DETERMINANTS OF NUTRITIONAL REQUIREMENTS OF SPINAL CORD INJURED ATHLETES

Ingrid Perols

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ERRATUM

Throughout this thesis, and particularly in the abstract, results and discussion sections, where reference is made to the dietary intake data collected in this study it should be acknowledged that due to the lack of validity and reliability of the method of dietary data collection used (self-reported intakes in the form of a food diary) these are reported intakes and cannot be considered as actual intakes.

In the “Abstract”, line 28 the unit for energy intake should be stated as kilojoules and not as incorrectly stated in kilocalories. The sentence should read: “The mean reported energy intake was 8678 ± 2411 kJ per day...“.
Abstract

Disability sport is attracting more participants and is becoming increasingly competitive. Athletes with spinal cord injury (SCI) resulting in quadriplegia face a number of physiological limitations such as reduction in lean body mass, increased fat mass and altered physiological responses to exercise, as a direct consequence of their injury. Long-term health consequences of spinal cord injury such as cardiovascular disease, type 2 diabetes and osteoporosis occur earlier than in the able-bodied population.

The aims of this study was to investigate the nutritional status and some of the physiological characteristics likely to impact on the nutritional requirements of SCI athletes.

Sixteen wheelchair rugby players currently playing for a regional team in the 1999 NZ National Wheelchair Rugby Tournament were recruited. Subjects completed a brief demographic questionnaire to provide information about their injury, and 7-day food diary for assessment of dietary intake during the competition season. Body composition and bone mineral density were assessed by dual-energy x-ray absorptiometry (DEXA) and an arm-cranking test was performed in a laboratory situation to assess heart rate response and oxygen uptake during exercise. Finally, monitoring of heart rate in a game situation was performed on three subjects.

Body composition data showed that subjects had a body mass index (BMI) (26.4 g/cm2) similar to that of the general New Zealand population and to paraplegic subjects in New Zealand. However, lean body mass was low (60.1% of total body mass) and body fat was relatively high (34.9% of body
mass) in comparison to both the general population and to other athletes. Mean whole body bone mineral density for the group was within the normal range however, five subjects were classified as having osteopenia and of those, two were classified as having osteoporosis. The mean energy intake was 8678 ± 2411 kcal per day, significantly below that of NZ males in the same age groups [1]. The contribution from carbohydrate, protein and fat to total energy intake was virtually identical to that of the general NZ population [1]. Many of the subjects did not meet the Recommended Dietary Intake (RDI) for a number of micronutrients.

Heart rate (HR) response and oxygen uptake (VO2) was similar to that found in other studies [2]. Interestingly, the relationship between HR and VO2 showed a linear increase during the graded exercise test resulting in a coefficient of determination above 0.923 for all subjects.

Heart rate profiles were recorded for three subjects in a game situation and energy expenditure was calculated from VO2 data from the laboratory exercise test. The three subjects had a calculated energy expenditure ranging from 3.7-7.2 kcal/minute of exercise.

It was concluded that BMI may not be an accurate method to assess obesity in quadriplegic subjects and body composition assessment by DEXA provides a more accurate method for determining changes in body composition and bone mineral density over time. It was further concluded that many subjects had low energy intakes resulting in suboptimal intakes of macronutrients. Many subjects would benefit from dietary advice with a view to improve general health and exercise performance. Heart rate monitoring for the
purpose of estimating energy expenditure should be investigated further in this population as should other factors likely to impact on both general health and exercise performance.
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1 Introduction

Competitive disability sport (sport for the disabled) is steadily attracting more participants. In New Zealand, disability sport is organised through Paralympics NZ and athletes can compete in regional, national and international events such as World Championships and the Paralympic games. In the Sydney 2000 Paralympic games the following sports will be included: archery, athletics, basketball, boccia (a game similar to petanque), cycling, equestrian, fencing, futsal (indoor soccer), goalball, judo, lawn bowling, power lifting, shooting, swimming, table tennis, tennis, wheelchair rugby, volleyball and yachting [3].

Within the group “disabled athletes” a wide range of disabilities are represented [4]. Of these athletes many are permanently wheelchair bound, as a result of injuries such as spinal cord injuries, a range of muscular disorders or other disease states, while others may have visual impairments, cerebral palsy, dwarfism, or be single or multiple amputees [5]. In some sports codes athletes are classified according to reduction in ability and compete against others with a similarly reduced ability [4]. Other codes such as wheelchair rugby are reserved almost exclusively for athletes with a specific disability (quadriplegics or others with a similar level of impaired functional ability) [6]. Disabled athletes are now more frequently competing in able-bodied events [4]. Particularly “fun runs” and marathons regularly offer wheelchair competitors to race over the same course together with the runners and walkers.

Depending on the type of disability or injury the athlete has, a number of physiological changes resulting from the disability may affect the nutritional needs of the athlete [5]. It is therefore important to be familiar with the physiological changes that may be present in disabled athletes.
Spinal cord injured (SCI) athletes are among those most likely to experience physiological changes directly resulting from their injury. The most notable physiological changes in this group are [3, 7]:

- impaired thermoregulation and an inability to sweat
- lack of innervation of muscle groups below the point where the spinal cord injury has occurred
- changes in body composition (both acute and gradual)
- changes in energy expenditure
- reduced resting and maximum heart rates
- reduced ability to maintain plasma volume

The potential differences in nutritional requirements compared to requirements estimated for able-bodied individuals can be seen as direct consequences of the altered physiological responses to acute exercise and to the normal process of ageing. The most likely differences are in estimations of daily energy requirements, recommendations for carbohydrate and protein intakes based on body weight and recommendations for fluid and electrolyte needs.

The metabolic alterations resulting from SCI are known to affect the long term health outcomes. It is well documented that, in particular, spinal cord injured individuals are more likely to experience early development of cardiovascular disease and type 2 diabetes [8]. The metabolic response to exercise has not been thoroughly investigated in spinal cord injured athletes [5] however, it could be hypothesised that the alterations in carbohydrate and lipid metabolism could have some effect on substrate utilisation during exercise. The inability to use the legs and trunk results in bone density values below those seen in an able-bodied population [9].

Health problems that commonly occur in the short term include an increased incidence of urinary tract infections [9], pressure sores [10], gastrointestinal
disturbances such as constipation [11] and over-use injuries in the upper body (shoulders, wrists) [12].

Both physical activity and specific nutrition recommendations are thought to prevent the development of both short and long term health problems in this population [13].

Nutrition guidelines for able-bodied athletes have been developed and tested based on research performed on competitive athletes in a wide range of sports [14]. There are currently few guidelines specifically developed from data collected about disabled athletes and spinal cord injured athletes in particular [5]. Recommendations based on questionnaires with American wheelchair athletes concluded that “nutritional guidance was necessary for most athletes” [15].

Recommendations for food and fluid intake during training and competition should be based on an assessment of the individual athlete’s disability and physiological changes resulting from this. As for able-bodied athletes, individual recommendations may need to be altered due to factors such as changes to the intensity and duration of training or after a re-assessment of energy expenditure or body composition has been made. Information about SCI athletes’ typical body composition, energy expenditure during rest and exercise, and dietary habits are valuable both for establishing dietary recommendations for particular sports and when designing nutrition plans for individual athletes.
2 Literature review

2.1 Wheelchair rugby

2.1.1 The history of wheelchair rugby in New Zealand
Wheelchair rugby has developed from a game called “murder ball” (invented in Canada) which was first introduced to New Zealand in 1989. The first national wheelchair rugby tournament was held in New Zealand in 1991 as part of the Parafed Nationals. In 1993 the first New Zealand wheelchair rugby nationals were played in Wellington. The seventh New Zealand nationals were played in Palmerston North in September 1999 where approximately 80 players participated.

In 1995 the first New Zealand wheelchair rugby team competed in the world championships in Switzerland where they finished in third place. This performance was repeated in the Atlanta Paralympic games in 1996 (Martin, P. personal communication). In the second world championships in Canada in 1998 the team finished in second place and would now be considered one of the favourites for the Paralympic games to be held in Sydney, Australia in 2000.

2.1.2 The rules of the game
The game is played on a basketball sized court, using a volleyball, with each team allowed four players on court. In order to make the teams compete on equal terms players are classified from 0.5 for the players with least functional ability to 3.5 for the players with most functional ability. (Classification can only be determined by an approved classifier, usually a doctor, physiotherapist or occupational therapist) [16] [4]. The combined classification for the players a team has on court at any one time cannot exceed eight. Players with a high classification are referred to as “high-pointers” and are responsible for most of the sprints, while the “low-pointers” are doing most of the blocking of the opposition’s players [6]. A game is played in four 8-minute quarters and substitutions are allowed during the game. Goals are scored when a player crosses the opposition’s goal line at the end of the court with...
the ball in their possession. A goal is usually worth one point however, in some tournaments a goal by a 0.5 classified player is worth double points. Special purpose built wheelchairs are used in wheelchair rugby. The design of the chair features a "tip-bar" at the front of the chair and cambered wheels. Players are not allowed contact with each other of any part of their body but full wheelchair contact is legal and wheelchair tackles are part of the game strategy [6].

Many participants in wheelchair rugby are ex-able-bodied rugby players, some of whom sustained their injury while playing rugby. The majority have a quadriplegic SCI, although athletes with other disabilities resulting in a comparable functional classification are allowed to play.

2.2 Spinal cord injury
A complete spinal cord injury (SCI) causes permanent motor paralysis and is accompanied by a loss of sensation below the level of the injury. As well as the inability to move and loss of feeling a spinal cord injured person experiences many other both physiological, psychological, social and often economical consequences of the injury, all of which contribute to a dramatic change in lifestyle for the individuals and their families [12]. A spinal cord injury would be considered by many as one of the most traumatic experiences imaginable and the dramatic life changes psychologically impossible to cope with. Many individuals with a SCI will confirm this and tell about their frustration, depression, loss of self-esteem and associated problems [12]. Despite this many people involved in disability sport also tell of the opportunities and positive changes to their lives they have experienced after the injury [17]. They discover opportunities to participate in competitive sport, to travel, to have an active social life and to build and increase self-esteem that they may not have had before the injury.

About 80% of all spinal cord injuries occur in males. In North America the majority (48%) of spinal cord injuries are caused by motor vehicle accidents,
followed by falls (21%) and sport and acts of violence in shared third place (14.5% each) [12]. More than 61% of new injuries occur in the 16-30 year age group with another 19.4% in the 31-45 year age group [12]. Statistical information for New Zealand could not be obtained, however it is likely to be similar to the data for North America.

2.2.1 The spinal cord

The spine consists of 33 individual and fused vertebrae and can be divided into four segments: the cervical (C1-8), the thoracic (T1-12), the lumbar (L1-5) and sacral (S1-5) spine (see Figure 2.1). The spinal cord runs through the canal in the middle of the vertebral column. Paraplegia is usually caused by a spinal cord lesion below the first thoracic vertebra and quadriplegia is usually the result of a lesion above this point [3].

Two terms are used to describe spinal cord injuries. Quadriplegia or tetraplegia which are both used to describe a complete or incomplete paralysis of all four limbs and the trunk. Paraplegia is used to describe a complete or incomplete paralysis of the lower limbs and the trunk [3]. The term “complete syndrome” refers to a loss of all sensory and motor nerve transmissions below the level of injury and is caused either by a complete rupture of the spinal cord, a total breakage of nerve fibres due to stretching of the cord, or interruption of the blood supply causing ischaemia of the cord [18]. An “incomplete” injury spares some motor and sensory function and can be caused by damage either to the central portion of the cord or to one side of the cord only.
Figure 2.1. The spinal cord and spinal nerves. [18]
Some functions return gradually after a spinal cord injury and it may take up to a year after an injury before it is fully known what functions have been lost [18]. As a result of the many possible combinations of injuries to the spinal cord individuals can have widely differing loss of function and seemingly similar injuries can result in different functional ability [3].

The major exercise related problems after SCI are [7]:
- reduced ability to voluntarily use the large muscle groups necessary to perform aerobic exercise
- inability to stimulate the cardiovascular system to support higher rates of aerobic metabolism
- decreased ability to exercise in environmental extremes due to impaired thermoregulation

The major long-term health related problems after SCI are [8] [9]:
- increased risk of obesity due to reduced energy expenditure both as a result of decreased muscle mass and relative inactivity
- increased risk of cardiovascular disease and type 2 diabetes, probably related to the increased incidence of obesity
- premature osteoporosis due to inactivity and relative inability to perform weight bearing exercise

2.3 Life and health expectancy after spinal cord injury
Before the second world war the majority of people who sustained spinal cord injuries were unlikely to survive the first 48 hours after the injury [12]. Those who did were likely to die of other complications or permanently suffer from infections, bedsores and other complications of the injury. With advancements in medical care and the relatively large numbers of war veterans returning with spinal cord injuries after the war the long-term management of the injuries and the quality of care for spinal cord injured patients were largely improved [19].
Intense research in the areas of acute treatment and rehabilitation of spinal cord injuries has contributed to a much greater body of knowledge than in the area of long-term care and health outcomes. Many SCI can now be managed in local hospital facilities [12]. In New Zealand there are two specialised spinal units (Burwood, Christchurch and Otara, Auckland) for treatment and rehabilitation of SCI patients.

The remaining life expectancy and quality of life is considered to be reduced after traumatic spinal cord injury. A study completed in 1983 in Canada reported that subjects injured at age 30 had an overall expected age at death of 64.5 years [20] cited in [19] significantly earlier than that of the general population. In a recent study by McColl et al [19] the life expectancy and long-term health outcomes for 286 spinal cord injured Canadian subjects (complete and incomplete para- and quadriplegic patients) were investigated. The study which included individuals injured between 1945-1990 and who experienced their injury between 25-34 years of age, estimated a median survival of 38 years post-injury with 43% of subjects surviving at least 40 years [19]. The researchers concluded that the life expectancy appeared to be increased by at least 5 years or by a 8-10% margin of increased survival compared to previous studies conducted 5-10 years earlier. It is difficult to know whether this represents a real increase in life expectancy for this population or is a reflection of the increased life expectancy in the general population [19]. There is a possibility that improvements in the medical care of SCI patients could have affected the differing results of these studies.

The most significant factors that affected life expectancy in the majority of studies on subjects with SCI were completeness and level of injury and age at injury [19]. Individuals sustaining their injury at an early age had much improved long-term health outcomes. Subjects with paraplegia had an estimated survival time approximately 9 years longer than those with quadriplegia. A similar pattern to that seen in the general population was found, with males having a shorter life expectancy than females [19].
A number of studies have attempted to estimate the SCI individuals' expectations of health compared to that of people in the general population. Consistent with general population data studies of Canadian and British SCI subjects indicated that there is a period of 5-7 years near the end of life when poor health can be expected [19]. Although subjects reported the same level of life satisfaction during a 3 year follow-up period, a number of health indicators had deteriorated. Musculo-skeletal complaints, pressure sores, cardiac and circulatory problems, bowel and bladder problems and fatigue were the most commonly reported symptoms of deteriorating health in the SCI study group [19].

2.4 Obesity, cardiovascular disease and type 2 diabetes mellitus
A sedentary lifestyle is clearly linked to the development of overweight and obesity in the general population. The combination of excess body weight, in the form of body fat, and low levels of physical activity is considered a strong risk factor in the development of cardiovascular disease and type 2 diabetes. Cellular insulin resistance and hyperinsulinaemia, a lipid profile with reduced HDL-concentration and increased triglyceride concentration, and abdominal obesity are thought to result in a physiological state referred to as the metabolic syndrome [21].

In spinal cord injury, where a significant amount of muscle mass is paralysed and therefore inactive, body composition changes such as a reduction in total muscle mass and an increase in fat mass often develop [22]. This clearly puts people with SCI at an increased risk of obesity, cardiovascular disease and type 2 diabetes. Currently cardiovascular disease is the leading cause of death in SCI individuals [23]. Furthermore, obesity is a factor that contributes to the level of independence that can be achieved by SCI patients. An observational study of two obese quadriplegic subjects (C7) showed that their functional ability was far below that expected for a C7 quadriplegic injury and this severely interfered with the rehabilitation process [24]. Both subjects had
to be discharged to a high level care institution rather than to their own homes.

In a recent cohort study by Bauman and Spungen [8] indicators of type 2 diabetes, insulin sensitivity, body composition assessment by DEXA, lipid profiles and maximal oxygen uptake were determined in 50 subjects with paraplegia, 50 with quadriplegia and 50 able-bodied controls. The results from this study show that 56% of the SCI study group had an abnormal glucose tolerance compared to 20% in the control group. Of the same SCI subjects 22% had developed type 2 diabetes compared to only 6% of the healthy controls (according to the World Health Organisation criteria). Subject with type 2 diabetes were more likely to be in the older age groups however, those with SCI developed glucose intolerance and diabetes at younger ages than the controls [8]. Both controls and SCI subjects with established type 2 diabetes had higher fasting plasma glucose levels than subjects with normal glucose tolerance. Interestingly, in the SCI group both individuals with normal and impaired glucose tolerance (but who did not fit the criteria for type 2 diabetes), had lower fasting plasma glucose values than the respective control subgroups. The authors suggested that this could indicate a reduced hepatic glucose output in the SCI group.

In the able-bodied controls % body fat showed the strongest correlation with insulin sensitivity while in the SCI study group maximal oxygen uptake rather than level of adiposity, was most strongly correlated to insulin sensitivity [8]. The SCI subjects were also found to have decreased HDL cholesterol levels (0.95 mmol/l) compared to the able-bodied control group (1.20 mmol/l).

