# The Effect of Vibration Exercise on Aspects of Muscle Physiology and Muscular Performance

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# Abstract

It has been proposed that the increases in muscle force and power following acute vibration exercise are similar to that of several weeks of conventional resistance or explosive power training. Further, it has been purported that vibration exercise operates via a stretch-reflex response which elicits a small change in muscle length. However, despite its wide use there remain gaps of knowledge on aspects such as physiological effects, mechanism of action, clinical effects, and even details of regimens for particular therapeutic use. Therefore, the aim of this thesis was to investigate the acute effects of vibration exercise on muscle performance and to examine the physiological aspects of its use in the young and older people, and competitive athletes. This thesis reported that acute upper-body vibration enhanced concentric peak power, but it was not significantly greater than concentric (arm-cranking) exercise. When matched for metabolic rate, vibration exercise elevated muscle temperature more quickly than traditional forms of warm-up by cycling or passive heating, but there were no significant differences in the increase in muscle power between the interventions, which suggested that the interventions were temperature dependent. There was no apparent benefit in performing a shallow, fast tempo dynamic squat with vibration because muscle temperature, cardiovascular indices, and metabolic rate were increased by the same amount and rate without vibration. Further, the Jendrassik manoeuvre did not potentiate the metabolic rate in young or older adults when superimposed with vibration exercise and the patellar reflex was not enhanced after vibration exercise, but muscle twitch potentiation was evident. However, low frequency vibration exercise induced a small change in muscle length and increased muscle activation, suggesting that spinal reflexes were involved. In conclusion, vibration exercise with a static squat could be used as a warm-up modality after interval breaks, as it would incur a low metabolic cost and be time efficient. It appears that the increases in muscle performance from vibration exercise are not caused by a neurogenic potentiation because patellar tendon reflex showed no significant augmentation and muscle twitch properties were enhanced. Vibration exercise elicited a small increase in metabolic rate and cardiovascular indices. Given that a main objective of a balanced exercise programme is to increase aerobic capcity it would be unwise to completely substitute conventional aerobic exercise with vibration. However, when conventional aerobic exercise is not possible, for example, in aged, cardiovascular compromised persons, vibration exercise could be implemented at an early stage because it could provide a safe induction of a low level of cardiovascular strain. Vibration exercise has the potential to benefit sport, exercise, and health however, it should be used to compliment other modalities but it should never be used in preference or in isolation to other programmes.

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# List of Publications and Presentations

Chapters 2 to7 have been submitted to international peer-reviewed journals. The details of these publications are as follows

***Peer Reviewed Journals***

***Cochrane D.J.***, Stannard S.R., Walmsley A., Firth E.C. (2008). The acute effect of vibration exercise on concentric muscular characteristics. *Journal of Science and Medicine in Sport* 11:527-534.

***Cochrane D.J.***, Stannard S.R., Sargeant T.J., Rittweger, J. (2008). The rate of muscle temperature increase during acute whole-body vibration exercise. *European Journal of Applied Physiology* 103:441-448.

***Cochrane D.J***., Stannard S.R., Firth E.C., Rittweger, J. (2010). The comparison of muscle temperatures during static and dynamic squatting with and without acute vibration exercise. *Clinical Physiology and Functional Imaging* (*In Press*).

***Cochrane D.J***., Sartor F., Winwood K., Stannard S.R., Narici M., Rittweger J. (2008). A comparison of the acute physiological effects of acute whole-body vibration exercise in young and older people. *Archives of Physical Medicine & Rehabilitation* 89:815-821.

***Cochrane D.J***., Stannard S.R., Loram I., Rittweger J. (2009). Changes in joint angle, muscle-tendon complex length, muscle contractile tissue displacement and modulation of EMG activity during acute whole-body vibration. *Muscle Nerve* 40:420-429.

***Cochrane D.J***., Stannard S.R., Firth E.C., Rittweger, J. (2010). Acute whole-body vibration elicits post-activation potentiation. *European Journal of Applied Physiology* 108:311-319.

***Conference Papers***

***Cochrane D.J.***, Stannard S.R., Sargeant TJ., Rittweger J. (2008). The rate of muscle temperature increase during acute vibration exercise. Australian Association for Exercise and Sports Science, 27-30 March 2008, Melbourne, Australia.

***Cochrane D.J.*** (2008). Warming up with vibration exercise – Does it increase muscle temperature? Massey University Post Graduate Sport Research Conference March 31-April 1, Palmerston North, New Zealand.

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***Cochrane D.J***., Stannard S.R., Walmsley A., Firth E.C. (2008). The acute effect of vibration exercise on concentric muscular characteristics. Australian Association for Exercise and Sports Science 27-30 March, Melbourne, Australia.

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# List of Abbreviations

**A**

| | |
|---|---|
| $a$ | Acceleration |
| $A$ | Amplitude (peak-to-peak) |
| AC | Arm cranking |
| AVI | Audio video interleave |

**B**

| | |
|---|---|
| BM | Body mass |
| BB | Barbell |
| BP | Blood pressure |
| DB | Dumbbell |

**C**

| | |
|---|---|
| $Ca^{2+}$ | Calcium |
| CD | Contractile length |
| CMJ | Countermovement jump |
| CMJ-PP | Countermovement jump peak power |
| CMJ-Ht | Countermovement jump maximum height |
| CMV | Calf muscle volume |
| CYCL | Stationary cycling |

**D**

| | |
|---|---|
| DS+ | Dynamic squatting with vibration exercise |
| DS– | Dynamic squatting without vibration exercise |
| DS | Dynamic squatting |

**E**

| | |
|---|---|
| EMD | Electromechanical delay |
| EMG | Electromyography |
| $EMG_{rms}$ | Electromyography root-mean-square |

**F**

| | |
|---|---|
| $f$ | Frequency |

**G**

| | |
|---|---|
| $g$ | Gravitational acceleration |

**H**

| | |
|---|---|
| Hz | Hertz |

**J**

| | |
|---|---|
| JH | Jump height |
| JVC | Jendrassik maximum voluntary contraction |

**M**

| | |
|---|---|
| Mean $T_{sk}$ | Mean skin temperature |
| MG | Medial gastrocnemius |
| MIC | Five second maximal isokinetic cycle test |
| MIC-PP | Maximal isokinetic cycle peak power |
| MTC | Muscle tendon complex |
| MVC | Maximal voluntary contraction |

**P**

| | |
|---|---|
| PAP | Post-activation potentiation |
| PAQ | Physical activity questionnaire |
| PBP | Prone bench pull |
| PEH | Peri-event histograms |
| PF | Muscle twitch peak force |
| PJP | Peak jump power |
| PP | Peak power |

**R**

| | |
|---|---|
| RFD | Rate of force development |
| RH | Relative humidity |
| RL | Patellar reflex latency |
| RP | Reflex potentiation |
| RPE | Ratings of perceived exertion |
| RM | Repetition maximum |

**S**

| | |
|---|---|
| SOL | Soleus |
| SS+ | Static squatting with vibration |
| SS– | Static squatting without vibration |
| SS | Static squatting |
| SV | Side-alternating vibration |

**T**

| | |
|---|---|
| $\Delta T_m$ | Change in muscle temperature |
| $T_{amb}$ | Ambient temperature |
| $T_c$ | Core temperature |
| $\dot{T}_m$ | Rate of muscle temperature change |
| $T_{sk}$ | Skin temperature |
| TA | Tibialis anterior |
| TLS | Thermal leg sensation |
| TMS | Transcranial magnetic stimulation |
| TMV | Thigh muscle volume |
| TP | Twitch potentiation |
| TPF | Time to peak force |
| TVR | Tonic vibration reflex |

**V**

| | |
|---|---|
| VBX+ | With vibration exercise |
| VBX− | Without vibration exercise |
| VBX | Vibration exercise |
| VJ | Vertical jump |
| VL | Vastus lateralis |
| $\dot{V}O_2$ | Rate of oxygen uptake |
| VV | Vertical synchronous vibration |

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# Chapter 1 – Introduction

## 1. Background

Vibration can be traced back to the time of the ancient Greeks who used it for therapeutic and health benefits. To hasten the recovery of injuries, a sheet of fabric was wrapped around the injured body part, with one end tied to a flexible saw to transmit mechanical vibrations. In the sixteenth century, the Japanese used percussion vibration and pressure to relax rigid and spasmodic muscle contractions. In the 1880s physician John Kellogg built vibration machines such as a chair, platform, and hand-held devices that were used to cure patients suffering from constipation, headaches, and lower back pain. However, Kellogg never performed any experimental trials to validate his claim. Whedon and co-workers (1949) were the first to conduct research on vibration, and reported that a custom built vibrating bed was capable of preventing bone loss by 50% in healthy bedridden participants. Numerous experts of that time criticised the methodology of the study and it wasn't until the 1960s that vibration resurfaced in sport and exercise disciplines. From 1960 to 1980 vibration was used extensively in sport by Eastern bloc countries, but the findings were kept a secret.

At best, these findings were often anecdotal and lacked scientific rigor. Scientific publications on the effectiveness and possible benefits of such a modality were scarce with only two publications (Atha & Wheatley, 1976; Nazarov & Spivak, 1985) cited from the 1970s and 1980s. Due to the fall of communism, information on vibration technology became available and made its way across to Europe to train sportspeople. It was in the early 1990s that other European athletes began using this modality to enhance performance, especially in the areas of strength and power training and for exercise and health, with more than 10,000 machines being sold annually. Despite its wide use there remain gaps of knowledge on aspects such as physiological effects, mechanism of action, clinical effects, and even details of regimens for particular therapeutic use. Additionally, anecdotal negative reports of overuse of vibration exercise are surfacing, indicating that people are being harmed by its incorrect use.

When the studies described in this thesis commenced there were approximately thirty publications on vibration exercise, many of them not internationally peer-reviewed. It has been reported that vibration exercise increases power, strength, balance, and flexibility; however, many of the descriptions of such effects were no better than anecdotal, while other reported findings have not been reproduced, or even analysed using credible scientific methods. A lack of consensus exists regarding the effectiveness of vibration exercise, which is primarily due to

some commercial companies marketing products on gifted promises with little scientific merit. Various manufacturers have seized the opportunity to make a quick profit and acclaim vibration exercise for its performance-enhancing and curative qualities without it actually being trialled and presented in scientific publications. The sales of vibration machines have taken precedence over research, with many machines being sold without proper and safe advice for the client, and without scientific evidence of their effectiveness. In some cases the marketing departments of companies have used scientific findings from their opposition to sell machines to potential customers. This is not ideal given that some vibration machines operate in different ways.

Early research found that muscle force and power was potentiated after acute vibration exercise. It was postulated that the effects of vibration were synonymous to that of several weeks of conventional resistance or explosive power training (Bosco et al., 1998; Bosco et al., 1999c). This claim has been based on the assumption that neural factors are responsible for the increases in force and power, which are similar to those neural changes seen in the initial stages of conventional resistance and power training. Further, it has been purported that vibration exercise causes a stretch-reflex response which elicits a rapid but small change in muscle length (Cardinale & Bosco, 2003). Similarities to the tonic vibration reflex have been used to propose that the muscle spindle plays a role in activating the muscle (Bosco et al., 1999c; Cardinale & Bosco, 2003). It appears that vibration exercise may act directly on muscle to increase force and power. The central role that muscle plays in producing movement means a clear understanding of neuromuscular physiology is required. However, the scientific enquiry of vibration exercise has lagged behind its application, with equivocal evidence produced by the few well-controlled studies. The purpose of this thesis is to provide scientific evidence on aspects of muscle physiology and muscular performance, and to make a significant original contribution to the knowledge of vibration exercise for those working in health, sport, exercise and rehabilitation fields.

As part of this thesis, a cohort of studies were undertaken in an attempt to bridge the gap of knowledge that currently exists in vibration exercise and to examine muscle physiology aspects of vibration exercise in trained, young and older people. Vibration exercise is worthy of serious consideration, and the physiological responses of muscular performance deserve to be addressed. The following review critiques contemporary literature in order to identify reliable biological knowledge, on which to propose sensible hypotheses to be tested through the studies that will be addressed later in this thesis.

# 2. Literature Review

## *Introduction*

Muscle is central to any exercise, without it force or movement would not occur. Similarly, muscle is integral to understanding the neuromuscular and performance aspects of vibration exercise. To provide a theoretical framework and background for later discussion of researching vibration exercise and its acute responses, an understanding of muscle physiology is required. This review will explore the structure and microstructure of skeletal muscle, the contraction process and the recruitment, sensory receptors and reflex activity of skeletal muscle. Following this a distinction will be made between occupational vibration and vibration exercise, including how vibration exercise evolved. The different types of vibration machines (platforms, dumbbells) and the vibration parameters (frequency, amplitude, acceleration, duration, posture) will be examined. The current proposed mechanisms that underpin vibration exercise will be discussed, which will focus on the muscle spindle and neural mechanisms. The review will then explore the effects of vibration exercise on reflex and muscle activity, as well as its effect on metabolism, cardiovascular, hormonal, body temperature and muscular performance such as force, power, speed, and flexibility. Finally, the review will reveal areas requiring specific investigation by proposing hypotheses to be tested through a series of experimental studies.

## 2.1 Structure and Microstructure of Skeletal Muscle

### 2.1.1 Muscle Connective Tissue Organisation

Muscle provides a structure allowing force production and movement to occur. Every skeletal muscle is composed of muscle fibres that are compartmentalised by a series of connective tissue membranes. Encasing the entire muscle is a membrane known as the epimysium (Figure 1). Each muscle fibre is covered with connective tissue known as the endomysium where arterioles, venules and some intramuscular nerve branches are found. The muscle fibres are grouped together into bundles that form fascicle, held together by a connective tissue called perimysium, through which blood vessels and nerves pass. The key functions of these connective tissues are to keep the muscle fibres together, to maintain shape of the muscle belly, allow a pathway for blood vessels and nerves to supply muscle fibres, and to distribute forces to minimise muscle fibre damage (MacIntosh, Gardiner, & McComas, 2006).

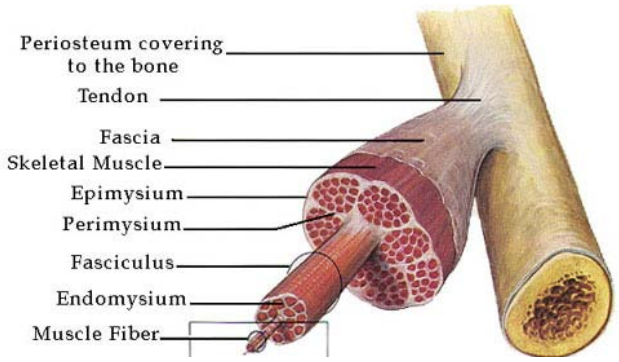



**Figure 1** The various connective tissue membranes of muscle (Van De Graaff & Fox 1995)

### 2.1.2 Contractile and Structural Proteins

The muscle fibre is composed tightly packed myofibrils and a contractile apparatus, the former being responsible for force generation and active shortening (Edman, 2003). Each myofibril is separated from its neighbour by mitochondria, sarcoplasmic reticulum and transverse tubular systems. The myofibril structure has two sets of protein filaments which are striated in appearance (Edman, 2003). The thicker fibrous filaments occupy the darker of the bands that are known as myosin filaments or A-filaments (Figure 2). In the A-band it has a M-filament containing M-protein (myomesin) and creatine kinase, which have struts that hold the myosin filaments together (MacIntosh et al., 2006). Part of a titin strand overlaps the myosin filament with the remaining part attaching to the I-band. The titin strands contribute to the elasticity of the muscle fibre when it is either passively stretched or when the sarcomere shortens (MacIntosh et al., 2006). It has been reported that titin is an organiser for myofibril assembly,

where it binds the two primary filaments of the sarcomere: actin and myosin (Littlefield & Fowler, 1998). The second set of filaments thinner and lighter in colour are known as actin filaments or I-filaments, and are located in the centre of the light band (MacIntosh et al., 2006). These filaments are anchored by the Z-disc,and extend from either side of the Z-disc and reach into the adjacent A-band where they overlap with the A-filaments (Hanson & Huxley, 1953).

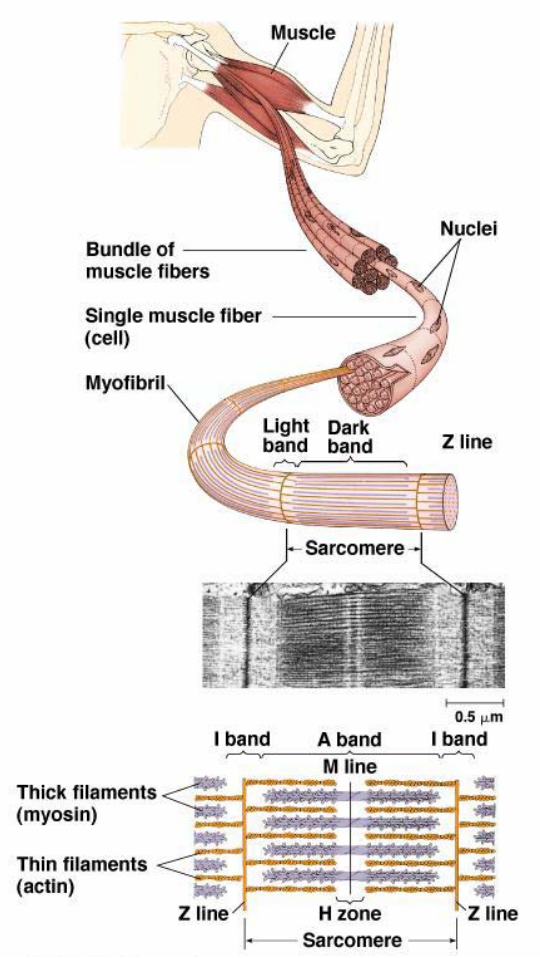



**Figure 2** Componets of a sarcomere (Marieb, 2009)

The A-filaments are surrounded by six I-filaments in a hexagonal arrangement that allows each I-filament to interact with three neighbouring A-filaments. This arrangement allows for stability during activity (Edman, 2003). During a contraction the A-filaments make contact with the I-filaments by molecular cross-bridges, which slide opposing I-filaments toward each other within each sarcomere, which diminishes the widths of the H-zone and the I-band. The I-filaments contain two additional regulatory proteins known as troponin and tropomyosin and a strengthening protein called nebulin. Troponin is thought to be composed of three subunits namely troponin-I (inhibitory), troponin-C (calcium binding) and troponin-T (tropomyosin binding). At rest, tropomyosin covers the seven actin monomers which prevent any actin-myosin interaction.

