THE EFFECTS OF BODY WEIGHT SUPPORT TREADMILL TRAINING ON AMBULATORY CAPACITY, BONE METABOLISM, AND BLOOD LIPID PROFILES IN PERSONS WITH SPINAL CORD INJURY

By

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Abstract

Common sequelae of chronic spinal cord injury (SCI) include ambulatory deficits, osteoporosis in sublesional bone, and dyslipidemia. Body weight support treadmill training (BWSTT) is recognized as an effective tool for ambulation retraining in persons with SCI. Until now, the ability of BWSTT to modify bone metabolism and blood lipid and lipoprotein profiles has not been investigated. Given that this protocol has the potential to stress both the skeletal and cardiovascular systems, we proposed that in addition to improving ambulation, a BWSTT program would favorably modify bone metabolism and blood lipid and lipoprotein profiles in persons with SCI.

To test these hypotheses, we completed a 3 month BWSTT study with 5 persons with chronic (19 months – 11 years post-injury), incomplete (ASIA C – D) SCI. The subjects trained 3X/week for a target duration of 40 minutes/session. Levels of deoxypyridinoline, a urinary marker of bone resorption, and osteocalcin, a serum marker of bone turnover/formation, were determined pre- and post-training. Bone mineral density (BMD) of the whole body, lumbar spine, and hips was assessed at the same time intervals with DEXA. Plasma concentrations of total cholesterol (TC), high density lipoprotein cholesterol (HDL-C), TC/HDL-C, low density lipoprotein cholesterol (LDL-C), and very low density lipoprotein cholesterol (VLDL-C) were evaluated pre- and post-training. % BWS, treadmill velocity, ambulatory endurance, independent steps, and required assistance were recorded at each training session.

Across the training period, all subjects experienced reductions in the amount of BWS and manual assistance that were required, and increases in independent stepping, treadmill velocity, and endurance. Ambulatory progress was related to the pre-training ASIA scores, with the greatest gains seen in those subjects with the highest residual motor capabilities. At the completion of the training program, urinary [deoxypyridinoline] was significantly increased ($p<0.05$) compared to pre-training. Serum [osteocalcin] and BMD were not significantly different pre-to post-training. With respect to lipid metabolism, only TC was significantly lower ($p<0.05$) post-training. The response of the lipoprotein fractions to the BWSTT protocol was variable between subjects.

The results of this study indicate that coincident with improving ambulation, BWSTT has the potential to increase bone remodelling in persons with chronic SCI. The effect of this protocol on lipid and lipoprotein profiles is uncertain.

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Introduction

On a cursory level, it is relatively easy to generate arguments that advocate body weight support treadmill training (BWSTT) as a tool for gait retraining in persons with spinal cord injury (SCI). BWSTT allows a person with para- or tetraplegia to be appropriately unloaded and consequently, it promotes symmetrical distribution of weight through the legs, it frees the arms for balance and swing, and it encourages gait patterns that produce afferent signals that the central nervous system (CNS) can recognize. Although these arguments are valid, they do not adequately explain the underlying neuronal basis of the locomotor response in persons with SCI who are exposed to BWSTT. In the section that follows, the basic principles/theories of human locomotion will be discussed with an emphasis on the locomotor potential of SCI patients.

Neuronal Control of Human Locomotion

According to current theories (Dietz 1992; Dietz et al. 1997; Hultborn et al. 1998), the neuronal control of human locomotion is based on the interactions of three systems: I) central programs or central pattern generators (CPGs) in the spinal cord, II) supraspinal systems, and III) afferent/proprioceptive systems. Under normal conditions, the interactions of the three systems can be described as follows: the tonic input necessary for driving lumbosacral CPGs for locomotion is generated by brain stem neurons and mediated by long-descending axons to the interneuronal surface of the lumbosacral spinal

cord; here, the tonic input converges with phasic peripheral input (see review in Dimitrijevic et al. 1998).

I) CPGs

The term CPG operationally defines an ensemble of spinal neurons whose membrane, synaptic, and network properties are capable of generating, in the absence of peripheral or descending inputs, a detailed motor pattern such as locomotion (Rossignol and Barbeau 1995). The concept of CPGs for locomotion stems from research with various animal models - the spinalized cat playing the leading role. After a complete spinal transection at the low thoracic level, cats display well-organized bilateral and reciprocal stepping movements of their hindlimbs when they are held in the air by the thorax and when they are placed on the ground with their body weight supported (Rossignol and Barbeau 1995). After a few weeks of daily treadmill training, these cats can walk on their hindlimbs without any further assistance (ie. body weight relief), while their forelimbs rest on a fixed platform and their tails are held to improve lateral stability (see reviews in Barbeau and Rossignol 1994; Rossignol and Barbeau 1995). While these findings suggest that the locomotor pattern is generated at the spinal level, they do not exclude the possibility that afferent signals from the periphery are conducting the locomotor response. In cats, evidence for *central*-spinal generation of locomotion has been obtained by recording the activity of motoneurons 1) after neuromuscular blockade ("fictive locomotion"), which prevents the locomotor movements themselves and therefore, all movement-related phasic afferent feedback, and 2) after spinal cord transection, which

abolishes all descending influences (see review in Rossignol and Barbeau 1995).

In contrast to the abundance of data in animals, evidence for locomotor CPGs in humans is less definitive, and is for the most part, limited to research published within the past decade. Much of the support comes from BWSTT studies and is detailed in a section below. Perhaps the simplest piece of evidence that is suggestive of CPGs for human gait are the innate steplike movements that are present at birth, either spontaneously initiated or triggered by peripheral stimuli. The central origin of these movements may be assumed since the electromyographic (EMG) bursts precede the actual mechanical events; spinal localization of these movements may be inferred based on the findings of stepping in anencephalic infants (see review in Dietz 1992).

In the absence of a model for fictive locomotion in humans, the strongest evidence for spinal CPGs (exclusive of BWSTT research) may be that non-patterned electrical stimulation of the posterior structures of the lumbar spinal cord in subjects with complete, long-standing SCI can induce patterned, locomotor-like activity (Dimitrijevic et al. 1998). These authors reported that epidural spinal cord stimulation over the second lumbar segment was effective in eliciting step-like EMG activity and locomotor synergies in subjects with complete paraplegia. Other evidence to support the existence of locomotor CPGs in humans, excluding that from BWSTT studies, is limited. A case study involving a person with chronic, incomplete SCI reported that involuntary, rhythmic, alternating, and forceful movements of the lower extremities could be reliably evoked by lying the subject supine and extending his hips (Calancie et al. 1994). The striking similarity between the movement and EMG patterns in this subject and those described in many reports using the

surgically reduced cat model led the authors to suggest that this was the first well-defined example of a central rhythm generator for stepping in the adult human. Confirmation of the existence of a spinal rhythm-generating network in this subject would have required two additional parameters however: 1) motor output in the absence of movement (fictive locomotion), so as to eliminate afferent feedback, and 2) a total elimination of supraspinal influence.

II) Supraspinal Systems

The contributions of supraspinal systems to locomotion can be dissolved into two main branches: 1) locomotor driving systems in the brain stem (Dimitrijevic et al. 1998; Grillner 1985) which control the overall intensity of the spinal locomotor mechanisms (ie CPGs) and bring them into operation; and 2) adaptive systems which modify the stereotyped spinally generated motor output according to the environmental context in which it takes place (see review by Dietz 1992).

In the absence of afferent feedback to the CPGs, locomotor movements do not take place spontaneously in persons with complete SCI. It has been suggested that this is due to the predominance of supraspinal over spinal neuronal mechanisms in humans (Kuhn 1950). The profound loss of tonic facilitative drive from supraspinal (brainstem) centers in persons with SCI might explain why it is so difficult to sufficiently excite the proportion of each of the motoneuron pools required for stepping (Dobkin et al. 1995).

Although spinal feedback systems can provide detailed compensation for perturbations, the perfection of locomotor movements, such as accurate placing of the

foot on the ground, depends on the cerebellum being intact (see review in Grillner and Wallen 1985). The cerebellum receives, via spino-cerebellar pathways, detailed information concerning the movements at each joint and also detailed copies of commands issued from CPGs (ie. efference copy information) (see review in Grillner and Wallen 1985). In each step cycle, the cerebellum provides phasic corrections via descending spinal pathways. The cerebellar contribution to motor control can be achieved by presetting and adjusting the gain of proprioceptive reflexes and by sequencing the programmed responses (see review in Dietz 1992).

Other sources of afferent information involved in the supraspinal modification of locomotion include signals from the visual and vestibular systems.

III) Afferent/Proprioceptive Systems

Although locomotor CPGs are believed to be capable of generating the complex pattern of gait, afferent feedback is crucial for the adaptation of the movement to what is actually happening to each limb during the step cycle (Dietz et al. 1997; Grillner 1985; Hultborn et al. 1998; Pearson et al. 1998). The afferent feedback allows the CPGs to modulate efferent output in a manner that is appropriate for the environment. The lumbosacral motor pools appear to respond to multiple sensory modalities simultaneously (including limb peak load, muscle tendon stretch, temporal and spatial distribution of load on the soles of the feet, limb unloading, hip extension, contralateral limb information and cutaneous stimulation (see review in Harkema et al. 1997)), and the type and magnitude of the response is dependent on the phase of the step cycle. Two aspects of the motor

pattern for gait that are particularly dependent on afferent signals are: 1) the magnitude of activity in knee and ankle extensor muscles, and 2) the duration of extensor bursts during stance (ie. the timing of the transition from stance to swing) (Pearson et al. 1998).

Afferent input may influence locomotor movements via "direct" pathways to the motoneurons, or more "indirect" pathways through the rhythm generator (Grillner 1985; Hultborn et al. 1998). This differentiation between afferent feedback that results in a reflex response "independent of CPGs" and feedback that is "interpreted" by locomotor CPGs, is a fundamental concept underpinning BWSTT theories. There is general agreement that the control of stance and gait is not based on reflex responses, but that a selection and integration of peripheral and supraspinal input occurs to generate an appropriate response pattern (Dietz 1992).

The interaction between supraspinal input and afferent reflex activity at the level of the spinal cord characterizes the interdependence of the neural components involved in locomotion. Spinal reflex responses are not stereotyped to a given sensory input; rather, depending on the descending and segmental conditions, different available efferent pathways are utilized (see review in Dietz 1992). An example is the maturation of locomotor patterns in which a descending inhibition of monosynaptic/short-latency reflexes (by presynaptic inhibition of group I afferents) and facilitation of polysynaptic/long-latency spinal reflexes becomes established (Dietz 1992). This regulation can be described as a reciprocal modulation of mono- and polysynaptic reflex mechanisms (Dietz 1992). When this control is either not yet matured (children) or impaired (spastic paresis), inhibition of monosynaptic stretch reflexes is missing in

combination with a reduced facilitation of polysynaptic reflexes (Dietz 1992).

Locomotion in SCI and BWSTT

Based on the preceding model of the neuronal control of human locomotion, the direct effects of a complete SCI on locomotor potential are:

I) the loss of facilitative drive from the brainstem to the CPGs,
II) the loss of cerebellar refinement of locomotor patterns, and
III) the loss of supraspinal descending pathways that modulate spinal reflexes.

The loss of facilitative drive is the most consequential of the three, as it effectively "turns off" the lumbosacral locomotor CPGs and subsequently eliminates any proprioceptive feedback that would have been generated by movement. Assuming the model is accurate, a person with a SCI retains the potential to ambulate due to the residual locomotor CPGs in the lumbosacral cord. However, the lack of both supraspinal drive (disrupted neuronal connections) and afferent input (paralysis) is paramount, and voluntary stepping movements are not realized. This assessment concurs with Kuhn's (1950) explanation for the reduction in locomotor potential in humans with SCI (see above).

Within the past decade, a group of research teams (Barbeau/Rossignol et al.; Dietz et al.; Dobkin/Edgerton/Harkema et al.; Wernig et al.) interested in human locomotor CPGs have focused their attention on persons with SCI. Because of limited or absent supraspinal input (depending on lesion severity), persons with SCI offer a valuable paradigm in which the theoretical, latent, locomotor CPGs can be isolated and assessed. The majority of the research uses a system of BWSTT adapted from experiments with cats. BWSTT reduces the weight bearing requirement of standing. This "unloading",

which is usually accomplished with a harness + suspension system, allows independent standing in SCI persons not capable of supporting 100% of their body weight. Physiotherapists can then assist with the stepping movements of the subjects in accordance with the speed of the treadmill. The ability of ambulation-associated afferent signals to elicit and train efferent locomotor EMG activity and movements of the leg is assessed and used as indirect support for spinal CPGs.

A review of the methods and apparatus used in BWSTT (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; Dietz et al. 1997; Dobkin et al. 1995; Wernig and Muller 1992; Wernig et al. 1995) can be summarized with the following general guidelines - note that these guidelines are intended for protocols in which recovery of gait is the primary goal. Unloading of the subject's body weight should be accomplished with a harness that suspends the body in a near-vertical position and allows an axial direction of contact forces with the treadmill. The harness should permit extension at the hip joint during the stance phase of gait. BWS should be set as the minimum amount that enables the subject to stand vertically with legs fully extended (ie. no buckling) and, depending on residual motor function, execute stepping movements. The speed should be adjusted with two goals in mind: to find an optimal rhythm for each subject and to minimize assistance. If required, assistance should be provided to encourage proper timing of the stance and swing phases of both legs, and to ensure that the knee and hip joints are fully extended during the stance phase to optimally support body weight. Hyperextension of the knees should be avoided however, to prevent damage to ligaments around the knee joint. The feet should pass under the vertical projection of the subject's center of gravity in order to

obtain a full activation of extensor muscles. The pelvis should be kept fixed so that loading of knee and ankle joints occurs in a physiological manner. The subject's hands, while they may be used for balance on the lateral frames of the treadmill, should be discouraged from bearing weight. Whenever possible, walking with reciprocal arm swinging should be encouraged.

The provision of BWS has been shown to have beneficial effects on the locomotor pattern of persons with incomplete SCI. Compared with full weight bearing ambulation, ambulation with BWS in this cohort produces more appropriate EMG timing in relation to the gait cycle, increases in single limb support time, stride length and comfortable walking speed, and a decrease in percentage total double support time. Joint angular displacement data also reveal straighter trunk and knee alignment during the weight bearing phase with BWS (Visintin and Barbeau 1989). The ability of the BWS apparatus to provide a context in which ambulation can approximate "normal" is advanced as a primary reason for the success of BWSTT. This is because the afferent volleys associated with these "normal" ambulatory patterns are more apt to be recognized by the lumbosacral CPGs.

The inability of SCI subjects to voluntarily generate locomotor EMG patterns when supine, even after BWSTT (Dobkin et al. 1995; Wernig and Muller 1992; Wernig et al. 1995), suggests that the sensory information associated with weight bearing stepping is required to initiate the locomotor pattern. A further indication of the importance of incoming afferent information during BWSTT is that when the flow of stepping is interrupted (ie. by an incomplete step), a proper starting position must be imposed to continue stepping (Wernig et al. 1995). In the same study, the authors (Wernig et al.

1995) also observed that most of the subjects with SCI could not start stepping from a parallel limb position with equal loading on both limbs. Instead, the subjects needed to load and unload the paralysed limb in a rhythmic and alternating manner according to the 'rules of spinal locomotion' to initiate stepping. These observations suggest that in persons with SCI, recognizable afferent patterns during BWSTT can assume the role previously held by the brainstem as the facilitator of the lumbosacral locomotor CPGs.

Several studies investigating the effectiveness of BWSTT in persons with SCI have yielded positive results with respect to ambulatory outcomes (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; Dobkin et al. 1992; Dobkin et al. 1995; Wernig et al. 1992; Wernig et al. 1995; Wernig et al. 1998). The improved ambulatory capacity following BWSTT is reflected by improvements in weight bearing, walking speed, walking endurance and gait pattern (see review in Barbeau et al. 1998). Underlying these external changes are neuromuscular adaptations; these include better synchronization of EMG bursts to the swing and stance phases of the step cycle (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; Dobkin et al. 1992), increased amplitude of EMG signals (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998), and more recently noted, non-specific musculo-tendinous adaptations (Dietz et al. 1998). The latter observation stems from BWSTT in patients with paraplegia due to a cauda (peripheral nerve) lesion. It was observed in this cohort that improvements in locomotor function were not connected with a corresponding change in leg muscle EMG activity. This led the authors to conclude that the locomotor improvements associated with BWSTT can be partially explained by adaptations within the locomotor apparatus - ie. muscular-tendon systems (Dietz et al.

1998).

The parsimonious explanation that the EMG activity observed during BWSTT is a reflection of rhythmic stretches of the leg muscles (Rossignol and Barbeau 1995; Stewart et al. 1991) (and not a manifestation of CPG output), has been repeatedly refuted (Dietz 1995; Dobkin et al. 1995; Harkema et al. 1997). The finding that leg muscle EMG activity during stepping is about equally distributed between muscle lengthening and shortening, both in subjects without neurological impairments and in subjects with complete paraplegia, indicates that stretch reflexes are unlikely to be the sole factor responsible for the EMG patterns (Dietz 1995; Dobkin et al. 1995).

The more reciprocally organized EMG patterns and increased EMG amplitudes seen over time with BWSTT in persons with SCI have been ascribed by some authors to the ability of the isolated human spinal cord to not only generate a locomotor pattern, but also "to learn" (Dietz 1995; Dietz et al. 1997). Although it is appealing, this "learning" theory does not have unanimous support within the BWSTT literature. It has been proposed that the increase in EMG amplitude seen with BWSTT is due to an increase in body loading (ie. decrease in BWS) which is interpreted and responded to by the lumbosacral spinal cord (Harkema et al. 1997). This argument is based on the observation that the modulation of EMG amplitude from the soleus and gastrocnemius muscles of untrained SCI patients during BWS ambulation is closely associated with limb peak load (Harkema et al. 1997). Thus, the principle of this argument is not that the spinal cord can learn, but rather that the lumbosacral spinal cord has the ability to perceive and respond to afferent stimuli (Harkema et al. 1997). To identify the relative contributions of chronic

CPG learning and acute lumbosacral responsiveness (to limb peak load) to the increased EMG activity seen with BWSTT, Dietz et (1998) used mathematical analyses. The investigators reported that when the effect of (un)loading was separated statistically, the effect of training on EMG activity was still significant. This result agrees with earlier studies that found that: 1) the slope of EMG increase with BWSTT did not parallel the amount of unloading in subjects with SCI, and 2) unloading of healthy subjects did not result in a similar reduction of EMG amplitude and activity (Dietz et al. 1994; Dietz et al. 1995). One benefit of this apparent spinal CPG "learning" is that the EMG activity in the extensor muscles becomes high enough to allow the extensors to take on more load during ambulation. In summary, the results to date demonstrate the importance of activity-dependent reorganization following CNS injury and suggest that significant plasticity is possible and may persist for many years following SCI (Barbeau et al. 1998).

As noted above, one of the consequences of a SCI (with respect to locomotor potential) is the loss of supraspinal modulation of reflex activity at the level of the spinal cord. A loss of descending control means that functionally essential polysynaptic reflexes are not facilitated and that monosynaptic stretch reflexes are not inhibited (Dietz 1992). As a result, hypertonia (in the form of spasticity) and exaggerated tendon reflexes (ie. clonus) can develop following SCI (Dietz et al. 1997). What is the relationship between these neuromuscular changes and locomotor potential in SCI? Dietz et al. (1997) reports that extensive investigations on functional movements of the lower extremity muscles have not shown any causal relationship between exaggerated reflexes and movement disorder. The pattern of muscle activation and development of increased muscle tone in spasticity

appears to be different in the active motor condition compared with passive muscle function (Dietz et al. 1997). That being said, the EMG activity in weight bearing leg muscles during gait in persons with spasticity is smaller in amplitude and less well-modulated compared with that of persons without neurological impairments. This is attributed to the impaired function of polysynaptic reflexes (Dietz 1992; Dietz et al. 1997). The loss of supraspinal facilitative drive to the CPGs likely also contributes to the reduced EMG amplitude observed in persons with SCI.

Despite the reduced levels of EMG activity, increased muscle tone is reported during BWSTT in patients with complete and incomplete SCI (when compared to persons without neurological impairments) (see review in Dietz et al. 1997). This incongruent observation precludes the increased muscle tone from being explained by increased activity of motoneurons (Dietz et al. 1997). Instead, it is thought that the change in tension development in spastic paresis is due to a morphological transformation of the motor units with their muscle fibers (Dietz 1992; Dietz et al. 1997), the details of which will not be discussed here. These authors theorize that the alteration to a simpler regulation of muscle tension could be advantageous because it enables the person with paresis (due to spinal lesion) to support the body during gait and, consequently, to achieve mobility.

Cardiovascular Disease (CVD) and SCI

With improved survival in the SCI population, the causes of morbidity and mortality are approximating those of the able-bodied population (Groah and Menter 1998; Krum et al. 1992; Whiteneck et al. 1992). Preventative measures have reduced the

incidence of early death (ie. from genitourinary causes - renal failure) (DeVivo 1989; DeVivo et al. 1993; DeVivo and Stover 1995; Frankel et al. 1999; Maynard and Weingarden 1989; Whiteneck et al. 1992) and consequently, more persons with SCI are at risk for CVD, particularly as this population ages (Bergman et al. 1997).

The literature is made confusing by inconsistent use of terminology with respect to CVD, coronary artery disease (CAD), and coronary heart disease (CHD), but the main points are clear. Although the prevalence of CHD in the population of persons with SCI is not established with certainty (Bauman et al. 1999), epidemiological reports repeatedly identify CAD/CVD as a leading cause of morbidity and mortality (Bergman et al. 1997; DeVivo et al. 1993; DeVivo and Stover 1995; Frankel et al. 1999; see review in Kocina 1997; Le and Price 1982; Whiteneck et al. 1992; Yekutiel et al. 1989). Merging of all the data from the US National Database, the collaborative studies and the Social Security Administration (n=17 349) revealed that heart disease (ischemic and non-ischemic combined) was the second leading underlying cause of death among tetraplegics and the leading underlying cause of death among paraplegics (DeVivo and Stover 1995). In addition, it has been shown that CVDs were the leading cause of death in persons with longstanding SCI (over 30 years) and among those with SCI age 60 and over (Whiteneck et al. 1992). In a review of survival studies (of persons with SCI), Washburn and Figoni (1999) stated that adults with SCI are at increased risk for CV morbidity and mortality compared with able-bodied populations. However, this review failed to include the research of DeVivo et al. (1993) and DeVivo and Stover (1995). These two studies reported that only nonischemic heart disease, and not ischemic HD, was associated with a

significant increase in mortality risk in SCI persons. Thus, although it is unclear if CVD causes greater morbidity and mortality in those with SCI compared to the able-bodied population, it is clear that CVD is increasing in prevalence as the lifespan of SCI persons is extended.

The prevalence of CHD in individuals with SCI is not explained by higher levels of several established CVD risk factors such as blood pressure, cigarette smoking or total serum cholesterol (Krum et al. 1992; see review in Washburn and Figoni 1999). Rather, it is believed that persons with SCI are at increased risk for CAD because of a constellation of other factors. These include: reduced glucose tolerance, decreased HDL cholesterol (HDL-C) levels, increased body fat percentage, reduced exercise tolerance, the potential for silent ischemia due to altered sensation, reduced cardiac output (see review in Bergman et al. 1997), decreased lean body mass, increased paralyzed muscle mass (Washburn and Figoni 1999), physical inactivity (Brenes et al. 1986; Dearwater et al. 1986) unfavorable apolipoprotein profiles (see review in Dallmeijer et al. 1999) hyperinsulinemia and elevated serum uric acid levels (Zhong et al. 1995). Some of these risk factors are thought to be linked. The metabolic parameters, specifically disordered lipid and carbohydrate metabolism, may be related to the paralysis/decreased physical activity and associated loss of lean body tissue and gain in adiposity (Bauman et al. 1999; Washburn and Figoni 1999). A recent review broadened the already multifactorial etiology of CVD risk in SCI, suggesting that hemostatic and autonomic dysfunction contributed to the pathology (Bauman et al. 1999).

A hypothetical model of accelerated CVD progression has been developed that

integrates the metabolic and somatotypical changes that pervade the SCI population (Washburn and Figoni 1999).

SCI
↓
muscle paralysis and autonomic decentralization
↓
inability to voluntarily exercise a "large" muscle mass
↓
↓ physical activity
↓
↓ exercise tolerance - fitness & mobility
↓
change in body composition - ↑ fat mass & ↓ lean mass
↓
↑ insulin resistance & hyperinsulinemia
↓
↑ triglycerides
↓
↓ HDL-C
↓
↑ CHD risk
↓
↑ secondary CV disabilities
↓
↑ functional impairment
↓
further deconditioning & ↓ functional capacity

In this schema, reduced [HDL-C] is advanced as the surrogate marker of increased CVD risk. It should be noted however, that the array of changes that contribute to this dyslipidemic profile may also be independently associated with CVD risk.

The elevated insulin resistance in the SCI population is suggested to be due to a combination of the following factors: I) reduced muscle mass, which is the primary peripheral site of insulin action; II) a predominance of type IIb muscle fibers which are less

sensitive to insulin action; III) denervation and concomitant reductions in physical activity, which appear to be responsible for a) a post-receptor defect in insulin action, as well as b) the loss of contraction-stimulated glucose disposal; and IV) increased adiposity and the resulting decreased response of the peripheral tissue to insulin (see reviews in Bauman et al. 1999; Washburn and Figoni 1999).

The mechanism(s) underlying the inverse relationship between serum triglycerides (TG) and HDL-C, a relationship that has been observed in SCI (Bauman et al. 1998; Bauman et al. 1994; Bauman et al 1992; Janssen et al. 1997; Zhong et al. 1995) and is generally believed to exist (Bauman et al. 1999), has not been conclusively expounded. It has been proposed that hyperinsulinemia may cause increased hepatic TG production, which tends to lower serum HDL-C possibly by enhancing clearance mechanisms (see review in Bauman et al. 1998). An alternative explanation is that the lower activity levels of SCI persons reduces the activity of lipoprotein lipase (LPL) (Janssen et al. 1997). LPL is an endothelial enzyme (eg. in heart, adipose tissue and skeletal muscle) responsible for the delipidation of TG-rich chylomicrons and lipoproteins. LPL action also enhances the uptake of the released fatty acid into extrahepatic tissue (Durstine and Haskell 1994). The TG-poor lipoprotein remnants that result from LPL action play an important role in HDL-C metabolism (see reviews in Berg et al. 1994; Durstine and Haskell 1994). These remnants are transferred to HDL-C, which acts as a sink for them. There is a general consensus that exercise and LPL activity are positively correlated in able-bodied persons (see reviews in Berg et al. 1994; Durstine and Haskell 1994).

The evidence in support of the proposed dysmetabolic sequelae of SCI is very

strong. Two comprehensive reviews reported consistently depressed levels of HDL-C in the SCI population (Bauman et al. 1999; Washburn and Figoni 1999). With the exception of reports by Cardus et al. (1992b) and Janssen et al. (1997), all the cross-sectional studies that were reviewed showed significantly lower levels of HDL-C in SCI individuals when compared with able-bodied controls (Washburn and Figoni 1999). The difference in [HDL-C] between the two populations represents a potentially large increase in risk for CVD in SCI persons (Washburn and Figoni 1999). Comprehensive reviews have also found that impaired glucose tolerance, increased insulin resistance, and diabetes mellitus are more prevalent in individuals with SCI than in able-bodied persons (Bauman et al. 1999; Kocina 1997; Washburn and Figoni 1999). Again, there are exceptions to this trend (Dearwater et al. 1986; Yekutiel et al. 1989)

In a cross-sectional survey by LaPorte et al. (1983), [HDL-C] across different cohorts was positively related to the level of physical activity, with marathon runners having the highest values and those with acute SCI the lowest. The findings also suggested that within the SCI cohort, HDL-C profiles improved as the time post-SCI increased. This was attributed to the acquisition of new functional abilities and increased activity in recovery. Other inferential reports of activity-induced changes in [HDL-C] in the SCI population have been equivocal however. Using lesion level as a representative measure of participation in physical activity, it has not been consistently shown that there exists higher HDL-C levels in paraplegics (theoretically more active) when compared with tetraplegics (see review in Washburn and Figoni 1999).