In a descriptive study conducted in conjunction with a routine medical follow-up of rehabilitated SCI patients a similar incidence of glucose intolerance (23% of subjects) and fasting hyperglycemia (19%) was found [25] (this study did not distinguish between impaired glucose tolerance and established type 2 diabetes). Although only 2% of subjects had abnormal total serum
cholesterol levels 58% had low HDL levels. The authors recommended that as HDL cholesterol is considered cardioprotective even when total cholesterol is within normal limits a complete lipid profile should always be done in spinal cord injured patients as the majority of individuals who are at cardiovascular risk could otherwise be undetected [25].

The authors of both studies reviewed above conclude that carbohydrate and lipid abnormalities are common in SCI and although these condition are common in the general population they appear to occur prematurely in SCI patients.

Hooker and Wells showed that total cholesterol was reduced by 8% and HDL cholesterol increased by 20% in men with SCI after eight weeks of moderate intensity wheelchair exercise [26] cited in [13].

A study by Brenes et al investigated the HDL concentrations in sedentary compared to physically active SCI patients. The subjects in physically active group were Olympic calibre wheelchair athletes involved in athletics, swimming, road racing and weight training. The sedentary SCI subjects in this study had a HDL cholesterol (0.86 mmol/l*) similar to that found in the studies by Tharion and Baumann reviewed above, while the physically active subjects had HDL levels of 1.01 mmol/l compared to the able-bodied controls (1.18 mmol/l) [27]. The authors of this study strongly recommend that the “dose” of physical activity required to provide an increase in HDL cholesterol and a reduction in cardiovascular disease risk needs to be established by further prospective studies [27].

Level of physical activity appears to be strongly related to HDL serum concentration with endurance athletes shown to have the highest values [28] cited in [27].

*For ease of interpretation serum HDL-cholesterol results were converted 1 mmol/l = 40.0 mg/dl.
In the sedentary population it has so far not been established whether the level of mobility has any effect on serum HDL levels. Baumann et al further investigated the relationship between lipid profile and level of neurological deficit (categorised as complete and incomplete paraplegia and quadriplegia respectively), and therefore mobility. Subjects with complete tetraplegia were shown to have the lowest serum HDL concentrations and the highest total / HDL ratio. The authors hypothesised that as well as level of inactivity, hyperinsulinaemia, which has been shown to be more prevalent in the quadriplegic population, may have contributed the lower HDL levels [29].

Weiss et al have formulated guidelines for prevention of cardiovascular disease and type 2 diabetes in SCI individuals and recommend that a combination of nutritional intervention and an aerobic exercise programme (modified from prevention guidelines for the general population) is likely to reduce the incidence of these conditions in the SCI population [13]. The effectiveness of dietary and exercise intervention programmes on reducing the incidence of cardiovascular disease and diabetes in this population has not been thoroughly evaluated.

Considering that the majority of the SCI population (>80%) is male [12], and that male gender in itself is a risk factor for cardiovascular disease, it would seem appropriate to recommend strategies for risk reduction in this population.

2.5 Osteoporosis

Osteoporosis and the accompanying increased risk for fractures is a feature of the normal ageing process [30]. As average life expectancy increases both in the general as well as the spinal cord injured population so does the risk for osteoporosis.

Prevention of osteoporosis should start early by taking measures to ensure that the genetic potential for peak bone mineral density (BMD) is achieved. A
number of environmental factors such as calcium intake, physical activity and sex steroid status influence bone accretion [30].

Premature development of osteoporosis is well documented in the spinal cord injured population. Two factors in particular; inability of the lower extremities to perform weight bearing activities and rapid demineralisation of bone in the acute phase of SCI, both contribute to the increased risk of osteoporosis in this population. Fractures of lower extremities and also collapse of the spine below the level of injury have been reported among World War II survivors of spinal cord injuries. When SCI occurs in children the long term risk of premature osteoporosis is thought to increase as they are unlikely to reach optimal genetic peak bone mass. The majority of SCI (66%) occur in people under the age of 30, the years when the majority of calcium is laid down in the bones [31].

There is currently considerable debate about the optimum calcium intake for prevention of osteoporosis in different age groups of the general population [31].

Dietary recommendations for calcium intake for SCI patients provided by rehabilitation centres are inconsistent [23, 32]. In the acute phase of SCI patients experience hypercalciuria as a result of paralysis and immobilisation. SCI patients are at increased risk of renal stone formation for a number of reasons such as urinary infections, alkaline urine, reduced urinary output, catheterisation and inactivity. It is therefore seen as prudent to reduce calcium intake as a measure to prevent renal stone formation. The practice of recommending calcium and vitamin D restriction appears to be based on findings of high urinary and serum levels of calcium in the acute phase of SCI. However, many authors now agree that due to the high risk of premature osteoporosis this should not be recommended as a life long dietary strategy [9, 23]. It has also been documented that increasing dietary calcium from 400 mg/day to 1,160 mg/day did not increase either urinary or serum calcium
concentrations in the acute and early rehabilitation phase of SCI [33] cited in [9].

Studies of bone mineral density (BMD) in the acute phase of SCI (14-114 days post-injury) showed losses of approximately 17% and 13% respectively in the distal femur and the proximal tibia but no significant decline in the lumbar spine or the hip [34]. The same group of subjects were followed up 16 months later when they were found to have significantly lower BMD in the pelvis and legs compared to a control group. At the same follow up 50% of subjects had BMD at the proximal tibia below the fracture threshold while in a group of subjects with chronic SCI (>10 year post-injury) 90% were below the fracture threshold.

A similar study where age matched able-bodied and SCI subjects were compared showed no difference in BMD levels within the first year of injury [35]. When different age groups were compared it was found that femoral bone density appeared to decrease most dramatically in the 20-39 year SCI group. Femoral bone density in paraplegic and quadriplegic subjects over the age of 40 decreased steadily over time however, bone loss in this region only became significant 10 years post-injury [35].

Long term studies of twins (one paraplegic / one able-bodied) have shown progressive losses of total BMD over decades after the injury [36]. In this study bone density in the upper extremities was unchanged while the spine showed a small but significant gain in density.

In a study by Demirel et al [37] a positive correlation between time since injury and the degree of BMD in the paralysed areas was found. The study, which compared paraplegic and quadriplegic subjects, also found that BMD scores for the upper extremities were significantly higher in the paraplegic group but BMD scores of the upper extremities were similar for both groups [37].
Regional differences in bone mineral density between male subjects with paraplegia and quadriplegia were also found by Tsuzuku et al [38]. This group found that BMD in the femoral neck, Ward's triangle (area within the femoral neck and inside the trochanter forming a triangle), head, pelvis and lower extremities were similar while significant differences were found in the lumbar spine, trochanter region and upper extremities [38].

A longitudinal study by Wilmet et al confirmed findings by other researchers of rapidly decreasing BMD in areas rich in trabecular bone with a slower but more steady decrease in BMD in cortical bone. The long term result was an approximate 50% loss of bone mineral [39].

In a New Zealand study of paraplegic patients similar losses of BMD were found as those reported in the studies above [40]. With a better understanding of the exact mechanisms of bone metabolism in the acute phase of SCI it is anticipated that in the future the dramatic initial bone loss may be prevented by use of specific pharmaceutical treatment [9].

Uebelhart et al have investigated changes in a number of biological markers of bone metabolism [41] however it is beyond the scope of this review to discuss these results.

Spinal cord injured individuals are not only exposed to the risk factors for osteoporosis common in the general population (lack of weight bearing exercise, low calcium intake, smoking, high sodium intake) but may also experience other risks more specific to their injury [9]. Low intake of both calcium and vitamin D due to previous dietary recommendations for prevention of renal stones is thought to be common in the SCI population [9]. However, other factors affecting calcium status such as a high sodium intake due to frequent intake of convenience foods and more frequent use of steroidal drugs may also contribute [9]. Early studies of calcium loss in SCI
showed that 3-5 days of sodium restriction (10 mmol/day) reduced daily calcium loss from 16 mEq/day to 3 mEq/day [9].

Vitamin D status may be affected by factors such as less time spent outside, use of anticonvulsants (accelerating vitamin D metabolism by inducing hepatic enzymes) and pressure sores [42]. In fact, the sub-group of SCI subjects with pressure sores had the lowest levels of 25-hydroxyvitamin D, however the mechanism for this is not well understood. The authors suggested that this may have been as a result of reduced cutaneous synthesis resulting from less time spent outside, possibly due to reduced mobility while suffering from pressure sores [42]. Subjects with SCI have been shown to have decreased levels of 25-hydroxyvitamin D levels, elevated serum 1,25-dihydroxyvitamin D levels and elevated levels of parathyroid hormone (PTH) compared to controls [43]. In a group of 100 SCI subjects investigated by Baumann, vitamin D deficiency was found in approximately a third [43]. Seventeen of those subjects also had serum calcium levels below the lower limit for normal [43]. The author concluded that many SCI individuals are prone to secondary hyperparathyroidism due to calcium and vitamin D deficiency. The author also suggests that patients identified with a deficiency state should receive aggressive treatment with calcium and vitamin D supplements [9].

Apart from dietary intervention and potentially new drug therapies to manipulate calcium and vitamin D metabolism in order to prevent bone loss, adapted physical activity may have a role in improving bone health in SCI. However, interventions using electrical-stimulation-induced leg cycling (ES-LC) have failed to improve BMD in the lower extremities after periods of six months [44] [45] to nine months [46]. A subset of SCI subjects in the study by Bloomfield [46] who were able to produce a higher power output (>18kW) than the other subjects did however show a significant increase in BMD in the distal femur after nine months of ES-LC.
A case study of two female paraplegic athletes showed increased bone density values in the forearm (21% and 16% higher in the ulnar and radial respectively) compared to able-bodied controls [47] indicating that physical activity may potentially improve BMD regionally.

In children with SCI the use of standing frames, tilt tables enabling weight bearing are thought to slow the loss of bone [48].

No conclusive evidence appears to exist that other types of exercise assists in the prevention of bone loss or increase in BMD. Wilmet et al found that in the rehabilitation phase their subjects had a significant increase in upper body muscle mass but did not significantly increase their BMD. They suggested that effect of exercise on bone mass may take longer than the intense rehabilitation period [39].

An interesting observation from the study by Wilmet et al was the dramatic increase in upper extremity lean muscle mass during the rehabilitation period, half of which was lost during the six months after discharge before reaching a plateau 12 months after the injury. This indicates a decrease in level of physical activity after discharge and it is possible that this could also impacts on post-injury bone status.

In theory, physical activity, incorporating some weight-bearing exercise, has the potential to prevent development of osteoporosis. However, there is very little information about the effectiveness of different types of physical activity in the SCI population. One study comparing physically active and non-active SCI subjects (paraplegic and quadriplegic) found bone density at all sites (whole body, total hip, radii) to be higher in the physically active subjects with paraplegia compared to subjects in the other groups. In quadriplegic subjects no significant difference in bone density was found at any site between active and non-active subjects [49]. No intervention studies investigating the effect of different types of exercise have been performed to this author’s knowledge.
2.6 Physiological Consequences of Spinal Cord Injury

The spinal cord can be seen as an extension of the brain and together with the brain it forms the central nervous system. The cranial and spinal nerves branch out over the entire body from the brain and spinal cord and together form the systemic or peripheral nervous system and is responsible for communication between the brain and peripheral body parts [18]. An injury to the spinal cord causes loss of innervation of the part of the body that is served by the spinal nerves from the level of the injury and down, as the means of communication has been cut off. The damage is irreversible due to the fact that neurons (or nerve cells) can not multiply or regenerate once they have been destroyed [18].

In quadriplegia (injury above T1) leg, abdominal and chest muscles as well as bowel, bladder and sexual function is affected. Depending on the exact level of injury hand, triceps, wrist, deltoid and biceps function may also be affected [18].

The autonomic nervous system (the sympathetic and the parasympathetic systems) consists of nerves outside the brain and spinal cord and is a function of the hypothalamus [18]. This system provides subconscious control of functions such as heart rate, blood pressure, temperature control, appetite, fluid balance, bladder emptying, gastrointestinal function, carbohydrate and fat metabolism, sleep and sexual function [18]. As a result of a quadriplegic SCI the sympathetic nervous system is “decentralised” ie it loses its ability to become activated during exercise. The peripheral nervous system is separated from control by the cardiovascular centres of the brain due to the interruption of efferent sympathetic outflow [50]. Organs such as the myocardium, the smooth muscle of the arteries and veins, and the adrenal medullae, that normally effect a sympathetic response can no longer be activated by exercise. This results in a severe impairment of physiological responses such as redistribution of blood to working muscles, increased stroke volume and cardiac output, blood pressure regulation and
thermoregulation that normally occur during exercise in able-bodied individuals [2].

Both the sympathetic and parasympathetic systems provide innervation of the heart [18]. During rest the heart rate is regulated by the parasympathetic system via the vagus nerve. As part of the body’s stress response the sympathetic system increases heart rate and respiration, strengthens cardiac contractions, often referred to as the “fight or flight” mechanism [18]. The adrenal gland is stimulated to release adrenalin and blood is redirected away from less important areas such as the gastrointestinal tract to cardiopulmonary circulation [51].

The parasympathetic system is more important for everyday non-stress functions and the outflow of parasympathetic nerves is from the cranio-sacral area. The gastrointestinal and bladder functions are regulated by the nerves from the sacral spine so quadriplegic injuries are likely to affect these functions [18].

Certain functions such as the voluntary muscles, skin, sweat glands adrenal gland and the spleen are regulated by the sympathetic system alone.

Some of the symptoms of impaired autonomic function seen in SCI can be controlled by adrenergic (sympathetic system) or cholinergic (parasympathetic system) drugs [18].

2.6.1 Cardiovascular System

Many of the complications seen in SCI are associated with changes in the functioning of the cardiovascular system. Some of these complications such as disturbed blood pressure regulation, heart rate and heart dysrhythmias, affect the injured person in the acute phase of SCI and gradually decline within 6 weeks post-injury [48]. This review will focus on the chronic changes
to the cardiovascular system and the effect these are likely to have on normal every-day activities, exercise and the long-term health of the SCI person.

The heart is innervated by both the sympathetic and the parasympathetic systems while the blood vessels only derive innervation from the sympathetic system. During rest the parasympathetic system has the major influence on the heart. The vasomotor centre together with the sympathetic vasoconstrictor system are responsible for vasomotor tone (vasoconstriction and vasodilation). Neural stimulation of blood vessels causes the rapid changes in vasoconstriction and changes the rate of blood flow to different tissues [51]. In able-bodied individuals this redistribution of blood flow from internal organs to muscles and skin serves to assist efficient muscle function and to dissipate heat during exercise without greatly affecting cardiac output or blood pressure [52]).

As a result of SCI above the T6 level neural control of vasomotor tone is impaired [52]. Due to the lack of sympathetic regulation of the heart, the contractile force of the heart is impaired and the heart rate is lowered. The ability to redistribute blood is reduced due to poor vasoconstriction below the level of injury, particularly in the abdominal and pelvic area [2].

2.6.2 Exercise responses
People with quadriplegia are at most risk of cardiovascular disturbances such as hypotension, hypertension and bradycardia while exercising [7]. The pathologic consequences of the injury are extensive and the sympathetic nervous system is often completely separated from brain control.

Due to impairment of the skeletal muscle venous pump, excessive venous pooling of blood in the abdomen and the legs may cause exercise induced hypotension. Hypotension during exercise may also be caused by vasodilation in active muscles which is not compensated by sympathetic
vasoconstriction [2]. Many quadriplegic athletes wear support stockings and an abdominal binder to prevent this during exercise [3].

On the other hand, extreme hypertension can also be caused by exercise. This usually occurs as a result of autonomic dysreflexia (or hyperreflexia) a syndrome caused by excessive activation of autonomic reflexes. The extreme blood pressure rise (>200/100 mmHg) is dangerous and needs to be recognised and treated immediately [48]. Some quadriplegic athletes induce hyperreflexia as the rapid blood pressure rise is believed to cause a "power surge" and therefore improve performance particularly in a sprint or speed phase of a competition, [53]. Athletes can voluntarily induce hyperreflexia by inflicting pain or by overfilling their catheter causing an increased bladder pressure (personal communication with wheelchair athletes).

In both paraplegia and quadriplegia the amount of total body muscle mass able to be used for exercise is reduced and this results in reduced peak power output, oxygen consumption (VO2) and cardiac output. In quadriplegia these values have been estimated to be about a quarter to a sixth of those achieved by able-bodied individuals [7]. Peak power output can be expected to range from 0-50 watts in athletes with quadriplegia and incremental exercise increases of 5-20 watts may be appropriate depending on the mode of exercise and the subject's completeness of injury [7]. In quadriplegia upper body muscle innervation, and therefore function, varies so that in higher level injuries arm muscle function is further reduced, often by lack of triceps function [2], author's interviews with athletes) causing further variation in power output between individuals with relatively similar injuries.

Maximum heart rates do not usually exceed 130 beats per minute in complete quadriplegia. A study by Hopman of heat stress during exercise also showed that average maximum heart rates in SCI above T6 reached about 110 beats per minute [54].
Although improvements of 10-20% in peak power output and VO2max can result from upper body exercise this does not equate to an increase in central cardiovascular fitness [2]. Therefore most of the physiological benefits of upper body exercise are peripheral (located within the muscle) and include increased number of mitochondria, myoglobin and oxygen storage, oxidative enzyme activity, muscle fibre size and capillary density [2].

Exercise position may alter the cardiovascular responses to exercise. Hypotension during exercise, also called "circulatory hypokinesis" has been found to be more severe during sitting arm exercise than during arm exercise in the supine position [2], [55]. Significantly higher values for heart rate, VO2, cardiac output and stroke volume can be achieved during exercise in the supine position [2] thereby increasing the likelihood of training effects on the cardiovascular system. However, the peripheral adaptations may still be valuable for performance both of everyday activities and sport specific tasks.

