Nutrition Issues Following Spinal Cord Injury

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Key Points

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.

Individuals with tetraplegia have higher rates of altered glucose metabolism.

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

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

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.

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

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

Docosahexaenoic acid and eicosapentaenoic acid supplementation increase upper body strength and endurance in persons with SCI.

Individuals with SCI should be screened for vitamin D deficiency and, if needed, 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.
Table of Contents

Abbreviations .............................................................................................................................................. i

1.0 Introduction ........................................................................................................................................... 1

2.0 Energy Imbalance ................................................................................................................................... 1

3.0 Dysphagia .................................................................................................................................................. 3

4.0 Nutrition-Related Complications ........................................................................................................ 3
  4.1 Altered Glucose and Lipid Metabolism ................................................................................................. 3
  4.2 Neurogenic Bowel ............................................................................................................................... 6
  4.3 Neurogenic Bladder ........................................................................................................................... 6
  4.4 Pressure Ulcers ................................................................................................................................... 7
  4.5 Osteoporosis ....................................................................................................................................... 7

5.0 Nutritional Intervention for Energy Imbalance and Wellness ................................................................ 7
  5.1 Diet and Exercise ............................................................................................................................. 7
  5.2 Health Promotion and Education .................................................................................................... 8

6.0 Nutritional Interventions for Dyslipidemia and Cardiovascular Disease Risk ................................... 10
  6.1 Nutrition Counseling ....................................................................................................................... 10
  6.2 Omega-3 Fatty Acid Supplementation .......................................................................................... 11

7.0 Nutritional Interventions for Vitamin Deficiencies and Supplementation ...................................... 13
  7.1 Vitamin D ......................................................................................................................................... 13
  7.2 Vitamin B₁₂ ..................................................................................................................................... 16
  7.3 Creatine ........................................................................................................................................... 16

8.0 Cardiovascular and Hormonal Responses to Food Ingestion ................................................................ 18

9.0 Effects of Nutrient Intake on Ambulation Performance .................................................................... 19

10.0 Post-Meal Resting Energy Expenditure ............................................................................................ 20

11.0 Cardiovascular, Endocrine and Renal Responses to Dietary Sodium Restriction .......................... 22

12.0 Summary ............................................................................................................................................ 24

References .................................................................................................................................................... 26
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>AISA</td>
<td>ASIA Impairment Scale</td>
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<tr>
<td>ANP</td>
<td>Atrial Natriuretic Peptide</td>
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<td>BG</td>
<td>Blood Glucose</td>
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<td>BMI</td>
<td>Body Mass Index</td>
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<td>BP</td>
<td>Blood Pressure</td>
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<td>CVD</td>
<td>Cardiovascular Disease</td>
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<td>DBP</td>
<td>Diastolic Blood Pressure</td>
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<td>DHA</td>
<td>docosahexaenoic acid</td>
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<td>DSC</td>
<td>Dissociated Sympathetic Control</td>
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<td>EE</td>
<td>Energy Expenditure</td>
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<td>EPA</td>
<td>eicosapentaenoic acid</td>
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<td>GRT</td>
<td>Grasp and Release Test</td>
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<td>HDL</td>
<td>High Density Lipoprotein Cholesterol</td>
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<td>HPLP-II</td>
<td>Healthy Promoting Lifestyle Profile II</td>
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<td>HR</td>
<td>Heart Rate</td>
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<td>ISC</td>
<td>Intact Sympathetic Control</td>
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<td>LDL</td>
<td>Low Density Lipoprotein Cholesterol</td>
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<td>OGTT</td>
<td>Oral Glucose Tolerance Test</td>
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<td>PADS</td>
<td>Physical Activities with Disability Questionnaire</td>
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<td>PD</td>
<td>Parkinson’s disease</td>
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<td>PO</td>
<td>Power Output</td>
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<td>PTH</td>
<td>Parathyroid Hormone</td>
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<td>RER</td>
<td>Respiratory Exchange Ratio</td>
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<td>SAHP</td>
<td>Self-rated Abilities for Health Practices Scale</td>
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<td>SAT</td>
<td>Subcutaneous Adipose Tissue</td>
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<td>SBP</td>
<td>Systolic Blood Pressure</td>
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<td>SCI</td>
<td>Spinal Cord Injury</td>
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<tr>
<td>SCS</td>
<td>Secondary Conditions Scale</td>
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<td>SRD</td>
<td>Sodium Restriction Diet</td>
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<td>TC</td>
<td>Total Cholesterol</td>
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<tr>
<td>VAT</td>
<td>Visceral Adipose Tissue</td>
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<tr>
<td>VE</td>
<td>Ventilation Exchange</td>
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<tr>
<td>VF</td>
<td>Ventilatory Frequency</td>
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<tr>
<td>VO₂</td>
<td>Oxygen Uptake</td>
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<tr>
<td>VT</td>
<td>Tidal Volume</td>
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</table>
Nutrition Issues Following Spinal Cord Injury

