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Nutrition Following Following Spinal Cord Injury

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Key Points

Adequate dietary consumption is important in maintaining bone health.

Age and gender, but not level of injury, predict total caloric intake in individuals with SCI.

Individuals with SCI are at a significant risk for malnutrition.

VFSS, BSE and FEES are all appropriate screening tools for diagnosing dysphagia in individuals with SCI.

Several risk factors for dysphagia in individuals with SCI exist, the most common being presence of a tracheostomy, ventilator use, increasing age, and presence of a nasogastric tube.

Individuals with tetraplegia have higher rates of altered glucose metabolism.

Impaired gallbladder emptying is seen in diabetic and obese SCI individuals.

Strict guidelines for diet and exercise interventions are important for patients with SCI due to the inconsistent nature of their energy balance causing weight gain leading to obesity.

A combined diet and exercise program can help patients reduce weight following SCI without compromising total lean mass and overall health.

A diet intervention focusing on anti-inflammatory foods and supplements can reduce inflammation serum markers, but may not show improvement in motor nerve conduction.

Participation in a holistic wellness program is positively associated with improved eating and weight-related behaviours in persons with SCI.

A combined nutrition, exercise and behaviour modification program can help persons with SCI increase metabolically active lean tissue, work efficiency, resting oxygen uptake and strength.

Having a high social participation is positively associated with better self-rated health, and receiving sufficient social support is positively associated with a greater dietary satisfaction, in persons with SCI.

Having a positive perceived food environment is associated with higher physical and mental scores of health-related quality of life, and higher diet satisfaction in persons with SCI.

Dietary counseling alone does significantly improve results in lipid profile; consultation with a registered dietitian should be obtained as individualized diets may enhance compliance.

Blood concentrations of DHA and EPA increased as a result of supplementation; however, no significant changes in lipid profile were identified.

DHA and EPA supplementation increase upper body strength and endurance in persons with SCI.

Vitamin D deficiency is highly prevalent in individuals with chronic SCI.

Individuals with SCI should be screened for vitamin D deficiency according to guideline practices and, if when necessary, replacement therapy should be initiated.

Clinicians should conduct early screening for and treatment of vitamin B₁₂ deficiency.

Creatine supplementation does not result in improvements in muscle strength, endurance or function in weak upper limb muscles.

Creatine supplementation enhances exercise capacity in persons with complete tetraplegia and may promote greater exercise training benefits.

Consumption of a standard liquid meal does not change blood pressure, heart rate or noradrenalin levels in individuals with tetraplegia and postural hypotension.

The consumption of a whey protein plus carbohydrate supplement following fatiguing ambulation improves subsequent ambulation by increasing distance, time to fatigue and caloric expenditure in persons with incomplete SCI.

Nutrient-induced thermogenesis is not decreased in tetraplegic individuals with low sympathoadrenal activity; efferent sympathoadrenal stimulation from the brain is not necessary for nutrient-induced thermogenesis.

Impairment of sympathetic control of the kidney secondary to SCI resulting in tetraplegia does not impact renal sodium conservation in response to dietary salt restriction.

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Nutrition Issues Following Spinal Cord Injury

1.0 Chapter Summary

The nutritional aspect following a spinal cord injury (SCI), is a complex and multifactorial challenge. SCIs can cause devastating short and long-term impairments in physical, cognitive, and psychological status. Specifically, post-SCI cognitive and oropharyngeal impairment may result in decreased tongue movement while chewing and reduced pharyngeal movement, all which ultimately delay the passage of food to the esophagus.

Resting metabolic rate, physical activity and the thermic effect of food are all factors that contribute to energy expenditure and a healthy weight, which can be challenging for patients to achieve and maintain post-SCI. The resting metabolic rate of people with chronic SCI is estimated to be 14-27% lower than their non-SCI counterparts, largely due to reductions in fat-free mass and reduced sympathetic nervous system activity. Physical activity levels of persons with SCI are also generally lower than that of non-SCI persons. Finally, individuals post-SCI can have substantial limitations to their mobility, and thus caregivers may play a large role in meal planning and diet adherence.

In persons with SCI, the usual clinical measures of total body fat, such as weight and body mass index (BMI), underestimate the degree of adiposity. The metabolic alterations related to adverse body composition changes, decreased physical activity and other factors in individuals with SCI are considered atherogenic.

Alterations in the central or peripheral nervous system can result in delayed gastric emptying, prolongation of intestinal transit time, and poor colonic motility, collectively known as neurogenic bowel. Neurogenic bowel has a significant impact on the quality of life of individuals with SCI, contributing to morbidity and even death. Pressure ulcers are common following SCI, and healing can be compromised by suboptimal nutrition status. After a SCI, patients with pressure ulcers have lower zinc, albumin and prealbumin levels than those without pressure ulcers. Impaired nutritional status contributes to delayed or incomplete wound healing. Osteoporosis is common in SCI and results in increased bone fragility and fracture risk (Warden et al. 2001). In addition to pharmacological and other management strategies, supplementation with nutrients such as calcium and vitamin D may play a role in bone health following SCI.

Individuals after SCI commonly experience chronic inflammation, which is associated with immune impairment which can lead to infections and other dysfunctions. Cardiovascular disease appears prematurely in persons with SCI. It is the most frequent cause of death among persons surviving more than 30 years following injury and accounts for 45% of all SCI deaths.

Although little work has been done examining the vitamin profiles of individuals following SCI, it is generally thought that vitamin deficiency is a significant issue. Studies have shown that 16% to 37% of community-dwelling SCI subjects had serum levels below the reference range for vitamins A, C, D and E compared with general population norms. Vitamin D deficiency promotes calcium deficiency and secondary hyperparathyroidism, resulting in further bone loss and exacerbating osteoporosis. Myopathy and nonspecific musculoskeletal pain may also develop as a consequence of vitamin D deficiency. Vitamin B₁₂ deficiency in persons with SCI is reported to be between 5.7% and 19%, with symptoms may include declining gait, depression or fatigue, upper limb weakness, memory loss and worsening pain.

There is a scarcity of intervention studies investigating nutritional status and associated risk for persons with SCI, and more research is needed in this field.

Gaps in the Evidence

- Further investigation of the mechanism underlying Vitamin D deficiency in patients with SCI and whether prophylactic treatment without screening is warranted
- Developing a practical and accessible method of determining energy requirements in patients with SCI, to better tailor individual diet plans
- Improved guidelines for exercise targets specific for patients with SCI that are correlated with reduced CVD risk

2.0 Introduction

Given that traumatic spinal cord injuries (SCI) often occur among young, previously well-nourished individuals, a decline in nutritional status most likely occurs after the injury. These declines are the result of the combined effects of altered metabolism and a change in lifestyle practices. Many secondary complications of SCI are related to changes in energy, glucose, lipid and vitamin metabolism, including undesirable weight gain, cardiovascular disease (CVD) risk, insulin resistance and osteoporosis. Additional nutrition-related complications which can negatively impact quality of life include pressure ulcers and neurogenic bowel and bladder.

Little is known about the most effective health promotion activities, including nutrition interventions, required to promote long-term wellness for persons after a SCI. However, it is clear that adequate nutrition following SCI will help reduce the likelihood of further morbidity associated with post-SCI physiological and metabolic changes. This chapter will summarize what is currently known regarding nutrition issues in the post-acute SCI population.

3.0 Energy Imbalance

To maintain a healthy weight, one must stay in energy balance whereby energy intake equals energy expenditure. Total daily energy expenditure is determined by three factors: resting metabolic rate, physical activity and the thermic effect of food. In addition to lifestyle practices (e.g., smoking) each of these factors is altered following a SCI, rendering it challenging for patients to achieve and maintain energy balance (De Groot et al. 2008). The resting metabolic rate of people with chronic SCI is estimated to be 14-27% lower than their non-SCI counterparts, largely due to reductions in fat-free mass and reduced sympathetic nervous system activity (Buchholz & Pencharz 2004). Physical activity levels of persons with SCI are generally lower than that of non-SCI persons (Buchholz & Pencharz 2004). In addition, a lower thermic effect of food has been reported in persons with a SCI compared to non-SCI controls (Monroe et al. 1998). Three studies have examined dietary intake and malnutrition in the SCI population (Pellicane et al. 2013; Sabour et al. 2012; Wong et al. 2012).

Table 1 Dietary Intake in Individuals with SCI

Author Year Country Research Design Sample Size	Methods	Outcomes
Sabour et al. 2016 Iran	Population: Mean age: 39.5 yr; Gender: males=86, females=17;	1. Measurements were taken at the femoral neck (FN), femoral trochanter (FT), femoral

<p>Observational N=103</p>	<p>Injury etiology: unspecified; Level of injury: cervical=23, thoracic=63, lumbar=17; Level of severity: AIS A=76, B=13, C=4, D=10. Intervention: Participants were assessed upon admission to a research centre. Outcome Measures: Caloric Intake, Protein Intake, Body Mass Index (BMI), Bone Mineral Density (BMD).</p>	<p>intertrochanteric zone (FIZ), lumbar vertebrae (LV), and hip.</p> <ol style="list-style-type: none"> BMD was significantly correlated with BMI at all measured points ($p<0.05$). BMD was significantly greater in female participants at all measured points ($p<0.05$), except at the FN. BMD of the LV was significantly greater in participants with incomplete injury ($p<0.05$) and with paraplegia ($p<0.05$). BMD of the FIZ was significantly greater in participants with AIS D ($p<0.05$). Caloric intake was not significantly correlated with BMD at any point. Protein intake was negatively correlated with BMD of the LV ($r=-0.24$, $p=0.03$). BMD of the LV was negatively correlated with intake of tryptophan, isoleucine, lysine, cysteine, tyrosine, threonine, leucine, methionine, phenylalanine, valine, and histidine ($p<0.05$).
<p>Gorgey et al. 2015 USA Observational N=16</p>	<p>Population: Mean age: 38 yr; Gender: males=16, females=0; Injury etiology: unspecified; Level of injury: C5-7=6, T3-10=10; Level of severity: AIS A=12, B=4; Time since injury: >1yr. Intervention: Participants from the community were assessed and dietary intake was recorded for 4wk. Outcome Measures: Dietary Record Frequency, Percentage of Macronutrients, Caloric Intake, Total Energy Expenditure (TEE), Basal Metabolic Rate (BMR), Fat-Free Mass (FFM), Fat Mass (FM).</p>	<ol style="list-style-type: none"> Caloric intake decreased over 4 wk, but the difference was not significant ($p=0.056$). There was no significant difference ($p=0.93$) or interaction ($p=0.54$) in measuring caloric intake among different dietary record frequencies (1, 3, or 5 d/wk). TEE was significantly higher than caloric intake using 1 d ($p=0.001$), 3 d ($p=0.015$), or 5 d ($p=0.005$) dietary frequency records. BMR was not significantly different from caloric intake for any dietary record frequency, and the two were not significantly correlated. BMR was significantly correlated with total FFM ($r=0.71$, $p=0.005$), leg FFM ($r=0.55$, $p=0.04$), and trunk FFM ($r=0.62$, $p=0.018$). Percentage of macronutrients consumed was not significantly different among dietary frequency records: fat ($p=0.92$), carbohydrates ($p=0.50$), or protein ($p=0.35$). Percentage of fat consumed was significantly different across 4 wk ($p=0.031$), particularly at 2-3 wk ($p=0.034$). There was no significant interaction among dietary record frequencies in measuring fat intake ($p=0.80$). Percentage of carbohydrates consumed was significantly different across 4 wk ($p=0.032$), particularly at 1-3 wk ($p=0.026$) and 2-3 wk ($p=0.014$). There was no significant interaction among dietary record frequencies in measuring carbohydrate intake ($p=0.30$). Percentage of protein consumed was significantly different across 4 wk ($p=0.021$), particularly at 1-3 wk ($p=0.008$). There was no significant interaction among dietary record frequencies in measuring protein intake ($p=0.025$). Percentage of fat consumed accounted for 29% of total FM ($r^2=0.29$, $p=0.037$), 34% of leg FM ($r^2=0.34$, $p=0.022$), and 24% of trunk FM ($r^2=0.24$, $p=0.066$). It was negatively correlated with total

		<p>FFM ($r=-0.53$, $p=0.04$), trunk FFM ($r=-0.54$, $p=0.036$), and BMR ($r=-0.52$, $p=0.059$).</p> <p>10. Percentage of carbohydrates was negatively correlated with % fat ($r=-0.92$, $p<0.0001$), % protein ($r=-0.67$, $p=0.005$), total FM ($r=-0.56$, $p=0.031$), leg FM ($r=-0.64$, $p=0.01$), and trunk FM ($r=-0.50$, $p=0.059$). It was positively correlated with total FFM ($r=0.54$, $p=0.037$) trunk FFM ($r=0.52$, $p=0.046$), and BMR ($r=0.55$, $p=0.04$).</p> <p>11. Percentage of protein was not correlated with FM, FFM, or BMR.</p>
<p>Tsunoda et al. 2015 Japan Observational N=841</p>	<p>Population: Mean age: 61 yr; Gender: males=718, females=123; Injury etiology: unspecified; Level of injury: cervical=245, thoracic=434, lumbar=162; Level of severity: unspecified; Mean time since injury: 27 yr.</p> <p>Intervention: Participants from the community were assessed via questionnaires, and categorized as superior (n=413) or subordinate (n=428) based on food intake score.</p> <p>Outcome Measures: Food Intake, Trans-Theoretical Model (TTM), Self-Efficacy (SE), Outcome Expectancy (OE).</p>	<p>1. Food intake frequency scores between the superior and subordinate groups were significantly different in age ($p<0.001$), gender ($p=0.002$), living situation ($p=0.002$), and care services status ($p=0.007$).</p> <p>2. In univariate analysis, all food intake variables were significantly correlated ($p<0.001$) with TTM (OR range: 2.55-5.89) SE (OR range: 1.93-4.08), and OE (OR range: 1.61-2.76).</p> <p>3. In multivariate analysis, TTM was significantly correlated with the following food intake variables: 'to eat vegetable dishes' (OR=2.76, $p<0.001$), 'to eat green/yellow vegetables' (OR=2.29, $p=0.003$), 'to eat dairy products' (OR=2.75, $p<0.001$), and 'to eat fruits' (OR=1.87, $p=0.003$).</p> <p>4. In multivariate analysis, SE was significantly correlated with the following food intake variables: 'to eat vegetable dishes' (OR=2.12, $p=0.008$), 'to eat dairy products' (OR=1.91, $p=0.001$), and 'to eat fruits' (OR=1.97, $p=0.001$).</p> <p>5. In multivariate analysis, OE was not significantly correlated with any food intake variable.</p>
<p>Lieberman et al. 2014 USA Observational N=100</p>	<p>Population: Mean age: 45.3 yr; Gender: males=78, females=22; Injury etiology: unspecified; Level of injury: paraplegia=43, quadriplegia=57; Level of severity: AIS A=66, B=16, C=18; Mean time since injury: 15.1 yr;</p> <p>Intervention: Participants from the community were assessed and compared to age- and gender-matched controls (n=100).</p> <p>Outcome Measures: Nutrient Intake, Food Intake, Dietary Guideline Adherence.</p>	<p>1. Nutrient intake: participants consumed significantly less calcium (means: 1049 versus 1415 mg; $p=0.004$) and Vitamin D (means: 223 versus 315 IU; $p=0.009$) when compared to controls.</p> <p>2. Food intake: participants consumed significantly fewer mean daily servings of dairy (2.10 versus 4.79, $p<0.0001$), fruit (2.01 versus 3.64, $p=0.002$), whole grains (1.20 versus 2.44, $p=0.007$), and sugars (1.46 versus 3.50, $p=0.002$) when compared to controls.</p> <p>3. Guidelines: fewer participants adhered to recommended daily servings of fruits and vegetables (≥ 5 cups; 40.3% versus 68.7%, $p<0.001$), whole grains (≥ 3 oz; 8.9% versus 21.1%, $p=0.01$), and dairy (≥ 3 cups; 23.4% versus 48.6%, $p<0.001$).</p>
<p>Wong et al. 2014 UK Observational N=150</p>	<p>Population: Median age: 44 yr; Gender: males=46, females=104; Injury etiology: trauma=107, non-trauma=43; Level of injury: cervical=57, thoracic=59, lumbar=22, sacral=1; Level of severity: AIS A=70, B=10, C=28, D=31; Mean time since injury: unspecified.</p>	<p>1. 44.6% of participants were at risk for undernutrition (SNST≥ 11 / MUST≥ 1).</p> <p>2. LOS was significantly higher in at-risk participants than those not at risk (129 versus 85 d, $p=0.012$).</p> <p>3. Increased LOS was associated with higher SNST score ($p=0.012$), higher MUST score ($p=0.013$), new admission ($p<0.01$), prior ICU stay</p>

	<p>Intervention: Participants were assessed upon admission to SCI centers.</p> <p>Outcome Measures: Spinal Nutrition Screening Tool (SNST), Malnutrition Universal Screening Tool (MUST), Length of Stay (LOS), Mortality.</p>	<p>($p<0.01$), low protein ($p=0.022$), low albumin ($p<0.01$), and weight loss $>10\%$ ($p<0.01$).</p> <ol style="list-style-type: none"> 4. Mortality rate at 1 yr was significantly higher in at-risk participants than those not at risk (10.2% versus 1.4%, $p=0.036$). 5. Higher mortality was associated with age ≥ 60 yr ($p<0.01$), readmission ($p=0.018$), pressure ulcers ($p=0.028$), and mechanical ventilation ($p=0.025$). 6. In univariate analyses, predictors of LOS were SNST score ($p=0.003$), MUST score ($p=0.003$), injury level ($p=0.027$), admission type ($p<0.001$), mechanical ventilation usage ($p=0.003$), prior ITU stay ($p<0.001$), serum protein ($p=0.002$), and serum albumin ($p<0.001$). 7. In multivariate analysis, predictors of LOS were admission type ($B=81.23$, $p<0.001$) and serum albumin ($B=-3.62$, $p=0.013$).
<p>Pellicane et al. 2013 USA Observational N=78</p>	<p>Population: SCI ($n=16$): Mean age=41.1 ± 21.2 yr; Gender: males=13, females=3; Level of injury: tetraplegia=8, paraplegia=8; Other injury etiologies: TBI=9, stroke=43, Parkinson's disease (PD)=10.</p> <p>Treatment: Rehabilitation inpatients were assessed by a Registered Dietitian for dietary intake once weekly.</p> <p>Outcome Measures: Calorie and protein intake.</p>	<ol style="list-style-type: none"> 1. Total calorie intake was significantly higher in individuals with SCI compared to stroke ($p<0.003$) and PD ($p<0.45$). 2. Calorie intake per body weight (cal/kg) was significantly higher in individuals with SCI compared to stroke ($p<0.025$). 3. There were no significant differences in total protein intake between varying etiologies. 4. Age ($p<0.001$), gender ($p=0.023$), were significant predictors of calorie and protein intake; admission weight also predicted calorie intake ($p=0.025$).
<p>Krempien & Barr 2012 Canada Observational N=32</p>	<p>Population: Mean age: 30.6 yr; Gender: males=24, females=8; Injury etiology: unspecified; Level of injury: paraplegia=12, quadriplegia=20; Level of severity: unspecified; Time since injury: unspecified.</p> <p>Intervention: Participants with professional athletic history were assessed.</p> <p>Outcome Measures: Three-Factor Eating Questionnaire (TFEQ), Body Mass Index (BMI), Sum of Skinfolds (SoS), Dietary Intake.</p>	<ol style="list-style-type: none"> 1. Participants with low dietary restraint (≤ 11; $n=16$) had significantly lower TFEQ disinhibition score (2.1 versus 3.5, $p<0.05$) and percentage of energy from protein (16.9% versus 18.4%, $p<0.05$) than those with high dietary restraint. 2. There were no significant differences in BMI, SoS, or other dietary intakes (i.e. calories, carbohydrates, fat, fibre) between high and low dietary restraint groups. 3. TFEQ dietary restraint score was not significantly associated with BMI, SoS, or dietary intakes ($p>0.05$). 4. TFEQ disinhibition score was significantly associated with SoS ($r=0.513$, $p=0.003$). 5. TFEQ hunger score was significantly associated with intake of calories ($r=0.354$, $p=0.047$), carbohydrates ($r=0.361$, $p=0.042$), and protein ($r=0.456$, $p=0.009$).
<p>Sabour et al. 2012 Iran Observational N=162</p>	<p>Population: Mean age=34.2 ± 0.7 yr; Gender: males=131, females=31; Level of injury: tetraplegia=94, paraplegia=68; Time since injury=8.0 ± 0.5 yr.</p> <p>Treatment: Face-to-face interviews examining habitual daily food intake patterns.</p> <p>Outcome Measures: Macronutrient intake, simple carbohydrate intake, total calorie intake.</p>	<ol style="list-style-type: none"> 1. Percentages of total energy intake derived from macronutrients were 53% vs. 52% carbohydrate, 10% vs. 11% protein, and 37% vs. 39% fat for men and women, respectively. 2. There was excessive consumption of simple carbohydrates (102.2 ± 40.4 g/d). 3. Males consumed a greater number of calories than women ($p<0.05$). 4. No difference in total intake between those with tetraplegia versus paraplegia. 5. Individuals with incomplete injuries consumed significantly more monounsaturated fatty

		<p>acids than those with complete injuries (p=0.03).</p> <p>6. Age, education and gender significantly predicted calorie intake; time since injury, education, and gender were significant predictors for carbohydrate intake.</p> <p>7. Smoking and level of injury were not related to any dietary variable, and there were no significant predictors for dietary protein and simple carbohydrate intake.</p>
<p>Wong et al. 2012 UK Observational N=150</p>	<p>Population: Age: <60 yr=109, >60 yr=38; Level of injury: C=41.1%, T=42.4%, L=15.8%, S=0.7%; Severity of injury: AISA A=50.4%, B=7.2%, C=20.1%, D=22.3%.</p> <p>Treatment: Assessment of nutritional risk on admission to SCI centers.</p> <p>Outcome Measures: Malnutrition Universal Screening Tool, Body Mass Index (BMI)</p>	<p>1. At the time of hospital admission, 40.0% of the sample were found to be nutritionally 'at risk' and 21.4% were assessed as being 'at high risk' of malnutrition.</p> <p>2. The highest prevalence of nutritional risk was found in groups with prior intensive care unit stays (p=0.035), mechanical ventilation (p=0.183) and 'artificial' nutritional support at the time of arrival (<0.001).</p> <p>3. Nutritional risk showed no significant difference with increased age (p=0.913).</p> <p>4. Compared with 'no-risk' patients, at-risk patients were found to have significantly lower concentrations of total protein, albumin, Hb, creatinine and Mg, with lower BMI and less appetite.</p> <p>5. 'At-risk' patients were found to be receiving more prescribed medications.</p>

Discussion

To ensure adequate dietary intake in a SCI population, regulating the constituents of one's diet is important. Sabour et al. (2016) found that a higher protein intake of essential amino acids is associated with a lower bone mineral density in the lumbar vertebrae of a SCI population. Lieberman et al. (2014) evaluated dietary guideline adherence in individuals with SCI, and found that they consume fewer daily servings of fruit, dairy and whole grains when compared to age-matched controls. This is of concern as Tsunoda et al. (2015) identified in their study of a Japanese SCI population, that total consumption of vegetables, dairy products and fruits is a differentiating factor between those with superior and subordinate healthy diet habits.

Pellicane et al. (2013) found that among four populations (i.e., SCI, stroke, traumatic brain injury, and Parkinson's disease), mean caloric intake, but not protein intake, was significantly higher in the SCI population compared to the others (p=0.004). Both Pellicane et al. (2013) and Sabour et al. (2012) reported that age and gender were significant predictors of calorie and protein intake. Further, Sabour et al. (2012) found that simple carbohydrate consumption was excessive among their sample. There were no differences in calorie intake between those with tetraplegia versus paraplegia.

Excessive or limited dietary intake can leave individuals at risk for malnutrition. Wong et al. (2012) examined rates of malnutrition among individuals with SCI on admission to hospital. The authors reported that 40.0% of the sample were found to be nutritionally 'at risk' and 21.4% were assessed as being 'at high risk' of malnutrition. Wong et al. (2014) also demonstrated that undernutrition is associated with worse clinical outcomes in the year after a SCI. Patients that were undernourished had significantly longer length of stays in rehabilitation (p=0.012), and a greater

12-month mortality rate ($p=0.036$). Thus, there are a significant number of individuals at risk of developing further nutrition-related complications post SCI.

A study by Krempien and Barr (2012) examined eating behaviours and attitudes of professional Canadian Paralympians with a SCI. The authors found that in reference to average individuals with SCI, these athletes had good control of: eating to maintain body weight and composition, knowledge of the types of food they were eating, and were less responsive to physiological hunger cues.

Given alterations in resting energy expenditure, it can be challenging to accurately estimate daily energy requirements for individuals with post-acute SCI. Equations validated and used in non-SCI populations to predict resting metabolic rate overestimate actual measured energy needs in the SCI population (Buchholz & Pencharz 2004). However, Gorgey et al. (2015) found that in a chronic SCI population, caloric intake was on average much lower than the total energy expenditure and basal metabolic rate of this population. Therefore, it has been suggested that energy needs following SCI are best assessed by indirect calorimetry using a metabolic cart (Hadley 2002). Because not all health care centers have access to metabolic carts to measure resting metabolic rate, validated equations specific to the SCI population are needed.