The I-filaments require positional support, which is achieved by nebulin and from their insertions into the Z-disc in the centre of each I-band (MacIntosh et al., 2006). The Z-discs are mainly composed of proteins namely, α-actinin, desmin, vimentin, and synemin. The α-actinin is thought to attach the ends of the actin filaments on one side of the Z-disc to those on the opposite side, while desmin, vimentin, and synemin are involved in the transverse connections of the Z-disc that keeps the myofibrils in register within a single muscle fibre (MacIntosh et al., 2006).

#### *2.1.2.1 Myosin Filament*

The A-filament consists of a globular head (S1 fragment) and ATPase activity, which is the portion that combines with actin (Figure 3). The S2 fragment contains one pair of myosin heavy chains (MHC's), two pairs of myosin light chains (MLC's) and a hinge region. The two pairs of MLC's are known as regulatory and essential myosin chains, which are located near the myosin head sub-fragment of the heavy chain, with the essential MLC being proximal to the head region (Figure 3). Both MLC's are wrapped around the lever arm of the myosin head sub-fragment, where they are in a position to transmit movements of the myosin head to the filamentous backbone of the myosin molecule (Figure 3). The essential MLC's are important for the stability of the myosin molecule, while the regulatory MLC's are phosphorylable and can be dissociated (Gordon, Homsher, & Regnier, 2000). The hinge region assists in bringing the cross-bridge to the surface of the A-filament (MacIntosh et al., 2006).

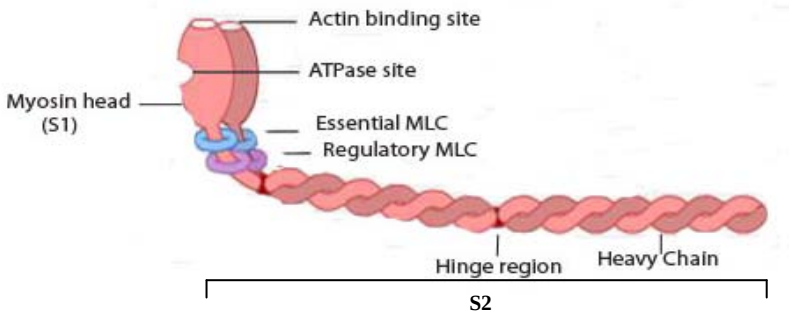



**Figure 3** Components of myosin filament (Sheffield, 2009)

The function of both the essential and regulatory MLC's is to stabilise the myosin head during force development (Gordon et al., 2000). Using a unique motility assay Lowey, Waller, and Trybus (1993) demonstrated that when light chains were removed from the myosin molecules the movement of actin across the myosin was reduced to zero. When one or the other light chains were added, the velocity of shortening was partially restored, however when both MLC's were added the original velocity was fully restored. Likewise, Vanburen *et al*. (1994) reported that when MLC's were removed, the force per cross-bridge was reduced by half,

suggesting that essential light chains may influence the maximal velocity of shortening. Research conducted by Metzger and Moss (1992) found that removing the regulatory MLC increased $Ca^{2+}$ sensitivity of the contractile proteins. Based on these findings researchers suggest that the phosphorylation of the regulatory light chains may contribute to an active potentiation.

### *2.1.2.2 Actin Filament*

The I-filament consists primarily of contractile actin protein and regulatory protein of tropomyosin and troponin. The actin consists of monomers known as G-actin (globular) and F-actin (filamentous) (Figure 4). The actin protein has a binding site that when exposed, attaches the myosin cross-bridge (Gordon et al., 2000). The cycling of the cross-bridges causes the development of muscular force. The filamentous actin is arranged in an α-helical arrangement, which creates a groove along the thin filament's length. In this groove lies the regulatory protein tropomyosin which blocks the binding site on actin for myosin during resting conditions (Brooks, Fahey, & Baldwin, 2005).

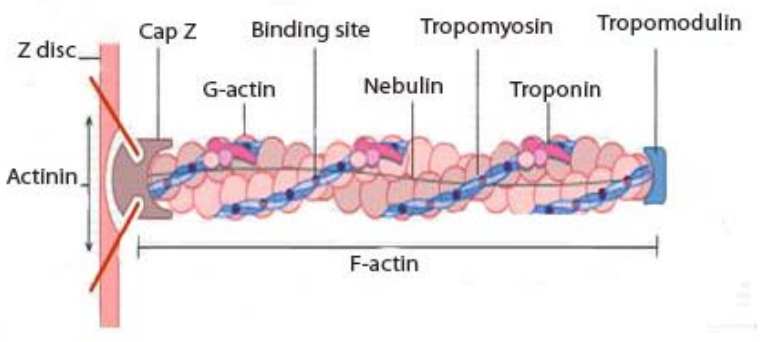



**Figure 4** Components of actin filament (Sheffield, 2009)

Troponin is made up of three subunits, which work together in moving tropomyosin away from the active binding sites on actin. In resting conditions, the tropomyosin covers the myosin-binding site on actin. Troponin-T (troponin-binding subunit) binds loosely onto the tropomyosin to prevent it from moving off the actin and troponin-I (troponin-inhibitory subunit) also positions tropomyosin on the binding site. When the intracellular $Ca^{2+}$ concentration rises to a critical level, four molecules of $Ca^{2+}$ bind to troponin-C causing the entire three-subunit configuration to change. The troponin-T then binds tightly to tropomyosin and the entire troponin protein physically moves on the actin. Once the myosin-binding site is exposed the myosin head is permitted to insert, which creates the cross-bridge (Brooks et al., 2005).

### 2.1.3 Sarcoplasmic Reticulum and Tubular Systems

Every myofibril is enveloped in an interlacing network containing the sarcoplasmic reticulum (SR) and transverse tubules (T-tubules). The function of the SR is to release $Ca^{2+}$ into the cytosol around the myofibrils, where it combines with the troponin C and allows contraction but when the electrical excitation ceases $Ca^{2+}$ in cytoplasm decreases and muscle relaxes. According to Toyoshima (2009), the SR has both $Ca^{2+}$ releasing channels and $Ca^{2+}$ ATPase pumps. The T-tubules form narrow channels which lie at the junctions of the A and I bands at each end of the thick filaments of the myofibril (Figure 5).

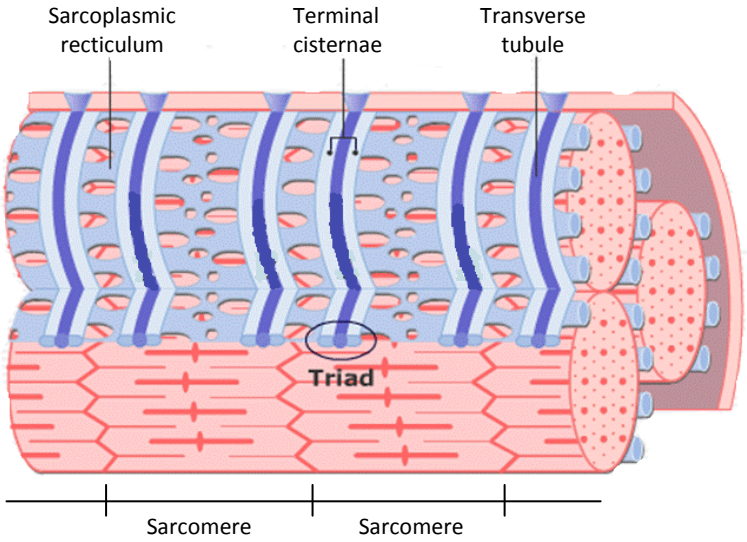



**Figure 5** Structure of sarcoplasmic reticulum (Sheffield, 2009)

The primary function of the T-tubules is to conduct electrical impulses from the surface to the interior of the muscle fibre, which initiate the release of $Ca^{2+}$ from the lateral sacs of the SR. This was first noted after application of weak stimulating currents at different points along the surface of frog muscle where an electrode tip was placed on the I-band of the T-tubule causing the myofibril contract (Huxley & Taylor, 1958). These protein structures were identified as junctional feet that straddled the gap between the T-tubule and the SR. They were sensitive to voltage changes that caused depolarisation of the T-tubule membrane, which triggered the opening of the $Ca^{2+}$ channels in the terminal cisternae.

## 2.2 The Contraction Process of Skeletal Muscle

### 2.2.1 Neuromuscular Junction and Post-Synaptic Membrane

Muscle contraction involves the central nervous system (CNS) sending signals to neurons in the spinal cord which travel along the efferent fibre. These fibres branch, and form terminals on the muscles that they innervate. Each terminal branch runs along a shallow groove in the muscle fibre surface. The sarcolemma in the junctional grooves is highly folded and is referred to as motor end plates. The tips of the terminal branches known as synaptic bulbs contain mitochondria and secretary vesicles filled with a neurotransmitter known as acetylcholine (ACh). A non-myelinated cell covers the terminal branches and a small space exists between the synaptic bulb and sarcolemma, which is known as the synaptic cleft. When the action potential arrives at the terminal branches of the motoneuron the $Ca^{2+}$ channels in the synaptic bulbs open in response and $Ca^{2+}$ diffuses into the neuron (MacIntosh et al., 2006).

The influx of $Ca^{2+}$ causes the synaptic vesicles to fuse with the neuron membrane and release ACh into the synaptic cleft, which diffuses across the cleft and binds to the receptors in the motor end plate of the muscle fibre. This causes the opening of ion channels, which temporarily allows $Na^{+}$ to enter the muscle fibre and $K^{+}$ to exit (Brooks et al., 2005). The gain of positive ions within the muscle fibre causes the sarcolemma to depolarise. When the neurotransmission of ACh stops and the residual ACh is catabolised by acetylcholineterase (AChE), the ion channels located on the sarcolemma close. This blocks the pathways through the sarcolemma for $Na^{+}$ and $K^{+}$ ions and no further depolarisation can take place. After the channels close, the $Na^{+}/K^{+}$ pump in the sarcolemma requires ATP to redistribute the $Na^{+}$ and $K^{+}$. With each ATP, three $Na^{+}$ move out of the muscle fibre and two $K^{+}$ move in; this unequal pumping brings the synaptic sarcolemma membrane potential back to its resting level (Jones, Round, & de Hann, 2004).

Neural stimulation of the muscle fibre opens the $Na^{+}$ channels (via the neurotransmitter [ACh] binding to the ACh receptors on the sarcolemma of the motor end-plate). The influx of $Na^{+}$ through the open channel depolarises the area producing, an end-plate potential (EPP), which was first described by Fatt and Katz (1952). The EPP generates action potentials in the sarcolemma next to the end-plate, which propagates along the sarcolemma and enters the muscle fibre at the T-tubules. As the action potentials travel down the T-tubules the calicum channels in the nearby terminal cisternae of SR open and $Ca^{2+}$ diffuses into the surrounding sarcoplasm. The $Ca^{2+}$ binds to the troponin proteins embedded along the actin filaments and the contraction process begins.

### 2.2.2 Sliding Filament Theory

The classical works of Hanson and Huxley (1953), and Huxley, (1953) found that actin and myosin filaments were constant in length during muscle contraction but the region of overlap between the two filaments changed with fibre length. They suggested that muscle contraction was cyclic and involved a sliding movement of myosin and actin. In support of this, Edman (2003) has reported that when myosin bridges make repeated contacts with adjacent thin filaments a force ensues from its contraction. However, the precise mechanism of how the force is generated by the cross-bridge remains equivocal, as the precise detail of cross-bridge action is not known. Previous works of Gordon *et al*. (2000) suggest a connection forms between the globular head (S1) of the bridge and an actin site that leads to a conformational change within the head region, which creates a strain on the shaft of the bridge. This causes the thin filament to move further into the thick filament, producing a force. The cross-bridge head is then detached from the thin filament, which occurs as an ATP molecule binds to the myosin head. The ATP is rapidly split and the bridge resumes its original shape. The bridge is then ready to attach again to the actin filament for a new cycle of contraction. For each cycle a cross-bridge requires the hydrolysis of one ATP molecule, which serves as an immediate source of energy for the contractile process (Huxley, 1990). But some researchers have suggested that multiple cycles are performed for each ATP hydrolysed (Yamaguchi, Takaki, Matsubara, Yasuhara, & Suga, 1996).

The number of cross-bridges formed is determined by the degree of activation of the contractile system and by the $Ca^{2+}$ ions (Edman, 2003). Bridges attach to the thin filament in a position where they are able to produce an active force. For an isometric contraction, cross-bridges dissociate spontaneously and are replaced with new ones. This keeps the total number of attached bridges at a given level, which results in some energy expenditure when, in fact, the muscle produces no work (Edman, 2003).

### 2.2.3 Twitch Contraction

When free $Ca^{2+}$ increases in response to a stimulus it binds to troponin and the cross-bridge is initiated. The smallest contractile response that can be elicited is the twitch response, which involves a time delay between the detection of the action potential to contractile activity (onset of shortening or force development). This is known as electromechanical delay (Isabelle, Sylvie, & Chantal, 2003). This delay occurs because of the time needed for conduction of activation into the T-tubules, release of $Ca^{2+}$ into the cytoplasm, binding of $Ca^{2+}$ to troponin, and initiation of cross-bridge cycling (MacIntosh et al., 2006). The T-tubule action potential results in brief opening of the ion gated channels and flow of $Ca^{2+}$ from the terminal cisternae into the cytoplasm. The released $Ca^{2+}$ binds to troponin C, permitting cross-bridge cycling.

Each troponin molecule can bind up to two $Ca^{2+}$, and the amount bound will depend on the magnitude and the duration of the calcium transient (MacIntosh et al., 2006). The number of cross-bridges involved in force development is determined by the amount of $Ca^{2+}$ binding to troponin. Calcium is removed from the cytosol by the pumping action of the $Ca^{2+}$ ATPase where cross-bridges stop cycling and the muscle relaxes (MacIntosh et al., 2006). The rate of twitch rise is dependent on the amount and speed of $Ca^{2+}$ released, the amount that binds to troponin, and how quickly the myosin heads bind with the actin. The rise and fall of twitch tension provides contractile aspects such as tension, peak rate of tension, contraction time, peak rate of relaxation, and half-relaxation time, which have been used to characterise contractile capabilities of muscle (Cannon, Kay, Tarpenning, & Marino, 2008).

## 2.3 Recruitment, Sensory Receptors, and Reflex Activity of Skeletal Muscle

### 2.3.1 Motor Unit Recruitment

A motor unit consists of the alpha (α) motoneuron in the spinal cord and the muscle fibres it innervates. The α motoneuron is the final destination of all descending and reflex inputs for muscle activity where its firing rate is caused by the membrane current of the motoneuron. According to Henneman's *et al*. (1965) classic work, motor unit recruitment occurs in an orderly fashion from small to large units, which has been coined the 'size principle'. According to the size principle, smaller cell bodies (slow motor units) have a higher level of dendritic input to generating an action potential compared to fast-fatigable and fast-fatigue resistant units, which are recruited last.

The size principle has been verified by Bawa and Lemon, (1993) and Rothwell *et al*. (1991). Using transcranial magnetic stimulation of the motor cortex, these investigators recruited motor units of the forearm and hand muscles and found that motor units were recruited in a normal and orderly fashion from small to large.

Motor unit recruitment and firing frequency are often dependent upon the required level of force and the velocity of action. In low force-generating activities low-threshold motor units are recruited, but as force increases recruitment of high-threshold fatigable units occurs (Henneman & Mendell, 1981). There is some debate that not all muscles follow the size principle. Grimby and Hannerz (1977) have suggested that preferential recruitment of high-threshold units occurs in relaxed muscle prior to twitch contraction. Additionally, it has been reported that during muscle lengthening, high-threshold motor units are selectively recruited with a simultaneous de-recruitment of slow-twitch motor units (Nardone, Romano, & Schieppati, 1989). Moreover, Romeny, Vandergon, and Gielen, (1982) have suggested that multi-directional movement may selectively activate certain motor units. It remains untested whether specific training, such as resistance and power training, can lead to selective recruitment, which is dependent on muscle action, velocity and movement pattern.

## 2.3.2 Receptors of Muscle, Tendon, Joint, Skin, and Others

### *2.3.2.1 Muscle Receptors – Muscle Spindle*

There are three groups of motoneurons: $\alpha$ motoneurons receive monosynaptic excitatory input from group Ia muscle spindle afferents that innervate extrafusal muscle; gamma ($\gamma$) motoneurons do not receive any Ia excitatory input but innervate intrafusal muscle fibres; and beta ($\beta$) motoneurons are capable of innervating both intra- and extrafusal muscle fibers (Bessou, Emonet-Dénand, & Laporte, 1965; Emonetdenand, Petit, & Laporte, 1992) by receiving monosynaptic group Ia excitation comparable to that in α motoneurons (Burke & Tsairis, 1977). The primary role of $\beta$ motoneurons is currently unknown because they are often difficult to identify and their influence cannot be removed without disrupting other structures.