During incremental exercise such as in a graded exercise test subjects with quadriplegia usually experience peripheral fatigue before cardiovascular pumping capacity reaches its maximum ability [2]. During aerobic exercise, when using only the smaller muscle groups of the upper body the demand for oxygen is not likely to exceed oxygen delivery capacity [2] either in quadriplegic or able-bodied individuals. Values measured for VO2 are therefore likely to be relatively low, even in persons who engage in regular physical activity and therefore are trained to perform the specific exercise. The measured VO2 is not necessarily a reflection of the individual's "true" maximum oxygen uptake capacity, only an indication of the oxygen required to perform that particular exercise.

Due to the difficulties in determining "true cardiovascular fitness" in this population current research in this area is focusing on exercise modifications such as combinations of horizontal posture, electrically stimulated leg cycle ergometry (ES-LCE, using skin surface electrodes) and the use of
sympathomimetic agents in order to stress the heart. Particularly the use of ES-LCE combined with arm-cranking exercise has the potential to significantly increase energy expenditure [2]. Krauss et al have shown that sedentary subjects with paraplegia and low-level tetraplegia can improve cardiovascular fitness as measured by peak VO2 after 12 weeks of either arm ergometry, ES-LCE, or a combination of both, with the combination protocol achieving the highest increase in fitness level [56].

Heart rates, VO2 values, power outputs and rate of perceived exertion (RPE) during exercise in sitting and supine position have shown large individual variability [55]. This is thought to be strongly related to level of injury with individuals with low-level injuries (C7 and below) having a stronger correlation between heart rate and VO2 (correlation coefficients 0.92-0.99) [55]. Individuals with high-level injuries (above C7) showed an average correlation between HR and VO2 of 0.80 [55].

The purpose of McLean's study was to investigate different recommended methods of exercise prescription commonly used for the able-bodied population (HR, % of VO2, PO and RPE). Subjects with low-level injuries obtained an average heart rate of 114 BPM and those with high-level injuries 115 BPM and VO2 values of 0.748 L/min and 0.473 L/min respectively in the sitting position [55]. Two of the subjects in the high-level injury study group did not show an increase in HR as VO2 increased, probably due to a drop in blood pressure throughout the exercise test. It was also concluded that significantly higher power outputs could be achieved in the supine position by both groups. This was mainly contributed to increased stability of the trunk and scapula while lying down. Based on these findings it was concluded that none of the suggested methods for exercise prescription were applicable to this population.

Comparisons between sedentary and physically active (PA) SCI subjects (paraplegics) have shown lower resting heart rates, higher peak heart rates
and higher peak workloads in the PA subjects in a graded arm-cranking exercise test [57]. The resting HR was lower and the peak HR and peak PO were also higher than in able-bodied sedentary controls [57] indicating a training effect in the PA group.

2.6.3 Thermoregulation

The documented drop in cardiac output due to decreased stroke volume and a relatively small rise in heart rate has implications for thermoregulation as the reduced cardiac output may not be sufficient to maintain blood pressure as well as transport blood to the skin surface for cooling [58]. This puts individuals with SCI above T6 at increased risk of heat stress, especially when exercising in the heat. In subjects with injuries below the T6 level cardiac output does not decrease during exercise and is similar to the "cardiovascular drift" described in able-bodied subjects exercising in the heat [59] cited in [54].

Hopman has also shown that skin temperature in the legs of SCI subjects (above T6) is about 4°C lower than in the torso (35°C) at rest, pointing to a reduced skin blood flow to the legs. At the end of a 45 minute exercise period skin temperature in the torso had risen to 38°C while the leg temperature had only increased to 34°C in the above T6 group while those with lower-level injuries and able-bodied subjects had similar skin temperatures on the torso and the legs [52]. This finding has been partly explained by the lower sweat production in subjects with SCI above T6. While able-bodied subjects lost on average 718 g of sweat those with SCI between T6-T2 only lost 172 g during the same exercise test [52].

However, in a study of quadriplegic subjects performing arm-cranking at 75% of peak VO2 no significant changes in heart rate, rectal or skin temperatures were found [60] (cited in [2]. The authors suggested that the amount of muscle mass involved in exercise was not large enough to cause an increase
in body temperature [60]. These conflicting findings may just indicate a difference between different environmental conditions.

A combination of perceived reduced sweat rate and the inconvenience of exercising with a catheter and urine bag (interviews with SCI athletes) may result in inadequate fluid intake both before and during exercise in this population. However, fluid intake during exercise is still essential as it improves body cooling [52] and prevents dehydration which may contribute to urinary infections frequently seen in spinal cord injured individuals [5]). Therefore other strategies such as acclimatisation and pre-cooling to reduce the likelihood of heat stress in this population may be useful.

2.7 Body composition

Estimation of body composition in able-bodied subjects can be performed by a number of techniques such as anthropometry, bioelectrical impedance analysis (BIA), hydrodensitometry, dual energy X-ray absorptiometry (DEXA) and total body potassium (TBK). Many of these methods have not been validated in different ethnic groups. The bioelectrical impedance method has been validated for both males and females from China, Ethiopia, Italy and the Netherlands [61] however, data for the typical New Zealand ethnic mix is not available.

While anthropometry and BIA are easy and relatively inexpensive to perform their accuracy and reliability have been questioned [62]. Hydrodensitometry has been replaced by DEXA as the method of choice when a high level of accuracy is required for determining the amount of fat mass and lean body mass [63]. The main reason for this is that DEXA relies less on assumptions regarding biological consistency of water, protein and mineral content of the body [63] but also its ease of use compared to hydrodensitometry.
Changes in body composition are known to occur in a number of disease states with changes in fat mass, lean mass, fluid space, and skeletal and hard-tissue seen [64].

Used for assessment of fat mass and lean body mass, DEXA has been shown to provide highly reproducible results when repeated measures are taken and when different scan speeds (20 minutes vs 10 minutes) are used [65]. However, concerns have been raised that DEXA may not be as accurate in the very young, the very old or during illness, mainly due to assumptions of normal hydration being used [66]. The variability in methods of calibration, data acquisition and data analysis used by different manufacturers contribute to the uncertainty regarding validity of results [63].

When comparing data for % body fat derived from DEXA with that from hydrodensitometry a large discrepancy can be seen. An in-depth comparison of the steps for information processing in DEXA and hydrodensitometry respectively shows that the estimated amounts of different tissues are very similar until the last step in the calculations [67]. The hydrodensitometry method uses information about the density of body tissues to estimate % body fat with either the Siri or Brozek equation. The DEXA method simply uses the measured weight of body tissues to compute the proportional contribution of FM to total body mass. The hydrodensitometry or under-water weighing equivalents for the Siri and Brozek equations are usually reported for comparison in the printed analysis from a DEXA scan.

Both anthropometry and hydrodensitometry have practical limitations for SCI individuals (unable to stand, difficulty moving onto weighing chair and balancing in chair). Furthermore these methods are probably inaccurate as equations used for the estimation are derived from able-bodied populations [68]. DEXA on the other hand is relatively easy to use for this population and have fewer variables likely to result in estimation errors as described above.
Although there are still some debate about the usefulness of the DEXA technique for the measurement of body composition the use of DEXA for assessment of bone mineral density (BMD) has been shown to have a very low precision error (<1%) [69, 70] cited in [66] and is currently considered to be the preferable method to use.

Because of the established increased risk of cardiovascular disease, type 2 diabetes and osteoporosis [13] in persons with SCI there is a need to accurately quantify and monitor changes in body composition in this population. The DEXA technique has been confirmed as a useful and well tolerated method for the assessment of regional as well as whole body composition in spinal cord injured subjects [40].

Changes in body composition have been documented in several studies using different assessment methods [71] [39]. Nuhlicek et al using dilution of 3H2O and Na2 35SO4 found that fat mass increased and lean body mass decreased the higher the level of injury (low paraplegia, high paraplegia, low quadriplegia, high quadriplegia) [71].

In a 1 year longitudinal study DEXA was used to assess changes in body composition of paraplegic subjects with the initial scan performed within 1-8 weeks post-injury [39]. Leg lean mass decreased by 15% after a year with the majority lost within 15 weeks of injury. On the other hand arm lean mass stabilised at 15% above the initial level. In this study changes in fat mass were too disparate to allow any comparison during the 1 year study period, however there was a clear trend for fat content to increase in the lower limbs and decrease in the upper limbs of some patients [39].

Jones et al found that compared to age and weight matched controls, SCI (paraplegics) subjects had 16% less lean body mass, 12% less total BMC and 47% more fat mass as measured by DEXA [40]. As expected, the greatest differences were found in the lower limbs with the study group
retaining only 70% of the BMC in the leg compared to the controls [40]. An interesting observation from this study was that despite having BMI values below 27.8 kg/m2 (BMI>30 considered as obese) the SCI subjects had an average body fat percentage above 25%, a factor that may increase their risk of developing both type 2 diabetes and cardiovascular disease [40]. The use of BMI as an indicator of overweight and obesity may not be appropriate in the SCI population.

In addition to the decreased amount of LBM, a transformation of skeletal muscle fibre type below the level of injury, has been documented after SCI. The composition of the remaining muscle mass gradually changes from the normally occurring combination of type 1 (slow twitch) and type 2 (fast twitch) muscle fibres to predominantly type 2 fibres [72]. This is thought to explain why SCI subjects who perform electrically stimulated leg cycle ergometry (ES-LCE) experience muscle fatigue earlier than expected.

Spungen et al compared the quality of fat free mass (muscle, viscera and bone) between controls and subjects with paraplegia by using dual photon x-ray absorptiometry (DPA) and total body potassium (TBK) by whole body counting [73]. The quality of fat free mass is indicated by the ratio of TBK/FFM-DPA which was found to be reduced in paraplegia compared to the control group [73]. Resting energy expenditure (REE) was also measured and it was concluded that in paraplegic subjects TBK and the ratio of TBK/FFM-DPA are better indicators of metabolic activity of the lean tissue while DPA is more appropriate measure of lean tissue mass. The lower ratio of potassium (K) per kg lean body tissue was thought to be due to the lower K concentration in visceral tissue compared to skeletal muscle indicating a relatively higher contribution from visceral tissue to total lean tissue [73].

Few studies have described body composition in SCI athletes. A comparison of body composition of two female wheelchair athletes to able-bodied controls revealed significantly increased bone densities in the forearm (21% in the
ulnar and 16% in the radial) [47] (cited in Bulbulian, 1987). The same study also found large discrepancies in % body fat when comparing data from hydrodensitometry and whole body potassium counting with several anthropometric prediction equations [47].

A more recent study by Bulbulian et al compared anthropometric data (5 diameters, 11 circumferences and 7 skinfolds) and hydrodensitometry data for 22 paraplegic men with two able-bodied control groups (ectomorphs and mesomorphs) [68]. The validity of 12 different body composition prediction equations were tested on the study group. As a result of their training all paraplegic subjects, had developed a mesomorph body type and it was therefore hypothesised that body composition data for the paraplegic group would be better predicted by equations developed for a mesomorph somatotype [68]. Most of the prediction equations overestimated body density and underestimated body fat and the authors concluded that anthropometric data based on any of the currently used prediction equations is likely to be unreliable for this population group [68].

Lastly, in a study of participants in the annual Oita wheelchair Marathon race over the past ten years anthropometric features of a total of 2,677 wheelchair competitors were described [74]. The aim of the study was to compare the anthropometric measurements (and other measurements such as lung vital capacity) to race performance. Apart from height, weight, chest girth and upper arm girth, skinfolds were measured at three sites (triceps, subscapula and supraillium) and body fat (%) was estimated based on the sum of the three sites.

Data from six of the ten years were reported and body fat (%) showed a non-significant variation between the fastest and the slowest performers. In fact, values were almost identical in the two groups (between 15.6 % - 19.7 %) except for one year when the slower competitors had a significantly higher level of body fat (23.7% vs 18.7%) [74]. Both chest girth and upper arm girth
were significantly larger in the faster competitors compared to the slower group. Although this study is an interesting attempt at correlating body composition with exercise performance many other factors could have affected the results. Factors such as type of injury, outside temperature (likely to affect SCI athletes differently to those with other injuries) and the lack of evidence that skinfold measurements can be used for the estimation of % body fat in a wheelchair bound population may all have confounded the results. Despite these short-comings it is still reasonable to assume that body composition will affect exercise performance in wheelchair athletes.

No further studies have been located describing the relationship between body composition and exercise performance in spinal cord injured athletes.

2.8 Energy expenditure during rest and exercise

Many factors have an impact on the nutritional requirements of athletes. Due to physiological changes as a result of SCI the nutritional requirements of these athletes are likely to be different to those of able-bodied athletes [5].

Energy and macronutrient requirements for able-bodied athletes can be estimated based on body weight and activity level [14] [75]. Daily energy requirements include energy expenditure during activities of daily life and any physical activity or exercise performed [76]. Resting energy expenditure is partially determined by the proportion of lean body mass while energy expenditure during exercise is a result of factors such as body mass, intensity of exercise and the amount of muscle mass being utilised during a particular activity [51].

Daily energy expenditure is known to be lower in spinal cord injured individuals as less muscle mass is innervated and levels of physical activity are generally lower [77]. Level of injury appears to have a much greater effect on denervation atrophy and therefore energy expenditure than time since injury [78].
In able-bodied populations a number of equations (Harris-Benedict, Quebbeman, Spanier and Shizgal) based on weight, body surface area, height, age or a combination of these measurements, are used to estimate energy requirements [79]. When compared to estimates of energy expenditure by indirect calorimetry (using the Weir equation) [80] methods such as those above, based mainly on body size, have been found to significantly overestimate energy expenditure in SCI subjects [79]. Cox et al measured an average energy requirement of 22.7 kcal/kg/day in the rehabilitation phase (>3 weeks post-injury) for individuals with quadriplegia. This represents 45-90% of the calculated energy requirements when recognised formulae are used [79]. Energy requirements for other trauma patients are typically 30-40 kcal/kg/day [81].

Similar findings have been reported in patients during the acute phase of SCI [82]. An interesting finding of the study by Rodriguez was that despite aggressive nutrition support (high energy/high protein/adequate nutrient intake) based on the recommended model for estimating energy requirements (RMR + activity and trauma factors) and preventing loss of lean tissue mass, nitrogen balance in the acute phase (1-4 weeks) remained negative [82]. This has led to recommendations being given to SCI patients to maintain a high protein intake after the acute phase of injury in order to prevent loss of muscle mass (author's experience). There is no evidence that this is effective for preservation of muscle mass either in the acute phase or in the long-term [82].

One of the obvious risks with consistently overestimating energy requirements is weight gain in the rehabilitation phase after injury. This was shown to be the case in quadriplegic subjects on an uncontrolled diet who gained an average of 1.84 kg per week during the first six months after the acute injury phase [79].
The only study identified by this researcher where the relationship between body composition and energy expenditure has been investigated was by Sedlock [22]. LBM was calculated based on hydrodensitometry and RMR measured after a 12-hour fast in a group of 4 paraplegic men. The correlation between LBM and RMR was high ($r=0.98$) however the measured RMR values were lower than those predicted from LBM by using the Cunningham equation ($\text{RMR (kcal/day)} = 501.6 + 21.6 \times (\text{kgLBM})$) [22].

Recommendations for estimating energy requirements in SCI athletes are therefore difficult to make. Requirements are likely to be affected by factors such as type of injury, time since injury, amount of innervated muscle mass and type of training performed.

2.8.1 Estimating energy expenditure by the use of heart rate monitoring

By correlating an individual’s heart rate (HR) to a certain oxygen consumption (VO2), 24-hour heart rate monitoring can be used in able-bodied populations to estimate energy expenditure over periods of several days [83, 84] [85]. The relationship between HR and VO2 from resting and during increasing workloads must first be established for each subject in laboratory conditions. The recorded heart rates during the test period can then be translated into oxygen uptake and energy expenditure estimated using the equation for indirect calorimetry (Weir equation = 1 litre of oxygen = 4.825 kcal [80]).

Due to the physiological differences in spinal cord injury described in section 2.6.1 it is uncertain whether this method is appropriate for SCI athletes. Heart rate monitoring to assess training intensity has been suggested as a useful tool for SCI athletes with an injury below T4, but is thought to be of limited use in athletes with an injury above T4 [3]. It is possible that this method is suitable for spinal cord injured athletes with an injury below a certain level and could then be used to better determine daily energy expenditure in this population. To this author’s knowledge no studies have been performed.
where heart rate monitoring has been used to estimate energy expenditure during both rest, activities of daily life (ADL) and exercise in SCI athletes.

2.9 Exercise testing

The two most common methods for laboratory exercise testing of wheelchair athletes are the arm-crank ergometer and the wheelchair ergometer. A number of different designs of arm-crank ergometers exist, some of which are specifically designed for wheelchair users (mounted on a table or wall with height adjustment possible) and others designed for upper-body training for able-bodied persons (secured to the floor and adjusted to adult standing height) (author's investigations). Some models have only a fan-wheel and do therefore not allow for adjustment of resistance as would be required in an exercise testing situation. Models used for exercise testing purposes in the laboratory need to allow for adjustment of resistance so that power output can be measured, be height adjustable to the individual’s sitting height, and have a relatively large fly-wheel for smoother cranking action. Less sophisticated models of arm-crank ergometers are used by many wheelchair athletes as alternatives to wheelchair exercise.

The wheelchair ergometer is more task specific for wheelchair athletes as it allows the athlete to exercise in the same mode as during competition. However, factors such as type of tyres, weight and efficiency of wheelchair, camber of wheels [86] and problems to quantify power outputs of individuals with different body mass [87] can affect the reproducibility and validity of wheelchair ergometer exercise. As a wheelchair ergometer is designed as either a treadmill or as a bicycle trainer where the wheels are fixed at the hubs neither design allows for “free-wheeling” as during normal wheelchair propulsion. This means that the athlete is working against constant resistance and has to push as in the acceleration phase of exercise at each “push”. As the resistance increases this becomes difficult and many athletes are unable to maintain the required pace. In all papers on exercise performance in
wheelchair athletes reviewed by this researcher arm-crank ergometers were used.