Cross-sectional studies correlating [HDL-C] and *direct indices* of physical activity

in persons with SCI have generated more consistent results, and have established a basis for exercise programs for persons with SCI. It has been shown that active SCI persons have significantly higher levels of HDL-C compared with sedentary SCI persons (Brenes et al. 1986; Dallmeijer et al. 1997; Dearwater et al. 1986). Two of these studies (Brenes et al. 1986; Dearwater et al. 1986) also found that SCI athletes had concomitant lower total cholesterol (TC) (significant) and TG (non-significant) levels than the SCI sedentary group. It should be noted that differences in lesion level and ASIA classification between groups were not controlled for in either of these studies (Brenes et al. 1986; Dearwater et al. 1986). In the third study (Dallmeijer et al. 1997), all subjects had a SCI between C5-C8 and regression analysis found that completeness of the lesion did not significantly influence the lipoprotein variables. Although the results were non-significant, Krum et al. (1992) assessed physical activity on an ordinal 5 point scale (0=no exercise to 4=wheelchair athlete), and also found a positive correlation between [HDL-C] and reported exercise in SCI subjects. The only dissenting results have been reported by Janssen et al. (1997), who found that activity level did not add significantly to the explanation of variance of [HDL-C] in SCI subjects. Recall however, that this was also one of only two reports that found that HDL-C levels were not significantly different between the SCI cohort and able-bodied controls. Janssen et al. (1997) did report that [TC] and [LDL-C] were significantly inversely related to sport participation in long-standing SCI, while the [HDL]/[LDL] ratio was significantly and directly related to sport participation. Collectively, this research appears to demonstrate that the dyslipidemia, and specifically the low concentrations of total HDL-C observed in sedentary SCI persons, can be at least partially explained by

inactivity.

Cross-sectional studies investigating the association between cardiovascular fitness and blood lipids/lipoproteins in persons with SCI also suggest a role for exercise in this population. Bostom et al. (1991) found that there was a significant, inverse correlation between absolute maximal oxygen uptake (VO2 max) and [TC]/[HDL-C] levels in long-standing SCI subjects. Similar results were reported by Janssen et al. (1997), with the exception that the VO2 max was relative to body weight. Neither group of authors found a significant correlation between aerobic power and HDL-C levels. A direct relationship between aerobic power and [HDL-C] has been reported in a subsample of chronic SCI subjects, though the correlation failed to achieve statistical significance (Bauman et al. 1992). These findings have been challenged by Dallmeijer et al. (1999) who argued that the cross-sectional relationship between physical capacity parameters (used as a measure of training status) and lipid and (apo)lipoprotein profiles is confounded by differences in active muscle mass, as a result of differences in lesion level.

The only recent longitudinal training study to investigate the effects of exercise training on lipids in persons with SCI reported exercise-induced improvements in the blood lipid profile. Following 8 weeks of wheelchair ergometer exercise training, the moderate intensity group (70-80% of maximal HR reserve) showed a significant increase in [HDL-C] and significant decreases in [TC]/[HDL-C], [LDL-C] and [TG] (Hooker and Wells 1989). Total cholesterol concentration was also lower, but did not reach statistical significance. Unfortunately, conclusions were limited because of a small sample size, lack of dietary control and the absence of a control group. It should also be noted that although

the authors of this study suggested that 70-80% of HR reserve was moderate intensity exercise, other classification systems (Wilmore and Costill 1994b) consider this intensity (specifically the 75-80% range) to be heavy. The significance of this point is commented on in the discussion. A longitudinal evaluation of lipid, lipoprotein, and apolipoprotein profiles in the first two years post-SCI found that sport activity and improvements in physical capacity can positively influence risk profile parameters (Dallmeijer et al. 1999). Regression analyses showed that sport activity one year after discharge from rehabilitation and changes in physical capacity were two of the three most important determinants of changes in the lipid and (apo)lipoprotein profiles, even when other possible confounders were taken into account.

While the results are not definitive, the available data does suggest a potentially important role for physical activity to improve the lipid and (apo)lipoprotein profiles, including the HDL-C subfraction, in the SCI population. Considering that:

I) a general consensus exists with regard to the finding of depressed levels of HDL-C in SCI,

II) there exists an inverse association between [HDL-C] and risk of CAD/CHD (see reviews in Bauman et al. 1999; Bauman et al. 1992; Berg et al. 1994; Bostom et al. 1991; Cardus et al. 1992b; Durstine and Haskell 1994; Hardman 1996; Hartung 1995; Sagiv and Goldbourt 1994; Washburn and Figoni 1999), and

III) HDL-C levels are responsive to physical activity (see reviews in Berg et al. 1994; Durstine and Haskell 1994; Hardman 1996; Hartung 1995; Sagiv and Goldbourt 1994),

exercise prescription may offer an antidote to the hypothesized heightened CVD risk of this cohort.

Osteoporosis and SCI

Osteoporosis is conceptually defined as a disease characterized by low bone mass and microarchitectural deterioration of bone tissue, leading to enhanced bone fragility and a consequent increase in fracture risk (World Health Organization 1998). The operational definition of osteoporosis for Caucasian women is a value for bone mineral 2.5 SD or more below the young adult mean (t-score ≤ -2.5) (World Health Organization 1998). It is suggested that a similar absolute value for bone mineral density to that used in women can be taken as a cut-off point for the diagnosis of osteoporosis in men - that is, a value 2.5 SD below the average for adult premenopausal women (World Health Organization 1998).

There is unanimity within the literature that SCI causes a deleterious sequence of events to occur in sublesional bone. This sequence is characterized by increased bone remodelling in which the degradation and synthesis of bone are uncoupled (Chantraine et al. 1979a; Pietschmann et al. 1992; Sharp et al. 1995; Uebelhart et al. 1994). In the acute post-injury time period (~ first 2-4 weeks), osteoblastic activity is in the depressed-to-normal range, while osteoclastic activity is elevated (Pietschmann et al. 1992; Uebelhart et al. 1994). Subsequently, bone formation increases (Pietschmann et al. 1992; Uebelhart et al. 1994), but not to a level where it can compensate for the continued high rate of osteoclastic destruction (Chantraine et al. 1986). Consequently, bone mass decreases and osteopenia develops. Following the very active period of bone remodelling where degradation outpaces repair, there is an evolution towards a new equilibrium between resorption and synthesis. Thus, although steady state is eventually re-achieved, it is at an

osteoporotic level (Chantraine et al. 1986; Garland et al. 1992). The osteoblastic/clastic relationship in the acute post-injury period has been determined with the use of biochemical markers that are sensitive and specific:

>bone formation - serum osteocalcin;
>bone resorption - urinary pyridinium crosslinks of collagen (deoxypyridinoline) (Pietschmann et al. 1992; Uebelhart et al. 1994).

The use of these markers is advocated as they dissociate between the two opposite but complimentary processes, and they provide quantification of the dynamic osteoblastic and osteoclastic activity (Uebelhart et al. 1994).

Because of methodological variation between studies, dating the return of bone mass homeostasis post-SCI is difficult. Until recently, most authors agreed that the major bone loss occurred during the first 6 months after SCI and that blast/clast activity stabilized between 12-16 months (Garland et al. 1992; Geusens et al. 1992; see review in Uebelhart et al. 1995). A few research groups reported that there was a longer window of up to 2 years before steady state returned (Biering-Sorensen et al. 1990; Chantraine et al. 1986). The slower turnover of cortical bone compared with trabecular bone (Biering-Sorensen et al. 1990; Wilmet et al. 1995) explains why it takes longer to re-achieve steady state (ie. > 3 years) in areas such as the femoral shaft (Biering-Sorensen et al. 1990). Recent studies are now questioning the re-establishment of an equilibrium. Using biochemical markers of bone metabolism (serum collagen I telopeptide and osteocalcin), Sharp et al. (1995) found that bone catabolism and formation remained significantly elevated and diminished respectively, at 104 weeks post-injury. In a large cross-sectional study, Szollar et al. (1997a) found that dual-energy x-ray absorptiometry (DEXA) scans

of the hip did not reveal evidence of bone mass loss until after the first year post-injury, and following that, the decline was gradual with the lowest bone mass at 19 years following injury, regardless of age or level of injury. The authors also found that bone loss in the femoral region occurred most dramatically in young men. Considering the variation in results, it is fair to state that at present, the temporal pattern of skeletal changes in chronic SCI is not known definitively.

Quantification of post-SCI bone loss with dual photon absorptiometry (DPA) has shown a range of values that appear to be bone-specific. Biering-Sorensen et al. (1988; 1990) reported that new steady-state levels for bone mineral content (BMC) of the proximal tibia and femoral neck were 40-50% and 60-75% respectively of normal values in cross-sectional and longitudinal studies. Garland et al. (1992) reported that the bone mineral density (BMD) of the distal femur stabilized at 63% of the baseline (control) value in chronic SCI. Using DEXA measures, Geusens et al. (1992) noted a similar trend with a 30 % loss of BMD in the legs compared to a 15% loss of BMD in the femoral neck of males with chronic paraplegia. Although not steady-state values (ie. results were pooled from subjects 1 month to 34 years post-injury), Szollar et al. (1997a; 1997b) reported that BMD of the proximal femur in SCI persons (aged 20-59) was between 82-84% of able-bodied age-matched controls. The total body loss of BMC and BMD in chronic paraplegia, as assessed by DEXA, is suggested to approximate 12% (Geusens et al. 1992; Jones et al. 1998). In the latter reference (Jones et al. 1998), it should be noted that three of five subjects were less than 2 years post-injury, and thus, may not yet have attained a new osteo-equilibrium.

While there is no disputing that the pathological consequence of a SCI on bone metabolism is osteoporosis, the underlying etiology is not as clear. It is suggested that generic terms such as disuse or immobilization osteoporosis, which traditionally emphasize the lack of biomechanical stress in paralysis, do not reflect the multifactorial nature of the condition (Chantraine et al. 1979a; Garland et al. 1992; Uebelhart et al. 1995). Significant differences in the chronology and intensity of the bone metabolic changes between osteoporosis resulting from a SCI and experimental disuse osteoporosis (Bergmann et al. 1977-1978; Chantraine et al. 1979a; Chantraine et al. 1986; see review in Goemaere et al. 1994; Pietschmann et al. 1992; Uebelhart et al. 1995; Van Ouwenaller et al. 1989), support the theory that other factors are involved. The current thinking is that SCI-induced osteoporosis is caused by a loss of normal biomechanical stress associated with some degree of neurovascular changes, the latter due to altered autonomic nervous system (ANS) activity (see review in Kocina 1992; Saltzstein et al. 1992; Uebelhart et al. 1995). The neurovascular changes are explained as follows: the ANS lesion (including sympathectomy) induces vasomotor paralysis which slows intraosseous circulation and causes venous stasis and tissue acidosis (Chantraine 1978-1979; Chantraine et al. 1979b). Concurrently, the opening of arteriovenous shunts increases venous pressure and therefore, intramedullary pressure (Bergmann et al. 1977-1978; Chantraine 1978-1979; Chantraine et al. 1979b). These circulatory modifications have metabolic consequences where bone tissue is one of the primary targets (Uebelhart et al. 1995). The altered environment induces a cellular reaction which manifests itself as an imbalance between osteoclastic and osteoblastic activity (Chantraine 1978-1979).

It is generally accepted that bone remodelling in SCI osteoporosis is not hormone dependent (see review in Chantraine 1978-1979; Chantraine et al. 1979a; see review in Chantraine et al. 1986; see review in Claus-Walker and Halstead 1982; Pietschmann et al. 1992; Vaziri et al. 1994), and that any hormonal changes are likely secondary effects (see review in Chantraine 1978-1979; Chantraine et al. 1979a; see review in Garland et al. 1992; see review in Van Ouwenaller et al. 1989; Vaziri et al. 1994). The depressed levels of parathyroid hormone (PTH) and calcitriol (active vitamin D) that have been observed after acute SCI have now been reported to persist in long-standing SCI, despite the re-establishment of normal ionized [calcium] (Vaziri et al. 1994). It has been proposed that the chronic suppression of PTH and calcitriol is an indicator of low-grade net bone resorption continuing for many years after SCI (Vaziri et al. 1994).

The implications of a predominantly neurovascular pathogenesis on strategies to limit the osteoporotic process in SCI are not clear. It is theorized that while mechanical factors are important in maintaining bone mineralization and structure, their role is likely limited when there is disruption of the nerve supply to the bone (Garland et al. 1992; Van Ouwenaller et al. 1989). This hypothesis may explain why neither spasticity (Biering-Sorensen et al. 1988; Wilmet et al. 1995) nor weight bearing, either with the daily use of long leg braces (Biering-Sorensen et al. 1988) or a standing frame (Kunkel et al. 1993), significantly influenced the BMC/BMD in the acute (till 1 year) (Wilmet et al. 1995) or chronic post-injury period (Biering-Sorensen et al. 1988; Kunkel et al.1993). An alternative explanation for the lack of benefit (as assessed by BMC) following daily mobilization, is that the use of long leg braces does not give rise to much direct weight

bearing on the long bones (Biering-Sorensen et al. 1988).

Results that are contradictory to those above have been reported by Demirel et al. (1998) and Goemaere et al. (1994). Demirel et al. (1998) found a significantly higher BMD in those SCI persons with spasticity when compared with flaccid patients. This suggests that muscle activation may be protective against BMD loss. The latter reported that passive weight-bearing standing with a device (long leg braces, standing frame or standing wheelchair) during the first year post-injury, significantly preserved BMD at the femoral shaft in persons with complete SCI. Interestingly, the authors also found that BMD was not preserved at the proximal femur in the same subjects. This is in agreement with the work of Kunkel et al. (1993) (reviewed above), who reported no change in BMD of the femoral neck following 5-6 months of standing frame "standing". Goemaere et al. (1994) concluded that passive mechanical loading can have a beneficial effect on the preservation of bone mass in osteoporosis-prone paraplegics. To account for the site-specific effect of weight bearing, two theories have been suggested (see review in Goemaere et al. 1994): I) possibly, the transmission of force through trabecular and cortical bone differs, so that the minimum effective strain for initiating bone remodelling is reached more rapidly in cortical bone; and II) perhaps there exists different strain thresholds to control bone modelling and remodelling.

Support for post-SCI mobilization also stems from the work of Kaplan et al. (1978) who found that ambulation, especially when initiated early post injury (ie. < 3 months), significantly decreased hypercalciuria and modified the calcium balance in a positive direction. Unfortunately, no description of the type or duration of ambulation was

provided. In a subsequent study, Kaplan et al. (1981) reported that tilt table exercises significantly reduced urinary calcium output and improved calcium metabolic balance in early (<6 months) and late (between 12-18 months) post-SCI periods. Again, the authors failed to report the frequency and duration of the "standing" program. Nevertheless, these findings indirectly suggest that early ambulation and weight-bearing may prevent bone loss.

A cross-sectional study of persons with chronic complete and incomplete SCI found a strong positive correlation between mobility and bone density of the distal tibia (Saltzstein et al. 1992). Mobility was gauged with a mobility index; the parameters ranged from 1, for complete immobility, to 9, for full mobility of the uninjured control population. Although the authors conceded that the results may have been due to differences in intra-osseous circulation (ie. ANS (dys)function) between completes and incompletes, it was argued that this was further evidence to support ambulatory training. Although 'verticality' has had both physiological and psychological (Gallien et al. 1995; Kunkel et al. 1993) benefits for persons with SCI, caution must be exercised with this regimen given the reduced BMD at important weight bearing sites in this population (Jones et al. 1998).

In summary, a review of the literature does not provide consistent evidence on the effects of musculoskeletal activation as it pertains to the osteoporotic process in acute and chronic SCI.

Summary

In a recent review of neurorehabilitation approaches (BWS, functional electrical stimulation (FES), and pharmacological), Barbeau et al. (1998) found that only BWS-assisted locomotor training had a greater effect on locomotor recovery than conventional or locomotor training alone in neurological populations. The effectiveness of BWSTT is suggested here to reflect the compatibility between the neuronal organization of locomotion in humans (including those with SCI), and the patterns of sensory stimuli ("afferent experience") generated with this form of training. In persons with SCI, the profound loss of descending excitatory pathways from the brainstem makes it difficult to voluntarily excite the proportion of each of the motoneuron pools required for stepping. This renders the lumbosacral CPGs inactive, although the CPGs retain the ability to interpret the sensory information associated with locomotion. When the CPGs are exposed to recognizable sensory information associated with locomotion (as in BWSTT), they can respond with patterned, basic locomotor synergy in which each muscle displays its own characteristics during the stepping cycle (see review in Dimitrijevic et al. 1998).

By providing an appropriate degree of unloading, the BWS apparatus generates the conditions in which the person with SCI may experience appropriate, recognizable afferent feedback during assisted ambulation. As is the case for chronic spinal cats, the specific manner of loading and the control of other sources of sensory inflow are crucial to the success of weight-bearing stepping (see review in Harkema et al. 1997). The moving treadmill for instance, encourages hip extension which is an afferent trigger to initiate the swing phase of gait (Calancie et al. 1994; Dobkin et al. 1995; see review in Pearson et al.

1998; Visintin and Barbeau 1989; Wernig et al. 1992). With BWSTT, the three components of gait (weight bearing, balance and stepping) can be retrained simultaneously under dynamic conditions (Visintin and Barbeau 1989). The unloading of body weight also enables gait retraining to be initiated soon after SCI at a time when patients lack balance and lower extremity strength, and when there is the most plasticity and potential to influence the recovery of mobility (Barbeau et al. 1998).

In persons with SCI, the recovery of locomotion with BWSTT is influenced by several factors. The cause, extent, and location of the central nervous system (CNS) lesion, as well as the chronicity of the injury can have an impact on the locomotor potential and on the degree of plasticity or adaptability within the neuromuscular system (Barbeau et al. 1998). The extent of the injury can be described with the ASIA Impairment Scale (see below), a modification of the Frankel Classification (Yarkony et al. 1997).

ASIA		Definition
A	complete	No sensory or motor function is preserved in the sacral segments S4-S5
B	incomplete	Sensory but not motor function is preserved below the neurologic level and extends through the sacral segments S4-S5
C	incomplete	Motor function is preserved below the neurologic level, and the majority of key muscles below the neurologic level have a muscle grade < 3
D	incomplete	Motor function is preserved below the neurologic level, and the majority of key muscles below the neurologic level have a muscle grade > or equal to 3
E	normal	Sensory and motor function is normal

Table 1: The American Spinal Injury Association Impairment Scale (Yarkony et al. 1997)

It has been demonstrated that the ASIA A (complete) designation is associated with a poorer prognosis for locomotor recovery than the ASIA B, C, and D (incomplete)

classifications (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; see review in Formal et al. 1997; Wernig et al. 1995). The ability to wean off the BWS apparatus appears to be limited to the patients with incomplete injuries, as does the ability to generate unsupported stepping movements on a stationary surface (Dietz et al. 1994; Dietz et al. 1995). These findings have led to the conclusion that apart from the positive effects on the cardiovascular and musculoskeletal systems, only patients with incomplete SCI appear to profit directly from locomotor training (Dietz et al. 1995). The assumed coincidental health benefits referred to by Dietz et al. (1995) have been virtually ignored in BWSTT research to date. Considering the rigor of some of the training protocols that have been used:

>Dietz et al (1995): 5 months, daily, 300m/day;
>Wernig et al. (1995): 10.5 weeks, 5 sessions/week, 30 minutes/session,

and the potentially modifiable medical risk factors inherent in SCI (reduced HDL-C and reduced BMD for example), the health benefits of BWSTT in this cohort merit investigation.

Purpose

The purpose of this study was to determine if BWSTT offered health related benefits for persons with SCI in addition to its role in locomotor recovery. Because the cardiovascular and skeletal systems are deleteriously affected by SCI, and both would seemingly benefit from weight-bearing exercise of this sort, these two systems served as foci for the investigation. It has been previously documented that the dyslipidemic profile (Brenes et al. 1986; Dallmeijer et al. 1997; Dallmeijer et al. 1999; Dearwater et al. 1986; Hooker and Wells 1989) and loss of BMD (Goemaere et al. 1994; Kaplan et al. 1978; Kaplan et al. 1981; Kunkel et al. 1993) in persons with SCI are modifiable with exercise and weight-bearing activity respectively. To our knowledge however, BWSTT has not been part of the training routine or activity history in any study looking at these outcomes in this population.

BWSTT is advanced as the leading tool for locomotor rehabilitation in the neurological population (Barbeau et al. 1998). In theory, BWSTT is also ideal for modifying some of the risk factors for CVD and osteoporosis in persons with SCI. By reducing the weight bearing demands of standing, BWSTT allows SCI persons to rise from their wheelchair and ambulate, with or without assistance. Across an intense 3 month BWSTT schedule, advances can be expected in ambulatory capacity (ie. reduced assistance, increased duration of ambulation intervals, increased treadmill speed, decreased BWS, etc.) (Barbeau et al. 1993; Dietz et al. 1994; Ladouceur et al. 1993; Wernig et al.

1992). As ambulatory capacity improves, the ability to stress the cardiovascular and musculoskeletal systems will also improve. It is hypothesized that the exercise and weight bearing stimuli of a 3 month BWSTT program will be sufficient to affect risk factors for CVD (specifically blood lipid profiles) and the osteoporotic process.

Hypotheses

Three months (36 sessions) of BWSTT in persons with chronic, incomplete SCI will result in:

I)improved ambulatory capacity
- a)decreased BWS requirements
- b)decreased assistance from the trainers
- c)increased endurance on the treadmill
- d)increase in maximum tolerable treadmill velocity
- e)decrease in inappropriate muscle tone (ie. spasticity)

II)improvements in the blood lipid/lipoprotein profile; specifically
- a)increased plasma [HDL-C]

III)modification of bone metabolism
- a)increase in bone turnover as evidenced by biochemical markers of osteoclastic and osteoblastic activity
- b)increase in BMD of weight bearing bones (likely limited by the length of the training period)

Methods

Subjects

Five individuals with incomplete SCI were recruited to participate in this study. The subjects presented a range in age (25-44 years), neurological lesion level (C2/C3 - T7/T8), time post-injury (19 months - 11 years) and ASIA classification (C-D). Although four of five subjects were diagnosed with ASIA C impairment, there was a range of muscle strength grades within this bracket. Grades of muscle strength were extracted from the most recent neurological assessment in each subject's medical chart. A pre-training profile of each subject is found in Table 2; from the top of the table, the subjects are listed in descending order of lower extremity muscle strength grades. Physical trauma was the cause of SCI in four of the five subjects (I, II, IV and V) with a vascular accident causing SCI in the other (III). Any reference to the subjects will not distinguish between the causes of spinal cord damage; all subjects will simply be classified as having a SCI.

The inclusion and exclusion criteria were formulated with the intention of recruiting a heterogeneous population of individuals with SCI. It was hoped that a diverse cohort would reveal those factors that predict a favourable response (in terms of bone metabolism and lipid/lipoprotein profile) to BWSTT.

Individuals were considered potential candidates if they had a chronic (>1 year post-injury), incomplete SCI (ASIA classification of B or higher), and normal respiratory

function. Incomplete injuries, encompassing the ASIA B, C, and D subgroups (Ditunno Jr et al. 1994), were preferentially selected because of greater success with locomotor retraining and specifically BWS training in this cohort as compared to individuals with complete (ASIA A) SCI (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; see review in Formal et al. 1997; Wernig et al. 1995).

Candidates were excluded on the basis of any of the following criteria: a history of heterotopic ossification, CAD, hypertension, diabetes mellitus or post-injury fracture. Use of lipid-modulating medication and an inability to tolerate 12 minutes on a vertical tilt table also served as reasons for exclusion. Eighteen and fifty years of age were set as the lower and upper limits for participation in the study. Because of the commitment that subjects were required to make, physicians and physiotherapists familiar with potential subjects were consulted to determine their reliability. This information was considered in the recruitment.

Subjects were made aware of the potential risks associated with the training program and all signed an informed consent document. This study was approved by the Research Ethics Board at McMaster University.

Subject	Age	Gender	Neurological Lesion Level	ASIA Classification	Time Post-Injury	Medications	Pre-Training Mobility
I	34	M	C5-C6	D	30 months	baclofen, ciprofloxacin*	unilateral cane (R hand)
II	38	M	C2-C3	C	34 months	baclofen, dantrolene sodium, senokot	wheelchair
III	44	F	T7-T8	C	21 months	baclofen, desipramine, gabapentin@, ibuprofen, oxybutynin-chloride, nitrofurantoins	wheelchair
IV	26	M	C4-C5	C	19 months	baclofen, carbamazepine divalproex sodium, docusate sodium	wheelchair
V	25	F	C4-C5	C	11 years	4AP	wheelchair

Table 2: Subject profiles

** - only for post-training testing*
@ - only for pre-training testing and first 7 weeks of training
$ - only for pre-training testing

Dependent Variables

The protocol for the collection of urine and blood samples and the laboratory assessment of the dependent variables was consistent across pre- and post-training. Post-training samples were collected 24-48 hours after completing the final training session; post-training DEXA scans were also performed at this time.

The Cholestech LDX system was used to assess plasma lipid, lipoprotein and glucose levels. Subjects were required to fast for 12 hours prior to giving a fingerstick blood sample. The procedures recommended within the Cholestech LDX manual for obtaining and analyzing blood samples were strictly followed.

Bone turnover was quantified with measures of serum osteocalcin and urinary

deoxypyridinoline. Osteocalcin or BGP (bone gla protein) is found exclusively in bone tissue and dentine (Garnero and Delmas 1998). It is an extrahepatic, vitamin K-dependent protein with a molecular weight of 5800 u that is produced by osteoblasts (NovoCalcin assay kit). The in vivo function of osteocalcin in bone metabolism has not been established (Delmas 1993; Kent 1997; NovoCalcin assay kit). Comparisons of the serum osteocalcin level with iliac crest histomorphology and calcium kinetics data have shown that, in most conditions, serum osteocalcin is a valid marker of bone turnover when resorption and formation are coupled and is a specific marker of bone formation when formation and resorption are uncoupled (see review in Garnero and Delmas 1998).

Pyridinium crosslinks (pyridinoline and deoxypyridinoline) provide rigidity and strength to the mature type I collagen in bone matrix (Pyrilinks-D assay kit). During osteoclast mediated bone resorption, pyridinium crosslinks are released from the bone matrix (Delmas 1993; Khosla and Kleerekoper). Of the two nonreducible markers, deoxypyridinoline has greater specificity (for the resorption of bone) because it is found in appreciable quantities only in bone collagen (Delmas 1993; Kent 1997; Khosla and Kleerekoper). This is compared with pyridinoline which is also present to some extent in type II collagen of cartilage and other connective tissues (Khosla and Kleerekoper). Following its release into the circulation, deoxypyridinoline is excreted unmetabolized in urine (Delmas 1993; Pyrilinks-D assay kit).

Concentrations of osteocalcin and deoxypyridinoline were measured with NovoCalcin and Pyrilinks-D kits from Metra Biosystems. For the former, a small sample of blood, separate from that used for the lipid assessment, was obtained via venipuncture.

This sample was allowed to clot and it was then separated by centrifuge and the serum was extracted and subsequently analyzed for osteocalcin. The time of day when the blood sample was drawn was consistent (within subjects only) from pre- to post-training and was always after 12:00pm. For the measurement of deoxypyridinoline, urine samples were taken from the first urination of the morning, except in subjects III and IV who provided afternoon samples for both the pre- and post-training testing. To correct for variations in urine concentration, the results obtained from the Pyrilinks-D assay were expressed per mmol creatinine. For each serum and urine sample, the appropriate assay was run in quadruplicate and the mean value from the four trials was used in the statistical analysis.

Dual energy x-ray absorptiometry (DEXA) scans were used to assess the BMD of the subjects pre- and post-training. The DEXA protocol is well tolerated by the SCI population (Jones et al. 1998), and despite concerns raised by Kocina (1997) and Nord (1998) with respect to soft tissue measurements, DEXA results are also reported to be accurate in the SCI cohort (Jones et al. 1998). In addition to the whole body scan with the Hologic QDR 4500, the BMD of the left and right hips (total hip area) and the lumbar spine (L1-L4) were isolated. The total hip region was selected to interpret femur BMD in accordance with the recent recommendations of the International Committee for Standards in Bone Measurements (ICSBM) (see review in Blake and Fogelman 1998). Total hip BMD is the area weighted mean BMD for the femoral neck, trochanter, and intertrochanteric sites (Blake and Fogelman 1998). Initial evidence in postmenopausal women suggests that the total hip site is as responsive to change as the femoral neck area and has the advantage of better precision (see review in Blake and Fogelman 1998).

Ambulatory progress was closely monitored and data from each training session was recorded in a log. The foci of interest were changes in BWS requirements, treadmill velocity, duration of training intervals, manual assistance with the gait cycle, independent stepping, muscle tone (spasticity), and tendon reflexes (clonus). At approximately every fourth session, a video recording was taken of the locomotor pattern.

Body Weight Support Treadmill Training (BWSTT) Apparatus

The BWS treadmill used in this investigation was the Loko S 55 Spezial developed by Woodway. The speed of the treadmill ranged from a minimum of 0.1 km/hr to a maximum of 5.0 km/hr and was adjustable in increments of 0.1 km/hr. Bars of adjustable height were positioned along the sides and in front of the treadmill. Although these bars could be used by the subjects for balance and support, this practice (especially support) was discouraged. In some cases, this was accomplished by raising the bars to a height that made it difficult for subjects to bear weight through their arms. The treadmill itself was raised approximately 35cm above the ground. This allowed the trainers to sit at the sides of the treadmill and manually adjust the gait pattern of the subjects as required. A slightly graded ramp at the back of the treadmill allowed the subjects to wheel up and onto the treadmill belt which was sufficiently wide to accommodate a wheelchair. The harness worn by each subject had four inter-connected components: I&II) two padded straps, one for each inner thigh, that coursed anteroposteriorly across the perineum; III) a padded gluteal belt that supported the gluteal region posteriorly; and IV) a padded abdominal belt/binder that completely surrounded the torso at the level of the inferior costal margin.