Muscle spindles are located within the muscle where their primary function is to detect muscle length and rate change in length, which is important for reflex responses, proprioception, and learned movement (Matthews, 1972). The muscle spindle is composed of two intrafusal fibres: the nuclear bag fibres, which have nuclei distributed around the centre of the fibre; and the nuclear chain fibres, were the nuclei are distributed more evenly along the length of the fibre. In terms of structure, the bag fibres are normally thicker and longer than the chain fibres. Additionally, there are approximately two bag fibres in each spindle, compared to 12 chain fibres (MacIntosh et al., 2006).

The muscle spindles lie parallel to the extrafusal fibre and are serviced by two types of motor nerve endings. The plate ending is associated with the nuclear bag fibre and the trail ending is associated with the nuclear chain fibres. Both endings are connected to the γ efferent fibre, however nuclear chain fibres have their own γ efferent fibre whilst the nuclear bag fibre shares the other γ efferent fibre with the chain fibres. The sensory γ afferent fibre has a primary and secondary ending. The primary (annulospiral, Ia ) nerve fibre is entwined in the mid region of the bag and chain fibres. The secondary (flowerspray, II) mainly connects to the chain fibres but it also attaches to the bag fibres (Figure 6).

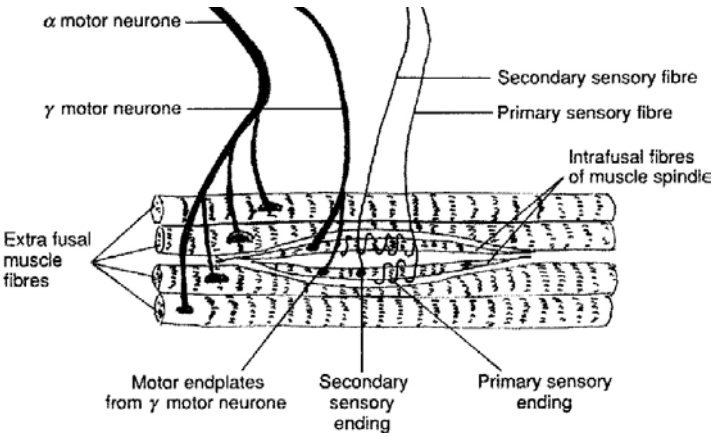



**Figure 6** The main components of muscle spindle (MacIntsoh et al., 2006)

The primary sensory endings (Ia) are sensitive to muscle length and velocity. Firing frequency is higher after a stretch than before. In rapid stretches the firing frequency is high, indicating its sensitivity to velocity, which is most likely due to the Ia fibre being connected to both the nuclear bag and chain fibre (Durbaba, Taylor, Ellaway, & Rawlinson, 2003). The secondary (II) endings are only responsive to muscle length.

The muscle spindle is capable of detecting both length and velocity of stretch, but its sensitivity can be enhanced by the γ efferent fibres of the central nervous system, which regulate the the length and sensitivity regardless of the overall length of the muscle. The adjustment to the γ efferent fibres enables the spindle to continuously monitor the length of muscles.

#### *2.3.2.2 Tendon Receptors – Golgi Tendon Organs*

Golgi tendon organs (GTO's) are located near the junction between the muscle and the tendon. They are high-threshold receptors, which exert an inhibitory effect on agonist muscles but facilitate antagonist muscles. GTO's primarily act as a protective mechanism, preventing excessive force from damaging or injuring muscles during contraction. They do this by sending sensory information from Ib motoneurons to the spinal cord where they synapse with inhibitory spinal interneurons, which reduces α motoneuron activity and prevents the full potential of force production of the agonists. This inhibition prevents excessive loading of the muscle from increased changes in stretch or tension. However, continual exposure to high levels of tension may reduce the GTO's sensitivity, an effect termed disinhibition. During resistance training, the high level of muscle tension reduces the inhibitory input and allows greater motor unit recruitment and higher firing frequency, in turn improving force production through increased agonist activation and concomitant changes in muscle stiffness (Aagaard, Simonsen, Andersen, Magnusson, & Dyhre-Poulsen, 2002a; Kamen, 1983).

#### *2.3.2.3 Joint and Skin Receptors*

Joint receptors are located in the articular capsules that enclose synovial joints, which are innervated by motoneurons of variable size. Joint receptors play a role in sensorimotor control and kinaesthesia, and are sensitive to changes in joint capsule tension, which corresponds to muscle force (Latash & Zatsiorsky, 2001). Skin has many receptors, such as the Meissner corpuscles and Merkle discs, located superficial to the skin surface. Merkle discs respond to vertical pressure, but not to lateral displacements, while Meissner corpuscles are sensitive to changes in pressure (Macefield, 2005). Ruffini endings, located in the dermis layer of the skin, respond to deformation of the skin. Pacinian corpuscles are located in the deep subcutaneous tissue of the skin, and react to mechanical deformation, such as vibration (Latash & Zatsiorsky,

2001). The human body has other receptors, such as thermoreceptors responding to temperature changes; nociceptors, responding to damaging stimuli that give rise to pain; chemoreceptors, responding to different molecules in the body; and mechanoreceptors that are sensitive to pressure. All of these receptors may contribute to the excitatory processes from vibration but at present, it is unknown what that contribution is.

### 2.3.3 Reflex Activity

Reflexes are automatic, involuntary, and predictable reactions to a sensory stimulus which can be modified by learning, and conscious effort. Reflex activities help to maintain muscle tone, body posture and movement. Reflex activity consists of a central processing unit, an afferent neuron that senses an external stimulus to induce muscle contraction. Reflexes containing one central synapse are known as monosynaptic; those involving two or three synapses are known as oligosynaptic; and those that involve many synapses are called polysynaptic. Each reflex involves a time delay between the reaction and the stimulus. This is known as the reflex latency, which consists of afferent and efferent time conduction, and central delay (MacIntosh et al., 2006). The conduction time depends upon the speed of the action potential and the length of the fibre. The central delay depends on the number of synapses involved in processing and generating the efferent command (MacIntosh et al., 2006).

Spinal reflexes take approximately 10-20ms; however, for supraspinal reflexes it can take 50ms or longer (Jones et al., 2004). One of the simplest monosynaptic reflexes is the tendon reflex or stretch reflex. When the muscle becomes stretched the muscle spindles transmit impulses to the spinal cord, where the sensory fibre directly synapses with the large anterior horn $\alpha$ motoneuron, which sends efferent signals to the muscle fibre. The afferent neurons also synapse with interneurons that inhibit motoneurons of the antagonist muscles, causing them to relax. Impulses sent to the dorsal white columns of the spinal cord are relayed to the brain, which provides information on the velocity and length of muscle shortening. The muscle spindles are innervated by efferent $\gamma$ motorneurons, which regulates the response of the intrafusal muscle fibres. Stimulation of the intrafusal fibres maintains the spindles' tension and sensitivity during muscle contraction, and allows the brain to continually advise the state of the muscle. The motor supply to the muscle spindle also allows the CNS to regulate the stretch reflex response by stimulating or inhibiting the $\gamma$ motoneurons.

### 2.3.3.1 Post-synaptic and Pre-synaptic Inhibition

Inhibition of the nervous system is caused by post-synaptic and pre-synaptic inhibition. Post-synaptic inhibition occurs on the post-synaptic membrane, where the excitatory synapses lead to a depolarisation of the post-synaptic membrane, decreasing the absolute value of the excitatory post-synaptic potentials (EPSPs). The inhibitory post-synaptic potentials (IPSPs) take the membrane potential further away from its threshold and make the membrane less likely to generate an action potential to an excitatory response (MacIntosh et al., 2006). Inhibition of the antagonists can be activated by Renshaw cell-mediated inhibition. Renshaw cells are interneurons that directly synapse on α motoneurons and Ia inhibitory interneurons. The Renshaw cell acts to inhibit α motoneurons of the contracting muscle and its synergists. It also inhibits the antagonist muscle's Ia inhibitory interneuron (disinhibition) (Moritani, 2003). The output of α motoneurons excites cells that inhibit the same α motoneurons. This is known as negative feedback, which allows the CNS to minimise the effects of external perturbations (Latash & Zatsiorsky, 2001). Inhibitory interneurons receive impulses from Ia spindle afferents. These interneurons send their impulses to the α motoneurons that control antagonist muscle. The Ia interneurons make inhibitory synapses on the membrane of the α motoneurons that belong to the antagonist pool (Figure 7). The Ia interneurons are inhibited by the Renshaw cells that create the negative feedback that decreases the Ia interneuron inhibitory effect on antagonist α motoneurons. This is known as disinhibition, and increases the activity of the antagonist muscle to counteract joint motion (Latash & Zatsiorsky, 2001).

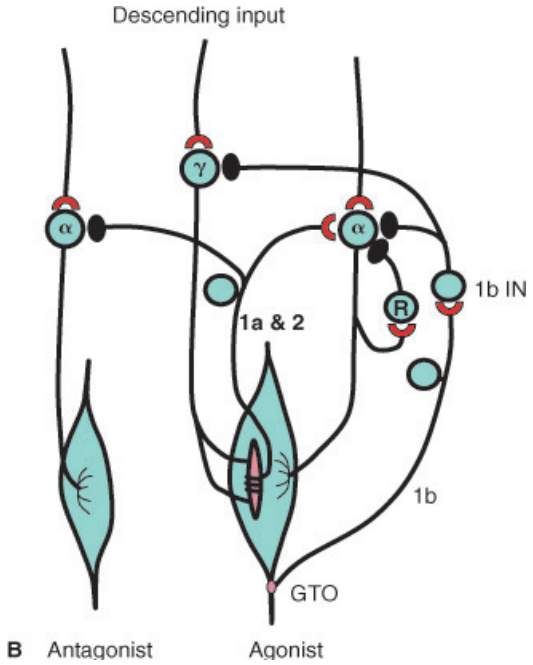



**Figure 7** Inhibitory network (Jones, Round, & de Hann 2004)
The Y-shaped endings indicate excitatory synapses; solid endings show inhibitatory synapses. GTO - Golgi Tendon Organ; R - Renshaw cell; 1b IN - Inhibitatory interneuron

## 2.4 Function of Skeletal Muscle

### 2.4.1 Muscle Model

Hill (1938) described this phenomenological model as having a contractile element (CE) that forms the basis of all active muscle behaviour. The CE embodies two main relationships, the length-tension and force-velocity interactions, which will be discussed shortly. Surrounding CE is a series elastic component (SEC) and parallel elastic component (PEC) (Figure 8). The SEC represents the tendinous structures and cross-bridge attachment where 50-85% of SEC exists in the tendinous structures, and the remainder reside intracellularly (Rack & Westbury 1984; Morgan, Proske, & Warren 1978). PEC is a passive component characterised by cytoskeletal structures and connective tissue sheaths. Vibration exercise has the ability to excite fascia, tendons, and active and passive cytoskeletal components, where the resting length of the CE and the rate and type of muscle action will influence the quantity of energy absorbed and the recoil imparted when stretched (Albasini, Krause, & Rembitzki 2010).

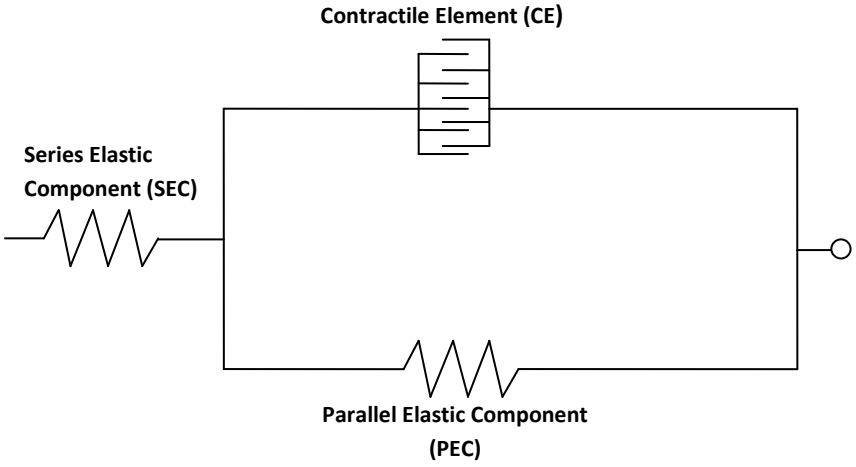



**Figure 8** Hill's muscle model of viscoelasticity

## 2.4.2 Muscle Fibre

### *2.4.2.1 Categories of Muscle Fibre*

Biochemical techniques, such as histochemical procedures have been used to classify fibre types by staining for the enzyme activity of myosin ATPase or myosin heavy chain (MHC) content. The staining procedure for ATPase activity has normally involved pre-incubating the tissue at a pH of 10.4. Muscle fibres are categorised into two types. Type I fibres show low ATPase activity following treatment. Type II fibres are those involved in high speed muscle shortening; they stain strongly and have high ATPase activity to degrade ATP rapidly (Engel, 1962). Further sub-division of muscle fibres has been reported by Brooke and Kaiser (1974). By pre-incubating the muscle tissue at various pH values (4.3 and 4.6), they successfully demonstrated a sub-division of type II fibres (A, B, C). These researchers identified type IIC as a precursor, with an ability to develop into type IIA or type IIB fibres. Recent evidence has suggested that IIC fibres contain a mixture of type I and type II myosin ATPase enzymes known as hybrid fibres (MacIntosh et al., 2006).

However, the method of ATPase histochemistry has limitations, as it may underestimate the true amount of different MHC isoforms in the muscle (Fry, Allemeier, & Staron, 1994). An alternative technique has emerged to overcome this problem. Known as immunohistochemistry, it was specifically developed to identify different molecular forms (isoforms) of myosin. As noted earlier the heavy chains are responsible for the cross-bridge that combine with the actin filament to produce muscle shortening (Bottinelli, Schiaffino, & Reggiani, 1991). The MHC controls the pH sensitivity of the ATP splitting reaction and is responsible for depth of histochemical staining (Brooke & Kaiser, 1974). It has been reported that the protein found in IIb fibres is closely linked to IIx, rather than to IIb and should, therefore be termed IIx (Smerdu, Karschmizrachi, Campione, Leinwand, & Schiaffino, 1994). Recently Stephenson, (2001) observed that large proportions of IIab (IIax) are found in human leg muscles, which contain both IIa and IIx MHC's.

### *2.4.2.2 Alteration of Muscle Fibre Types*

It is generally accepted that exercise training results in changes in neuromuscular properties, recruitment of different motor units, energy metabolism, and hormonal responses. This may lead to changes in MHC isoform composition, but it will depend on the status and type of training, muscle groups, energy metabolism, and the type muscle analysis performed. It is well known that that a contimuum of muscle fibre types exist and that transformation within a practicular muscle fibre subtype is a common adaptation to resistance training (Staron et al., 1994). It appears that when MHC IIB are stimulated they start a process of transformation toward MHC IIA profile by changing the quality of proteins and expressing different amount

of types and combinations of myosin asensodine triphosphatase (Fleck & Kraemer 2004). Using MHC technique, an eight week resistance training programme transformed MHC IIB into MHC IIA, which resulted in a 7% reduction in type IIX fibres (Staron et al., 1994). Longer studies of heavy resistance training have examined the changes in muscle fibre type and cross-sectional size with training (Staron et al., 1991). In this particular study skeletal muscle of women was examined over a 20 week period, detrained for 2 weeks followed by 6 weeks of retraining. The investigators reported that an increase in muscle fibre cross section from training with a decrease in MHC IIB (16% to 0.9%). Additionally, after a short period of detraining the muscle fibre started to return to pre-training values of muscle fibre cross-sectional areas, especially of MHC IIB, and a coversion of MHC IIA back to MHC IIB fibres. Further, it was reported that retraining period resulted in a faster change in muscle size and conversion to MHC IIA than when it started in an untrained condition. Therefore, the concept used by conditioning coaches of 'muscle memory' may have some legitimacy in the retraining of a person after a period of detraining. Hather, Tesch, Buchanan, Dudley (1991) reported that a significant increase in strength was accompanied by an increase percent in MHC IIA and a decrease in percent of MHC IIB after 19 weeks in all three groups that undertook different resistance training programmes. Additionally, they found that the increases in MHC I area occurred only in the group that performed concentric/eccentric muscle contractions, and MHC II area increased in groups that peformed concentric/eccentric or concentric-only muscle contractions, where the fibre subtypes paralleled an increase in MHC IIA and decrease in MHC IIB.

Recently, Liu, Schlumberger, Wirth, Schmidtbleicher, and Steinacker (2003) conducted a six week resistance training programme with maximum contractions for one group, while another group of similar age performed combination training of maximum contractions of ballistic and stretch-shortening movements. The group that performed resistance training with maximum contractions produced a shift in MHC isoform composition from IIX to IIA. In comparison, the combined resistance training group produced a MHC isoform composition, which shifted from slow to IIa. Transformation of muscle fibres is not confined to resistance training. Demirel, Powers, Naito, Hughes, and Coombes (1999) reported that after 10 weeks of endurance training the MHC isoform was capable of shifting from fast to slow. In this study, rats were trained at 75% $\dot{V}O_2$ max for 90 minutes. The investigators noted a decrease in percentage of MHC IIB and an increase percentage of MHC I.