Most authors agree that conditions can more easily be controlled and results are highly reproducible when using the arm-crank ergometer for investigations of physiological responses during exercise in paraplegic and quadriplegic athletes [54].

In a number of studies investigating the response to different modes of exercise, HR and VO2 have been measured. In an investigation of valid measures of exercise intensity, VO2, HR and rating of perceived exertion (RPE) during assisted leg exercise were recorded [88]. This study of paraplegic subjects found a strong linear relationship between HR and VO2 during this type of exercise. It has also been demonstrated that persons with paraplegia below T4 performing arm ergometry exercise show a linear relationship between HR and VO2 [89] (cited in [88]).

Raymond et al also investigated physiological responses to arm-ergometry alone and to combined arm-ergometry / electrical-stimulation-induced leg cycling (ES-LC) in SCI subjects [90]. The exercise protocol consisted of 5 minutes on the combined protocol followed by 5 minutes of arm-cranking alone. VO2 was shown to be lower during arm-cranking alone while HR, on the other hand, was higher during the first minute of arm-cranking after transition from the combined protocol. [90]. This was thought to be due to a drop in venous return when the leg cycling was discontinued [90]. During wheelchair sport this response is less likely as only the upper body muscles are being used (unless the athlete used ES-LC for warm-up prior to arm exercise alone).

Heart rate monitoring has also been used to assess physical strain experienced by relatively sedentary wheelchair users [91]. The effort required to perform normal activities of daily life (ADL) in SCI subjects (paraplegic and
quadriplegic) was assessed by recording HR over a 24-hour period. Level of physical effort was calculated as % heart rate reserve (%HRR) (HHR is expressed as peak HR - HR at rest) and reported as time spent at different levels of %HRR [91]. Maximal HR for each individual had previously been determined during an incremental exercise test in laboratory conditions [92]. The authors concluded that the effort required for ADL was not great enough to result in any significant improvement of physical activity. It was also clear from this study that the subjects with quadriplegia performed all ADL at a higher %HHR than the other SCI subjects with lower level injuries. In inactive individuals the physical strain of ADL is thought to lead to fatigue particularly later in the day, and these authors suggest that persons with high level injuries such as quadriplegia can benefit from improving their physical capacity thus reducing the fatigue associated with ADL [91].

2.10 Current nutrition recommendations for wheelchair sport
As mentioned above there are currently no nutrition recommendations for SCI athletes and any nutritional advice given would be based on recommendations for able-bodied athletes.

Based on the known physiological changes in spinal cord injured individuals a number of assumptions can be made regarding the likely changes in nutritional requirements. However, these assumed changes in requirements have not, in most cases, been investigated and quantified to an extent where recommendations based on research findings can be made.

In summary the following areas, at least, are likely to be affected by changes resulting from the spinal cord injury:
- impaired thermoregulation and inability to sweat may affect fluid and electrolyte balances
- immobility causes loss of calcium and reduced bone mineral density and premature osteoporosis
• the increased calcium loss may be a contributing factor to an increased risk of kidney stone formation
• reduced innervation of muscle causes a reduction in muscle mass and a reduction in daily energy expenditure caused by the immobility
• changes in body composition between lean body mass (LBM) and fat mass results in changes in energy expenditure
• changes in body composition may also be linked to the increased risk of abdominal obesity, cardiovascular disease and type 2 diabetes
• the smaller size of upper body muscles compared to lower body muscle may affect the ability of the muscle to store muscle glycogen for use during exercise resulting in a need to consume carbohydrate more frequently during exercise
• regular use of laxatives may affect nutrient absorption

The following dietary recommendations for disabled athletes have been found in the literature.

Although total energy requirements are considered to be reduced the need for carbohydrate in the diet of SCI athletes to provide energy for repeated bouts of exercise is still likely to be high [5]. Rice et al have suggested that fat intake should not exceed 20% of total energy intake in people with immobilising physical disabilities such as SCI in order to prevent excess weight gain [93] cited in [5]. Recommendations for calcium intake have been controversial in the past as calcium was thought to contribute to the already increased risk of renal stone formation (see section 2.5). However, most authors now agree that due to the increased risk for fractures and osteoporosis immobilised persons such as the SCI population should follow guidelines for calcium intake for the general population [94].

Recent focus on the possible link between adequate calcium intake and reduced risk of hypertension [95] [96] and therefore cardiovascular disease
may have implications for recommended calcium intake for the male SCI population who are already at high-risk of developing cardiovascular disease.

Dietary recommendations for the prevention of chronic disease have been discussed in the section on long-term health risks (section 2.4).

2.10.1 Dietary intake of spinal cord injured individuals

Information about dietary intake of spinal cord injured individuals appears to be lacking. The majority of the data has been collected during the acute and rehabilitation phases when subjects are hospitalised and investigations generally focus on energy and protein requirements [9, 23, 79, 97].

The quality and quantity of dietary intake post-hospitalisation will likely depend on factors such as ability to prepare meals, level of support from care givers, dietary information at discharge, access to shops and food outlets and motivation to follow healthy eating guidelines (author's observations). Specific nutrition related problems described in rehabilitated patients are hypoalbuminaemia particularly in patients with pressure ulcers, reduced immune function, subnormal levels of vitamin K, chronic gastrointestinal problems and electrolyte disturbances (particularly sodium and calcium) [10] [11, 98, 99].

Little is known about the dietary intake of persons with SCI in general and SCI athletes in particular.

A study of 39 North American wheelchair athletes (25 of whom were SCI athletes) investigated the training practices of athletes who participated in the National Wheelchair Athletic Association’s training camps [15]. Part of the questionnaire completed by the participants surveyed dietary practices however, did not attempt to quantify dietary intake. Subjects estimated the composition of their diet to be 50.5% carbohydrate, 19.0% fat and 30.5% protein. No attempt was made to verify these estimates. Sixty percent of
participants said they were following high carbohydrate diets, 51.4% low fat diets, 46.0% high fibre diets, and 40.3% said they followed a high protein diet ("high" and "low" were not defined). The study also found that 16.2% used protein supplements, 27.0% CHO supplements and 62.2% used vitamin supplements.

2.11 Assessment of Dietary Intake

Dietary intake can be assessed by a number of methods such as 24-hour diet recall, 3 or 7-day food diaries, food frequency questionnaires, weighed food intake and double portion methods [100]. All of these methods are considered to have limitations and the accuracy of reported food intakes is often questioned [101].

Energy intake as estimated by self-report does not correspond well with actual energy expenditure measured by the doubly-labelled water technique during a study period. Several studies have confirmed that certain population groups such as the overweight, women with disordered eating behaviour and female athletes in certain sports tend to under-report food intake while other groups may over-report dietary intake [101]. Under-reporting of food intake can also occur due to failure to remember all foods consumed during the day, not accurately recording serving sizes or not accurately describing the consumed food items [100].

The use of a 7-day food diary where subjects are instructed to weigh all food and fluid consumed is often used in smaller research studies while food frequency questionnaires are commonly used in larger population based surveys [100]. In order to obtain useful data it is important that the research subjects understand the purpose of the dietary data collection and are given detailed instructions on how to record their intake [100].

In research studies factors such as within subject variation and between subject variation in intake of different nutrients should be taken into account.
when determining the number of days required for dietary data collection in order to most accurately estimate energy, macro- and micronutrient intakes in the study population [102]. The accuracy of estimated dietary intake may be affected by seasonal changes in food intake due to variation in food supply [1].

The number of days of food records required in order to estimate the true average intake for a group of subjects within a given statistical confidence varies for different nutrients [100]. Results from a 1-year dietary intake study by the Beltsville Human Nutrition Research Center (U.S. Department of Agriculture) determined the number of days required to estimate dietary intake with a precision of ±10% and a confidence interval of 95% in males [103]. Estimates of energy, macronutrients iron, phosphorus, potassium, sodium niacin and riboflavin could all be achieved in a 7-day period [103]. For other nutrient the number of days required varied from 8 to 39 days.

Errors may also occur in the interpretation or processing of the collected data. A recent New Zealand study showed a significant difference in estimated energy and nutrient intake when the same food records were analysed by different operators (NZ registered dietitians) [104].

Due to a lack of more accurate methods food frequency questionnaires and 7-day food records continue to be used, despite the problems described above, for the purpose of dietary assessment both in the clinical and the research setting.
3 Aims of the study

This study was undertaken to investigate the nutritional status and some of the physiological characteristics likely to impact on the nutritional requirements of spinal cord injured (SCI) athletes.

Wheelchair rugby players were selected for the study as this ensured that the group was as homogenous as possible with regard to level and completeness of injury, level of fitness, and type of training performed.

The aim of the study was firstly to investigate the nutritional habits of the study group. Secondly, physical characteristics such as anthropometric data and bone mineral status (bone mineral content and bone mineral density) was estimated. The third part of the study aimed to investigate heart rate (HR) response and oxygen uptake (VO2) at rest and during increasing levels of exercise intensity to assess whether a linear relationship exists between HR and VO2 in this group of athletes.

Finally, the overall aim of the study was to create a better picture of some of the factors that affect nutritional status and what specific nutritional recommendations may be appropriate for quadriplegic athletes.
4 Methodology

The subjects for this study were recruited from players who represented one of the regional teams in the 1999 national New Zealand wheelchair rugby tournament. Subjects were invited to participate in the study (appendix 1) by advertisements placed in “Paraflash” (the newsletter for Paralympics NZ members) and by contacting area representatives who informed players in their region about the study. The researcher was present at some of the pre-tournament games where further information about the study was provided. Players who agreed to participate gave their informed consent (appendix 2) before the start of the study.

The criteria for inclusion in the study were that players were male, had a quadriplegic spinal cord injury and had represented a regional wheelchair rugby team for a minimum of two years. In total 16 players were recruited (n=16).

Ethical approval for this study was sought and given by the Waikato Ethics Committee (Ethics Committee of the Regional Health Authority) and by the Human Ethics Committee of Massey University, Albany.

A brief questionnaire was completed by all subjects (appendix 3) to provide information about their injury and their involvement in wheelchair sport.

4.1 Dietary Assessment

Participants were asked to complete a 7-day food diary (appendix 4). Instructions on how to weigh and/or measure food items, fluids and supplement products consumed were given verbally and in writing. Subjects were provided with sets of metric spoon and cup measures for measuring food and fluids. Household scales were available for participants to borrow. However, these were only used by a few as most participants found weighing food items on scales was too difficult due to poor hand and finger function.
Some participants received assistance from their care-takers with measuring quantities of food and fluids as well as recording of daily food intake.

The food records were analysed using SERVE-NZ dietary analysis software (M. Williams Pty Ltd, Sydney, Australia) with the NZ Food Files version 10 database (Crop & Food Research, Palmerston North). The dietary analysis included total energy intake, intake of macronutrients and a selected range of micronutrients. Nutrient intake from dietary sources as well as from supplement products listed in the database or for which nutrient content could be obtained (meal replacement drink, sports bars) were included in the analysis. One subject was taking a dietary supplement not listed in the database and this could not be included in the nutrient analysis.

The results from the nutrient analysis were compared with the Recommended Dietary Intakes (RDI) for adult New Zealanders [105] and nutrition guidelines for athletes [14].

4.2 Body Composition and Bone Mineral Density

Body composition; bone mineral content (BMC), lean body mass (LBM) and fat mass (FM) as well as bone mineral density (BMD) were determined by dual energy x-ray absorptiometry (DEXA) scan (Norland TXR-26/391A051, Norland Corporation, Fort Atkinson, WI, USA) (see appendix 5 for technical details of the DEXA technique). A whole body scan was performed to determine body composition and whole body BMD and a scan of the right forearm was performed to determine BMD at the distal wrist. All subjects had their right forearm scanned regardless of which was their dominant hand. It was not considered likely that there would be any significant difference between left and right forearm as the major force on the forearm in wheelchair users comes from wheelchair propulsion (performed with both arms) rather than the comparatively minor strain from additional daily activities regularly performed with one hand.
Additional anthropometric data was collected in conjunction with the DEXA scan. Due to the subjects inability to stand unassisted, height was measured in the supine position on the x-ray bed. The recorded weight is that estimated by the body composition data (BMC+LBM+FM= body weight) and has been shown to be within a fraction of a kilogram of the weight as measured on electronic scales [106].

4.3 Exercise Test

Subjects were instructed not to perform any intense physical activity in the 24 hours prior to any laboratory testing session. Subjects were also requested not to consume any food or caffeinated products or smoke in the four hour period preceding a test session. They were also reminded to empty their catheter bags immediately prior to the test. Laboratory environmental conditions were maintained at 20°C and 50-60% relative humidity throughout all testing procedures.

Subjects were required to perform an incremental arm exercise test to volitional fatigue. The test was performed using a Monark 238 bicycle ergometer (Monark AB, Varberg, Sweden) which had been modified for use as an arm-crank ergometer. The foot pedals were replaced by hand-grips and the bicycle was secured to a specially modified support table with height-adjustable legs. The table design allowed subjects to individually adjust their position to enable them to perform the exercise session whilst seated in their own wheelchairs. In order to maximise hand grip, subjects were allowed to wear either exercise gloves or to have their hands secured with strapping tape to the hand-grips during the test. See figure 4.1.
Figure 4.1  Set-up for the exercise test

During the test respiratory gases were collected continuously on-line using a breath by breath metabolic cart system (Sensor Medics, Vmax 29, USA). Samples were immediately analysed and results displayed on an IBM compatible Compaq Pentium computer. Heart rate was continuously monitored via a four lead ECG system connected directly to the metabolic cart.

The exercise protocol started with a calibration period of two minutes during which baseline data for HR and VO2 were collected. The exercise protocol consisted of two-minute exercise periods alternated with two-minute periods of complete rest. Subjects were required to crank at 50 revolutions per minute (RPM) through the duration of the test. During the exercise periods a digital RPM display and audible metronome were positioned so that subjects could check the cranking pace. Subjects were also verbally instructed to slow down or speed up to keep the required pace through the test. The first two-minute
work period was performed at a work rate of 12.5 watts (W) and was increased by 12.5 W for each subsequent two-minute work period. Subjects continued until they were no longer able to maintain the required cranking pace. The main reason subjects gave for discontinuing the test was muscle fatigue.

Subjects remained in the exercise laboratory for 30 minutes post-exercise for monitoring of any side effects as a result of the test.

4.4 Heart Rate Monitoring in a Game Situation

An attempt was made to record heart rates during a game situation. The trial was carried out during a wheelchair rugby tournament (Waikato regional games) where eleven of the sixteen subjects were playing in their home teams. The games were 4x12 minutes in duration with one-minute rest periods between quarters and two minute intervals between games. Subjects were fitted with heart rate monitors (Polar Sports Tester, Polar Electro Oy, Finland) and the recording wrist watch was secured under the seat of the wheelchair to protect it from inadvertent interference during the game. Three subjects in each team were wearing monitors during each game. Heart rates were recorded throughout the game including the rest periods between quarters and any substitutions when players were not on the court. This was done in order to simulate as closely as possible the HR profile during a competitive game rather than that in a training situation.

Due to technical problems complete heart rate profiles had only been recorded for three of the eleven subjects. The other profiles had interruptions during the recording period where no heart rate signals had been received. The reason for this has not been clarified. All watches were fitted with new batteries prior to the testing situation and the chest straps were fitted securely and checked between each quarter. All watches were started simultaneously and were functioning at the start of the game. The distance between the chest strap and the recording watch did not exceed the 100 centimetres
prescribed by the manufacturer when the monitors were fitted prior to the start of the game. However, the most likely explanation for the failure to record throughout the game is that this distance was exceeded at certain points during the game thereby interrupting the heart rate signals for sufficient time to completely discontinue the recording.

A laboratory technician experienced in the use of the Polar heart rate monitors for exercise research was consulted but was not able to provide any clarification of the technical problems experienced.
5 Results

5.1 Demographic Data and Physical Characteristics

In table 5.1 information about age, type and duration of injury, the amount of time subjects have been involved in wheelchair sport and whether their injury was sustained through sport is presented. The mean age of subjects was 29.8 ± 6.8 years. The mean duration of injury was 10.8 ± 6.2 years. Seven subjects had C5/6 (complete) injuries, three had C6/7 (complete) injuries, three had incomplete injuries (one C4/5, one C5/6 and one C6/7), and three did not provide specific information about their injuries. The mean amount of time subjects had been involved in wheelchair sport was 6.4 ± 4.4 years. Five subjects reported starting to participate in wheelchair sport within six months of being injured. The type or intensity of sport specific training was not investigated. Six of the 16 subjects had sustained their injury through playing rugby union or rugby league.