Conclusion

There is level 5 evidence (from one observational study; Sabour et al. 2016) that elevated protein intake can lower bone mineral densities in individuals with SCI.

There is level 5 evidence (from two observational studies; Tsunoda et al. 2015; Lieberman et al. 2014) that consumption of whole grains, vegetables, fruits and dairy products are important in maintaining adequate dietary intake.

There is level 5 evidence (from two observational studies; Pellicane et al. 2013; Sabour et al. 2012) that age and gender, but not level of injury, predict total caloric intake in individuals with SCI; further, level 5 evidence (from one observational study; Gorgey et al. 2015) suggests that individuals with chronic SCI often have a negative energy balance, consuming fewer calories than they burn.

There is level 5 evidence (from two observational studies; Wong et al. 2014; Wong et al. 2012) that individuals with SCI are at a significant risk for malnutrition and are at risk of worse clinical outcomes in the first year after injury.

Adequate dietary consumption is important in maintaining bone health.

Age and gender, but not level of injury, predict total caloric intake in individuals with SCI.

Individuals with SCI are at a significant risk for malnutrition.

4.0 Dysphagia

Swallowing traditionally occurs in three phases. In the oral preparatory phase, food is inserted into the mouth mixed with saliva and broken down to form a bolus, this bolus is then transferred to the pharynx initiating the swallowing reflex. While the preparatory phase is completely voluntary, the latter two phases, once initiated, are involuntary and irreversible. The pharyngeal phase is the shortest but most complex of the phases. In the pharyngeal phase, the following series of movements occur to ensure successful deglutition of the bolus: the tongue blocks entry to oral cavity, the soft palate blocks entry to the nasal cavity, the vocal folds close to protect the airway to the lungs, the epiglottis covers entry to the trachea and the upper esophageal sphincter opens to allow passage of the bolus to the esophagus. Finally, in the esophageal phase, the bolus is directed via primary and secondary peristalsis toward the stomach. Dysphagia is a swallowing disorder characterised by unsuccessful or incomplete deglutition during any phase of eating, from the detection of food in the buccal cavity to manipulation and swallowing of food of every consistency (Papadopolou et al. 2013). Dysphagia is most common in the pharyngeal phase, but can occur during any of the three phases (Chaw et al. 2012).

Dysphagia occurs in a variety of clinical populations (Khan et al. 2014, Martino et al. 2005, Suttrup & Warnecke, 2016). In SCI, dysphagia is most often present as oropharyngeal type, seen in those with an injury to the cervical spine (Chaw et al. 2012). Damage along the cervical spine impedes the pathways of the glossopharyngeal, vagus and hypoglossal cranial nerves, all of which are fundamental in swallowing (Finsterer and Grisold, 2015). The glossopharyngeal cranial nerve provides efferent motor fibers to the stylopharyngeus muscle which controls the elevation of the larynx and pharynx, and is involved in parasympathetic control of the salivary gland (Ong and Chong, 2010). The vagus cranial nerve innervates the palatoglossus muscle which is involved in the initiation of swallowing, the palatopharyngeal muscle which controls the movement of the food bolus, and the pharyngeal constrictor muscles which pushes food into the esophagus and allows for shortening and widening of the pharynx during swallowing (Ong and Chong, 2010). Finally, the hypoglossal nerve innervates nearly all the muscles of the tongue (Papadopolou et al. 2013). Location of the injury is often a primary determinant of dysphagia severity; injuries to C1-C2 vertebrae may result in no sensory awareness of swallowing whilst damage at C4-C6 has a tendency to cause poor laryngeal movement and reduced cricopharyngeal opening (Logemann, 1998).

The prevalence of dysphagia varies considerably with studies reporting incidences of 16-30% among SCI patients (Takizawa et al. 2016; Shin et al. 2011; Seidl et al. 2010; Stinneford et al. 1993) whilst other articles have reported rates of 36-60% (Gordan et al. 2009). Dysphagia was reported in one study to be most prevalent among injuries within the mid-cervical level (C3-C5) with 40% of dysphagic patients exhibiting an injury to the C4 vertebrae (Seidl et al. 2010).

For the majority of SCI patients who present with dysphagia during acute care, the problem tends to be transient and a natural recovery usually occurs as rehabilitation progresses (Shin et al. 2011). However, for patients with persistent dysphagia, complications may result including malnutrition, dehydration, aspiration, and increased hospitalization (Papadopolou 2013). Aspiration is the greatest clinical concern; it occurs in the pharyngeal phase of swallowing, and is defined as the movement of the bolus from the oropharynx past the vocal cords into the trachea and respiratory tract, as a result of a dysfunctional epiglottis inversion during the swallowing reflex. Aspiration can lead to life threatening complications including pneumonia, chemical pneumonitis, respiratory insufficiency, transient hypoxemia, atelectasis, bronchospasm and death (Chaw 2012, Shem et al. 2012b).

Due to the nature of dysphagia, there is a varied symptomology amongst SCI patients including: coughing, choking, repeated clearing of the throat after swallowing, drooling, watery-eyes or runny nose during or after feeding, audible swallowing, regurgitation, and aspiration (Chaw et al. 2012). Logemann (1998) also adds that SCI patients can also experience a number of oral-motor deficits such as reduced tongue motion, delayed pharyngeal swallowing, and unilateral or bilateral pharyngeal wall dysfunction. Symptoms can be observed via a plethora of measures including bedside swallowing evaluation (BSE) assessments, Videofluoroscopy Swallow Study (VFSS), Fiberoptic Endoscopic Evaluation of Swallowing (FEES), and barium swallow tests (Papadopoulou et al. 2013).

4.1 Diagnosis of Dysphagia

Dysphagia can be diagnosed and monitored with many different screening tools. Common to all these screening tools is the ability to differentiate patients at risk for aspiration and laryngeal penetration from those who can consume solids and/or liquids safely (Papadopoulou 2013). One of the most common and initial forms of screening for dysphagia is the Bedside Swallowing Evaluation (BSE). The BSE is important in helping clinicians decide the degree of safety in oral feeding, and foresee warnings about potential complications. The BSE allows speech-language pathologists to conduct a case history interview about the patient's nutritional and respiratory medical status, and an examination of oral motor abilities including labial, palatal, lingual, pharyngeal wall, and laryngeal muscle control; along with an assessment of swallowing (Workman and Treole, 2002). Patients are diagnosed with dysphagia if the speech language pathologist performing the BSE observes signs of aspiration, such as coughing, choking, or liquid and/or food present in or around the tracheostomy stoma, limited or uncoordinated laryngeal movement, or a wet sound in vocal quality after drinking (Shem et al. 2012).

Videofluoroscopic Swallow Studies (VFSS) are considered to be the gold standard for evaluation of oropharyngeal dysphagia as it provides direct visualisation of the movement of the jaw, palate, pharynx, larynx, esophagus and tongue during the swallowing motion (Shem et al. 2012b). Videofluoroscopy works by ingestion of different types of foods ranging from very liquid to solid made radiopaque with a small dose of barium sulfate, via syringe or spoon in order to measure muscle movements during swallowing (Ryu et al. 2012). Not only can it be used to diagnose the existence of dysphagia in a patient, it can also be used to determine the etiology of dysphagia and under what conditions the patient can swallow safely (Shem et al. 2012b). Dysphagia is diagnosed if a patient undergoing the VFSS presents with any of the following: pooling of the test material in pyriform sinuses/valleculae, decreased laryngeal elevation, lack of epiglottic inversion, laryngeal penetration and aspiration (Shem et al. 2012b). Not only does this assessment require specially-trained technicians, but patients who are in intensive care, unable to sit in the correct position, or those with a high risk of aspiration may not be suitable to undergo VFSS (Papadopoulou et al. 2013).

The Fiberoptic Endoscopic Evaluation of Swallowing (FEES) is a widely used diagnostic procedure in exploring the swallowing process when patients are not suitable for VFSS. It is performed using a flexible laryngoscope that is fed through the nasal cavity that allows imaging of the epiglottis, sinus piriformis and arytenoid cartilages; consumption of a bolus (liquid or semi-liquid food) allows for examination of the swallowing reflex and presence of aspiration, retention and laryngeal penetration can be observed (Wolf & Meiners, 2003). FEES can be performed in a sitting eating position, or for bed-ridden patients the head of the bed is raised 45 degrees or more to compensate (Papadopoulou et al. 2013). FEES is well tolerated, administrable to bed-ridden patients, and can be used repeatedly to monitor therapy progress. FEES is disadvantaged in that

the oral and esophageal phase of swallowing cannot be examined; additionally, documentation and reporting of FEES can be time-consuming and difficult to complete (Hey et al. 2009).

Table 2 Diagnostic Measures of Dysphagia

Author Year Country Research Design Sample Size	Methods	Outcomes
Shem et al. 2012 USA Prospective Cohort N=40	<p>Population: Mean age: 41.0 yr; Gender: males=31, females=9; Injury etiology: Motor vehicle accident=9, Fall=7, Gunshot wound=3, Diving=6, Bicycle accident=4, Motorcycle accident=2, Other=9; Level of injury: C4 or higher=29, C3 or lower=11; Level of severity: Not reported; Mean time since injury: 14.3 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a Videofluoroscopy Swallow Study (VFSS).</p> <p>Outcome Measures: Dysphagia incidence.</p>	<ol style="list-style-type: none"> 16/40 patients were diagnosed with dysphagia by the BSE. While VFSS found 12 cases of dysphagia and four cases of aspiration.
Shem et al. 2012b USA Prospective Cohort N=39	<p>Population: Mean age: 41.6 yr; Gender: males=30, females=9; Injury etiology: Motor vehicle accident=9, Fall=7, Gunshot wound=3, Diving=6, Bicycle accident=4, Motorcycle accident=2, Other=8; Level of injury: C4 or higher=28, C3 or lower=11; Level of severity: Not reported; Mean time since injury: 14.1 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a Videofluoroscopy Swallow Study (VFSS). Sensitivity and specificity of the BSE was determined in reference to the VFSS.</p> <p>Outcome Measures: Dysphagia incidence, sensitivity, specificity, positive predictive value, negative predictive value of the BSE in reference to VFSS.</p>	<ol style="list-style-type: none"> 15 participants were diagnosed with dysphagia based on the BSE. 11 participants were diagnosed with dysphagia, and four participants with aspiration, based on the VFSS. Of the 26 participants who underwent both BSE and VFSS, a dysphagia diagnosis was different in only one case. When comparing the BSE to the VFSS, the BSE's sensitivity was 100% (95% CI: 71.5% to 100%), specificity was 93.3% (95% CI: 68.1% to 99.8%), positive predictive value was 91.7% (95% CI: 61.5% to 99.8%), and the negative predictive value was 100% (95% CI: 76.8% to 100%). For diet recommendations, when comparing the BSE to the VFSS, the BSE's sensitivity was 76.9% (95% CI: 46.2% to 95%), specificity was 84.6% (95% CI: 54.6% to 98.1%), positive predictive value was 83.3% (95% CI: 51.6% to 97.9%), and the negative predictive value was 78.6% (95% CI: 49.2% to 95.3%).
Shem et al. 2011 USA Prospective Cohort N=29	<p>Population: Mean age: 41.0 yr; Gender: males=22, females=7; Injury etiology: Motor vehicle accident=5, Fall=7, Gunshot wound=3, Diving=3, Bicycle accident=3, Motorcycle accident=3, Other=5; Level of injury: C1=1, C2=3, C3=7, C4=10, C5=4, C6=2, C7=2; Level of severity: Not reported; Mean time since injury: 12.9 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a</p>	<ol style="list-style-type: none"> 12/29 patients were diagnosed with dysphagia by BSE, and 9/29 with VFSS. VFSS also showed that 4/29 patients had aspiration.

	Videofluoroscopy Swallow Study (VFSS). Outcome Measures: Dysphagia incidence.	
Seidl et al. 2010 Germany Retrospective Cohort N=175	Population: Mean age: 43.5 yr; Gender: males=144, females=31; Injury etiology: Fracture 1 vertebral body=73, Fracture 2 vertebral bodies=47, Fracture >2 vertebral bodies=16, Spondylodiscitis=15, Contusio spinalis=10, Tumour=5, Spinal stenosis=4, Nuclear pulposus prolaps=3, Knife wound=1, Postoperative=1; Level of injury: C0=1, C1=1, C2=4, C3=14, C4=58, C5=53, C6=33, C7=6, C8=5; Level of severity: Frankel A=103, Frankel B=19, Frankel C=21, Frankel D=24, Frankel E=8; Time since injury: Participants were recruited within 8 wk of their injury. Intervention: Swallowing ability was examined with a clinical bedside bolus-swallowing test by a speech therapist, and an endoscopic-swallowing test. Outcome Measures: Dysphagia incidence.	<ol style="list-style-type: none"> Swallowing disorders were identified in 28/175 patients. Swallowing disorders were most common in patients with the highest grade of sensorimotor deficit, however this was not significant ($p>0.05$).
Kirshblum et al. 1999 USA Case Control N=187	Population: Mean age: 44.3 yr; Gender: males=156, females=31; Injury etiology: Fall=64, Motor vehicle accident=65, Gunshot wound=9, Diving=31, Other=18; Level of injury: C7 and below=15, C6=21, C5=43, C4=63, C3=25, C2 and above=20; Level of severity: AIS A=71, AIS B=5, AIS C=59, AIS D=48, AIS E=4; Median time since injury: 30 days (range: 5-264 days). Intervention: Videofluoroscopic Swallowing Study (VFSS) to confirm the presence of dysphagia. Outcome Measures: Dysphagia incidence.	<ol style="list-style-type: none"> VFSS confirmed the presence of dysphagia in 31 patients.
Chaw et al. 2012 USA Observational N=68	Population: Mean age: 43.0 yr; Gender: males=57, females=11; Injury etiology: Motor vehicle accident=18, Fall=13, Diving=9, Bicycle accident=5, Gunshot wound=5, Motor cycle accident=4, Medical=4, Myelopathy=4, Trauma=4, Other=2; Level of injury: C1=2, C2=6, C3=14, C4=6, C4 (incomplete)=21, C5 (incomplete)=10, C6 (incomplete)=4, C7 (incomplete)=3, C8 (incomplete)=2; Level of severity: Complete=28, Incomplete=40; Mean time since injury: 31.8 days. Intervention: Bedside Swallowing Examination (BSE), which was followed by Videofluoroscopy Swallow Study (VFSS) within 72 hr. Outcome Measures: Dysphagia incidence.	<ol style="list-style-type: none"> BSE results found 21 cases of dysphagia. Of these 21, 14 were diagnosed with dysphagia via VFSS.
Abel, Ruf & Spahn 2004 Germany Observational N=73	Population: Mean age: 42.9 yr; Gender: males=51, females=22; Injury etiology: Trauma=56, Spondylitis=5, Tumour=3, Other=9; Level of injury range: C1-C7; Level of severity: AIS A (complete)=41, incomplete=32; Mean time since injury: Not reported. Intervention: Patients with cervical SCI admitted to an initial care facility between January 1997 to December 2000. Prevalence of pneumonia via x-rays, and dysphagia via methylene blue test and videofluoroscopic	<ol style="list-style-type: none"> 26 cases of dysphagia were confirmed: three cases of severe impairment of deglutition, eight cases of moderate impairment, and 15 of minimal impairment. 24 patients had only one episode of pneumonia, 11 patients experienced early and multiple episodes of late pneumonia.

	swallowing, were determined at intake and discharge. Changes in dysphagia status were observed after tracheostomies, surgery to the cervical spine and dietary restrictions. Outcome Measures: Dysphagia and pneumonia incidence.	3. Incidence of late or multiple late pneumonia was significantly higher for patients with dysphagia ($p<0.01$).
Wolf & Meiners 2003 Germany Cross-sectional N=51	Population: Mean age: 43.4 yr; Gender: males=35, females=16; Injury etiology: Not reported; Level of injury: C2=20, C4=19, C5=7, C6=4, C7=1; Level of severity: AIS A=30, AIS B=13, AIS C=7, AIS D=1, AIS E=0; Time since injury: Less than 3 months. Intervention: Presence of dysphagia was determined by Fiberoptic Endoscopic Examination of Swallowing (FEES) upon admission and was followed-up at intervals of 4 to 6 wk until discharge. Patients were given artificial respiration and supplementary treatments (tracheotomy, nasogastral tube, speech therapy) dependent on their level of dysphagia determined by admission FEES. Outcome Measures: Prevalence and severity of dysphagia by five levels of laryngeal function (level 1-complete dysfunction, level 2-severely impaired, level 3-moderate impairment, level 4-mild dysfunction, level 5-unimpaired function), risk factors for dysphagia.	1. The use of the FEES was able to detect severe dysphagia with major aspiration in 21 patients, mild dysphagia with a leading symptom of either laryngeal edema or mild aspiration with sufficient coughing reflex in 20 patients, and no detectable dysphagia in 10 patients. 2. FEES was not only found to be effective in detecting and classifying patients within the levels of dysphagia, it was also found to be useful in evaluating the treatment program with a high number of patients experiencing successful treatment outcomes. 3. Multiple FEES examinations were performed with a range of one to nine examinations conducted per patient.

Discussion

The majority of studies assessing the diagnosis of dysphagia in a SCI population relied on either the BSE or VFSS as their screening tools. Four studies used both measures for diagnosing dysphagia (Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011). The incidence of dysphagia for these studies was on average, 40%. Shem et al. (2012b) performed a novel, prospective, psychometric comparison between the BSE and VFSS, for patients recently admitted to acute care for their SCI, to determine if the BSE was as accurate as the VFSS. The sensitivity, specificity, positive predictive and negative predictive values were calculated for the BSE in reference to the VFSS. The BSE performed comparatively well with the VFSS, with a sensitivity of 100% and a specificity of 93.3%. There was only one case of a misdiagnosis of dysphagia by the BSE, which the VFSS ruled out, giving a positive predictive value of 91.7%. Importantly, the BSE was able to rule out to the same extent as the VFSS individuals not at risk of dysphagia, with a negative predictive value of 100%. The results of this study indicate that the BSE can be an excellent screening tool for dysphagia in a SCI population that performs nearly as well as the VFSS. This is important given that the VFSS is more expensive, invasive, requires patients to be seated in a certain way, and protocols for VFSS may vary from facility to facility. However, the authors found when comparing diet recommendations, the BSE did not perform nearly as well as the VFSS, with diets recommended by BSE being more stringent and limiting the amount of safe foods patients can eat. Therefore, the VFSS still has an important clinical role in determining nutrition recommendations after a dysphagia diagnosis.

Wolf and Meiners (2003), was the only study that examined FEES as a method of diagnosis for dysphagia in a SCI population. The authors used FEES to diagnose dysphagia based on five levels of swallowing dysfunction, where levels 1 and 2 indicated severe swallowing impairment, level 3

was presence of aspiration, level 4 was mild aspiration or laryngeal edema, and level 5 was no signs of laryngeal dysfunction. They found a high incidence of dysphagia of 80%; however, only 40% had severe dysphagia whereas 50% had mild dysphagia. Treatment was given to dysphagic individuals proportional to their level of swallowing impairment that included: artificial respiration, positive end expiratory pressure, tracheostomy, nasogastric tubes, and breathing exercises. FEES was useful in that it allowed for repeated administration to track patients' treatment progress in ICU settings.

Conclusion

There is level 5 evidence (from several observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Seidl et al. 2010, Abel, Ruf & Spahn 2004, Kirshblum et al. 1999) that VFSS and BSE are adequate measures of diagnosing dysphagia in a SCI population.

There is level 5 evidence (from four observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011) that VFSS and BSE are comparable in diagnosing dysphagia in a SCI population.

There is level 5 evidence (from one observational study: Wolf and Meiers 2003) that FEES is an adequate tool to diagnose dysphagia and monitor treatment progress in a SCI population.

VFSS, BSE and FEES are all appropriate screening tools for diagnosing dysphagia in individuals with SCI.

4.2 Risk Factors for Dysphagia

The etiology of dysphagia in relation to acute cervical spinal cord injury is not completely understood. In addition to damaged nerves, the consortium of spinal cord medicine clinical practice guidelines on respiratory management following spinal cord injury (2005) has identified the following risk factors for dysphagia: supine position, spinal shock, slowing of gastrointestinal tract, gastric reflux, inability to turn the head to spit out regurgitated material, nausea inducing medications, recent anterior cervical spine surgery, presence of a tracheostomy, and age. Proper identification of risk factors in individuals at risk of dysphagia, can allow for early dietary intervention that could prevent secondary complications.

Table 3 Risk Factors for Dysphagia

Author Year Country Research Design Sample Size	Methods	Outcomes
Ihalainen et al. 2017 Finland Prospective Cohort N=37	Population: Mean age: 61.2 yr; Gender: males=31, females=6; Injury etiology: Sport=2, Transport=6, Fall=28, Unknown=1; Level of injury: C1-C4=32, C5-C8=4, Unknown=1; Level of severity: AIS A=8, AIS B=3, AIS C=5, AIS D=21; Mean time since injury: 16.4 days. Intervention: Clinical swallowing trial and Videofluoroscopic	<ol style="list-style-type: none"> Results of the PAS divided patients into two groups: penetrator/aspirators (n=19) and non-penetrator/aspirators (n=18). The two groups were significantly different in necessity for bronchoscopy (p=0.042), coughing

	<p>Swallowing Study (VFSS) to identify laryngeal penetration or aspiration.</p> <p>Outcome Measures: Rosenbek's Penetration-Aspiration Scale (PAS), risk factors for laryngeal penetration or aspiration.</p>	<p>(p=0.007), and changes in voice quality (p=0.004).</p>
<p>Shem et al. 2012 USA Prospective Cohort N=40</p>	<p>Population: Mean age: 41.0 yr; Gender: males=31, females=9; Injury etiology: Motor vehicle accident=9, Fall=7, Gunshot wound=3, Diving=6, Bicycle accident=4, Motorcycle accident=2, Other=9; Level of injury: C4 or higher=29, C3 or lower=11; Level of severity: Not reported; Mean time since injury: 14.3 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a Videofluoroscopy Swallow Study (VFSS).</p> <p>Outcome Measures: Risk factors for dysphagia, medical complications resulting from dysphagia.</p>	<ol style="list-style-type: none"> 1. Significant risk factors for dysphagia included: age (p=0.016), tracheostomy tube (p=0.013), mechanical ventilation (p=0.003), and nasogastric tubes (p=0.027). 2. There was a trend towards significance for halo vest usage as a risk factor (p=0.076). 3. In terms of medical complications, individuals with dysphagia have significantly higher occurrences of pneumonia (p=0.004). 4. There was a statistical trend for longer length of stays for individuals with dysphagia (p=0.064), and days to BSE was marginally significant (p=0.047).
<p>Shem et al. 2012b USA Prospective Cohort N=39</p>	<p>Population: Mean age: 41.6 yr; Gender: males=30, females=9; Injury etiology: Motor vehicle accident=9, Fall=7, Gunshot wound=3, Diving=6, Bicycle accident=4, Motorcycle accident=2, Other=8; Level of injury: C4 or higher=28, C3 or lower=11; Level of severity: Not reported; Mean time since injury: 14.1 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a Videofluoroscopy Swallow Study (VFSS). Sensitivity and specificity of the BSE was determined in reference to the VFSS.</p> <p>Outcome Measures: Risk factors for dysphagia, medical complications resulting from dysphagia.</p>	<ol style="list-style-type: none"> 1. Dysphagia diagnosis was significantly associated with: mechanical ventilation (p=0.005), presence of pneumonia (p=0.007), older age (p=0.015), a tracheostomy (p=0.019), a nasogastric tube (p=0.023), and a greater length of stay (p=0.023). 2. There was a trends towards significance for halo vest use as a risk factor (p=0.066), and days to BSE (p=0.068) as a complication.
<p>Shem et al. 2011 USA Prospective Cohort N=29</p>	<p>Population: Mean age: 41.0 yr; Gender: males=22, females=7; Injury etiology: Motor vehicle accident=5, Fall=7, Gunshot wound=3, Diving=3, Bicycle accident=3, Motorcycle accident=3, Other=5; Level of injury: C1=1, C2=3, C3=7, C4=10, C5=4, C6=2, C7=2; Level of severity: Not reported; Mean time since injury: 12.9 days.</p> <p>Intervention: Presence of dysphagia was determined in patients using a Bedside Swallowing Examination (BSE), and a Videofluoroscopy Swallow Study (VFSS).</p> <p>Outcome Measures: Risk factors for dysphagia, medical complications resulting from dysphagia.</p>	<ol style="list-style-type: none"> 1. Significant risk factors for dysphagia included: age (p=0.028), presence of a tracheostomy (p=0.047), and use of a nasogastric tube (p=0.029). 2. Non-significant risk factors included: presence of a halo vest (p=0.081), posterior spine surgery (p=0.090), anterior spine surgery (p=0.82), gender (p=0.43), presence of a head injury (p=0.26), high versus low tetraplegia (p=0.79), complete injury (p=0.30) and presence of a collar (p=0.97). 3. Individuals with dysphagia had significantly higher occurrences of pneumonia (p=0.016). 4. As well for individuals with dysphagia, complications that trended towards significance