### 2.4.3 Muscle Architecture

The prime function of skeletal muscle is for movement; however, the size and shape of our muscles reflect the task that is being performed. The orientation of the fibre has an important role for force production and range of motion, where skeletal muscles can be categorised as fusiform or pennate. Fusiform are muscles whose fibres run parallel to and are found in the extremities. Pennate muscles arrange their fibres at an angle to the muscle's longitudinal axis and produce larger forces, but over a shorter range than fusiform muscles. In general, a fusiform will facilitate muscle shortening and a pennate fibre arrangement will promote force production because they can contain more fibres per unit of muscle volume (Hall, 2006). Muscles such as hamstrings and ankle dorsiflexors have their fibres arranged in parallel, which is advantageous for velocity of shortening. In contrast, muscles such as the quadriceps and gastrocnemius provide an advantage for force production due to the fibres being aligned at an angle to the muscle's longitudinal axis (Brooks et al., 2005).

When sarcomeres are arranged in series, the velocities become additive and the overall velocity of shortening at the tendon is increased. This maintains the length of the individual sarcomeres while increasing the overall velocity of shortening. Alternatively, a greater force production is evident when more sarcomeres are arranged in parallel, as more sarcomeres can be packed in between the origin and insertion of the muscle this way (Wickiewicz, Roy, Powell, & Edgerton, 1983). Exercise, especially resistance training is capable of changing the muscle architecture; namely the pennation angle, and fascicle length. Kawakami, Abe, Kuno, and Fukunaga (1995) reported that 16 weeks of unilateral resistance training in the elbow triceps brachii produced a greater pennation angle compared to untrained muscle. Likewise, Seynnes *et al*. (2007) reported that when untrained males and females performed 35 days of isoinertial knee extension a significant increase of 10% and 8% in fascicle length and pennation angle was evident in the vastus lateralis muscle. The researchers postulated that these increases may be due to the addition of sarcomeres both in series and in parallel. However, other researchers (Abe, Kumagai, & Brechue, 2000) have reported that in the absence of resistance training, sprint and plyometric training were associated with increases in fascicle length and reductions in pennation angle. Furthermore, these same investigators have reported that male elite sprinters have a smaller pennation angle of the lower limb muscles compared to elite distance runners. They suggest that faster sprinting speed coincides with the determinants of maximum velocity of shortening. However, it remains equivocal whether or not muscle architecture changes can occur in the initial stages of resistance training (Blazevich, Gill, Deans, & Zhou, 2007; Seynnes et al., 2007).

### 2.4.4 Length-Tension Relationship

Early investigations of the muscle length-tension relationship were difficult to perform, because the sarcomere pattern was not precisely uniform within a muscle fibre – it varied from one region to another along the fibre (Edman, 2003). However, a technique was developed to record isometric force from an intact fibre. Gordon and associates (1966) excluded the end regions of the fibre by length-clamping the middle portion of the muscle fibre during tetanus. They discovered that when the sarcomeres were stretched to greater lengths, force decreased until a sarcomere length of 3.65µm where the muscle developed no active force (Figure 9). At this length the investigators reported no overlap between the actin (2.0µm) and myosin filament (1.65µm) suggesting no active cross-bridge sites were evident in generating any force. Additionally, these researchers reported that the peak of the length-tension curve showed a plateau region with constant tension over the range of 2.0-2.2µm sarcomere length. However, Edman and Reggani (1987) found no distinct plateau between 2.0-2.2µm in a length-tension curve where its shape was sigmoidal compared to polygonal shape as previously described by Gordon *et al*. (1966). The discrepancy between the studies is probably due to Gordon *et al*. (1966) length-clamping fibre segment at 7-10mm, which could have created a higher degree of sarcomere instability and tension compared to the creep-free tension used by Edman and Reggani (1987).

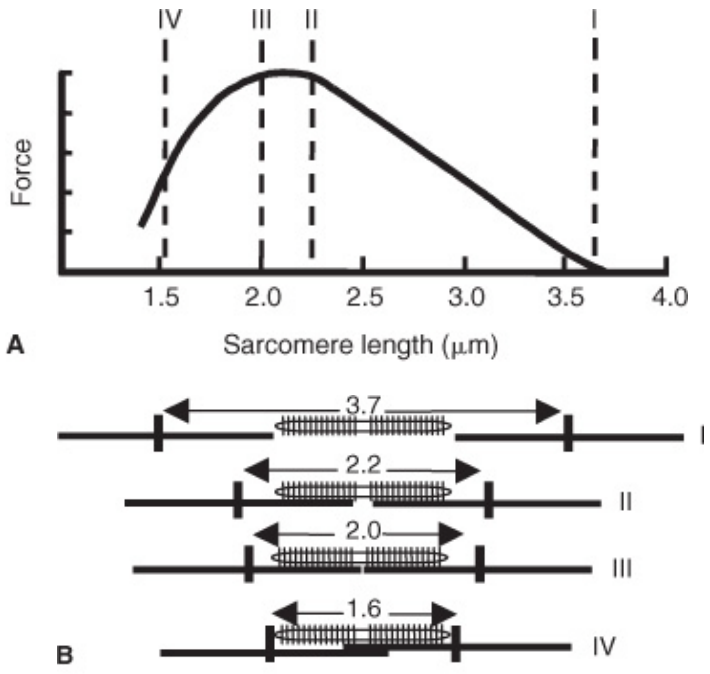



**Figure 9** Length-tension relationship and the arrangement of filaments at different lengths (Jones, Round, & de Hann 2004)

### 2.4.5 Force-Velocity Relationship

Using an isolated whole frog muscle, (Hill, 1938) demonstrated the existence of the force-velocity relationship as a hyperbolic curve; the maximum speed of shortening occurred when the load was zero, and the maximum force occurred when the muscle was stationary. Since then, contemporary single muscle fibre experiments have found that the force-velocity relationship is more complex than first described by Hill (1938). Joyce and Rack (1969) reported that the velocity of movement was modified by changes in muscle performance that occur at different lengths, and by the history of the movement. Research conducted by Edman (1988) found two distinctive curves either side of a 'breakpoint' at approximate to 75% of isometric force. Edman (1988) stated that there was a flat region of the force-velocity relationship that occurred around maximum force. This has a functional significance for promoting stability to keeping the sarcomere pattern uniform when the muscle works at high loads. Additionally, it prevents muscle from being inappropriately stretched when a load is suddenly increased above its isometric level.

The force-velocity relationship can, in part, be explained, by the cross-bridge mechanism. At higher shortening velocities the cross-bridge cycling rate increases, which results in less cross-bridges being attached, thus producing less force (Huxley & Simmons 1971). At maximal shortening velocity little force is being exerted, as most of the cross-bridges are cycling rather than staying attached. Therefore, on average the cross-bridges are stretched less, generating less force (Figure 10). Edman (1988) reported that when maximal shortening velocity occurred, a high correlation between myofibrillar ATPase activity and maximum cross-bridge cycling rate was present. The faster the sarcomeres were shortening and cross-bridges were cycling, the less force they produced. However, when the load is greater than the contractile force the muscle lengthens, it absorbs rather than generates mechanical energy (Kandel, Schwartz, & Jesse, 2000).

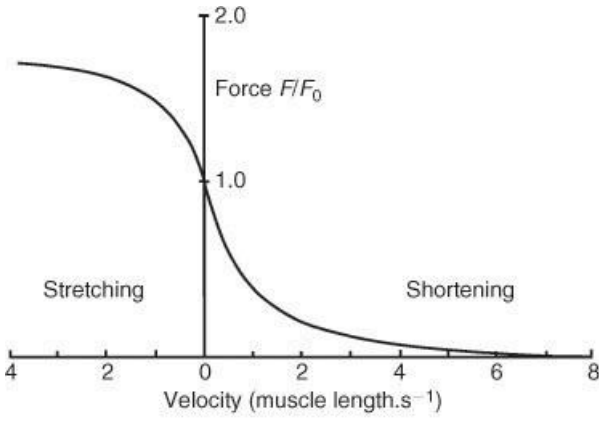



**Figure 10** Force-velocity relationship (Jones, Round, & de Hann 2004)

During lengthening at slow velocity, the cross-bridges go through the normal cycle of attachment. However, at faster velocities of lengthening fewer cross-bridges are unable to complete the cycle and the majority of myosin heads remain in contact with actin, which causes the cross-bridge to stretch and requires considerable force detachment (Figure 11) (Jones et al., 2004). Therefore, a high level of sustained force that occurs in muscle lengthening is most likely due to the myosin heads rapidly re-attaching to actin. Finally, at the same velocity a greater force is elicited in an eccentric compared to a concentric muscle action. The eccentric action supposedly provides less movement of cross-bridges (tearing apart instead of cycling), therefore, more cross-bridges are attached at any time, which generates greater torques (Jones et al., 2004) and is likely to be under greater neural control (Duchateau & Enoka, 2008).

During rapid shortening little force is generated, as the rate of energy consumption is very high because the cross-bridge dephosphorylates one ATP molecule in the process of detaching at the end of the power stroke (Geeves & Holmes, 1999). The rate of energy consumption by the cross-bridge is proportional to their velocity not to the force they produce. The classical work of Abbott, Bigland, and Ritchie (1952) noted that the energy cost was higher when performing concentric work compared to the eccentric work during stationary cycling. Therefore, in muscle lengthening the rate of energy consumption is much lower because the cross-bridges are pulled apart without ATP hydrolysis (Flitney & Hirst, 1978).

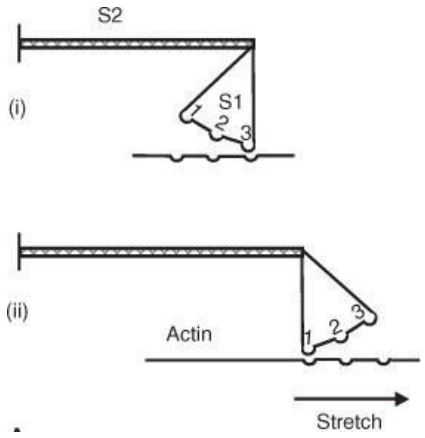



**Figure 11** Behaviour of cross-bridges during stretch (Jones, Round, & de Hann 2004)
Movement of actin from left to right produces greater stretch of the S2 compponent than in the isometric condition. For slow stretches (i) some myosin heads can reach position 3 and complete the cycle, but for fast stretches (ii) most will remain in position 1.

### 2.4.6 Post-Activation Potentiation

Depending on the extent of prior work, the muscles' performance can either be impaired through fatigue or enhanced through a phenomenon known as post-activation potentiation (PAP) (Sale, 2002). PAP is an increase in muscle twitch and tetanic tension following contractile activity such as a series of evoked twitches, sustained maximal voluntary contraction (MVC) (Sale 2002), or performing a heavy resistance exercise prior to an explosive movement (Hodgson, Docherty, & Robbins, 2005). It has been shown to increase isometric twitch and the rate of force development, and decrease time to peak twitch force (O'Leary, Hope, & Sale, 1997).

Additionally, it has been purported that PAP has the capacity to move the force-velocity curve to the right (Sale 2002), and to increase power and strength performance (Hodgson et al., 2005). Applied studies have either showed that performing a heavy pre-loaded activity such as heavy resistance training (HRT) potentiates muscular performance (Gourgoulis, Aggeloussis, Kasimatis, Mavromatis, & Garas, 2003; Young, Jenner, & Griffiths, 1998) or causes no change (Gossen & Sale, 2000; Scott & Docherty, 2004; Smith & Fry, 2007). The dissonance between the studies is most likely due to methodological issues such as the rest interval prior to the performance, training status of the participants, relative strength of the participants, and the type and duration of the HRT (Hodgson et al., 2005; Sale 2002; Robbins 2005).

The mechanism for explaining PAP has been demonstrated using electrically-induced muscle twitch contraction(s) and is considered to involve phosphorylation of myosin regulatory light chains, making actin and myosin more sensitive to the intracellular $Ca^{2+}$ signal (Moore & Stull, 1984; Sweeney, Bowman, & Stull, 1993; Zhi et al., 2005). The result is a greater rate of cross-bridge attachment for the same $Ca^{2+}$ concentration, which in turn increases twitch tension (Metzger, Greaser, & Moss, 1989). Twitch potentiation is larger in type II muscle fibres due to a pronounced increase in $Ca^{2+}$ sensitivity of the myofilaments, which leads to greater phosphorylation of myosin light chains (Moore & Stull, 1984).

### 2.4.7 Muscle Temperature

As demonstrated by Asmussen and Boje (1945), raising body temperature prior to exercise has the potential to enhance short-term performance. Active and passive modalities have frequently been used to elevate muscle and/or core temperature. Passive modalities normally involve using an approach such as hot water baths and saunas where externally applied heat is absorbed by the body's tissues (Bishop, 2003a). In comparison, active modalities involving dynamic and rhythmic muscular work such as running, cycling, and skipping, raise metabolic heat production, increasing body temperature (Bishop, 2003a).

Exercising muscles generate heat; the amount of which is dependent on the work rate/intensity of the activity and muscle temperature at any given time (Kenny et al., 2003). Early work from Saltin, Gagge, and Stolwijk (1968) found that when a thermocouple was placed in the quadriceps muscle at varying depths (2-5 cm), it produced a range of resting values (33.0-36.5°C). Saltin *et al*. (1968) reported that on the commencement of steady-state ergometry cycling, muscle temperatures at all depths rapidly increased more than rectal temperature and plateaued within 10-20 minutes. They observed that the location of the thermocouple and posture of the participant were crucial to taking valid and reliable muscle temperature readings. Changing the cycling position from seated to standing caused a variation of 0.5°C, indicating that certain areas of the muscle may be more actively involved in muscular contraction. This may explain why a large variation in blood flow is seen in different parts of the muscle during steady-state exercise.

It is widely known that increasing tissue temperature can enhance muscular performance (Asmussen & Boje, 1945; Bennett, 1984; Davies & Young, 1983; Dolan, Greig, & Sargeant, 1985b; O'Brien, Payne, Gastin, & Burge, 1997). Asmussen and Boje (1945) reported that stationary cycling at 98W for five minutes raised muscle temperature by 1.4°C, which improved short term cycling performance by 4.2%. Sargeant (1987) observed that passively warming legs in a hot tub (44°C) raised muscle temperature by 2.7°C and increased isokinetic cycle peak power by 11%, which equated to 4% per °C rise in muscle temperature compared to resting conditions. Sargeant (1987) conducted additional experiments to investigate four different muscle temperatures (at rest, water bath 12°C, 18°C, 44°C) with three cycling velocities of 54, 95, 140 rpm. He found that the muscle temperature effect was velocity dependent, where peak power increased by 2% per °C for the slowest pedalling velocity, while the peak power increased by 10% per °C for the fastest pedalling velocity. This finding suggests that the force-velocity relationship may be greater at faster contraction velocities, which are influenced by temperature-related changes. Davies and Young (1983) found that by immersing the triceps surae in hot (46°C) or cold water (0°C) muscle temperature rose 3.1°C or decreased by 8.4°C. Muscle contractile properties were assessed using muscle stimulation and MVC. The rise in muscle temperature produced a decrease time to peak tension and half

relaxation time, with no effect on twitch and tetanic tension and MVC. Conversely, the cooling decreased all the contractile parameters, indicating that mechanical and contractile properties of short-term performance are muscle temperature dependent. However, the authors did not document the fact that the heating increment was not identical between the passive heating and cooling condition (3.1°C vs. 8.4°C).

In conclusion, muscle temperature is directly proportional to the exercise intensity due to the heat generated by the working muscle (Saltin et al., 1968). Both passive and dynamic modalities increase the rate of muscle temperature rise, however the exact mechanism remains equivocal (Barcroft & King, 1909; Bishop, 2003b). Comparing the various muscle temperature studies has been difficult; most of the studies used varying depths when inserting the muscle thermocouples, and were statistically underpowered due to the invasive nature of the method.

## 2.5 Vibration

*Introduction*

This review has explored the structure and microstructure of skeletal muscle, the contraction and the recruitment process, as well as the function of skeletal muscle, which is central to the background of vibration exercise. The focus of the review will now shift to the operationalisation of vibration. Occupational and exercise vibration will be differentiated, and the history, definition, types, and parameters of vibration exercise discussed. Currently, the proposed mechanisms of vibration exercise are based on neurogenic potentiation. The first mechanism has centred on spinal reflex known as the tonic vibration reflex that causes excitatory responses of the muscle spindle, which augments muscle activity. The second is based on a muscle tuning response, and the final mechanism is founded on neural responses synonymous with resistance and power training. All of these will be explored and critiqued. The subsequent sections of the review will examine the effects of vibration exercise on muscle activity, cardiovascular, metabolism, and muscle performance.

### 2.5.1 Occupational Vibration

Vibrations have, for centuries, been part of our daily lives. In the 17th century, back pain was prevalent among drivers of horse carriages, and to alleviate kidney stones patients were prescribed a drive over an uneven road (Mester, Spitzenpfeil, Schwarzer, & Seifriz, 1999). Today, automated machinery dominates our lives – most people come into daily contact with some sort of vibration, whether it is by car, rail or bus. Sport activities such as downhill skiing, mountain biking, horse riding, and inline skating are sources of vibration. Individuals can be exposed to several types of vibration, such as periodic (sinusoidal and multi-sinusoidal); non-periodic (transient, shock); stationary (random); and non-stationary (random) vibration (Mester et al., 1999). However, most machinery and devices produce measurable oscillatory motion that is periodic and sinusoidal (Mester et al., 1999).