Table 5.1 Demographic data for each subject

<table>
<thead>
<tr>
<th>SUBJECT NO.</th>
<th>AGE</th>
<th>YEARS IN W/C</th>
<th>YEARS IN W/C SPORT</th>
<th>SPORTS INJURY</th>
<th>TYPE OF INJURY</th>
<th>* PLAYER CLASSIFICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41</td>
<td>6</td>
<td>2</td>
<td>N</td>
<td>C4/5 (I)</td>
<td>2</td>
</tr>
<tr>
<td>2</td>
<td>25</td>
<td>3.5</td>
<td>2</td>
<td>Y</td>
<td>C5/6 (C)</td>
<td>0.5</td>
</tr>
<tr>
<td>3</td>
<td>37</td>
<td>9.5</td>
<td>9</td>
<td>N</td>
<td>C6/7 (C)</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>37</td>
<td>19</td>
<td>4</td>
<td>N</td>
<td>C5/6 (C)</td>
<td>0.5</td>
</tr>
<tr>
<td>5</td>
<td>37</td>
<td>18</td>
<td>3</td>
<td>N</td>
<td>C5/6 (C)</td>
<td>0.5</td>
</tr>
<tr>
<td>6</td>
<td>23</td>
<td>4</td>
<td>3.5</td>
<td>Y</td>
<td>C5/6 (I)</td>
<td>1</td>
</tr>
<tr>
<td>7</td>
<td>37</td>
<td>22</td>
<td>14</td>
<td>Y</td>
<td>C5/6 (C)</td>
<td>1</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>13</td>
<td>5</td>
<td>N</td>
<td>N/A (I)</td>
<td>3.5</td>
</tr>
<tr>
<td>9</td>
<td>28</td>
<td>17</td>
<td>14</td>
<td>N</td>
<td>N/A (I)</td>
<td>3.5</td>
</tr>
<tr>
<td>10</td>
<td>24</td>
<td>13</td>
<td>10</td>
<td>N</td>
<td>N/A (I)</td>
<td>2</td>
</tr>
<tr>
<td>11</td>
<td>32</td>
<td>13</td>
<td>8</td>
<td>N</td>
<td>C6/7 (C)</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>30</td>
<td>3</td>
<td>2</td>
<td>N</td>
<td>C5/6 (C)</td>
<td>1.5</td>
</tr>
<tr>
<td>13</td>
<td>32</td>
<td>15</td>
<td>13</td>
<td>Y</td>
<td>C5/6 (C)</td>
<td>1</td>
</tr>
<tr>
<td>14</td>
<td>29</td>
<td>4.5</td>
<td>3</td>
<td>Y</td>
<td>C5/6 (C)</td>
<td>1</td>
</tr>
<tr>
<td>15</td>
<td>22</td>
<td>7</td>
<td>6.5</td>
<td>Y</td>
<td>C6/7 (C)</td>
<td>2.5</td>
</tr>
<tr>
<td>16</td>
<td>24</td>
<td>6</td>
<td>4</td>
<td>N</td>
<td>C6/7 (I)</td>
<td>2.5</td>
</tr>
</tbody>
</table>

Mean 29.8 10.8 6.4
SD 6.75 6.2 4.4

* See explanation of classification system in section 2.1.2 of the literature review.
5.2 Body Composition and Bone Density

Information about height, weight, body mass index (BMI), weight of lean body mass (LBM), fat mass (FM) and bone mineral content (BMC) respectively, and % body fat is presented in table 5.2.

Table 5.2 Anthropometric data for the study group

<table>
<thead>
<tr>
<th>HEIGHT</th>
<th>WEIGHT</th>
<th>BMI</th>
<th>LBM</th>
<th>FM</th>
<th>%FAT</th>
<th>BMC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>1.74</td>
<td>80.4</td>
<td>26.4</td>
<td>48.3</td>
<td>29.1</td>
<td>34.9</td>
</tr>
<tr>
<td>SD</td>
<td>0.13</td>
<td>19.5</td>
<td>6.3</td>
<td>10.6</td>
<td>12.6</td>
<td>10.0</td>
</tr>
</tbody>
</table>

The mean height was 174.4 ± 12.6 cm and mean body weight was 80.4 ± 19.5 kg. This resulted in a mean BMI of 26.4 ± 6.3 kg/m². The mean weight of LBM was 48.3 ± 10.6 kg, the mean weight of FM was 29.1 ± 12.6 kg, and the mean BMC was 3.0 ± 0.5 kg. The mean % body fat based on the direct measurements of tissue weight from the DEXA scan was 34.9 ± 10 %. Neither time since injury nor time involved in wheelchair sport showed any correlation with body weight, LBM and FM.

The values for whole body bone mineral density (BMD) (g/cm²), the t-scores (BMD) for total body and for the distal wrist are presented in table 5.3. The mean value for total BMD was 1.054 ± 0.149 g/cm². The mean total body BMD t-score was -0.20 ± 1.87 and the mean BMD t-score at the distal wrist was -0.12 ± 0.95. All measurements were compared to the Australian norm as this was considered more appropriate than the American norm.

Table 5.3 Data for BMD and t-scores for the study group

<table>
<thead>
<tr>
<th></th>
<th>TOTAL BMD</th>
<th>t-SCORE TOTAL BODY</th>
<th>t-SCORE DISTAL WRIST</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mean</strong></td>
<td>1.05</td>
<td>-0.20</td>
<td>-0.12</td>
</tr>
<tr>
<td><strong>SD</strong></td>
<td>0.15</td>
<td>1.87</td>
<td>0.95</td>
</tr>
</tbody>
</table>

Normal values for BMD are 0 to 1
Osteopenia is classified as BMD -1 to -2.5
Osteoporosis is classified as BMD <-2.5
In order to estimate the bone mineral status of an individual the t-score can be used. A BMD t-score between 0 and -1 is classified as normal. The definition of osteopenia is a t-score between -1 and -2.5 while the definition of osteoporosis is a t-score below -2.5. In this study five subjects (31.2%) had total body t-scores below -1 and were classified as having osteopenia and of those two (12.5%) had t-scores below -2.5 and were therefore classified as being at risk of osteoporosis.

Values for the distal wrist were higher and only two subjects (12.5%) were found to have t-scores below -1. None of them had a t-score below -2.5. See appendix 6 for individual data for bone mineral status and appendix 7 for a sample of a DEXA scan.

Total body t-score were not significantly different to the t-scores for the distal wrist in this study group. Furthermore time since injury showed no correlation with bone mineral density.

5.3 Dietary Intake
5.3.1 Energy and Macronutrient Intake
Twelve of the 16 subjects (75%) returned completed 7-day food diaries. Where food records were unclear the subject was contacted and additional information was sought in order to make the food record as accurate as possible. All subjects completed the food diaries during a period when they were training regularly therefore making the data representative of a "typical training diet".

The average daily energy, macronutrient, fibre and alcohol intake for the 7-day period is presented in table 5.4 (carbohydrate and protein intakes are presented both as intake in grams and as grams per kg body weight). The contribution of each macronutrient as well as alcohol to total intake is presented in figure 5.1. (See appendix 8 for a summary of individual intakes).
The mean daily energy intake was $8678 \pm 2411$ kiloJoules/day (kJ/day). Rather large variations are seen in the mean daily intakes (range 5386-14324 kJ).

Table 5.4 Daily intake of energy, macronutrients, alcohol and fibre

<table>
<thead>
<tr>
<th></th>
<th>Energy (kJ)</th>
<th>CHO (g)</th>
<th>g/kg BW</th>
<th>Protein (g)</th>
<th>g/kg BW</th>
<th>Fat (g)</th>
<th>ETOH (g)</th>
<th>Fibre (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>8678</td>
<td>242</td>
<td>3.3</td>
<td>86</td>
<td>1.2</td>
<td>76</td>
<td>12</td>
<td>18.5</td>
</tr>
<tr>
<td>SD</td>
<td>2411</td>
<td>101</td>
<td>1.0</td>
<td>15</td>
<td>0.3</td>
<td>17</td>
<td>21</td>
<td>7.2</td>
</tr>
<tr>
<td>Range</td>
<td>5386-14324</td>
<td>116-508</td>
<td>1.2-6.2</td>
<td>60-117</td>
<td>0.6-1.8</td>
<td>49-99</td>
<td>0-56</td>
<td>5.9-30.9</td>
</tr>
</tbody>
</table>

![Figure 5.1 Contribution of carbohydrate, protein, fat and alcohol to total energy intake](image)

Figure 5.1 Contribution of carbohydrate, protein, fat and alcohol to total energy intake

Energy requirements are largely dependant on the amount of LBM and to a lesser extent on BMI. No correlation was found between energy intake and BMI or energy intake and LBM for the subjects in this study.
Mean daily carbohydrate (CHO) intake was 242 grams (g) representing on average 45 ± 7% of the total daily energy intake. This intake is below the 50-55% of total energy intake recommended for the general population [107] and even further below the >55% of total energy recommended for athletes [14].

The mean CHO intake expressed as grams of CHO per kg body weight was 3.3 ± 1 g/kg BW (range 1.2-6.2 g/kg BW). The recommended CHO intake per kg body weight for athletes who perform moderate intensity training for an hour daily is 5-7 g/kg BW [75]. There are no specific recommendations for spinal cord injured (SCI) athletes and it is unknown whether recommendations for able-bodied athletes should be used.

Mean daily intake of dietary fibre was 18.5 ± 7.2 grams per day (range 5.9-30.9 g/day) compared with the recommended intake for New Zealanders of 25-30 g/day [107].

Mean daily protein intake was 86 ± 15 grams representing a mean percentage contribution to total daily energy intake of 17 ± 3%. All subjects had an intake above the general RDI of 55 grams per day for males [105]. Expressed as grams protein per kg body weight the mean daily intake was 1.2 ± 0.3 g/kg BW (range 0.6-1.6 g/kg BW). The recommended dietary intake (RDI) for protein based on body weight is 0.8 g/kg BW for the general population [105] or 12-15% of total daily energy intake [107]. In the present study two subjects (17%) had mean daily protein intakes below 0.8 g/kg BW one of whom (8%) had an average daily protein intake of only 0.6 g/kg BW.

For athletes the recommended intake is 1.2-1.6 g/kg BW depending on the specific training performed while 1.0 g/kg BW is generally recommended for individuals performing moderate intensity exercise daily [108]. As mentioned above it is not known whether recommendations for able-bodied athletes should be applied to this group.
Mean dietary fat intake for the 7-day period was 76 ± 17 g/day (range 49-99g) representing 34 ± 7% of total daily energy intake. Six subjects (50%) had a daily fat intake above 33% of total energy intake.

In the NZ dietary guidelines it is currently recommended that fat should contribute no more than 30-33% of total daily energy intake [107]. Of the total energy intake 8-12% should come from saturated fatty acids (SFA), 6-10% from polyunsaturated fatty acids (PUFA) and up to 20% from monounsaturated fatty acids (MUFA).

Mean 7-day intakes of SFA, PUFA and MUFA (as contribution to total energy intake) are presented in table 5.5. The mean contribution of each group of fatty acids to total energy intake was for SFA 14.3 ± 3.6% (range 9.9-19.5%), of PUFA 4.6 ± 1.3% (range 2.6-6.8%) and of MUFA 11.6 ± 2.7% (range 7.5-15.8%).

Table 5.5 Contribution of different fatty acids to total energy intake*

<table>
<thead>
<tr>
<th></th>
<th>SFA (%)</th>
<th>MUFA (%)</th>
<th>PUFA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>14.3</td>
<td>11.6</td>
<td>4.6</td>
</tr>
<tr>
<td>SD</td>
<td>3.6</td>
<td>2.7</td>
<td>1.3</td>
</tr>
<tr>
<td>Range</td>
<td>9.9-19.5</td>
<td>9.9-15.8</td>
<td>2.6-6.8</td>
</tr>
</tbody>
</table>

* The figures for fatty acid intake does not include the glycerol fraction. This accounts for the discrepancy between %fatty acids of total energy intake and %fat of total energy intake.

Four subjects (33%) consumed alcohol during the 7-day period in which they completed their food diary. The mean alcohol intake for the whole study group was 12 ± 21 g/day (range 0-56 g/day) representing 3 ± 7% (range 0-19%) of total energy intake. The mean alcohol intake for the four subjects who consumed alcohol was 35 ± 25 g/day (range 7-56 g) representing 11 ± 8% of total energy intake (range 2-19%). For those who consumed alcohol, intake was restricted to one or two days only. The highest recorded daily individual alcohol intake was 378 grams representing 71% of the total energy intake for
that day. In comparison it is recommended that daily alcohol intake for males
does not exceed 30 g/day [107].

5.3.2. Micronutrient Intake

Intake of selected micronutrients is summarised in table 5.6. For ease of
comparison the RDI value is indicated and both the percentage of the RDI
met by the group and the proportion of subjects who did not meet the RDI for
each nutrient are indicated. For the purpose of this study the RDI values for
adult males (19-64 years) was used although one subject was 18.6 years at
the time of the study.

Table 5.6 Micronutrient intake in comparison to the RDI for New
Zealanders

<table>
<thead>
<tr>
<th>NUTRIENTS</th>
<th>MEAN ± SD</th>
<th>RANGE</th>
<th>RDI</th>
<th>GROUP % OF RDI</th>
<th>% OF SUBJECTS BELOW RDI</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>VITAMINS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin A Eq.(ug)</td>
<td>751±242</td>
<td>519-1286</td>
<td>750</td>
<td>100%</td>
<td>58%</td>
</tr>
<tr>
<td>Thiamin (mg)</td>
<td>2.0±0.7</td>
<td>1.2-2.9</td>
<td>1.1</td>
<td>182%</td>
<td>0</td>
</tr>
<tr>
<td>Riboflavin (mg)</td>
<td>1.6±0.4</td>
<td>1.0-2.2</td>
<td>1.7</td>
<td>94%</td>
<td>58%</td>
</tr>
<tr>
<td>Niacin Eq.(mg)</td>
<td>35.7±8.2</td>
<td>19.1-47.7</td>
<td>19</td>
<td>188%</td>
<td>0</td>
</tr>
<tr>
<td>Vitamin B6 (mg)</td>
<td>1.6±0.5</td>
<td>0.9-2.5</td>
<td>1.3</td>
<td>100%</td>
<td>17%</td>
</tr>
<tr>
<td>Folate (ug)</td>
<td>254±69</td>
<td>95-336</td>
<td>200.0</td>
<td>127%</td>
<td>17%</td>
</tr>
<tr>
<td>Vitamin B12 (ug)</td>
<td>4.0±1.2</td>
<td>2.4-6.8</td>
<td>2</td>
<td>299%</td>
<td>0</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>109±81</td>
<td>26-276</td>
<td>40</td>
<td>272%</td>
<td>25%</td>
</tr>
<tr>
<td>Vitamin E (mg)</td>
<td>7.3±3.0</td>
<td>4.2-12.4</td>
<td>10</td>
<td>73%</td>
<td>92%</td>
</tr>
<tr>
<td><strong>MINERALS</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>740.0</td>
<td>501-980</td>
<td>800</td>
<td>92%</td>
<td>58%</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>13.4</td>
<td>9.2-18.8</td>
<td>7</td>
<td>191%</td>
<td>0</td>
</tr>
<tr>
<td>Magnesium (mg)</td>
<td>282.5</td>
<td>148-462</td>
<td>320</td>
<td>88%</td>
<td>75%</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>1358/228</td>
<td>981-1778</td>
<td>1000</td>
<td>136%</td>
<td>8%</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>3061/800</td>
<td>1676-4851</td>
<td>1950-5460</td>
<td>100%</td>
<td>8%</td>
</tr>
<tr>
<td>Selenium (ug)</td>
<td>53.0/15.4</td>
<td>25.3-79.7</td>
<td>85</td>
<td>62%</td>
<td>100%</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>3214/1107</td>
<td>2121-5886</td>
<td>920-2300</td>
<td>100%</td>
<td>0</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>11.1/2.5</td>
<td>7.5-16.2</td>
<td>12</td>
<td>92%</td>
<td>58%</td>
</tr>
</tbody>
</table>

For the B-group vitamins thiamin, riboflavin, niacin and B6 the group RDI
value suggested in the RDI tables (based on average energy intake) has
been used. Individual requirements of these vitamins can be estimated as a
quantity per 1000 kJ (thiamin, riboflavin and niacin) and per gram of protein
(B6) and based on individual requirements of energy and protein. As it is uncertain whether estimates of energy requirements for able-bodied persons should be used for SCI individuals it was not considered appropriate to use individually calculated RDI values.

Individual intakes of micronutrients varied greatly (see range). The group mean met or exceeded the RDI for vitamin A, thiamin, niacin, vitamin B6, folate, vitamin B12, vitamin C, iron, phosphorus, potassium and sodium. However, the mean intake of the group was below the RDI for the following nutrients: riboflavin (94% of RDI), calcium (92%), zinc (92%), magnesium (88%), vitamin E (73%) and selenium (62%).

On an individual basis all subjects met or exceeded the RDI for thiamin, niacin, vitamin B12, iron and sodium. For the remaining micronutrients there were varying numbers of subjects whose mean dietary intake for the 7-day period did not meet the RDI. One subject (8%) did not meet the RDI for phosphorus and potassium, two subjects (17%) for vitamin B6 and folate, four subjects (25%) for vitamin C, seven subjects (58%) for vitamin A, riboflavin, calcium and zinc, eight subjects (75%) for magnesium, eleven subjects (92%) for vitamin E and not surprisingly no one met the RDI for selenium. No correlation was found between calcium intake and bone mineral content or bone mineral density.

5.4 Exercise Test
Fifteen of the sixteen players completed the exercise test. Table 5.7 shows a summary of number of stages completed, power output, oxygen uptake and heart rate for the group.

Subjects completed between 4-10 stages of two-minute exercise periods (mean 5.7 ± 1.9). Five of the six subjects with incomplete injuries achieved heart rates above 130 beats per minute (bpm). The highest heart rate for a subject with a complete injury was 124 bpm.
Table 5.7  Oxygen uptake, heart rate and power output for the group

<table>
<thead>
<tr>
<th>STAGES</th>
<th>Max power (W)</th>
<th>Max VO2 (L.min⁻¹)</th>
<th>Max HR (bpm)</th>
<th>Incomplete</th>
<th>Complete</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>5.7</td>
<td>70.8</td>
<td>1.28</td>
<td>127</td>
<td>155</td>
</tr>
<tr>
<td>SD</td>
<td>1.9</td>
<td>23.5</td>
<td>0.52</td>
<td>28</td>
<td>25</td>
</tr>
</tbody>
</table>

Figure 5.2 shows the HR/VO2 graphs for two subjects, one with a complete injury and one with an incomplete injury. See appendix 9 for individual HR/VO2 graphs. The coefficient of determination for VO2/HR was above 0.923 for all subjects (range 0.923-0.998). This clearly indicates that HR and VO2 follow a linear increase during increasing exercise intensities.