		included: need for bronchoscopy (p=0.054) and length of stay (p=0.064).
Shin et al. 2011 Korea Retrospective Cohort N=121	<p>Population: Mean age: 44.9 yr; Gender: males=105, females=16; Injury etiology: Motor vehicle accident=81, Fall=26, Diving=4, Other traumatic=7, Non-traumatic=3; Level of injury: Cervical SCI; Level of severity: AIS A=72, AIS B=20, AIS C=19, AIS D=10; Mean time since injury: 178.35 days.</p> <p>Intervention: Presence of dysphagia/aspiration was determined using Videofluoroscopy Swallow Study (VFSS).</p> <p>Outcome Measures: Aspiration prevalence, risk factors for aspiration.</p>	<ol style="list-style-type: none"> 1. VFSS found aspiration in 10 patients. 2. Aspirators compared to non-aspirators were significantly older in age (p=0.044). 3. Aspiration was more common for patients with tracheostomy (p=0.011). 4. Significantly higher incidences of aspiration were found for patients with symptoms of dysphagia (p=0.002) and signs of dysphagia (p=0.001).
Seidl et al. 2010 Germany Retrospective Cohort N=175	<p>Population: Mean age: 43.5 yr; Gender: males=144, females=31; Injury etiology: Fracture 1 vertebral body=73, Fracture 2 vertebral bodies=47, Fracture >2 vertebral bodies=16, Spondylodiscitis=15, Contusio spinalis=10, Tumour=5, Spinal stenosis=4, Nuclear pulposus prolaps=3, Knife wound=1, Postoperative=1; Level of injury: C0=1, C1=1, C2=4, C3=14, C4=58, C5=53, C6=33, C7=6, C8=5; Level of severity: Frankel A=103, Frankel B=19, Frankel C=21, Frankel D=24, Frankel E=8; Time since injury: Participants were recruited within 8 wk of their injury.</p> <p>Intervention: Swallowing ability was examined with a clinical bedside bolus-swallowing test by a speech therapist, and an endoscopic-swallowing test.</p> <p>Outcome Measures: Risk factors for dysphagia.</p>	<ol style="list-style-type: none"> 1. Swallowing disorders were most common in patients with the highest grade of sensorimotor deficit, however this was not significant (p>0.05). 2. Posterior and combined approach surgery patients had a non-significant higher rate of swallowing disorders (p>0.05). 3. Swallowing disorders increased significantly with lower levels of tetraplegia (p<0.05), tracheotomies (p<0.05), and duration of ventilation (p<0.05).
Brady et al. 2004 USA Case Control N=131	<p>Population: <i>Dysphagia Group (n=72):</i> Mean Age=55.5yr; Gender: Male=13, Female=59; Type of Injury: Traumatic injury (66.4%), Non-traumatic (30.6%); Respiratory Status: Tracheotomy tube=24; Type of Cervical Spine Surgery: Anterior=31, Posterior=11, Combined=10, None=20. <i>Non-Dysphagia Group (n=59):</i> Mean Age=55.9yr; Gender: Not Reported; Type of Injury: Traumatic=40, Non-traumatic=19; Respiratory Status: Tracheotomy tube=6; Type of Cervical Spine Surgery: Anterior=14, Posterior=18, Combined=4, None=23.</p> <p>Intervention: Chart reviews were conducted on patients admitted to two rehabilitation hospitals within a 27mo period. All patients were screened for dysphagia upon admission. Those clinically suspected of experiencing dysphagia were referred for further evaluation with a speech-language pathologist.</p> <p>Outcome Measures: American Speech-Language-Hearing Association National Outcomes Measurement swallowing level</p>	<ol style="list-style-type: none"> 1. The presence of a tracheostomy tube to assist the patient was significantly predictive of the occurrence of dysphagia (p=0.002) with one in three dysphagic patients having had a tracheostomy inserted compared to one in ten non-dysphagic patients. 2. There was a significant difference between the two groups concerning the type of surgery. Dysphagic patients were significantly more likely to have undergone cervical surgery, particularly the anterior approach, compared with the non-dysphagic patients (p=0.02). 43.1% of patients with dysphagia underwent anterior approach surgery. 3. ASHA NOMS scores at discharge revealed that the presence of a tracheostomy tube (p=0.02), receiving fewer days of treatment

	scale (ASHA NOMS), type of surgery, respiratory status. Reviewed data had been collected at admission and at discharge from rehabilitation.	(p=0.04) and demonstrating aspiration (p=0.0001) were negative predictors of dysphagia recovery.
Kirshblum et al. 1999 USA Case Control N=187	<p>Population: Mean age: 44.3 yr; Gender: males=156, females=31; Injury etiology: Fall=64, Motor vehicle accident=65, Gunshot wound=9, Diving=31, Other=18; Level of injury: C7 and below=15, C6=21, C5=43, C4=63, C3=25, C2 and above=20; Level of severity: AIS A=71, AIS B=5, AIS C=59, AIS D=48, AIS E=4; Median time since injury: 30 days (range: 5-264 days).</p> <p>Intervention: Videofluoroscopic Swallowing Study (VFSS) to confirm the presence of dysphagia.</p> <p>Outcome Measures: Risk factors for dysphagia.</p>	1. Significant predictors for dysphagia included: older age (p=0.028), history of tracheostomies (p<0.0001), ventilator status (p<0.001), anterior approach cervical spine surgery (p=0.016), higher level injuries (p=0.012), and ASIA impairment classification (p=0.02).
Hayashi et al. 2017 Japan Retrospective case series N=298	<p>Population: Median age: 64.0 yr; Gender: males=256, females=42; Level of injury: C3-C7; Level of severity: AIS A=98, AIS B=38, AIS C=127, AIS D=35; Time since injury: approximately 3 days.</p> <p>Intervention: Retrospective review of patients with acute cervical spinal cord injury for presence of dysphagia or not. Suspected associated risk factors for dysphagia were also recorded and put into a multivariable logistic regression.</p> <p>Outcome Measures: Risk factors for dysphagia.</p>	1. Multivariable logistic regression analyses revealed that the following were significant risk factors for dysphagia: age greater than 72 yr (p=0.02), AIS A or B (p=0.008), and presence of a tracheostomy (p<0.001).
Shem et al. 2005 USA Case Series N=68	<p>Population: Mean Age=33yr; Severity of Injury: ASIA A=36, ASIA B=7, ASIA C=12, ASIA D=13; Level of Injury: C2=1, C3=11, C4=56; Mean Time Post-Injury=8.8d.</p> <p>Intervention: Chart reviews were conducted on patients' medical records existing on the SCI National Database and were admitted to acute inpatient rehabilitation between 1998 and 2002. Data collected upon patient admission was analysed for contributing risk factors and predictors of dysphagia.</p> <p>Outcome Measures: Bedside Swallow Evaluation, type of surgery, presence of tracheostomy, ventilator status.</p>	<ol style="list-style-type: none"> 1. Although patients who also underwent cervical fusion surgery reported more cases of dysphagia and a longer mean time returning to a regular diet, cervical fusion was not a statistically significant risk factor for dysphagia. 2. Patients who had been intubated with a tracheostomy tube demonstrated a higher incidence of dysphagia than patients that did not receive a tracheostomy tube (p=0.0002). 3. Furthermore, patients with a tracheostomy tube took a significantly longer amount of time returning to their regular diet than non-tracheostomy patients (p<0.0001). 4. Patients who required ventilator support were also significantly more likely to experience dysphagia post-surgery (p=0.04) and took significantly longer to return to regular diet (p=0.0083). 5. The use of a Halo Skeletal Fixator was not significantly associated with diagnosis of dysphagia or mean time to eating a regular diet.

Chaw et al. 2012 USA Observational N=68	<p>Population: Mean age: 43.0 yr; Gender: males=57, females=11; Injury etiology: Motor vehicle accident=18, Fall=13, Diving=9, Bicycle accident=5, Gunshot wound=5, Motor cycle accident=4, Medical=4, Myelopathy=4, Trauma=4, Other=2; Level of injury: C1=2, C2=6, C3=14, C4=6, C4 (incomplete)=21, C5 (incomplete)=10, C6 (incomplete)=4, C7 (incomplete)=3, C8 (incomplete)=2; Level of severity: Complete=28, Incomplete=40; Mean time since injury: 31.8 days.</p> <p>Intervention: Bedside Swallowing Examination (BSE), which was followed by Videofluoroscopy Swallow Study (VFSS) within 72 hr.</p> <p>Outcome Measures: Risk factors for dysphagia.</p>	<ol style="list-style-type: none"> 1. Risk factors that were significantly related to dysphagia included: ventilator status ($p=0.012$), presence of tracheostomy ($p=0.028$), and use of a NG tube ($p=0.049$). Other risk factors were non-significant ($p>0.05$). 2. Individuals with dysphagia had significantly higher occurrences of pneumonia ($p<0.001$). 3. There were no significant differences between those with and without dysphagia on: bronchoscopy need ($p=0.23$), rate of re-intubation ($p=0.14$) or length of stay ($p=0.087$).
Abel, Ruf & Spahn 2004 Germany Observational N=73	<p>Population: Mean age: 42.9 yr; Gender: males=51, females=22; Injury etiology: Trauma=56, Spondylitis=5, Tumour=3, Other=9; Level of injury range: C1-C7; Level of severity: AIS A (complete)=41, incomplete=32; Mean time since injury: Not reported.</p> <p>Intervention: Patients with cervical SCI admitted to an initial care facility between January 1997 to December 2000. Prevalence of pneumonia via x-rays, and dysphagia via methylene blue test and videofluoroscopic swallowing, were determined at intake and discharge. Changes in dysphagia status were observed after tracheostomies, surgery to the cervical spine and dietary restrictions.</p> <p>Outcome Measures: Risk factors for dysphagia.</p>	<ol style="list-style-type: none"> 1. Patients with higher levels of cervical injury are more significantly likely to have a swallowing disorder ($p<0.05$). 2. Tracheostomy was strongly associated with the incidence of dysphagia ($p=0.00014$). 3. Anterior spinal surgery did not result in a significant contribution to dysphagia ($p=0.42$). 4. Patients with oral phase problems required dietary modifications at a significantly higher rate than those with problems in the pharyngeal phase ($p=0.044$). 5. At discharge, 9/26 cases had their dysphagia resolved.

Several risk factors have been associated with dysphagia incidence in individuals with SCI. Risk factors commonly identified were presence of a tracheostomy, ventilator use, age, nasogastric tube use, level of cervical injury, and presence of pneumonia.

Tracheostomies appear to be the most common factor for dysphagia. Tracheostomy tubes are used in individuals with SCI to facilitate ventilation and impaired cough reflexes (Shem et al. 2011). Prolonged use of tracheostomy tubes reduces respiratory volume and subglottic pressure inadvertently increasing the risk of aspiration (Hayashi et al. 2017). Aspiration can also occur due to leakage of secretions around the cuff depending on how well the tracheostomy tube is sealed (Chaw et al. 2012). The utilisation of a tracheostomy tube may have a disruptive presence on motor and sensory functioning with aspiration potentially caused by glottis injury, loss of protective reflexes, fixation of the trachea to the anterior neck skin, and esophageal obstruction due to the cuff's contact with the esophagus and hypopharynx (Kirshblum et al. 1999). Shem et al. (2011) also point out that the poor secretion is a risk factor for both pneumonia and dysphagia and this is managed with tracheostomy tubes; therefore, the connection between dysphagia and tracheostomy tubes may be better explained through management of secretion difficulties.

Ventilator use was also a frequent risk factor for dysphagia. There exists a multitude of ventilation techniques all with goal of assisting in respiration and secretion management to simulate coughing (Wong et al. 2012). Chaw et al. (2012) explain that confirming a causal relationship between dysphagia and ventilator dependence and/or tracheostomy is difficult due to the latter's link with pneumonia and as such, patients may require greater intensive intervention in order to improve their health. This assertion is supported by Wong et al. (2012) who stated that with aspiration and pneumonia being major complications of dysphagia, the need for ventilator support increases. This in turn complicates directionality as to the definitive cause of dysphagia with ventilator support and pneumonia acting as both independent and interrelated risk factors.

Age has been identified as risk factor for many diseases and conditions. Age is believed to increase the risk of dysphagia because increasing age is associated with changes in the physiology of the upper esophageal sphincter and pharyngeal region, blunting individuals' sensation and motility during the swallowing reflex (Hayashi et al. 2017).

Nasogastric tubes are used for acute SCI for gastrointestinal decompression. The nasogastric tubes impede swallowing as it traditionally passes through the lumen of the pharynx. Nasogastric tubes can consequently lead to aspiration as they: lower the anatomical integrity of the upper and lower esophageal sphincters, increase the frequency of transient lower esophageal sphincter relaxations, and lead to a desensitization of the pharyngoglottal adduction reflex (Chaw et al. 2012).

The consortium guidelines should be updated to reflect these more commonly identified risk factors present in the literature.

Conclusion

There is level 5 evidence (from several observational studies; Hayashi et al. 2017, Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Shin et al. 2011, Seidl et al. 2010, Shem et al. 2005, Abel, Ruf & Spahn 2004, Brady et al. 2004 Kirshblum et al. 1999) that presence of a tracheostomy is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from six observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Seidl et al. 2010, Shem et al. 2005) that ventilator use is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from six observational studies; Hayashi et al. 2017, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Shin et al. 2011, Kirshblum et al. 1999) that increasing age is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from four observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011) that presence of nasogastric tubes are a risk factor for dysphagia in individuals with SCI.

Several risk factors for dysphagia in individuals with SCI exist, the most common being presence of a tracheostomy, ventilator use, increasing age, and presence of a nasogastric tube.

5.0 Nutrition-Related Complications

5.1 Altered Glucose and Lipid Metabolism

In persons with SCI, the usual clinical measures of total body fat, such as weight and body mass index (BMI), underestimate the degree of adiposity (Bauman et al. 1997; Mollinger et al. 1985; Spungen et al. 1993; Spungen et al. 2000; Spungen et al. 2003). The metabolic alterations related to adverse body composition changes, decreased physical activity and other factors in individuals with SCI are considered atherogenic (Maki et al. 1995; National Cholesterol Education Program 2001, 2002). Even a mild decline in glucose tolerance is associated with insulin resistance and hyperinsulinemia, which are also considered atherogenic (Haffner et al. 1990).

Table 4 Lipid Lipoprotein Profiles

Low-density lipoprotein (LDL)	Lipid protein complex that transports cholesterol from the liver to other tissues within the body. LDL is often referred to as the “bad” cholesterol. LDL levels above 160 mg/dL (4.1 mmol/L) are considered to be high.
High-density lipoprotein (HDL)	Lipid protein complex that transports cholesterol from the tissues to the liver for excretion and re-utilization. HDL is often referred to as the “good” cholesterol. HDL levels of <40 mg/dL (<1.03 mmol/L) are associated with an increased risk for CVD.
Total cholesterol (TC)	Total amount of all cholesterol in the blood (increased TC related to increased risk for CVD)
Triglycerides (TG)	High energy fatty acids which form much of the fat stored by the body

Many factors contribute to increased risk of insulin resistance and hyperinsulinemia, glucose intolerance, CVD and obesity in persons with SCI. These factors tend to correlate with the severity and level of the neurological deficit (Javierre et al. 2005). It is hypothesized that the decreased lean muscle mass and increased adipose tissue which follow a SCI lead to impaired glucose uptake and an imbalance in whole body glucose homeostasis (Javierre et al. 2005). Pathogenesis of SCI combined with lifestyle practices impact blood glucose management thereby increasing the risk of morbidity and mortality due to CVDs, a frequent cause of death among persons with SCI (Arrowwood et al. 1987; Javierre et al. 2005; Yekutieli et al. 1989). Abnormalities in lipid metabolism in SCI develop early following injury and tend to progress over time (Brenes et al. 1986; Bauman et al. 1992; Kocina 1997; Szlachcic et al. 2001). Insulin resistance and exaggerated hyperinsulinemia in response to an oral glucose challenge are associated with the development of type II diabetes mellitus, atherosclerosis and ischemic heart disease (Bauman et al. 1992; Defronzo et al. 1991; Duckworth et al. 1983; Mohr et al. 2001). Conventional risk factors for coronary heart disease should be identified and treated aggressively in individuals with SCI according to current standards of care (Bauman & Spungen 2008).

Table 5 Altered Glucose and Lipid Metabolism

Author Year Country Research Design PEDro Score Total Sample Size	Methods	Outcome									
Glucose											
Battram et al. 2007 Canada RCT PEDro=6 N=14	<p>Population: Mean age=44.9 yr; Mean weight=82.1 kg; Level of injury: C4-C6; Level of severity: AIS A=5, AIS B and C=9; Mean time since injury=15.0 yr.</p> <p>Intervention: Participants were randomized to receive a 4 mg/kg dose of caffeine capsule or gelatin placebo capsules. Both groups then consumed a standard 75 gram glucose solution, and then a oral glucose tolerance test (OGTT) was performed. Analyses were performed between intervention groups, and subgroup analyses between SCI severity (complete vs. incomplete).</p> <p>Outcome Measures: Glucose response area under the curve (AUC), Insulin levels, Proinsulin levels, Proinsulin to insulin (PI/I) ratio, glucagon-like-peptide-1 (GLP-1), Epinephrine concentrations, Free fatty acid levels, glycerol concentrations, Mean arterial pressure (MAP),</p>	<ol style="list-style-type: none">1. The caffeine and placebo groups were not significantly different in glucose response AUC during the OGTT (p>0.05).2. The complete SCI subgroup had a 50% greater glucose response AUC compared with the incomplete SCI subgroup (p<0.05).3. Proinsulin levels were 40% lower in the complete group compared to the incomplete group (p<0.05).4. There were no treatment or subgroup effects on insulin levels (p>0.05), proinsulin levels or PI/I ratio (p>0.05), GLP-1 (p>0.05), epinephrine concentrations (p>0.05), free fatty acid (p=0.07), glycerol (p>0.05),5. The caffeine group had a significantly higher MAP compared to the placebo group (p<0.05).									
	<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%C.I.) as calculated from pre- and post-intervention data.</p> <table><caption>Forest Plot Data: Battram et al. 2007; Caffeine vs Gelatin</caption><thead><tr><th>Outcome</th><th>Standardized Mean Difference (95% C.I.)</th></tr></thead><tbody><tr><td>Caffeine</td><td>0.45 (-0.30, 1.20)</td></tr><tr><td>Glycerol</td><td>15.44 (11.33, 19.55)</td></tr><tr><td>MAP</td><td>2.83 (1.87, 3.88)</td></tr><tr><td>HR</td><td>0.33 (-0.41, 1.08)</td></tr></tbody></table>		Outcome	Standardized Mean Difference (95% C.I.)	Caffeine	0.45 (-0.30, 1.20)	Glycerol	15.44 (11.33, 19.55)	MAP	2.83 (1.87, 3.88)	HR
Outcome	Standardized Mean Difference (95% C.I.)										
Caffeine	0.45 (-0.30, 1.20)										
Glycerol	15.44 (11.33, 19.55)										
MAP	2.83 (1.87, 3.88)										
HR	0.33 (-0.41, 1.08)										
Bennegard & Karlsson 2008 Sweden Prospective Controlled Trial N=19	<p>Population: SCI (n=9): Mean age=40.8 yr; Mean weight=71.2 kg; Level of injury: C=2, T=7; Severity of injury: AISA A=8, B=1; Non-SCI controls (n=10): Mean age=31.9 yr; weight=75.9 kg.</p> <p>Intervention: Blood flow and overnight fasting glucose.</p> <p>Outcome Measures: Glucose uptake, plasma flow, lean tissue mass, and lactate.</p>	<ol style="list-style-type: none">1. SCI individuals were found to have significantly higher glucose uptake than those in the control group (p<0.05).2. Plasma flow was higher in legs of SCI individuals than the controls.3. Control subjects had higher lean tissue mass in their legs compared to the SCI subjects who only had 2/3 of the lean mass of the control subjects.4. For non-SCI individuals glucose uptake was lower in legs than arms in the control group whereas venous glucose concentration was higher in the leg (p<0.05); no differences were observed for those with SCI.5. Control subjects had a higher lactate production in arms than legs (p<0.05), while SCI subjects did not.									

Author Year Country Research Design PEDro Score Total Sample Size	Methods	Outcome
Bauman & Spungen 1994 USA Cohort N=150	<p>Population: Paraplegia (n=50): Mean age=51±2 yr; Time since injury=19±2 yr; Tetraplegia (n=50): Mean age=47±2 yr; Time since injury=17±2 yr; Controls (n=50): Mean age=51±2 yr; SCI and controls were age- and BMI-matched.</p> <p>Intervention: Oral glucose tolerance test (OGTT).</p> <p>Outcome Measures: Mean plasma glucose and insulin values, serum lipid levels.</p>	<ol style="list-style-type: none"> 82% of controls had normal oral glucose tolerance vs. 38% of those with tetraplegia and 50% with paraplegia. Subjects with SCI had significantly higher mean glucose and insulin values during the OGTT when compared to controls. Serum lipid levels in subjects with SCI showed a decreased HDL cholesterol level (38±1 mg/dL).
Bauman et al. 1999 USA Pre-Post N=201	<p>Population: Mean age=39 yr; Gender: males=169, females=32; Mean duration of injury=13 yr; Mean weight=75.9 kg; Mean BMI=25; Level of injury: tetraplegia=81, paraplegia=120; Severity of injury: complete=140, incomplete=61.</p> <p>Intervention: Oral glucose tolerance test (OGTT).</p> <p>Outcome Measures: Serum glucose concentration, plasma insulin levels, hyperinsulinemia, and serum uric acid.</p>	<ol style="list-style-type: none"> Individuals with complete tetraplegia had higher values for serum glucose concentration at 60 min, 90 min and 120 min and for plasma insulin at 90 min and 120 min after OGTT. Levels of serum glucose were similar in both men and women; however, plasma insulin levels were greater in men than women at all time points (p<0.05). Individuals with complete tetraplegia also had an increased frequency of diabetes mellitus compared to others. Individuals with tetraplegia had a significantly higher rate of hyperinsulinemia than individuals with paraplegia (p<0.05). A significant relationship was found between serum uric acid and BMI (p<0.0001), peak serum glucose (p=0.001) and peak plasma insulin (p=0.01).
Lipid		
Ketover et al. 1996 USA Prospective Controlled Trial N=58	<p>Population: SCI (n=29): Mean age=51 yr; Gender: males=28, females=1; Obesity (BMI>27)=11; Non-SCI controls (n=29): Mean age=36 yr; Gender: males=13, females=16; Obesity (BMI>27)=14.</p> <p>Intervention: All individuals were administered a 20 g fat liquid meal.</p> <p>Outcome Measures: Gallbladder emptying.</p>	<ol style="list-style-type: none"> No significant difference was seen in gallbladder emptying and volumes between SCI individuals and non-SCI subjects. In SCI subjects with diabetes and obesity, poor gallbladder emptying was observed. Age and injury level had no effect on gallbladder emptying.

Note: AISA=ASIA Impairment Scale; BMI=Body Mass Index

Four studies have examined altered glucose metabolism in individuals after a SCI (Bauman et al. 1999; Bauman & Spungen 1994; Bennegard & Karlsson 2008, Battram et al. 2007). Significantly higher serum glucose concentration and diabetes mellitus was seen in persons with complete tetraplegia than any other classification of SCI (Bauman et al. 1999). Gender had no effect on level of serum glucose; however, men had greater insulin levels than women (p<0.05; Bauman et al. 1999). In the remaining two studies, fasting glucose levels were compared between individuals with and without SCI. Bauman and Spungen (1994) reported that 38% and 50% of individuals with tetraplegia and paraplegia, respectively, had normal oral glucose tolerance compared to 83% of the non-SCI control group. Their findings were supported by Bennegard and Karlsson (2008) who reported a significantly higher glucose uptake in individuals with SCI compared to non-SCI

controls. Those with SCI had higher plasma flow rate in their legs compared to the controls; however, lean tissue mass was lower than those without SCI (Bennegard & Karlsson 2008). Battram et al. (2007) found that caffeine ingestion does not impair glucose tolerance in tetraplegics as well.

Recent research has begun to examine the altered glucose response in relation to visceral adipose tissue (VAT) and subcutaneous adipose tissue (SAT). Gorgey and Gater (2011) examined 32 males with SCI (mean body mass=74±14 kg; mean BMI=23.5±4.5) after an overnight glucose fast and found that both leg and trunk fat mass were associated with an altered metabolic profile. Further, those with tetraplegia (n=7) had greater leg, trunk and body fat mass than those with paraplegia (n=25). Fasting glucose was higher and the resting metabolic rate was 18% lower in those with tetraplegia than in those with paraplegia (p<0.05). Similarly, Gorgey et al. (2011) assessed VAT and SAT among 13 males with SCI (mean body mass=74±13 kg; mean BMI=23±4) after an overnight glucose fast. The authors reported that individuals with VAT >100 cm² had higher fasting plasma glucose compared to those with <100 cm²; further, VAT and SAT were associated with an altered metabolic profile. Although Gorgey and Gater (2011) and Gorgey et al. (2011) have presented a connection between fat mass and altered glucose response in individuals with SCI, the findings were conflicted with regard to lipid profile.