Those who operate industrial machinery on a full-time basis are at risk of vibration exposure and the ailments that may follow. Long-term exposure to hand-held machines such as motor chain saws, jackhammers, and drills, may result in a vibration side-effect known as ‘white finger’ (Voelter-Mahlknecht et al., 2008). Trucks, tractors, and heavy-equipment are capable of producing whole-body vibration, which increases the risk of lower back disorders by a factor of 1.2–39.5 (Bernard, 1997). The adverse health effect from prolonged vibration is an issue because of its impact on workers’ health and the potential cost to the health system. Zimmermann, Cook, and Rosecrance (1997) found engineers who were continually exposed to vibration reported increases in work-related musculoskeletal symptoms of the lower back

(60%), neck (44%), shoulders (37%), and knees (32%). Additionally, it was observed that operators using older equipment reported a higher percentage of missed work due to musculoskeletal symptoms than those using newer equipment. In 2002, Parliament and the Commission of the European Community set guidelines for International Standards for hand-transmitted vibration and industrial whole-body vibration exposure. They recommended that the daily exposure limit be standardised to an eight hour day of 5m/s$^2$ rms for hand-transmitted vibration and 1.15 m/s$^2$ rms for whole-body vibration (Griffin, 2004). These guidelines advocate that adequate rest periods need to be scheduled throughout the day, along with limiting the duration and intensity of the exposure by making sure machinery is correctly maintained. However, such vibration safety guidelines do not exist in sport, exercise and physical rehabilitation.

#### *2.5.1.1 Resonance Frequency*

During vibration exercise energy is transferred from the vibrating device to the human body, where body parts are accelerated, which causes reactive forces within the body. These forces have the potential to be harmful, but also beneficial. The human body is a spring-mass system where tendons and muscles act like springs to store and release mechanical energy. The stiffness and mass of these body parts will determine the natural frequency (Rittweger, 2010).

The body can accumulate mechanical energy when the frequency of the vibrating device parallels the frequency of the body (Rittweger, 2010). Resonance frequency for a given part of the body is defined by the frequency at which the transmissibility from the vibration source to that part of the body reaches maximum (Yue & Mester, 2004). Herterich and Schnauber (1992) reported that for whole-body the resonance frequency is approximately 5 Hz, but other structures within the body have different resonance frequencies such as, eyeball (~20 Hz), head (~18 Hz), spine (~8 Hz), lung (~8 Hz), and muscle (~7-15 Hz).

Resonance frequency should be avoid otherwise it can lead to resonance catastrophe, which will cause a detrimental effect on the body. But resonance frequency can be controlled by adjusting body position and changing muscle stiffness (Mester, Kleinoder, & Yue, 2006). According to Wakeling *et al*. (2002) muscles have damping characteristics that can absorb energy and generate heat.

Controlling the external vibration source may have a beneficial effect on muscle activity that could be used as a physical training modality (Wakeling & Nigg, 2001). There is an abundance of commercialised vibration machines that have been targeted towards sport, exercise, and health sectors. These machines differ to the occupational vibration, where low frequency vibration ($f$=5-45Hz) exposure occurs over a short duration either intermittently or continuously. Therefore, vibration exercise should not be confused with occupational vibration,

where workers are continually exposed to very high frequencies from machinery such as chain saws, jackhammers, and heavy vehicles. Although occupational vibration may produce undesirable side-effects and musculoskeletal ailments, there is growing evidence from both anecdotal and research sources that vibration exercise may enhance muscular performance.

## 2.6 Vibration Exercise

### 2.6.1 History of Vibration Exercise

Vibration has been classified as an occupational hazard that causes pain and discomfort when used for prolonged periods. However, there have been several attempts to utilise vibration as a therapeutic modality, dating back to ancient Greece, when vibration was used to treat injuries. In the sixteenth century, the Japanese used percussion vibration and pressure, as well as passive motion, to relax rigid and spasmodic muscle contractions. In more modern times, Dr John Kellogg, best known for his development of cornflakes, but also a skilled surgeon, developed a vibrating chair (Figure 12) that was capable of producing vibrations at 60 cycles per second to the lower and upper parts of the spinal column (Kellogg, 1895). Kellogg observed that 3-10 minutes of exposure was an optimal time to induce the desired physiological effects. Unfortunately, his findings were never formally documented.

**Figure 12** Kellogg's vibrating chair (Lifestyle Laboratory, 2010)

In addition to the vibrating chair, Kellogg devised a vibrating platform and bar. From these devices he observed that with each oscillation, muscular contractions were prevalent. He also noted that when the vibration exposure continued beyond a given time, the muscular contractions become less distinct, and merged, becoming continuous, or tetanic. Kellogg administered mechanical vibrations to his patients to cure constipation, headaches and lower back pain, but Kellogg never performed any controlled studies to test his observations.

The therapeutic benefits of vibration continued to interest people. A custom made vibrating bed (0.01 Hz) was used to treat cardiovascular disease (Sanders, 1936). Using the same bed, Whedon *et al*. (1949) reported that it could prevent bone loss by 50% in young, healthy bedridden participants. Experts at the time questioned the methodology and results, and research was halted. It wasn't until 1960 that vibration exercise reappeared in the form of rhythmic neuromuscular stimulation (RNS) when Professor. W. Biermann, from the former East German Republic, described the so-called "cyclical vibrations" as capable of improving joint conditions. This type of research then found its way into the former USSR space programme to counter the effects of spaceflight for cosmonauts to prevent strength and bone loss.

In the 1970s, the Eastern Bloc countries recognised the potential use of vibration for sport, and developed a vibration system to train Olympic athletes. Dr. Vladimir Nazarov, a Russian sports scientist at the time, was one of the first to develop the vibration technology, which he coined Biomechanical Stimulation (BMS). In his first publication he reported that vibration was able to prevent osteoporosis and strengthen bones by 34%. He subsequently introduced this to ballerinas, and found that jump height, strength and flexibility increased, with reductions of injury rates. As a result of these findings vibration was introduced to a number of sports in the former USSR. However, due to the fall of communism, the vibration technology for training sportspeople was made public and made its way across Europe.

In the early 1990s other European athletes began using the modality to enhance sport performance. A Dutch Olympic coach, Guus van der Meer, saw the potential of this training not only for enhancing human performance but for medical and therapeutic benefits. The difficulty that Van der Meer found was that the majority of the Russian research was either published in abstract form or was unavailable. However, through trial and error, Van der Meer found that low frequency with high amplitude led to discomfort and undesirable organ resonance. He continued to make modifications by improving the individual functions and parts of the vibration technology, which allowed him to build a vibrating platform that he considered to provide the greatest benefit. Since then, much anecdotal evidence has surfaced from coaches and athletes suggesting that athletic performance is enhanced by vibration exercise. However, it has been difficult to prove, given that carefully designed and well controlled research studies were scarce, and the training prescription of the vibration programmes were never documented.

A lot of the testimonials from vibration training were performed in-training and tended to be case studies in uncontrolled conditions, therefore it was difficult to determine if the benefits of vibration were authentic. In the late 1980s, the first study was published on the beneficial effects of vibration exercise on strength abilities in athletes (Nazarov & Spivak, 1985); however, it lacked the necessary research design to differentiate the placebo and experimental effects of vibration training.

Since the turn of the 21st century, there has been a resurgence of vibration technology. This is not surprising, given that many athletes are looking for gains in performance to provide maximum benefits with minimal effort. The application of vibration has found its way into medical, clinical and rehabilitation fields; for example, space agencies, such as the European Space Agency and NASA, have developed vibration training as a way to negate the effects of the gravity-free environment on bone and muscle tissue loss. This technology has been sent in spacecrafts where astronauts have exercised on low frequency and low magnitude vibration devices to provide enough gravity resistance to maintain and, in some cases, increase muscle strength, mass, and bone density during space flight. However, the embargo on this research has led to little dissemination of these findings.

In summary, there has been a renewed interest in vibration in sport, exercise, and health; however, given its potential benefit, research to gain an understanding of vibration exercise has often lagged behind its application. To date, there have been varying anecdotal reports of the effects of vibration exercise and documentation of well controlled studies investigating its mechanisms and benefits has been sparse.

### 2.6.2 Definition of Vibration Exercise

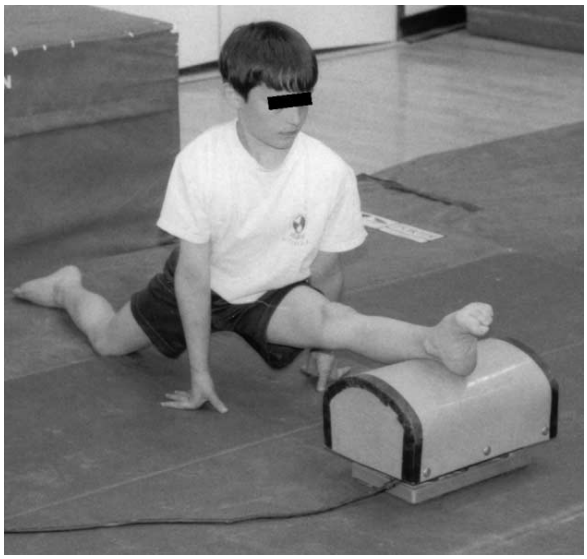
Vibration exercise is also known as biomechanical stimulation, biomechanical oscillation, vibration training, vibration therapy, and whole-body vibration. Initially, vibratory devices (actuator) were directly placed on specific parts of the body. Later, oscillating platforms were designed to stand on so that the whole body was stimulated with sinusoidal vibration and became a popular modality where the term whole-body vibration and vibration training were coined. However, vibration exercise provides a better description of the modality because it differentiates between that of occupational whole-body vibration, which can be a health hazard when individuals are exposed to machinery over a long time period. Further, dynamic and/or static exercises are often performed on vibrating platforms or with other vibrating devices (vibrating dumbbells). Therefore, this thesis will use the term vibration exercise, which is defined as a mechanical stimulus that delivers vertical sinusoidal oscillations to the body while simultaneously performing dynamic and/or static exercises. Future research may want to adopt this as universal standard term, which is necessary if this method is to be accepted as a

legitimate exercise modality. With anticipation, this term will need to be approved by an international task force for vibration classification and standardisation, which ultimately will lead to less confusion and greater consistency within the literature.

### 2.6.3 Types of Vibration Exercise

Vibration exercise has taken on many different forms. Kellogg (1895) built a vibrating chair and an oscillating platform; others have used small vibratory units placed directly on the muscle or tendon (Jackson & Turner, 2003; Warman, Humphries, & Purton, 2002) and larger custom built units have been constructed for flexibility training (Kinser et al., 2008; Sands, McNeal, Stone, & Haff, 2008a; Sands, McNeal, Stone, Russell, & Jemni, 2006; Figure 10). Vibrating units have also been attached to resistance training equipment to elicit vibration transmission through the cables of various machines (Issurin & Tenenbaum, 1999; Figure 13) and a cycle ergometer has also been mounted on a custom built vibrating table (Samuelson, Jorfeldt, & Ahlborg, 1989). Vibration has long been acknowledged as a potential modality in the sport, exercise and health sectors, and there has been a rapid increase in commercially made vibrating machines.

Currently, there are two commercial forms of vibration platforms manufactured for the health and fitness industry. The first type of platform (e.g. Galileo®) has a teeterboard that produces side-alternating vertical sinusoidal vibration (SV) to the body. It rotates about an anteroposterior horizontal axis, so when the feet are further from the axis it results in larger vibration amplitude (Figure 14). The side-alternating movement is asynchronous where the unilateral vibration is applied alternately to the left and right foot. The other commercial machines (Power Plate®, Nemes®, Vibra Pro®,Vibrafit®, Fit Vibe®, Pneu-vibe®, Vibrogym®, Soloflex®, Bodypulse®, Juvent 1000®) produce vertical synchronous vibration (VV) where both legs are vibrated as the plate moves predominately in the vertical direction. This results in simultaneous and symmetrical movement of both sides of the body during the exposure (Figure 10). Hand-held powered vibrating dumbbells have also been commercially manufactured for exercising the upper-body (Galileo TOP®, Mini-VibraFlex®) where the central handle piece of the dumbbell rotates and produces oscillatory movements to the body of varying frequencies (0-30 Hz) (Figure 15).



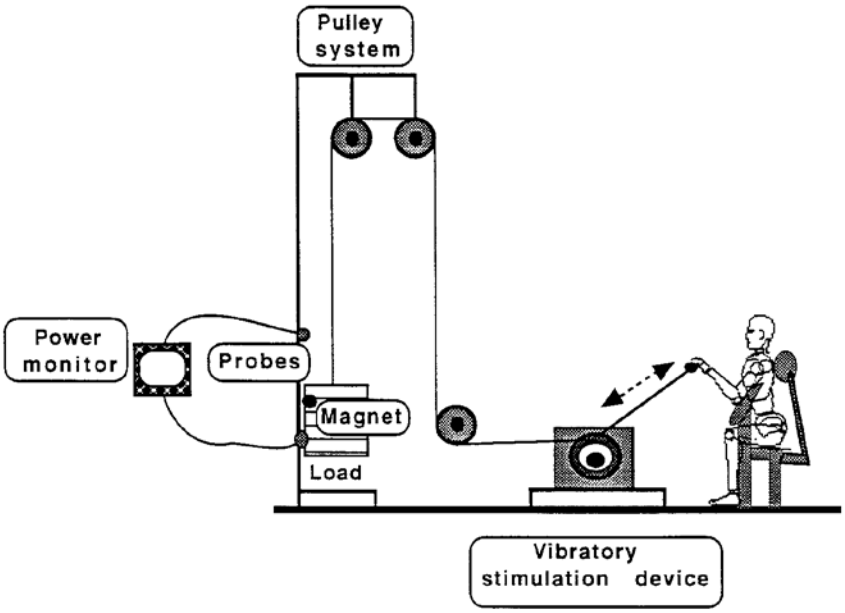



**Figure 13** Above: custom built vibration unit used for flexibility training (Sands et al., 2006), and below: vibratory unit secured to resistance training equipment (Issurin et al., 1994)

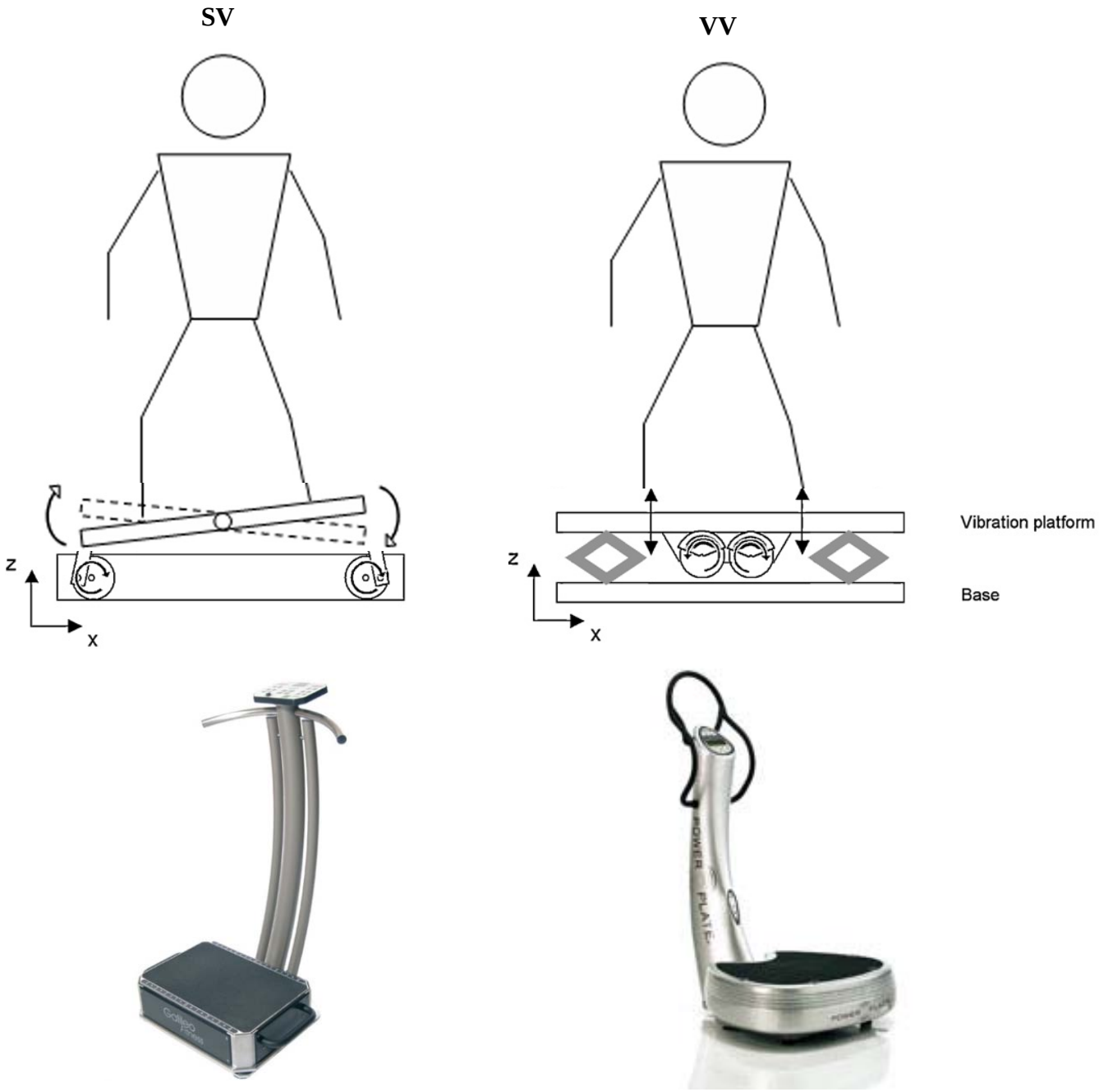



**Figure 14** The two different types of commercially manufactured vibrating platforms: SV and VV (Adapted from Pel et al., 2009). The SV oscillates around a central axis, where a crankshaft on each side of the platform translates to a rotating motion of the electro-motor into a vertical displacement, inducing a seesaw motion, of which the amplitude is either small, closer to axis or larger near the edge of the platform.