Figure 5.2  Heart rate and oxygen consumption data for two subjects

5.5  Heart Rate Monitoring in a Game Situation

Due to technical limitations described in the methodology section complete heart rate recordings during a whole game were obtained for only three subjects. The lack of data for the rest of the study group means that no comprehensive results can be reported. Incidentally, all three subjects for
whom HR were recorded had incomplete injuries resulting in HR profiles higher than what would be expected for the whole group.

The time the players spent in each ten beat heart rate interval was recorded and depended on time spent on the court in each game. (See appendix 10 for a sample of an individual HR recording.)

One of the subjects reached the same maximum heart rate as that achieved in the laboratory test while the other two subjects reached heart rates significantly higher than that in the laboratory.

The time spent in each ten beat HR interval was multiplied by the average VO2 for that heart rate interval, as estimated from the laboratory exercise test, then multiplied by 4.825 kcal (Weir equation - 1 L oxygen = 4.825 kcal [80]) to calculate total energy expenditure during the game. Total energy expenditure was then divided by the total game time so that energy expenditure per minute could be derived. For the two subjects who recorded HR above the maximum reached in the laboratory VO2 was estimated by extrapolation for the higher HR recordings.

The three subjects recorded energy expenditures of 3.7, 4.7 and 7.2 kcal/minute respectively. One subject played two consecutive games resulting in a total estimated energy expenditure of 602 kcal while the other two subjects only played one game resulting in energy expenditures of 244 kcal and 441 kcal per game.
6 Discussion

This study was undertaken to investigate the nutritional status and some of the physiological characteristics likely to impact on the nutritional requirements of spinal cord injured (SCI) athletes. Dietary habits, body composition and bone mineral status were assessed and an exercise test was performed to determine the relationship between heart rate (HR) and oxygen uptake (VO2) during rest and exercise. Wheelchair rugby players were selected for this study as this ensured that the group was as homogenous as possible with regard to level and completeness of injury and type of training performed. Most other studies of disabled subjects have included people with different types of disabilities [15], different levels of SCI [38], sedentary and active subjects [19], and athletes participating in widely different sports [15] in the same study. This may be useful if differences between groups are being investigated or comparisons are being made. However, for the purpose of accurately describing the characteristics of a specific group, selecting a homogenous group was considered to be essential.

Research from other studies has shown that SCI individuals experience changes in body composition [68], reduction in bone mineral density [46], and have altered responses to exercise [2] compared to able-bodied persons. Information about dietary intake of SCI individuals (both sedentary and athletes) is scarce. In this study comparisons of dietary intake have therefore been made with other NZ population groups, with able-bodied athletes in similar sports and with recommendations for able-bodied athletes.

In order to make recommendations for daily energy intake the energy cost of both sedentary activities and physical activity must first be determined. A few studies have reported that energy requirements in SCI populations are lower than in age, height and weight matched controls both in the rehabilitation phase [79] and in the longer term [77]. No such data appears to be available for SCI athletes who are regularly physically active.
6.1 Anthropometric Characteristics and Body Composition

Few studies estimating body composition specifically of athletes with quadriplegic spinal cord injuries have been carried out. Studies on body composition of SCI subjects have not reported on the subjects level of activity or exercise history [71].

The mean BMI of the wheelchair rugby players in this study of 26.4 kg/m² was above the WHO definition for “healthy” BMI of 20-25 [109] cited in [110] but similar to that found in a recent study of paraplegic subjects (24.8 kg/m²) [40]. There are several possible explanations for this result. Firstly, it is well documented that the average weight for height is increasing in the NZ population. In 1994 it was estimated that 42% of New Zealand males had a BMI above 25 and 10% were in the above 30 (obese) category [110]. The recently published 1997 New Zealand National Nutrition Survey (NNS97) reported that in the 19-24 year and 25-44 year age groups BMI was 24.8 and 26.1 respectively and that 34.9% and 39.2% respectively of men in those population groups were overweight [1]. Fifty six percent of the players in this study had a BMI above 25 with 31% in the 30-35 BMI category.

The appropriateness of using the same BMI ranges for all ethnic groups has been discussed and current recommendations from the South Pacific Commission suggest that the “healthy” BMI range for people of Polynesian descent should be 22-27 and the “overweight” range 27-32 [111] cited in [112]. The rugby players in this study were not asked to state their ethnic group in the demographic questionnaire however, this information was recorded for the purpose of the DEXA scan. Six subjects or 37.5% identified themselves as being Maori or Polynesian, which is a significantly higher proportion than in the average NZ population [1]. This could have accounted for the higher average BMI of the study group.

Despite being physically active the subjects would still be considered to have a largely sedentary lifestyle due to their injury. This could also have
contributed to the higher BMI. Lastly, BMI is not considered an appropriate measure of "healthy weight" in athletes due to their often larger amount of muscle mass compared to the general population [113]. Able-bodied Australian rugby union and league players are reported on average to have BMIs between 28-30 [113] due to a higher than average muscle mass. In this study six of the 16 subjects (37.5%) reported having sustained their SCI while playing rugby and it is possible that others had been active rugby players pre-injury. This over-representation of rugby players in the study group (it is estimated that approximately 14-15% of SCI are caused by sport) could to some extent explain the higher than average BMI values because many subjects due to natural selection already had a high BMI.

Of note is the large variation in BMI of the study group (17.6-39.2 kg/m²) with players' BMIs ranging from the underweight to obese categories. This finding may be particular to this relatively small sample size. Such large variation is not normally seen in most able-bodied sports. Perhaps, as wheelchair rugby becomes more competitive, an "anthropometric ideal" may emerge as is currently happening for talent identification purposes in other sports [113].

Body composition data however, shows that the players had an average body fat percent of 34.9% which is also higher than the 8-18% average body fat level of 17-30 year old North American males [114] indicating that a higher proportion of the weight is body fat. The range for individual body fat levels was wide (16-49%) although it shows that even the leanest wheelchair rugby subject had a relatively high level of body fat compared to that of able-bodied rugby players of 6-16% [115]. Levels of body fat estimated by sum of six skinfolds in able-bodied rugby union players also show a similar variation (40-75 mm) [113].

Interestingly, of the four wheelchair players who had BMIs below 20, the variation in % body fat ranged from 19-41%. This is most likely an indication of the extent of loss of lean muscle mass in many athletes with quadriplegia.
It must be remembered that in most data on % body fat, figures have been derived from any one of a number of different equations. Percent body fat as measured by DEXA is known to consistently estimate a higher level of body fat [67] compared to that estimated by the most commonly used anthropometric equations.

A recent New Zealand study [40] that also used dual-energy x-ray absorptiometry (DEXA) to assess bone, lean tissue content and fat mass in paraplegic subjects found significantly higher levels of body fat than in matched able-bodied controls. Compared to the paraplegic group the wheelchair rugby players in this study were of similar age and height but weighed on average 4.2 kg more. Average lean tissue mass was identical in both groups and bone mineral content was only slightly lower in the paraplegic group. The difference in body mass was accounted for by the higher level of body fat in the wheelchair rugby players. The higher level of fat mass in the wheelchair rugby group is most likely explained by the higher level of injury (quadriplegic compared to paraplegic). The level of body fat has been shown in other studies to increase the higher the level of spinal injury is [71]. The able-bodied controls in the study by Jones had significantly higher lean tissue mass and bone mineral content than the paraplegic subjects and an average body fat level of 16.6% [40].

In a descriptive study of four male paraplegic subjects the level of body fat was 23% [22] which is also significantly lower than that of the rugby players in this study.

In a longitudinal study of paraplegics leg lean mass was shown to decrease while arm lean mass increased during the first year of injury [39]. The authors did not report on the level of physical activity but did mention that the lower increase in arm muscle mass in some subjects was likely due to failure to adhere to the rehabilitation programme.
A study by Ide et al [74] of SCI wheelchair marathon racers showed levels of body fat of 15.6-23.7% however, body fat was assessed by sum of three skinfolds (triceps, subscapula and suprailium) and may not accurately represent total body fat as no measurements were taken of the inactive (non-innervated) body parts. It is also possible that athletes in aerobic endurance sports such as marathon racing have lower levels of body fat. The study did not report on any differences in body composition between athletes with different levels of spinal injury [74].

No information could be found on body composition specifically of quadriplegic athletes. It would be interesting to compare the levels of lean mass, bone mass and fat mass in sedentary and physically active quadriplegic subjects. It could be hypothesised that more physically active people are able to maintain lean muscle mass and bone mass while minimising gains in body fat. This may have beneficial effects in terms of reducing risk of chronic disease such as cardiovascular disease, insulin resistance and osteoporosis [13]. It also has the potential to assist with performance optimisation in disability sport.

Other implications for SCI athletes and quadriplegic athletes in particular is that the relatively high level of body fat may further hinder effective thermoregulation which is known to be impaired in SCI [52]. Higher levels of body fat reduces the ability to dissipate body heat and the body surface area-to-body mass ratio for sweat evaporation is smaller [116] thus reducing exercise tolerance.

### 6.2 Bone Mineral Density

A rapid drop in bone mineral density (BMD) post-injury, an increased incidence of osteoporosis and an increased risk of fractures are well-known complications of SCI [9] [37]. Many factors are known to affect BMD however, in SCI the almost complete removal of weight bearing exercise is thought to be the most significant factor [9, 48]. Time since injury has been shown to be
positively correlated to BMD [37]. The reduction in total bone mass is most severe in the legs and the pelvis however, some bone mass is also lost from the arms and trunk [34].

The wheelchair rugby players in this study had a mean whole body BMD of 1.05 g/cm², only slightly below that of a group of paraplegic twins (1.11 g/cm²). The non-paraplegic twins (controls) had a mean BMD of 1.27 g/cm² [9]. This compares to the Australian norm for bone density of 1.08 g/cm² used for assessment of BMD in New Zealand [117]. To this author's knowledge there is no data for normal bone density in the New Zealand population. Studies conducted in this country have mainly been on at-risk groups such as the elderly, post-menopausal women and young girls [118].

Although regional differences in BMD have been found between paraplegics and quadriplegics most studies have failed to find any significant difference in whole body density [35, 38].

It has not been confirmed that physical activity can reduce the loss of bone mineral or reverse the drop in BMD experienced in the acute phase. Comparisons between physically active and sedentary SCI subjects (matched for age, lesion level and years post-injury) show that the physically active subjects had higher values for BMD at all sites although statistically not significantly higher than the inactive groups [49]. Results from intervention studies using electrical-stimulation-induced leg cycling (ES-LC) for periods of 6-9 months are contradictory. Subjects who were able to achieve a work output above 18 W showed some increases in BMD of the femur [46] while a group of quadriplegic subjects failed to show any changes in BMD [44]. BeDell et al found no significant increase in lower body bone density after 25 weeks of ES-LC in a group of paraplegic and quadriplegic subjects [45].

The use of DEXA to determine bone mineral status, monitor the rate of bone loss and assess the effect of different types of intervention is useful [40]. The
increased availability of this technology in New Zealand should be utilised by researchers in this area.

As a group the wheelchair rugby players had a total body t-score in the normal range (0 to -1). No correlation was found between the time since injury or the time subjects had participated in wheelchair sport and the values for whole body t-score or distal wrist t-score.

In this study five subjects (31.2%) had total body t-scores below -1 and were classified as having osteopenia and of those two (12.5%) had t-scores below -2.5 and were therefore classified as being at risk of osteoporosis. Values for the distal wrist were higher and only two subjects (12.5%) were found to have t-scores below -1. No one had a wrist t-score below -2.5.

In wheelchair sport the upper body and arms are the body parts performing the majority of the work. It was therefore hypothesised that the BMD in the forearm may be higher than the whole body BMD. This has been shown to be the case in other sports such as in jockeys [119] where the physical stress on the forearm is thought to contribute to the higher BMD. No significant difference was found however between t-scores for distal wrist and whole body t-scores in the wheelchair rugby players. Some subjects with a normal to high whole body t-score had below normal t-scores for the wrist while the opposite was true for other subjects. There is large variation in hand function between players and as mentioned earlier players are classified between 0.5 and 3.5 according to functional ability. It was hypothesised that this may have affected the amount of work that can be performed with the forearm and therefore the wrist t-score. However, when the data was analysed no correlation was found between player classification and wrist t-score.

Some authors have suggested that early rehabilitation may have the most beneficial effect in preventing bone loss and development of premature
osteoporosis as most of the bone loss occurs during the first few months post-injury.

The close to normal values for BMC and BMD in the SCI players may be explained by the higher than average proportion of Maori and Polynesian subjects compared to the general NZ population. Until reference data is available for these population groups it is impossible to compare the relative bone mineral status of SCI people from different ethnic groups. It is also unclear at this point whether it is a higher total BMC or a higher BMD that results in a lower incidence of osteoporosis in these ethnic groups [117].

Another possible explanation for the near normal bone mineral status is that being physically active post-injury has resulted in a higher than expected bone mineral status in this study group. Examples of other factors that may affect peak bone mass are calcium intake during childhood, vitamin D intake, lifestyle factors such as alcohol intake and smoking, weight-bearing exercise and certain medications such as steroidal drugs [118]. As many of the subjects in this study had been injured while playing rugby it is reasonable to assume that they had been performing regular physical activity up until the time of injury and this may have contributed to a high peak bone mass compared with others of the same age. Some subjects however, sustained their injuries before the age when they would have reached peak bone mass. It could be speculated that this may have had a negative impact on their adult peak bone mass [118].

A closer examination of individual results shows that the two subjects with whole body t-scores below -2.5 both described themselves as being Maori/Pacific Island while the three highest t-scores were also found in this group. This indicates that factors other than ethnicity are partly responsible for variations in BMD.
A larger sample population would be required in order to fully investigate the typical changes in bone metabolism that occur after spinal cord injury. Alterations in parathyroid hormone levels, 1,25 dihydroxyvitamin D and nephrogenous cyclic adenosine monophosphate levels are known to occur in the acute and rehabilitation phases of SCI [9]. Hormonal changes and other factors such as heredity, whether peak bone mass had been reached at the time of injury, smoking and alcohol consumption, dietary factors, use of medications and the normal process of ageing may all have an effect on bone mineral status in this population [118].

6.3 Dietary Assessment
The lack of data on dietary intake of SCI populations in general and athletes in particular prompted the assessment of dietary habits in this study. Able-bodied athletes are known to have nutritional requirements that differ from the general (largely sedentary) population [14]. It is reasonable to assume that SCI athletes also differ in their nutritional needs compared with both able-bodied sedentary and physically active individuals as well as with non-active SCI people. An assessment of typical dietary practices is useful in order to establish nutrient intake compared to recommended dietary intakes (RDI), to identify areas that may require particular attention and to use as a starting point when investigating specific dietary changes and their effect on performance in this group of athletes. The known physiological changes in SCI must also be taken into account when recommending particular dietary practices for this group.

The increased risk of cardiovascular disease, mature onset diabetes and osteoporosis was described in the literature review section. With this in mind recommendations for appropriate dietary changes based on the assessment of current dietary intake for individual players as well as for the group can be made.
Recent information from the 1997 New Zealand National Nutrition Survey (NNS97) provides current data on the dietary intake of the New Zealand population and of specific age, gender and ethnic groups within the population [1]. Results from this study showed a mean energy intake for males of 13247 kJ/day (19-24 years) and 12904 kJ/day (25-44 years) [1]. The mean energy intake for the group of SCI athletes (8678 kJ/day) is similar to that of NNS97 subjects in the 10th percentile (8879 kJ/day) [1] indicating a much lower than average energy intake.

The mean energy intake represents an intake of 117 ± 36 kJ/kg/day (range 54-174 kJ/kg/day). Studies of rehabilitating quadriplegic subjects have estimated energy expenditure by indirect calorimetry to 96 kJ/kg/day [79]. The wheelchair rugby players would be expected to have a higher level of physical activity and therefore energy expenditure than patients rehabilitating in hospital. In a study by Monroe et al at 24-hour energy expenditure of a group of paraplegic and quadriplegic subjects who were at least 2 years post-injury was estimated to 7824 kJ/day or 111.6 kJ/kg/day [77]. Despite not being allowed to perform any strenuous physical activity while in the metabolic chamber SCI subjects had a significantly lower energy expenditure compared with the able-bodied controls (9941 kJ/day). The extra 800 kJ/day consumed by the wheelchair rugby players compared to that of the subjects in Monroe's study may be required for the regular sports training performed by this group.

Although body weight of the wheelchair rugby players was not recorded daily over the 7-day period or over a longer period of time at least two subjects mentioned that they were attempting to lose body fat while one subject mentioned that he wanted to increase muscle mass. It is possible that other subjects were making similar attempts and this may be the cause of the large variation in energy intake in the study group.

Lean body mass is strongly related to resting metabolic rate and therefore largely responsible for individual differences in energy requirements [77,
In individuals with stable body weight a correlation between amount of lean body mass and energy intake exists [114], however no such correlation was found in this study group. As mentioned above some participants may have attempted to gain or lose body weight and therefore adjusted energy intake accordingly. Other possible explanations is that the sample size was too small to detect such a relationship or that differences in energy expenditure from physical activity had an influence on the results.