The perineal straps connected anteriorly with the abdominal belt and posteriorly with the gluteal belt. The gluteal belt was connected to the abdominal belt anteriorly with buckles and posteriorly with Velcro straps. All components of the harness were adjustable, thus ensuring a tight and "comfortable" fit for the subjects. Two additional straps, originating on the left and right sides of the harness, ascended across the shoulders of the subject to link the harness with the suspensory system above.

BWS was provided with adjustable weight stacks mounted bilaterally on the treadmill. Cables ran from the weight stacks, through pulley systems above the treadmill, and were fastened onto the straps of the harness. *Theoretically*, the BWS provided through the harness was equivalent to the sum of the settings on the two weight stacks. The support was adjustable in increments of 8kg (4kg per stack). A maximum of 80 kg could be supported using the weight stacks. Previous experience with the BWS treadmill had revealed that there were unexplainable inconsistencies between the supposed BWS provided by the stacks and the actual BWS as determined by placing a scale beneath the subject. Discrepancies were also evident in this study and a consistent relationship between the two values could not be identified. Thus, in this study, the *actual* amount of weight that a subject was supporting through the legs at a given level of BWS was determined by placing an electronic scale on the surface of the treadmill. It is these scale values that were used in the calculations of %BWS.

As a precaution, the harness was also connected to two additional overhead cables that were capable of suspending all of the subject's weight. During normal treadmill training these safety ropes were slack and did not 'remove' any of the subject's body

weight. However, if the subject moved too far back on the treadmill or started to buckle under his or her own weight, the slack was removed from the system and the ropes became taut.

Training Program

All subjects participated in a training program consisting of 36 sessions over a 12 week period. When a session was cancelled, an attempt was made to reschedule it within the same week so as to maintain a frequency of 3 sessions/week. When this was not feasible, the session was added on to the end of the training program. The goal for each training session was to have the subject exercise on the treadmill for a total of 40 minutes. Although subjects were encouraged to complete the 40 minutes consecutively, this was not a requirement. At the onset of training, an interval approach was most successful with exercise bouts alternating with periods of recovery. Gradually, the intervals of treadmill training increased in length and the rest periods became shorter and fewer in number, until subjects were capable of completing 40 minutes of exercise without interruption. Although a total of 40 minutes of treadmill training was set as the target, pain, exhaustion, headache and inappropriate blood pressure responses were all sufficient reasons to terminate an exercise session prematurely. Sessions were considered valid if they contained a minimum of 20 minutes of treadmill training. Alternatively, subjects were not discouraged from surpassing the 40 minute mark if they felt strong and had a normal blood pressure response to the exercise stimulus.

To conform with previous BWSTT studies that used ~40% BWS as the starting

value for subjects with incomplete SCI (Dietz et al. 1994; Wernig and Muller 1992; Wernig et al. 1995), the investigators in this study established a target range of 25-50% BWS for the first training session. A target range of BWS was used (instead of a fixed value for all subjects) to accommodate the heterogeneous subject pool in this trial. A previous study that showed that BWS of 40% elicited a more biomechanically appropriate gait cycle in chronic, incomplete SCI subjects when compared with full weight bearing (Visintin and Barbeau 1989), also influenced the target range of BWS proposed for this trial. Based on the target range, subjects were provided with a minimum of 25% BWS to begin the first training session even if they were able to remain vertical on the treadmill with less BWS. If however, subjects required greater than 50% BWS in order to stand, the upper limit of the target range was ignored and support was provided as necessary to allow training to proceed.

The target range proved too narrow, and the amount of BWS that was provided at the beginning of the training program (i.e. session 1) varied from 22% to 69% depending on the subject. The range of values can be partially ascribed to inter-subject differences in pre-training motor capabilities. The upper limit of the target range was exceeded in two subjects (IV and V) in order to prevent uncontrollable buckling at the knee and hip joints during stance.

As the subjects progressed with the treadmill training, BWS was adjusted regularly based on the following general principle: the minimum amount of BWS that allows the subject to stand upright on the treadmill without buckling at the knees or hips (with arms removed from the support bars) will be provided. Other factors that influenced the amount

of BWS provided were lower extremity pain, fatigue, lack of progress, and regressive changes in the gait cycle; BWS was increased in all four situations. When a subject experienced unaccustomed musculoskeletal pain, weakness, or arthralgia in the lower limbs, BWS was temporarily increased to reduce the strain. When independent stepping was not forthcoming, BWS was also increased with the hope that reducing the weight on the lower limbs would facilitate voluntary movement.

Manual assistance with the locomotor pattern was provided as needed by trainers during BWSTT. The trainers were all MSc. candidates in the department of Human Biodynamics at McMaster University. There was a team of 3 regular trainers and 2 substitutes. All were educated of the desired gait pattern and were comfortable dealing with clonus, hypertonia (spasticity), and weakness in the lower limbs. Three trainers were present and provided assistance at the initial training sessions. As the subjects weaned off the assistance, the number of trainers was reduced accordingly. The trainers provided help with the gait cycle of the legs (lifting +/- directional guidance +/- support during stance), pelvic rotation, weight transfer, stabilization of the upper body, balance, and the timing of the arm swing. Subjects were continuously encouraged to do as much work on their own, and to not rely on the assistance of the trainers. A mirror was positioned directly in front of the treadmill to encourage subjects to look forward as opposed to down at their feet while 'ambulating'. As an aside, the term ambulation is used in this document to refer to the gait pattern that results from the combined efforts of the subject, the trainers, and the weight stacks; it is not synonymous with walking, which is used herein to describe completely independent gait (i.e. without external help from trainers and without BWS).

Treadmill velocity was gradually increased within and across sessions to the maximum value that would sustain a coordinated gait cycle, either unassisted or assisted. The dynamics of this progression were subject-specific. In the training portion of this study, treadmill velocity was deemed to be more important than independent stepping, and for some subjects, the latter was sacrificed in order to increase the velocity. This hierarchy was established in an attempt to attain a training stimulus that was sufficient to exact changes in lipid/lipoprotein metabolism. Although independent stepping (which in some cases was only forthcoming at very slow velocities) was sometimes sacrificed for higher velocity, the subjects' contributions to the ambulatory effort were never compromised. In addition, velocity never took priority over smooth, coordinated ambulation. Recall that it is the latter outcome that generates the recognizable afferent signals that are thought to underlie the success of BWSTT. Despite the focus on high velocity training, some slow velocity intervals were included in the training program of each subject. The slower velocity gave the subjects more time to make corrections in their gait cycle based on the afferent feedback they were receiving, and allowed greater independence in ambulation. These intervals also gave the trainers the opportunity to accurately assess each subject's contribution to the ambulatory effort.

At the end of each training interval while the subject was still standing, heart rate and brachial blood pressure were measured with an automatic blood pressure cuff. Blood pressure was monitored out of concern for autonomic dysreflexia and postural hypotension. Heart rate was recorded for interest sake as an indicator of exercise intensity (in those subjects with residual functional sympathetic cardiac innervation). Statistics were

not performed on either variable. During the rest intervals, subjects were seated and provided with water and/or Gatorade.

Overground walking was substituted for treadmill training when subjects no longer required BWS and manual assistance from the trainers. To progress to overground walking, subjects must also have demonstrated adequate balance during treadmill sessions. Overground walking sessions took place on a 400m track. Distances and times were recorded and speed was subsequently calculated.

Statistical Analysis

Using Statistica, dependent samples (correlated samples) t-tests were performed on the pre- and post-training values of all outcome variables. Significance was set at $p<0.05$.

Results

Note that all of the figures are found at the end of the results section.

Bone Metabolism

The three month program of BWSTT appeared to modify bone metabolism in the SCI subjects. Urinary deoxypyridinoline was significantly increased ($p<0.05$) following the training period (Figure 2), and there was a non-significant trend for serum osteocalcin to be increased ($p=0.167$) (Figure 1). DEXA scans did not reveal significant changes in total body BMD, nor in BMD of the lumbar spine, right hip or left hip (Figure 3). There was, however, a trend for reduced BMD of both the left and right total hip regions ($p=0.061$ & $p=0.186$ respectively).

Subject	Deoxypyr Pre	Deoxypyr Post	Osteocal Pre	Osteocal Post	Lumbar Spine BMD Pre	Lumbar Spine BMD Post	R Hip Total BMD Pre	R Hip Total BMD Post	L Hip Total BMD Pre	L Hip Total BMD Post
I	3.91	4.40	7.69	8.66	1.158	1.177	0.944	0.926	1.104	1.052
II	7.00	11.57	8.04	7.79	0.960	0.966	0.700	0.691	0.724	0.710
III	7.06	9.79	4.69	5.72	0.979	0.977	0.730	0.721	0.821	0.790
IV	9.05	11.40	10.78	12.44	0.936	0.932	0.620	0.618	0.636	0.621
V	9.96	12.48	9.49	9.26	1.104	1.075	0.724	0.730	0.674	0.673
Mean +- SD	7.40 +- 2.33	9.93 +- 3.24	8.14 +- 2.29	8.77 +- 2.45	1.027 +- 0.098	1.025 +- 0.100	0.744 +- 0.120	0.737 +- 0.114	0.792 +- 0.188	0.769 +- 0.170

Table 3: Biochemical markers of bone turnover (deoxypyridinoline - nmol/mmol creatinine; osteocalcin - ng/mL) and DEXA measures of BMD (g/cm2)

Blood Lipids, Lipoproteins, and Glucose

Total plasma cholesterol concentrations were significantly lower post-training ($p<0.05$) (Figure 4). The response of all other lipid and lipoprotein measures ([total cholesterol]/[HDL-C] ratio, [HDL-C], [LDL-C], [VLDL-C] and [TG]) to the training protocol was highly variable across the subject pool (Figure 4).

There was a trend for fasting plasma glucose concentrations to be lower in all subjects following the BWSTT program ($p=0.052$).

Subject	TG Pre	TG Post	TC Pre	TC Post	HDL Pre	HDL Post	LDL Pre	LDL Post	VLDL Pre	VLDL Post	TC/ HDL Pre	TC/ HDL Post	GLU Pre	GLU Post
I	0.63	0.90	3.63	3.55	0.98	0.98	2.36	2.16	0.29	0.41	3.70	3.65	4.64	4.46
II	1.19	0.94	2.67	<2.59	1.31	1.16	0.81	N/A	0.55	0.43	2.00	N/A	5.25	5.10
III	0.87	1.01	4.92	4.73	1.37	0.81	3.15	3.45	0.40	0.46	3.60	5.83	4.81	4.27
IV	1.14	0.89	2.83	2.61	1.30	1.32	1.01	0.89	0.52	0.41	2.20	2.00	4.65	4.57
V	1.15	0.97	2.81	2.59	0.95	0.83	1.33	1.32	0.53	0.45	3.00	3.10	4.74	4.58
Mean +- SD	1.00 +- 0.24	0.94 +- 0.05	3.37 +- 0.94	3.21 +- 0.94	1.18 +- 0.20	1.02 +- 0.22	1.96 +- 0.98	1.96 +- 1.13	0.46 +- 0.11	0.43 +- 0.02	3.13 +- 0.69	3.65 +- 1.61	4.82 +- 0.25	4.60 +- 0.31

Table 4: Lipid, lipoprotein, and glucose profiles (mmol/L)

Ambulatory Capacity

Across the 36 BWSTT sessions, there were marked improvements in the ambulatory capacity of each of the 5 SCI subjects. Some gains were observed in all subjects, while other gains were less pervasive and limited to a group of subjects or one individual. The timing of the improvements also varied between subjects. The individualized responses to the training protocol reflect the heterogeneity of the subject pool. In retrospect, pre-training ASIA classifications and muscle grading scores (taken

from medical charts) were effective predictors of training progress; in general, the greater the baseline leg muscle strength, the more quickly and completely BWS and manual assistance were reduced.

Figures 5-9 detail the ambulatory progress of the subjects. The data is presented in a case study format.

Endurance

By the end of the training program, all subjects had improved their endurance on the treadmill. Each subject was capable of ambulating for a minimum of 40 consecutive minutes. This was accomplished at levels of BWS and manual assistance that were equivalent to, or less than that used during the first session. For comparison, no subject had a training interval longer than 15 minutes in their first two sessions.

BWS Requirements

With training, all subjects were able to reduce the level of BWS that they required to remain vertical during treadmill ambulation. This progress was more consistent and profound in the ASIA D (I) and C subjects (II, III) with greater baseline leg strength. These three subjects were able to wean off BWS and bear 100% of their own weight during treadmill walking. This was accomplished in the fifth (I), ninth (II), and tenth (III) weeks respectively. In the ASIA C subjects with lower baseline leg muscle strength (IV & V), the reductions in BWS reached a plateau at ~ 31% and ~ 41% BWS respectively. Further decreases in BWS in these subjects resulted in a greater reliance on the arm bars, and an increased workload for the trainers (i.e. maintaining knee extension during stance).

In fact, for subjects IV and V, BWS was increased towards the end of the training program as the training team felt that lower levels of BWS were compromising all other aspects of ambulatory progress and potentially limiting changes in blood lipids and bone metabolism. In Figures 5 through 9, the % BWS values are the distance weighted mean values for each week (ie. to calculate weekly means for each subject, the different levels of BWS that were used were weighted according to how much distance was covered at that level of BWS).

Treadmill Velocity

For all subjects, there was an increase in the maximum treadmill velocity that could be tolerated without compromising the baseline (ie. session #1) gait pattern. At levels of BWS and manual assistance that were equivalent to, or less than baseline, all subjects progressed to velocities greater than 2.2 km/hr. In addition, three of the five subjects (I, II, V) were competent at velocities greater than or equal to 3.3 km/hr. Generally, the improvements in velocity parallelled the reductions in BWS. Thus, peak velocities were observed close to, or at minimum levels of BWS; one exception to this trend is discussed below*. The relationship between velocity and manual assistance was not as consistent. For three of the subjects (II, III, V), high treadmill velocities were not compatible with self-initiated, unassisted stepping movements. Although the higher velocity did not necessitate an increase in manual assistance compared with baseline in these subjects, it did not permit the independent stepping that had developed at lower velocities since the beginning of the BWSTT program.

*In the case of subject III, ambulation without BWS became an achievable goal as training progressed. However, it was only achievable at treadmill velocities which were lower than those in previous sessions when there had been greater BWS. Eliminating BWS was deemed to be the most important outcome in this case, and therefore, velocity and distance measures suffered slightly from sessions 29-36. This is reflected in Figure 7.

Required Assistance / Independent Stepping

Qualitative improvements in the gait patterns were observed by the trainers who facilitated, only as necessary, the locomotor movements of the subjects. Over the course of 12 weeks, all subjects became capable of independently initiating stepping movements on the treadmill. This includes the two ASIA C subjects (IV & V) who, at baseline, required significant manual assistance with all aspects of the gait cycle. With appropriate unloading (no greater than baseline), these two ASIA C subjects were able to generate sufficient power to independently toe-off and initiate the swing-through phase of walking. Independent stepping in these two subjects was erratic and limited to a few consecutive steps; following these sequences, there was continued force generation in the legs, but it was insufficient to allow unassisted stepping. For one of the subjects (V), the self-initiated toe-off and swing-through were predominantly unilateral phenomena. In these two subjects (IV & V), it cannot be reported that independent walking on the treadmill was achieved with the BWSTT program.

By the eleventh week, the other three subjects (2 ASIA C's with higher baseline muscle strength grades and the ASIA D) were capable of unassisted and unsupported (0%

BWS) walking on the treadmill. Subjects I & II could walk freely for the majority of the session, while subject III was successful only at low velocities (0.5 km/hr) and for a maximum of three minutes.

Overground Walking

Overground walking was substituted for some of the treadmill walking sessions in the training program of one subject (I) beginning at the tenth week. The walking was performed without any manual assistance from the trainers. Initially, a cane was used for balance, but this aid was gradually withdrawn and the subject was able to complete a full lap of the 400m track without the cane at the beginning of the 11th week. Although the time spent walking per session was not compromised with the integration of overground training into the protocol, velocity and thus distance were slightly reduced. The overground gait profile of the subject was qualitatively very similar to that observed during treadmill training.

Other qualitative changes in the ambulatory patterns that were observed by the trainers are summarized in Table 5. Also included in the table are the subject's self-reported daily living changes that were noted during the training period.

Subject	Ambulatory Changes	Daily Living Changes
I	-increase in active knee flexion during swing -increase in clearance of feet during unassisted swing through -increase in active eversion of R foot -reduction in compensatory abduction of L hip during swing through -improved upper body balance during hands-free walking -increase in active dorsiflexion of R and L feet allowing heel strike -improved arm swing	-easier to rise out of a chair or from the floor -easier to pick up objects from the floor -easier to climb stairs -better swimmer -improved balance -confidence to walk without cane
II	-improved coordination of arm swing -increase in active knee and hip flexion -increase in clearance of feet during unassisted swing through -improved control of knee extension during stance (not snapping into extension) -decreased tendency for legs to scissor during unassisted walking -unassisted heel strike with L; assisted heel strike with R	-improved control of swing through (of legs) when walking behind a shopping cart -easier to rise out of a chair
III	-decreased tendency to buckle at the knee during stance (R & L) -improved control of L leg during unassisted swing through; unassisted heel strike with L -improved trunk control during ambulation (ie. lumbar spine is less hyperlordotic) -improved timing of swing/stance phases in unassisted walking -reduced clonus and hypertonicity of leg muscles	-increase in leg strength -decreased swelling in the legs -decrease in leg muscle spasticity -decreased time for cuts on legs and feet to heal -improved trunk control and balance -better proprioceptive sense (ie.legs)
IV	-improved arm swing -reduced hypertonicity of leg muscles -decrease in assistance required to maintain knee extension during stance	-decrease in leg muscle spasticity -increase in leg strength -increase in leg mobility -easier transfers into bed
V	-assisted heel strike possible -reduced clonus and hypertonicity of leg muscles -decrease in assistance required to maintain knee extension during stance	-decrease in muscle spasticity -improved trunk balance -improved posture -improved standing endurance

Table 5: Benefits of BWSTT: qualitative improvements in ambulatory capacity and self-reported changes in daily living (R=right; L=left)

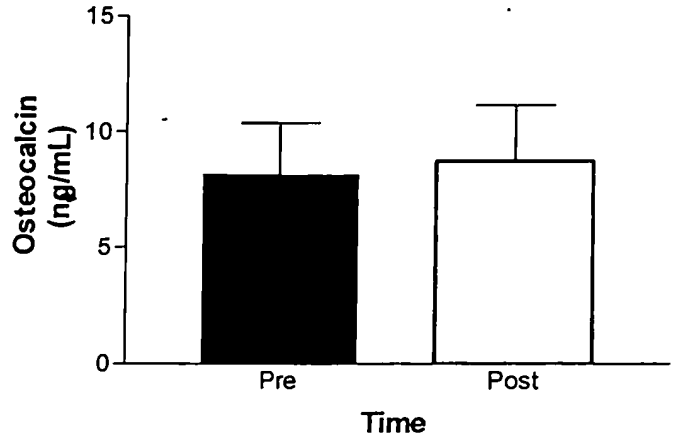

Figure 1. Serum osteocalcin concentration before and after 12 weeks of BWSTT. Values are means ± SD.

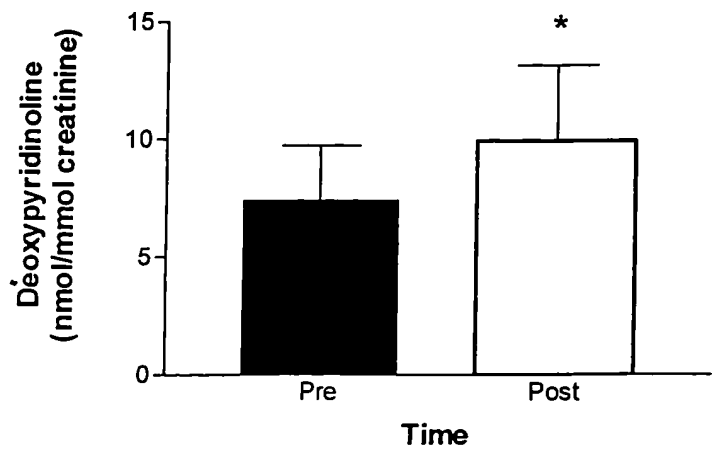

Figure 2. Urinary deoxypyridinoline concentration before and after 12 weeks of BWSTT. Values are means ± SD. * Significantly different from values before training ($p < 0.05$).

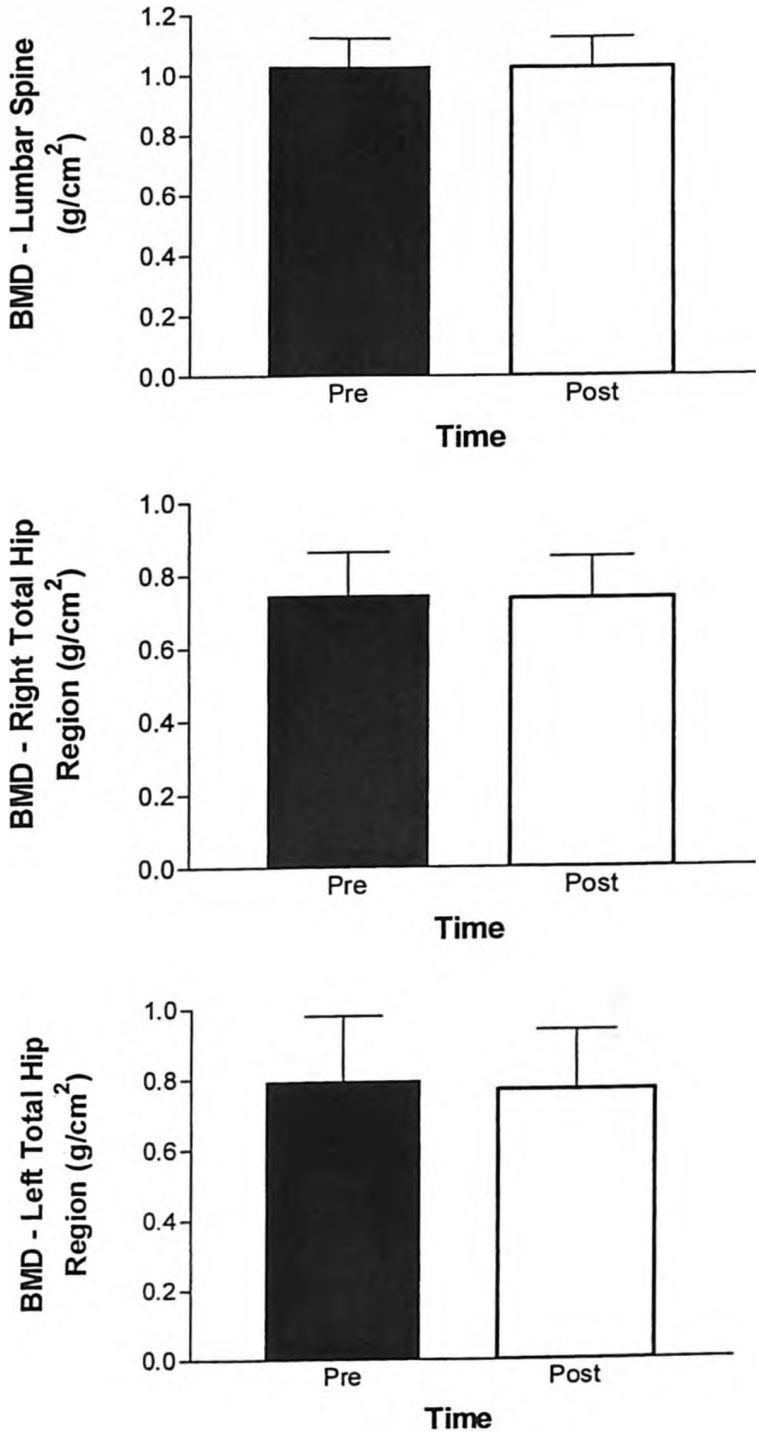

Figure 3. BMD before and after 12 weeks of BWSTT. Values are means ± SD.

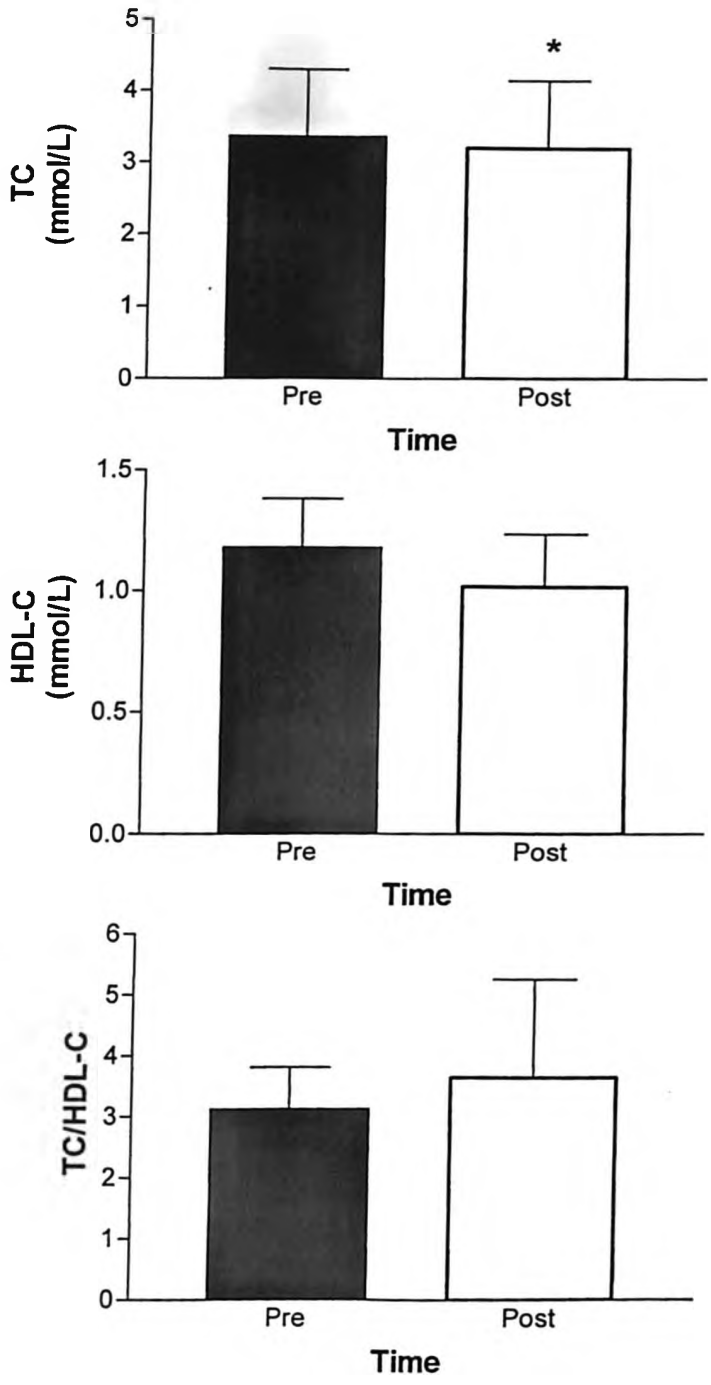

Figure 4. Total cholesterol (A), high density lipoprotein cholesterol (B), and TC/HDL-C ratio (C) before and after 12 weeks of BWSTT. Values are means ± SD. * Significantly different from values before training ($p < 0.05$).