For VV two electro-motors with an eccentric mass induce vertical vibrations where the amplitude has two predetermined settings: low or high. Indicted are the vertical (*Z*), and horizontal (*X*) directions


**Figure 15** Galileo TOP® device, which produces vibration to shoulders and arms from an electric-powered dumbbell (DB), weighing 2.5kg. The central handle piece rotates and produces oscillatory movements to the body of varying frequencies (0 to 30 Hz) with amplitude of 3mm around a horizontal axis.

The debate over which platform is superior is currently equivocal. Research performed by Abercromby *et al*. (2007a) reported that the lower limb extensors (vastus lateralis and gastrocnemius) were activated significantly more during SV than VV; however, the activation of the tibialis anterior was significantly greater during VV than SV. Furthermore, during dynamic (from 10° to 35°of knee flexion, at a tempo of 4s up 4s down) and static squatting (18.5° knee flexion), SV produced a greater activation of the lower limb muscles compared to VV. In a later study, the same authors (Abercromby et al., 2007b) reported that across different knee angles (5-35°) vibration transmitted to the upper-body and head was 71 to 189% greater during VV than SV. The authors concluded that during SV the pelvis damps the vibration energy more than the VV. Earlier, Rittweger, Schiessl and Felsenberg (2001) had proposed a similar hypothesis: in SV the feet are alternated between up and down positions, causing rotation of pelvis and flexion of the spinal column, which decreases the vibration transmission to the head. However, no kinematic analyses have been performed on SV and VV to validate this claim. It should be noted that in Abercomby's *et al*. (2007a) study the dynamic squat was only performed through a limited range of knee flexion (10-35°). Therefore, it would be of interest to conduct additional experiments over a larger range of knee flexion to identify whether further differences exist between VV and SV. From a practical perspective, only a certain number of conditioning exercises can be performed on the SV compared to the VV platform. The alternating nature of the SV machine makes it difficult to perform abdominal crunches, prone and lateral bridge holds, tricep dips, various massage exercises, and some types of stretches that involve lying or sitting on the vibrating plate.

### 2.6.4 Transmission of Vibration Exercise

There are many types of industrial vibration, including sinusoidal, multi-sinusoidal, transient, shock, stationary random or non-stationary random vibration (Figure 16) (Mester, Spitzenpfeil, & Yue 2003). People participating in sport or physical activity are also exposed to different vibration waveforms from activities, such as inline skating, mountain biking, downhill skiing, walking, running, or the impact of ball and bat. The majority of contemporary vibration machines produce periodic sinusoidal oscillations, where energy is transferred from the vibratory machine to the human body. The vibratory load is dependent on four parameters: frequency, amplitude, acceleration, and duration. The number of cycles of oscillation per second determines the frequency; the amplitude refers to the displacement of the oscillatory motion (Figure 17); the acceleration ($m/s^2$ or $g$) determines the magnitude; and duration refers to the exposure time. Normally, vibration exercise is administered in the range of 0-45 Hz, amplitude of 0-12mm (peak-to-peak amplitude) and acceleration of 0-18$g$.

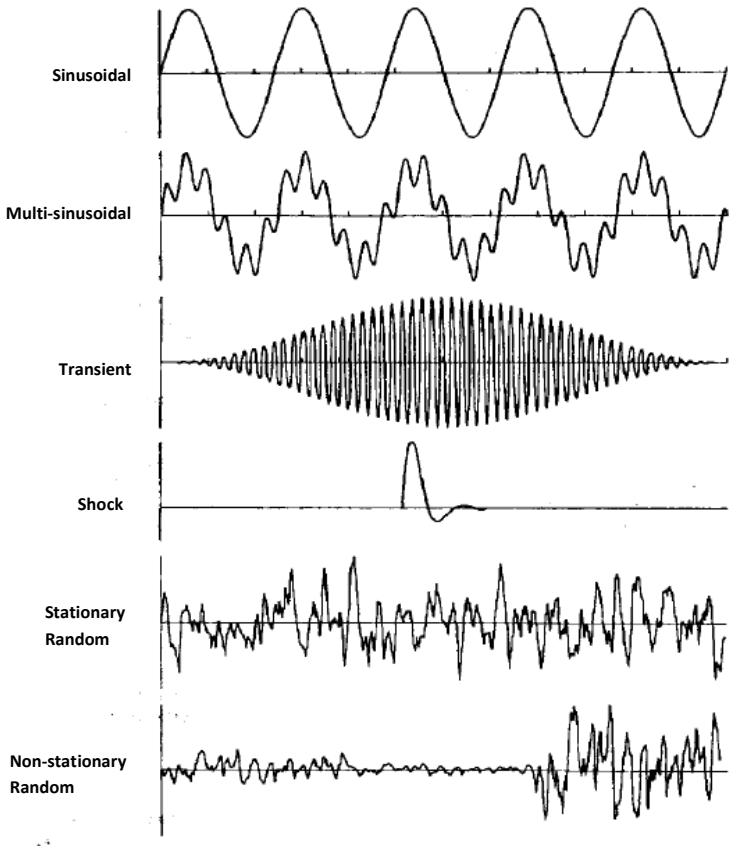



**Figure 16** Different types of vibration waveforms (Griffin, 1994)

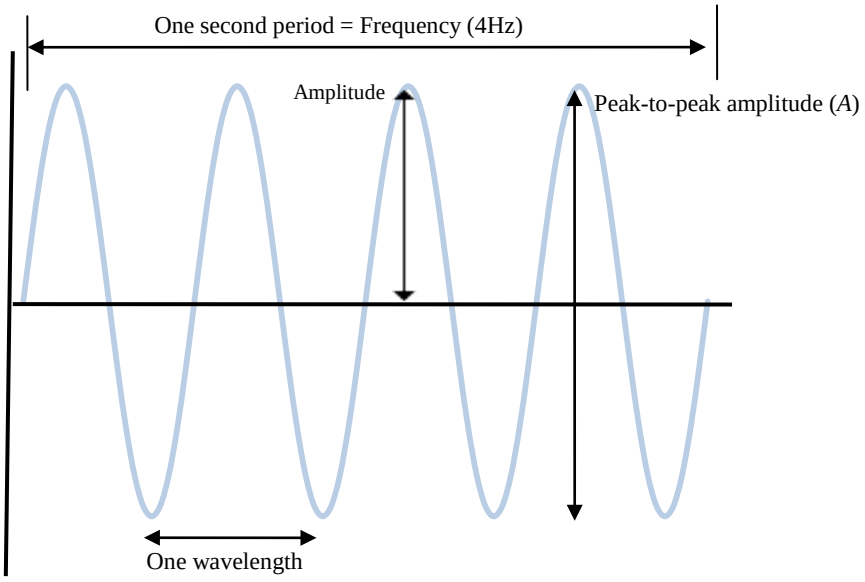



**Figure 17** Parameters of sinusoidal oscillation

The motion of vibration training is sinusoidal. The acceleration transmitted to the body is based on the principle of peak angular acceleration (*a*) being the product of angular velocity ($\omega^2$) and peak-to-peak amplitude (*A*)

$$a_{peak} = \omega^2 A$$

where angular velocity ($\omega^2$) is the product of vibration frequency (*f*) and 2 π squared

$$\omega^2 = (2 \pi f)^2$$

Changes in vibration frequency or amplitude will determine the changes in acceleration transmitted to the body. The greatest acceleration (or gravitational load) is high when both frequency and peak-to-peak amplitude are at their maximum. For example, a vibration frequency of 26 Hz, at a peak-to-peak amplitude of 6mm, will produce a peak angular acceleration of 160 $ms^{-2}$ or 16.3*g*. The vibration frequency and amplitude can be manipulated to determine the desired peak acceleration; however, only a few studies have investigated the dose relationship on neuromuscular and performance aspects.

Damping and stiffness adjustments are two other factors that need to be considered when vibration is transmitted from the vibratory device to the human body. Wakeling, Nigg, and Rozitis (2002) have reported that vibration is likely to be damped by tissues and fluids, such as synovial fluid, where the mechanical energy is absorbed by structures, leading to heat generation. The damping response of these structures has been termed 'muscle tuning' (see further description of this in the proposed mechanism for vibration exercise).

Anecdotal reports claim that standing erect, as opposed to squatting, evokes a stronger transmission of the vibration to the head, and shifting body weight to the forefront of the foot will reduce the vibration transmission. Lafortune, Lake, and Hennig (1996) confirmed this, reporting that when the knees were extended the vibration frequency above 10 Hz was more effective in transmitting to the hip compared to a frequency of less than 5 Hz. Rubin (2003) found that at low vibration frequency the vibration transmission to the hip was dissipated; however, when the frequency was raised to 15-35 Hz, a higher response at the hip was evident. A scientific model (Yue & Mester, 2002) has been used to describe the transmission of vibration; however, this model has lacked the inclusion of the three lower limb joints (ankle, knee and hip) and the researchers have not considered the effect of the range of joint motion on vibration transmission. Abercromby *et al*. (2007a) stated that the damping of mechanical energy by the legs depends not only on the compliance of ankle, knee, and hip joints, but also on the modulation of leg muscle activation. These researchers found that the greatest mechanical impedance (as determined by a decrease in joint compliance and an increase in the absorption of vibration energy) occurred at a knee angle of 10-15°. They also found that acceleration of the head decreased as knee angle increased from 10 to 30°, and was greater in

VV than SV (*f*=30 Hz, *A*=4mm). The authors concluded that squatting at a knee angle of 26–30° dissipated head vibration and that the use of small knee flexion angles during vibration exercise increased the likelihood of negative side effects as the greatest mechanical energy is likely to be transmitted to the upper-body and head and should, therefore, be avoided.

Additionally, Abercromby *et al*. (2007b) reported that undertaking a vibration protocol of 10 minutes per day (*f*=30 Hz, *A*=4mm) exceeded the recommended daily whole-body vibration exposure as defined by the International Organisation for Standardisation (ISO 2631-1). However, the health risk of vibration exercise cannot be accurately calculated using ISO health standards, because of the intermittent nature of vibration exercise. More research is required to develop a new method of assessing the potential negative side effects for intermittent vibration exercise.

## 2.6.5 Parameters of Vibration Exercise

### *2.6.5.1 Duration*

Most vibration exercise studies have either been performed acutely for single or multiple sessions, either intermittently (30-60s exposure) or continuously (3-5 minutes) (Adams et al., 2009; Bosco, Cardinale, & Tsarpela, 1999a; Bosco et al., 1998; Bosco et al., 2000; Bullock et al., 2008; Cardinale et al., 2006; Cardinale & Lim, 2003; Cochrane & Stannard, 2005; Cormie, Deane, Triplett, & McBride, 2006; de Ruiter et al., 2003a; Di Loreto et al., 2004; Erskine et al., 2007; Hopkins et al., 2008; Rittweger et al., 2002a; Rittweger et al., 2001; Torvinen et al., 2002c). It appears that Bosco's (1998; 1999c) intermittent protocol of 10 repeated exposures of one minute interspersed with one minute rest has been a common duration used in subsequent vibration studies, but with little justification. Currently, there is little scientific evidence on what the optimal duration is for intermittent and continuous sessions. Nevertheless, Adams *et al*. (2009) found no significant differences in VJ peak power when untrained participants were exposed to vibration durations of 30s, 45s, or 60s (*f*=30-50 Hz *A*=2-4, 4-6mm). Moreover Stewart *et al*. (2009) reported that standing (5° knee flexion) on a SV, isometric peak torque increased by 3.8% after two minutes of continuous vibration, compared to decrements in peak torque at four and six minutes (*f*=26 Hz, *A*=4mm). In long-term vibration training studies, various exposure times have been reported for studies conducted over 6-12 weeks and 3-8 months (Delecluse et al., 2003; Roelants et al., 2004a; Torvinen et al., 2002b; Torvinen et al., 2003).

The research to date indicates that there is a limited window of opportunity to enhance muscular performance following acute vibration exercise (Adams et al., 2009; Bazett-Jones et al., 2008; Bosco et al., 1999c; Cormie et al., 2006; Lohman et al., 2007; Torvinen et al., 2002a). Cormie *et al*. (2006) found a significant increase in CMJ height immediately following

vibration exercise (*f*=30 Hz, *A*=2.5mm, VV), but this increase fell below baseline at 5 minutes post-treatment. Bosco *et al*. (1999c) also found that after acute vibration exercise (*f*=26 Hz, *A*=10mm, SV) showed temporary increases (lasting 10 minutes) in leg press average force, average velocity, and average power of the treated leg. Further, in healthy men and women Torvinen *et al.* (2002a) reported a significant increase in jump height (2.5%), isometric knee extension force (3.2%) and balance (15.7%) 2 minutes post acute vibration exercise (*f*=15-30Hz, *A*=10mm, SV), which returned to baseline levels post 60 minutes. Likewise, a 2.3% increase in vertical jump height has been recorded in untrained females post-5 minutes of acute vibration exercise (Bazet-Jones et al. 2008) while an acute vibration exercise bout (*f*=30-50 Hz, *A*=2-4;4-6mm, VV) increased CMJ peak power at 1 minute post-treatment and remained elevated at 5 minutes post-treatment but declined below significant levels at 10 minutes (Adams et al., 2009). Additionally, Lohman *et al*. (2007) noted that skin blood flow was significantly elevated 10 minutes post acute vibration (*f*=30 Hz, *A*=5-6mm, VV).

In summary, the majority of vibration exercise studies have been performed intermittently of 60s exposures with 60s rest, with some studies being conducted continuously from 90s to 6 minutes. However, there seems to be a knowledge deficit regarding the ability of continuous vibration exercise to enhance performance measures and more work is required to determine whether there is an optimal duration exposure time for vibration exercise in acute and long-term studies. Moreover, from the limited research, the transient increases from acute vibration exercise appear to last from 1-10 minutes and future research should focus on the dosage (vibration frequency, amplitude, and duration) and the latency post-vibration.

#### *2.6.5.2 Frequency*

Prior to the inception of commercialised vibration exercise, vibration studies focused on high vibration frequency; vibratory units were applied directly to the muscle or tendons of animals or humans for a very short duration. McCloskey, Matthews, and Mitchell (1972) applied direct vibration of 100-200 Hz to the hindlimb of a cat's tricep surae muscle, and Bongiovanni and Hagbarth (1990) applied two minutes of direct vibration of 150 Hz to human tendon ankle dorsiflexors to stimulate the tonic vibration reflex (TVR). It is not exactly known how the scientific merit of low frequency vibration was validated, but Vladimir Nazarov may have had a part in determining it. He used an arbitrary frequency of 23 Hz because he feared that the vibration would disappear during transmission in the tissue if a higher frequency was selected. Another explanation states that from a natural frequency lower limb muscles respond between a range of 5-65 Hz (Nigg & Wakeling, 2001). There is little scientific documentation on the appropriate vibration frequency (*f* ); however, Bosco *et al*. (1998) exposed handball and waterpolo athletes to 10 days of intermittent vibration (*f*=26 Hz, SV, Galileo) and reported an increase in vertical jump height by 12%, but gave no rationale as to why 26 Hz was selected. In

a follow up study, Bosco *et al*. (1999c) confirmed their earlier findings that vibration performed at 26 Hz on an SV (Galileo) platform had a positive effect on muscular performance by shifting the force-velocity and power-force relationship to the right, enhancing average force, velocity and power in a sample size of six elite volleyball players. Akin to their previous study, no rationale was given for the selection of the vibration frequency.

It took Cardinale and Lim (2003) to provide some insight on the optimal vibration frequency. Using electromyography (EMG) to validate the vibration frequency, these authors found that the EMG response of the vastus lateralis, as analysed by the root mean square (rms) was significantly higher in 30 Hz compared to 40 and 50 Hz when standing in a half squat position (knee angle 100 deg) on a VV (Nemes) platform for 60 seconds. Delecluse, Roelants, and Verschueren, (2003) also reported that standing in a static half squat position on a vibrating platform ($f$=35 Hz, Amplitude ($A$)= 5mm, acceleration = 3.9$g$) increased $EMG_{rms}$ of the rectus femoris and medial gastrocnemius muscles compared to the placebo condition ($f$=35 Hz, $A$= 5mm acceleration = 0.4$g$). However, both Delecluse *et al*. (2003) and Cardinale and Lim (2003) studies were conducted on a VV platform, with no comparison being made to a SV machine.

Recently, a number of studies have examined whether a dose relationship exists between different vibration frequencies and muscular performance, where the majority have used vertical jump (VJ) height as the performance measure. Da Silva *et al*. (2006) found that an intermittent vibration protocol performed at a frequency of 30 Hz ($A$=4mm) increased vertical jump height and leg power more than 20 Hz and 40 Hz. However, this study lacked a control condition; the body posture during vibration was not described; and no measures of EMG were collected. Likewise, Bazett-Jones Finch, and Dugan (2008) reported a significant increase of 9% and 8.3% in VJ height in young untrained women at 40 Hz and 50 Hz compared to the control condition, but there was no increase of jump height of untrained men. However, the small sample size (n=11) of the women compared to the men (n=33) may have incurred a type I error, and the vibration exposure time of 45s may have been insufficient to elicit the required neuromuscular responses for the males.