1.0 Introduction

Given that traumatic spinal cord injuries (SCI) tend to 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 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.

2.0 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

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
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<tbody>
<tr>
<td>Pellicane et al. 2013</td>
<td>USA</td>
<td>Observational</td>
<td>N=78</td>
<td>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.</td>
<td>Treatment: Rehabilitation inpatients were assessed by a Registered Dietitian for dietary intake once weekly.</td>
<td>1. Total calorie intake was significantly higher in individuals with SCI compared to stroke (p&lt;0.003) and PD (p&lt;0.45). 2. Calorie intake per body weight (cal/kg) was significantly higher in individuals with SCI compared to stroke (p&lt;0.025). 3. There were no significant differences in total protein intake between varying etiologies. 4. Age (p&lt;0.001), gender (p=0.023).</td>
</tr>
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</table>
were significant predictors of calorie and protein intake; admission weight also predicted calorie intake (p=0.025).

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.
Treatment: Face-to-face interviews examining habitual daily food intake patterns.
Outcome Measures: Macronutrient intake, simple carbohydrate intake, total calorie intake.

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 acids than those with complete injuries (p=0.03).
6. Age, education and gender significantly predicted calorie intake; time since injury, education, and gender were significant predictors for carbohydrate intake.
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.

Wong et al. 2012
UK
Observational
N=150

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: AIS A=50.4%, B=7.2%, C=20.1%, D=22.3%.
Treatment: Assessment of nutritional risk on admission to SCI centers.
Outcome Measures: Malnutrition Universal Screening Tool, BMI

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.
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).
3. Nutritional risk showed no significant difference with increased age (p=0.913).
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.
5. 'At-risk' patients were found to be receiving more prescribed medications.

Discussion

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 nutritional 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. Thus, there are a significant number of individuals at risk of developing further nutrition-related complications post SCI.

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). 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 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.

There is level 5 evidence (from one observational study; Wong et al. 2012) that individuals with SCI are at a significant risk for malnutrition.

| 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. |

### 3.0 Dysphagia

This section is currently under review; please check back soon.

### 4.0 Nutrition-Related Complications

#### 4.1 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).

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, which are the principal cause of death among persons with SCI (Arrowood et al. 1987; Javierre et al. 2005; Yekutiel 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; Defranzo 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 2 Altered Glucose and Lipid Metabolism**