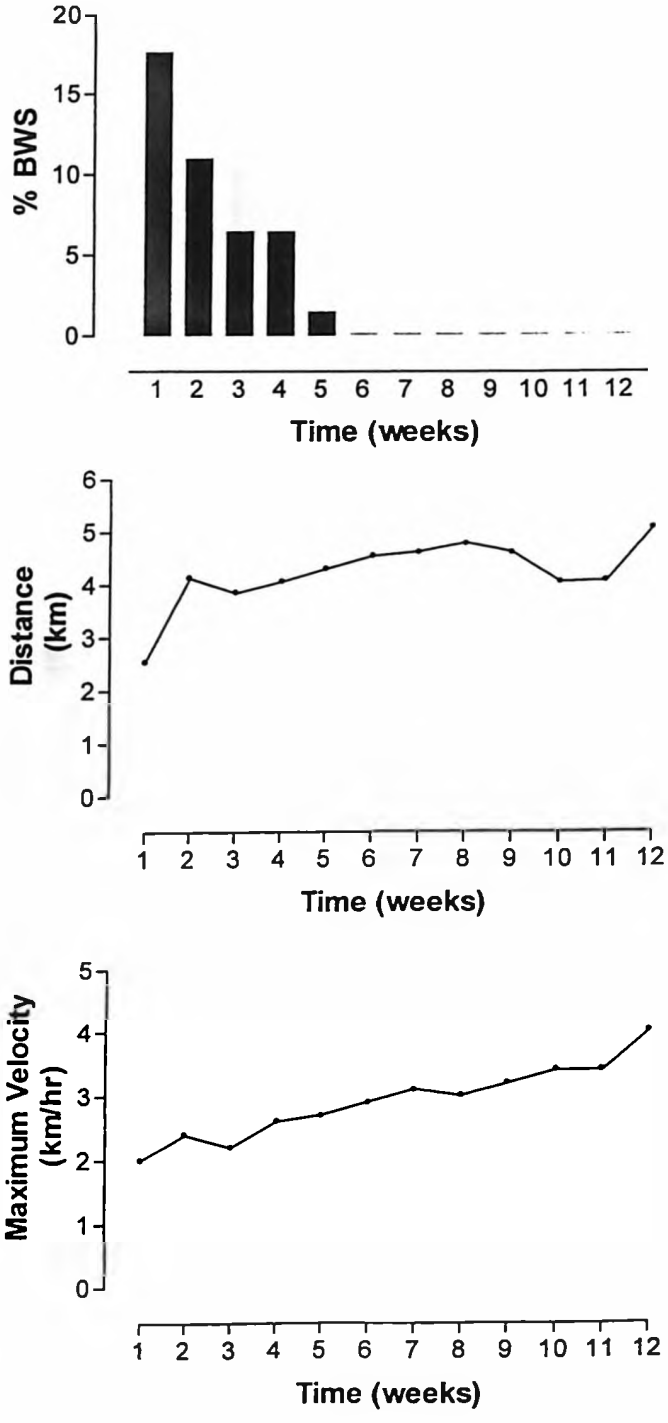

Figure 5. Ambulatory progress across 12 weeks of BWSTT – subject 1.

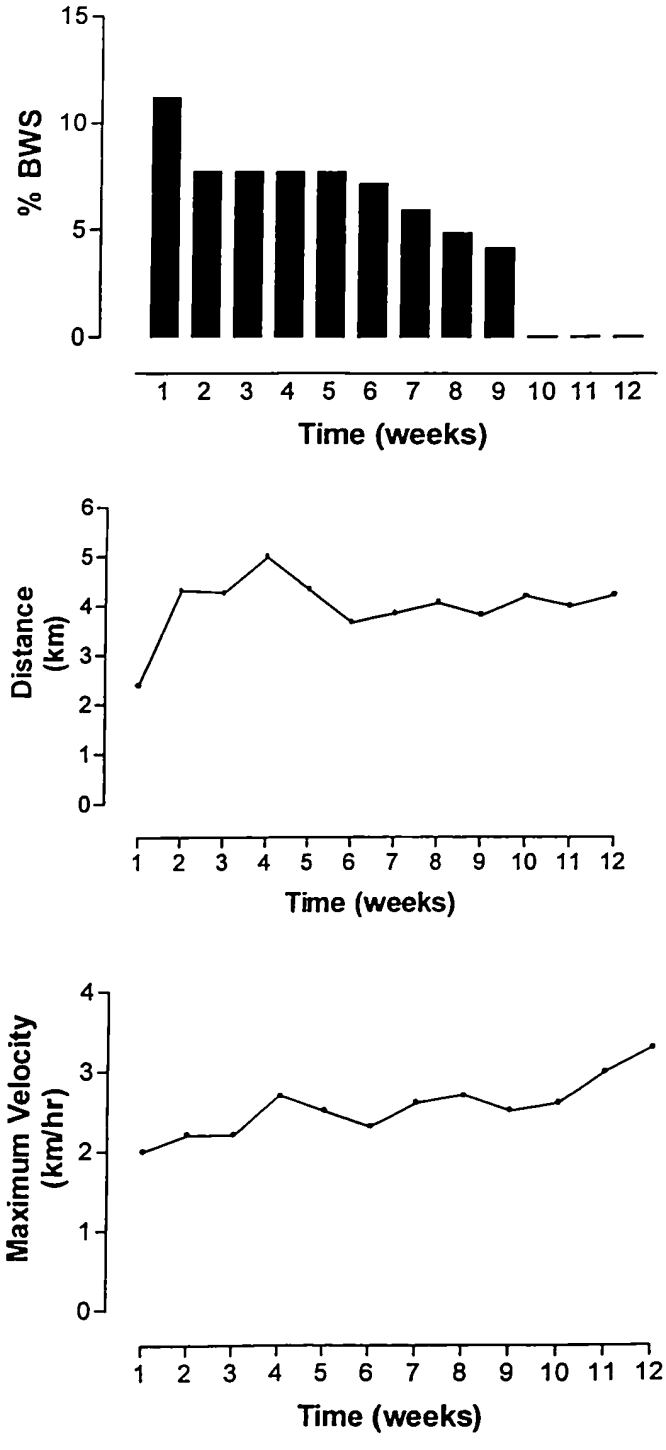

Figure 6. Ambulatory progress across 12 weeks of BWSTT – subject 2.

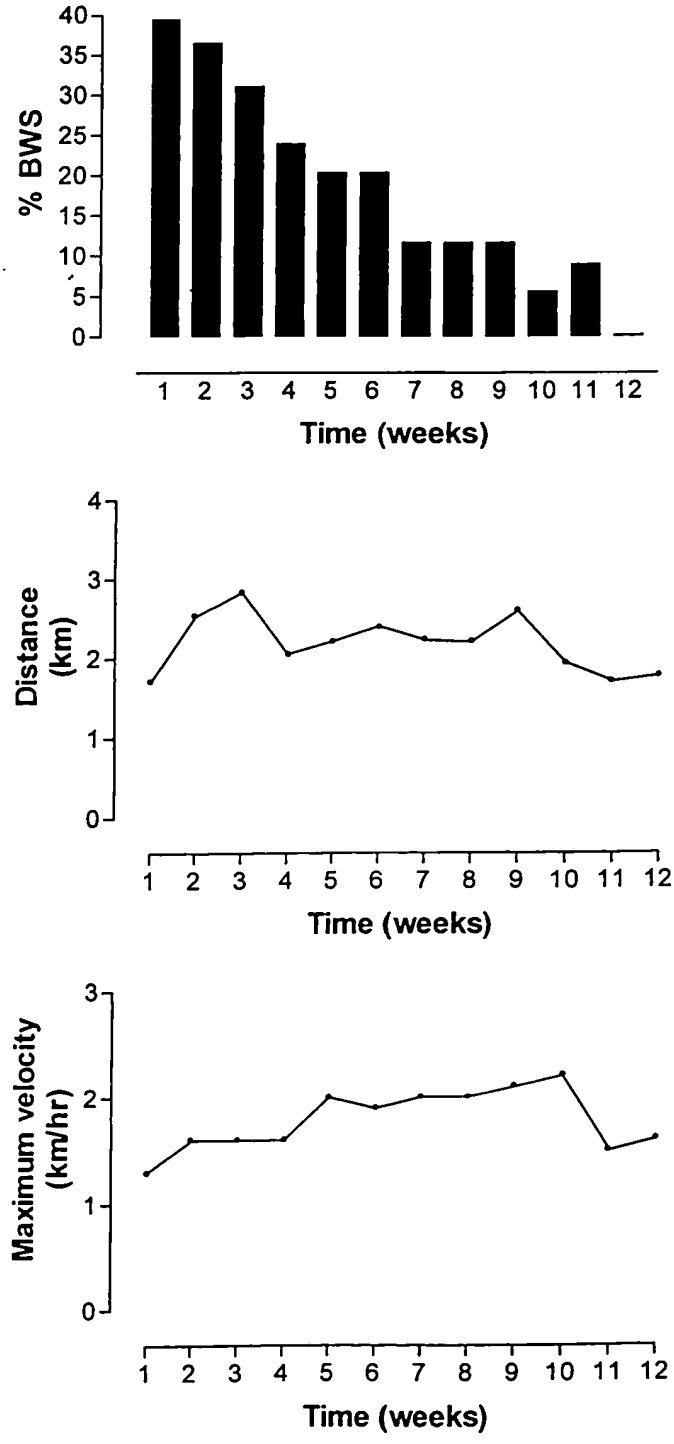

Figure 7. Ambulatory progress across 12 weeks of BWSTT – subject 3.

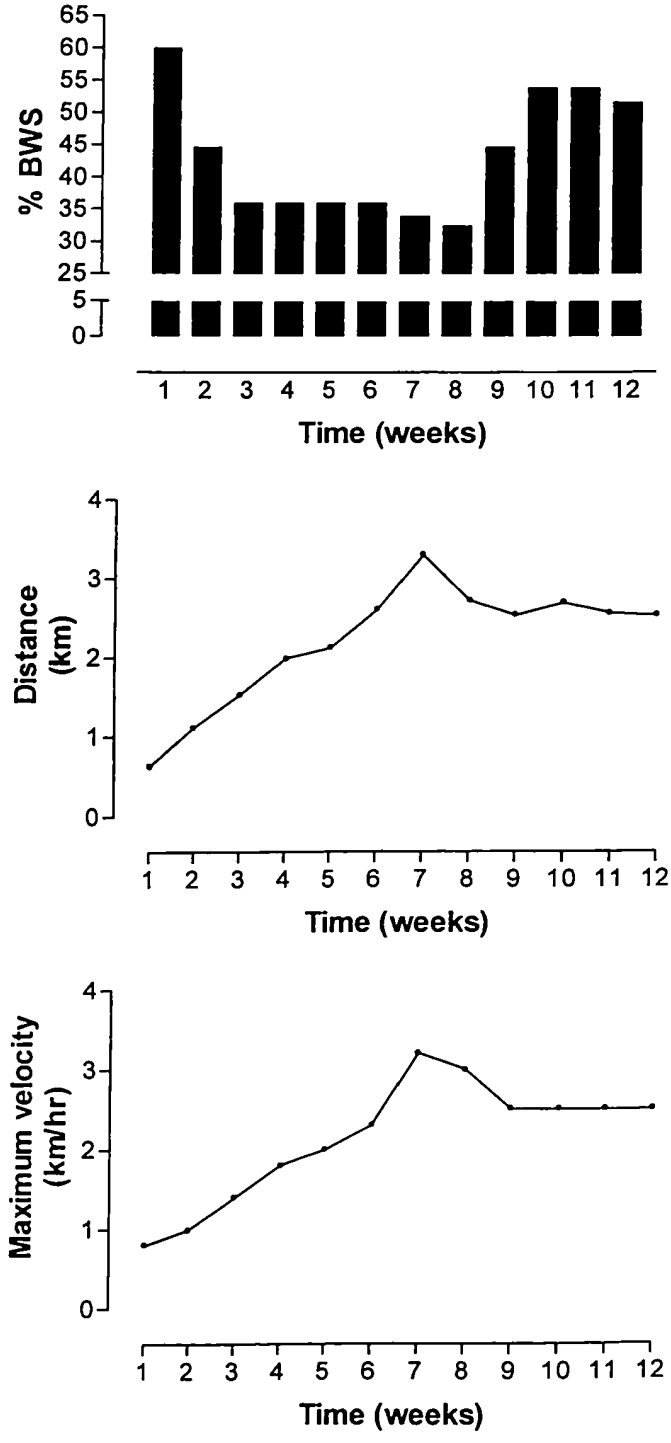

Figure 8. Ambulatory progress across 12 weeks of BWSTT – subject 4.

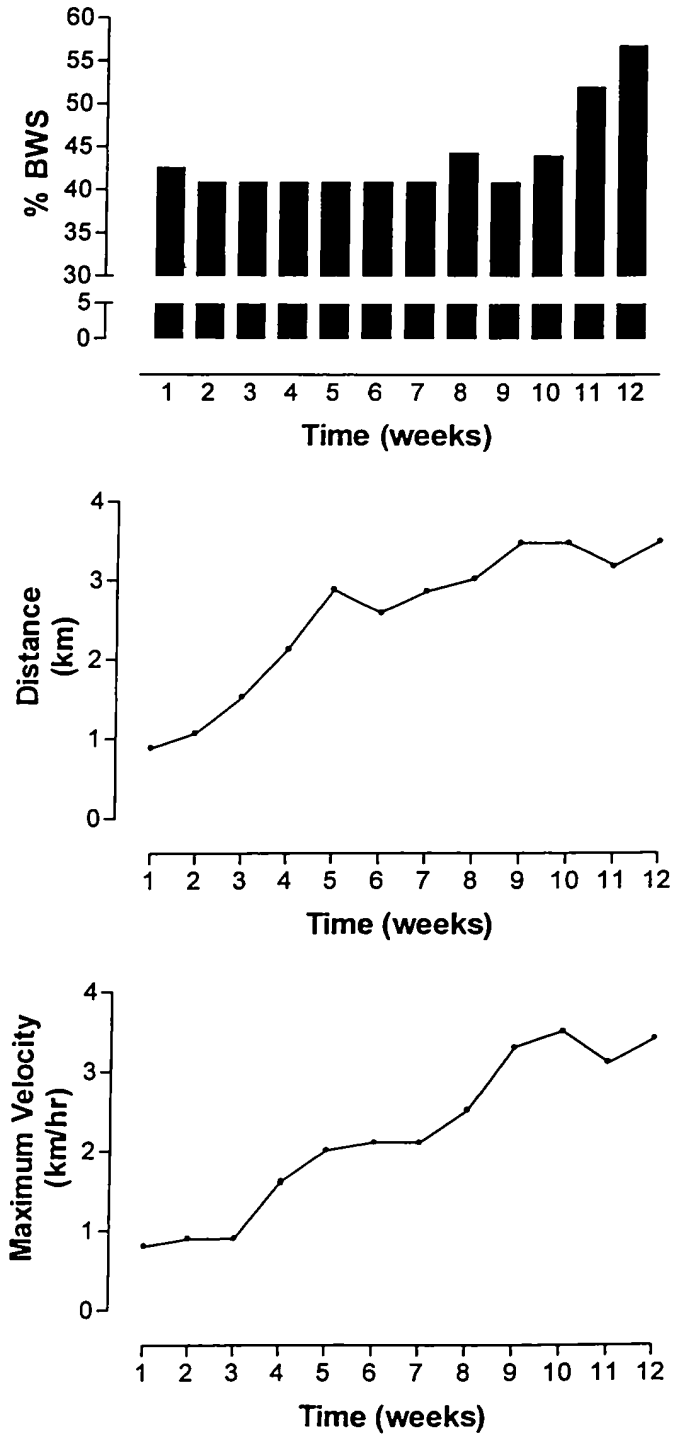

Figure 9. Ambulatory progress across 12 weeks of BWSTT – subject 5.

Discussion

Background

Common sequelae of SCI are osteoporosis in sublesional bone (see reviews in Kocina 1997; Uebelhart et al. 1995) and disordered metabolism of lipids and carbohydrates (see review in Bauman et al. 1999; Bauman and Spungen 1994; see reviews in Kocina 1997; Washburn and Figoni 1999). The etiology of each condition has not been definitively described. The decrease in BMD is attributed to a combination of neurovascular alterations and the loss of mechanical stress on bone that is normally generated with weight bearing and muscle activity (Kocina 1997; Saltzstein et al. 1992; Uebelhart et al. 1995). The dysmetabolic response, which includes impaired glucose tolerance and suppressed [HDL-C], is believed to be due to changes in body composition and reduced levels of physical activity (Washburn and Figoni 1999). Thus, according to prevailing theories, post-SCI immobilization contributes to the development of both of these conditions which increase morbidity in the SCI population.

A limited number of studies have investigated the potential benefits of weight bearing activity on BMC, BMD, and calcium balance in SCI persons (Biering-Sorensen et al. 1988; Goemaere et al. 1994; Kaplan et al. 1978; Kaplan et al. 1981; Kunkel et al. 1993; Saltzstein et al. 1992). These studies have generated inconsistent results. Questionable methodology has contributed to this variability and has limited the influence of this

research. Perhaps the major weakness is the lack of a consistent definition of weight bearing activity. Ambulation (Kaplan et al. 1978), a "mobility index" (Saltzstein et al. 1992), and the use of long leg braces, a tilt table, a standing frame, and a standing wheelchair (Biering-Sorensen et al. 1988; Goemaere et al. 1994; Kaplan et al. 1981; Kunkel et al. 1993) have all been used to identify those subjects exposed to weight bearing activity. Studies have also failed to quantify the amount of weight bearing training that subjects experienced. Inconsistent results have also been reported on the relationship between spasticity and BMC (and BMD); one group found that SCI persons with spasticity had significantly higher BMD than flaccid patients (Demirel et al. 1998), while others have found that spasticity has no effect on BMC (Biering-Sorensen 1988; Wilmet et al. 1995). Thus, although it has been suggested that the role of mechanical factors in preserving BMD is likely minimal when the neurovascular system is altered (Garland et al. 1992; Van Ouwenaller et al. 1989), the hypothesis has not been adequately addressed.

More consistent results have been reported in studies correlating exercise and lipid/lipoprotein parameters in persons with SCI. The available data suggests a potentially important role for physical activity in improving the lipid/lipoprotein profiles (including increased [HDL-C]) in this population (Brenes et al. 1986; Dallmeijer et al. 1997; Dallmeijer et al. 1999; Dearwater et al. 1986; Hooker and Wells 1989; LaPorte et al. 1983). HDL-C concentration is frequently selected as a dependent variable in these studies. The reasons for this selection are likely four-fold:

I) HDL-C levels have consistently been shown to be lower in the SCI population as compared with able-bodied controls (see reviews in Bauman et al. 1999; Washburn and Figoni 1999);

II) in the dysmetabolic schema proposed by Washburn and Figoni (1999) for SCI persons, [HDL-C] is the terminal marker in the cascade of deleterious changes that occur;

III) in able-bodied persons, exercise has been identified as an important determinant of [HDL-C] (see reviews in Berg et al. 1994; Durstine and Haskell 1994; Hardman 1996; Hartung 1995; National Cholesterol Education Program 1993; Sagiv and Goldbourt 1994); and

IV) there is an inverse association between [HDL-C] and the risk of CAD/CHD (see reviews in Berg et al. 1994; Durstine and Haskell 1994; Hardman 1996; Hartung 1995; National Cholesterol Education Program 1993; Sagiv and Goldbourt 1994).

As with the weight bearing-BMD research, there is a lack of uniformity in the indices of physical activity and physical fitness between studies. Consequently, there is no consensus on the mode, frequency, intensity or duration of activity that is required to manifest changes in lipid and carbohydrate metabolism in persons with SCI.

Rationale for this Study

Although BWSTT has been used in the research domain as a tool to improve ambulation after SCI for over 10 years, no study to date has investigated the secondary effect of this protocol on BMD or lipid and glucose metabolism. Simply described, the BWS apparatus consists of a tight fitting harness that is connected through a suspension system above the treadmill to a series of adjustable weight stacks. The suspension system allows "unloading" of subjects on the treadmill. Consequently, SCI persons who are incapable of supporting 100% of their body weight are able to stand upright. This provides the framework in which SCI persons can be (re)exposed to the afferent input associated with walking. Depending on the residual motor capabilities of the SCI subject, ambulation is accomplished with or without external assistance to the legs and pelvis. In theory, the

flux of afferent signals to the locomotor CPG in the spinal cord mobilizes latent motor patterns. The efferent signals manifest themselves in appropriate but unrefined EMG patterns that have been recorded in the leg muscles of SCI subjects during BWSTT (Dietz 1995; Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; Dobkin et al. 1992; Dobkin et al. 1995; Harkema et al. 1997). As described elsewhere (Barbeau et al. 1993 - paretic SCI subjects; Barbeau et al. 1998; Barbeau and Rossignol 1994; Barbeau et al. 1987 - paretic SCI patients; Dietz et al. 1997; Finch and Barbeau 1986 - hemiplegic patients; Finch et al. 1991; Gardner et al. 1998 - SCI subject; Hassid et al. 1997 - hemiparetic stroke patients; Hesse et al. 1995 - hemiparetic stroke patients; Hesse et al. 1994 - hemiparetic stroke patients; Hesse et al. 1997 - hemiparetic stroke patients; Norman et al. 1995; Pillar et al. 1991 - stroke patients; Visintin and Barbeau 1994 - paretic SCI subjects; Visintin and Barbeau 1989 - paretic SCI subjects; Visintin et al. 1998 - stroke patients; Visintin et al. 1988 - hemiplegic patient; Wainberg and Barbeau 1985 - paraparetic patients), the advantages of this approach to gait retraining are manifold. The advantages however, are not limited to gait retraining. We hypothesized that the depressed BMD in sublesional bone and the disordered metabolic profiles that characterize SCI would be responsive to an extended program of BWSTT. The rationale is as follows:

It has been shown that the provision of BWS delays the onset of fatigue in ambulation training and allows for longer exercise intervals compared with other locomotor rehabilitation modalities (Visintin and Barbeau 1994). Although this finding has been challenged (Visintin et al. 1998), we subscribe to the former theory and believe that the longer training intervals in BWSTT provide a greater cumulative stimulus for the

cardiovascular system. Ambulation training with BWS also offers the potential to activate the (formerly) large muscle mass of the legs, though this capability is likely limited to subjects with ASIA C and D classifications. In principle, the benefits of this are twofold:

I) if recruitment of the leg musculature is significant, exercise with the large muscle mass is likely to a yield an effective aerobic challenge (Glaser et al. 1996); and

II) it is argued by some that the skeleton is most responsive to the mechanical loading (and bone strain) that results from muscle contractions (Frost 2000; Frost 1997); others have argued that the skeleton is most responsive to the combination of gravitational and muscular forces (Henderson et al. 1998; Rutherford 1990). Either way, the mechanical loads generated by muscle contractions are a key contributor to preservation of bone (if the bone strain is below the modelling threshold), and to bone anabolism (if the strain is above the modelling threshold) (Frost 2000; Frost 1997).

With respect to bone metabolism, there is a general consensus that the magnitude and rate of bone strain are more important in stimulating anabolic activity than the number of strain cycles or the duration of exposure to the strain (see reviews in Chilibeck et al. 1995; Frost 1997; Henderson et al. 1998). Although the mechanical load, which is a primary determinant of bone strain (Frost 1997), is initially reduced in BWSTT, this is done out of necessity (ie. to allow verticality). The expectation is that with a program of BWSTT, progress will be made over time and subjects will support increasing percentages of body weight. In turn, the load on bone due to both muscular contractions and gravity will increase (as will bone strain magnitude), ideally to a level that is sufficient to exact positive changes in bone metabolism.

Because of the stability and security that the harness provides, SCI subjects are less likely to rely on the upper extremities to bear weight (compare with parallel bar training). Thus, progress with weight bearing can be confidently attributed to the legs. The

stability that the harness provides also allows subjects to focus their attention on the ambulatory pattern; this should enhance locomotor gains. Finally, it should be noted that when compared with long leg braces (Biering-Sorensen et al. 1988), BWSTT allows weight to be borne in a biomechanically appropriate manner.

An inherent advantage of the BWSTT approach is that it allows a fairly accurate quantification of the exercise stimulus. The protocol identifies the weight that is borne by the subject's legs during stance (although the counterbalance weight stacks may not always tell the truth), and the duration of the training sessions. Treadmill velocity can be used as a gauge of exercise intensity although the intensity is probably more dependent upon the ability of the subject to contribute to the ambulation outcome. Unfortunately, quantifying the relative contributions of the trainers and the subject to ambulation is subjective, unless the ambulation is independent. Nevertheless, the information drawn from BWSTT facilitates interstudy comparisons and it is envisioned that this information will be used in the development of exercise and weight bearing guidelines for the SCI population.

Based on these premises, we designed a pilot study with the following goals:

I)to determine the effect of a 3 month BWSTT program on
    a)bone metabolism,
    b)lipid/lipoprotein profiles, and
    c)ambulatory recovery in a diverse cohort of chronic (> 1 year) SCI persons, and

II)to assess the feasibility of this form of rehabilitation for future larger scale investigations.

Inclusion/exclusion criteria for the study restricted participation to those subjects classified as incompletes (ASIA B or higher). This was based on previous reports of limited success

with BWSTT in persons with complete (ASIA A) injuries (Dietz et al. 1994; Dietz et al. 1995; Dietz et al. 1998; Wernig et al. 1995).

Discussion of Results

Ambulatory Capacity

The five subjects in this study each completed the 36 training sessions over a span of 12 weeks. BWS was evaluated regularly and following the first session was set at the minimum level that would allow the subject to stand upright on the treadmill without buckling at the knee or hip joints. Exceptions to this general rule are listed in the methods and results sections. Manual assistance from the trainers was provided only as required at the ankle, knee and hip joints. Subjects were continuously encouraged to maintain a coordinated arm swing and to refrain from resting the arms on the horizontal bars. The majority of the sessions were 40 minutes in duration. The goal was to ambulate for 40 consecutive minutes and although all subjects accomplished this at least once, the interval format was preferred by most (only subject I was able to consistently complete the 40 minutes in one interval).

Over the training period, all subjects experienced reductions in required BWS and manual assistance, and increases in ambulatory endurance and velocity on the treadmill. This indicates that the protocol was effective in re-training the gait pattern. Between subjects, progress was more pronounced in those with higher baseline muscle grade scores. Only subject I made gains that were transferrable to overground walking.

In light of the ambulatory improvements, the HR responses to BWSTT (see

training records in the appendix), and the daily self-reports of generalized fatigue and muscular fatigue (unreported data from the study), it appears as though the protocol was also able to stress the skeletal and cardiovascular systems of the subjects. The biochemical markers of bone metabolism support this contention, while the lipid/lipoprotein measures do not.

Bone Metabolism

Following three months of BWSTT, urinary deoxypyridinoline (a marker of bone resorption) was increased significantly ($p<0.05$) and there was a non-significant trend for serum osteocalcin (a marker of bone turnover and formation - see methods and see below) to be increased ($p=0.167$). The elevated deoxypyridinoline levels suggest that the biomechanical strain on bone generated with BWSTT was sufficient to increase bone remodelling. The lack of congruence between the two markers may be a reflection of the normal sequence of events in bone remodelling. Typically, a remodelling site is initiated by the appearance of osteoclasts (and precursors) following any of several humoral or local stimuli to resorption. The osteoclasts proceed to resorb a small amount of bone which produces a small resorption pit. This resorptive phase is followed by an active reversal phase when the cement line is deposited. During the subsequent formative phase, actively synthesizing cuboidal osteoblasts appear and begin to deposit uncalcified matrix (osteoid) which is later mineralized (Ng et al. 1997). Resorption and formation occur successively in accordance with the six stage cycle of a bone remodelling unit (BMU) (Jee 1999; Vigorita 1999). Thus, the non-significant trend reported for osteocalcin in this study may be due to

the delayed onset of bone formation in the remodelling cycle. A review of the literature found no reports on the time frame of the BMU response to remobilization efforts in the SCI population. Subsequent trials should extend the BWSTT period beyond three months to determine if time is the limiting factor for observing significant changes in bone formation with this protocol.

It is unlikely that the observed changes in biochemical markers were a reflection of the normal evolution of bone following SCI. The majority of evidence suggests that osteoequilibrium is re-established between 12-16 months post-injury (Garland et al. 1992; Geusens et al. 1992; see review in Uebelhart et al. 1995), and all subjects in this study were beyond that window. It should be noted however, that recent studies have re-evaluated the time frame of bone metabolic changes in the post-SCI period and have indicated that osteoequilibrium may not be established within two years, let alone ever (Sharp et al. 1995; Szollar et al. 1997a). Thus, there remains a possibility that the changes in deoxypyridinoline and osteocalcin were independent of the exercise protocol. The consistency of the deoxypyridinoline changes across subjects, regardless of their duration of injury (19 months - 11 years), is suggestive however, of a common response to a training stimulus.

Considering that the increased concentrations of deoxypyridinoline represent an increase in bone resorption, it should be confirmed that the trend towards increased levels of osteocalcin is not a coincident manifestation of this resorption. Khosla and Kleerekoper argue that because osteocalcin is incorporated into bone matrix and is released into the circulation from the matrix during resorption, the serum level of osteocalcin at any one

time has a component of both bone formation and resorption. Consequently, osteocalcin is suggested to be a more appropriate marker of bone turnover rather than a specific marker of bone formation (Khosla and Kleerekoper). This argument is countered with considerable experimental evidence (eg. by using vitamin K antagonists) that indicates that the osteocalcin found in the circulation is derived from newly synthesized osteocalcin rather than from that released during bone resorption (see review in Kent 1997). It is thought that the release of many proteolytic enzymes during osteoclast-mediated bone resorption precludes the release of intact osteocalcin into the circulation (Kent 1997). Further, the antibody used in the NovoCalcin assay is believed to be conformationally dependent and as such, it should recognize only intact osteocalcin and not fragments from resorbed bone tissue (NovoCalcin assay kit). Therefore, the levels of serum osteocalcin recorded in this study are likely attributable to osteoblastic activity.

To correct for variations in urine concentration, values of deoxypyridinoline were expressed per mmol creatinine. There exists a possibility that this correction may have introduced a confounding factor. Khosla and Kleerekoper indicate that alterations in muscle mass, which cannot be excluded considering the training protocol in this study, can produce artifactual changes in urinary markers that are normalized to creatinine.