Vibration exercise is like any other form of training – every individual will have a response to an optimal intensity, and/or training load. In physical conditioning, most fitness programmes consider individual responses, and are tailored to suit the individual; however, in vibration exercise there has been a tendency for one vibration frequency to be used by all. Until recently, this was the status quo; however, Di Giminiani, Tihanyi, Safar, and Scrimaglio (2009) observed that after eight weeks (3x/week) of training with the vibration frequency individualised by determining the $EMG_{rms}$ activity of the vastus lateralis performed in a half squat position, the mean power of squat jump and jump height from a continuous rebound jumping test increased 11% and 18% respectively in comparison to the fixed (30 Hz), or no vibration. These results clearly indicate that vibration frequency should be individualised to fully maximise the benefits

of vibration training. Just as other fitness and resistance programmes are individualised for the client, vibration training should follow suit. However, it is unknown if $EMG_{rms}$ is the most appropriate measure to individualise each person's optimal vibration frequency. It may not be practical, as EMG equipment is often expensive, and may not be affordable or readily available in practical settings, such as clinics and gyms. It also requires a certain level of expertise. Further, it should be noted that in Di Giminiani's *et al*. (2009) study determined the optimal vibration frequency from analysing the EMG of one muscle (vastus lateralis). In the future it would be beneficial to record EMG activity from other muscle groups such as the knee flexors, ankle plantarflexors and dorsiflexors to confirm whether the same optimal vibration frequency is similar among the different muscle groups.

Vertical jump performance is not the only measure that has been used to determine optimal vibration frequency. Recently, a study used a range of frequencies to ascertain leg blood flow of participants that were exposed to one minute vibration bouts on an SV (Galileo) platform. The investigators reported that increasing vibration frequency from 10 Hz to 30 Hz significantly increased mean blood cell velocity in the femoral artery, with 30 Hz frequency showing the greatest increase of approximately 50% compared to resting levels (Lythgo, Eser, de Groot, & Galea, 2009).

In summary, many investigators have either used a fixed frequency, such as 18 Hz (Rittweger, Just, Kautzsch, Reeg, & Felsenberg, 2002b), 26 Hz (Bosco et al., 1998; Bosco et al., 1999c; Bosco, Iacovelli, Tsarpela, & Viru, 2000; Kerschan-Schindl et al., 2001; Rittweger, Beller, & Felsenberg, 2000; Rittweger, Mutschelknauss, & Felsenberg, 2003; Rittweger et al., 2001) and 30 Hz (de Ruiter, van der Linden, van der Zijden, Hollander, & de Haan, 2003a), for acute or long-term bouts of exposure. Other researchers have increased the vibration frequency during or after the completion of a session(s) (Delecluse et al., 2003; Roelants, Delecluse, Goris, & Verschueren, 2004a; Roelants, Delecluse, & Verschueren, 2004b; Torvinen et al., 2002a; Torvinen et al., 2002b; Torvinen et al., 2002c) but the optimal frequency remains equivocal. In an attempt to elicit the so-called neurogenic responses, contemporary research has used low vibration frequencies of 25-30 Hz for SV and 30-45 Hz for VV. However, there has been no agreement on determining the optimal vibration frequency, which has been confounded by the different protocols used in research. Only a few studies have directly measured neuromuscular activity (EMG), while the majority have focused on muscular performance, such as VJ height and power. The dissonance can be explained by the different types of vibration (SV, VV), the participant characteristics (body/muscle mass, training status, muscle strength and stiffness, age, gender), the different permutations of vibration parameters (amplitude, exposure duration, rest interval) and the prescription (number of repetitions, sets, exercises). Therefore, optimum dose relationship of frequency remains equivocal, influenced by various protocol designs, participants and outcome measures.

### *2.6.5.3 Amplitude*

Amplitude is defined as the maximum displacement of a vibration point from a mean position, compared to peak-to-peak amplitude (*A*), which is referred to as the height from the lowest to highest vibration wave (Figure 17). For SV, the amplitude is dependent on where the feet are placed on the plate. When the feet are close to the middle of the plate it equates to a small peak amplitude (~3mm). A wide stance equates to a greater amplitude (~12mm) (Figure 18). This differs to VV, where foot placement is independent of amplitude and has a pre-setting of 0-2mm or 4-6mm. Cardinale, Leiper, Erskine, Milroy, and Bell (2006) found no differences in insulin growth factor 1 (IGF-1) and testosterone levels when participants were exposed to high amplitude (*A*=3mm), low amplitude (*A*=1.5mm) and zero amplitude (*A*=0mm) at a fixed vibration frequency (*f*=30 Hz). The authors failed to document the type of vibration plate; therefore, if a VV was used with a small amplitude this may not have elicited the required response. Using a SV (Galileo) platform where participants stood in an upright stance (10° knee flexion) at a fixed frequency of 26 Hz, Rittweger *et al*. (2002a) reported that the oxygen cost increased in all three amplitudes (2.5, 5, 7.5mm) compared to baseline levels, with the highest amplitude (7.5mm) having the greatest oxygen cost (7.3 ml/kg/min compared to resting 3.6 ml/kg/min). For muscular power, Adams *et al*. (2009) showed that high vibration frequency (50 Hz, VV, [Power Plate]) with high amplitude (4-6mm), and low frequency (30 Hz) with low amplitude (2-4mm) were effective for increasing VJ power. Moreover, Lythgo *et al*. (2009) observed that low vibration amplitude ranging from 2.5mm to 4.5mm (SV) was able to elicit an increase in mean blood cell velocity by 27%.

To date, no study has directly compared the different amplitudes between the VV and SV platforms. As discussed earlier, the placement of the feet determines the amplitude in SV, which may affect the transmission of vibration to the various regions of the body. There has been some speculation that different body masses may alter the amplitude of vibration platforms; a heavier mass may decrease the amplitude of the platform. This presumption has been rejected based on recent evidence from Pel *et al*. (2009) who reported no change in amplitude when a SV (Galileo) and a VV (Power Plate) platform were loaded with two different body masses (62kg and 81kg). But the authors observed that the acceleration (*g*) in the vertical direction of the SV platform was reduced when vibration frequency was increased from 30 to 40 Hz. However, a wider range of body masses and the interaction of body mass and stance (knee angle) on amplitude requires further investigation.

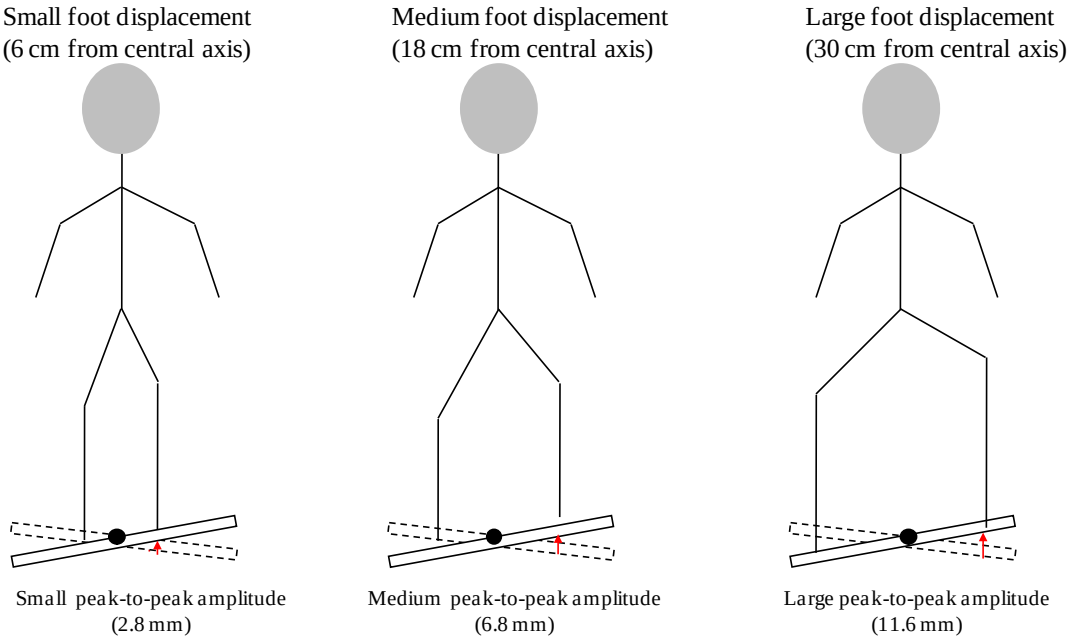



**Figure 18** Illustration of the different foot positions and the corresponding peak-to-peak amplitudes of SV (Galileo, Sport) performed at 26 Hz.

A current criticism of the literature is that most studies do not provide such details on how amplitude is calculated to whether it is measured by an accelerometer or computed by a mathematical equation. Much confusion surrounds the terms amplitude, peak-to-peak amplitude, and displacement because they are used interchangeably and standardisation of terminology is required.

In conclusion, the variable of amplitude has lacked the necessary consistency and scientific rigor to enable comparisons between studies. It is advised that a consensus be established in an attempt to gain consistency between research protocols. Lorenzen, Maschette, Koh and Wilson *et al*. (2009) have recommended that the term 'peak-to-peak amplitude' be used, and for vibrating platforms an anatomical landmark, such as the middle toe of the foot, be used to standardise the amplitude measurement, which needs be measured and reported.

#### *2.6.5.3 Acceleration*

VbX generates mechanical vibration resulting in acceleration, which is the product of angular velocity $(2 \pi f)^2$ and amplitude ($A$), where it has also been termed magnitude and the unit is either expressed as $m/s^2$ or as multiples of terrestric gravitation in $g$ (where $1g = 9.81$ $m/s^2$). Acceleration is proportional to the force applied. Therefore, increasing acceleration relies on changing the frequency and amplitude to increase acceleration of vibration being transmitted to the body (Cardinale & Wakeling 2005), which is similar to adding extra load in conventional resistance training ($F = m\ a$). VbX relies on increasing acceleration to increase force, where the force is likely to be the primary stimulus to promote changes within the body. However, as the vibrations travel through the body the effect of the force is likely to be damped by muscles, tissues and fluids (Wakeling, Nigg, & Rozitis, 2002).

A large number of studies have reported acceleration but have not documented how they measured or calculated it (Bosco et al., 1999c; Bosco et al., 2000; Cardinale, Ferrari, & Quaresima, 2007; Cardinale et al., 2006; Erskine, Smillie, Leiper, Ball, & Cardinale, 2007; Issurin, Liebermann, & Tenenbaum, 1994; Kerschan-Schindl et al., 2001; Rittweger et al., 2000; Russo & Lauretani, 2004; Torvinen et al., 2002a; Torvinen et al., 2002b). A few studies have directly measured the acceleration source by fixing accelerometers to vibration plates (Delecluse, Roelants, Diels, Koninckx, & Verschueren, 2005; Delecluse et al., 2003; Di Giminiani et al., 2009; Lythgo et al., 2009; Roelants et al., 2004a), and some researchers have placed accelerometers on body landmarks to determine the acceleration transmission of the vibration that passes through the various joints (Abercromby et al., 2007b; Crewther, Cronin, & Keogh, 2004). According to Lorenzen *et al*. (2009) all studies should document maximum acceleration ($m/s^2$ or $g$) and provide an explanation of how peak acceleration was determined.

#### *2.6.5.5 Exercises and Posture*

Static and dynamic squats are common exercises performed on vibration platforms (Abercromby et al., 2007a, 2007b; Bosco et al., 1999c; Bosco et al., 2000; Bullock et al., 2008; Cardinale et al., 2006; Cardinale & Lim, 2003; de Ruiter et al., 2003a; Di Loreto et al., 2004; Erskine et al., 2007; Hopkins et al., 2008); however, a combination of lower- and upper-body exercises have also been performed on various vibrating plates (Bosco et al., 1998; Cochrane & Stannard, 2005; Mahieu et al., 2006; Torvinen et al., 2002a).

The knee joint angle is a critical factor when performing a static or dynamic squat on a vibrating platform. Abercromby *et al.* (2007a) reported that a static squat at 18.5° provided greater muscle activation compared to dynamic squatting (10-35°knee angle). Abercromby *et al*. (2007a) also observed that during dynamic squatting on SV and VV platforms, $EMG_{rms}$ of vastus lateralis, gastrocnemius, and tibialis anterior activity were higher during knee flexion of 10-15° compared knee flexion of 31-35°. Caution should be used when interpretating Abercromby's *et al*. (2007a) findings, because the range of knee angle was only from 10-35° and only one knee angle (18.5°) was assessed for the isometric squat position. Further, it is unknown whether greater knee angles would continue to decrease muscle activity in dynamic squatting, and it is unknown what effect EMG activity has on high (120°) or low (90°) isometric squats. It remains equivocal whether a greater knee angle elicits a decrement in EMG activity for dynamic and static squatting, and if amplitude or frequency provides the greatest stimulus for muscle activity change. Using two knee flexion angles of 10° and 70° ($f$=20 Hz, $A$=5-9mm, VV, [Galileo 900]) Savelberg *et al*. (2007) reported that after 4 weeks (3x/week) of vibration exercise the 10° knee angle shifted to a more extended knee joint angle. In contrast, the larger knee angle (70°) shifted to a more flexed knee joint position, and the authors concluded that vibration caused a change in muscle length which shifted the knee angle. However, to confirm this finding a control condition should have been included. Therefore, future research needs to focus on different isometric knee angles in response to larger knee angles in dynamic squatting and its application to muscle performance.

### 2.6.6 Safety of Vibration Exercise

Occupational vibration can be detrimental to one's health, especially for workers who are constantly and continually exposed to vibrations from different types of machinery (Mester et al., 1999). However, most exercise vibration studies are conducted acutely and intermittently with no incidences of ill-effects having being reported. Conversely, Crewther *et al*. (2004) observed that untrained participants in vibration studies suffered from side-effects, such as hot feet, itching of the lower limbs, vertigo and severe hip discomfort. Likewise, Cronin, Oliver, & McNair (2004) reported that untrained participants suffered from vibration pain of jaw, neck and lower limbs, which subsided after 7-10 days of physiotherapy treatment. However,

Crewther *et al*. (2004) and Cronin *et al*. (2004) did not fully disclose how the participants were familiarised on the vibration platform, or whether they used an exclusion criteria for possible vibration side-effects. Both studies required the participants to slightly flex their knees; this small knee angle may have increased the vibration transmission to areas such as the head or hip. Recent research has concluded that the smaller the knee angle, the greater the vibration transmission to the head (Pel et al., 2009; Abercromby et al., 2007b). The findings of Crewther *et al*. (2004) and Cronin *et al*. (2004) are very uncommon, but highlight the need for researchers and exercise specialists to be fully trained on the use of vibration technology before participants take part in vibration led exercise, research and rehabilitation programmes.

Additionally, it has been reported by Rittweger *et al*. (2000), Kerschan-Schandl *et al*. (2001), Russo *et al*. (2003), Roleants *et al*. (2004b), Hazell, Thomas, DeGuire, and Lemon (2008), and Broadbent *et al*. (2008) that vibration exercise can elicit an erythema of the lower limbs, with anecdotal reports from participants suggesting that vibration exercise causes a hot sensation of the legs and acute itchiness, which normally subsides within minutes and has no deleterious effect on the body. However, it is unknown what causes the itchiness; one proposal suggests that the increase in blood flow is the main contributor, while another thought is that vibration induces skin shear forces which promotes vasodilation and is mediated by release of histamine (Rittweger, 2010). Likewise, Broadbent *et al*. (2008) has postulated that vibration may cause an excitatory response on mast cells to produce histamine, which causes vasodilation and promotes erythema itchiness. Results from their study, however, indicate that histamine levels were lower in the leg receiving vibration ($f$=40 Hz, $A$=5mm, VV) after muscle damage was elicited from downhill running, compared to those with muscle damage who received no vibration. Further, they speculate that the increase in blood flow from the vibration could have increased the clearance rate of histamine. Therefore, the mechanism causing erythema remains unidentified, and warrants further investigation.

## 2.6.7 Proposed Mechanisms of Vibration Exercise

*Introduction*

The mechanism(s) of vibration exercise currently remains equivocal because early theories were based on findings from direct muscle or tendon vibration. This differs to vibration exercise, where the whole body or parts of the body are vibrated, stimulating various muscles, tendons, organs and bones. Caution is therefore required when using evidence gained from direct vibration to explain the possible mechanism(s) of vibration exercise.

Vibration exercise has been reported to increase EMG activity, muscle force, and power (Bosco, Cardinale, & Tsarpela, 1999a; Bosco et al., 1999c; Cardinale & Lim, 2003; Torvinen et al., 2002a) where the excitatory response of the muscle spindle is speculated to play a role in enhancing muscle activation, involving the spinal reflex mechanism. The muscle tuning response is another possible mechanism where the muscular system damps the vibration stimuli to promote muscle activity. Additionally, the neural adaptations that occur from resistance and power training have been purported to be similar to that of acute vibration exercise. Neural aspects, such as motor unit recruitment, synchronisation, and co-contraction may be responsible for force and power increases following vibration exercise. Other mechanisms of vibration, such as a warm-up effect, where friction between the vibrating tissues may raise muscle temperature (Issurin et al., 1999), together with a vibratory induced increase in blood flow (Kerschan-Schindl et al., 2001) may contribute to enhancing muscle performance. It is unlikely that muscle hypertrophy occurs after an acute bout of vibration exercise due to the small load placed on the muscle-tendon unit (Issurin, 2005). However, muscle hypertrophy has not been directly measured from long-term vibration studies and remains an area worthy of further consideration. To date there has been no direct attempt to identify which mechanism(s) may contribute to the enhancement of neuromuscular performance. Therefore, the following sections will explore and critique the current proposed mechanisms of vibration exercise.