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<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Total Sample Size</th>
<th>Methods</th>
<th>Outcome</th>
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<tr>
<td>Bennegard &amp; Karlsson 2008 Sweden Prospective Controlled Trial N=19</td>
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<td>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. Treatment: Blood flow and overnight fasting glucose.</td>
<td>1. SCI individuals were found to have significantly higher glucose uptake than those in the control group (p&lt;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&lt;0.05); no differences were observed for those with SCI. 5. Control subjects had a higher lactate production in arms than legs (p&lt;0.05), while SCI subjects did not.</td>
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<tr>
<td>Bauman et al. 1999 USA Pre-Post N=201</td>
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<td>Population: Mean age=39 yr; Gender: males=169, females=32; Mean duration of injury=13 yr; Mean BMI=25; Level of injury: tetraplegia=81, paraplegia=120; Severity of injury: complete=140, incomplete=61. Treatment: Oral glucose tolerance test (OGTT).</td>
<td>1. 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. 2. 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&lt;0.05). 3. Individuals with complete tetraplegia also had an increased frequency of diabetes mellitus compared to others. 4. Individuals with tetraplegia had a significantly higher rate of hyperinsulinemia than individuals with paraplegia (p&lt;0.05). 5. A significant relationship was found between serum uric acid and BMI (p&lt;0.0001), peak serum glucose</td>
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<tr>
<td>Author Year</td>
<td>Country</td>
<td>PEDro Score</td>
<td>Research Design</td>
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<td>Bauman &amp; Spungen 1994</td>
<td>USA</td>
<td>5</td>
<td>Cohort</td>
<td>N=150</td>
<td>Populations: 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. Treatment: Oral glucose tolerance test (OGTT). Outcome Measures: Mean plasma glucose and insulin values, serum lipid levels.</td>
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<tr>
<td>Ketover et al. 1996</td>
<td>USA</td>
<td>5</td>
<td>Prospective Controlled Trial</td>
<td>N=58</td>
<td>Populations: SCI (n=29): Mean age=51 yr; Gender: males=28, females=1; Obesity (BMI&gt;27)=11; Non-SCI controls (n=29): Mean age=36 yr; Gender: males=13, females=16; Obesity (BMI&gt;27)=14. Treatment: All individuals were administered a 20 g fat liquid meal. Outcome Measures: Gallbladder emptying.</td>
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Note: AISA=ASIA Impairment Scale; BMI=Body Mass Index

**Discussion**

Three studies have examined altered glucose metabolism in individuals after a SCI (Bauman et al. 1999; Bauman & Spungen 1994; Bennegard & Karlsson 2008). Significantly higher serum glucose concentration and diabetes mellitus was seen in persons with complete tetraplegia (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).

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 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.

| Individuals with tetraplegia have higher rates of altered glucose metabolism. |
| Impaired gallbladder emptying is seen in diabetic and obese SCI individuals. |

4.2 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.

4.3 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.
4.4 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.

4.5 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.

5.0 Nutritional Intervention for Energy Imbalance and Wellness

5.1 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 rate were reported to be 43.4% in those with physical disabilities and 34.6% for those without physical disabilities (Oh et al. 2012). 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. In addition, there are limited educational resources available on nutrition issues and weight control for this high-risk group (Chen et al. 2006).

Table 3 Diet and Exercise Program for Overweight/Obesity

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chen et al. 2006</td>
<td>USA</td>
<td>Pre-post</td>
<td>N_initial 17; N_Final 16</td>
<td>Population: Gender: males=9, females=7; Injury etiology: SCI=15, spina bifida=1; Severity of injury: AISA A–D; Family history of overweight/obesity: yes=11, no=5.</td>
<td>Treatment: Patients attended classes on nutrition, exercise and weight control/reduction for 12 wk (90 min/wk) and exercised for 6 wk (30-min).</td>
<td>1. During the intervention 14 subjects lost weight (mean age=4.2 kg). 2. Decreases were noted in BMI (p&lt;0.050), waist circumference (p&lt;0.001), neck circumference (p&lt;0.020), and skinfold thickness (p&lt;0.001). 3. HDL decreased significantly (p&lt;0.030). 4. At follow-up, 6 continued to lose weight, 4 stabilized, and 3 gained.</td>
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</table>

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

Discussion

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.