Altered lipid metabolism is apparent in the SCI population. In a prospective controlled trial, Ketover et al. (1996), evaluated gallbladder emptying in non-SCI individuals compared to persons with SCI after administering liquid meal (20 g fat). Both groups demonstrated similar gallbladder emptying and volumes post interventions; however, diabetic and obese subjects with SCI showed poor gallbladder emptying (Ketover et al. 1996).

There are a significant number of other biochemical changes in serum concentrations that occur after a SCI. Studies that examine these trends without specific regard to nutritional status or intervention have been placed in either the Aging Chapter or Cardiovascular Chapter. Please review those chapters for further exploration of resting glucose, insulin, lipid and other blood levels post SCI.

Conclusion

There is level 2 evidence (from one prospective controlled trial and one cohort study; Bennegard & Karlsson 2008; Bauman & Spungen 1994) that glucose uptake is higher in paralysed, spastic legs of SCI subjects than in able-bodied controls. There is a regional difference in glucose uptake in able-bodied with a lower uptake in legs than in arms.

There is level 2 evidence (from one cohort study and one pre-post study; Bauman & Spungen 1994; Bauman et al. 1999) that SCI individuals with tetraplegia have higher rates of altered glucose metabolism than other SCI individuals.

There is level 2 evidence (from one prospective controlled trial; Ketover et al. 1996) that diabetic and obese SCI individuals show impaired gallbladder emptying in response to a high fat meal compared to healthy SCI individuals.

Individuals with tetraplegia have higher rates of altered glucose metabolism.

Impaired gallbladder emptying is seen in diabetic and obese SCI individuals.

5.2 Neurogenic Bowel

Alterations in the central or peripheral nervous system can result in delayed gastric emptying, prolongation of intestinal transit time, and poor colonic motility, collectively known as neurogenic bowel. Neurogenic bowel has a significant impact on the quality of life of individuals with SCI, causing morbidity and even death (Correa & Rotter 2000). Modifications to dietary fiber consumption may assist with the management of neurogenic bowel following SCI. For further discussion on neurogenic bowel and specific nutrition interventions see the Neurogenic Bowel Chapter.

5.3 Neurogenic Bladder

Functional foods are products that are demonstrated to have health benefits and/or reduce the risk of chronic disease beyond their basic nutritional functions (Health Canada 1998). Cranberry juice, as it pertains to urinary tract infection risk, may fall under the category of a functional food. Refer to the Neurogenic Bladder Chapter for further information on the potential impact of cranberry juice on urinary tract infection in the SCI population.

5.4 Pressure Ulcers

Pressure ulcers are common following SCI, and healing can be compromised by suboptimal nutrition status. After a SCI, patients with pressure ulcers have lower zinc, albumin and prealbumin levels than those without pressure ulcers (Cruse et al. 2000a). Impaired nutritional status contributes to delayed or incomplete wound healing (Cruse et al. 2000b). Refer to the Pressure Ulcer Chapter for additional information regarding pressure ulcers in the SCI population.

5.5 Osteoporosis

Osteoporosis is common in SCI and results in increased bone fragility and fracture risk (Warden et al. 2001). In addition to pharmacological and other management strategies, supplementation with nutrients such as calcium and vitamin D may play a role in bone health following SCI. Refer to the Bone Health Chapter for further details.

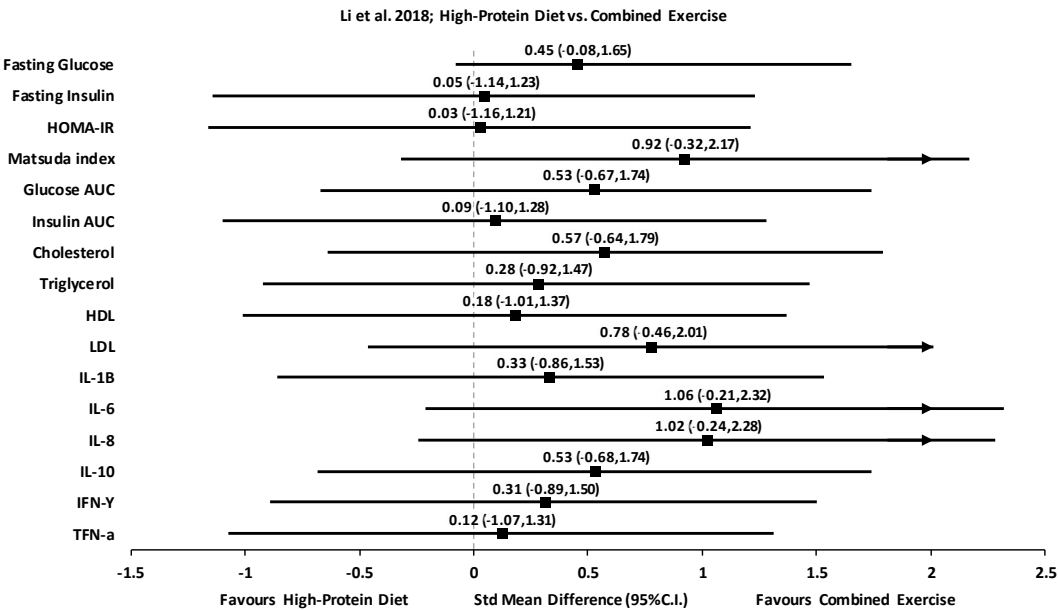
6.0 Nutritional Intervention for Energy Imbalance and Wellness

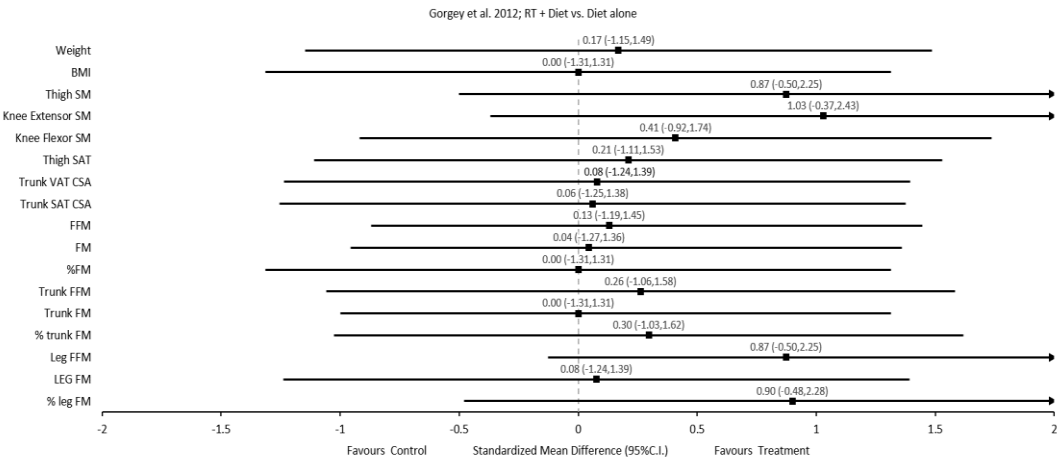
6.1 Diet and Exercise

Without appropriate modification of dietary intake following SCI, energy intake readily exceeds daily energy expenditure, predisposing persons with SCI to undesirable weight gain (Cox et al. 1985). Obesity is a common secondary complication of chronic SCI and is associated with adverse metabolic sequelae. In a large South Korean sample of individuals with SCI, obesity rates were reported to be 43.4% in those with physical disabilities and 34.6% for those without physical disabilities (Oh et al. 2012). An observational study from the United Kingdom found that energy imbalance leading to weight gain could be caused by the very inconsistent daily energy expenditure and daily energy intake and controlling this through intervention is important in weight loss for individuals with SCI (Nightingale et al. 2017). Despite widespread emphasis on obesity-related health risks in persons with SCI, limited research has been carried out to address this problem. There is a lack of information regarding the health outcomes of weight loss in this population, as well as limited educational resources available on nutrition issues and weight control for this high-risk group (Chen et al. 2006). However more recent studies (Betts et al. 2017 and Brochetti et al. 2018) have shown that combining diet and exercise in a structured lifestyle manner with accommodations for low-ambulatory SCI patients increases the compliance and retention post-intervention for longer periods of time.

Table 6 Diet and Exercise Program for Overweight/Obesity

Author Year Country Research Design PEDro Score Sample Size	Methods	Outcomes
Li et al. 2018 USA RCT PEDro=4 N=11	<p>Population: Mean age=46.0±7.8 yr; Gender: males=10, females=1; Time since injury=21.8±6.3 yr; Level of injury: C=4, T=7, L=0; Severity of injury: AIS A=3, B=8, C=0, D=0.</p> <p>Intervention: Participants randomized into an 8-week combined exercise (Comb-Ex) group designed to challenge strength, power, and endurance, or an 8-week high protein (HP) Diet group that focused on maintaining a carbohydrate to protein ratio <1.5 and limiting fat to ~30% of total energy intake.</p> <p>Outcome Measures: Exercise adaptations (oxygen consumption (VO₂), strength, muscle fiber type), body composition (body mass, lean mass, fat mass, android fat mass), glucose homeostasis, fasting glucose concentration (FGC), insulin sensitivity, lipid profile (total cholesterol (TC), triacylglycerol, high-density lipoprotein (HDL), low-density lipoprotein (LDL)), inflammation profile (IFN-γ, IL-1B, IL-6, IL-8, IL-10, IL-12, and TNF-α), and intracellular muscle signaling (GLUT 4, AMP-activated protein kinase (AMPK), calcium/calmodulin-dependent protein kinase type II (CAMPKII), protein kinase B (Akt), and Atk substrate (AS-160)).</p>	<ol style="list-style-type: none"> Comb-ex participants improved VO₂ peak significantly from baseline (p<0.05). Comb-Ex participants improved their maximum voluntary upper body strength (p<0.05). Comb-Ex participants had significant hypertrophy in the deltoid (p<0.05) and vastus lateralis (VL) muscles (p<0.05). Comb-Ex participants had a significant shift in myofiber type from type IIx to type IIa muscle fibers (p<0.05). Both groups significantly reduced their total body mass (p<0.05) and fat mass (p<0.05). Neither group had significant changes in lean body mass or android fat mass (p>0.05). Significant reduction in FGC in Comb-Ex group (p<0.05) but not in the HP Diet group (p>0.05). Both groups saw a significant reduction for insulin area under the curve (AUC) (p<0.05). No correlation between changes in body composition and changes in FGC and insulin AUC's. No significant changes in TC, triacylglycerol, HDL or LDL (p>0.05). No significant changes in IFN-γ, IL-1B, IL-6, IL-8, IL-10 and IL-12 for inflammatory profile in either group (p>0.05). Significant reduction in TNF-α in both groups (p<0.05). Comb-Ex group had significantly higher deltoid AMPK and CAMPKII than the HP diet group (p<0.05). GLUT4 levels were significantly higher in the nonparalytic deltoid compared to the paralytic VL muscle in the Comb-Ex group (p<0.05). Atk and AS-160 phosphorylation was significantly higher in the paralytic VL compared to the nonparalytic deltoid muscle in the Comb-Ex group (p<0.05).

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	<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%C.I.) as calculated from pre- and post-intervention data.</p> <p>Li et al. 2018; High-Protein Diet vs. Combined Exercise</p>  <table><thead><tr><th>Marker</th><th>SMD (95% C.I.)</th></tr></thead><tbody><tr><td>Fasting Glucose</td><td>0.45 (-0.08, 1.65)</td></tr><tr><td>Fasting Insulin</td><td>0.05 (-1.14, 1.23)</td></tr><tr><td>HOMA-IR</td><td>0.03 (-1.16, 1.21)</td></tr><tr><td>Matsuda index</td><td>0.92 (-0.32, 2.17)</td></tr><tr><td>Glucose AUC</td><td>0.53 (-0.67, 1.74)</td></tr><tr><td>Insulin AUC</td><td>0.09 (-1.10, 1.28)</td></tr><tr><td>Cholesterol</td><td>0.57 (-0.64, 1.79)</td></tr><tr><td>Triglycerol</td><td>0.28 (-0.92, 1.47)</td></tr><tr><td>HDL</td><td>0.18 (-1.01, 1.37)</td></tr><tr><td>LDL</td><td>0.78 (-0.46, 2.01)</td></tr><tr><td>IL-18</td><td>0.33 (-0.86, 1.53)</td></tr><tr><td>IL-6</td><td>1.06 (-0.21, 2.32)</td></tr><tr><td>IL-8</td><td>1.02 (-0.24, 2.28)</td></tr><tr><td>IL-10</td><td>0.53 (-0.68, 1.74)</td></tr><tr><td>IFN-γ</td><td>0.31 (-0.89, 1.50)</td></tr><tr><td>TFN-a</td><td>0.12 (-1.07, 1.31)</td></tr></tbody></table>	Marker	SMD (95% C.I.)	Fasting Glucose	0.45 (-0.08, 1.65)	Fasting Insulin	0.05 (-1.14, 1.23)	HOMA-IR	0.03 (-1.16, 1.21)	Matsuda index	0.92 (-0.32, 2.17)	Glucose AUC	0.53 (-0.67, 1.74)	Insulin AUC	0.09 (-1.10, 1.28)	Cholesterol	0.57 (-0.64, 1.79)	Triglycerol	0.28 (-0.92, 1.47)	HDL	0.18 (-1.01, 1.37)	LDL	0.78 (-0.46, 2.01)	IL-18	0.33 (-0.86, 1.53)	IL-6	1.06 (-0.21, 2.32)	IL-8	1.02 (-0.24, 2.28)	IL-10	0.53 (-0.68, 1.74)	IFN-γ	0.31 (-0.89, 1.50)	TFN-a	0.12 (-1.07, 1.31)	
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Gorgey et al. 2012 USA RCT PEDro=6 N=9	<p>Population: <i>Treatment group (n=5):</i> Mean age: 36 yr; Gender: males=5, females=0; Injury etiology: unspecified; Level of injury: cervical=4, thoracic=1; Level of severity: AIS A=3, B=2; Mean time since injury: 16 yr. <i>Control (n=4):</i> Mean age: 33 yr; Gender: males=4, females=0; Injury etiology: unspecified; Level of injury: thoracic; Level of severity: AIS A=3, B=1; Mean time since injury: 8 yr.</p> <p>Intervention: Participants were randomized to receive neuromuscular electrical stimulation resistance training and diet (treatment) or diet alone (control) over 12 wk. Training sessions were delivered 2 x/wk and involved leg extensions with increasing ankle weights (4 sets, 10 reps). Stimulation was delivered during training at 30 Hz and 450 μ, with 50 sec on and 50 sec off. Diets were composed of 45% carbohydrates, 30% fat, and 25% protein. Outcomes were assessed before and after treatment.</p> <p>Outcome Measures: Weight, Body Mass Index (BMI), Cross-Sectional Area (CSA), Skeletal Muscle, Adipose Tissue, Fat-Free Mass (FFM), Fat Mass (FM), Cholesterol, Triglycerides (TG), Low-Density Lipoproteins (LDL), High-</p>	<ol style="list-style-type: none">1. Weight and BMI were not significantly different between groups post intervention.2. Based on MRI findings, there were significant differences ($p<0.001$) between treatment and placebo groups post intervention in mean skeletal muscle CSA for the thigh (78 versus 53 cm²), knee flexor (35 versus 26 cm²), and knee extensor (22 versus 16 cm²).3. Based on MRI findings, there was a significant difference between treatment and control groups post intervention in intramuscular fat (15% versus 31%, $p=0.009$); there were no significant differences between groups in CSA of visceral and subcutaneous adipose tissues.4. Based on DXA findings, there were significant differences between treatment and placebo groups post intervention in mean leg FFM (7.5 versus 6.2 kg, $p=0.03$), leg %FM (28% versus 36%, $p=0.02$), and ratio of leg FFM to whole body FFM (0.15 versus 0.12, $p=0.043$); there were no significant differences between groups in FFM or FM of the whole body or trunk.5. Based on DXA findings, significant interactions were observed due to increases in trunk FFM by 1kg in the treatment group ($p=0.0001$) and decreases																																		

Author Year Country Research Design PEDro Score Sample Size	Methods	Outcomes																																				
	Density Lipoproteins (HDL), Free Fatty Acid (FFA), Glucose, Insulin, Insulin-Like Growth Factor 1 (IGF-1).	<p>in trunk %FM by 2% in the control group (p=0.0003).</p> <p>6. Lipid profiles showed a significant difference between treatment and placebo groups post intervention in mean levels of TG (87 versus 125 mg/dL, p=0.045) and cholesterol to HDL ratio (4.8 versus 5.2, p=0.017); there were no significant differences between groups in total cholesterol, LDL, HDL, or FFA.</p> <p>7. Carbohydrate metabolism showed a significant difference between treatment and placebo groups post intervention in ratio of plasma insulin to plasma glucose (p=0.04); there were no other significant differences between groups in glucose or insulin.</p> <p>8. IGF-1 was significantly correlated with knee extensor CSA (r=0.53, p=0.037) and visceral adipose tissue CSA (r=-0.56, p=0.023).</p>																																				
	<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%CI.) as calculated from pre- and post-intervention data.</p>  <table border="1"><caption>Forest Plot Data (Approximate)</caption><thead><tr><th>Measure</th><th>SMD (95%CI)</th></tr></thead><tbody><tr><td>Weight</td><td>0.17 (-1.15, 1.49)</td></tr><tr><td>BMI</td><td>0.00 (-1.31, 1.31)</td></tr><tr><td>Thigh SM</td><td>0.87 (-0.50, 2.25)</td></tr><tr><td>Knee Extensor SM</td><td>1.03 (-0.37, 2.43)</td></tr><tr><td>Knee Flexor SM</td><td>0.41 (-0.92, 1.74)</td></tr><tr><td>Thigh SAT</td><td>0.21 (-1.11, 1.53)</td></tr><tr><td>Trunk VAT CSA</td><td>0.08 (-1.24, 1.39)</td></tr><tr><td>Trunk SAT CSA</td><td>0.06 (-1.25, 1.38)</td></tr><tr><td>FFM</td><td>0.13 (-1.19, 1.45)</td></tr><tr><td>FM</td><td>0.04 (-1.27, 1.36)</td></tr><tr><td>%FM</td><td>0.00 (-1.31, 1.31)</td></tr><tr><td>Trunk FFM</td><td>0.26 (-1.06, 1.58)</td></tr><tr><td>Trunk FM</td><td>0.00 (-1.31, 1.31)</td></tr><tr><td>% trunk FM</td><td>0.30 (-1.03, 1.62)</td></tr><tr><td>Leg FFM</td><td>0.87 (-0.50, 2.25)</td></tr><tr><td>LEG FM</td><td>0.08 (-1.24, 1.39)</td></tr><tr><td>% leg FM</td><td>0.90 (-0.48, 2.28)</td></tr></tbody></table>		Measure	SMD (95%CI)	Weight	0.17 (-1.15, 1.49)	BMI	0.00 (-1.31, 1.31)	Thigh SM	0.87 (-0.50, 2.25)	Knee Extensor SM	1.03 (-0.37, 2.43)	Knee Flexor SM	0.41 (-0.92, 1.74)	Thigh SAT	0.21 (-1.11, 1.53)	Trunk VAT CSA	0.08 (-1.24, 1.39)	Trunk SAT CSA	0.06 (-1.25, 1.38)	FFM	0.13 (-1.19, 1.45)	FM	0.04 (-1.27, 1.36)	%FM	0.00 (-1.31, 1.31)	Trunk FFM	0.26 (-1.06, 1.58)	Trunk FM	0.00 (-1.31, 1.31)	% trunk FM	0.30 (-1.03, 1.62)	Leg FFM	0.87 (-0.50, 2.25)	LEG FM	0.08 (-1.24, 1.39)	% leg FM	0.90 (-0.48, 2.28)
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Brochetti et al. 2018 USA Pre-Post N=18	<p>Population: Mean age=55.7±13.0 yr; Gender: Not reported; Time since injury=9.1±10.7 yr; Level of injury: C=11, T=4, L=1; Severity of injury: AIS A=3, B=1, C=5, D=7. *2 participants had multiple sclerosis, so their data is not represented in level or severity of injury.</p> <p>Intervention: 18 overweight participants with SCI engaged in a 12-week interdisciplinary weight, food and lifestyle management program to</p>	<ol style="list-style-type: none">Significant difference in weight from the program start to end (p<0.001).Significant difference in waist circumference measurements from program start to end (p<0.001).Pearson Moment Correlation determined that those with a higher BMI did not lose proportionally more weight than participants with a lower BMI (p<0.05).Pearson Moment Correlation determined there was no relationship between age, weight loss and waist circumference (p>0.05).																																				

Author Year Country Research Design PEDro Score Sample Size	Methods	Outcomes
	observe its effects physical measurements. Outcome Measures: Body weight, waist circumference, body mass index (BMI), and diabetes risk scores.	5. Significant reduction in diabetes risk scores from program start to end ($p<0.001$).
Betts et al. 2017 USA Pre-Post N=10	Population: Mean age= 48.6 ± 13.1 yr; Gender: males=6, females=4; Time since injury=18.8 yr; Injury etiology: SCI=6, Spina Bifida=2, Osteoarthritis/Joint Disease/Orthopedic Problems=2; Level of injury: Not reported; Severity of injury: Not reported. Intervention: Participants took part in a modified diabetes prevention program group lifestyle balance program to facilitate weight loss through lower caloric intake and higher aerobic activity. Outcome Measures: Body Weight, Body Mass index (BMI), Waist Circumference, Systolic Blood Pressure, Diastolic Blood Pressure	1. There were significant improvements over time for weight loss and BMI ($p<0.05$). 2. There were no significant changes over time for blood pressure and waist circumference ($p>0.05$).
Chen et al. 2006 USA Pre-post N _{Initial} =17; N _{Final} =16	Population: Gender: males=9, females=7; Injury etiology: SCI=15, spina bifida=1; Severity of injury: AIS A-D; Family history of overweight/obesity: yes=11, no=5. Intervention: Patients attended classes on nutrition, exercise and weight control/reduction for 12 wk (90 min/wk) and exercised for 6 wk (30-min). Outcome measures: Physiologic measures (weight loss, body mass index [BMI]), high density lipoprotein (HDL).	1. During the intervention 14 subjects lost weight (mean age=4.2 kg). 2. Decreases were noted in BMI ($p<0.050$), waist circumference ($p<0.001$), neck circumference ($p<0.020$), and skinfold thickness ($p<0.001$). 3. HDL decreased significantly ($p<0.030$). 4. At follow-up, 6 continued to lose weight, 4 stabilized, and 3 gained.
Nightingale et al. 2017 United Kingdom Observational N=33	Population: Mean age= 44.0 ± 9.0 yr; Gender: males=27, females=6; Time since injury= 15.0 ± 10.0 yr; Level of injury: C=0, T=33, L=0; Severity of injury: AIS A=28, B=5, C=0, D=0. Intervention: Participants wore a physical activity monitor and completed a weighed food diary for 7 days consecutively. Outcome Measures: Physical activity monitor wear time, body mass, resting metabolic rate (RMR), energy expenditure variables (total energy expenditure (TEE), physical activity energy expenditure (PAEE), and physical activity level (PAL)), energy intake (total energy intake, macronutrient composition(percent protein, percent carbohydrate, percent fat)), number of days required to	1. Significant main effect of physical activity monitor wear time for TEE ($p=0.02$), PAEE ($p<0.001$), PAL ($p=0.003$), sedentary time ($p=0.017$), and light-intensity activity ($p=0.003$). 2. Level of injury was a significant covariate for TE ($p=0.03$), PAL ($p=0.04$), sedentary time ($p=0.03$) and moderate-to-vigorous physical activity (MVPA) ($p=0.03$). 3. Sex was also a significant predictor for TEE, PAEE, sedentary time, light and MVPA (all $p<0.001$). 4. Total energy intake decreased significantly from day 1 to 7 ($p=0.01$). 5. No significant change from day 1 to 7 on energy expenditure variables ($p>0.23$) or diet macronutrient composition ($p>0.70$). 6. Alcohol consumption was significantly higher for Friday and Saturday compared to the rest of the week ($p<0.05$).

Author Year Country Research Design PEDro Score Sample Size	Methods	Outcomes
	reliably estimate energy expenditure variables.	<ol style="list-style-type: none"> 7. Day of the week did not significantly affect energy expenditure variables or total energy intake ($p>0.05$). 8. Concluded that 1-4 days are required to reliably estimate energy expenditure variables and 4-24 days are required to estimate total energy intake and macronutrient composition.

An observational study by Nightingale et al. (2017) provides insight into what characteristics to focus on when designing diet and exercise interventions for SCI patients. 33 individuals wore a physical activity monitor for 7 consecutive days and kept a log of their daily food intake to assess their energy balance using their energy intake and energy expenditure. 31 participants successfully wore the physical activity monitor for at least the minimum required amount of time, and 32 completed the food intake log. The results revealed that both energy expenditure and energy intake were very inconsistent and reported that it takes 1-4 days of monitoring energy expenditure to reliably estimate energy expenditure variables and 4-24 days to reliably estimate total energy intake. The study also reported which covariates effect energy expenditure and energy intake and how much. These results are useful in designing future interventions as it gives researchers the ability to target specific characteristics in order to produce the most useful data regarding diet and exercise and how to generate the best results for patients with SCI.