To account for the circadian rhythms of osteocalcin and deoxypyridinoline, post-training samples of urine and blood were collected at approximately the same time of the day as the baseline samples in all subjects (note: this time did vary between subjects - see methods). Although this was thought to obviate any effects of diurnal flux (Pyrilinks-D assay kit), it has been reported that for urine-based markers, the best methodology is to

obtain a 24 hour urine collection (Khosla and Kleerekoper). This should be kept in mind for future investigations

DEXA scans of the whole body, lumbar spine (L1-L4), and right and left hips (total hip region) did not reveal any significant differences in BMD across the training period. There was however, a trend for the BMD of both the left and right hips (total hip region) to be lower following BWSTT ($p=0.061$ & $p=0.186$ respectively). The presence of even a trend was an unexpected result given the 3 month duration of the study. It has been reported that in low bone turnover states, as long as two years may be required to detect a significant change in bone mass with x-ray absorptiometry (Kent 1997). In contrast, in high bone turnover states (which are not defined), significant changes in bone mass can be detected within three months (Kent 1997). In a review of longitudinal strength training studies with able-bodied human subjects, Chilibeck et al. (1995) found that training of 8-12 months duration results in much smaller increases in BMD than expected, and that a training duration of greater than 1 year may be needed before physiologically significant gains in bone mass are noticed. Where the BWSTT stimulus fits on the continuum of "bone turnover rates" is not known.

Even though the biochemical markers indicate an increase in bone turnover in this study, it is doubtful that the turnover rate was so rapid and pervasive as to affect BMD within 3 months. We argue instead, that the observed trends in BMD are artifactual, and are likely due to the intrinsic precision error of DEXA scanning combined with the small subject number; the trends may also be due to the inconsistent alignment of boundaries between pre- and post-training DEXA scans. With respect to the latter point, subsequent

studies should explicitly define the boundaries of each of the compartments to be analysed with DEXA. This will allow an accurate assessment of changes not only in BMD, but also in lean tissue, which is of interest given the disposition of SCI persons to develop sublesional muscular atrophy (Jones et al. 1998; see review in Kocina 1997).

One additional recommendation for future BWSTT studies using DEXA scanning is that because the regular bone remodelling cycle lasts from 4-6 months, studies should be continued for at least 1 year to ensure that the training effect is measured over an equilibrium period (see review in Chilibeck et al. 1995).

Blood Lipids, Lipoproteins, and Glucose

On the basis of the lipid/lipoprotein profiles, the BWSTT protocol did not appear to have a beneficial effect on lipid metabolism. The only measure that differed significantly ($p<0.05$) across the training period was plasma [TC]; post-training values for [TC] were lower than pre-training. Any potential reductions in CVD risk that could be attributed to this change are likely nullified by the fact that in three of five subjects, reductions in plasma [HDL-C] contributed notably to the decreased [TC].

The lack of improvement in the lipid/lipoprotein profile is surprising given the consistency of earlier cross-sectional reports that correlated physical activity with more favorable lipid/lipoprotein levels (including increased [HDL-C]) in the SCI population (Brenes et al. 1986; Dallmeijer et al. 1997; Dearwater et al. 1986). Questionable methodology may have contributed to the results that were obtained in this current study. Potential confounding factors and possible explanations for the lack of improvement in

lipid and lipoprotein values following 3 months of BWSTT include:

I)*diet* - Diet, which has the potential to influence lipid and lipoprotein profiles acutely and chronically (National Cholesterol Education Program 1993; see reviews in Berg et al. 1994; Durstine and Haskell 1994; Pronk 1993), was not controlled for. Diet records should be considered in future studies;

II)*medications* - Although the medications of each subject were recorded (see methods), there was no attempt to control for between- or within-subject variability. The pharmacological profile of one of the medications (carbamazepine - anticonvulsant) that was taken during the study (subject IV) includes a known interaction with blood lipoprotein concentrations - specifically the HDL-C subfraction (Henkin et al. 1992). In this case, carbamazepine was taken consistently by the subject across the duration of the study, including the pre- and post-training testing windows. The duration of treatment with this drug prior to the study is not known.

Within-subject medication consistency was not seen in subjects I and III (see methods). In both cases, medications varied across the testing periods although the effect, if any, that the drugs in question have on lipid metabolism is not known. Thus, another potential confounder may be the lack of a consistent drug regimen between and within subjects during pre- and post-training testing;

III)*analytical error*- The total error of Cholestech LDX measurements has been reported to be slightly higher than acceptable standards (Bard et al. 1997); consequently, the analyzer may be more suitable for categorizing a patient's results rather than determining the exact numerical value (Bard et al. 1997);

IV)*acute effects* - Because the post-training lipid/lipoprotein analysis was performed within 48 hours of the final training session, there is a possibility that a chronic change in lipid metabolism was veiled with the residual effects of the last training session. Review articles (able-bodied subjects) have concluded that a single bout of exercise has the potential to induce short-term, transient changes in plasma lipid and lipoprotein concentrations, and that the duration, intensity, and total volume of the exercise bout influences these changes (Durstine and Haskell 1994; Pronk 1993). Although the exercise prescription necessary to cause acute changes in [HDL-C] is not definitively known, an earlier review of the literature (Durstine and Haskell 1994) suggests that time is likely a factor (a period >1.5 hours), with a minimum energy expenditure of 1000 kcal. Recent work (Crouse et al. 1995; Gordon et al. 1998; Visich et al. 1996) however, has significantly lowered this threshold. In fact, Crouse et al. (1995) reports that a minimum energy expenditure of 350 kcal (with exercise performed at moderate intensity - 50% VO2max) is needed to promote favorable changes in [HDL-C] in hypercholesterolemic men. It appears that inter-study differences in baseline training levels and HDL-C levels of the subjects may explain some of the discrepancies within the literature.

The time of onset and the temporal decay of the acute lipid/lipoprotein response varies widely across studies. Pronk (1993) suggests that measurement of blood lipids and lipoproteins should only be accepted when taken following at least 48 hours abstinence from exercise. Forty-eight hours is lobbied as the maximum time course for short-term changes (Pronk 1993), despite the fact that greater than 50% of the studies reviewed by Pronk (1993) found that significant differences persisted at 48 hours. The review by

Durstine and Haskell (1994) indicates that acute changes in lipoprotein metabolism may extend 72 hours into the post-exercise period.

It should be noted that the acute response to exercise in able-bodied men is reported to consist primarily of an increase in [HDL-C] and a decrease in [TG] (Pronk 1993). Neither of these trends were consistently observed in the post-training samples (b/w 24-48 hrs post-exercise) of our SCI subjects. Thus, although a different population was involved in this study, it appears as though this potential confounder can be ruled out;

V)*intra-individual variation* - Beyond the analytical error (III), it has been shown that there is considerable intra-individual biologic variation in daily and monthly measures of blood lipids and lipoproteins in able-bodied persons (Nazir et al. 1999; see review in Pronk 1993);

VI)*alcohol* - Alcohol consumption has the potential to interact with blood lipid/lipoprotein profiles (Marrugat et al. 1996; Pronk 1993), and this confounder was not controlled for;

VII)*menstruation and oral contraceptives*: Females present additional variables that have the potential to interact with the lipid/lipoprotein profile. These factors, which include the stage of the menstrual cycle and the use of oral contraceptives, should be considered when interpreting results (see reviews in Durstine and Haskell 1994; Pronk 1993). No attempt was made to control for these factors in this study;

VIII)*baseline values within the normal/desirable range* - Based on standards advocated by the Heart and Stroke Foundation of Canada (1997) and the National Cholesterol Education Program (associated with National Institutes of Health - USA)

(1993), all subjects in this study had pre-training TG, TC, HDL-C and LDL-C values that were normal/desirable for their age. This is of significance because a meta-analysis has shown that in able-bodied persons, the modifiability of lipid/lipoprotein profiles with exercise is directly related to the extent of baseline dyslipidemia (ie. subjects with higher initial levels show greater decreases) (Matson et al. 1993). Therefore, the normal pre-training lipid/lipoprotein profiles of the subjects may have limited any potential benefits that BWSTT has with respect to modifying lipid metabolism.

The fact that the SCI subjects in this study all had normal pre-BWSTT lipid/lipoprotein profiles is surprising given the general consensus (see reviews in Bauman et al. 1999; Washburn and Figoni 1999) that dyslipidemia (specifically lower [HDL-C]) is common in this population. Perhaps this is an example of a sampling bias, where active and health conscious persons with SCI self-selected themselves for this study. Prior to engaging in the BWSTT program, 4 of the 5 subjects were exercising or attending physiotherapy two or more times per week. The only subject not involved in formal physiotherapy was subject I, who was able to ambulate with a cane.

IX)*plasma volume changes* - When the concentrations of plasma lipids and lipoproteins are measured over time, a change in blood volume as a direct result of a change in plasma volume will result in artificial inverse changes in the levels of the measured lipid parameters (Pronk 1993). A change in plasma volume may be acute and transient, as when following a bout of exercise (Crouse et al. 1995; see review in Durstine and Haskell 1994; Krum et al. 1991; see review in Pronk 1993; Visich et al. 1996), or it may represent a chronic adaptation to habitual aerobic exercise (McArdle et al. 1994;

Wilmore and Costill 1994a). Failing to account for plasma volume fluctuations can lead to spurious and misleading conclusions regarding changes in lipid and lipoprotein concentrations. The Cholestech LDX manual (analyzer used in this study) did not indicate if readings were corrected based upon the plasma volume;

X)*inadequate training stimulus* - Perhaps the BWSTT protocol was an inadequate exercise stimulus (dose) in terms of intensity of sessions, frequency of sessions, duration of sessions, length of training period, and/or total training volume for long-term modification of lipid and lipoprotein profiles. The current literature (able-bodied population) suggests that a dose-response relationship exists between aerobic physical activity and favorable changes in [HDL-C] (see reviews in Durstine and Haskell 1994; Hardman 1996; Kokkinos and Fernhall 1999). The relative contribution of each exercise component (intensity, frequency, and duration of training sessions, along with the length of training period) to the dose-response relationship has not been clearly established. It is likely however, that the dose is determined by an interaction between these 4 exercise variables (see review in Kokkinos and Fernhall 1999). It is also likely that a baseline threshold exists for each of these exercise components, but again these have not been clearly identified (see review in Kokkinos and Fernhall 1999).

If there was a limiting factor in attaining a sufficient exercise stimulus (dose) with BWSTT in persons with SCI, it would presumably be a lack of intensity. The intensity of BWSTT is limited by the ability of the subjects to I) activate their own leg musculature and II) ambulate at a reasonably high velocity. In the able-bodied literature, there is no consensus on the exercise intensity that is required to elicit favorable changes in [HDL-C].

Based loosely on exercise intensity classifications from Wilmore and Costill (1994b),

Intensity	HRmax	VO2max or HRmax reserve
light	*35-59%*	*30-49%*
moderate	*60-79%*	*50-74%*
heavy	*80-89%*	*75-84%*

there is support for each of low (Cook et al. 1986 - walking; intensity not quantified), moderate (Spate-Douglas and Keyser 1999 - walking @ 60% HR reserve; Stein et al. 1990 - cycle ergometry @ 75% HRmax; Sunami et al. 1999 - cycle ergometry @ 50% VO2max), and high intensity exercise (Marrugat et al. 1996 - exercise @ >7 kcal/min) as being the minimum intensity required for [HDL-C] modification. This inconsistency is likely due to a combination of factors that includes: variations in study protocol (ie. other determinants of exercise dose: training frequency, length of training program), variations in subject age, gender and dietary intake, variations in pre-training lipid profiles and fitness levels, and variations in concomitant weight loss and changes in body composition.

In an attempt to avoid the uncertainty associated with the intensity threshold, it has been proposed that total energy expenditure or training volume (see reviews in Durstine and Haskell 1994; Kokkinos and Fernhall 1999) be used as the determinant of the exercise dose. Another reason for reporting total energy expenditure (training volume) is that there is some evidence to suggest that this variable may be more important than exercise intensity with respect to modifying [HDL-C] (see review in Hardman 1996; Spate-Douglas and Keyser 1999). However, efforts to define the relationship between energy expenditure (training volume) and lipid/lipoprotein parameters have also been confounded by the variability between study protocols and subjects (Kokkinos and Fernhall 1999),

although not to the same extent as seen with exercise intensity. Despite the limitations, some general guidelines for energy expenditure (training volume) have been formulated from the research in this area:

An older review of the literature indicates that an additional weekly energy expenditure of >1000 kcal is the threshold for effecting plasma lipid and lipoprotein changes (Haskell 1984). Although this value is still supported as the approximate threshold for bringing about changes in lipids/lipoproteins (see reviews in Berg et al. 1994 - 1000 kcal/wk; Durstine and Haskell 1994 - 1000-1200 kcal/wk; Kokkinos and Fernhall 1999 - 1200-1600 kcal/wk), it is also suggested that a supplementary metabolic output of 1500-2000 kcal/week is necessary to actuate changes that are medically preventative; i.e. changes that reduce the incidence of CHD and prolong life expectancy (see review in Berg et al. 1994). Of significance for those SCI persons who are less mobile and less active, is the proposal that the level of energy expenditure necessary to cause changes in lipids/lipoproteins may be less in bed-rested patients (see review in Durstine and Haskell 1994). Perhaps even more important for the SCI cohort is the evidence (detailed above) from able-bodied studies that shows that low-to-moderate intensity exercise has the potential in some circumstances (perhaps if total energy expenditure is sufficient), to exert a measurable level of benefit on a negative risk factor for CAD (ie. [HDL-C]). Consider that subsequent to a SCI, there is usually a reduced capacity to exercise at high intensities. This is partly because of interrupted motor pathways and partly because of diminished sympathetic nervous system outflow (Glaser et al. 1996). Thus, low to moderate intensity exercise is often the only option in this population.

Whether the findings from able-bodied research are even applicable to the SCI population is not known. The sole longitudinal study to investigate the effects of exercise intensity on lipoprotein levels in SCI persons found that 8 weeks (3 sessions/week; 20 minutes/session) of *moderate intensity* (70-80% maximal HR reserve) wheelchair ergometry training was sufficient to elicit a significant increase in [HDL-C] and significant reductions in [TG], [LDL-C] and the [TC]/[HDL-C] ratio (Hooker and Wells 1989). Blood lipid/lipoprotein levels remained unaltered in the *low intensity* (50-60% maximal HR reserve) group. Although the authors of this study suggested that 70-80% of maximal HR reserve was moderate intensity exercise, other classification systems (Wilmore and Costill 1994b) consider this intensity (specifically the 75-80% range) to be heavy. In addition, the 50-60% maximal HR reserve exercise bracket that is classified by Hooker and Wells (1989) as low intensity falls into the moderate range in the classification from Wilmore and Costill (1994b). Thus, one needs to be wary when interpreting the results of the Hooker and Wells (1989) study.

In summary, there is insufficient evidence to identify the exercise parameters that are important and/or necessary for inducing favorable changes in lipid metabolism in the SCI population. Thus, if the BWSTT stimulus was indeed lacking, it is difficult to determine where this protocol fell short: ie. training intensity, training frequency, training duration, length of training program, and/or total energy expenditure. The able-bodied research seems to suggest that low-moderate intensity exercise (like BWSTT) has the potential to modulate lipid/lipoprotein levels, although the findings are inconsistent. It is possible that the total energy expenditure (training volume) of the BWSTT protocol was

inadequate. It is also possible that at 12 weeks in length, the BWSTT program was too short. Kokkinos and Fernhall (1999) have reported that in general, training studies of > 12 weeks duration have found some favorable changes in HDL-C levels; this puts the current study on the threshold. In comparison, Hartung (1995) suggests that aerobic training should extend for a minimum of 6 months in trials looking at [HDL-C]. Thus, it reasonable to suggest that the length of the training period may have been a limiting factor for lipid/lipoprotein changes in this investigation.

Although fasting plasma glucose concentration was not a primary outcome in this investigation, it was calculated simultaneously with the lipid/lipoprotein values by the Cholestech analyzer, and the results will be discussed briefly. It should be noted that some of the confounding factors that may have affected the lipid/lipoprotein profiles may also have affected the glucose measures. Pre- to post-training there was a non-significant trend ($p=0.052$) for [glucose] to be lower. Because of the increased prevalence of glucose intolerance, insulin resistance, and diabetes mellitus in the SCI population (see reviews in Bauman et al. 1999; Kocina 1997; Washburn and Figoni 1999), this trend is engaging and may warrant greater attention in subsequent BWSTT studies.

Although the literature indicates that persons with SCI have an increased susceptibility to impairments in glucose metabolism, all subjects in this study had pre-training fasting plasma [glucose] ≤ 5.25 mmol/L (mean +- SD = 4.82 +- 0.25). According to the Canadian clinical practice guidelines, these values are normal (Meltzer et al. 1998). In fact, they lie well below the range (6.1-6.9 mmol/L) that is indicative of impaired

glucose tolerance. The combination of normal pre-training lipid/lipoprotein profiles and normal pre-training fasting plasma [glucose] demonstrates that the subjects in this study did not have any underlying metabolic disorders. Why the subjects in this study differed in this regard from the majority of the SCI population is not known.

Summary and Additional Recommendations

The design of this pilot study (small cohort (n=5), confounding factors, and lack of a control group) precludes any bold, wide-ranging conclusions. The results of the study do lend support however, to the hypothesis that BWSTT is an effective stimulus for bone remodelling in persons with chronic SCI. The deoxypyridinoline findings should provide the impetus for further investigations. Subsequent trials using BWSTT should extend the training period to determine if the changes in biochemical markers of bone metabolism are the antecedent of positive changes in sublesional BMD (as determined by DEXA), and consequently, a reduced fracture risk. More frequent sampling of the biochemical markers would also delineate the time frame of skeletal adaptations in response to the BWSTT stimulus.

While BWSTT has been used safely and effectively to retrain ambulatory patterns in the acute post-injury time period, it is not known if early BWSTT minimizes the deleterious sequence of events that occurs in sublesional bone. Future research should determine if the introduction of BWSTT in the acute post-injury period affects the level at which osteo-equilibrium is re-established. This line of investigation may also provide some insight into the relative contributions of neurovascular changes and (the loss of)

biomechanical strain in the sublesional osteoporosis of persons with SCI.

The results from this study gave no indication that BWSTT had a salutary effect on lipid metabolism. The significance of the potential confounding factors (reviewed above) in this study is debatable, but they likely contributed to the lipid/lipoprotein responses that were observed. Because dyslipidemia (specifically decreased [HDL-C]) is a concern in this population (see reviews in Bauman et al. 1999; Washburn and Figoni 1999) (but not in our subjects) and is a risk factor for CVD, future studies should re-evaluate the [HDL-C] over a longer training period and with greater attention to methodological control. In addition to total [HDL-C], the subfractions of HDL-C should be measured, as their protective value, with respect to CHD, differs (see reviews in Hartung 1995; Sagiv and Goldbourt 1994). Finally, an attempt should be made to calculate the caloric expenditure of each subject during BWSTT in order to assess the metabolic demands placed on the body with this form of training. This should be done on each subject to gauge the effect of ASIA classification and muscle strength grade on caloric expenditure. This information will provide some indication as to whether BWSTT is an adequate stimulus for the modification of lipid metabolism in persons with SCI.

The feasibility of a follow-up BWSTT study of longer duration and with a greater number of subjects needs to be addressed. Based on experiences from the pilot project, a couple of concerns are raised and suggestions offered:

I) The physical demands that are placed on the trainers assisting with the leg movements and torso stablilization of the subjects should not be underestimated. The degree of difficulty for the trainer(s) varies inversely with the ASIA classification of the

subject (ie. those subjects with less strength and motor control require greater assistance). Even within a given ASIA level there are a range of capabilities: ASIA C subjects with low muscle strength grades require significantly more assistance than ASIA C subjects with high muscle strength grades. In addition to providing support during stance and assisting with swing through, significant upper body strength is required by the trainers to overcome the periodic hypertonia (spasticity) that can interrupt the gait cycle of a subject. It is no exaggeration to state that the task is physically exhausting, and that the trainers can be the limiting factor in a BWSTT session. The physical demands should be considered when selecting trainers and when determining the number of subjects a trainer will work with per day.

II) Each subject with an incomplete SCI possesses a unique (but incomplete) set of residual ambulatory abilities that must be recognized and exploited by the trainers over the course of a BWSTT program. Trainers should be familiar with the ambulatory response of a subject to changes in treadmill velocity and manual assistance. Trainers should also know how to minimize spasticity and clonus in the subject they are working with. Reciprocally, the SCI subject needs to develop confidence in the assistance provided by the trainers during BWSTT. It takes time to establish such a working relationship between trainers and subject. Thus, BWSTT is most effective if the subject works with the same group of trainers at each session. This strategy also allows for regular and consistent evaluations of ambulatory progress.

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Appendix I

Raw Data and Statistics

Raw Data and Statistics

Conversion between initials and subject #:

KL = 1
DR = 2
CS = 3
JL = 4
EB = 5

Osteocalcin (ng/ml)

0 CASE NAME	1 PRE	2 POST
JL	10.780	12.440
KL	7.690	8.660
EB	9.490	9.260
DR	8.040	7.790
CS	4.690	5.720

STAT. T test	T-test for Dependent Samples (new.sta) Marked differences are significant at p < .05000					
Osteocalcin	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t
PRE POST	8.138000 8.774000	2.286213 2.448669	5	-.636000	.844145	-1.68471

STAT. T test	T-test for Dependent Samples (new.sta) Marked differences are significant at p < .05000	
Osteocalcin	df	p
PRE POST	4	.167328

Deoxypyridinoline (nmol/mmol creatinine)

0 CASE NAME	1 PRE	2 POST
JL	9.050	11.400
KL	3.910	4.400
EB	9.960	12.480
DR	7.000	11.570
CS	7.060	9.790

STAT. STAT T test	T-test for Dependent Samples (deoxy.sta) Marked differences are significant at p < .05000					
Deoxypyridinoline	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t
PRE POST	7.396000* 9.928000*	2.330865* 3.238575*	5*	-2.53200*	1.448765*	-3.90797*

STAT. STAT T test	T-test for Dependent Samples (deoxy.sta) Marked differences are significant at p < .05000	
Deoxypyridinoline	df	p
PRE POST	4*	.017424*

Bone Mineral Density (g/cm2) WHOLE BODY

0 CASE NAME	1 PRE	2 POST
JL	1.081	1.079
KL	1.333	1.335
EB	1.035	1.045
DR	1.121	1.097
CS	1.087	1.107

WHOLE BODY

STAT. STAT T test	T-test for Dependent Samples (bmd.sta) Marked differences are significant at p < .05000						
BMD	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t	df
PRE POST	1.131400 1.132600	.116785 .115580	5	-.001200	.016407	-.163542	4

STAT. STAT T test	T-test for Dependent Samples (bmd.sta) Marked differences are significant at p < .05000
BMD	p
PRE POST	.878022

Bone Mineral Density (Lumbar Spine) (g/cm2)

0 CASE NAME	1 PRE	2 POST
JL	.936	.932
KL	1.158	1.177
EB	1.104	1.075
DR	.960	.966
CS	.979	.977

STAT. Stat T test	T-test for Dependent Samples (bmdlumbar.sta) Marked differences are significant at p < .05000					
BMD Lumbar Spine	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t
PRE POST	1.027400 1.025400	.097677 .100046	5	.002000	.017593	.254205

STAT. Stat T test	T-test for Dependent Samples (bmdlumbar.sta) Marked differences are significant at p < .05000	
BMD Lumbar Spine	df	p
PRE POST	4	.811870

Bone Mineral Density (R & L Hips) (g/cm2) TOTAL HIP

0 CASE NAME	1 R_PRE	2 R_POST	3 L_PRE	4 L_POST
JL	.620	.618	.636	.621
KL	.944	.926	1.104	1.052
EB	.724	.730	.674	.673
DR	.700	.691	.724	.710
CS	.730	.721	.821	.790

STAT. Stat T test	T-test for Dependent Samples (bmdhip.sta) Marked differences are significant at p < .05000						
BMD R Hip	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t	df
R_PRE R_POST	.743600 .737200	.120320 .114349	5	.006400	.008961	1.597008	4

STAT. Stat T test	T-test for Dependent Samples (bmdhip.sta) Marked differences are significant at p < .05000
BMD R Hip	p
R_PRE R_POST	.185502

STAT. Stat T test	T-test for Dependent Samples (bmdhip.sta) Marked differences are significant at p < .05000						
BMD L Hip	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t	df
L_PRE	.791800	.187796					
L_POST	.769200	.169655	5	.022600	.019578	2.581214	4

STAT. Stat T test	T-test for Dependent Samples (bmdhip.sta) Marked differences are significant at p < .05000
BMD L Hip	p
L_PRE	
L_POST	.061249

BMD Ward's (g/cm2)

0 CASE NAME	1 LWARDPRE	2 LWARDPST	3 RWARDPRE	4 RWARDPST
EB	.520	.523	.685	.664
DR	.554	.534	.653	.611
JL	.415	.422	.379	.419
KL	.675	.657	.665	.669
CS	.473	.471	.435	.508

STAT. BASIC STATS	T-test for Dependent Samples (bmdhipwards.sta) Marked differences are significant at p < .05000						
Variable	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t	df
LWARDPRE	.527400	.097649					
LWARDPST	.521400	.087990	5	.006000	.012309	1.090009	4

STAT. BASIC STATS	T-test for Dependent Samples (bmdhipwards.sta) Marked differences are significant at p < .05000
Variable	p
LWARDPRE	
LWARDPST	.336974

STAT. BASIC STATS	T-test for Dependent Samples (lipidata.sta) Marked differences are significant at p < .05000						
Variable	Mean	Std.Dv.	N	Diff.	Std.Dv. Diff.	t	df
GLU_PRE	4.818000	.251336					
GLU_POST	4.596000	.308107	5	.222000	.181714	2.731803	4

STAT. BASIC STATS	T-test for Dependent Samples (lipidata.sta) Marked differences are significant at p < .05000
Variable	p
GLU_PRE	
GLU_POST	.052346

Ambulatory capacity - raw data.

	subject 1			subject 2			subject 3			subject 4			subject 5		
week	1-bws	1-distance	1-max speed	2-bws	2-distance	2-max speed	3-bws	3-distance	3-max speed	4-bws	4-distance	4-max speed	5-bws	5-distance	5-max speed
1	17.7	2.54	2	11.23	2.38	2	39.6	1.7	1.3	60	0.63	0.8	42.6	0.88	0.8
2	11	4.13	2.4	7.76	4.32	2.2	36.6	2.53	1.6	44.6	1.12	1	40.9	1.07	0.9
3	6.5	3.85	2.2	7.76	4.27	2.2	31.2	2.84	1.6	36	1.53	1.4	40.9	1.52	0.9
4	6.5	4.06	2.6	7.76	4.99	2.7	24.1	2.06	1.6	36	1.99	1.8	40.9	2.12	1.6
5	1.5	4.3	2.7	7.76	4.33	2.5	20.5	2.22	2	36	2.12	2	40.9	2.88	2
6	0	4.54	2.9	7.23	3.67	2.3	20.5	2.41	1.9	36	2.6	2.3	40.9	2.59	2.1
7	0	4.63	3.1	5.98	3.86	2.6	11.8	2.23	2	33.9	3.29	3.2	40.9	2.86	2.1
8	0	4.8	3	4.92	4.07	2.7	11.8	2.21	2	32.4	2.72	3	44.3	3.02	2.5
9	0	4.63	3.2	4.21	3.82	2.5	11.8	2.6	2.1	44.7	2.53	2.5	40.9	3.48	3.3
10	0	4.07	3.4	0	4.21	2.6	5.7	1.95	2.2	53.9	2.69	2.5	44	3.48	3.5
11	0	4.1	3.4	0	4.01	3	9.1	1.71	1.5	53.9	2.56	2.5	52	3.19	3.1
12	0	5.1	4	0	4.23	3.3	0	1.78	1.6	51.7	2.53	2.5	60.5	3.5	3.4