**Conclusion**

*There is level 4 evidence (from one pre-post study; Chen et al. 2006) that an intervention program combining diet and exercise is effective for reducing weight among overweight persons with SCI.*

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

### 5.2 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).

#### Table 4 Nutritional Education Intervention and Long-Term Wellness

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zemper et al. 2003</td>
<td>USA</td>
<td>PEDro=4</td>
<td>RCT</td>
<td>NInitial=76; NFinal=43</td>
<td>Population: Age range=22-80 yr; Gender: males=30, females=13; Level of injury: complete, incomplete; Time since injury=1-49 yr. Treatment: Subjects attended a series of six 4-hr workshop sessions over 3 mo. <strong>Outcome measures:</strong> 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).</td>
<td>1. Treatment groups scores on the SAHP improved following treatment (p&lt;0.05) as well as on the HLPD-II (total score) &amp; the HLPD-II health related subscale score (p&lt;0.001). 2. Treatment group scores improved post-treatment on the HLPD-II (nutrition subscale) (p&lt;0.05). 3. Mean scores for the treatment groups improved significantly for the HLPD-II stress management subscale (p=0.001). Treatment group’s stress scores also improved, indicating less stress (p&lt;0.05). 4. HLPD-II physical activity scores improved post treatment for the treatment group only (p=0.001). No significant differences were noted for</td>
</tr>
</tbody>
</table>
**Discussion**

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.

Conclusion

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.

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.

6.0 Nutritional Interventions for Dyslipidemia and Cardiovascular Disease Risk

6.1 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 5 Effect of Nutrition Counseling on Dyslipidemia and Cardiovascular Disease Risk

<table>
<thead>
<tr>
<th>Author Year Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Szlachcic et al. 2001 USA Prospective Controlled Trial N=222</td>
<td></td>
<td></td>
<td></td>
<td>Population: Gender: males=198, females=24; Level of injury: complete, incomplete; Time since injury=&gt;2 yr. Treatment: Subjects who had a cholesterol level &gt;5.2mmol/L (n=86) were referred to either a dietary consultation where they were advised to modify daily intakes as follows: total fat&lt;30% of kcal, saturated fat&lt;10% of kcal, cholesterol&lt;300 mg, carbohydrate=60% of kcal, or no treatment. Outcome Measures: Total cholesterol (TC), high-density lipoprotein cholesterol</td>
<td>1. TC decreased in the dietary counseling group from 6.1 mmol/L to 5.8 mmol/L (p&lt;0.001) and slightly increased in the control group from 4.2 mmol/L to 4.3 mmol/L (p=0.006). 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. 3. Neither group experienced significant changes in HDL or triglyceride values.</td>
</tr>
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</table>
Discussion

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 (>5.2mmol/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 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 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.

Conclusion

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.

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

6.2 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 6 Omega-3 Fatty Acid Supplementation on Lipid Profile and Physical Performance
### Author Year Country PEDro Score Research Design Sample Size

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Javierre et al.</td>
<td>2005</td>
<td>Spain</td>
<td>5</td>
<td>Pre-post</td>
<td>N=19</td>
</tr>
<tr>
<td>Javierre et al.</td>
<td>2006</td>
<td>Spain</td>
<td>5</td>
<td>Pre-post</td>
<td>N=21</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Population:</strong> Severity of injury: AISA A–D; Time since injury=&gt;12 yr.</td>
<td>1. Plasma EPA and DHA increased significantly (p&lt;0.05) in response to the intake of the supplement at 3 months and 6 months (p&lt;0.05).</td>
</tr>
<tr>
<td><strong>Treatment:</strong> 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.</td>
<td>2. No differences in all types of cholesterol, triglycerides, or glucose were observed.</td>
</tr>
<tr>
<td><strong>Outcome Measures:</strong> Plasma DHA, EPA, total, very low density, low density, and high density lipoprotein, triglycerides, and overnight fasting glucose.</td>
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</table>

**Note:** AISA=ASIA Impairment Scale

### 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.

### 7.0 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.

#### 7.1 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.

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 7 Vitamin D Supplementation Post SCI**

<table>
<thead>
<tr>
<th>Author Year Country PEDro Score Research Design Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hummel et al. 2012 Canada Case Series N=62</td>
<td><strong>Population:</strong> Mean age=49±12 yr; Gender: males=51 male, females=14; Time since injury: &gt;2 yr; Cause of injury= traumatic=62, non-traumatic=0. <strong>Treatment:</strong> Blood draw for serum sample. <strong>Outcome Measures:</strong> Serum 25(OH)D and PTH.</td>
<td>1. 39% of the cohort had suboptimal serum 25(OH)D levels. 2. 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). 3. 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).</td>
</tr>
</tbody>
</table>