Li et al. (2018), an RCT in which 11 participants (10 males, 1 female) with SCI were randomized to either 8 wks of combined exercise (Comb-Ex) consisting of resistance and aerobic exercise training with supervision or 8 wks of a high-protein (HP) diet with phone meetings to record compliance and study retention. At the end of the intervention, the HP diet group saw a significantly larger decrease in Matsuda Index scores compared to the Comb-Ex group, whereas the Comb-Ex group saw a their fasting plasma glucose levels decrease significantly more than the HP diet group. Values for area under the curve for insulin and for the pro-inflammatory cytokine TNF- α decreased significantly as well. Results reveal that combined exercise has a significant effect on fasting plasma glucose and high-protein diet has a significant effect on Matsuda Index scores, and both groups improve the area under the curve for insulin and TNF- α for individuals with SCI. It is possible that a regime that incorporates both an HP diet and a Comb-Ex program could potentially provide the best results and this should be a focus of future research.

In an RCT by Gorgey et al. (2012), nine males with chronic SCI were randomized to 12 wk of resistance training plus a diet program or a diet program alone. After the intervention, groups were comparable in body weight and levels of subcutaneous and visceral adipose tissue. However, the treated group had significantly greater increases in skeletal muscle cross sectional areas, insulin growth factor, and fat-free mass; they also experienced reductions in intramuscular fat percentage, the ratio of visceral to subcutaneous adipose tissue, the ratio of plasma insulin to plasma glucose, and triglyceride and HDL-C levels. Resistance training therefore has the benefits of increasing favourable body composition regions through skeletal muscle hypertrophy, which in turn can lead to improvements in carbohydrate and lipid metabolism.

Chen et al. (2006) conducted a study to assess the effect of a weight-loss program on body weight, BMI, waist and neck circumference, skinfold thickness, fat versus lean mass, bone mineral

content, blood pressure (BP), serum lipids, hemoglobin, albumin, eating habits, nutrition knowledge, bowel function and indicators of psychosocial well-being. A total of 16 subjects with chronic SCI who were overweight or obese completed the intervention program. Subjects attended 90-minute counseling sessions once per week for 12 weeks, led primarily by a Registered Dietitian. The dietary approach emphasized high-fiber, nutrient-dense foods (e.g., fruits, vegetables, grains, cereals) and the moderation of meats, cheeses, sugars and fats (Weinsier et al. 1983). The program included exercise and behaviour modification. Reported results included an average weight loss of 3.5 kg, significant reductions in BMI, anthropometric measures and fat mass. Lean mass, hemoglobin, albumin and bone mineral content were maintained. There was no significant change in BP or low-density lipoprotein cholesterol (LDL), although there was a significant decrease in high density lipoprotein cholesterol (HDL). There was a trend between weight lost and decrease in waist circumference, increase in nutritional quality of diet, increase in fiber consumption and decrease in time required for bowel movements. Changes in psychosocial and physical functioning were also reported.

Brochetti et al. (2018) is a pretest-post test study similar in design to Chen et al. (2006), as it looks at weight loss, waist circumference with the additional outcome measures of intervention compliance and retention. 18 participants completed a 12-wk interdisciplinary weight management program called Working on Healthy Eating, Exercise and Life Style (WHEELS) that consisted of a dietary component and an exercise program. Those who could not attend meetings or exercise sessions at the program headquarters were able to complete their workouts via satellite sessions, so the program was very accommodating. From baseline to intervention's end 15 of the 18 participants lost at least 4.5 ± 0.5 kg and 17 of the 18 experienced weight loss of some sort. Also from baseline to post-intervention 16 of the 18 participants experienced a decrease in waist circumference. At the 6-mo follow-up, there were no significant differences in weight or waist circumference with 12/18 participants continuing to lose weight or barely gaining any weight. 11 of the 18 participants had decreased Diabetes Risk scores as well. Results from the WHEELS intervention indicate that the program may have clinical benefits as well as show promise for compliance and retention from post-intervention to 6-mo follow-up, which is something that studies to come should seek to duplicate.

While previous studies assessed the physiological effects of nutrition-based weight loss programs, a study by Betts et al. (2017) evaluated the effectiveness of a Diabetes Prevention Program Group Lifestyle Balance (DPP GLB) program with regards to not only weight loss, but program retention and prolonged lifestyle change as well. Ten participants began the program, and seven were retained, attending 79.3% of the conference calls over the 20 wk intervention. Along with the 70% retention, participants rated the program a $6.3 \pm 0.3/7$ for helplessness and a $6.2 \pm 0.6/7$ for satisfaction with a significant mean weight loss of 8.86 ± 8.37 kg and significant reduction in BMI in those that completed the intervention. Despite the small sample size making difference detection challenging, the DPP GLB program completers experienced significant weight loss finding the intervention helpful and satisfying.

Conclusions

There is level 1b evidence (from one RCT; Gorgey et al. 2012 and three pre-post studies; Chen et al. 2006, Betts et al. 2017 and Brochetti et al. 2018) that an intervention program combining diet and exercise is effective for reducing weight among overweight persons with SCI.

There is level 1b evidence (from two RCTs; Gorgey et al. 2012 and Li et al. 2018) that interventions targeting just diet and just exercise are both effective for reducing metabolic markers for obesity and associated metabolic diseases like diabetes and heart disease.

There is level 4 evidence (from two pre-post studies; Betts et al. 2017 and Brochetti et al. 2018) that lifestyle interventions that combine diet and exercise and provide accommodation to low-ambulatory participants are effective in participant compliance and retention during and post-intervention.

There is level 5 evidence (from one observational study; Nightingale et al. 2017) that it is important to incorporate strict dietary and exercise guidelines for patients with SCI because of how inconsistent those with SCI are with their daily energy intake and daily energy expenditure.

Strict guidelines for diet and exercise interventions are important for patients with SCI due to the inconsistent nature of their energy balance causing weight gain leading to obesity.

A combined diet and exercise program can help patients reduce weight following SCI without compromising total lean mass and overall health.

6.2 Diet and Inflammatory Mediators

Individuals after SCI commonly experience chronic inflammation (Allison & Ditor, 2015). This prolonged state of elevated inflammation is associated with immune impairment which can lead to infections and other dysfunctions (Allison et al., 2017). Inflammation is measured by serum levels of certain pro-inflammatory cytokines, such as interleukin and interferon gamma (Hayes et al., 2002). Two methods of reducing inflammation are exercise and diet modification however, SCI individuals often have difficulty participating in exercise (Allison et al., 2018). Several supplements have been identified to have anti-inflammatory properties in uninjured individuals (Galland, 2010). These supplements, such as omega-3 and fatty acids, and anti-inflammation dietary changes have not been studied at length in SCI populations. The following studies have been identified and meet inclusion criteria, examining anti-inflammation diet changes and supplements in SCI.

Table 7 Diet and Inflammatory Mediator Effects

Author Year Country Research Design PEDro Score Sample Size	Methods	Outcomes
Allison et al. 2018 Canada Secondary Analysis of a previous RCT(Alison et al. 2017) N=20	<p>Population: Mean age=48.7±14.0 yr; Gender: males=10, females=10; Time since injury=13.1±10.8 yr; Level of injury: C=12, T=6, L=2; Severity of injury: AIS A=7, B=2, C=3, D=8.</p> <p>Intervention: Participants were randomly assigned to a three-month control group or a three-month anti-inflammatory diet treatment group.</p> <p>Outcome Measures: Change in nutrient intake, and corresponding changes to various inflammatory mediators (C reactive protein (CRP), interleukin (IL-2, IL-6, IL-1β), tumor necrosis factor alpha (TNF-α), interferon gamma (IFN-γ), prostaglandin E2 (PGE2) and kynurenine/tryptophan ration (KYN/TRP).</p>	<ol style="list-style-type: none"> 1. No significant change in total energy intake from baseline to the end of the intervention (p=0.10). 2. Significant reduction in fat intake (p=0.02) and significant increase in protein intake (p=0.02), however carbohydrate intake was not significantly changed (p=0.23). 3. Well-established anti-inflammatory nutrients vitamin A, carotenoids, vitamin C, vitamin E, and omega-3 fatty acids all showed significant increases from baseline to intervention's end (p<0.01 for each). 4. Pro-inflammatory nutrients showed significant reductions from baseline to intervention's end including trans fatty

	<p>*NOTE: This was a statistical analysis of a previous randomized controlled trial (Alison et al. 2017).</p>	<p>acids ($p=0.05$), caffeine ($p<0.01$) and sodium ($p=0.02$).</p> <ol style="list-style-type: none"> 5. No significant observable changes in total energy intake, macronutrient intake, or nutrients with anti-inflammatory or pro-inflammatory properties in the control group ($p>0.05$ for all). 6. Mann-Whitney test showed change scores from baseline – 3-months between treatments group and control group were significantly different for IFN-γ ($p=0.01$), IL-1β ($p=0.01$), and I-2 ($p=0.01$). 7. No other significant differences reported by the Mann-Whitney test for inflammatory mediators in the treatment or control groups. 8. Friedman test indicated that in the treatment group there was a statistically significant reduction in IFN-γ ($p=0.01$), IL-1β ($p<0.01$) and IL-6 ($p<0.05$). 9. No other significant changes reported by the Friedman test for the inflammatory mediators in the treatment or control group. 10. Wilcoxon signed-rank test reported significant reductions from baseline to 3 months in IFN-γ ($p=0.01$) and IL-1β ($p<0.01$) in the treatment group. 11. No other significant changes reported by the Wilcoxon signed-rank test in the treatment or control group. 12. Non-significant trends towards group X time interactions for TNF-α ($p=0.10$) and PGE2 ($p=0.07$) by two-way repeated measures ANOVA. 13. No trends observed for KYN/TRP by two-way measured ANOVA. 14. Change in total caloric intake and individual macronutrient intake was not significantly correlated with any inflammatory mediator. 15. Significant negative correlation observed between the change in vitamin A and the change in CRP ($p=0.02$), IL-1β ($p=0.02$), IFN-γ ($p=0.04$), and KYN/TRP ($p<0.01$). 16. Significant negative correlation observed between the change in carotenoids and the change in CRP ($p<0.01$), IL-1β ($p<0.01$), PGE2 ($p=0.05$) and KYN/TRP ($p<0.01$). 17. Significant negative correlation observed between the change in omega-3 and the change in IL-1β ($p=0.03$) and KYN/TRP ($p=0.01$). 18. Significant negative correlation observed between the change in zinc and the change in IL-2 ($p=0.04$), IL-6
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		<p>($p=0.05$), IL-1β ($p<0.01$), TNF-α ($p=0.02$), IFN-γ ($p=0.03$) and KYN/TRP ($p<0.01$).</p> <p>19. Significant negative correlation observed between the change in vitamin C and the change in the KYN/TRP ratio ($p=0.05$) as well as the change in iron and the change in the KYN/TRP ratio ($p<0.01$).</p> <p>20. Significant positive correlation observed between the change in iodine and the change in IL-2 ($p=0.03$), IL-6 ($p=0.01$) and IFN-γ ($p=0.02$).</p>																										
Allison et al. 2017 Canada RCT PEDro=7 N=20	<p>Population: Mean age=47.8\pm13.8 yr; Gender: males=10, females=10; Time since injury=13.1\pm10.8 yr; Level of injury: C=12, T=6, L=2; Severity of injury: AIS A=7, B=2, C=3, D=8.</p> <p>Intervention: Participants were randomized into a control group, who ate foods they would normally eat and a treatment group, which eliminated inflammatory inducing foods from their diet and added anti-inflammatory foods. Nerve conduction was tested at baseline, 1 and 3 months.</p> <p>Outcome Measures: Inflammatory Mediators (pro-inflammatory cytokines (IL-2, IL-1β, IL-6, TNF-α and IFN-γ), acute phase protein (CRP), anti-inflammatory cytokines (IL-4, IL-10 and IL-1RA), and pro-inflammatory eicosanoid (PGE2), nerve conduction velocity (NCV), and M-wave amplitude.</p>	<p>1. Mann-Whitney test showed significant difference between treatment and control groups for IFN-γ ($p=0.01$), IL-1β ($p=0.01$) and IL-2 ($p=0.01$).</p> <p>2. Friedman test showed a significant reduction over time for the treatment group in IFN-γ ($p=0.01$), IL-1β ($p=0.01$), and IL-6 ($p<0.05$).</p> <p>3. Mann-Whitney and Friedman tests did not show any significant reductions for an inflammatory mediator in the control group ($p>0.05$).</p> <p>4. Wilcoxon signed-rank test showed significant reduction over time for the treatment group in IFN-γ ($p=0.01$) and IL-1β ($p<0.01$).</p> <p>5. Wilcoxon signed-rank test showed no significant reduction over time at 3 months from baseline in IL-6 ($p=0.08$) but did at 1 month ($p=0.02$).</p> <p>6. No other significant changes in pro-inflammatory or anti-inflammatory cytokines ($p>0.05$).</p> <p>7. No significant Group X Time interactions observed for motor NCV ($p=0.77$) or M-wave amplitude ($p=0.61$).</p>																										
<p>Effect Sizes: Forest plot of standardized mean differences (SMD \pm 95% C.I.) as calculated from pre- and post-intervention data.</p> <table><caption>Forest Plot Data: Standardized Mean Differences (SMD) and 95% C.I.</caption><thead><tr><th>Marker</th><th>SMD (95% C.I.)</th></tr></thead><tbody><tr><td>CRP</td><td>0.52 (0.40, 1.42)</td></tr><tr><td>IL-2</td><td>0.13 (0.77, 1.02)</td></tr><tr><td>IL-6</td><td>0.39 (0.51, 1.28)</td></tr><tr><td>IL-1β</td><td>0.74 (0.35, 1.66)</td></tr><tr><td>TNF-α</td><td>0.69 (0.23, 1.61)</td></tr><tr><td>IFN-γ</td><td>0.47 (0.44, 1.38)</td></tr><tr><td>IL-4</td><td>0.13 (0.77, 1.02)</td></tr><tr><td>IL-10</td><td>0.14 (0.75, 1.09)</td></tr><tr><td>IL-1RA</td><td>0.41 (0.48, 1.32)</td></tr><tr><td>PGE2</td><td>0.45 (0.46, 1.35)</td></tr><tr><td>Motor Nerve Conduction Velocity</td><td>0.10 (0.80, 0.98)</td></tr><tr><td>M-Wave Amplitude</td><td>0.38 (0.52, 1.29)</td></tr></tbody></table>			Marker	SMD (95% C.I.)	CRP	0.52 (0.40, 1.42)	IL-2	0.13 (0.77, 1.02)	IL-6	0.39 (0.51, 1.28)	IL-1 β	0.74 (0.35, 1.66)	TNF- α	0.69 (0.23, 1.61)	IFN- γ	0.47 (0.44, 1.38)	IL-4	0.13 (0.77, 1.02)	IL-10	0.14 (0.75, 1.09)	IL-1RA	0.41 (0.48, 1.32)	PGE2	0.45 (0.46, 1.35)	Motor Nerve Conduction Velocity	0.10 (0.80, 0.98)	M-Wave Amplitude	0.38 (0.52, 1.29)
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Discussion

Allison et al. (2017) conducted an RCT investigating the effects of an anti-inflammatory diet intervention in SCI individuals. The control group individuals continued their normal diets, where

the treatment group eliminated inflammation inducing foods such as foods with high glycemic indices (refined wheat and sugar products), food with common intolerances (milk), and unhealthy food such as hydrogenated oils (Allison et al., 2017). The treatment group participants also consumed anti-inflammatory diet supplements (such as omega-3, chlorella, and antioxidants) up to three times a day depending on the supplement and dosage (Allison et al., 2017). The diet intervention program lasted for 12-weeks. The participants completed detailed 7-day diet records at baseline, and 3-day records at one, two, and three month follow-ups. Outcomes included measuring inflammatory serum markers and nerve conduction (Allison et al., 2017). While the authors found significant reduction in inflammation serum markers in the treatment group, there was no significant interaction effect found between-groups over time for motor nerve conduction (Allison et al., 2017). This study highlights a reduction in known inflammatory serum markers in SCI individuals after an anti-inflammatory dietary intervention, but no significant improvement was seen on motor nerve conduction (Allison et al., 2017). The authors followed-up this study with a secondary analysis investigating the changes in nutrient intake and their relationship with different inflammatory serum markers (Allison et al., 2018). The main findings of this secondary analysis indicate that significantly reducing fats and increasing protein intake lead to reductions in inflammatory serum biomarkers, while carbohydrates and caloric restriction have no effect. When looking at supplements, vitamins A, carotenoids, zinc, and omega-3 were associated with a reduction in serum biomarkers indicating inflammation (Allison et al., 2018). The authors discuss similarities of their diet intervention to the ‘Mediterranean diet’ which has been studied in healthy controls. The Mediterranean diet consists of lowering excess sugar, and red and processed meats, emphasizing ingestion of olive oil, fresh fruits and vegetables, fish and white meats (Estruch et al., 2013). The authors highlight a need for more studies to be conducted with anti-inflammatory diet changes in SCI individuals to better understand the potential benefits.

Conclusion

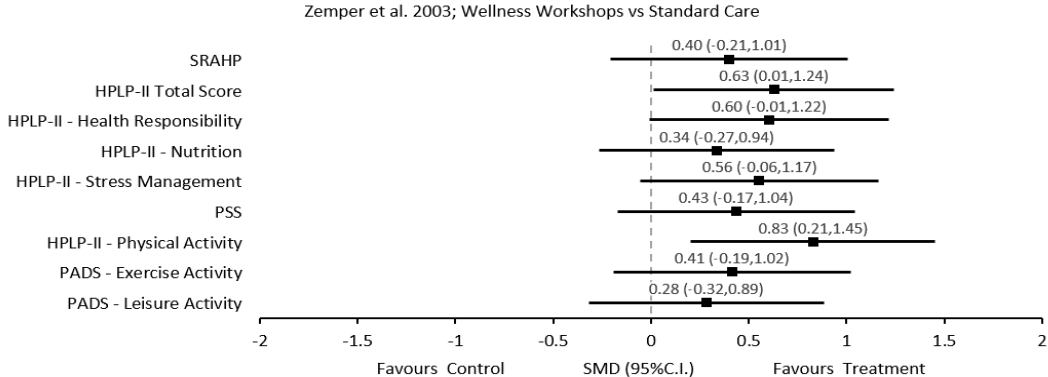
There is level 1b evidence (from one RCT and one secondary RCT analysis; Allison et al., 2017;2018) that a diet intervention focusing on anti-inflammatory diet changes and supplements can reduce inflammatory serum markers, but did not show improvement in motor nerve conduction compared to a control group.

A diet intervention focusing on anti-inflammatory foods and supplements can reduce inflammation serum markers, but may not show improvement in motor nerve conduction.

6.3 Health Promotion and Education

Little is known about the most effective health promotion activities, including nutrition interventions, to meet the long-term wellness needs for persons after SCI. A holistic wellness program intervention was developed, conducted and assessed by Zemper et al. (2003). Improvement in healthy diet involves a number of factors. One important factor of health promotion and education is improving the ‘food environment’. This involves diet education, expanding current food choices, and providing better access to healthy foods (Hata, Inayama, & Yoshiike, 2017).

Table 8 Nutritional Education Intervention and Long-Term Wellness

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes																				
Zemper et al. 2003 USA RCT PEDro=4 N _{Initial} =76; N _{Final} =43	<p>Population: Age range=22-80 yr; Gender: males=30, females=13; Level of injury: complete, incomplete; Time since injury=1-49 yr.</p> <p>Intervention: Subjects attended a series of six 4-hr workshop sessions over 3 mo.</p> <p>Outcome measures: Wellness survey, Health Promoting Lifestyle Profile-II (HPLP-II); Secondary Conditions Scale (SCS), Self-Rated Abilities for Health Practices Scale (SAHP), Perceived Stress Scale, and Physical Activities with Disability Questionnaire (PADS).</p>	<ol style="list-style-type: none">1. Treatment groups scores on the SAHP improved following treatment (p<0.05) as well as on the HLPH-II (total score) & the HPLP-II health related subscale score (p<0.001).2. Treatment group scores improved post-treatment on the HPLP-II (nutrition subscale) (p<0.05).3. Mean scores for the treatment groups improved significantly for the HPLP-II stress management subscale (p=0.001). Treatment group's stress scores also improved, indicating less stress (p<0.05).4. HPLP-II physical activity scores improved post treatment for the treatment group only (p=0.001). No significant differences were noted for the PADS score for either group post treatment.5. SCS score decreased for the treatment group, post treatment (p=0.001), indicating fewer and less severe problems with secondary conditions. Number of secondary conditions was decreased for the treatment group post treatment (p<0.01).																				
<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95% C.I.) as calculated from pre- and post-intervention data.</p> <p>Zemper et al. 2003; Wellness Workshops vs Standard Care</p>  <table><thead><tr><th>Outcome</th><th>SMD (95% C.I.)</th></tr></thead><tbody><tr><td>SRAHP</td><td>0.40 (-0.21, 1.01)</td></tr><tr><td>HPLP-II Total Score</td><td>0.63 (0.01, 1.24)</td></tr><tr><td>HPLP-II - Health Responsibility</td><td>0.60 (-0.01, 1.22)</td></tr><tr><td>HPLP-II - Nutrition</td><td>0.34 (-0.27, 0.94)</td></tr><tr><td>HPLP-II - Stress Management</td><td>0.56 (-0.06, 1.17)</td></tr><tr><td>PSS</td><td>0.43 (-0.17, 1.04)</td></tr><tr><td>HPLP-II - Physical Activity</td><td>0.83 (0.21, 1.45)</td></tr><tr><td>PADS - Exercise Activity</td><td>0.41 (-0.19, 1.02)</td></tr><tr><td>PADS - Leisure Activity</td><td>0.28 (-0.32, 0.89)</td></tr></tbody></table>			Outcome	SMD (95% C.I.)	SRAHP	0.40 (-0.21, 1.01)	HPLP-II Total Score	0.63 (0.01, 1.24)	HPLP-II - Health Responsibility	0.60 (-0.01, 1.22)	HPLP-II - Nutrition	0.34 (-0.27, 0.94)	HPLP-II - Stress Management	0.56 (-0.06, 1.17)	PSS	0.43 (-0.17, 1.04)	HPLP-II - Physical Activity	0.83 (0.21, 1.45)	PADS - Exercise Activity	0.41 (-0.19, 1.02)	PADS - Leisure Activity	0.28 (-0.32, 0.89)
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Liusuwan et al. 2007 USA Pre-post N _{Initial} =20; N _{Final} =14	<p>Population: SCI=14; Mean age=15.4 yr; Gender: males=7, females=7.</p> <p>Intervention: Individuals participated in the BENEFit program which included an interactive lecture on nutrition, physical activity and participation in activity, games, prizes and other motivational techniques; parents engaged in a discussion group.</p> <p>Outcome measures: Weight, body mass index (BMI), cholesterol, high- (HDL) and low-density lipoprotein (LDL), triglycerides, VO_{2 REST AND MAX}, power output</p>	<ol style="list-style-type: none">1. There was no change in weight, BMI, cholesterol, LDL, HDL, and triglyceride levels, and HR2. Total lean tissue increased 2.1%.3. 27% ↑ in VO_{2rest} increased 27% (p<0.002); no change in VO_{2max}.4. 22% ↑ in PO_{max} increased 22% (p=0.014)5. Aerobic efficiency increased 35%.6. There was an increase in peak shoulder extension (p=0.016); shoulder and elbow flexion and extension did not change significantly.7. Shoulder extension strength increased.																				

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
	(PO), heart rate (HR), shoulder and elbow flexion and extension.	
Hata et al. 2017 Japan Observational N=506	<p>Population: Age groups: <49 yr=81, 50-59 yr=98, 60-69 yr=175, >70 yr=152; Gender: males=424, females=82; Time since injury: <9 yr=39, 10-19 yr=92, 20-29 yr=114, 30-39 yr=122, >40 yr=136; Level of injury: Cervical=147, Thoracic=270, Lumbar=89; Severity of injury: Not reported. *Mean age not reported.</p> <p>Intervention: Potential participants were sent a questionnaire regarding their sociodemographic variables, health related quality of life (HRQOL), dietary satisfaction and perceived food environment which they had the option to fill out and return, those that completed it were included in the study. Logistic regression models were conducted to examine the association between HRQOL/dietary satisfaction and perceived food environment.</p> <p>Outcome Measures: Physical and mental summary scores from the HRQOL and dietary satisfaction and perceived food environment.</p>	<ol style="list-style-type: none"> 1. HRQOL and dietary satisfaction were differentially associated with perceived food environment. 2. Acquired dietary information was associated with both physical and mental summary scores. 3. Mental summary score was related to price and access to food. 4. Dietary satisfaction was positively related to access to food and access to information. 5. Perceived food environment was positively associated with dietary satisfaction. 6. Access to food in the household was related to mental summary score. 7. Home-cooked meals related more to mental summary scores than physical summary scores.
Hata et al. 2016 Japan Observational N=625	<p>Population: Mean age: 62.7 yr; Gender: males=625, females=0; Injury etiology: unspecified; Level of injury: cervical=183, thoracic=323, lumbar=119; Severity of injury: unspecified; Mean time since injury: 28.1 yr.</p> <p>Intervention: Participants from the community were assessed via questionnaires.</p> <p>Outcome Measures: Dietary Satisfaction (DS), Self-Rated Health (SRH), Social Participation (SP), Social Support (SS).</p>	<ol style="list-style-type: none"> 1. Sufficient SP was reported in 67.5% of participants and sufficient SS was reported in 55.4%. 2. High DS was reported in 26.4% of participants and high SRH was reported in 67.0%. 3. High DS was significantly more likely in participants with SS than those without (OR=6.46, p<0.001). 4. When compared to participants without SP and SS, high DS was significantly more likely in participants with SS, either with SP (OR=8.64, p<0.001) or without SP (OR=6.99, p<0.001). 5. High SRH was significantly more likely in participants with SP than those without (OR=1.80, p=0.003) and in participants with SS than those without (OR=1.83, p=0.003). 6. When compared to participants without SP and SS, high SRH was significantly more likely in those with SP but without SS (OR=1.78, p=0.43) and those with both SP and SS (OR=3.28, p<0.001).