BWS Treadmill Training
Ken Lamb

Session	Interval	Speed (km/hr)	Time (min)	Time (hr)	Distance (km)	BP	HR	3 session km	BWS (kg)	%wt Supported	3 session avg % wt Supported
1	a	0.3	7	0.11667	0.035				32	27.3	
	b	1.3	6	0.1	0.13				32	27.3	
	c	1.3	10	0.16667	0.216666667				32	27.3	
	d	1.3	6	0.1	0.13	91/68	84		24	24	
			29		0.511666667						
2	a	1.3	5	0.08333	0.108333333				24	24	
	b	1.4	15	0.25	0.35				16	15	
	c	1.5	16	0.26667	0.4	108/64	103		16	15	
			36		0.858333333						
3	a	1.5	10	0.16667	0.25				16	15	
	b	1.7	15	0.25	0.425				16	15	
	c	2	15	0.25	0.5	116/64	104		16	15	
			40		1.175			2.54			17.70%
4	a	2	15	0.25	0.5				16	15	
	b	2.4	10	0.16667	0.4	88/61	112		16	15	
	c	2.4	15	0.25	0.6	137/60	110		16	15	
			40		1.5						
5	a	2	20	0.33333	0.666666667				16	15	
	b	2	16	0.26667	0.533333333	100/56	114		8	6.5	
			36		1.2						
6	a	2.2	20	0.33333	0.733333333	87/52	120		8	6.5	
	b	2.1	20	0.33333	0.7	68/41	128		8	6.5	
			40		1.433333333			4.13			11.00%
7	a	2	20	0.33333	0.666666667	88/56	113		8	6.5	
	b	2.2	21	0.35	0.77	77/48	122		8	6.5	
			41		1.436666667						
8	a	2	25	0.41667	0.833333333	93/61	119		8	6.5	
			25		0.833333333						
9	a	2.2	22	0.36667	0.806666667	92/55	114		8	6.5	
	b	2.1	22	0.36667	0.77	85/72	128		8	6.5	
			44		1.576666667			3.85			6.50%
10	a	1.8	11	0.18333	0.293333333				8	6.5	
	b	1.3	8	0.13333	0.173333333	99/61	115		8	6.5	
	c	2	15	0.25	0.5	79/56	126		8	6.5	
			34		0.966666667						
11	a	2.2	25	0.41667	0.916666667	80/52	119		8	6.5	
	b	2.1	19	0.31667	0.665				8	6.5	
		2.8	2	0.03333	0.086666667	69/54			8	6.5	
			46		1.668333333						
12	a	1.8	10	0.16667	0.266666667				8	6.5	
		2.4	6	0.1	0.24				8	6.5	
	b	2.5	22	0.36667	0.916666667	91/59	105		8	6.5	
			38		1.423333333			4.06			6.50%
13	a	2	18	0.3	0.6				8	6.5	
		2.6	9	0.15	0.39	94/60	117		8	6.5	
	b	1.7	8	0.13333	0.226666667				0	0	
		2.6	7	0.11667	0.303333333	97/60	108		0	0	
			42		1.52						
14	a	1.9	10	0.16667	0.316666667				0	0	
		2.1	10	0.16667	0.35				0	0	
		2.2	10	0.16667	0.366666667				0	0	
		2.5	10	0.16667	0.416666667	102/64	118		0	0	
			40		1.45						
15	a	1.7	24	0.4	0.68				0	0	
		2.3	8	0.13333	0.306666667				0	0	
		2.5	6	0.1	0.25				0	0	
		2.7	2	0.03333	0.09	99/64	127		0	0	
			40		1.326666667			4.3			1.50%
16	a	1.7	23	0.38333	0.651666667				0	0	
		2.1	4	0.06667	0.14				0	0	
		2.3	5	0.08333	0.191666667				0	0	
		2.5	11	0.18333	0.458333333	89/61	124		0	0	
			43		1.441666667						
17	a	1.7	11	0.18333	0.311666667				0	0	
		1.9	11	0.18333	0.348333333				0	0	
		2.5	17	0.28333	0.708333333				0	0	
		2.9	3	0.05	0.145	114/68	132		0	0	
			42		1.513333333						
18	a	1.4	8	0.13333	0.186666667				0	0	
		1.7	5	0.08333	0.141666667				0	0	
		1.9	12	0.2	0.38				0	0	
		2.7	11	0.18333	0.495				0	0	
		2.9	8	0.13333	0.386666667	89/60	122		0	0	
			44		1.59			4.54			0%
19	a	1.4	8	0.13333	0.186666667				0	0	
		1.8	11	0.18333	0.33				0	0	
		2.9	10	0.16667	0.483333333				0	0	
		3.1	3	0.05	0.155				0	0	
		2.7	10	0.16667	0.45				0	0	

		1.7	2	0.03333	0.056666667	78/57	124		0	0		
			44		1.661666667							
20	a	1.4	10	0.16667	0.233333333				0	0		
		1.8	5	0.08333	0.15				0	0		
		2	8	0.13333	0.266666667				0	0		
		2.2	4	0.06667	0.146666667				0	0		
		2.6	8	0.13333	0.346666667				0	0		
		2.9	7	0.11667	0.338333333	76/56	128		0	0		
			42		1.481666667							
21	a	1	2	0.03333	0.033333333				0	0		
		1.4	6	0.1	0.14				0	0		
		1.6	5	0.08333	0.133333333				0	0		
		1.8	3	0.05	0.09				0	0		
		2	5	0.08333	0.166666667				0	0		
		2.2	3	0.05	0.11				0	0		
		2.6	12	0.2	0.52				0	0		
		2.9	8	0.1	0.29	79/56	132		0	0		
			42		1.483333333			4.03				0%
22	a	1.4	3	0.05	0.07				0	0		
		1.7	6	0.1	0.17				0	0		
		2	6	0.1	0.2				0	0		
		2.2	5	0.08333	0.183333333				0	0		
		2.4	5	0.08333	0.2				0	0		
		2.7	12	0.2	0.54				0	0		
		3	3	0.05	0.15	88/54	124		0	0		
			40		1.513333333							
23	a	1	2	0.03333	0.033333333				0	0		
		1.6	3	0.05	0.08				0	0		
		1.9	7	0.11667	0.221666667				0	0		
		2.2	11	0.18333	0.403333333				0	0		
		2.5	10	0.16667	0.416666667				0	0		
		2.8	9	0.15	0.42	88/52	120		0	0		
			42		1.575							
24	a	1.4	2	0.03333	0.046666667				0	0		
		1.7	3	0.05	0.085				0	0		
		2	12	0.2	0.4				0	0		
		2.2	9	0.15	0.33				0	0		
		2.5	8	0.13333	0.333333333				0	0		
		2.7	6	0.1	0.27				0	0		
		3	5	0.08333	0.25	89/58	142		0	0		
			45		1.715			4.8				0%
25	a	1.5	3	0.05	0.075				0	0		
		1.8	4	0.06667	0.12				0	0		
		2.1	8	0.13333	0.28				0	0		
		2.3	12	0.2	0.46				0	0		
		2.5	9	0.15	0.375				0	0		
		2.8	4	0.06667	0.186666667	91/56	133		0	0		
			40		1.496666667							
26	a	1.5	3	0.05	0.075				0	0		
		2	14	0.23333	0.466666667				0	0		
		2.4	13	0.21667	0.52				0	0		
		2.8	7	0.11667	0.303333333				0	0		
		3	4	0.06667	0.2	71/42	128		0	0		
			41		1.565							
27	a	1.5	3	0.05	0.075				0	0		
		1.8	7	0.11667	0.21				0	0		
		2.1	6	0.1	0.21				0	0		
		2.4	5	0.08333	0.2				0	0		
		2.6	11	0.18333	0.476666667				0	0		
		2.8	4	0.06667	0.186666667				0	0		
		3.2	4	0.06667	0.213333333	81/58	132		0	0		
			40		1.571666667			4.03				0%
28	a:track	1.94	13	0.21667	0.420333333		121		0	0		
		1.94	13	0.21667	0.420333333		115		0	0		
	b	1.5	2	0.03333	0.05				0	0		
		2	4	0.06667	0.133333333				0	0		
		2.5	4	0.06667	0.166666667				0	0		
		3	3	0.05	0.15				0	0		
		3.4	2	0.03333	0.113333333	75/44			0	0		
			41		1.454							
29	a:track	2.1	12	0.2	0.42		110		0	0		
		2.29	11	0.18333	0.419833333		105		0	0		
	b	1.6	3	0.05	0.08				0	0		
		2.1	3	0.05	0.105				0	0		
		2.6	10	0.16667	0.433333333	87/57	118		0	0		
			39		1.458166667							
30	a	1.4	8	0.13333	0.186666667				0	0		
		1.8	4	0.06667	0.12				0	0		
		2.1	4	0.06667	0.14				0	0		
		2.4	8	0.13333	0.32				0	0		
		2.6	2	0.03333	0.093333333				0	0		
		3	2	0.03333	0.1				0	0		
		3.2	2	0.03333	0.106666667				0	0		
		2.8	2	0.03333	0.093333333	77/54	114		0	0		
			32		1.16			4.07				0%
31	a:track	2.1	12	0.2	0.42				0	0		
		2.52	10	0.16667	0.42		127		0	0		
	b:track	2.29	11	0.18333	0.419833333		120		0	0		
			33		1.259833333							

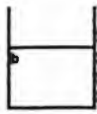
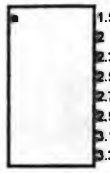
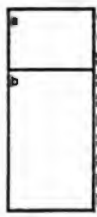
32	a:track	2.1	12	0.2	0.42					0	0	
		1.8	14	0.23333	0.42					0	0	
		1.8	14	0.23333	0.42		108			0	0	
			40		1.26							
33	a	1.5	5	0.08333	0.125					0	0	
		1.8	5	0.08333	0.15					0	0	
		2.1	5	0.08333	0.175					0	0	
		2.4	9	0.15	0.36					0	0	
		2.7	8	0.13333	0.36					0	0	
		3	6	0.1	0.3					0	0	
		3.4	2	0.03333	0.113333333	85/53	116			0	0	
			40		1.583333333			4.1				0%
34	a	1.5	4	0.06667	0.1					0	0	
		2.5	2	0.03333	0.083333333					0	0	
		2	2	0.03333	0.066666667					0	0	
		3	1	0.01667	0.05					0	0	
		2	2	0.03333	0.066666667					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		2	2	0.03333	0.066666667					0	0	
		3	1	0.01667	0.05					0	0	
		2.3	2	0.03333	0.076666667					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		4	1	0.01667	0.066666667					0	0	
		2	3	0.05	0.1					0	0	
		2.8	2	0.03333	0.093333333					0	0	
		2.2	2	0.03333	0.073333333					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		2.4	3	0.05	0.12					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		2.3	2	0.03333	0.076666667					0	0	
		3	1	0.01667	0.05					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		3	1	0.01667	0.05					0	0	
		2.2	2	0.03333	0.073333333					0	0	
		3.8	2	0.03333	0.126666667	82/58	141			0	0	
		1.2	3	0.05	0.06					0	0	
			43		1.751666667							
35	a:track	2.1	12	0.2	0.42					0	0	
		2.1	12	0.2	0.42					0	0	
		2.29	11	0.18333	0.419833333					0	0	
		2.29	11	0.18333	0.419833333					0	0	
			46		1.679666667							
36	a	1.5	2	0.03333	0.05					0	0	
		2.3	2	0.03333	0.076666667					0	0	
		2.9	2	0.03333	0.096666667					0	0	
		2	1	0.01667	0.033333333					0	0	
		3.8	1	0.01667	0.063333333					0	0	
		2.5	2	0.03333	0.083333333					0	0	
		3.3	1	0.01667	0.055					0	0	
		2.2	1	0.01667	0.036666667					0	0	
		3.4	1	0.01667	0.056666667					0	0	
		2	1	0.01667	0.033333333					0	0	
		2.6	2	0.03333	0.086666667					0	0	
		3.1	1	0.01667	0.051666667					0	0	
		2.6	1	0.01667	0.043333333					0	0	
		3.3	1	0.01667	0.055					0	0	
		2.2	1	0.01667	0.036666667					0	0	
		3.3	1	0.01667	0.055					0	0	
		2.5	2	0.03333	0.083333333					0	0	
		3.5	1	0.01667	0.058333333					0	0	
		2.2	1	0.01667	0.036666667					0	0	
		2.8	1	0.01667	0.046666667					0	0	
		2.4	1	0.01667	0.04					0	0	
		2.8	3	0.05	0.14					0	0	
		3.1	1	0.01667	0.051666667					0	0	
		2.1	1	0.01667	0.035					0	0	
		2.6	1	0.01667	0.043333333					0	0	
		2.9	1	0.01667	0.048333333					0	0	
		2	2	0.03333	0.066666667					0	0	
		2.7	1	0.01667	0.045					0	0	
		3.4	1	0.01667	0.056666667	96/58	133			0	0	
			38		1.665			5.1				0%

BWS Treadmill Training
Des Regehr

Session	Interval	Speed (km/hr)	Time (min)	Time (hr)	Distance (km)	BP	HR	3 Session km	BWS (kg)	%wt Supported	3 Session avg % wt Supported
1	a	0.1	5	0.08333	0.00833				32	22.11	
	b	0.5	10	0.16666	0.08333				32	22.11	
	c	0.6	15	0.25	0.15				32	22.11	
	d	0.6	10	0.16666	0.1		84		24	15.99	
			40		0.34166						
2	a	0.8	12	0.2	0.16				24	15.99	
	b	1.3	15	0.25	0.325				24	15.99	
	c	1.5	15	0.25	0.375	108/66	95		16	7.76	
			42		0.86						
3	a	1.6	10	0.16666	0.26666				16	7.76	
	b	1.9	15	0.25	0.475				16	7.76	
	c	2	13	0.21666	0.43333				16	7.76	
			38		1.17499			2.38			11.23
4	a	2	15	0.25	0.5				16	7.76	
	b	2	10	0.16666	0.33333				16	7.76	
	c	2.2	15	0.25	0.55	132/78	96		16	7.76	
			40		1.38333						
5	a	2	15	0.25	0.5				16	7.76	
	b	2	15	0.25	0.5				16	7.76	
	c	2	10	0.16666	0.33333	135/75	100		16	7.76	
			40		1.33333						
6	a	2	15	0.25	0.5				16	7.76	
	b	2.2	15	0.25	0.55	141/73	88		16	7.76	
	c	2.2	15	0.25	0.55	135/72	100		16	7.76	
			45		1.6			4.32			7.76
7	a	2	20	0.33333	0.66666				16	7.76	
	b	2	20	0.33333	0.66666		100 (radial)		16	7.76	
			40		1.33332						
8	a	2	25	0.41666667	0.833333333	113/67	95		16	7.76	
	b	2.2	15	0.25	0.55	137/80	99		16	7.76	
			40		1.383333333						
9	a	2	15	0.25	0.5				16	7.76	
	b	2	15	0.25	0.5				16	7.76	
	c	2.2	15	0.25	0.55	123/74	99		16	7.76	
			45		1.55			4.27			7.76
10	a	2.3	20	0.33333333	0.766666667				16	7.76	
		2.5	5	0.08333333	0.208333333				16	7.76	
	b	2.5	17	0.28333333	0.708333333	140/83	108		16	7.76	
			42		1.683333333						
11	a	2.5	20	0.33333333	0.833333333	128/74	103		16	7.76	
	b	1.9	13	0.21666667	0.411666667				16	7.76	
		2.5	5	0.08333333	0.208333333				16	7.76	
		2.7	2	0.03333333	0.09	112/64	111		16	7.76	
			40		1.543333333						
12	a	2	5	0.08333333	0.166666667				16	7.76	
		2.3	20	0.33333333	0.766666667	111/66	94		16	7.76	
	b	2.5	20	0.33333333	0.833333333	116/64	95		16	7.76	
			45		1.766666667			4.99			7.76
13	a	2.2	40	0.66666667	1.466666667	116/58	101		16	7.76	
14	a	2.2	35	0.58333333	1.283333333				16	7.76	
		2.5	10	0.16666667	0.416666667	132/72	108		16	7.76	
			45		1.7						
15	a	1.3	7	0.11666667	0.151666667				16	7.76	
		2	6	0.1	0.2				16	7.76	
		2.2	20	0.33333333	0.733333333				16	7.76	
		2.5	2	0.03333333	0.083333333	116/66	94		16	7.76	
			35		1.168333333			4.33			7.76
16	a	1.3	7	0.11666667	0.151666667				16	7.76	
		1.6	2	0.03333333	0.053333333				16	7.76	

		2	28	0.46666667	0.933333333				16	7.76	
		2.3	3	0.05	0.115	112/62	91		16	7.76	
			40		1.253333333						
17	a	1.2	12	0.2	0.24				16	7.76	
		2	8	0.13333333	0.266666667				16	7.76	
		2.2	9	0.15	0.33				16	7.76	
		1.2	5	0.08333333	0.1				16	7.76	
		2.2	10	0.16666667	0.366666667	107/58	90		16	7.76	
			44		1.303333333						
18	a	1	7	0.11666667	0.116666667				8	5.98	
		1.2	8	0.13333333	0.16				8	5.98	
		2	12	0.2	0.4				8	5.98	
		2.2	11	0.18333333	0.403333333				8	5.98	
		1.1	2	0.03333333	0.036666667	109/62	88		8	5.98	
			40		1.116666667			3.67			7.23
19	a	1	8	0.13333333	0.133333333				8	5.98	
		1.2	14	0.23333333	0.28				8	5.98	
		1.9	8	0.13333333	0.253333333				8	5.98	
		2.2	6	0.1	0.22				8	5.98	
		1.1	5	0.08333333	0.091666667				8	5.98	
		2.2	3	0.05	0.11				8	5.98	
		2.4	2	0.03333333	0.08	115/67	84		8	5.98	
			46		1.168333333						
20	a	1	3	0.05	0.05				8	5.98	
		1.2	3	0.05	0.06				8	5.98	
		1.4	6	0.1	0.14				8	5.98	
		1.6	9	0.15	0.24				8	5.98	
		1.8	2	0.03333333	0.06				8	5.98	
		2.3	6	0.1	0.23				8	5.98	
	b	2.2	6	0.1	0.22				8	5.98	
		2.4	7	0.11666667	0.28	95/54	97		8	5.98	
			42		1.28						
21	a	0.8	2	0.03333333	0.026666667				8	5.98	
		1.2	2	0.03333333	0.04				8	5.98	
		1.4	10	0.16666667	0.233333333				8	5.98	
		1.6	6	0.1	0.16				8	5.98	
		1.8	5	0.08333333	0.15				8	5.98	
		2.3	5	0.08333333	0.191666667				8	5.98	
	b	2.4	12	0.2	0.48				8	5.98	
		2.6	3	0.05	0.13	102/61	93		8	5.98	
			45		1.411666667			3.86			5.98
22	a	1.1	4	0.06666667	0.073333333				0	0	
		1.4	21	0.35	0.49				0	0	
		1.9	5	0.08333333	0.158333333	98/57	84		0	0	
	b	1.4	2	0.03333333	0.046666667				8	5.98	
		1.6	4	0.06666667	0.106666667				8	5.98	
		2.1	7	0.11666667	0.245				8	5.98	
		2.5	3	0.05	0.125	106/54	93		8	5.98	
			46		1.245						
23	a	1	3	0.05	0.05				8	5.98	
		1.3	3	0.05	0.065				8	5.98	
		1.6	14	0.23333333	0.373333333				8	5.98	
		1.8	3	0.05	0.09				8	5.98	
		2.2	5	0.08333333	0.183333333				8	5.98	
		2.4	12	0.2	0.48				8	5.98	
		2.6	5	0.08333333	0.216666667	120/65	91		8	5.98	
			45		1.458333333						
24	a	1	3	0.05	0.05				8	5.98	
		1.4	3	0.05	0.07				8	5.98	
		1.7	12	0.2	0.34				8	5.98	
		1.9	2	0.03333333	0.063333333				8	5.98	
		2.2	10	0.16666667	0.366666667				8	5.98	
		2.5	5	0.08333333	0.208333333				8	5.98	
		2.7	6	0.1	0.27	117/66	88		8	5.98	
			41		1.368333333			4.07			4.92
25	a	1	4	0.06666667	0.066666667				8	5.98	
		1.4	6	0.1	0.14				8	5.98	
		1.8	17	0.28333333	0.51				8	5.98	
		2.2	8	0.13333333	0.293333333				8	5.98	

		2.5	5	0.08333333	0.208333333	121/69	82		8	5.98	
			40		1.218333333						
26	a	1.1	4	0.06666667	0.073333333				8	5.98	
		1.5	5	0.08333333	0.125				8	5.98	
		1.7	9	0.15	0.255				8	5.98	
		1.8	4	0.06666667	0.12				8	5.98	
		2.2	12	0.2	0.44				8	5.98	
		2.5	11	0.18333333	0.458333333	133/67	84		8	5.98	
			45		1.471666667						
27	a	1.1	5	0.08333333	0.091666667				0	0	
		1.3	7	0.11666667	0.151666667				0	0	
		1.5	7	0.11666667	0.175				0	0	
		1.7	7	0.11666667	0.198333333				0	0	
		1.9	4	0.06666667	0.126666667				0	0	
	b	1.7	6	0.1	0.17				0	0	
		2.2	6	0.1	0.22	102/65	80		0	0	
			42		1.133333333			3.82			4.21
28	a	1.2	4	0.06666667	0.08				0	0	
		1.5	8	0.13333333	0.2				0	0	
		1.8	10	0.16666667	0.3				0	0	
	b	1.6	3	0.05	0.08				0	0	
		1.8	4	0.06666667	0.12				0	0	
		2.2	6	0.1	0.22				0	0	
		2.4	5	0.08333333	0.2	114/66	82		0	0	
			40		1.2						
29	a	1.2	4	0.06666667	0.08				0	0	
		1.5	4	0.06666667	0.1				0	0	
		1.8	10	0.16666667	0.3				0	0	
		2.1	6	0.1	0.21				0	0	
		2.4	6	0.1	0.24				0	0	
		2.6	5	0.08333333	0.216666667	122/66	97		0	0	
	b	2	2	0.03333333	0.066666667				0	0	
		2.4	2	0.03333333	0.08				0	0	
		2.6	3	0.05	0.13				0	0	
	c	2.4	6	0.1	0.24	106/61	88		0	0	
			48		1.663333333						
30	a	1.5	5	0.08333333	0.125				0	0	
		1.8	9	0.15	0.27				0	0	
		2.1	6	0.1	0.21				0	0	
	b	2	5	0.08333333	0.166666667				0	0	
		2.3	15	0.25	0.575	96/55	82		0	0	
			40		1.346666667			4.21			0
31	a	1.4	5	0.08333333	0.116666667				0	0	
		1.6	5	0.08333333	0.133333333				0	0	
		1.7	5	0.08333333	0.141666667				0	0	
		1.9	5	0.08333333	0.158333333	112/60	86		0	0	
	b	1.8	3	0.05	0.09				0	0	
		2	2	0.03333333	0.066666667				0	0	
		2.2	8	0.13333333	0.293333333				0	0	
		2.4	2	0.03333333	0.08				0	0	
		2.5	5	0.08333333	0.208333333	109/58	85		0	0	
			40		1.288333333						
32	a	1.2	4	0.06666667	0.08				0	0	
		1.5	4	0.06666667	0.1				0	0	
		1.8	27	0.45	0.81				0	0	
		2.4	5	0.08333333	0.2	112/67	86		0	0	
			40		1.19						
33	a	1.5	5	0.08333333	0.125				0	0	
		1.8	10	0.16666667	0.3				0	0	
		2	10	0.16666667	0.333333333				0	0	
		2.2	1	0.01666667	0.036666667	112/64	96		0	0	
	b	2.1	4	0.06666667	0.14				0	0	
		2.2	5	0.08333333	0.183333333				0	0	
		2.6	5	0.08333333	0.216666667				0	0	
		2.8	2	0.03333333	0.093333333				0	0	
		3	2	0.03333333	0.1	104/62	92		0	0	
			44		1.528333333			4.01			0
34	a	1.5	5	0.08333333	0.125				0	0	

		1.8	10	0.16666667	0.3				0	0
		2	12	0.2	0.4	112/68	84		0	0
		2.1	6	0.1	0.21				0	0
		2.4	3	0.05	0.12				0	0
		2.7	4	0.06666667	0.18	103/63	90		0	0
			40		1.335					
35		1.5	7	0.11666667	0.175				0	0
		2	6	0.1	0.2				0	0
		2.3	4	0.06666667	0.153333333				0	0
		2.5	4	0.06666667	0.166666667				0	0
		2.7	4	0.06666667	0.18				0	0
		2.9	5	0.08333333	0.241666667				0	0
		3.1	3	0.05	0.155				0	0
		3.3	2	0.03333333	0.11	113/68	105		0	0
			35		1.381666667					
36		1.5	4	0.06666667	0.1				0	0
		1.9	6	0.1	0.19				0	0
		2.2	10	0.16666667	0.366666667	124/65	96		0	0
		3.1	3	0.05	0.155				0	0
		2.9	3	0.05	0.145				0	0
		2.7	3	0.05	0.135				0	0
		2.5	3	0.05	0.125				0	0
		2.3	3	0.05	0.115				0	0
		2.1	3	0.05	0.105				0	0
		2.4	2	0.03333333	0.08	119/63	99		0	0
			40		1.516666667			4.23		0

BWS Treadmill Training
Cathy Schmuck

Session	Interval	Speed (km/hr)	Time (min)	Time (hr)	Distance (km)	BP	HR	3 Session km	BWS (kg)	%wt Supported	3 Session avg % wt Supported
1	a	0.7	10	0.16666667	0.116666667				32	42.2	
	b	0.7	10	0.16666667	0.116666667				32	42.2	
	c	0.7	10	0.16666667	0.116666667	155/87	119		32	42.2	
			30		0.35						
2	a	0.9	15	0.25	0.225				32	42.2	
	b	1.3	15	0.25	0.325	145/104	124		32	42.2	
			30		0.55						
3	a	1.2	20	0.33333333	0.4	160/112	128		24	36.6	
	b	1.2	20	0.33333333	0.4				24	36.6	
			40		0.8			1.7			39.6
4	a	1.2	20	0.33333333	0.4				24	36.6	
	b	1.3	20	0.33333333	0.433333333	124/95	99		24	36.6	
			40		0.833333333						
5	a	1.1	20	0.33333333	0.366666667				24	36.6	
	b	1.3	14	0.23333333	0.303333333				24	36.6	
		1.6	5	0.08333333	0.133333333				24	36.6	
			39		0.803333333						
6	a	1.3	20	0.33333333	0.433333333	162/108	152		24	36.6	
	b	1.3	15	0.25	0.325				24	36.6	
		1.6	5	0.08333333	0.133333333	140/95	158		24	36.6	
			40		0.891666667			2.53			36.6
7	a	1.5	18	0.3	0.45	164/94	120		24	36.6	
	b	1.6	18	0.3	0.48	159/108			24	36.6	
			36		0.93						
8	a	1.4	18	0.3	0.42				24	36.6	
	b	1.6	20	0.33333333	0.533333333				24	36.6	
			38		0.953333333						
9	a	1.6	18	0.3	0.48	139/91	154		16	20.5	
	b	1.6	18	0.3	0.48		156		16	20.5	
			36		0.96			2.84			31.2
10	a	0.9	10	0.16666667	0.15				16	20.5	
		1.2	5	0.08333333	0.1				16	20.5	
		1.4	3	0.05	0.07				16	20.5	
	b	0.7	20	0.33333333	0.233333333				16	20.5	
			38		0.553333333						
11	a	0.7	25	0.41666667	0.291666667				16	20.5	
	b	0.7	5	0.08333333	0.058333333				16	20.5	
		1.2	5	0.08333333	0.1				16	20.5	
		1.4	5	0.08333333	0.116666667				16	20.5	
		1.6	2	0.03333333	0.053333333	160/105	140		16	20.5	
			42		0.62						
12	a	0.7	3	0.05	0.035				16	20.5	
		1.4	17	0.28333333	0.396666667				16	20.5	
	b	0.7	3	0.05	0.035				24	36.6	
		1.5	17	0.28333333	0.425	128/88	122		24	36.6	
			40		0.891666667			2.06			24.1
13	a	0.8	21	0.35	0.28				16	20.5	
		1.1	4	0.06666667	0.073333333				16	20.5	
		1.3	7	0.11666667	0.151666667				16	20.5	
		1.6	8	0.13333333	0.213333333	155/97	166		16	20.5	
			40		0.718333333						
14	a	0.8	17	0.28333333	0.226666667				16	20.5	
		1.1	7	0.11666667	0.128333333				16	20.5	
		1.4	7	0.11666667	0.163333333				16	20.5	
		1.7	7	0.11666667	0.198333333				16	20.5	
		2	2	0.03333333	0.066666667	152/94	150		16	20.5	
			40		0.783333333						
15	a	0.8	17	0.28333333	0.226666667				16	20.5	
		1.1	7	0.11666667	0.128333333				16	20.5	
		1.4	7	0.11666667	0.163333333				16	20.5	
		1.7	7	0.11666667	0.198333333	121/78	117		16	20.5	
			38		0.716666667			2.22			20.5
16	a	0.8	17	0.28333333	0.226666667				16	20.5	
		1.1	7	0.11666667	0.128333333				16	20.5	
		1.4	16	0.26666667	0.373333333	164/150?	129		16	20.5	
			40		0.728333333						
17	a	0.5	5	0.08333333	0.041666667				16	20.5	
		0.8	5	0.08333333	0.066666667				16	20.5	
		1.1	10	0.16666667	0.183333333				16	20.5	
		1.4	12	0.2	0.28				16	20.5	
		1.6	8	0.13333333	0.213333333	137/91	144		16	20.5	
			40		0.785						
18	a	0.7	8	0.13333333	0.093333333				16	20.5	
		1.1	8	0.13333333	0.146666667				16	20.5	
		1.5	16	0.26666667	0.4				16	20.5	
		1.9	8	0.13333333	0.253333333	134/88	160		16	20.5	
			40		0.893333333			2.41			20.5