The intake of macronutrients and their contribution to total energy intake was calculated. The mean contribution of carbohydrate (CHO), protein and fat to total intake was almost identical to that reported in the NNS97 for the 19-24 and 25-44 year age groups; CHO = 45%, protein = 14/15% (NNS97) and 17% (rugby), fat = 36% (NNS97) and 34% (rugby). Nutrition guidelines for the general population recommend a diet consisting of 50-55% CHO, 12-15% protein and no more than 30-33% from fat [107]. Guidelines for able-bodied athletes recommend that more than 55% is contributed by CHO, 12-15% by protein and that fat makes up the balance [14].

The mean carbohydrate intake of 242 g/day for the rugby players compares to 362 g/day (19-24 years) and 338 g/day (25-44 years) for males in the NNS97 study [1]. This represents 45% of total energy intake in all three groups. Despite recommendations that athletes should consume >55% of energy from CHO studies have shown that able-bodied team sport player often do not follow these recommendations [76].

When carbohydrate intake is calculated per kg of body weight (g/kg BW) the mean intake is 3.3 g/kg BW for the wheelchair rugby players. For able-bodied athletes this would be considered inadequate to perform regular physical training [75].

Although it is known that energy requirements are reduced after SCI there does not appear to be any information about the carbohydrate requirements
of this group of athletes. To this author's knowledge no studies have been published reporting on the muscle glycogen content or predominant muscle fibre type in the upper body of SCI athletes. Muscle fibre samples of the deltoid muscle for example are more difficult to obtain than samples from the quadriceps as an open biopsy must be performed [120]. In order to be able to provide recommendations for CHO intake for wheelchair athletes and SCI in particular questions about the amount of muscle mass used during exercise and the amount of carbohydrate utilised during different intensity and duration of exercise must be addressed. The ability of the upper body muscles to increase their storage of carbohydrate as a response to regular endurance training in SCI athletes is unknown. The possibility of optimising performance by modifying quantity and timing of carbohydrate should also be investigated in this population. Until this has been done no exact recommendations for carbohydrate intake for SCI athletes can be provided. If the storage capacity for CHO is indeed limited it is likely that more frequent feedings of CHO would be beneficial during longer bouts of exercise.

As a proportion of total energy intake the wheelchair rugby players' protein intake represented on average 17%, above the recommended intake of 12-15% for athletes. The mean intake in grams was 86 grams and this compares to 110-117 g/day (19-24 and 25-44 years) of total energy intake for males in NNS97 [1]. Despite a lower daily intake of protein all subjects in the wheelchair study were well above the RDI for protein of 55 g/day for men (range 60-117 g/day). The mean protein intake per kg of body weight of 1.2 g/kg BW was well below the reported 1.5-2.1 g/kg BW estimated in able-bodied players in team sports such as soccer, Australian rules football and gridiron [121].

One of the wheelchair rugby players recorded a protein intake below (0.6 g/kg BW) the 0.8 g/kg BW stated in the RDI and one further subject was below the recommended intake for general sports activity of 1 g/kg BW [108]. The subject with the lowest protein intake also had the lowest recorded mean
energy intake over the 7-day period. A meal replacement drink was used to substitute some meals indicating that this practice was used either temporarily to reduce energy intake or as a matter of convenience.

Protein requirements for SCI athletes do not appear to have been established and a number of issues should be considered when investigating this area. The requirement for protein is determined by the requirements for repair and maintenance of cells including muscle cells [108]. The reduced amount of lean body mass in SCI athletes may result in a reduced requirement for protein however, this has not been established. When energy and/or carbohydrate intake is below that required to fuel exercise protein is used to a larger extent as a source of energy [108]. The relatively low carbohydrate and energy intakes of the study group may lead to an increased use of protein to cover energy requirements of daily living and physical activity. This is difficult to assess before energy and carbohydrate requirements have been established for this group.

Some subjects may have reduced energy and carbohydrate intake severely in order to reduce levels of body fat however, this may instead lead to difficulties in maintaining lean body mass [108, 110] which is already reduced in this group. A vicious circle of decreasing levels of lean body mass, fluctuating body weight, reduced energy expenditure and recurring weight gain may occur, similar to that described in dieters on very low calorie diets [110].

The mean contribution of fat to total energy of 34% (representing a mean intake of 76 grams) in the wheelchair rugby group was slightly below that estimated in NNS97 in the 19-24 and 25-44 year groups of 36% [1]. The highest recorded individual fat intake was 99 grams, well below the mean intake of 129 and 126 g/day recorded in the corresponding age groups in NNS97. It would of course be desirable to reduce fat intake to a level closer to the recommended <30% of total energy for athletes [14]. A reduction in fat intake would make increasing the intake of carbohydrate easier to achieve.
Reducing fat intake would also be desirable from a long-term health perspective. As body fat has been shown to be increased in this population [40] a reduction in dietary fat intake may facilitate body fat loss and thereby reduce the already high risk of developing mature onset diabetes which is thought to be related to abdominal obesity [8, 13].

Development of guidelines for estimating energy expenditure in SCI athletes is crucial in order to more accurately calculate required energy and macronutrient intake for this group.

The contribution of saturated (SFA), monounsaturated (MUFA) and polyunsaturated fatty acids (PUFA) to total energy intake for the study group is consistent with that for New Zealand males in the 19-24 and 25-44 year groups for SFA = 16 and 15% respectively, for PUFA = 5% and for MUFA = 12% [1]. Current recommendations state that of the total energy intake 8-12% should come from SFA, 6-10% from PUFA and up to 20% from MUFA [107]. In groups at increased risk of cardiovascular disease or individuals with raised serum cholesterol levels SFA intake should be restricted to the lower end of the range (6%) [96]. Considering the increased risk of cardiovascular disease in SCI it would be logical to recommend a drastically reduced intake of saturated fat.

Similar recommendations for fat intake have been made previously in studies on the long-term health status of SCI people [13].

The mean intake of dietary fibre in the study group was 18.5 ± 7.2 g/day (range 5.9-30.9). The mean fibre intake for the same age groups in the NNS97 study was 23-25 g/day. New Zealand Nutrition Guidelines recommend 25-30 g/day. This is most likely a reflection of the lower energy intake in the study group rather than a major difference in food selection. Constipation is one of the complications of SCI and laxatives are commonly used for bowel elimination [11]. Lack of innervation of the bowel musculature is the main reason for the long-standing bowel problems and will not be
resolved by an increased fibre intake however, other aspects of bowel health and general health may benefit from inclusion of more dietary fibre. Dietary fibre has been shown to have positive effects in cardiovascular disease as well as in mature onset diabetes [13]. A high fibre intake is often recommended as a way of reducing calcium absorption [32] however the appropriateness of reducing calcium intake in SCI has been discussed in the literature review. Combining an increased fibre intake with the recommended calcium intake appears to be the most appropriate advice. This would combine the benefits of an adequate calcium intake for bone health with the beneficial effects of fibre on bowel health and general long-term health.

The mean alcohol intake for the whole study group was 12 g/day although 2/3 of the subjects did not consume any alcohol. The mean alcohol intake for the four subjects who consumed alcohol was 35 g/day (range 7-56 g) a contribution of 11% to total energy intake (range 2-19%). For those who consumed alcohol, intake was restricted to one or two days only. The highest recorded daily individual alcohol intake was 378 grams representing 71% of the total energy intake for that day. It is recommended that daily alcohol intake for males does not exceed 30 g/day [107]. In the comparable general population groups mean daily alcohol intake was 18-21 g/day [1]. It is difficult to accurately assess alcohol intake when a relatively short study period (7 days) is used. Some of the subjects who drank no alcohol during the 7 day period may do so at other times. For example more players may regularly consume alcohol in the off-season than they do during the rugby season.

Excessive alcohol intake is known to negatively affect bone mineral status [118] so considering the increased risk of premature osteoporosis a recommendation to limit alcohol intake in the SCI population may be prudent. The 7-day period during which dietary habits were surveyed is not considered long enough to give an accurate indication of whether alcohol intake had any effect on bone mineral status in this study group.
Micronutrient intakes for the study group showed great variation. The group mean met or exceeded the RDI for vitamin A, thiamin, niacin, vitamin B6, folate, vitamin B12, vitamin C, iron, phosphorus, potassium and sodium. However, the mean intake of the group was below the RDI for riboflavin, calcium, zinc, magnesium, vitamin E and selenium.

The relatively low energy intake compared with the same age groups in the general population explains why more of the wheelchair rugby players did not meet the RDI for a number of micronutrients. The lower the energy intake the higher the nutrient density must be in order to meet the recommended intakes [100].

As individuals, all subjects met or exceeded the RDI for thiamin, niacin, vitamin B12, iron and sodium. For all other micronutrients there were subjects who did not meet the RDI when the average intake was calculated for the 7-day study period. Relatively few subjects did not consume enough phosphorus, potassium, vitamin B6 and folate to meet the RDI however, the number of subjects who did not meet the RDI for vitamin C, vitamin A, riboflavin, calcium, zinc, magnesium, vitamin E and selenium was of more concern. All nutritional supplements recorded by participants have been included in the dietary analysis. These result clearly indicate that the majority of the subjects would benefit from nutritional advice. One of the limitations of this study was the amount of time for which dietary intake was recorded. Seven days may not have been long enough to accurately estimate the intake of certain micronutrients such as vitamin A and vitamin C for which a longer study period is required [103].

Direct comparisons with the NNS97 data for intake of each macronutrient have not been made. Different methodologies may affect the intake of micronutrients to a larger extent than for macronutrient intake. The food frequency questionnaire used in NNS97 is likely to cover seasonal variation in food intake better [100] than the 7-day food diary used in this study. The
wheelchair rugby study was conducted between June and August (NZ winter).

As mentioned in the literature review the appropriateness of recommending a reduced dietary intake of calcium to prevent formations of renal stones is not supported by current research [122, 123]. It is suggested that a number of other dietary strategies such as decreased oxalate intake, inclusion of some calcium rich items in each meal, increased intake of fibre, increased fluid intake and possibly a reduction in sodium intake may be more successful in preventing calcium oxalate renal stones [123].

Food records only provide an indication of potential nutritional problems. It is important to remember that failure to meet the RDI for a nutrient does not signify a deficiency of that nutrient. RDIs are determined with a large safety margin in order to cover the nutritional needs of 97.5% of the population [105]. They can be used to identify groups and individuals at risk of nutritional deficiency however diagnosis of a deficiency can only be made after biochemical and clinical assessments have also been carried out.

6.4 Exercise Testing

The main aim of the exercise test was to investigate whether a linear relationship exists between oxygen uptake (VO2) and heart rate (HR) from resting and during increasing intensity of exercise. If such a relationship exists heart rate monitoring could potentially be used to assess energy expenditure both at rest and during exercise in SCI individuals [77, 83].

It is well-established that individuals with complete quadriplegic injuries have a low heart rate response to exercise [2] and that they, due to the reduced amount of total muscle mass they can use, are unable to perform aerobic exercise at an intensity that requires any substantial increase in oxygen use [7]. It was therefore not surprising that subjects with complete SCI were unable to reach heart rates above 130 bpm. The mean maximum HR for the
whole group was 127 ± 28 bpm however, if mean HR is calculated separately for subject with complete and incomplete injuries the mean for the subjects with complete injuries is 109 ± 7 bpm and for the group with incomplete injuries 155 ± 25 bpm.

The mean VO2 for the group was 1.28 L.min⁻¹. When results are analysed for players with complete and incomplete injuries the same trend is seen for VO2 as for HR. The mean VO2 for the incomplete injury group was 1.62 L.min⁻¹ and for the complete injury group 1.05 L.min⁻¹.

This is consistent with findings from other studies where HR and VO2 have been monitored during exercise [2, 3, 52, 89].

Power outputs reached by the wheelchair rugby players were also similar to those achieved by quadriplegic athletes in other studies [92] [7]. However, power output did not show the same relationship with completeness of injury as HR and VO2. The power output that is achieved is likely to be a result of training status and some athletes who regularly train on an arm-crank ergometer are more likely to be able to produce high power outputs.

For the purpose of this study HR and VO2 were the variables of interest and power output was simply measured to ensure that workload was increased in a similar fashion for all subjects.

There was some doubt among the players that HR response during an exercise test would show an increasing pattern as most of them felt that their heart rates remained low throughout the duration of any exercise session. This was mentioned particularly with reference to the HR players felt they had reached during exercise prior to their SCI injury.

As the individual graphs show (see appendix 10) the coefficient of determination for VO2/HR was above 0.923 for all subjects (range 0.923-
0.998). This clearly indicates that HR and VO2 follow a linear increase during increasing exercise intensities. Other studies that have measured these two parameters in an exercise laboratory situation have reported on only HR [92] or VO2 [124] separately. A study by Jacobs et al found a linear relationship between the two parameters, however the study group consisted only of paraplegic subjects [88].

There are some concerns that HR may not be a valid method of estimating energy expenditure during rest or low levels of activity due to relatively large variations in daily resting HR in able-bodied subjects [125]. However, the linear relationship between HR and VO2 found in the quadriplegic rugby players in this study indicate that heart rate monitoring may be a useful method for estimating energy expenditure during exercise. In order to confirm the findings from this study a larger sample of quadriplegic athletes should be investigated. Validation against more accurate methods such as the doubly labelled water technique should be performed [126].

Another factor that may affect the HR response in quadriplegic subjects in particular is the effect of blood pressure changes and blood pooling during prolonged exercise [2]. Heart rate may also be affected by the phenomenon referred to as “cardiovascular drift” seen in able-bodied athletes as a response to increased body temperature during submaximal exercise [127] when the heart rate rises in an effort to circulate more blood to the skin for cooling.

The HR and VO2 profiles during other forms of exercise such as endurance exercise, short anaerobic exercise and exercise on a wheelchair ergometer may be valuable to investigate.

6.5 Heart Rate Monitoring in a Game Situation

Heart rate monitoring has been used in able-bodied populations to estimate energy expenditure during rest and exercise [83, 125]. Due to technical
problems described in section 5.5 complete heart rate data was only obtained for three subjects, all of whom had incomplete injuries. The recorded HR profiles showed that one subject reached the same maximum HR in the game situation while the other two subjects reached heart rates significantly higher in the game situation than that reached in the laboratory test. This may be explained either by the effect of nervousness and therefore neural stimulation on heart rate in a competitive situation [128] or by the fact that the set-up of the arm-crank ergometer in the laboratory could not be optimally adjusted to each subject's sitting height and arm length.

The individual energy expenditure for the three subjects whose HR data was available showed a relatively large variation between individuals (3.7-7.2 kcal/min). Possible explanations for this variation include factors such as body mass, functional capacity, efficiency of the wheelchair and the subject's training status.

In order to compare energy expenditure estimates found in this study to other studies the values for energy expenditure were converted to kcal per kilogram (kg) of body weight per day. The values for the three subjects in this study are then estimated to 98.7, 56.4 and 130.0 kcal/kg/day respectively.

Few studies exist with which meaningful comparisons of estimated energy expenditure can be made. Twenty-four-hour energy expenditure (daily living, no vigorous exercise allowed) was estimated to 26.6 kcal/kg/day in rehabiliated SCI subjects (paraplegic and quadriplegic) in a respiratory chamber [77]. A similar study by Cox et al found energy expenditure in stable rehabilitated quadriplegic subjects to be 22.7 kcal/kg/day [79] while Sedlock et al estimated an energy expenditure of 20.9 kcal/kg/day in paraplegic subjects during complete rest [22].

Compared to the data for resting and moderately active paraplegic and quadriplegic subjects in other studies the wheelchair rugby players in this
study expended two to five times as much energy per kg body weight per hour during exercise. The increased energy expenditure would have a significant effect on daily energy expenditure if performed regularly. Daily exercise of similar intensity and duration would result in an additional energy requirement of 240-440 kcal per hour of exercise undertaken.

In order to fully assess 24-hour energy expenditure and provide recommendations for daily energy intake in this population further studies should be performed. The heart rate monitoring technique should be validated against a more reliable method such as the doubly-labelled water method before it is recommended as a viable method for estimating energy expenditure in the SCI population.

6.6 Recommendations
In summary, based on the information from the literature review and the findings from this study, some recommendations that may improve health outcomes and ensure optimum sports performance in the quadriplegic population can be made.

In order both to prevent chronic disease such as cardiovascular disease, insulin resistance and osteoporosis and, to enhance sports performance it is important to maintain lean body mass, bone mineral content and prevent body fat gain. Dietary recommendations should focus on nutrient density in order to reach an intake of essential nutrients that is closer to the RDI than what is currently achieved. Advice should be given against calcium restriction unless the individual has been identified as being at risk of forming calcium oxalate renal stones. Individual recommendations are necessary in order to adjust each subject's energy, carbohydrate, protein and fat intake to match their level of physical activity.

Considering the increased risk of cardiovascular disease, specific advice regarding limiting saturated fat intake and replacing some fat with mono- and
polyunsaturated fat appears sensible. Spinal cord injured people would benefit from individualised dietary advice and follow-up during the rehabilitation phase of SCI to prevent unnecessary body weight gain.

Other dietary manipulations such as using high doses of vitamin C to provide an acidic environment in the bladder to reduce infection [81] or drinking cranberry juice [129] appear to be harmless and may be worth considering for individuals who have frequent bladder infections.

Recommendations for physical activity should also be individualised and focus on maintenance of lean body mass, functional strength and aerobic capacity. Combining wheelchair exercise with arm cranking and/or electrical-stimulation-induced leg cycling may result in an increased oxygen requirement that is large enough to stimulate an improvement in oxygen uptake.

Regular monitoring of bone mineral status is useful in order to identify individuals at risk of premature osteoporosis.

Further work is required regarding the possibility of using heart rate monitoring for the estimation of energy expenditure. This would enable physiologists, nutritionists and coaches to more accurately assess the energy requirements of individual athletes in different sports. Based on the findings in this study it appears that heart rate monitoring can at least be used in order to estimate energy expenditure during exercise.