In the Zemper et al. (2003) study, 43 adults with SCI were randomly assigned to intervention or control groups. The intervention group attended 6 half-day wellness workshops over a 3-month period which included nutrition, physical activity, lifestyle management and prevention of secondary conditions. Among other measurements, total cholesterol and BMI were assessed.

Health Promoting Lifestyle Profile-II (HPLP-II) was used to assess nutrition and other health promotion habits. There was improvement in the HPLP-II nutrition subscale mean score for the intervention group. Mean BMI values actually increased for both groups. Total cholesterol values rose for both groups; changes in HDL and LDL cholesterol values were not reported. There were significant improvements in reported eating and weight-related behaviours.

A study was conducted by Liusuwan et al. (2007) which investigated the effects of behavioural intervention, exercise and nutrition education to improve health and fitness in adolescents with spinal cord dysfunction as the result of myelomeningocele and SCI. Among twenty adolescent subjects, fourteen completed all testing sessions conducted prior to and after completing a 16-week intervention program. Testing included measurements of aerobic fitness, heart rate (HR), oxygen uptake, peak isokinetic arm and shoulder strength, body composition, BMI and blood work assessment which included total, HDL and LDL cholesterol and triglycerides. Participants were given a schedule of aerobic and strengthening exercises and attended nutrition education and behaviour modification sessions every other week accompanied by their parents. Results suggested that there was no significant overall change in weight, BMI or blood work. There was a significant increase in whole body lean tissue without a concomitant increase in whole body fat. Fitness measures revealed a significant increase in maximum power output, work efficiency and resting oxygen uptake. Shoulder extension strength increased. There were no significant changes in total, HDL or LDL cholesterol or triglycerides during the 16-week program.

In a cross-sectional study Hata et al. (2016) examined a Japanese chronic male SCI population about their perceptions of their social participation in the community and the social support they receive from relatives and friends, in relation to their health and dietary satisfaction. The authors reported that individuals with sufficient social participation and social support are more likely to have greater self-rated health and dietary satisfaction. Resultantly, health promotion programs should emphasize these two social determinants of health. In particular, the authors found an odds ratio of having high dietary satisfaction seven times greater for those with high social support when comparing between individuals with low social participation (OR=6.99 vs OR=1.00), and six times greater for those with high social support when comparing between individuals with high social participation (OR=8.64 vs OR=1.38).

Hata, Inayama, and Yoshiike (2017) conducted another cross-sectional study, this time investigating perceived food environment and its relationship with health-related quality of life (HRQOL) and diet satisfaction of community-dwelling SCI individuals. The perceived food environment was studied, looking at how readily available and accessible healthy food is (Hata, Inayama, & Yoshiike, 2017). HRQOL was divided into physical and mental scores, and the authors found they were differently associated with factors of food environment. Physical scores were related to: stores with safe and reliable food, dietary information in the community, and accurate diet information from the media (ORs= 1.61, 1.67, and 1.74, respectively). Mental scores were related to: balanced meals in the household, nutritious foods at reasonable prices, and acquired diet information from the community (ORs= 1.86, 1.62, and 2.30, respectively). Lastly, diet satisfaction was related to: balanced meals in the household, food information available from the family, and accurate diet information from the media (ORs= 6.50, 2.70, and 1.75, respectively). This study highlights the importance of the perceived food environment of SCI individuals and its role in HRQOL and diet satisfaction while living in the community (Hata, Inayama, & Yoshiike, 2017).

Conclusions

There is level 1b evidence (from one RCT; Zemper et al. 2003) that improved health-related behaviours are adopted following a holistic wellness program for individuals with SCI.

There is level 4 evidence (from one pre-post study; Liusuwan et al. 2007) that an education program combining nutrition, exercise and behaviour modification is effective in increasing whole body lean tissue, maximum power output, work efficiency, resting oxygen uptake and shoulder strength in persons with SCI.

There is level 5 evidence (from one observational study; Hata et al. 2016) that social participation and social support have beneficial effects on an individual with SCI's self-rated health and dietary satisfaction.

There is level 5 evidence (from one observational study; Hata, Inayama, & Yoshiike, 2017) that the perceived food environment is associated with health related quality of life and diet satisfaction of community-dwelling SCI individuals.

Participation in a holistic wellness program is positively associated with improved eating and weight-related behaviours in persons with SCI.

A combined nutrition, exercise and behaviour modification program can help persons with SCI increase metabolically active lean tissue, work efficiency, resting oxygen uptake and strength.

Having a high social participation is positively associated with better self-rated health, and receiving sufficient social support is positively associated with a greater dietary satisfaction, in persons with SCI.

Having a positive perceived food environment is associated with higher physical and mental scores of health-related quality of life, and higher diet satisfaction in persons with SCI.

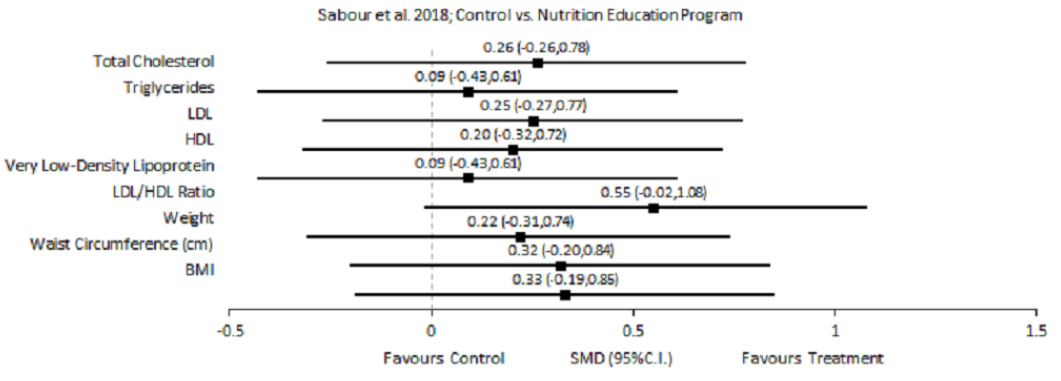
7.0 Nutritional Interventions for Dyslipidemia and Cardiovascular Disease Risk

7.1 Nutrition Counseling

CVD appears prematurely in persons with SCI. It is the most frequent cause of death among persons surviving more than 30 years following injury and accounts for 45% of all SCI deaths (Devivo et al. 1999). Abnormalities in lipid metabolism develop shortly after injury and tend to progress over time (Bauman et al. 1992; Brenes et al. 1986; Kocina 1997; Szlachcic et al. 2001). Despite the high risk for CVD morbidity and mortality in individuals with SCI, few studies have addressed the benefits of risk reduction interventions aimed at modifiable factors and those that exist have been limited to exercise interventions. This section discusses what is known about the value of nutrition counseling in improving dyslipidemia in persons with SCI.

Table 9 Effect of Nutrition Counseling on Dyslipidemia and Cardiovascular Disease Risk

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Sabour et al. 2018 Iran RCT	Population: Mean age: Not reported; Gender: males=45, females=12; Time since injury=>2 yr; Level of injury: C=9,	1. ANOVA showed no significant effect of the intervention on TG, TC, HDL, and LDL levels (p>0.05).

<p>PEDro=7 N=57</p>	<p>T=41, L=7; Severity of injury: Not reported.</p> <p>Intervention: Participants were randomized to a control group and a nutritional education group in which they attended a total of 5 education session over a 7-month period.</p> <p>Outcome Measures: Body weight and serum concentrations of total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL). Two-way repeated measure of analysis (ANOVA) was used.</p>	<p>2. Test of within subject contrast revealed no significant effect of the intervention on level of LDL (p=0.159), TC (p=0.224), TG (p=0.172) or HDL (p=0.081).</p> <p>3. Changes in TC level were not significantly different between intervention and control group (p=0.224).</p> <p>4. No significant differences in weight (p=0.64), height (p=0.10), waist circumference (p=0.92), or BMI (p=0.71) between intervention and control groups at baseline.</p> <p>5. No significant changes in weight (p=0.97) or waist circumflex (p=0.361) observed in the intervention group compared to the control group.</p> <p>6. No significant influence of nutritional education on anthropometric measurements, lipid profile, and blood pressure in patients with spinal cord injury.</p>																				
	<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%C.I.) as calculated from pre- and post-intervention data.</p>  <table><caption>Data from Forest Plot: Sabour et al. 2018; Control vs. Nutrition Education Program</caption><thead><tr><th>Measure</th><th>SMD (95% C.I.)</th></tr></thead><tbody><tr><td>Total Cholesterol</td><td>0.26 (-0.26, 0.78)</td></tr><tr><td>Triglycerides</td><td>0.09 (-0.43, 0.61)</td></tr><tr><td>LDL</td><td>0.25 (-0.27, 0.77)</td></tr><tr><td>HDL</td><td>0.20 (-0.32, 0.72)</td></tr><tr><td>Very Low-Density Lipoprotein</td><td>0.09 (-0.43, 0.61)</td></tr><tr><td>LDL/HDL Ratio</td><td>0.55 (-0.02, 1.08)</td></tr><tr><td>Weight</td><td>0.22 (-0.31, 0.74)</td></tr><tr><td>Waist Circumference (cm)</td><td>0.32 (-0.20, 0.84)</td></tr><tr><td>BMI</td><td>0.33 (-0.19, 0.85)</td></tr></tbody></table>		Measure	SMD (95% C.I.)	Total Cholesterol	0.26 (-0.26, 0.78)	Triglycerides	0.09 (-0.43, 0.61)	LDL	0.25 (-0.27, 0.77)	HDL	0.20 (-0.32, 0.72)	Very Low-Density Lipoprotein	0.09 (-0.43, 0.61)	LDL/HDL Ratio	0.55 (-0.02, 1.08)	Weight	0.22 (-0.31, 0.74)	Waist Circumference (cm)	0.32 (-0.20, 0.84)	BMI	0.33 (-0.19, 0.85)
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<p>Szlachcic et al. 2001 USA Prospective Controlled Trial N=222</p>	<p>Population: Gender: males=198, females=24; Level of injury: complete, incomplete; Time since injury=>2 yr.</p> <p>Intervention: Subjects who had a cholesterol level >5.2mmol/L (n=86) were referred to either a dietary consultation where they were advised to modify daily intakes as follows: total fat<30% of kcal, saturated fat<10% of kcal, cholesterol<300 mg, carbohydrate=60% of kcal, or no treatment.</p> <p>Outcome Measures: Total cholesterol (TC), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL), triglycerides.</p>	<p>1. TC decreased in the dietary counseling group from 6.1 mmol/L to 5.8 mmol/L (p<0.001) and slightly increased in the control group from 4.2 mmol/L to 4.3 mmol/L (p=0.006).</p> <p>2. LDL was reduced from 4.1 mmol/L to 3.9 mmol/L (p=0.004) in the dietary counseling group; there was no change for controls.</p> <p>3. Neither group experienced significant changes in HDL or triglyceride values.</p>																				
<p>Koutrakis et al. 2018 USA Observational N=174</p>	<p>Population: Mean age=57.6±12.4 yr; Gender: males=141, females=33; Time since injury=20.9±12.9 yr; Level and Severity of injury: Cervical motor complete and AIS C=40, Other motor complete and AIS C=65, AIS D=69.</p>	<p>1. Statistically significant positive association between 25(OH)D and total vitamin D intake, age, years of education, total calcium intake, wine consumption, total hours of planned exercise, female sex, white race, non-smokers, and users of sunscreen (p<0.05).</p>																				

	<p>*Level and severity of injury reported such that neither could be extracted independent of one another.</p> <p>Intervention: Participants completed food frequency and health questionnaires, gave blood samples and had percent total body fat measured via dual x-ray absorptiometry (DXA). Linear regression models were conducted to evaluate cross-sectional relationships between personal, lifestyle, and nutritional factors with blood plasma levels of 25-hydroxyvitamin D [25(OH)D].</p> <p>Outcome Measures: Dietary factors, lifestyle factors, clinical factors, and 25(OH)D.</p>	<ol style="list-style-type: none"> 2. No significant relationship between 25(OH)D and SCI level of injury, completeness of injury, body mass index, % total body fat, mobility mode, comorbid medical conditions, time outside between sunrise and sunset or season ($p>0.05$). 3. A multivariable model showed age, total vitamin D intake, total hours of planned exercise, sex, race, wine use, and smoking status remained statistically significantly associated with 25(OH)D. 4. Race and total vitamin D intake were the most statistically significant predictors ($p<0.0001$). 5. Impacts of supplementary vitamin D intake was statistically significant ($p<0.0001$) whereas dietary vitamin D intake was not ($p=0.305$). 6. In a univariable model, stretching, range of motion, and physical therapy was not significantly associated with 25(OH)D.
Javidan et al. 2017 Iran Observational N=265	<p>Population: Mean age=36.2±10.8 yr; Gender: males=217, females=48; Time since injury: Not reported; Level of injury: paraplegia=157, tetraplegia=108; Severity of injury: AIS A=132, B=133, C=0, D=0.</p> <p>Intervention: Participants completed 24-hour dietary recalls with a Nutritionist IV 3.5.3. for analysis.</p> <p>Outcome Measures: Dietary intakes (Amino Acids, Fasting plasma glucose (FPG), Triglyceride (TG), Systolic blood pressure (SBP), diastolic blood pressure (DBP), Total cholesterol (TC), High-density lipoprotein (LDL), and Low-density lipoprotein (LDL).</p>	<ol style="list-style-type: none"> 1. Majority of the participants in the study showed low protein intake (<1.5 g/kg when 1.5-2 g/kg is recommended). 2. Higher intake of isoleucine associated with higher levels of FPG ($p=0.007$), TG ($p=0.014$), SBP ($p=0.012$) and DBP ($p=0.04$). 3. Dietary intake of lysine was positively related to levels of FPG ($p<0.0001$), TG ($p=0.046$), SBP ($p=0.002$) and DBP ($p=0.009$). 4. A significant positive relationship observed between intake of cysteine and levels of TG ($p=0.027$) and SBP ($p=0.048$). 5. FPG was significantly positively related to intake of all amino acids except Cysteine, Glutamic acid, Threonine, Leucine, and Histidine ($p<0.05$). 6. TG was significantly higher among men ($p=0.015$), significantly higher in those with incomplete SCI ($p=0.016$), and significantly higher in those with paraplegia ($p=0.035$). 7. Blood pressure (BP) was significantly higher in quadriplegics ($p<0.0001$). 8. Older participants had significantly higher SBP ($p<0.0001$) and DBP ($p<0.0001$). 9. Higher SBP ($p=0.002$) and DBP ($p=0.001$) seen in those with higher total energy intake. 10. BP significantly higher in those with higher carbohydrate and cholesterol intake ($p<0.05$). 11. Higher tryptophan intake associated with lower SBP ($p=0.03$). 12. Significant correlation between glutamic acid and SBP ($p=0.007$) and DBP ($p=0.006$). 13. Low BP associated with higher lysine/arginine ratio: SBP ($p=0.009$) and DBP ($p=0.01$). 14. Women had significantly higher HDL ($p<0.0001$). 15. Age was significantly positively related to TC ($p=0.015$) and LDL ($p<0.0001$).

		16. Weight was significantly positively related to TC ($p<0.0001$) and LDL ($p<0.0001$).
		17. Higher BMI significantly related to TC ($p<0.0001$) and LDL ($p<0.0001$).

Discussion

Sabour et al. (2018) conducted a high-level RCT to examine the efficacy of a nutritional education program on SCI patients' body lipid profile and body weight. There was no significant difference in lipid profile levels (total cholesterol ($p=0.224$), triglyceride ($p=0.172$), low density lipoprotein cholesterol ($p=0.107$) high-density lipoprotein cholesterol ($p=0.081$) when comparing the intervention group receiving the nutritional education to the control group. Furthermore, there were no significant weight or waist circumflex changes between the two groups. Thus, study authors concluded that the nutritional education program alone was not adequately effective to have a beneficial influence on weight and lipid profile in patients post-SCI. The authors comment on the short duration of the seven-month study as being a potential factor for the insignificant results, as well as the small sample size of 57 participants. This study did not take into consideration caregiver involvement in eating for the patients studied. Persons with SCI are substantially limited in mobility, and thus caregivers play a large role for feeding (Sabour et al. 2018), which was not evaluated in this study, but may have changed the outcomes.

Szlachcic et al. (2001) evaluated the effects of dietary education for individuals with SCI at least two years post-injury who had moderately elevated total cholesterol levels ($>5.2\text{mmol/L}$) and reported significant decreases in total and low-density lipoprotein cholesterol (LDLC). Individuals who were assessed at baseline as having total cholesterol values $>5.2\text{ mmol/L}$ (200 mg/dL) were referred to the staff registered dietitian for counseling. Specifically, individuals were advised to limit daily fat intake to $<30\%$ of total daily calories (kcal), daily saturated fat intake to $<10\%$ of daily calories, daily cholesterol intake to $<300\text{ mg}$ and to consume 60% of total daily calories as carbohydrate. Subjects in the treatment group were seen by a dietitian at least twice to assess their dietary compliance. The remaining 136 subjects (control group) did not receive nutrition consultation. Subjects in the treatment group were significantly older and were a greater number of years post injury than those in the control group; therefore, changes in lipid profile were analyzed controlling for differences in age and duration post-injury. A greater number of individuals in the treatment group demonstrated a significant decrease in TC (69%) compared to the control group (43%). These declines were also demonstrated for LDL in the treatment and control group (67% versus 47% , respectively). There were no significant changes in HDL for either group although one third of all subjects in both groups had HDL values below the recommended range at baseline. Finally, 60% of the treatment group and 45% of control group had declines in triglycerides levels.

In a cross-sectional, observational study by Kourtrakis et al. (2018), 174 chronic SCI patients were studied to determine dietary, lifestyle and clinical factors associated with plasma 25-hydroxyvitamin D [25(OH)D] levels. Answers from the food frequency questionnaire (FFQ) were converted to daily intake of various nutrients. Independent factors positively associated with higher plasma 25(OH)D included vitamin D intake, age, hours of planned exercise, female sex, white race, wine consumption, and if a never or former smoker. The most important predictor of 25(OH)D was supplement intake. The majority of subjects (98.9% for vitamin D and 74.1% for calcium) did not meet the recommended daily allowance for adults from their diet alone. Level and completeness of SCI, injury duration, mobility, % total body fat, time outside, and comorbid diseases were not associated with plasma 25(OH)D (Kourtrakis et al, 2018). It was concluded that plasma 25(OH)D level in chronic SCI patients is not associated with clinical factors specific to SCI

such as injury level and completeness, injury duration, and mobility mode, but related to supplement intake and other lifestyle factors.

Javidan et al. (2017) evaluated the probable effect of dietary amino acid intake patterns on serum lipid profiles and BP in chronic SCI participants. Dietary intakes were assessed by recording consumed foods by 24-hour dietary recall interviews. Dietary intake of lysine was positively related to levels of fasting plasma glucose (FPG), triglyceride (TG), systolic blood pressure (SBP) and diastolic blood pressure (DBP) ($p < 0.0001$, 0.046, 0.002 and 0.009, respectively). There was a positive significant relationship between the intake of cysteine and levels of TG and SBP as well ($p = 0.027$ and 0.048, respectively). Higher intakes of threonine and leucine had a negative relationship with TG level ($p = 0.001$ and 0.026, respectively). Furthermore, tyrosine, threonine and leucine were inversely correlated to blood pressure. Total cholesterol level was only related to intake of threonine and leucine ($p = 0.004$ and 0.012, respectively). FPG was positively associated with intake of all amino acids except for cysteine, glutamic acid, threonine, leucine and histidine.

Conclusion

There is level 1b evidence (from one RCT; Sabour et al. 2018) that a nutritional education program alone does not influence body weight or lipid profile compared to a control group.

There is level 2 evidence (from one prospective controlled trial; Szlachic et al. 2001) that standard dietary counseling (total fat <30% of kcal, saturated fat <10% of kcal, cholesterol <300 mg, carbohydrate 60% of kcal) can reduce total and low density lipoprotein cholesterol among individuals with SCI who have total initial cholesterol >5.2 mmol/L.

There is level 5 evidence (from one observational study; Kourtrakis et al. 2018) that plasma 25(OH)D level in chronic SCI patients is not associated with clinical factors specific to SCI.

There is level 5 evidence (from one observational study; Javidan et al. 2017) that dietary intake of lysine may positively relate to levels of FPG, TG, SBP and DBP; intake of cysteine may positively relate to levels of TG and SBP; higher intakes of threonine and leucine may have a negative relationship with TG level; tyrosine, threonine and leucine may be inversely correlated to BP. However, these correlations may be due to an increase in total calories.

Dietary counseling alone does significantly improve results in lipid profile; consultation with a registered dietitian should be obtained as individualized diets may enhance compliance.

Dietary intake of plasma 25(OH)D is not associated with clinical factors specific to SCI; however dietary intake of amino acids may impact serum lipid profile and BP in chronic SCI patients.

7.2 Omega-3 Fatty Acid Supplementation

Studies suggest that n-3 polyunsaturated (omega-3) fatty acids have beneficial effects on cardiovascular disorders including anti-inflammatory, antithrombotic, hypolipemic and vasodilatory effects and contribute to primary and secondary prevention of ischemic heart disease in the general population (Hirafuji et al. 2003; Simopoulos 1999). Omega-3 fatty acids are found primarily in fatty fish and in smaller amounts in flax, soy, canola, olive and wheat germ oils and black walnuts. Studies have also suggested that eicosapentaenoic acid (EPA) and

docosahexaenoic acid (DHA) supplementation may result in changes in red blood cells (Andersson et al. 2002; Bruckner et al. 1987; Cartwright et al. 1985; Terano et al. 1983) which in turn may improve oxygen delivery to working muscles. Another study has shown that fish oil supplementation may facilitate fat oxidation (Delarue et al. 2003). Supplementation with EPA and DHA may improve VO₂ max and aerobic performance.

Table 10 Omega-3 Fatty Acid Supplementation on Lipid Profile and Physical Performance

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Javierre et al. 2006 Spain Pre-Post N=21	<p>Population: Mean age=34 yr; Gender: males=21, females=0; Level of injury: paraplegia=18, tetraplegia=3; Severity of injury: AISA A=57%, B=28%, C=10%, D=5%; Time since injury=8.5 yr.</p> <p>Intervention: Omega 3-fatty acid supplementation for 6 mo: 1.5 g/day docosahexaenoic acid (DHA), 0.60 g/day eicosapentaenoic acid (EPA) and 9 mg/day alpha-tocopherol combined in a pill. Two pills were taken three times daily.</p> <p>Outcome measures: Lab exercise test (O₂ uptake, CO₂ production, ventilation exchange [VE], heart rate, skin temperature, arm and leg blood pressure), dynamometry test, and aerobic field test.</p>	<ol style="list-style-type: none"> 1. At 3 months, the study showed an increased plasma concentration of DHA and EPA (p<0.05). 2. As the study progressed a significant decrease was seen in O₂ consumption (p<0.05) and systolic blood pressure (p=0.012). 3. Body weight, glucose levels, uric acid and lactate remained constant. 4. As workload increased, O₂ uptake increased; however, this trend progressively declined over time (p<0.001). 5. Time to complete 20 reps at 70% maximum load declined by 28% from baseline to 3mo testing , 13% from 3mo testing to 6mo testing and 41% from baseline to 6mo testing. 6. All muscle groups showed a significant improvement (p<0.05). 7. No improvement was noted for the aerobic test although the anaerobic test showed improvement in time for a 90 meter distance between day 1 and 2 only (p<0.05).
Javierre et al. 2005 Spain Pre-post N=19	<p>Population: Severity of injury: AISA A–D; Time since injury=>12 yr.</p> <p>Intervention: Subjects were administered daily doses of 1.5 g docosahexaenoic acid (DHA) and 0.75 g eicosapentaenoic acid (EPA) in the form of gelatin pearls, 6 per day to be taken with their principal meals.</p> <p>Outcome Measures: Plasma DHA, EPA, total, very low density, low density, and high density lipoprotein, triglycerides, and overnight fasting glucose.</p>	<ol style="list-style-type: none"> 1. Plasma EPA and DHA increased significantly (p<0.05) in response to the intake of the supplement at 3 months and 6 months (p<0.05). 2. No differences in all types of cholesterol, triglycerides, or glucose were observed.