19	a	0.5	3	0.05	0.025				8	11.8		
		0.8	3	0.05	0.04				8	11.8		
		1.1	10	0.16666667	0.183333333				8	11.8		
		1.4	10	0.16666667	0.233333333				8	11.8		
		1.7	10	0.16666667	0.283333333				8	11.8		
		2	4	0.06666667	0.133333333	143/88	120		8	11.8		
			40		0.898333333							
20	a	0.5	4	0.06666667	0.033333333				8	11.8		
		0.8	4	0.06666667	0.053333333				8	11.8		
		1.1	5	0.08333333	0.081666667				8	11.8		
		1.4	7	0.11666667	0.161333333				8	11.8		
	b	0.7	3	0.05	0.035				8	11.8		
		1	3	0.05	0.05				8	11.8		
		1.3	5	0.08333333	0.108333333				8	11.8		
			31		0.535							
21	a	0.5	5	0.08333333	0.041666667				8	11.8		
		0.8	5	0.08333333	0.066666667				8	11.8		
		1.1	5	0.08333333	0.091666667				8	11.8		
		1.4	5	0.08333333	0.116666667				8	11.8		
		1.7	17	0.28333333	0.481666667	155/91	132		8	11.8		
			37		0.798333333			2.23				11.8
22	a	0.5	5	0.08333333	0.041666667				8	11.8		
		0.9	5	0.08333333	0.075				8	11.8		
		1.2	5	0.08333333	0.1				8	11.8		
		1.4	5	0.08333333	0.116666667	145/92	146		8	11.8		
	b	1	5	0.08333333	0.083333333				8	11.8		
		1.4	5	0.08333333	0.116666667				8	11.8		
		1.7	5	0.08333333	0.141666667				8	11.8		
		2	2	0.03333333	0.075	152/94	136		8	11.8		
			37		0.741666667							
23	a	0.5	5	0.08333333	0.041666667				8	11.8		
		0.8	8	0.13333333	0.106666667				8	11.8		
		1.1	7	0.11666667	0.128333333				8	11.8		
		1.5	7	0.11666667	0.175				8	11.8		
		1.8	8	0.13333333	0.24	140/92	164		8	11.8		
			35		0.691666667							
24	a	0.5	7	0.11666667	0.058333333				8	11.8		
		0.8	7	0.11666667	0.093333333				8	11.8		
		1.1	7	0.11666667	0.128333333				8	11.8		
		1.4	9	0.15	0.21				8	11.8		
		1.7	10	0.16666667	0.283333333	146/89	153		8	11.8		
			40		0.773333333			2.21				11.8
25	a	0.8	8	0.13333333	0.106666667				8	11.8		
		1.1	8	0.13333333	0.146666667				8	11.8		
		1.4	8	0.13333333	0.186666667				8	11.8		
		1.7	6	0.1	0.17	157/96	138		8	11.8		
	b	1.2	2	0.03333333	0.04				8	11.8		
		1.5	4	0.06666667	0.1				8	11.8		
		1.8	4	0.06666667	0.12				8	11.8		
		2	2	0.03333333	0.066666667				8	11.8		
			42		0.936666667							
26	a	0.5	3	0.05	0.025				8	11.8		
		0.8	3	0.05	0.04				8	11.8		
		1.1	9	0.15	0.165				8	11.8		
		1.4	10	0.16666667	0.233333333				8	11.8		
	b	1.1	3	0.05	0.055				8	11.8		
		1.4	6	0.1	0.14				8	11.8		
		1.7	3	0.05	0.085				8	11.8		
		2	3	0.05	0.1				8	11.8		
			40		0.843333333							
27	a	0.5	4	0.06666667	0.033333333				8	11.8		
		0.8	4	0.06666667	0.053333333				8	11.8		
		1.2	8	0.13333333	0.16				8	11.8		
		1.4	6	0.1	0.14				8	11.8		
		1.7	1	0.01666667	0.028333333	128/88	164		8	11.8		
	b	1.2	4	0.06666667	0.08				8	11.8		
		1.5	4	0.06666667	0.1				8	11.8		
		1.8	3	0.05	0.09				8	11.8		
		2.1	4	0.06666667	0.14	141/91	175		8	11.8		
			38		0.825			2.6				11.8
28	a	0.3	4	0.06666667	0.02				8	11.8		
		0.7	4	0.06666667	0.046666667				8	11.8		
		1.1	4	0.06666667	0.073333333				8	11.8		
		1.5	4	0.06666667	0.1				8	11.8		
		1.7	4	0.06666667	0.113333333	142/85	145		8	11.8		
	b	1.3	4	0.06666667	0.086666667				8	11.8		
		1.6	4	0.06666667	0.106666667				8	11.8		
		1.9	8	0.13333333	0.253333333				8	11.8		
		2.2	4	0.06666667	0.146666667	139/89	159		8	11.8		
			40		0.946666667							
29	a	0.3	5	0.08333333	0.025				0	0		
		0.6	6	0.1	0.06				0	0		
		0.9	6	0.1	0.09				0	0		
	b	0.3	3	0.05	0.015				0	0		
		0.7	4	0.06666667	0.046666667				0	0		
		1.1	6	0.1	0.11				0	0		
			30		0.346666667							
30	a	0.3	1	0.01666667	0.005				0	0		
		0.5	3	0.05	0.025				0	0		
		0.8	4	0.06666667	0.053333333				0	0		

		1.1	5	0.083333333	0.091666667				0	0	
		1.4	7	0.116666667	0.163333333	140/87	161		0	0	
	b	0.8	2	0.033333333	0.026666667				0	0	
		1.1	3	0.05	0.055				0	0	
		1.4	10	0.166666667	0.233333333				0	0	
			35		0.853333333			1.95			5.7
31	a	0.5	3	0.05	0.025				0	0	
		0.8	10	0.166666667	0.133333333				0	0	
		1.1	2	0.033333333	0.036666667	151/81	143		0	0	
	b	0.9	5	0.083333333	0.075				8	11.8	
		1.3	7	0.116666667	0.151666667				8	11.8	
		1.5	5	0.083333333	0.125	140/84	140		8	11.8	
			32		0.546666667						
32	a	0.5	5	0.083333333	0.041666667				0	0	
		0.8	10	0.166666667	0.133333333				0	0	
		1	5	0.083333333	0.083333333	152/92	159		0	0	
	b	0.8	6	0.1	0.08				8	11.8	
		1.1	13	0.216666667	0.238333333	136/91	147		8	11.8	
			39		0.576666667						
33	a	0.5	6	0.1	0.05				0	0	
		1	6	0.1	0.1				0	0	
		1	6	0.1	0.1				8	11.8	
		1.3	2	0.033333333	0.043333333	132/92	159		8	11.8	
	b	1	7	0.116666667	0.116666667				16	20.5	
		1.3	8	0.133333333	0.173333333	128/88	156		16	20.5	
			35		0.583333333			1.71			9.1
34	a	0.8	5	0.083333333	0.05				0	0	
		0.9	5	0.083333333	0.075				0	0	
		1.2	8	0.133333333	0.16				0	0	
		1.5	2	0.033333333	0.05	152/92	149		0	0	
	b	0.5	3	0.05	0.025				0	0	
		0.8	3	0.05	0.04				0	0	
		1.1	7	0.116666667	0.128333333				0	0	
		1.3	2	0.033333333	0.043333333	149/87	147		0	0	
			35		0.571666667						
35	a	0.5	7	0.116666667	0.058333333				0	0	
		0.9	7	0.116666667	0.105				0	0	
		1.1	6	0.1	0.11				0	0	
	b	0.7	3	0.05	0.035				0	0	
		1	6	0.1	0.1				0	0	
		1.3	6	0.1	0.13				0	0	
		1.6	3	0.05	0.08	143/88	120		0	0	
			38		0.618333333						
36			37		0.595				0	0	
								1.78			0

BWS Treadmill Training
Jake Lawless

Session	Interval	Speed (km/hr)	Time (min)	Time (hr)	Distance (km)	BP	HR	3 Session km	BWS (kg)	%wt Supported	3 Session avg % wt Supported
1	a	0.5	5	0.083333333	0.041666667				48	69.4	
	b	0.5	5	0.083333333	0.041666667				48	69.4	
			10	0.16666667	0.083333333						
2	a	0.3	7	0.11666667	0.035				48	69.4	
	b	0.8	13	0.21666667	0.13				48	69.4	
			20		0.165						
3	a	0.6	11	0.183333333	0.11				40	53.9	
	b	0.7	12	0.2	0.14				40	53.9	
	c	0.8	10	0.16666667	0.133333333				40	53.9	
			33		0.383333333			0.631666667			60
4	a	0.8	10	0.16666667	0.133333333				40	53.9	
	b	0.7	10	0.16666667	0.116666667	132/85	99		40	53.9	
	c	0.8	11	0.183333333	0.146666667	132/86	98		40	53.9	
			31		0.396666667						
5	a	0.7	9	0.15	0.105				32	36	
	b	0.8	7	0.11666667	0.093333333				32	36	
	c	0.8	5	0.083333333	0.066666667				32	36	
			21		0.265						
6	a	0.6	13	0.21666667	0.13				32	36	
	b	0.7	7	0.11666667	0.081666667				40	53.9	
		0.8	4	0.06666667	0.08				40	53.9	
	c	0.6	4	0.06666667	0.04				32	36	
		0.9	2	0.033333333	0.03				32	36	
		1	7	0.11666667	0.116666667	108/73	104		32	36	
			37		0.458333333			1.12			44.6
7	a	0.7	9	0.15	0.105				32	36	
		0.9	6	0.1	0.09	85/54	114		32	36	
	b	0.7	6	0.1	0.07				32	36	
		0.9	6	0.1	0.09				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.3	5	0.083333333	0.108333333	117/82	104		32	36	
			38		0.536666667						
8	a	0.8	8	0.1	0.08				32	36	
		1	4	0.06666667	0.066666667				32	36	
		1.3	5	0.083333333	0.108333333	124/58	102		32	36	
	b	0.7	7	0.11666667	0.081666667				32	36	
		1	5	0.083333333	0.083333333				32	36	
		1.4	3	0.05	0.07	87/62	105		32	36	
	c	0.7	2	0.033333333	0.023333333				32	36	
		1	4	0.06666667	0.066666667				32	36	
		1.4	8	0.1	0.14	102/81	93		32	36	
			42		0.7						
9	a	0.5	5	0.083333333	0.041666667				32	36	
		0.8	5	0.083333333	0.066666667				32	36	
		1.1	5	0.083333333	0.091666667				32	36	
	b	1.1	5	0.083333333	0.091666667				32	36	
			20		0.291666667			1.528333333			38
10	a	0.5	4	0.06666667	0.033333333				32	36	
		0.7	4	0.06666667	0.046666667				32	36	
		1	4	0.06666667	0.066666667				32	36	
		1.3	2	0.033333333	0.043333333	86/61	97		32	36	
	b	0.5	4	0.06666667	0.033333333				32	36	
		0.8	4	0.06666667	0.053333333				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.3	2	0.033333333	0.043333333	97/69	96		32	36	
	c	0.8	5	0.083333333	0.066666667				32	36	
		1.4	8	0.1	0.14	112/82	82		32	36	
			39		0.8						
11	a	0.6	4	0.06666667	0.04				32	36	
		0.9	4	0.06666667	0.06				32	36	
		1.2	4	0.06666667	0.08				32	36	
		1.5	5	0.083333333	0.125	85/54	103		32	36	
	b	0.9	4	0.06666667	0.06				32	36	
		1.2	4	0.06666667	0.08				32	36	
		1.5	8	0.133333333	0.2				32	36	
		1.8	4	0.06666667	0.12	86/60	118		32	36	
			37		0.765						
12	a	0.5	5	0.083333333	0.041666667				32	36	
		0.8	5	0.083333333	0.075				32	36	
		1.4	4	0.06666667	0.093333333	83/52	104		32	36	

	b	0.7	6	0.1	0.07				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.5	4	0.06666667	0.1	91/59	105		32	36	
	c	1	4	0.06666667	0.066666667				32	36	
		1.5	3	0.05	0.075				32	36	
		1.8	1	0.01666667	0.03	107/70	95		32	36	
			38		0.825			1.99			36
13	a	0.5	3	0.05	0.025				32	36	
		0.8	3	0.05	0.04				32	36	
		1.1	9	0.15	0.165				32	36	
		1.4	3	0.05	0.07	96/64	94		32	36	
	b	0.5	3	0.05	0.025				32	36	
		0.8	3	0.05	0.04				32	36	
		1.1	3	0.05	0.055				32	36	
		1.4	2	0.03333333	0.046666667				32	36	
	c	0.8	2	0.03333333	0.026666667				32	36	
		1.1	2	0.03333333	0.036666667				32	36	
		1.4	2	0.03333333	0.046666667				32	36	
		1.7	1	0.01666667	0.028333333	118/66	84		32	36	
			38		0.605						
14	a	0.6	4	0.06666667	0.04				32	36	
		0.9	4	0.06666667	0.06				32	36	
		1.2	5	0.08333333	0.1				32	36	
	b	0.9	3	0.05	0.045				32	36	
		1.3	3	0.05	0.065				32	36	
		1.5	6	0.1	0.15				32	36	
		1.8	7	0.11666667	0.21	109/73	92		32	36	
			32		0.67						
15	a	0.5	3	0.05	0.025				32	36	
		0.8	4	0.06666667	0.053333333				32	36	
		1.1	3	0.05	0.055				32	36	
		1.4	2	0.03333333	0.046666667				32	36	
	b	0.8	3	0.05	0.04				32	36	
		1.1	3	0.05	0.055				32	36	
	c	0.8	3	0.05	0.04				32	36	
		1.1	3	0.05	0.055				32	36	
		1.4	8	0.15	0.21	103/66	112		32	36	
	d	0.8	2	0.03333333	0.026666667				32	36	
		1.1	2	0.03333333	0.036666667				32	36	
		1.4	2	0.03333333	0.046666667				32	36	
		1.7	3	0.05	0.085				32	36	
		2	2	0.03333333	0.056666667	110/76	108		32	36	
			44		0.841666667			2.116666667			36
16	a	0.5	5	0.08333333	0.041666667				32	36	
		0.8	7	0.11666667	0.093333333				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.4	4	0.06666667	0.093333333				32	36	
		1.7	2	0.03333333	0.056666667	100/81	112		32	36	
	b	0.5	2	0.03333333	0.016666667				32	36	
		0.8	2	0.03333333	0.026666667				32	36	
		1.1	5	0.08333333	0.091666667				32	36	
		1.4	5	0.08333333	0.116666667				32	36	
		1.7	4	0.06666667	0.113333333				32	36	
		2	4	0.06666667	0.133333333				32	36	
		2.3	2	0.03333333	0.076666667	88/61	105		32	36	
			46		0.933333333						
17	a	0.5	5	0.08333333	0.041666667				32	36	
		0.8	5	0.08333333	0.066666667				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.4	5	0.08333333	0.116666667	92/58	107		32	36	
	b	0.8	2	0.03333333	0.026666667				32	36	
		1.1	2	0.03333333	0.036666667				32	36	
		1.4	4	0.06666667	0.093333333				32	36	
		1.7	6	0.1	0.17				32	36	
		2	4	0.06666667	0.133333333	112/64	108		32	36	
			37		0.758333333						
18	a	0.5	5	0.08333333	0.041666667				32	36	
		0.8	4	0.06666667	0.053333333				32	36	
		1.1	5	0.08333333	0.091666667				32	36	
		1.4	4	0.06666667	0.093333333				32	36	
		1.7	4	0.06666667	0.113333333	88/58	115		32	36	
	b	0.8	3	0.05	0.04				32	36	
		1.1	3	0.05	0.055				32	36	
		1.4	5	0.08333333	0.116666667				32	36	
		1.7	3	0.05	0.085				32	36	
		2	3	0.05	0.1				32	36	
		2.3	3	0.05	0.115	113/74	114		32	36	
			42		0.905			2.596666667			36

19	a	0.5	3	0.05	0.025				32	36	
		0.8	3	0.05	0.04				32	36	
		1.1	6	0.1	0.11				32	36	
		1.4	6	0.1	0.14				32	36	
		1.7	6	0.1	0.17				32	36	
		2	6	0.1	0.2				32	36	
		2.3	2	0.033333333	0.076666667	104/70	112		32	36	
	b	1	2	0.033333333	0.033333333				32	36	
		1.3	2	0.033333333	0.043333333				32	36	
		1.6	2	0.033333333	0.053333333				32	36	
		1.9	2	0.033333333	0.063333333				32	36	
		2.2	2	0.033333333	0.073333333	128/81	88		32	36	
			42		1.026333333						
20	a	0.4	4	0.06666667	0.026666667				32	36	
		0.8	4	0.06666667	0.053333333				32	36	
		1.2	4	0.06666667	0.08				32	36	
		1.6	4	0.06666667	0.106666667				32	36	
		2	4	0.06666667	0.133333333	88/52	128		32	36	
	b	1	2	0.033333333	0.033333333				32	36	
		1.3	2	0.033333333	0.043333333				32	36	
		1.6	3	0.05	0.08				32	36	
		1.9	3	0.05	0.095				32	36	
	c	1.2	2	0.03333333	0.04				32	36	
		1.5	2	0.03333333	0.05				32	36	
		1.8	2	0.03333333	0.06				32	36	
		2.1	2	0.03333333	0.07				32	36	
		2.4	2	0.03333333	0.08	107/80	111		32	36	
			40		0.951666667						
21	a	0.5	2	0.03333333	0.016666667				24	30.6	
		0.8	2	0.03333333	0.026666667				24	30.6	
		1.1	2	0.03333333	0.036666667				24	30.6	
		1.4	6	0.1	0.14				24	30.6	
		1.7	8	0.13333333	0.226666667				24	30.6	
		2	4	0.06666667	0.133333333				24	30.6	
		2.3	4	0.06666667	0.153333333				24	30.6	
		2.6	4	0.06666667	0.173333333				24	30.6	
		2.9	4	0.06666667	0.193333333				24	30.6	
		3.2	4	0.06666667	0.213333333	112/75	98		24	30.6	
			40		1.313333333			3.293333333			33.8
22	a	0.5	3	0.05	0.025				24	30.6	
		0.8	3	0.05	0.04				24	30.6	
		1.1	3	0.05	0.055				24	30.6	
		1.4	3	0.05	0.07				24	30.6	
		1.7	3	0.05	0.085				24	30.6	
		2	6	0.1	0.2				24	30.6	
		2.3	1	0.01666667	0.038333333	90/64	115		24	30.6	
	b	1.5	4	0.06666667	0.1				24	30.6	
		1.9	4	0.06666667	0.126666667				24	30.6	
		2.3	4	0.06666667	0.153333333				24	30.6	
		2.7	5	0.08333333	0.225	108/74	103		24	30.6	
			38		0.893333333						
23	a	0.5	3	0.05	0.025				24	30.6	
		0.8	3	0.05	0.04				24	30.6	
		1.1	3	0.05	0.055				24	30.6	
		1.4	3	0.05	0.07				24	30.6	
		1.7	3	0.05	0.085				24	30.6	
		2	3	0.05	0.1				24	30.6	
		2.4	6	0.1	0.24				24	30.6	
		2.7	12	0.2	0.54				24	30.6	
		3	2	0.03333333	0.1	113/80	102		24	30.6	
			38		1.255						
24	a	0.5	2	0.03333333	0.016666667				24	30.6	
		0.8	3	0.05	0.04				24	30.6	
		1.1	2	0.03333333	0.036666667				24	30.6	
	b	0.7	2	0.03333333	0.023333333				24	30.6	
		1	2	0.03333333	0.033333333				24	30.6	
		1.3	2	0.03333333	0.043333333				24	30.6	
		1.6	1	0.01666667	0.026666667	100/65	109		24	30.6	
	c	0.5	2	0.03333333	0.016666667				32	36	
		0.8	2	0.03333333	0.026666667				32	36	
		1.1	2	0.03333333	0.036666667				32	36	
		1.4	2	0.03333333	0.046666667				32	36	
		1.7	2	0.03333333	0.056666667	136/72	109		32	36	
	d	0.8	2	0.03333333	0.026666667				40	53.9	
		1.1	2	0.03333333	0.036666667				40	53.9	
		1.4	2	0.03333333	0.046666667				40	53.9	
		1.7	1	0.01666667	0.023333333				40	53.9	
		2	1	0.01666667	0.033333333	125/81	100		40	53.9	
			32		0.575			2.725333333			32.4

25	a	0.4	3	0.05	0.02				24	30.6
		0.7	5	0.083333333	0.058333333				24	30.6
		1	2	0.033333333	0.033333333	102/64	114		24	30.6
	b	0.6	3	0.05	0.03				32	36
		0.9	3	0.05	0.045				32	36
		1.2	3	0.05	0.06				32	36
		1.5	3	0.05	0.075	90/56	117		32	36
	c	0.8	2	0.033333333	0.026666667				32	36
		1.4	6	0.1	0.14				32	36
		1.8	3	0.05	0.09				32	36
			33		0.578333333					
26	a	0.4	4	0.06666667	0.026666667				32	36
		0.7	4	0.06666667	0.046666667				32	36
		1	4	0.06666667	0.066666667				32	36
		1.3	4	0.06666667	0.086666667				32	36
		1.6	4	0.06666667	0.106666667				32	36
		1.9	4	0.06666667	0.126666667				32	36
		2.2	4	0.06666667	0.146666667				32	36
		2.5	2	0.033333333	0.083333333	108/78	90		32	36
	b	0.5	1	0.01666667	0.008333333				40	53.9
		1	2	0.033333333	0.033333333				40	53.9
		1.5	2	0.033333333	0.05				40	53.9
		2	2	0.033333333	0.066666667				40	53.9
		2.5	3	0.05	0.125	121/75	75		40	53.9
			40		0.973333333					
27	a	0.5	5	0.083333333	0.041666667				40	53.9
		1	5	0.083333333	0.083333333				40	53.9
		1.5	10	0.16666667	0.25				40	53.9
		2	6	0.1	0.2	100/60	107		40	53.9
	b	0.7	2	0.033333333	0.023333333				40	53.9
		1.2	2	0.033333333	0.04				40	53.9
		1.7	4	0.06666667	0.113333333				40	53.9
		2.2	4	0.06666667	0.146666667				40	53.9
		2.5	2	0.033333333	0.083333333	122/78	96		40	53.9
			40		0.881666667			2.533333333		44.7
28	a	0.4	1	0.01666667	0.006666667				40	53.9
		0.7	3	0.05	0.035				40	53.9
		1	3	0.05	0.05				40	53.9
		1.3	3	0.05	0.065				40	53.9
		1.6	6	0.1	0.16				40	53.9
		1.9	3	0.05	0.095				40	53.9
		2.2	3	0.05	0.11				40	53.9
		2.5	3	0.05	0.125		118		40	53.9
	b	0.8	3	0.05	0.04				40	53.9
		1.5	3	0.05	0.075				40	53.9
		2	3	0.05	0.1				40	53.9
		2.5	4	0.06666667	0.166666667	117/73	108		40	53.9
			38		1.028333333					
29	a	0.3	1	0.01666667	0.005				40	53.9
		0.6	2	0.033333333	0.02				40	53.9
		0.9	3	0.05	0.045				40	53.9
		1.2	3	0.05	0.06				40	53.9
		1.5	8	0.13333333	0.2	107/71	96		40	53.9
	b	0.9	3	0.05	0.045				40	53.9
		1.2	6	0.1	0.12				40	53.9
		1.5	6	0.1	0.15				40	53.9
		1.8	4	0.06666667	0.12	96/64	125		40	53.9
			36		0.765					
30	a	0.5	3	0.05	0.025				40	53.9
		0.9	3	0.05	0.045				40	53.9
		1.3	3	0.05	0.065				40	53.9
		1.7	6	0.1	0.17				40	53.9
		2.1	3	0.05	0.105				40	53.9
		2.5	2	0.033333333	0.083333333	96/64	112		40	53.9
	b	0.9	3	0.05	0.045				40	53.9
		1.3	3	0.05	0.065				40	53.9
		1.7	3	0.05	0.085				40	53.9
		2.1	6	0.1	0.21	103/64	112		40	53.9
			35		0.898333333			2.691666667		53.9
31	a	0.5	4	0.06666667	0.033333333				40	53.9
		0.9	4	0.06666667	0.06				40	53.9
		1.3	4	0.06666667	0.086666667				40	53.9
		1.7	8	0.13333333	0.226666667				40	53.9
		2.1	5	0.08333333	0.175	78/56	111		40	53.9
	b	0.9	3	0.05	0.045				40	53.9
		1.5	3	0.05	0.075				40	53.9
		2.1	4	0.06666667	0.14				40	53.9
			35		0.841666667					

32	a	0.5	3	0.05	0.025				40	53.9	
		0.9	3	0.05	0.045				40	53.9	
		1.3	6	0.1	0.13				40	53.9	
		1.7	3	0.05	0.085				40	53.9	
		2.1	3	0.05	0.105				40	53.9	
		2.5	3	0.05	0.125	108/68	119		40	53.9	
	b	0.8	5	0.083333333	0.066666667				40	53.9	
		1.1	5	0.083333333	0.091666667				40	53.9	
		1.4	5	0.083333333	0.116666667				40	53.9	
		1.7	2	0.033333333	0.056666667	121/75	104		40	53.9	
			38		0.846666667						
33	a	0.3	2	0.033333333	0.01				40	53.9	
		0.7	2	0.033333333	0.023333333				40	53.9	
		1.1	6	0.1	0.11				40	53.9	
		1.5	6	0.1	0.15				40	53.9	
		1.9	4	0.06666667	0.126666667	96/56	118		40	53.9	
	b	0.5	2	0.033333333	0.016666667				40	53.9	
		1	4	0.06666667	0.066666667				40	53.9	
		1.5	6	0.1	0.15				40	53.9	
		2	2	0.033333333	0.066666667				40	53.9	
		2.3	4	0.06666667	0.153333333	117/76	108		40	53.9	
			38		0.873333333			2.561666667			53.9
34	a	0.5	3	0.05	0.025				40	53.9	
		0.9	3	0.05	0.045				40	53.9	
		1.3	3	0.05	0.065				40	53.9	
		1.7	6	0.1	0.17				40	53.9	
		2.1	4	0.06666667	0.14				40	53.9	
	b	1	5	0.083333333	0.083333333				40	53.9	
		1.5	5	0.083333333	0.125				40	53.9	
		2	5	0.083333333	0.166666667				40	53.9	
		2.5	5	0.083333333	0.208333333	112/68	105		40	53.9	
			39		1.028333333						
35	a	0.3	2	0.033333333	0.01				40	53.9	
		0.7	8	0.133333333	0.093333333				40	53.9	
		1.1	6	0.1	0.11				40	53.9	
		1.5	10	0.16666667	0.25				40	53.9	
		1.9	9	0.15	0.285				40	53.9	
			35		0.748333333						
36	a	0.6	3	0.05	0.03				40	53.9	
		0.9	6	0.1	0.09				40	53.9	
		1.2	3	0.05	0.06				40	53.9	
		1.5	3	0.05	0.075				40	53.9	
		1.8	3	0.05	0.09				40	53.9	
		2.1	3	0.05	0.105	112/67	104		40	53.9	
	b	0.5	4	0.06666667	0.033333333				32	36	
		0.8	4	0.06666667	0.053333333				32	36	
		1.1	4	0.06666667	0.073333333				32	36	
		1.4	4	0.06666667	0.093333333				32	36	
		1.6	2	0.033333333	0.053333333	126/79	94		32	36	
			39		0.756666667			2.533333333			51.7