<p>| Bauman et al. 2005 USA Pre-post N_{Study 1}=10; N_{Study 2}=40 | <strong>Population:</strong> Study 1: Mean age=53 yr; Study 2: Mean age=43 yr. <strong>Treatment:</strong> Study 1: All patients were given 50 μg (2000 IU) vitamin D₃ 2x/wk and 1500 mg elemental calcium daily for 2 wk. Study 2: 10 μg (400 IU) vitamin D₃, a multivitamin with an additional 10 μg (400 IU) vitamin D₃, and 500 mg | <strong>Study 1:</strong> 1. After 2 weeks, serum 25(OH)D increased (p&lt;0.005) but 8 of 10 subjects still had values below the normal range (&lt;16 ng/mL). 2. Serum PTH decreased from 35 to 18 pg/mL (p&lt;0.05), serum calcium was not significantly different, and urinary... |</p>
<table>
<thead>
<tr>
<th>Author Year Country PEDro Score Research Design Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>elemental calcium daily for 12 mo. <strong>Outcome Measures:</strong> Changes in serum 25(OH)D, calcium and parathyroid hormone (PTH), and urinary calcium.</td>
<td>calcium increased from 103 to 239 mg/d (p=0.010). <strong>Study 2:</strong> 1. At baseline, 33 subjects were vitamin D deficient (&lt;16 ng/mL) compared to 9 after 12 months. 2. After 6 and 12 months, serum 25(OH)D increased (p&lt;0.0001). 3. Serum PTH decreased (p&lt;0.005), but serum calcium did not change.</td>
</tr>
</tbody>
</table>

**Discussion**

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 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.*

Individuals with SCI should be screened for vitamin D deficiency and, if needed, replacement therapy should be initiated.
7.2 Vitamin B\textsubscript{12}

The prevalence of vitamin B\textsubscript{12} 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 B\textsubscript{12} deficiency usually responds to supplementation.

Petchkrua et al. (2002) conducted a retrospective chart review of patients with SCI who had received serum vitamin B\textsubscript{12} testing over a 10 year period. The most common symptoms among subjects identified as having deficient, subnormal or low normal vitamin B\textsubscript{12} 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 B\textsubscript{12} 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 B\textsubscript{12} supplementation. It is recommended that clinicians conduct early screening and treatment of vitamin B\textsubscript{12} 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 B\textsubscript{12} deficiency in persons with SCI. Biochemical vitamin B\textsubscript{12} 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 B\textsubscript{12} deficiency was 40-59 years; among subjects older than 59 years, 9% had B\textsubscript{12} 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 B\textsubscript{12} deficiency. Additional investigations into the predisposing risk factors for vitamin B\textsubscript{12} deficiency in persons with SCI are warranted.

Clinicians should conduct early screening for and treatment of vitamin B\textsubscript{12} deficiency.

7.3 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 8 Creatine Administration Post SCI
### Table