Discussion

Javierre et al. (2005) assessed the effects on lipid profile and fasting blood glucose in 19 adult males with SCI at 3 and 6 months following daily supplementation of 1.5 grams DHA and 0.75 grams of EPA. Despite significant increases in the plasma concentration of DHA and EPA, plasma concentrations of glucose, total cholesterol, HDL, LDL, very low density lipoprotein cholesterol (VLDL), and triglycerides did not show differences as the result of n-3 fatty acid supplementation.

In a follow-up study Javierre et al. (2006) determined whether omega-3 fatty acid supplementation contributed to improved muscle strength and endurance capacity in persons with SCI. Twenty-one males, 18 with paraplegia and three with tetraplegia, underwent global physical evaluations at baseline, three months and six months of receiving omega-3 fatty acid supplementation. Participants continued with their usual diet while taking 1.5 grams per day of DHA and 0.60 grams per day of EPA plus 9 mg of alpha tocopherol provided in capsules; two capsules at each of three meals were consumed. No adverse effects were observed during the supplementation period. Increases in the concentrations of plasma DHA and EPA were observed. Body weight of the participants was stable during the study. There was an observed improvement in the functional capacity of the neuromuscular system as shown in enhanced strength and endurance of the upper-body musculature in the tests performed by the subjects.

Conclusion

There is level 4 evidence (from one pre-post study; Javierre et al. 2005) that daily supplementation with DHA (1.5 g) and EPA (0.75 g) increases plasma DHA and EPA levels but does not alter total cholesterol, very low-, low-, or high-density lipoprotein, triglycerides, or glucose.

There is level 4 evidence (from one pre-post study; Javierre et al. 2006) that DHA and EPA supplementation increases upper body strength and endurance in persons with SCI.

Blood concentrations of DHA and EPA increased as a result of supplementation; however, no significant changes in lipid profile were identified.

DHA and EPA supplementation increase upper body strength and endurance in persons with SCI.

8.0 Nutritional Interventions for Vitamin Deficiencies and Supplementation

Although little work has been done examining the vitamin profiles of individuals following SCI, it is generally thought that vitamin deficiency is a significant issue. Moussavi et al. (2003) reported that 16% to 37% of community-dwelling SCI subjects had serum levels below the reference range for vitamins A, C and E compared with general population norms.

A case-controlled study by Lynch et al. (2002) assessed complete blood count, white blood cells, iron status, ferritin, red blood cell folate, vitamin B12, magnesium, zinc, albumin and prealbumin in persons with chronic SCI and compared values to those of age and gender-matched non-SCI controls. Results were not outside the normal ranges for either group; however, the SCI group had significantly different median values than the control group for hemoglobin, white blood cell count, albumin, pre-albumin, serum iron and percentage saturation.

8.1 Vitamin D

Vitamin D deficiency is widespread and may result in a vast array of health consequences including osteoporosis, falls, increased cancer risk and altered glucose and lipid metabolism – the pathogenesis of diabetes and CVD. It plays an essential role in muscle and bone health, immunity and muscle signaling and has been linked with autoimmune disorders such as multiple sclerosis (Cantorna et al. 2006; Cherniak et al. 2008; Ford et al. 2005; Mathieu et al. 2005). Obesity has been associated with decreased bioavailability of vitamin D, and percentage body fat is inversely

related to vitamin D levels and directly correlated with parathyroid hormone (PTH) levels (Snijder et al. 2005; Wortsman et al. 2000).

The skeletal effects of hypovitaminosis D are evidenced in progressive stages such as calcium malabsorption with secondary elevation of PTH, increased bone remodeling and osteoporosis and further histologic changes related to continued lack of calcium and poor mineralization (Heaney 1999).

Individuals with SCI have an increased occurrence of vitamin D deficiency, resulting from a number of factors including decreased exposure to sunlight, inadequate dietary intake and the effect of medications (Hummel et al. 2012). In turn, vitamin D deficiency promotes calcium deficiency and secondary hyperparathyroidism, resulting in further bone loss and exacerbating osteoporosis. Myopathy and nonspecific musculoskeletal pain may also develop as a consequence of vitamin D deficiency (Bauman et al. 2005; Holick 2005).

Bauman et al. (1995) reported that 32 of 100 SCI subjects had 25(OH)D levels below normal range and 11 of 32 had elevated serum PTH levels. Zhou et al. (1993) measured the 25(OH)D, serum calcium, magnesium and albumin concentrations of 92 men with SCI, 38 of whom had single or multiple pressure ulcers, and compared these values with those of non-SCI controls. The SCI group had lower serum 25(OH)D, total calcium, and albumin concentrations. Individuals with tetraplegia had lower 25(OH)D levels than those with paraplegia. Additionally, the SCI subgroup with pressure ulcers demonstrated significantly lower serum 25(OH)D, calcium and magnesium levels than the SCI subjects without ulcers.

Serum 25-hydroxyvitamin D is the best test for assessing vitamin D levels in the following clinical scenarios where patient vitamin D levels may be abnormal including (Thatcher & Clark 2011):

- Significant renal or liver disease
 - Osteomalacia, osteopenia or osteoporosis
 - Malabsorption syndromes
 - Hypo or hypercalcemia/hyperphosphatemia
 - Hypo or hyperparathyroidism
 - Prescribed medications that affect vitamin D metabolism such as phenobarbital, carbamazepine, phenytoin and valproate
 - Prescribed medications that might interfere with vitamin D absorption such as cholestyramine, colestipol and orlistat
 - Unexplained increased levels of serum alkaline phosphatase
- Intake of high dose vitamin D combined with symptoms suggesting vitamin D toxicosis, i.e., hypervitaminosis D

There is increasing support for vitamin D supplementation beyond present recommendations. Additional studies are needed to establish the best diagnostic and supplementation guidelines for different populations (Cherniak et al. 2008).

Table 11 Vitamin D Supplementation Post SCI

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Amorim et al. 2018 Portugal	Population: Mean Age=47.0±10.6 yr; Gender: males=13, females=1; Time since	1. Over the 8-wk study, the amount of 25(OH)D improved significantly (p<0.05).

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
RCT PEDro=9 N=14	<p>injury=26.1±34.2 mo; Level of injury: C=3, T=6, L=5; Severity of injury: AIS A=4, B=0, C=5, D=5.</p> <p>Intervention: Participants were randomized to creatine (3g daily), vitamin D (25000 IU per two weeks) or placebo group and completed a double-blinded eight-week progressive resistance training program.</p> <p>Outcome Measures: Amount of 25-hydroxyvitamin D (25(OH)D), Sum four skinfolds, Arm muscle area, Manual wheelchair slalom test (MWST), Medicine ball throw, Handgrip strength, Chest press, Triceps, Pec deck, Lat pulldown.</p>	<div><div>2.</div><div>The amount of 25(OH)D improved significantly (p<0.05) when compared to the control group.</div></div> <div><div>3.</div><div>No significant improvements in any variable for the control group.</div></div> <div><div>4.</div><div>All variables improved significantly (p<0.05) over time in the creatine group except for the MWST.</div></div> <div><div>5.</div><div>In the vitamin D group, the correct arm muscle area, medicine ball throw, and chest press improved significantly (p<0.05) over time.</div></div> <div><div>1.</div><div>Corrected arm muscle area improved significantly (p<0.05) in the creatine group compared to the control group.</div></div>
<p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%C.I.) as calculated from pre- and post-intervention data.</p> <div><div><div>Amorim et al. 2018; Control vs. Creatine</div><div><div><div>Vitamin D (25(OH)D)</div><div>0.59 (-0.76,1.98)</div></div><div><div>Arm Muscle Area</div><div>0.52 (-0.82,1.85)</div></div><div><div>Manual Wheelchair Slolem Test</div><div>0.33 (-0.99,1.66)</div></div><div><div>Medicine Ball Throw</div><div>0.51 (-0.83,1.84)</div></div><div><div>Handgrip Strength</div><div>0.01 (-1.30,1.33)</div></div><div><div>Pec Deck</div><div>0.01 (-1.30,1.33)</div></div></div><div><div>-1.5</div><div>-1</div><div>-0.5</div><div>0</div><div>0.5</div><div>1</div><div>1.5</div><div>2</div></div><div><div>Favours Control</div><div>SMD (95%C.I.)</div><div>Favours Creatine</div></div></div><div><div><div>Amorim et al. 2018; Control vs. Vitamin D</div><div><div><div>Vitamin D (25(OH)D)</div><div>2.05 (0.43,3.67)</div></div><div><div>Arm Muscle Area</div><div>0.10 (-1.21,1.42)</div></div><div><div>Manual Wheelchair Slolem Test</div><div>0.06 (-1.25,1.38)</div></div><div><div>Medicine Ball Throw</div><div>0.09 (-0.25,1.13)</div></div></div><div><div>-1.5</div><div>-0.5</div><div>0.5</div><div>1.5</div><div>2.5</div><div>3.5</div></div><div><div>Favours Control</div><div>SMD (95%C.I.)</div><div>Favours Vitamin D</div></div></div></div></div>		

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes														
	<p style="text-align: center;">Amorim et al. 2018; Creatine vs. Vitamin D</p> <table><thead><tr><th>Outcome</th><th>SMD (95% C.I.)</th></tr></thead><tbody><tr><td>Vitamin D (25(OH)D)</td><td>1.70 (0.26, 3.15)</td></tr><tr><td>Sum Four Skinfolts</td><td>0.09 (-1.15, 1.33)</td></tr><tr><td>Arm Muscle Area</td><td>0.78 (-0.51, 2.07)</td></tr><tr><td>Manual Wheelchair Siolem Test</td><td>0.29 (-0.96, 1.53)</td></tr><tr><td>Medicine Ball Throw</td><td>0.45 (-0.81, 1.70)</td></tr><tr><td>Pec Deck</td><td>0.17 (-1.07, 1.42)</td></tr></tbody></table> <p style="text-align: center;">-1.5 -1 -0.5 0 0.5 1 1.5 2 2.5 3 3.5</p> <p style="text-align: center;">Favours Creatine SMD (95% C.I.) Favours Vitamin D</p>		Outcome	SMD (95% C.I.)	Vitamin D (25(OH)D)	1.70 (0.26, 3.15)	Sum Four Skinfolts	0.09 (-1.15, 1.33)	Arm Muscle Area	0.78 (-0.51, 2.07)	Manual Wheelchair Siolem Test	0.29 (-0.96, 1.53)	Medicine Ball Throw	0.45 (-0.81, 1.70)	Pec Deck	0.17 (-1.07, 1.42)
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Medicine Ball Throw	0.45 (-0.81, 1.70)															
Pec Deck	0.17 (-1.07, 1.42)															
Beal et al. 2018 USA Case-Control N=20	<p>Population: Mean age=47.0±11.8 yr; Gender: males=20, females=0; Time since injury=18.9±12.4 yr; Level of injury: T3-L11; Severity of injury: AIS A=17, B=3.</p> <p>Intervention: Participants were sorted into a high vitamin D consumption group and a low vitamin D consumption group based on their vitamin D consumption prior to the beginning of the study and different body measurements were recorded based on their vitamin D consumption.</p> <p>Outcome Measures: Vitamin D intake, Calcium intake, Total caloric intake, Percentage macronutrients, Total percent fat, Region percent fat, Fat mass, Lean mass, Sitting waist circumference, Sitting abdominal circumference, and metabolic profile (Fasting glucose, Low density lipoprotein (LDL), High density lipoprotein (HDL), Total cholesterol, Triglycerides (TG), Insulin sensitivity (Si), Glucose effectiveness (Sg)).</p>	<ol style="list-style-type: none">2. Total Vitamin D intake significantly different (p=0.0001) between groups.3. Calcium intake significantly different (p=0.0157) between groups.4. Total caloric intake significantly different between groups (p=0.02).5. Vitamin D intake positively related to total caloric intake (p=0.0001) and total caloric intake adjusted to body weight (p=0.0001).6. Vitamin D intake adjusted to total dietary intake positively related to Si adjusted to body weight (p=0.004) and Si adjusted to lean mass (p=0.012).7. Vitamin D intake adjusted to total dietary intake positively related to Sg (p=0.016) and Sg adjusted to body weight (p=0.018).8. Percentage macronutrients not significantly different between groups (p>0.05).9. No significant difference in total percent fat, region percent fat, fat mass, lean mass, sitting waist circumference, sitting abdominal circumference between groups (p>0.05).10. Insulin sensitivity not significantly different between the groups (p=0.13).11. Glucose effectiveness not significantly different between two groups (p=0.1257).12. Glucose effectiveness still not significantly different when controlled for body weight (p=0.1337) or lean mass (p=0.2044).13. Total cholesterol significantly different between the groups (p=0.0354).14. No significant difference in LDL (p=0.0654) or HDL (p=0.3993) between the two groups.15. Total cholesterol to HDL ratio not significantly different between groups (p=0.2645).16. TG not significantly different between groups (p=0.3934).														
Bauman et al. 2005 USA	<p>Population: Study 1: Mean age=53 yr; Study 2: Mean age=43 yr.</p>	Study 1:														

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Pre-post N _{Study 1} =10; N _{Study 2} =40	<p>Intervention: <i>Study 1:</i> All patients were given 50 µg (2000 IU) vitamin D₃ 2x/wk and 1500 mg elemental calcium daily for 2 wk. <i>Study 2:</i> 10 µg (400 IU) vitamin D₃, a multivitamin with an additional 10 µg (400 IU) vitamin D₃, and 500 mg elemental calcium daily for 12 mo.</p> <p>Outcome Measures: Changes in serum 25(OH)D, calcium and parathyroid hormone (PTH), and urinary calcium.</p>	<ol style="list-style-type: none"> After 2 weeks, serum 25(OH)D increased ($p<0.005$) but 8 of 10 subjects still had values below the normal range (<16 ng/mL). Serum PTH decreased from 35 to 18 pg/mL ($p<0.05$), serum calcium was not significantly different, and urinary calcium increased from 103 to 239 mg/d ($p=0.010$). <p><i>Study 2:</i></p> <ol style="list-style-type: none"> At baseline, 33 subjects were vitamin D deficient (<16 ng/mL) compared to 9 after 12 months. After 6 and 12 months, serum 25(OH)D increased ($p<0.0001$). <p>Serum PTH decreased ($p<0.005$), but serum calcium did not change.</p>
Hummel et al. 2012 Canada Case Series N=62	<p>Population: Mean age=49±12 yr; Gender: males=51 male, females=14; Time since injury: >2 yr; Cause of injury=traumatic=62, non-traumatic=0.</p> <p>Intervention: Blood draw for serum sample.</p> <p>Outcome Measures: Serum 25(OH)D and PTH.</p>	<ol style="list-style-type: none"> 39% of the cohort had suboptimal serum 25(OH)D levels. Factors associated with suboptimal vitamin D levels included having vitamin D assessed in the winter months (odds ratio (OR)=7.38, $p=0.001$), lack of calcium supplement (OR=7.19, $p=0.003$), lack of vitamin D supplement (OR=7.41, $p=0.019$), younger age (OR= 0.932, $p=0.010$), paraplegia (OR=4.22, $p=0.016$), and lack of bisphosphonate (OR=3.85, $p=0.015$). Significant associations were observed between serum PTH and 25(OH)D ($r=-0.304$, $p=0.032$) and between PTH and C-telopeptide of type I collagen (CTX-I) ($r=0.308$, $p=0.025$).

Discussion

A high-level RCT by Amorim et al. (2018) determined the effects that creatine (3g daily) versus vitamin D (25000 IU each two weeks) supplementation had on muscle strength in 13 chronic SCI individuals undergoing resistance training. The mean value of 25(OH)D from all participants prior to the intervention was 13.3ng/ml. 71.4% were deficient in vitamin D (<20 ng/ml), 28.6% had insufficient values of vitamin D (20–30ng/ml) with no participant being reported with sufficiency status. The corrected arm muscle area improved significantly ($p<0.05$) in creatine group relatively to the control group. There was a significant correlation ($p<0.05$) between the one repetition maximum Pec deck and levels of vitamin D. It was concluded that supplementation with creatine may improve muscle strength parameters in individuals with spinal cord injury. Vitamin D deficiency is highly prevalent in this population. It is recommended an initial screening of vitamin D levels at the beginning of the physical rehabilitation process.

A case-control study by Beal et al. (2018) determined that persons with SCI consume much less than the recommended guidelines for daily vitamin D consumption. 20 male chronic SCI patients were assigned to either a high vitamin D intake group, or a low vitamin D intake group. The high vitamin D group had an average intake of 5.33 ± 4.14 mcg compared to low vitamin D group, 0.74 ± 0.24 mcg. None of the participants met the recommended guidelines for daily vitamin D intake. The higher vitamin D group had a significantly lower ($p=0.035$) total cholesterol

(148.00±14.12 mg/dl) than the lower vitamin D group (171.80±36.22 mg/dl). Vitamin D adjusted to total dietary intake was positively correlated to improvement in Si and Sg ($p<0.05$). These results suggest that the high vitamin D intake group consumed greater total caloric intake with similar macronutrients compared to the low vitamin D intake. Serum vitamin D levels were not evaluated in this study; however, study authors expect that their population to have 25(OH)D levels within a similar range to Bauman et al. (2005) [10.2–19.6 ng/mL.52]. The calcium intake for the higher vitamin D intake group was within the daily calcium recommendation (700–1300 mg/d),¹ while the calcium intake for the lower vitamin D intake group was significantly lower than that daily recommendation

Bauman et al. (2005) determined that healthy individuals with chronic SCI living in the community had vitamin D deficiency. Ten subjects with chronic SCI and a diagnosis of absolute vitamin D (25(OH)D) deficiency received 50 ug (2000 IU) of vitamin D₃ twice per week for two weeks in addition to 1.5 grams (1500 mg) of elemental calcium daily. Serum 25(OH)D levels significantly increased by day 14; however, levels remained below normal range in eight out of ten subjects. Serum calcium level was not significantly different, urinary calcium significantly increased, and serum PTH levels significantly decreased. In their second study Bauman et al. (2005) gave forty subjects 10 ug (400 IU) of vitamin D₃ daily in addition to a multivitamin that contained 10 ug (400 IU) vitamin D₃ daily for 12 months. All subjects received this treatment regardless of their initial serum vitamin D status. Subjects were encouraged to have at least 0.8 grams (800 mg) of calcium in their daily diet and were supplemented daily with 0.5 grams (500 mg) elemental calcium. Vitamin D levels significantly increased between baseline and follow-up at 6 and 12 months. There was no significant association between level of injury and baseline 25(OH)D levels. Serum and ionized calcium were not significantly different after 12 months of treatment although serum PTH was significantly reduced at 6 and 12 months. It is important to note that at baseline, 33 of the 40 subjects had 25(OH)D levels that were below the lower limit of normal, and that after 12 months of supplementation at 800 IU, only eight of the 40 subjects had serum 25(OH)D values greater than 30 ng/mL. These levels are not adequate in reversing elevated parathyroid levels and reducing bone turnover, despite significant decreases in PTH at 12 months. In conclusion, vitamin D₃ supplementation resulted in significant increases in 25(OH)D levels and reductions in PTH; however, suboptimal 25(OH)D levels persisted, suggesting the need for higher doses of vitamin D₃ supplementation and/or longer periods of administration.

Conclusion

There is level 1b evidence (from one RCT; Amorim et al. 2018) that supplementation with creatine, as well as vitamin D may improve muscle strength parameters in individuals with spinal cord injury.

There is level 3 evidence (from one case-control study; Beal et al. 2018) that chronic SCI patients suffer from hypovitaminosis of vitamin D. Increasing the daily intake of vitamin D improves glucose homeostasis, and may also improve total cholesterol.

There is level 4 evidence (from one pre-post study; Bauman et al. 2005) that vitamin D supplementation raises serum 25(OH) D levels in persons with chronic SCI. However, the dose and duration required to ensure vitamin D sufficiency remains unclear.

Vitamin D deficiency is highly prevalent in individuals with chronic SCI.

Individuals with SCI should be screened for vitamin D deficiency according to guideline practices and, if when necessary, replacement therapy should be initiated.

8.2 Vitamin B₁₂

The prevalence of vitamin B₁₂ deficiency in persons with SCI is reported to be between 5.7% and 19% (Petchkrua et al. 2002). Symptoms may include declining gait, depression or fatigue, upper limb weakness, memory loss and worsening pain (Petchkrua et al. 2002; Petchkrua et al. 2003). Vitamin B₁₂ deficiency usually responds to supplementation.

Petchkrua et al. (2002) conducted a retrospective chart review of patients with SCI who had received serum vitamin B₁₂ testing over a 10 year period. The most common symptoms among subjects identified as having deficient, subnormal or low normal vitamin B₁₂ levels were declining gait, depression, fatigue, upper limb weakness, memory loss or worsening pain. In this report, greater than half of the cases of probable vitamin B₁₂ deficiency occurred in young persons with no known risk factors. Neurologic and/or psychiatric symptoms improved in 88% of SCI subjects following high-dose oral or monthly parenteral vitamin B₁₂ supplementation. It is recommended that clinicians conduct early screening and treatment of vitamin B₁₂ deficiency.

In a follow-up cross-sectional study, Petchkrua et al. (2003) prospectively collected blood samples and reviewed medical records to assess the prevalence of vitamin B₁₂ deficiency in persons with SCI. Biochemical vitamin B₁₂ deficiency was reported in 13% of the subjects. While hematologic abnormalities were infrequent, neuropsychiatric symptoms were observed in half of the subjects. The age range most associated with vitamin B₁₂ deficiency was 40-59 years; among subjects older than 59 years, 9% had B₁₂ deficiency. No deficiency was noted in subjects within the age range of 20-39 years. Deficiency was more predominant in subjects with a complete SCI.

Given the potential for permanent neurological deficits, the relatively low cost of screening and the low cost and high efficacy of high-dose oral supplementation, Petchkrua et al. (2002) suggest that clinicians conduct early screening and treatment of vitamin B₁₂ deficiency. Additional investigations into the predisposing risk factors for vitamin B₁₂ deficiency in persons with SCI are warranted.

Clinicians should conduct early screening for and treatment of vitamin B₁₂ deficiency.

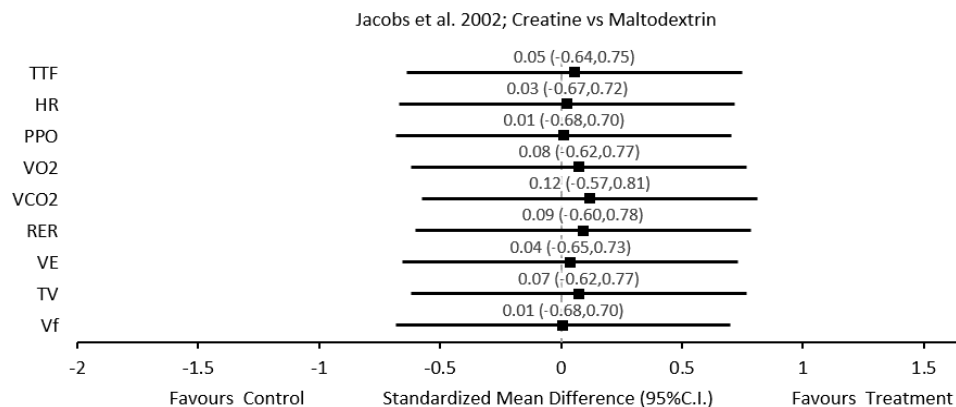
8.3 Creatine

Synthesized by the liver, kidney and pancreas, creatine occurs naturally and is found primarily in skeletal muscle. It can be obtained from eating foods rich in creatine such as meat and fish or be consumed in the form of supplement powders. The most predominant form of creatine is phosphocreatine which contributes to the rapid re-synthesis of adenosine triphosphate (ATP)

during short-term, high-intensity exercise. Dietary supplementation of creatine has been shown to improve strength, power and recovery from high-intensity exercise in the non-SCI population (Balsom et al. 1995; Casey et al. 1996; Earnest et al. 1995; Harris et al. 1993). Creatine serves as a short duration reservoir for the energy required for muscle contraction in skeletal muscle. Low levels of intramuscular creatine are seen in some neuromuscular diseases. Creatine supplementation improves muscle strength in some patient populations with neurological disorders (Kendall et al. 2005).