6.7 Limitations

In this study male wheelchair rugby players with quadriplegia were selected in order to make the study group as homogenous as possible. This resulted in a relatively small study group (n=16) however, most other studies of spinal cord injured subjects where body composition and exercise parameters have been
measured have been of similar size or smaller. As this study only included male subjects the data may not be applicable to female subjects.

The measurement of body composition by DEXA can be affected by the subjects' hydration status. However, DEXA is considered less sensitive to this than most other methods of body composition assessment [65]. In order to minimise this effect subjects were instructed to ensure adequate hydration on the day of the DEXA scan.

The accuracy of the estimated dietary intake is limited. As food intake was recorded for 7 days the results are only likely to be valid for energy and macronutrient intake. A longer study period is required to more accurately estimate micronutrient intake. Accurate recording of quantities and type of food is always a concern in any dietary study. Due to the subjects' disability the measurement of both food items and liquids may have been more difficult than for able-bodied individuals. An example of this is that many subjects did not use the scales provided as weighing food was too difficult due to poor hand-function.

The accuracy of the dietary analysis may be affected by limitations in the food data base. Some substitutions were made when the food item recorded could not accurately be matched or when recipes for home-made food were not available. Errors in data input is another factor that may have affected the results.

The results from the exercise testing may have been affected by the set-up of the arm ergometer. Although the table on which the ergometer was fixed could be height adjusted differences in height and arm length between subjects may have resulted in some subjects achieving below their potential in the exercise test. This may have affected the maximum HR, VO\textsubscript{2} and power output that was reached however, it should not have any impact on the correlation between HR and VO\textsubscript{2} values.
7 Conclusions

Many of the physiological changes that occur as a result of a quadriplegic spinal cord injury (SCI) are likely to affect the nutritional status and the nutritional requirements of those individuals. The known long-term health risks associated with SCI such as cardiovascular disease, type 2 diabetes mellitus and osteoporosis may to some extent be preventable by appropriate dietary modifications and regular exercise. Prevention of obesity is also indicated for maintaining independence and achieving an optimum rehabilitation outcome [24].

The results from this study showed that subjects had an average height, weight and BMI in the same range as that of able-bodied New Zealand males in the same age groups [1] and similar to that of New Zealand paraplegic subjects [40]. Overweight and obesity is considered a common complication of SCI. The proportion of subjects in this study group who had a BMI above 25 g/cm2 (56%) was similar to that of the general New Zealand population (51%) [110] however, a higher number of SCI subjects (31%) were in the obese category (BMI >30) compared to the general NZ male population (10%) [110].

Dual-energy x-ray absorptiometry (DEXA) showed that, as a percentage of body mass, lean tissue mass was lower and fat mass was higher than that of able-bodied athletes in similar sports (rugby union and league) and that of sedentary paraplegic males. This finding was not surprising and confirms that of other researchers [40, 74].

DEXA revealed that as a group the wheelchair rugby players in this study had a bone mineral status in the normal range. Previous studies have found BMD to be reduced regionally (lower extremities) while insignificant reductions in whole BMD have been noted [35, 38]. In the present study five subjects had t-scores below -1 (definition of osteopenia) and only two subjects (12.5%) had
t-scores below -2.5 (definition of osteoporosis). Possible explanations for this finding are:

- higher proportion of Maori/Pacific Island subjects in the study group than in the general population
- subjects may have had a higher bone mineral density pre-injury as a result of their involvement in sport (more than a third had sustained their injury through rugby and others indicated that they had been involved in regular sport pre-injury)
- subjects' current involvement in sport has provided some protection against bone mineral loss post-injury

The dietary analysis revealed that the wheelchair rugby players had a significantly lower energy intake than NZ males of the same age in the general population [1] however, the contribution of macronutrients to total energy intake was almost identical. The pattern of consumption of saturated, mono- and poly-unsaturated fatty acids was similar to that of the general population [1].

The reduced energy intake resulted in lower intakes of most micronutrients with a significant number of subjects not meeting the RDI for vitamins A, C, E, riboflavin, calcium, zinc, magnesium and selenium.

Based on the findings of a high incidence of cardiovascular disease, type 2 diabetes and osteoporosis in previous studies and the findings of low nutrient intakes in this study it would be appropriate to apply recommendations of reduced intake of fat and particularly saturated fat, an increased intake of nutrient dense foods, and an emphasis on increased calcium intake to the SCI population.

Heart rate (HR) and oxygen uptake (VO2) were found to be similar to that found in other studies of quadriplegic athletes [2, 88, 124]. The most
interesting finding in this study was the linear increase in HR and VO2 as measured in the laboratory exercise test.

The attempt at measuring heart rates during a game situation could not be completed due to technical limitations. However, the energy expenditure estimated from the HR data for three subjects indicated that subjects expended between 3.7-7.2 kcal per minute of exercise resulting in an additional energy requirement of 220-430 kcal per hour of exercise. This is between two to five times that estimated in daily activities in resting paraplegic and quadriplegic subjects in other studies.

Limitations to this study include the relatively small study group, the number of days for which dietary data was collected and the arm-crank ergometer set-up used in the exercise test.

Many factors that affect the nutritional requirements of SCI people and SCI athletes in particular should be investigated. The following list suggests some areas where future research is needed:

- the effect of exercise on body composition and bone mineral density
- muscle glycogen stores and utilisation in different groups of SCI athletes
- changes in glycogen synthesis in SCI subjects with glucose intolerance
- energy cost of different forms of exercise
- heart rate and oxygen uptake responses during different types of exercise
- substrate utilisation in SCI athletes during rest and exercise
- validation of methods for estimating energy expenditure against the doubly-labelled water technique
- effect of dietary modifications on sports performance in SCI athletes
- effect of dietary modifications on long-term health outcomes and disease risk

There are many potential benefits of regular physical activity and dietary modifications such as reduced risk for obesity and its accompanying health
risks [13]. Further studies should be carried out in order to evaluate the long-term effects on health outcomes from regularly participating in physical activity of different intensity and duration.
References


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Appendix 1

Invitation to Participate in a Research Study
INVITATION TO PARTICIPATE IN A RESEARCH STUDY

My name is Ingrid Perols and I am a sports dietitian currently studying towards a Master of Science (Nutritional Science) at the Institute for Food, Nutrition and Human Health at Massey University. This year I am completing a research study with the aim to investigate the nutritional needs of wheelchair rugby players. I am writing this letter to invite athletes to participate in the study. Potential participants for this study will be approached either by Paralympics NZ area representatives or by myself during one of the wheelchair rugby training camps.

The criteria for taking part in the study is that you have played wheelchair rugby for at least two seasons, that you are a male spinal cord injured athlete and that you are representing a regional team playing in the national wheelchair rugby tournament this year.

Ethical approval for this study has been sought and given by the Massey University Human Ethics Committee, Albany Campus and the Waikato Ethics Committee.

THE STUDY

Two of the many factors that can affect an athlete's nutritional needs are energy expenditure and body composition. In order to be able to give nutrition information to athletes it is important to have information about these factors. In this study data about energy expenditure, body composition and current food and fluid intake will be collected. It is envisaged that this study will contribute towards the formulation of sports nutrition guidelines for spinal cord injured athletes.

Participants will be asked to participate in the following procedures:

- Record their food and fluid intake for a period of seven (7) days.
- Have their body composition assessed by DEXA scan (Dual X-ray Absorptiometry).
- Perform a 30 minute game simulation exercise test where energy expenditure will be estimated by indirect calorimetry (breath analyser technique). The exercise test will be performed in the exercise laboratory at the Waikato Polytechnic.
- Wear a heart rate monitor for 2x36 hours in order to record heart rate during this period. This data will be used to estimate average 24 hour energy expenditure.
- Provide basic demographic data about themselves.
It is planned that all data collection will take place in conjunction with the wheelchair rugby games that are played in Hamilton and Morrinsville this season. Exercise testing will not take place on the same day as a game is played. There will be no cost to the participants. All test results will be made available to participants and the data remains the property of the study participants. All information about participants will be strictly confidential between the researcher and the participant and any laboratory staff who assist with the collection of data.

Data collected from participants in the study will only be presented in a cumulative form and data about individual participants will not be used for any purpose. All data collected about the participant remains the property of the participant. Data collected about individual participants will be securely stored in a locked office throughout the study and destroyed at the end of the research project.

Before the start of the study subjects will be given verbal and written information about the study and asked to sign an informed consent form. Participants have the right to withdraw from the study at any time without further explanation and have the right to refuse to answer any questions at any time. The participants will remain anonymous throughout the study and individual participants will not be able to be identified.

In the unlikely event of a physical injury as a result of your participation in this study, you will be covered by the accident compensation legislation with its limitations. If you have any questions about ACC please feel free to ask the researcher for more information before you agree to take part in this trial.

If you have any queries or concerns about your rights as a participant in this study you may wish to contact a Health and Disability Services Consumer Advocate, telephone (07) 834 3960.

If you are interested in participating in the research study please contact me for further details. You can either mail me the reply card enclosed with this letter or I can be contacted either at my workplace (The Waikato Polytechnic) on phone: 07-834 8800 ext. 8600 or by fax: 07-858 0201, or mobile 025-527 481. My research supervisor, Dr Clare Wall can be contacted on phone 09-443 9748 or fax: 09-443 9640. Thank you for taking the time to read this information sheet.

Ingrid Perols
Appendix 2
Consent Form
CONSENT FORM

I, ___________________________, give permission for Ingrid Perols, a student at the Institute for Food, Nutrition and Human Health, Massey University, Albany, to use information collected about me during the course of this research project to be utilised for the purpose of completing the research project stated above. The Massey University and the Waikato Ethics Committee guidelines for Human Ethics in Research will be followed.

I have read and I understand the information sheet dated 7 April 1999 for volunteers taking part in the study designed to investigate the nutritional needs of spinal cord injured athletes. I have had the opportunity to discuss this study with the principal researcher and I am satisfied with the answers I have been given.

I understand that taking part in this study is voluntary (my choice), that I agree to participate under the conditions set out in the information sheet, and that I may withdraw from the study at any time up until a time when data collection has been completed.

I understand that my participation in this study is confidential and that no material which could identify me will be used in any reports on this study. I understand that the project will be submitted as part fulfilment of the completion of a Master of Science degree.

I understand the compensation provisions for this study.
I have had time to consider whether to take part.
I know whom to contact if I have any side effects to the study.
I know whom to contact if I have any questions about the study.

I would like the researcher to discuss the outcomes of the study with me.

YES/NO
I would like the researcher to discuss the outcomes of the study with me.

YES/NO

I consent to my GP being informed of my participation in this study/the results of my participation in this study.

YES/NO

I, _______________________________, hereby consent to take part in this study.

Date

Signature

Full name of Researcher: Ingrid Perols
Contact Phone Number for researcher: 07-834 8800 ext. 8600 (work), 025-527 481
Project explained by: Ingrid Perols

Signature

Name of witness

Signature of witness
Appendix 3
Demographic Questionnaire
DEMOGRAPHIC DATA

The information collected on this form is for the purpose of providing some background information about the participants in the study “Nutritional requirements of spinal cord injured athletes”.

1. Name:

2. Address:

3. Contact phone number:

4. Age:

5. Number of years participated in wheelchair sport:

6. Type of spinal cord injury:

7. Years wheelchair bound:

Thank you for providing this information.
Appendix 4

Food Diary
FOOD DIARY

Date: __________________________ Name: __________________________

- Record all food and fluids consumed during each 24 hour period. Start each day on a new sheet.
- Describe all food and fluid as accurately as possible eg. untoasted muesli (brand if known or brief description of ingredients), sports drink (brand and concentration of carbohydrate if known).
- Estimate serving sizes as accurately as possible eg. slice of bread; toast thickness, cup of rice, serving spoon of mashed potato, size of pizzas or slices etc.
- Also list any nutritional supplements taken. This includes sports drinks, other carbohydrate supplements (squeezies, gels), sports bars, protein supplements, vitamin and mineral supplements or any other nutritional supplement products.
- If possible record the approximate time food or fluids were consumed.

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<th>DESCRIPTION OF FOOD/FLUID</th>
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Appendix 5

Technical Description of the DEXA Technique
DEXA (dual-energy x-ray absorptiometry)

Dual-energy x-ray absorptiometry is a technique used for measurement of both bone mineral content, bone density and the composition of soft tissue. DEXA was first developed for measurement of bone mineral content and as such has been accepted as a method with a high degree of accuracy. A DEXA scan also provides information about the composition of soft tissue (proportions of fat/lean tissue). The manufacturers of DEXA equipment have recently refined the technique by improving the calibration standards for fat and lean tissue and by applying a fat distribution model thereby increasing the reliability of the results produced by the DEXA scanner [67].

Fundamentals of DEXA

The DEXA scan measures the attenuation of an x-ray beam, at two energies (40 and 70 or 100 kV), at every point throughout the scanning of the whole body or selected body part. The resulting measurements are analysed for bone mineral content and also produce pixels, or picture elements.

Bone mineral, fat (lipid) and lean (non-fat soft tissue) differ in their x-ray attenuation properties mainly due to their differing amounts of high atomic number elements. Bone mineral has a high percentage of calcium and phosphorus while soft tissue is almost entirely made up of hydrogen, carbon and oxygen. Fat and lean components of soft tissue can be differentiated by the presence of the electrolytes potassium, chlorine, sulphur and calcium in lean tissue. These are not present in fat tissue.

DEXA can only differentiate between TWO different materials or body tissues at once. All three body compartments (bone, fat and lean tissue) can therefore not be measured directly. For the calculation of body composition the DEXA technique has been developed to sort the pixels into those with and without bone tissue. This is possible as the bone mineral in the body is concentrated in dense local regions (the bones) of the body. Pixels with bone are then analysed for bone and soft tissue while non-bone pixels are analysed for fat and lean tissue. An estimate of the composition of the lean tissue in the bone-
containing pixel then has to be made. This is done by assuming that the soft tissue closest to the bone has a similar composition to that in the next closest pixel of no-bone tissue.

The distribution of fat in the remaining tissues of the body is estimated using a linear fat distribution model based on actual in vivo fat distribution in different body layers from the bone to the skin (body fat is generally distributed in increasing amounts from the bone towards the surface of the skin) [67].

DEXA, like most other methods for the estimation of body composition, is sensitive to the hydration status of the subject.

Operation of the DEXA scanner
A whole body DEXA scan is performed with the subject in the supine position on the x-ray bed. The required length of the scan is pre-set by the operator prior to starting the scan [62]. The scanner is connected to a computer which stores and analyses the data.

The results for bone mineral density can be compared to reference values for both an Australian or a North American reference population. There are currently no reference values available for specific ethnic groups within the population (eg. Maori, Pacific Island, Asian) [117].
Appendix 6
Individual Bone Mineral Data
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<th>t-SCORE DISTAL WRIST</th>
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Appendix 7

Sample DEXA Scan
Total BMD (g/cm²) : 0.964
Total BMC (g) : 2745
Total Lean Mass (g) : 41507
Total Fat Mass (g) : 10404
Total Fat % : 19
Siri UWE Fat % : 12
Brozek UWE Fat % : 13
Soft Tissue Fat % : 20
% TBMC/FFM : 6

STD CV for Total BMD: 1.0  See Guide for other CVs.
6.5 x13.0 mm, 260 mm/s, 66.30 cm  Rev. 3.8.0/2.0.0  Calib. 01/11/99

COMMENTS
RF Arm C on 01/11/99 16:52

Dist. R+U CAUCASIAN NORLAND

Prox. R+U CAUCASIAN NORLAND

% Young Ref. 103.1 % Young Ref. 100.6
T - Score 0.21 T - Score 0.06
% Age Matched 102.2 % Age Matched 100.5
Z - Score 0.15 Z - Score 0.05

Bone image not for diagnosis

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STD CV for Distal R+U BMD: 1.4 See Guide for other CVs.
1.0 x 1.0 mm, 20 mm/s, 8.00 cm Rev. 3.8.0/2.0.0 Calib. 01/11/99

COMMENTS
Appendix 8 A

Individual Subjects' Dietary Intake of Energy, Macronutrients and Alcohol (graph)
No. | 1  | 2  | 3  | 4  | 5  | 6  | 7  | 8  | 9  | 10 | 11 | 12  
--- |----|----|----|----|----|----|----|----|----|----|----|     
kcal | 10607 | 8729 | 14324 | 7881 | 10322 | 6856 | 7727 | 6756 | 10465 | 7062 | 5386 | 8019  
%CHO | 41 | 42 | 59 | 46 | 49 | 54 | 36 | 40 | 50 | 50 | 36 | 45  
%protein | 16 | 14 | 14 | 17 | 16 | 20 | 21 | 18 | 14 | 22 | 19 | 16  
%fat | 29 | 25 | 26 | 37 | 35 | 27 | 44 | 43 | 31 | 29 | 46 | 40  
%ETOH | 15 | 19 | 2 | 0 | 0 | 0 | 0 | 0 | 6 | 0 | 0 | 0  

The table shows the energy intake (kcal) and the percentage distribution of energy intake among carbohydrates (%CHO), protein (%protein), fat (%fat), and ethanol (%ETOH) for twelve subjects. The bar chart above the table illustrates the energy intake across these nutrients for each subject.
Appendix 8 B

Individual Subjects' Dietary Intake of Energy, Macronutrients and Alcohol
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Appendix 9

Individual Heart Rate/Oxygen Uptake Values for the Exercise Test
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Appendix 10

Sample of a Heart Rate Recording from a Wheelchair Rugby Game
HR: 72
Time: 0:00:00.0

Average: 98 bpm
Recovery: -12 bpm

Duration of exercise: 2:08:42.6
Selected period: 0:00:00.0 - 2:08:40.0 (2:08:40.0)