrients in diets with persons with SCI, but rather focus on providing a healthy *dietary pattern* (Table 7; Fig. 2). The authors of this review further recommend adopting ≤ 2400 mg of Na for all individuals, regardless of hypertension status given the elevated consumption of Na-dense foods reported in the literature. We also emphasise the importance of lean poultry consisting of a moderate 3 to 4 oz portion, and the consumption of fish two times per week. Vegetables should be consumed between three and four servings per d. They should consist of the five vegetable subgroups (including dark green, red and orange, legumes (beans/peas), starchy, and others). Fruits should favour whole fruits at three least servings per d and significantly limit 100 % fruit juice because of their added/high sugar content and limited fibre content. Emphasis should be placed on low-fat dairy products in the form of milk, yogurt and cheese in small amounts while limiting saturated fat intake below 5–6 %. High-fat, sugary-based sweets and drinks should be replaced with fresh fruit and water, respectively. Flavoured and unflavoured carbonated water and zero energy liquid water enhancers can be used to provide variety and flavour to drinks. Red meat and sweets should be consumed only on special occasions such as birthdays, weddings, holidays, etc. By following the above-referenced dietary patterns, persons with SCI will naturally limit their intake of refined/simple carbohydrates, Na, and saturated fat and increase the consumption of unsaturated fats and fibre. Such dietary patterns will also promote optimal ingestion of micronutrients.

We recommend and recognise the significance of annual dietary assessments (minimally) and nutrition education with a registered dietitian as part of the medical assessment and standard of care for individuals with SCI (Fig. 2). It is our recommendations that in addition to assessing body composition by 3- or 4-compartment modelling[29], registered dietitians should: (1) assess resting metabolism through indirect calorimetry, or, when unavailable, assess RMR/BMR with the Nightingale and Gorgey[116], Chun

et al.[105] or Buchholz et al.[79] SCI-specific prediction equation (2) calculate TDEE using the prediction equation and correction factor by Farkas et al.[9] to determine accurate energy needs, (3) encourage adherence to the SCI dietary guidelines detailed above and balanced nutrition (Table 7, Fig. 2) as an overall healthy lifestyle choice, (4) prescribe dietary supplements when *specific* vitamin and/or mineral deficiencies have been detected or to prevent them when appropriate nutrition/healthy dietary patterns are unsuccessful, and (5) explore dietary anomalies specific to SCI, such as avoidance of food groups that may affect bowel/bladder function post-injury. Periodic assessments with the health care team, including a registered dietitian, should be implemented to manage neurogenic obesity, cardiometabolic risk and nutrition status after SCI and allow the individual themselves to take an active role in their overall health.

Future research

Expert advice advocates for pairing diet and physical activity to mitigate neurogenic obesity and reduce the cardiometabolic risk after SCI. However, the interaction of feeding and activity in SCI has only recently begun to be studied and is likely unique in this population. Furthermore, several barriers, such as transportation, overuse injuries, pain, access to facilities, financial restraints, educational knowledge, disability/SCI-specific resources on exercise, and fear of musculoskeletal or integumentary injury, can limit exercise/physical activity engagement. To mitigate barriers to physical exercise and facilitate improvements in overall health, research focused exclusively on dietary intervention may provide a large-scale 'cure' to chronic diseases in persons with SCI.

Rather than a nutrient (i.e., low-fat diets) focus, nutrition research is turning its attention to dietary patterns as we consume a whole range of foods and food groups, not just nutrients. Evidence suggests that vegetarian and/or plant-based diets create a negative energy balance and decrease the risk of obesity, diabetes and other chronic health ailments[195,196]. Fardet and Boirie[196] examined diet and chronic disease risk from over 300 meta-analyses and systematic reviews published in the last 63 years. The authors reported that plant-based foods were more protective against the risk of developing chronic disease compared with animal-based foods, reinforcing fundamental dietary patterns for good health. Among plant-based foods, whole-grain-based foods had a small edge over fruits and vegetables, while for animal-based foods, dairy products overall were considered neutral on health, and fish was considered protective. Red and processed meats were correlated with elevated chronic disease risk as were sugar-sweetened beverages and highly refined low-fibre grains[196]. These data were further supported by evidence published by the AHA that diets higher in plant-based foods and lower in animal-based products were associated with a lower risk of cardiovascular morbidity and mortality[197]. Plant-based diets may provide an alternative nutritional plan for cardiometabolic risk factors after SCI and minimise overeating by enhancing satiety relative to their need. Future research should focus on the effects of such diets in the SCI population but also attend to energy balance.

Final considerations

In summary, the loss of metabolically active tissue below the LOI, endometabolic pathophysiology, environmental and physical barriers, and poor dietary choices contribute to the suboptimal dietetics, poor body habitus and cardiometabolic risk in SCI[198]. Individuals with SCI meet several evidence-based recommendations published by authoritative guidelines for non-disabled individuals, although they are likely underestimated or overestimated for this population. These guidelines do not account for SCI-induced changes in body composition[199,200], reduced metabolic requirements[19,34,79,87,121], gut dysmotility[41,201] and sympathetic nervous system dysfunction[33,34] and need to be interpreted and used with caution. The PVA guidelines are currently the strongest evidence-based dietary guidelines for the population with SCI and should be followed by persons with SCI and their health care team. Stakeholders and practitioners should have an understanding that the currently limited evidence-based means that guidelines contain might not fully capture the unique nutritional needs of persons with SCI. Therefore, clinical nutritional strategies should also rely on strong inferences from existing studies and use routine monitoring of individual responses to interventions. Because diet-related comorbidities are related to anatomical and physiological changes after SCI, annual nutritional analysis and indirect calorimetry (or, minimally using an SCI-specific BMR/RMR prediction method) with a registered dietitian are encouraged in clinical practice. Future multicentre controlled trials are needed (with less reliance on cross-sectional study design) to collect large data sets on energy expenditure, energy needs and total energetic intake after SCI. These data are needed to help develop comprehensive, evidence-based *dietary reference values* for nutrients specific for persons with SCI aimed at reducing the secondary complications of the injury.

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