Table 12 Creatine Administration Post SCI

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Kendall et al. 2005 USA Cross-over RCT PEDro=9 N _{Initial} =9; N _{Final} =8	<p>Population: Level of injury: C5/C6; Severity of injury: AIS A-C; Time since injury=16.5 yr.</p> <p>Intervention: Subjects were randomized into one of two groups receiving either 10 g creatine orally twice daily for six days, then maintained on 5 g daily until testing, or placebo. After a wash-out period they crossed over to the other arm.</p> <p>Outcome Measures: Grasp and Release Test (GRT) and Functional Independence Measure.</p>	1. There were no significant between-group differences for GRT or FIM scores.
Jacobs et al. 2002 USA Cross-over RCT PEDro=8 N=16	<p>Population: Mean age=35.3 yr; Gender: males=16 females=0; Level of injury: tetraplegia; Mean weight=71.4 kg.</p> <p>Intervention: Individuals received 20 g of creatine monohydrate 4x/day mixed with 8 oz water or placebo powder for 1 wk. A washout period occurred for 3 wk and then individuals crossed over to receive the alternate treatment protocol.</p> <p>Outcome Measures: Power output, time to fatigue, heart rate (HR), oxygen uptake (VO₂), minute ventilation (V_E), ventilatory frequency (V_F), respiratory exchange ratio (RER), tidal volume (V_T).</p> <p>Effect Sizes: Forest plot of standardized mean differences (SMD ± 95%CI.) as calculated from pre- and post-intervention data.</p>	<p>1. No adverse effects were reported.</p> <p>2. There was no change in HR, RER and V_E, although there were significant difference in VO₂, VCO₂, V_F and V_T between trials (p<0.001).</p> <p>3. VO₂ increased by 18.6% with creatine treatment versus placebo.</p> <p>4. After creatine consumption, VO₂, VCO₂ and V_T reached their highest peak.</p>



Discussion

Kendall et al. (2005) reported findings of a study that sought to determine whether creatine supplementation improves muscle strength, endurance and function in weak upper limb muscles in person with tetraplegia. Eight individuals with tetraplegia and mild wrist extensor weakness were randomized to receive creatine or a placebo in a double-blind crossover design. During creatine supplementation, participants received oral doses of creatine monohydrate powder. There was no change in any of the functional tests performed by the participants and none of the participants had a change in self-reported motor Functional Independence Measure scores.

Persons with SCI have decreased upper extremity work capacity. Individuals with cervical SCI have limited proficiency in the repeated tasks of daily living that require endurance and strength (Hopman et al. 1992; Jehl et al. 1991; Lin et al. 1993; Van Loan et al. 1987). A study by Jacobs et al. (2002) sought to determine the effects of oral creatine monohydrate supplementation on upper-extremity work capacity in persons with complete cervical SCI. Sixteen men with complete tetraplegia (C5-7) were randomly assigned to one of two groups and received either 20g of creatine monohydrate supplement powder daily or placebo for the first treatment phase; treatment was reversed in the second phase. Each treatment phase lasted for 7 days with a 21-day washout period. Peak power output, time to fatigue, HR, and metabolic measures including oxygen uptake, minute ventilation, tidal volume and respiratory frequency were assessed. Significantly greater values of oxygen uptake, tidal volume and carbon dioxide production were observed in the groups receiving the creatine monohydrate supplementation. The investigators concluded that creatine supplementation enhances exercise capacity in persons with complete tetraplegia and may promote greater exercise training benefits.

Conclusion

There is level 1a evidence (from one RCT; Kendall et al. 2005) that creatine supplementation did not result in improvements in wrist extensor strength or muscle function.

There is level 1a evidence (from one RCT cross-over trial; Jacobs et al. 2002) that creatine supplementation enhances exercise capacity in persons with complete tetraplegia and may promote greater exercise training benefits.

Creatine supplementation does not result in improvements in muscle strength, endurance or function in weak upper limb muscles.

Creatine supplementation enhances exercise capacity in persons with complete tetraplegia and may promote greater exercise training benefits.

9.0 Cardiovascular and Hormonal Responses to Food Ingestion

Persons with chronic primary autonomic failure and widespread sympathetic denervation and postural hypotension often have postprandial hypotension (Mathias 1991). Food consumption often exacerbates symptoms and the degree of postural hypotension in certain groups. The cardiovascular responses to food ingestion in individuals with tetraplegia have been investigated.

Table 13 Cardiovascular and Hormonal Responses to Food Ingestion

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Baliga et al. 1997 USA Prospective Controlled Trial N=11	<p>Population: Tetraplegia (N=6): Mean age=33 yr; Gender: males=6, females=0; Severity of injury: complete; Paraplegia (N=5): Mean age=36 yr; Gender: males=4, females=1; Severity of injury: complete.</p> <p>Intervention: Individuals with tetraplegia received a milk-based liquid meal including Complan and glucose supplements (66 g carbohydrates, 22 g fat, 18 g protein, 550 Kcal, 300 mL). Individuals with paraplegia served as controls.</p> <p>Outcome Measures: Blood pressure [mean arterial (BP), systolic (SBP) and diastolic (DBP)]; plasma noradrenaline and adrenaline levels; heart rate (HR); serum osmolality, renin activity, glucose, and other electrolytes.</p>	<ol style="list-style-type: none"> 1. Those with tetraplegia had higher basal SBP and HR, but lower basal DBP compared to controls; after the meal, BP decreased in the treatment group (not significant) but not for controls 2. Basal plasma noradrenaline levels were lower for the treatment group compared to the controls; after the meal, the controls group's plasma noradrenaline levels increased ($p<0.05$), while the treatment group's did not. 3. Basal renin activity and glucose level among the treatment group increased after ingestion ($p<0.05$), but the control group's did not. 4. HR and all other measures did not change in either group.

Discussion

A fall in BP following the ingestion of food has been described in individuals with secondary autonomic failure of various causes. A single study has assessed cardiovascular and hormonal responses to food ingestion among individuals with tetraplegia from cervical spinal cord transection. Baliga et al. (1997) investigated the effects of a standard liquid meal (300 mL total liquid volume, 550 kilocalories, 66 grams carbohydrate, 22 grams fat, 18 grams protein) on BP, HR and neurohormonal levels. Five individuals with paraplegia with complete lesions (T12-L3) and essentially intact sympathetic nervous systems who did not experience postural hypotension served as the control group. The experimental group consisted of six individuals with tetraplegia (C4-7) with chronic and complete cervical spinal cord transection. All had postural hypotension. After food ingestion there was no change in BP or HR in either group. Plasma noradrenaline was unchanged for those with tetraplegia but rose in those with paraplegia. Conversely, plasma renin activity rose among those with tetraplegia but not paraplegia.

Conclusion

There is level 2 evidence (from one prospective controlled trial; Baliga et al. 1997) that consumption of a standard liquid meal does not change blood pressure, heart rate or noradrenalin levels in individuals with tetraplegia and postural hypotension.

Consumption of a standard liquid meal does not change blood pressure, heart rate or noradrenalin levels in individuals with tetraplegia and postural hypotension.

10.0 Effects of Nutrient Intake on Ambulation Performance

Reconditioning exercises pursued by persons with incomplete SCI have shown to reverse the decline in function imposed by the paralysis (Jacobs et al. 2001). Nutrition-related modifications

that optimize physical performance for individuals with SCI have not been studied extensively compared to that of individuals without disability.

Table 14 Nutrient Intake on Ambulation Performance

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Nash et al. 2007 USA PEDro=5 Cross-over RCT N=3	<p>Population: Mean age=38.7 yr; Gender: males=2, females=1; Level of injury: paraplegia=1, tetraplegia=2; Severity of injury: AIS C=2, D=1; Time since injury=11.3 yr.</p> <p>Intervention: On a 24m oval track, subjects walked to fatigue consecutively over 5 days. In trial 1, once fatigued, subjects consumed 48 g of vanilla whey and 1g/kg patient body weight of carbohydrate (CHO). Control subjects received a soy placebo solution. Subjects rested over the weekend then repeated the procedure. After a two week washout period, individuals crossed over to receive the alternate treatment.</p> <p>Outcome Measures: Ambulation time, distance walked, and energy expenditure.</p>	<p>1. Regardless of testing order, the average ambulation time was 17.8% longer (32.0 min versus 27.1 min), distance walked was 37.9% longer (470 m versus 341 m), and energy expenditure to fatigue was 12.2% greater (731k J [174kcal] versus 651 kJ [155kcal]) with the whey and CHO supplement than placebo.</p>

Discussion

Dietary, pharmacologic and nutrient modifications that may optimize physical performance for individuals with SCI have not been extensively studied. In the non-SCI population an effective nutrient supplementation combination to hasten recovery from intense activity and to improve performance in subsequent bouts of exercise is whey protein and carbohydrate (Ivy 1998; Ivy 2001). Nash et al. (2007) investigated the effect of protein and carbohydrate intake on ambulation in three persons with incomplete SCI (C5-T4). The subjects walked to fatigue on five consecutive days; upon fatigue, participants consumed 48 grams of whey plus 1 gram per kilogram of body weight of carbohydrate. The process was repeated following a weekend of rest. Following a 2-week wash-out period the process was repeated using 48 grams of soy supplement. The authors concluded that the combination of whey protein plus carbohydrate supplement ingestion following fatiguing ambulation improved subsequent ambulation by increasing distance, time to fatigue and caloric expenditure compared to soy supplement consumption.

Conclusion

There is level 2 evidence (from one RCT cross-over trial; Nash et al. 2007) that the consumption of a whey protein plus carbohydrate supplement following fatiguing ambulation improves subsequent ambulation by increasing distance, time to fatigue and caloric expenditure in persons with incomplete SCI.

The consumption of a whey protein plus carbohydrate supplement following fatiguing ambulation improves subsequent ambulation by increasing distance, time to fatigue and caloric expenditure in persons with incomplete SCI.

11.0 Post-Meal Resting Energy Expenditure

Food ingestion causes the metabolic rate to rise above the basal level (Jequier 1986; Lusk 1930). This rise in metabolic rate in the non-SCI population is initiated within minutes following meal ingestion, reaches its maximum after approximately one hour, and lasts up to 6 hours after food consumption. The mechanisms whereby nutrients stimulate energy expenditure are not fully understood. The potential role of the central sympathoadrenal system in the stimulation of nutrient-induced thermogenesis requires investigation.

Table 15 Post-Meal Resting Energy Expenditure

Author Year; Country PEDro Score Research Design Sample Size	Methods	Outcomes
Asknes et al. 1993 Sweden Prospective Controlled Trial N=13	<p>Population: SCI (N=7): Age range=21-34 yr; Gender: males=7, females=0; Severity of injury: Frankel A=7, complete=7, incomplete=0; Time since injury=1-11 yr; Non-SCI controls (N=6): Age range=18-30 yr; Gender: males=6, females=0.</p> <p>Intervention: Patients were divided into two groups and received either a liquid form mixed meal (52% carbohydrates, 37% fat, and 11% protein) or water.</p> <p>Outcome Measures: O₂ uptake, respiratory exchange (RE), blood glucose (BG), insulin, catecholamines, heart rate (HR), and energy expenditure (EE).</p>	<ol style="list-style-type: none"> 1. Basal O₂ uptake, EE, BG, insulin and noradrenaline levels was lower in the treatment group compared to controls ($p<0.01$ for all); HR was high for both groups before ingestion ($p<0.05$). <p>Post-meal:</p> <ol style="list-style-type: none"> 2. Both groups had increased O₂ uptake although there were no between group differences. 3. The treatment group's RE rate reached a maximum at 90 min ($p<0.05$) while the controls had an extremely high rate at 15 min which dropped to a rate similar to the treatment group. 4. Mean EE was higher in the treatment (17 W) versus control (14 W) group. 5. HR increased 6-7 beats/min in both groups ($p<0.05$). 6. The treated group had higher BG and insulin levels compared to controls ($p<0.05$); noradrenaline levels did not change.

Discussion

An increase in metabolic rate above basal levels following food ingestion is known as nutrient-induced thermogenesis (Jequier 1986; Lusk 1930). This post-meal rise in metabolic rate is significant to daily heat production and body weight homeostasis and may have a potential role in counteracting the development of obesity. In many obese individuals and in other conditions of insulin resistance, nutrient-induced thermogenesis is reduced below normal levels (Brundin et al. 1992; Pitt et al. 1976; Segal et al. 1985; Segal et al. 1990; Shetty et al. 1981). The rise in resting energy expenditure following food consumption has been generally considered to be mediated by central activation of the sympathoadrenal system. The purpose of a study by Asknes et al. (1993) was to determine the possible role of central sympathoadrenal stimulation for thermogenesis after ingestion of a normal mixed meal, in liquid form, in seven male subjects with chronic complete lesions of the cervical spinal cord (C4-C7). The thermogenic responses were compared to those in healthy males as well as to the responses in a control group of tetraplegic patients who received equal volumes of water instead of the liquid meal. The authors concluded that nutrient-induced thermogenesis in tetraplegic individuals with low sympathoadrenal activity is not diminished compared to healthy controls; efferent sympathoadrenal stimulation from the brain is not necessary for nutrient-induced thermogenesis.

Conclusion

There is level 3 evidence (from one prospective controlled trial; Asknes et al. 1993) that nutrient-induced thermogenesis is not decreased in individuals with tetraplegia with low sympathoadrenal activity; efferent sympathoadrenal stimulation from the brain is not necessary for nutrient-induced thermogenesis.

Nutrient-induced thermogenesis is not decreased in tetraplegic individuals with low sympathoadrenal activity; efferent sympathoadrenal stimulation from the brain is not necessary for nutrient-induced thermogenesis.

12.0 Cardiovascular, Endocrine and Renal Responses to Dietary Sodium Restriction

The kidneys are richly innervated by the sympathetic nervous system (Sutters 1992). The role of this sympathetic innervation in the adaptation to changes in dietary sodium intake in persons with spinal cord injury and impaired sympathetic nervous systems warrant study.

Table 16 Responses to Dietary Sodium Restriction

Author Year Country PEDro Score Research Design Sample Size	Methods	Outcomes
Sutters et al. 1992 USA Case Control N=15	<p>Population: Mean age=28 yr; Gender: males=15, females=0; Level of injury: paraplegia=6, tetraplegia=9; Time since injury=2 mo-1 yr.</p> <p>Intervention: All individuals received a sodium restriction diet over 5 days consisting of 90 g protein, 90 mmol potassium, and 300 g carbohydrates. Sodium intake was 260 mmol/day for days 1-3 but was reduced to 20 mmol/day on day 4 and 5.</p> <p>Outcome Measures: Total and fractional urinary sodium excretion, mean arterial pressure, creatinine clearance, plasma renin activity and atrial natriuretic peptide concentration compared between those with tetraplegia (dissociated sympathetic control (DSC)) and paraplegia (intact sympathetic control, (ISC)).</p>	<ol style="list-style-type: none"> 1. Total and fractional urinary sodium excretion fell in response to sodium restriction in both groups, but the fall in fractional sodium excretion was greater in the DSC group compared to ISC group. 2. Supine mean arterial pressure fell during the low salt period in the DSC group but was unaffected by salt restriction in the ISC group. 3. In the DSC group, creatinine clearance remained constant throughout the low salt period but fell during salt restriction in the ISC group. 4. Plasma renin activity was lower during salt loading in DSC subjects but increased more rapidly and to higher levels in response to salt restriction. 5. Plasma atrial natriuretic peptide concentration was higher in the DSC group during salt loading and salt restriction.

Discussion

In a study by Sutters et al. (1992) the effects of change from a high to low sodium diet on renal sodium and water excretion and hormonal responses were studied in nine individuals with tetraplegia (dissociated sympathetic control) and in six individuals with paraplegia (intact sympathetic systems). Given the results, the authors suggested that direct sympathetic control of the kidney is not required for renal sodium conservation in response to dietary salt restriction; however, is likely involved in the hemodynamic and hormonal responses.

Conclusion

There is level 3 evidence (from one case control study; Sutters et al. 1992) that sympathetic control of the kidney is not required for renal sodium conservation in response to dietary salt restriction.

Impairment of sympathetic control of the kidney secondary to SCI resulting in tetraplegia does not impact renal sodium conservation in response to dietary salt restriction.

13.0 Summary

There is a paucity of intervention studies investigating nutritional status and associated risk for persons with SCI. Many descriptive and observational publications address the risk for obesity, dyslipidemia and cardiovascular disease, impaired glycemic control and diabetes mellitus. Blood lipid profiles and indicators of impaired glucose tolerance and hyperinsulinemia of persons with SCI have been compared with those of non-SCI controls. Despite the high risk for CVD, and subsequent morbidity and mortality in individuals with SCI, few studies have assessed the benefits of nutrition-based risk reduction interventions. The majority of interventions have been limited to exercise. Other studies have investigated vitamin and mineral status of persons with SCI and compared values to those of non-SCI controls or to general population norms and have found lower levels of a variety of nutrients in the SCI population. Few publications have suggested screening and supplementation strategies to address these trends.

There is level 5 evidence (from one observational study; Sabour et al. 2016) that elevated protein intake can lower bone mineral densities in individuals with SCI.

There is level 5 evidence (from two observational studies; Tsunoda et al. 2015; Lieberman et al. 2014) that consumption of whole grains, vegetables, fruits and dairy products are important in maintaining adequate dietary intake.

There is level 5 evidence (from two observational studies; Pellicane et al. 2013; Sabour et al. 2012) that age and gender, but not level of injury, predict total caloric intake in individuals with SCI; further, level 5 evidence (from one observational study; Gorgey et al. 2015) suggests that individuals with chronic SCI often have a negative energy balance, consuming fewer calories than they burn.

There is level 5 evidence (from two observational studies; Wong et al. 2014; Wong et al. 2012) that individuals with SCI are at a significant risk for malnutrition and are at risk of worse clinical outcomes in the first year after injury.

There is level 5 evidence (from several observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Seidl et al. 2010, Abel, Ruf & Spahn 2004, Kirshblum et al. 1999) that VFSS and BSE are adequate measures of diagnosing dysphagia in a SCI population.

There is level 5 evidence (from four observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011) that VFSS and BSE are comparable in diagnosing dysphagia in a SCI population.

There is level 5 evidence (from one observational study: Wolf and Meiers 2003) that FEES is an adequate tool to diagnose dysphagia and monitor treatment progress in a SCI population.

There is level 5 evidence (from several observational studies; Hayashi et al. 2017, Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Shin et al. 2011, Seidl et al. 2010, Shem et al. 2005, Abel, Ruf & Spahn 2004, Brady et al. 2004 Kirshblum et al. 1999) that presence of a tracheostomy is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from six observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Seidl et al. 2010, Shem et al. 2005) that ventilator use is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from six observational studies; Hayashi et al. 2017, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011, Shin et al. 2011, Kirshblum et al. 1999) that increasing age is a risk factor for dysphagia in individuals with SCI.

There is level 5 evidence (from four observational studies; Chaw et al. 2012, Shem et al. 2012, Shem et al. 2012b, Shem et al. 2011) that presence of nasogastric tubes are a risk factor for dysphagia in individuals with SCI.

There is level 2 evidence (from one prospective controlled trial and one cohort study; Bennegard & Karlsson 2008; Bauman & Spungen 1994) that glucose uptake is higher in SCI individuals compared to non-SCI individuals.

There is level 2 evidence (from one cohort study and one pre-post study; Bauman & Spungen 1994; Bauman et al. 1999) that SCI individuals with tetraplegia have higher rates of altered glucose metabolism than other SCI individuals.

There is level 2 evidence (from one prospective controlled trial; Ketover et al. 1996) that diabetic and obese SCI individuals show impaired gallbladder emptying in response to a high fat meal compared to healthy SCI individuals.

There is level 1b evidence (from one RCT; Gorgey et al. 2012 and three pre-post studies; Chen et al. 2006, Betts et al. 2017 and Brochetti et al. 2018) that an intervention program combining diet and exercise is effective for reducing weight among overweight persons with SCI.

There is level 1b evidence (from two RCTs; Gorgey et al. 2012 and Li et al. 2018) that interventions targeting just diet and just exercise are both effective for reducing metabolic markers for obesity and associated metabolic diseases like diabetes and heart disease.

There is level 4 evidence (from two pre-post studies; Betts et al. 2017 and Brochetti et al. 2018) that lifestyle interventions that combine diet and exercise and provide accommodation to low-ambulatory participants are effective in participant compliance and retention during and post-intervention.

There is level 5 evidence (from one observational study; Nightingale et al. 2017) that it is important to incorporate strict dietary and exercise guidelines for patients with SCI because of how inconsistent those with SCI are with their daily energy intake and daily energy expenditure.

There is level 1b evidence (from one RCT and one secondary RCT analysis; Allison et al., 2017;2018) that a diet intervention focusing on anti-inflammatory diet changes and supplements can reduce inflammatory serum markers, but did not show improvement in motor nerve conduction compared to a control group.

There is level 1b evidence (from one RCT; Zemper et al. 2003) that improved health-related behaviours are adopted following a holistic wellness program for individuals with SCI.

There is level 4 evidence (from one pre-post study; Liusuwan et al. 2007) that an education program combining nutrition, exercise and behaviour modification is effective in increasing whole body lean tissue, maximum power output, work efficiency, resting oxygen uptake and shoulder strength in persons with SCI.

There is level 5 evidence (from one observational study; Hata et al. 2016) that social participation and social support have beneficial effects on an individual with SCI's self-rated health and dietary satisfaction.

There is level 5 evidence (from one observational study; Hata, Inayama, & Yoshiike, 2017) that the perceived food environment is associated with health related quality of life and diet satisfaction of community-dwelling SCI individuals.

There is level 1b evidence (from one RCT; Sabour et al. 2018) that a nutritional education program alone does not influence body weight or lipid profile compared to a control group.

There is level 2 evidence (from one prospective controlled trial; Szlachic et al. 2001) that standard dietary counseling (total fat<30% of kcal, saturated fat<10% of kcal, cholesterol<300 mg, carbohydrate 60% of kcal) can reduce total and low density lipoprotein cholesterol among individuals with SCI who have total initial cholesterol >5.2 mmol/L.

There is level 5 evidence (from one observational study; Kourtrakis et al. 2018) that plasma 25(OH)D level in chronic SCI patients is not associated with clinical factors specific to SCI.

There is level 5 evidence (from one observational study; Jayidan et al. 2017) that dietary intake of lysine may positively relate to levels of FPG, TG, SBP and DBP; intake of cysteine may positively relate to levels of TG and SBP; higher intakes of threonine and leucine may have a negative relationship with TG level; tyrosine, threonine and leucine may be inversely correlated to BP.

There is level 4 evidence (from one pre-post study; Javierre et al. 2005) that daily supplementation with DHA (1.5 g) and EPA (0.75 g) increases plasma DHA and EPA levels but does not alter total cholesterol, very low-, low-, or high-density lipoprotein, triglycerides, or glucose.

There is level 4 evidence (from one pre-post study; Javierre et al. 2006) that DHA and EPA supplementation increases upper body strength and endurance in persons with SCI.

There is level 1b evidence (from one RCT; Amorim et al. 2018) that supplementation with creatine, as well as vitamin D may improve muscle strength parameters in individuals with spinal cord injury.

There is level 3 evidence (from one case-control study; Beal et al. 2018) that chronic SCI patients suffer from hypovitaminosis of vitamin D. Increasing the daily intake of vitamin D improves glucose homeostasis, and may also improve total cholesterol.

There is level 4 evidence (from one pre-post study; Bauman et al. 2005) that vitamin D supplementation raises serum 25(OH) D levels in persons with chronic SCI. However, the dose and duration required to ensure vitamin D sufficiency remains unclear.

There is level 1a evidence (from one RCT; Kendall et al. 2005) that creatine supplementation did not result in improvements in wrist extensor strength or muscle function.

There is level 1a evidence (from one RCT cross-over trial; Jacobs et al. 2002) that creatine supplementation enhances exercise capacity in persons with complete tetraplegia and may promote greater exercise training benefits.

There is level 2 evidence (from one prospective controlled trial; Baliga et al. 1997) that consumption of a standard liquid meal does not change blood pressure, heart rate or noradrenalin levels in individuals with tetraplegia and postural hypotension.

There is level 2 evidence (from one RCT cross-over trial; Nash et al. 2007) that the consumption of a whey protein plus carbohydrate supplement following fatiguing ambulation improves subsequent ambulation by increasing distance, time to fatigue and caloric expenditure in persons with incomplete SCI.

There is level 3 evidence (from one prospective controlled trial; Asknes et al. 1993) that nutrient-induced thermogenesis is not decreased in individuals with tetraplegia with low sympathoadrenal activity; efferent sympathoadrenal stimulation from the brain is not necessary for nutrient-induced thermogenesis.

There is level 3 evidence (from one case control study; Sutters et al. 1992) that sympathetic control of the kidney is not required for renal sodium conservation in response to dietary salt restriction.

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Abbreviations

AISA	ASIA Impairment Scale
ANP	Atrial Natriuretic Peptide
BG	Blood Glucose
BMI	Body Mass Index
BP	Blood Pressure
CVD	Cardiovascular Disease
DBP	Diastolic Blood Pressure
DHA	docosahexaenoic acid
DSC	Dissociated Sympathetic Control
EE	Energy Expenditure
EPA	eicosapentaenoic acid
GRT	Grasp and Release Test
HDL	High Density Lipoprotein Cholesterol
HPLP-II	Healthy Promoting Lifestyle Profile II
HR	Heart Rate
ISC	Intact Sympathetic Control
LDL	Low Density Lipoprotein Cholesterol
OGTT	Oral Glucose Tolerance Test
PADS	Physical Activities with Disability Questionnaire
PD	Parkinson's disease
PO	Power Output
PTH	Parathyroid Hormone
RER	Respiratory Exchange Ratio
SAHP	Self-rated Abilities for Health Practices Scale
SAT	Subcutaneous Adipose Tissue
SBP	Systolic Blood Pressure
SCI	Spinal Cord Injury
SCS	Secondary Conditions Scale
SRD	Sodium Restriction Diet
TC	Total Cholesterol
VAT	Visceral Adipose Tissue
VE	Ventilation Exchange

VF
VO₂
VT

Ventilatory Frequency
Oxygen Uptake
Tidal Volume