BWB Treadmill Training
Eva Buttignon

Session	Interval	Speed (km/hr)	Time (min)	Time (hr)	Distance (km)	BP	HR	3 Session km	BWS (kg)	%wt Supported	3 Session avg % wt Supported
1	a	8.1	5	0.083333	0.675				46	52.2	
	b	8.3	8	0.133333	1.04				46	52.2	
	c	8.5	10	0.166667	1.483333333				46	52.2	
	d	8.8	7	0.116667	1.07				32	40.9	
			30		4.201666667						
2	a	8.4	5	0.083333	0.833333333				32	40.9	
	b	8.8	5	0.083333	0.95				32	40.9	
	c	8.4	8	0.133333	1.053333333				32	40.9	
	d	8.8	8	0.133333	1.08				32	40.9	
	e	8.8	5	0.083333	0.666666667				32	40.9	
			31		4.213333333						
3	a	8.8	8	0.1	0.96				32	40.9	
	b	8.8	8	0.133333	1.106666667				32	40.9	
	c	8.8	7	0.116667	0.963333333				32	40.9	
	d	8.8	5	0.083333	0.666666667				32	40.9	
	e	8.8	5	0.083333	0.95				32	40.9	
			31		4.396666667			8.610005			42.59
4	a	8.5	9	0.15	0.975				32	40.9	
	b	8.9	10	0.166667	1.15				32	40.9	
	c	8.7	8	0.1	0.87				32	40.9	
	d	8.8	8	0.133333	1.106666667				32	40.9	
			33		4.401666667						
5	a	8.4	3	0.05	0.42				32	40.9	
	b	8.8	10	0.166667	1.4				32	40.9	
	c	8.8	7	0.116667	1.07				32	40.9	
	d	8.8	9	0.15	1.09				32	40.9	
			29		3.28						
6	a	8.5	14	0.233333	1.190666667				32	40.9	
	b	8.7	12	0.2	1.74				32	40.9	
	c	8.7	11	0.183333	1.579333333				32	40.9	40.9
			37		4.385			1.00666666			
7	a	8.5	7	0.116667	0.958333333				32	40.9	
	b	8.7	13	0.216667	1.851666667				32	40.9	
	c	8.7	13	0.216667	1.851666667				32	40.9	
	d	8.7	8	0.133333	1.093333333				32	40.9	
			41		6.455						
8	a	8.7	18	0.3	2.61				32	40.9	
	b	8.8	17	0.283333	2.526666667				32	40.9	
			35		5.136666667						
9	a	8.9	22	0.366667	3.33				32	40.9	
	b	8.9	20	0.333333	3				32	40.9	
			42		6.63			1.521666666			40.9
10	a	8.9	20	0.333333	3				32	40.9	
	b	8.7	8	0.133333	1.083333333				32	40.9	
	c	1	9	0.15	2.15				32	40.9	
			37		6.543333333						
11	a	11	19	0.316667	3.483333333				32	40.9	
	b	12	12	0.2	2.4				32	40.9	
	c	13	8	0.133333	1.773333333				32	40.9	
			39		7.656666667						
12	a	14	12	0.2	2.8				32	40.9	
	b	15	13	0.216667	3.325	108			32	40.9	
	c	16	8	0.133333	2.213333333				32	40.9	
			33		8.818333333			2.123333333			40.9
13	a	13	10	0.166667	2.216666667				32	40.9	
		15	5	0.083333	1.125				32	40.9	
		18	5	0.083333	1.35	104			32	40.9	
	b	13	8	0.133333	1.773333333				32	40.9	
		15	5	0.083333	1.125				32	40.9	
		18	5	0.083333	1.35				32	40.9	
		2	2	0.033333	0.099666667	120			32	40.9	
			40		7.006666667						
14	a	12	6	0.1	1.32				32	40.9	
	b	08	4	0.066667	0.84				32	40.9	
		03	4	0.066667	0.95				32	40.9	
		12	5	0.083333	1.1				32	40.9	
		15	9	0.15	2.295				32	40.9	
		19	15	0.25	4.475	112			32	40.9	
			43		11.02						
15	a	03	1	0.016667	0.005				32	40.9	
		08	5	0.083333	0.95				32	40.9	
		09	3	0.05	0.645				32	40.9	
		12	17	0.283333	3.34				32	40.9	
		15	14	0.233333	3.35				32	40.9	
		18	2	0.033333	0.68				32	40.9	
			42		9.95			2.917666666			40.9
16	a	07	7	0.116667	0.681666667				32	40.9	
		11	4	0.066667	0.673333333				32	40.9	
		14	14	0.233333	3.256666667				32	40.9	
	b	11	3	0.05	0.655				32	40.9	
		14	3	0.05	0.87				32	40.9	
		17	9	0.15	2.255	108			32	40.9	
			40		8.861666667						
17	a	08	6	0.1	0.96				32	40.9	
		09	6	0.1	0.99				32	40.9	
		12	8	0.1	1.12				32	40.9	
		15	6	0.1	1.35				32	40.9	
		19	3	0.05	0.99				32	40.9	
	b	09	1	0.016667	0.015				32	40.9	
		14	5	0.083333	1.116666667				32	40.9	
		15	4	0.066667	1.1	82			32	40.9	
			37		7.741666667						
18	a	07	5	0.083333	0.583333333				32	40.9	
		1	5	0.083333	0.083333333				32	40.9	
		13	10	0.166667	2.216666667				32	40.9	
		18	10	0.166667	2.266666667				32	40.9	
		19	7	0.116667	2.221666667				32	40.9	
		21	4	0.066667	0.14	120			32	40.9	
			41		9.645666667			2.53666666			40.9
19	a	05	3	0.05	0.225				32	40.9	
		09	10	0.166667	0.15				32	40.9	
		13	7	0.116667	0.151666667				32	40.9	
	b	13	7	0.116667	0.151666667				32	40.9	
		17	7	0.116667	0.196333333				32	40.9	

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			41		0 s521666667						
20	a	0 5	5	0 083333	0 0416666667				32	40 9	
		0 8	5	0 083333	0 066666667				32	40 9	
		1 1	12	0 2	0 22				32	40 9	
		1 4	7	0 116667	0 1633333333	108			32	40 9	
	b	1 1	4	0 066667	0 0733333333				32	40 9	
		1 3	5	0 083333	0 1083333333				32	40 9	
		1 5	4	0 066667	0 1				32	40 9	
		1 8	3	0 05	0 09				32	40 9	
			45		0 883333333						
21	a	0 5	5	0 083333	0 0416666667				32	40 9	
		0 8	5	0 083333	0 066666667				32	40 9	
		1 1	5	0 083333	0 091666667				32	40 9	
		1 4	5	0 083333	0 1 116666667				32	40 9	
		1 7	15	0 25	0 425				32	40 9	
		2	10	0 166667	0 333333333	88			32	40 9	
			45		1 075		2 85999998				40 9
22	a	0 5	4	0 066667	0 0333333333				40	32 2	
		0 8	4	0 066667	0 0533333333				40	32 2	
		1 1	5	0 083333	0 091666667				40	32 2	
		1 4	7	0 116667	0 1633333333				40	32 2	
		1 7	8	0 133333	0 226666667				40	32 2	
		2	8	0 133333	0 266666667				40	32 2	
		2 3	2	0 033333	0 076666667	84			40	32 2	
			38		0 911666667						
23	a	0 3	5	0 083333	0 025				32	40 9	
		0 7	5	0 083333	0 0583333333				32	40 9	
		1 1	5	0 083333	0 091666667				32	40 9	
		1 5	10	0 166667	0 25				32	40 9	
	b	1 2	5	0 083333	0 1				32	40 9	
		1 6	5	0 083333	0 1333333333				32	40 9	
		2	5	0 083333	0 166666667				32	40 9	
		2 2	4	0 066667	0 1466666667				32	40 9	
		2 4	3	0 05	0 12	36			32	40 9	
			47		1 291666667						
24	a	0 5	5	0 083333	0 0416666667				32	40 9	
		0 8	5	0 083333	0 066666667				32	40 9	
		1 1	5	0 083333	0 091666667				32	40 9	
		1 4	5	0 083333	0 116666667				32	40 9	
	b	1 4	5	0 083333	0 116666667				32	40 9	
		1 7	5	0 083333	0 141666667				32	40 9	
		2	5	0 083333	0 166666667				32	40 9	
		2 3	5	0 083333	0 191666667				32	40 9	
		2 5	2	0 033333	0 0953333333	80			32	40 9	
			42		1 916666667		3 018999999				44 31
25	a	0 5	5	0 083333	0 0416666667				32	40 9	
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		1 1	5	0 083333	0 091666667				32	40 9	
	b	1 4	5	0 083333	0 116666667				32	40 9	
		1 7	5	0 083333	0 141666667				32	40 9	
		2	5	0 083333	0 166666667				32	40 9	
		2 3	5	0 083333	0 191666667				32	40 9	
		2 6	3	0 05	0 13				32	40 9	
		2 9	4	0 066667	0 1933333333	90			32	40 9	
			42		1 14						
26	a	0 4	5	0 083333	0 0333333333				32	40 9	
		0 8	5	0 083333	0 066666667				32	40 9	
		1 4	7	0 116667	0 1633333333				32	40 9	
		1 7	3	0 05	0 065				32	40 9	
		2	5	0 083333	0 166666667				32	40 9	
		2 3	5	0 083333	0 191666667				32	40 9	
		2 6	5	0 083333	0 216666667				32	40 9	
		3	3	0 05	0 15	72			32	40 9	
			38		1 071333333						
27	a	0 5	4	0 066667	0 0333333333				32	40 9	
		0 9	4	0 066667	0 08				32	40 9	
		1 3	4	0 066667	0 0866666667				32	40 9	
		1 7	8	0 133333	0 2266666667				32	40 9	
		2 1	8	0 133333	0 28				32	40 9	
		2 5	4	0 066667	0 166666667				32	40 9	
		2 9	4	0 066667	0 1333333333				32	40 9	
		3 3	4	0 066667	0 22	90			32	40 9	
			40		1 206666667		3 47999998				40 9
28	a	0 5	2	0 033333	0 0166666667				32	40 9	
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		1 5	5	0 083333	0 125				32	40 9	
		2	3	0 05	0 1				32	40 9	
	b	2	5	0 083333	0 166666667				32	40 9	
		2 5	3	0 05	0 125				32	40 9	
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			31		1 016666667						
29	a	0 5	2	0 033333	0 0166666667				32	40 9	
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		1 1	2	0 033333	0 036666667				32	40 9	
		1 4	4	0 066667	0 093333333				32	40 9	
		1 7	4	0 066667	0 113333333				32	40 9	
		2	4	0 066667	0 133333333				32	40 9	
		2 3	4	0 066667	0 153333333				32	40 9	
		2 6	4	0 066667	0 173333333				32	40 9	
		2 9	4	0 066667	0 193333333				32	40 9	
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		1 4	12	0 2	0 28				40	32 2	
		1 7	3	0 05	0 065				40	32 2	
		2	3	0 05	0 1				40	32 2	
		2 3	3	0 05	0 115				40	32 2	
		2 8	8	0 1	0 28				40	32 2	
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		2	4	0 066667	0 133333333				32	40 9		
		2 3	4	0 066667	0 153333333				32	40 9		
		2 6	4	0 066667	0 173333333				32	40 9		
		0 2	3	0 05	0 01				32	40 9		
		2	4	0 066667	0 133333333				32	40 9		
		2 3	4	0 066667	0 153333333				32	40 9		
		0 2	2	0 033333	0 006666667				32	40 9		
		2 6	2	0 033333	0 046666667				32	40 9		
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			36		1 116666667							
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		2 2	2	0 033333	0 073333333				40	53 2		
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		1 6	2	0 033333	0 053333333				40	53 2		
		1 9	2	0 033333	0 063333333				40	53 2		
		2 2	2	0 033333	0 073333333				40	53 2		
		2 5	1	0 016667	0 041666667	98			40	53 2		
		0 2	2	0 033333	0 006666667				48	63 6		
		1 3	2	0 033333	0 043333333				48	63 6		
		1 6	2	0 033333	0 053333333				48	63 6		
		1 9	2	0 033333	0 063333333				48	63 6		
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		1 7	3	0 05	0 085				40	53 2		
		2 1	3	0 05	0 105				40	53 2		
		2 5	3	0 05	0 125	96			40	53 2		
		1 6	3	0 05	0 08				48	63 6		
		1 9	3	0 05	0 095				48	63 6		
		2 2	3	0 05	0 11				48	63 6		
		2 5	4	0 066667	0 166666667				48	63 6		
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		1 9	3	0 05	0 095				48	63 6		
		2 3	3	0 05	0 115				48	63 6		
		2 7	3	0 05	0 135				48	63 6		
		3 1	3	0 05	0 155				48	63 6		
		2 9	12	0 2	0 58				48	63 6		
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		0 7	3	0 05	0 035				40	53 2		
		1	3	0 05	0 05				40	53 2		
		1 3	3	0 05	0 065				40	53 2		
		1 6	3	0 05	0 08				40	53 2		
		1 9	3	0 05	0 095				40	53 2		
		2 2	3	0 05	0 11				40	53 2		
		0 4	1	0 016667	0 006666667	104			40	53 2		
		0 4	1	0 016667	0 006666667				48	63 6		
		0 9	1	0 016667	0 015				48	63 6		
		1 4	5	0 083333	0 116666667				48	63 6		
		1 9	5	0 083333	0 158333333				48	63 6		
		2 4	5	0 083333	0 2				48	63 6		
		2 9	3	0 05	0 145				48	63 6		
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			45		1 273333333		3 4766666					60 48
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		1 1	3	0 05	0 055				40			
		1 4	12	0 2	0 28				40			
		1 7	3	0 05	0 085				40			
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Appendix II

Subject Information Package, Consent Form, and Ethics Application

HAMILTON HEALTH SCIENCES CORPORATION

Information Package
Study: Bodyweight Supported Treadmill Training

Required Visits

Visit 1 Holbrook Building, Chedoke Hospital: explanation of study protocol, signing of consent form, medical history and 12 minute tilt-table test (screening procedure - must pass)

note: ASIA D subjects will not perform the tilt-table test; the remaining objectives of visit 1 will be combined into visit 2 for the ASIA D subjects

*Visit 2 Ivor Wynne Centre, McMaster University: resting blood pressure, blood lipid profile, quality of life questionnaire

#Visit 3 Nuclear Medicine, McMaster University Medical Centre: DEXA scan (body composition & weight), urine and blood sample for assessment of bone metabolism

Visit 4 Ivor Wynne Centre, McMaster University: videotaping of pre-training mobility, 1st training session

Visits 5-38 Ivor Wynne Centre, McMaster University: 1 hour training sessions

Visit 39 Ivor Wynne Centre, McMaster University: 36th training session, videotaping of post-training mobility (before fatigued)

*Visit 40 Ivor Wynne Centre, McMaster University: resting blood pressure, blood lipid profile, quality of life questionnaire

#Visit 41 Nuclear Medicine, McMaster University Medical Centre: DEXA scan, urine and blood sample

***subjects must arrive for visits #2 and #40 having fasted for the previous 12 hours**
#subjects must bring a sample of their first urination of the day to visits #3 and #41

Outline of Objectives, Procedures and Risks

You have been asked to participate in a study conducted by Rob Pineau (MSc candidate), Cathy Craven (MD), Neil McCartney (PhD), Audrey Hicks (PhD) and Dave Ditor (MSc candidate) that is investigating the effects of bodyweight supported treadmill training on blood lipid profiles, bone metabolism, body composition and quality of life in persons with spinal cord injury (SCI). This study will determine the effectiveness of a 3 month program of bodyweight supported treadmill training in reducing risk for coronary heart disease and osteoporotic fracture in a population that is susceptible to both conditions.

In able-bodied persons, research has shown that regular, endurance-type exercise leads to improved blood lipid profiles, specifically decreases in triglycerides and LDL cholesterol ("bad cholesterol") and increases in HDL cholesterol ("good cholesterol"). These changes correspond to a reduced risk for coronary heart disease. It has also been shown that weight-bearing activity positively influences bone mass in able-bodied persons. These relationships have not been thoroughly investigated in individuals with spinal cord injuries. This study will provide some preliminary information.

Based on previous research, it is anticipated that there will be a gradual recovery of some mobility as subjects progress through the 3 month training period. This recovery will be individual, and will depend upon the baseline type of SCI that each subject presents. There is however, no guarantee that improved mobility will result from this training program. If you do make gains, the bodyweight support that you receive will gradually decline across the training period. This means that you will be supporting more of your own bodyweight during treadmill training. This increase in weight-bearing activity may have the potential to stimulate positive changes in bone. The increased workload will also place more demands on the cardiovascular system and may bring about positive changes in blood lipid profile. The effects of 3 months of bodyweight supported treadmill training on quality of life will also be investigated.

The trainers are aware that the physical demands of the training protocol will be high. You need to provide feedback during the exercise sessions indicating when you are tired and require a break. A reduction in mobility will also signal that a rest period is needed. With this in mind, the 1 hour training sessions will consist of both exercise, and rest periods. It is anticipated that the amount of time spent training in each session will increase across the duration of the 3 month study.

The success of this experiment depends on the compliance of the subjects. It is important that you meet the requirement of 3 training sessions per week. The investigators realize that due to uncontrollable circumstances you may have to cancel a training session. If this is the case, please notify Rob as soon as possible and the session will be rescheduled. If you were planning any extended holidays (greater than 4 days) over the next three months, please inform Rob before you begin the study, as this excludes you as a candidate.

Participation in this study requires that the following measures be taken before, and at the end of the 12 week training period:

i) Blood lipid and glucose profile (total cholesterol + LDL and HDL subfractions, triglycerides and glucose)

You will be asked to provide a small blood sample from your fingertip to determine this information.

ii) Body composition

You will be asked to undergo a DEXA (dual energy x-ray absorptiometry) scan to assess body composition. The procedure will take place in Nuclear Medicine at McMaster University Medical Centre. The procedure usually takes less than 20 minutes and the exposure to radiation is approximately one tenth of that from a chest x-ray.

iii) Bone metabolism

You will be asked to provide a urine sample and a small blood sample to assess levels of deoxypyridinoline and osteocalcin respectively. Both are indicators of bone activity. The urine sample must be collected on the day of the DEXA scanning. The sample must be from your first urination of that morning. This sample must be brought to the DEXA scanning session. The blood sample will be withdrawn by syringe at McMaster University Medical Centre on the day of the DEXA appointment.

iv) Quality of life

You will be asked to complete a questionnaire that assesses quality of life.

To help prevent skin irritation, the investigators ask you to regularly check your skin in the groin and buttock areas where the harness provides support. Let Rob know if you are experiencing any discomfort or chafing. The trainers will need to adjust the harness straps as they pass across the groin to help ensure that pressure sores do not develop in this area.

Other potential risks associated with participation in this study include:

- *autonomic dysreflexia*: the likelihood of this occurring can be reduced with proper bladder and bowel management; you must immediately inform the trainers if you are experiencing any of the symptoms of autonomic dysreflexia (i.e. headache); if you were to experience symptoms of dysreflexia, exercise would be discontinued, you would be removed from the harness, and your blood pressure would be monitored; if symptoms persisted or worsened, a physician would be called

- *hypotension (low blood pressure)*: the likelihood of this occurring during training was reduced with the tilt-table screening procedure which excluded those individuals who could not tolerate being upright for 12 minutes; blood pressure will be monitored periodically during training to ensure that it does not fall too low; you must immediately inform the trainers if you begin to feel lightheaded; if you were to experience hypotension during training, you would be lowered to a sitting position

- *skeletal fracture*: due to the combination of osteoporosis and weight-bearing activity

- there is also a chance that you could *fall or be injured* during transfers and during treadmill training; the likelihood of this occurring is minimal, as the investigators are all well trained; there will always be two to three investigators present to assist with the exercise sessions

The investigators reserve the right to remove a subject from the study based on the following criteria:

i) pressure sore that is aggravated by treadmill training
ii) persistent illness
iii) poor tolerability of treadmill training
iv) poor attendance/compliance
v) inability to tolerate the harness
vi) Dr. Craven's request

We advise that you wear shorts, a t-shirt and running shoes to the training sessions.

Finally, you will receive compensation for your transportation costs following successful completion of the training program.

Important Contact Number

Rob Pineau 308-8366

HAMILTON HEALTH SCIENCES CORPORATION

McMASTER UNIVERSITY

DEPARTMENTS OF KINESIOLOGY & MEDICINE: PHYSICAL MEDICINE AND REHABILITATION DIVISION

CONSENT FORM

The Effects Of Bodyweight Supported Treadmill Training On Blood Lipid Profile, Bone Metabolism, Body Composition and Quality Of Life In Persons With Spinal Cord Injury

I, _____, consent to take part in a study conducted by Neil McCartney PhD, Audrey Hicks PhD, Cathy Craven MD, and MSc candidates Rob Pineau and Dave Ditor, which will examine the effects of 3 months of exercise training on blood lipid profile, bone turnover, body composition and quality of life.

I am aware that the exercise training will consist of bodyweight supported treadmill training and will take place 3 times per week, for a 12 week period. A harness will be suspended from the frame of the treadmill and fitted around my torso and legs to provide bodyweight support. Each training session will last approximately 1 hour and will be supervised by trained staff. If I am unable to attend a scheduled training session, I will contact Rob Pineau (308-8366), and the session will be rescheduled. The study details are described in the subject information package.

I certify that I have read and understood the contents of the information package including the pre- and post-training measures, the schedule of required visits, the potential risks associated with participation and the criteria for removal from the study.

I will notify the trainers immediately if I am experiencing symptoms of autonomic dysreflexia or if I am having any other problems during the training sessions. I will monitor my skin for pressure sores and any other sort of irritation that may be aggravated by the harness.

I also give consent to the investigators to videotape my mobility prior to, and following the training period.

My participation in this study is voluntary. I have been told that I may withdraw from the study at any time, even after signing this form, without prejudice. Any information that is collected about me during the study will be kept confidential, and if the results are published, I will not be identified in any way. Confidentiality ensures that only those persons involved with the study will have access to data that is collected.

I have received a copy of this consent form _____ (initial). A copy of this form will become part of my health records.

Name (print)	Signature	Date
Witness	Signature	Date

I have explained the nature of the study to the individual and believe he or she has understood it.

Name	Signature	Date

McMASTER UNIVERSITY

FACULTY OF HEALTH SCIENCES AND AFFILIATED INSTITUTIONS

APPLICATION FOR REVIEW BY COMMITTEE FOR ETHICS FOR RESEARCH

(Forms must be typewritten: Form is on disk for use with WP5.1)

A. GENERAL INFORMATION:

For Office Use at St. Joseph's Hospital:
M.C.C.#
R.P

1) Title of Project: The effects of bodyweight supported treadmill training on blood lipid profile, bone metabolism, body composition and quality of life in persons with spinal cord injury

2) NAME(S) & DEGREE(S) of INVESTIGATOR(S)	TITLE(S) or POSITION(S)	ADDRESS(ES)	TELEPHONE#
Rob Pineau	M.Sc. candidate	98 Ainslie Dr. Hamilton, ON. L8S 2K2	(905) 308-8366
(Principal/Local responsible investigator)	*After May 1 1999:*	*60 Markland Dr. Etobicoke, ON. M9C 1N3*	*(416) 695-1606*
Neil McCartney	Ph.D., Assistant Professor - Kines.	Ivor Wynne Center AB116	X24468
Audrey Hicks	Ph.D., Assistant Professor - Kines.	Ivor Wynne Center AB120	X24643
Cathy Craven	M.D., FRCPC, Research Fellow	Lyndhurst Hospital, 520 Sutherland Dr. Toronto, ON. M4G 3V9	(416) 422-5551 X1222
Dave Ditor	M.Sc. candidate	Ivor Wynne Center	X27390

3) **Proposed Date of Commencement:** April 5, 1999 **Completion:** July 5, 1999

4) **Indicate where research will be conducted:** **Hospital(s):** McMaster University Medical Centre
Community: Ivor Wynne Center, Biomechanics Lab

5) **Has application been reviewed by other hospital Research Committees? If yes, attach copy of decision.**
No

6) **Hospital Appointment:** Cathy Craven, MD: dual appointment at Chedoke and Lyndhurst Hospitals
* *When studies are being conducted in the hospital, and involve patients, the responsible investigator or a designate must have a hospital appointment*

7) **Status of Funding** *(check one)*:

Applied	[ ]	Date
Funded	[ ]	Date
Other	[ ]	Date
None	[x]	Date

* *For Hamilton Civic Hospitals Corporation, attach copy of Budget Summary*

8) **Research Sponsor/Funding Agency:** NSERC studentship **Amount:**
(if applicable)

9) **Support requested from Hospital Research Funds?** *(check one)* **Yes [ ] Date** **No [x]**

(Please refer to the MRC Guidelines on Research Involving Human Subjects, 1987, prior to completion of form)

B. PART I: BRIEF SUMMARY (four or five lines) OF PROPOSED RESEARCH FOR REVIEW BY LAY PERSONS AND BY MEMBERS OF MEDICAL ADVISORY COMMITTEE

A new approach to the rehabilitation of individuals with spinal cord injury (SCI) uses bodyweight supported treadmill walking. Persons with SCI are outfitted with a harness that is attached to a pully system. Consequently, they can be raised to a standing position on the treadmill. Depending on their clinical status, bodyweight is fully or partially supported by a counterbalance. The unloading of bodyweight, combined with the assistance of therapists, makes it easier to initiate and modulate walking patterns in these individuals. It is our intention to do a pilot study to assess the effects of three months of bodyweight supported treadmill walking in subjects with SCI. Before and after the training period we will evaluate blood lipid profiles, bone metabolism, body composition and quality of life.

PART II: BRIEF OUTLINE OF THE PROPOSED RESEARCH AND ITS OBJECTIVE:

We propose to train individuals with spinal cord injuries (SCI) for three months using bodyweight supported treadmill walking. The apparatus that will be used in this trial is a *Woodway* treadmill equipped with a harness, pulley system, and counterbalance. It is capable of raising and supporting subjects above the walking surface. The amount of bodyweight support that each individual receives will be adjusted in accordance with their clinical status and progress. Previous investigations have demonstrated that bodyweight supported treadmill walking is effective in improving locomotion in individuals with SCI, especially those with residual sensory and motor function below the level of spinal cord trauma. It is well documented that individuals with chronic SCI are prone to poor lipid profiles as well as muscle atrophy and osteoporosis below the lesion level. This protocol will provide some insight into the protective and remedial effects of habitual treadmill walking on serum lipids, bone turnover and body composition in SCI subjects. As this is a pilot study, we intend to recruit subjects that present a range in clinical status in order to assess the differential response to this form of training.

(Items 1-7 inclusive are not required by St. Joseph's Hospital if this information is in the protocol)

1) **Sample size:** (Indicate how the sample size was determined) n=6: ASIA classes B, C and D will each be represented by 2 subjects; one male and one female. The ASIA classification scale indicates the degree of residual sensory and motor function in an individual with spinal cord injury.

2) **Design:** longitudinal (12 week training period), pilot study

3) **Setting:** Biomechanics Lab; Ivor Wynne Center

4) **Participants/Subjects:** community dwelling individuals with traumatic SCI; ASIA classes B, C and D will each be represented by 2 subjects: one male and one female; subjects will be greater than 1 year post-injury, and between 18 and 55 years of age

5) **Interventions:** full or partial bodyweight supported treadmill walking; 3X/week; 12 weeks; the amount of time spent walking during each training session will depend on the progress of the subject, but will not exceed one hour

6) **End-Point:** completion of 36 treadmill walking training sessions

7) **Measurements:** blood lipid & glucose profile (total cholesterol + HDL & LDL subfractions, triglycerides, and glucose), biochemical markers of bone metabolism (osteocalcin & deoxypyridinoline), body composition (DEXA), quality of life inventory and self-selected walking speed

C. ESTIMATE OF THE RISKS AND BENEFITS OF THE PROPOSED RESEARCH:

1) What are the proposed benefits to the subjects, the scientific community and/or society that would justify asking subjects to participate? Insight into the viability and efficacy of bodyweight supported treadmill training in chronic SCI patients will be obtained. The protocol will determine if regular treadmill exercise improves risk factors for cardiovascular disease and if it offers any protection against osteoporosis in this cohort. The study will also determine if treadmill walking affects the quality of life ratings of persons with SCI. The range in clinical status of the subjects recruited for this pilot study (ASIA classes B, C and D) will help identify the group that best responds to this form of training. A further benefit may be improved ambulation for the subjects.

2) (a) What inducement or compensation is offered to subjects?
None

(b) Will they be reimbursed for expenses? Yes [x] No [ ]

If yes, provide details: The only expenses the subjects are expected to incur is transportation and/or parking. The subjects will be remunerated for this expense.

3) Comment on the risks to subjects involved in this study: There is a possibility that subjects may experience postural and/or exertional hypotension when they are upright and walking on the treadmill. To reduce the likelihood, potential subjects will be screened and excluded if they cannot remain vertical on a tilt table for 12 minutes. During the training sessions, blood pressure will be monitored in rest periods to ensure that it does not begin to decline. If a subject were to experience hypotension during training, he/she would immediately be lowered to a sitting position. It is also remotely possible that the treadmill walking protocol will trigger autonomic dysreflexia with a subsequent rise in blood pressure. Patients are familiar with the onset of this condition however, and will be instructed to notify the investigators if they are experiencing any symptoms (eg. headache). The subjects will also be asked to monitor their urinary and fecal movements to ensure that neither their bladder or rectum is distended during the training session, as either could trigger a dysreflexic response. If a subject began to experience any symptoms of dysreflexia, exercise would be discontinued and the subject would be removed from the harness and their blood pressure would be monitored. If symptoms persisted or worsened, a physician would be called.

D. PLAN FOR OBTAINING INFORMED CONSENT:
(Please refer to Instructions for Preparation of Consent Form, enclosed, prior to completion of this section)

1) Describe how subjects are to be recruited including use of advertisements. Potential volunteers will be recruited through advertisements posted at the SCI rehabilitation clinic (Chedoke site), at the local peer support group meeting (the Vertebraves), and at various sites at McMaster University.

2) Describe the relationship between the investigator(s) and the subject(s). Who will obtain consent? There is a possibility that a subject may have been or is currently under the care of Dr. Craven. It will be made clear that participation (or lack of) in this study will in no way affect current or future care.

3) Are subjects competent to consent? Yes [x] No [ ] If not, describe the alternate source of consent.

If a minor, describe the procedure to be used. All the subjects will be above the age of consent.

4) What procedures will be followed for subjects who wish to withdraw at any point during or after study?

Subjects are free to withdraw from the study at any point. If a subject decides to withdraw, his/her data will be destroyed.