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kendall et al. 2005</td>
<td>USA</td>
<td>PEDro=9</td>
<td>Cross-over RCT</td>
<td>N&lt;sub&gt;initial&lt;/sub&gt;=9; N&lt;sub&gt;final&lt;/sub&gt;=8</td>
<td>Population: Level of injury: C5/C6; Severity of injury: AIS A-C; Time since injury=16.5 yr. Treatment: 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. Outcome Measures: Grasp and Release Test (GRT) and Functional Independence Measure.</td>
<td>1. There were no significant between-group differences for GRT or FIM scores.</td>
</tr>
<tr>
<td>Jacobs et al. 2002</td>
<td>USA</td>
<td>PEDro=8</td>
<td>Cross-over RCT</td>
<td>N=16</td>
<td>Population: Mean age=35.3 yr; Gender: males=16 females=0; Level of injury: tetraplegia; Mean weight=71.4 kg. Treatment: 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. Outcome Measures: Power output, time to fatigue, heart rate (HR), oxygen uptake (VO₂), minute ventilation (V&lt;sub&gt;E&lt;/sub&gt;), ventilatory frequency (V&lt;sub&gt;F&lt;/sub&gt;), respiratory exchange ratio (RER), tidal volume (V&lt;sub&gt;T&lt;/sub&gt;).</td>
<td>1. No adverse effects were reported. 2. There was no change in HR, RER and V&lt;sub&gt;E&lt;/sub&gt;, although there were significant difference in VO₂, VCO₂, V&lt;sub&gt;F&lt;/sub&gt; and V&lt;sub&gt;T&lt;/sub&gt; between trials (p&lt;0.001). 3. VO₂ increased by 18.6% with creatine treatment versus placebo. 4. After creatine consumption, VO₂, VCO₂ and V&lt;sub&gt;T&lt;/sub&gt; reached their highest peak.</td>
</tr>
</tbody>
</table>

*Note: AIS=ASIA Impairment Scale*

### 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.

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baliga et al. 1997</td>
<td>USA</td>
<td>Prospective Controlled Trial</td>
<td>N=11</td>
<td></td>
</tr>
</tbody>
</table>

**Methods**

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.

Treatment: Individuals with tetraplegia received a milk-based liquid meal including Compan and glucose supplements (66 g carbohydrates, 22 g fat, 18 g protein, 550 Kcal, 300 mL). Individuals with paraplegia served as controls.

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.

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 noradrenaline levels in individuals with tetraplegia and postural hypotension.

9.0 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 10 Nutrient Intake on Ambulation Performance

<table>
<thead>
<tr>
<th>Author Year</th>
<th>Country</th>
<th>PEDro Score</th>
<th>Research Design</th>
<th>Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nash et al. 2007</td>
<td>USA</td>
<td>PEDro=5</td>
<td>Cross-over RCT</td>
<td>N=3</td>
<td>Population: Mean age=38.7 yr; Gender: males=2, females=1; Level of injury: paraplegia=1, tetraplegia=2; Severity of injury: AIS A C=2, D=1; Time since injury=11.3 yr. Treatment: 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</td>
<td>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.</td>
</tr>
</tbody>
</table>
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.

10.0 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 11 Post-Meal Resting Energy Expenditure

<table>
<thead>
<tr>
<th>Author Year; Country; PEDro Score; Research Design; Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>alternate treatment.</td>
<td><strong>Outcome Measures:</strong> Ambulation time, distance walked, and energy expenditure.</td>
</tr>
<tr>
<td>Author Year; Country</td>
<td>PEDro Score</td>
<td>Research Design</td>
</tr>
<tr>
<td>----------------------</td>
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<tr>
<td>Asknes et al. 1993</td>
<td>4</td>
<td>Sweden</td>
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<tr>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

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.

### 11.0 Cardiovascular, Endocrine and Renal Responses to Dietary Sodium Restriction

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

#### Table 12 Responses to Dietary Sodium Restriction

<table>
<thead>
<tr>
<th>Author Year Country PEDro Score Research Design Sample Size</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sutters et al. 1992 USA Case Control N=15</td>
<td>Population: Mean age=28 yr; Gender: males=15, females=0; Level of injury: paraplegia=6, tetraplegia=9; Time since injury=2 mo-1 yr. Treatment: 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 280 mmol/day for days 1-3 but was reduced to 20 mmol/day on day 4 and 5. 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)).</td>
<td>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.</td>
</tr>
</tbody>
</table>

#### 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.
12.0 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 morbidity and mortality in individuals with SCI as evidenced by blood values, metabolic and lifestyle factors, few studies have addressed the benefits of risk reduction interventions aimed at modifiable factors and 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 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.

There is level 5 evidence (from one observational study; Wong et al. 2012) that individuals with SCI are at a significant risk for malnutrition.

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 4 evidence (from one pre-post study; Chen et al. 2006) that an intervention program combining diet and exercise is effective for reducing weight among overweight persons with SCI.

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 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 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 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.
References