### *2.6.7.1 Spinal Reflex Mechanism*

As a result of muscles being directly vibrated, an ensuing muscle contraction occurs with reciprocal inhibition of its antagonists, which has been termed the tonic vibration reflex (TVR) (Eklund & Hagbarth, 1965; Eklund & Hagbarth, 1966; Matthews, 1966). This response causes an excitatory response of the muscle spindle Ia fibres (Burke, Hagbarth, Lofstedt, & Wallin, 1976b; De Gail, Lance, & Neilson, 1966b; Roll, Vedel, & Ribot, 1989) that is mediated by monosynaptic and polysynaptic pathways (Matthews, 1966; Romaiguere, Vedel, Azulay, & Pagni, 1991). The excitability of the motoneurons innervating the antagonist muscles is depressed by reciprocal inhibition (De Gail et al., 1966; Lance, Degail, & Neilson, 1966), and

the monosynaptic stretch reflexes of the vibrated muscle are suppressed during the vibration (De Gail et al., 1966; Marsden, Meadows, & Hodgson, 1969).

Burke *et al.* (1976b) found that when direct vibration of 20-220 Hz ($A$=1.5 mm) was applied to a tendon of non-contracting muscle, the primary endings responded more during high frequency vibration than secondary endings, which corresponds with earlier findings from cat studies conducted by Bianconi and van der Meulen (1963) and Brown, Engberg, and Matthews (1967). However, Burke *et al*. (1976b) also found that both primary and secondary endings respond by discharging in synchrony to the vibration stimulus, which contradicts Bianconi *et al*. (1963) findings. The explanation for this discrepancy is that Bianconi *et al*. (1963) did not differentiate between endings that were unresponsive and those capable of being activated from vibration.

When the vibratory stimulus is applied to an agonist muscle it causes reciprocal inhibition of the antagonist motoneurons (Bishop, 1974); when vibration is applied simultaneously to the agonist and antagonist muscles they cancel each other's facilitatory effect Hagbarth, (1967). However, when both muscles are independently vibrated they show a strong TVR (Bishop, 1974). Vibration also causes suppression of the muscle's phasic stretch reflexes (De Gail et al., 1966; Marsden et al., 1969). Arcangel, Johnston, and Bishop (1971) observed that during tendon vibration, the Achilles tendon reflex and the Hoffmann (H response) were totally suppressed, but in the post-vibratory period the reflexes were potentiated, indicating that pre-synaptic inhibition of the Ia afferent terminals occurred (Gillies, Lance, Neilson, & Tassinar, 1969). Desmedt *et al*. (1978) confirmed that motor unit recruitment is suppressed by high vibration amplitudes, because directly applied high frequency vibration is likely to cause pre-synaptic inhibition of Ia afferents.

According to Bishop (1974), four factors affect the TVR response: 1) the location of the vibration; 2) the excitability state of the CNS; 3) the initial length of muscle i.e. pre-stretch; and 4) the vibration frequency and amplitude. Research has suggested that the initial length of muscle influences the strength of the TVR where further lengthening of a muscle will induce a stronger TVR (Bishop, 1974). Vibration amplitude can determine how much stretch is imparted to the muscle. Matthews (1966) and Brown *et al*. (1967) reported that in decerebrated cats increasing the vibration amplitude caused an increase in TVR, because it activates a larger number of muscle-spindle endings that causes more α motoneurons to be activated (Luo, McNamara, & Moran, 2005). However, the range of amplitudes which this augmentation occurred was between 25-150 μm (Bishop 1974). Moran, McNamara, and Luo (2007) have argued that higher vibration amplitudes may only benefit sub-maximal contractions and have proposed that in maximal voluntary contractions the Ia afferent discharge may reach a saturation threshold, where vibration is unable to cause further increases in Ia afferent inflow. Evidence for this is based on observations that vibration can only increase maximal isometric contraction force and EMG activity when fatigue is present in the intrafusal fibres

(Bongiovanni & Hagbarth, 1990) or when α-fibre are blocked (Hagbarth, Kunesch, Nordin, Schmidt, & Wallin, 1986).

When high frequency (>100 Hz) muscle vibration is applied for a prolonged time, the force-generating capacity decreases because it elicits a suppression of Ia afferent activity. It has been reported that prolonged vibration applied to a single synergist muscle decreases knee extensor MVC for 30 minutes post-vibration (Kouzaki, Shinohara, & Fukunaga, 2000). The proposed mechanism for the reduction in MVC following prolonged vibration is a reduced firing rate of high-threshold motor units compared to low-threshold motor units (Bongiovanni & Hagbarth, 1990). This has been supported by Ushiyama, Masani, Kouzaki, Kanehisa, and Fukunaga (2005), who reported that after prolonged vibration (30 minutes) on the human Achilles tendon at 100 Hz ($A$=1.5mm), plantar flexion torque declined due to decreased EMG activity in the medial and lateral gastrocnemius compared to soleus. Given that both medial and lateral gastrocnemius muscles contain a larger portion of high-threshold units than soleus (Johnson, Polgar, Weightma, & Appleton, 1973), it suggests that Ia afferent activation is required to maintain recruitment of high-threshold motor units to increase force-generating capacity. Another possibility is that prolonged vibration causes excitability of the motor cortex, which could influence the voluntary drive. It has been previously been noted that when transcranial magnetic stimulation and electrical stimulation were applied before and after muscle vibration ($f$=80 Hz, $A$=0.5 mm) to the right extensor carpi radialis (Kossev, Siggelkow, Kapels, Dengler, & Rollnik, 2001), the elicited motor-evoked potentials were increased by transcranial magnetic stimulation, but not by the electrical stimulation. This suggests that cortical mechanisms may play a role in muscle vibration to augment motor-evoked potentials.

In summary, it has been proposed that vibration exercise causes a response similar to that of tonic vibration reflex (TVR). The basis of this theory is that vibration elicits excitatory responses in the Ia endings of muscle spindles, which cause the extrafusal fibres contract via the α motoneurons to contract (Cardinale & Bosco 2003), where muscle spindles fire in synchrony with the vibration cycle (Bishop 1974), which increases muscle activation (Eklund & Hagbarth 1966; Brown et al., 1967). It has been reported that force production is enhanced in stretch-shortening activities from the facilitation of stretch-reflex activity initiated by Ia afferents to produce an excitatory effect on the α motoneurons (Brown et al., 1967; Burke, Hagbarth, Lofstedt, & Wallin, 1976a). However, TVR requires directly applied vibration to the muscle or tendon at high vibration frequencies (>100 Hz) but typically, vibration exercise is not directly applied to the body and it is lower in frequency (20-45 Hz) and longer in exposure (>30s). Therefore, it is difficult to uphold the view that vibration exercise may act through a spinal mechanism, such as TVR. However, vibration may cause an indirect involvement of muscle spindles, where other sensory inputs may influence γ motoneuron activity, causing changes to spindle input (Gandevia, 2001).

### *2.6.7.2 Muscle Tuning*

There is evidence to suggest that the body is capable of tuning its muscle activity in order to reduce the vibrations that are passing through the soft tissue that may produce a detrimental effect (Nigg, 1997). The amount of muscle activity required is dependent on the level of vibration, where maximal muscle activation can reduce or purge oscillations within the tissues. Everyday activities such as walking, running and jumping result in impact forces, from the collision of the heel with the ground produce vibrations of 10–20 Hz to the lower limbs (Wakeling & Nigg, 2001). It has been proposed that an input signal from the impact force produces muscle activity or 'tuning' response to reduce soft tissue vibrations. This activation or tuning supposedly occurs shortly before the heel strikes the ground (Nigg, 1997). Consequently, muscle tuning relies on three components: the frequency and amplitude of the input force, the vibration resonance of the soft tissue and the level of muscle activity. In an attempt to test if additional muscle activity is used to minimise the vibrations, a hydraulic actuator was used to mimic the force that occurs during running, by directly evoking vibrations onto the sole of a foot using a range of vibration frequencies (10-65 Hz) (Wakeling al., 2002). The outcome of the study found that lower limb soft tissues were damped, and that resonance was reduced at heel strike. However, the natural frequency of the soft tissues did not change in way that paralleled the frequency of the input. The mechanism of this remains unclear, but if damping does occur in soft-tissues where resonance is minimised at heel strike and the changes in frequency are a consequence of altered muscle activity then energy absorbed by the muscle is likely to involve cross-bridge cycling (Ettema & Huijing, 1994; Wakeling et al., 2002). Consequently, damping vibration will depend on the individual's neuromuscular response of muscle spindle, the sensitivity of joint and skin receptors, the proportion of muscle fibre types and viscoelastic (stiffness) elements (Bazzett-Jones et al., 2008). As a result of impact forces there is possibility of vibrations occuring in soft tissues that have viscoelastic properties. Mechanical energy from the vibration can be stored and returned from the elastic structures of the muscle-tendon complex where damping of vibrations result in a net dissipation of mechanical energy that can be absorbed by activated muscle (Albasini, Krause, & Rembitzki, 2010).

In conclusion, impact forces create vibrations in the foot, and the vibration travels proximally through the lower limb. To prevent resonance, the vibrations are damped by soft tissues, which cause sensory organs to send impulses to the central nervous system (CNS). The CNS responds by increasing muscle activity and adjusting joint stiffness.

#### 2.6.7.3 Neural

It has been proposed that the increases in muscle force and power following vibration exercise are similar to that of resistance training (Bosco et al., 1999a; Bosco et al., 1998; Bosco et al., 1999c; Cardinale & Bosco, 2003; Delecluse et al., 2003). According to Cardinale and Bosco (2003), resistance training and vibration exercise both place load on the neuromuscular system. In resistance training this is achieved by adding an extra load to increase the influence of gravity, in the form of barbells, dumbbells, elastic or manual resistance. For vibration, the extra load is not achieved by increasing the mass (unless barbells and dumbbells are used) but by increasing acceleration by adjusting vibration frequency and/or amplitude. Increasing the load has the ability to modify the neuromuscular aspects through neurogenic and myogenic factors. The initial changes seen from resistance and power training are often attributed to neural aspects (Aagaard et al., 2002a; Gabriel, Kamen, & Frost, 2006; Sale, 1988; Staron et al., 1994), and although protein synthesis, a precursor for hypertrophy, can occur after just one resistance training session (Chesley, Macdougall, Tarnopolsky, Atkinson, & Smith, 1992) it is not until approximately six weeks of training that hypertrophy is fully realised (Kraemer et al., 2002). However, this has recently been challenged, as the fascicle length has been shown to increase within 35 days of performing an isoinertial knee extension exercise (Seynnes et al., 2007). Therefore, a closer examination of how vibration exercise may influence neural responses of motor unit firing, motor unit synchronisation, inter-muscular co-ordination and central motor command is required.

##### 2.6.7.3.1 Motor Unit Firing Frequency

Motor unit firing frequency refers to the number of impulses per second that the muscle fibre of a motor unit receives from the motoneuron. The force output of every motor unit can be varied by the firing frequency from 10 to 60 impulses per second during sustained contractions (Sale, 2003). However, in high speed movements firing rates of 60-120 impulses per second are common (Desmedt & Godaux, 1977), which may last for 100ms and end before force production (Desmedt & Godaux, 1979). As force increases, more motor units may be recruited and/or the firing frequency increases. All motor units must be recruited and fired at an optimal rate for the force of contraction to be maximal. However, not all muscles show the same characteristics; the firing frequency and recruitment differ from muscle to muscle. Rapid changes in motor unit firing frequency have been reported in both young and older adults, after completing six weeks of resistance training (Kamen & Knight, 2004). The authors found that vastus lateralis motor unit firing frequency increased by 15% and 49% in young and older adults respectively during maximal isometric knee extension. Likewise, Patten, Kamen, and Rowland (2001) reported that the firing frequency of the abductor digiti minimi was significantly increased 48 hours after completing maximal isometric contractions. These results

indicate that an increased firing frequency, with a corresponding increase in force output, suggests that the early gains in force production from resistance training are partly due to motor unit firing frequency. However, when high frequency vibration (150 Hz) was applied over the tendons of ankle dorsiflexor muscles, Bongiovanni and Hagbarth, (1990) reported that the firing frequency in non-fatigued muscle did not change, but the vibration did increase force, EMG, and firing frequency during fatiguing isometric maximal voluntary contractions. On the contrary, Griffin, Garland, Ivanova, and Gossen (2001) reported that the firing frequency increased when direct vibration was applied to the distal tendon of the triceps brachii following a two minute sustained 20% maximal voluntary contraction (MVC). The authors suggest that during the isometric contraction muscle spindle activity declined to support the motoneuron pool, and consequently firing frequency decreased. However, muscle spindles are responsive to vibration, especially the Ia afferents (Burke et al., 1976b; De Gail et al., 1966; Roll et al., 1989), which may prevent a decline in muscle spindle activity to support the motoneuron pool (Griffin et al., 2001).

#### *2.6.7.3.2 Motor Unit Synchronisation*

Motor unit synchronisation relies on the number of motor units firing at any one time. Synchronisation occurs from branched inputs of pre-synaptic neurons that produce a common synaptic input to increase the chance of motoneurons discharging simultaneously (Fling, Christie, & Kamen, 2009). Increased motor unit synchronisation has been used to explain an increase in force and power production following resistance training (Gabriel et al., 2006; Sale, 1988; Semmler, 2002). Milner-Brown *et al*. (1975) investigated the discharge rates of the first dorsal interosseous muscle and found that synchronisation was greater in weightlifters than control participants. However, synchronisation was estimated using an indirect method of averaging surface EMG signal with respect to motor unit discharge, which has since been challenged (Yue, Fuglevand, Nordstrom, & Enoka, 1995). By using direct motor unit measurements, Semmler and Nordstrom (1998) reported that resistance trained weightlifters produced a higher level of synchronisation compared to highly skilled musicians, indicating that synchronisation may be an adaptation from resistance training to producing greater force production. Yao, Fuglevand, and Enoka (2000) found that during motor unit synchronisation, EMG increased 65-135% with corresponding force fluctuations. Martin and Park (1997) reported that when direct vibration was applied to finger and wrist muscles, harmonic synchronisation decreased; however, sub-harmonic synchronisation increased when vibration frequency increased from 40 to 200 Hz, but high vibration frequency (>150 Hz) produced less motor unit synchronisation.

Following acute vibration exercise, vertical jump performance in females remains enhanced 5 minutes post-vibration (Bazett-Jones et al., 2008), while skin blood flow remains elevated 10

minutes after vibration (Lohman et al., 2007). The enhancement in EMG, force, and power following acute vibration exercise has been used as indirect evidence of improved motor unit recruitment (Bosco et al., 1999a; Bosco et al., 1998; Bosco, Colli, Cardinale, Tsarpela, & Bonifazi, 1999b; Bosco et al., 2000; Cardinale & Bosco, 2003; Delecluse et al., 2003). However, according to Yue *et al*. (1995) surface EMG is a poor indicator of motor unit synchronisation and caution is required when interpreting results. Therefore, it remains untested whether motor unit synchronisation occurs during vibration exercise, because no study has assessed single motor unit discharge.

#### *2.6.7.3.3 Inter-muscular Co-ordination*

Inter-muscular co-ordination is the interaction of a number of muscle groups during a muscular activity. Contraction of the agonists may be associated with simultaneous contraction of their antagonists (Sale, 1988). Tyler and Hutton (1986) reported antagonist co-contraction reduced agonist force production with a decrease in EMG. When performing new and complex tasks the co-contraction of antagonists, through reciprocal inhibition, may impair agonist contraction (Sale, 1988). However, through continual practice and training the level of co-contraction may be reduced, which allows a greater activation of the agonists. Carolan and Cafarelli (1992) reported that after eight weeks of knee extensor resistance training, force increased in the knee extensors, with a reduction in antagonist (bicep femoris) co-contraction shown by a decrease in bicep femoris EMG activity.

It has been stated that vibration exercise causes neural changes synonymous to that of resistance and power training (Bosco et al., 2000). If this is true, an improvement in agonist activation and increased inhibition of antagonist muscles should also exist in vibration exercise. It is well known that direct vibration excites the muscle spindle, which activates the Ia afferents. These afferents, in turn, excite the α motoneurons of the homonymous muscle, which may uncouple the co-contraction of agonist-antagonists (Eklund & Hagbarth, 1966). Rothmuller and Cafarelli (1995) reported that antagonist co-contraction increased during fatigue, and that vibration elicited a greater co-contraction than the control condition, but the co-contraction did not change during the fatigue of either condition.

Recently, Abercromby *et al*. (2007a) suggested that vibration exercise may elicit neuromuscular responses that may be partly modulated by leg muscle co-contraction at the knee joint. In a study conducted by Mischi and Cardinale (2009), they investigated muscle activation and co-contraction of biceps and triceps brachii muscles during isometric exercise with and without vibration. Using an electromagnetic actuator, mechanical sinusoidal vibrations were generated for 15s at 28 Hz to the biceps and triceps brachii. They observed that co-contraction occurred at low levels of muscle force (20% and 40% maximum sustained force) during elbow extension, suggesting that co-contraction may serve to stabilise the joint, but it does not modulate agonist

force production. The authors concluded that vibration may be a viable option in the early stages of rehabilitation where low levels of muscle force are required due to limited joint mobility.

#### *2.6.7.3.4 Central Motor Command*

It has been shown that direct application of high frequency vibration on muscle and tendon activates Ia afferents of the muscle spindles and, to a lesser extent, the secondary afferents and Ib afferents of GTO (Roll et al., 1989). However, there is some evidence that cortical areas of the brain receive and process proprioceptive information when direct high frequency vibration is applied, which generates evoked cortical potentials (Munte et al., 1996). It appears that muscle afferent input to the cerebral cortex has a role in motor control (Wiesendanger & Miles, 1982); 30% of central motor drive is attributed to muscle afferent excitability (Macefield, Gandevia, Bigland-Ritchie, Gorman, & Burke, 1993). Moreover, it has been reported in humans that Ia afferent input has the ability to excite the corticospinal pathway (Carson et al., 2004) and activate the cortical motor areas (Lewis, Byblow, & Carson, 2001). To examine the potential changes of the motor cortex, the transcranial magnetic stimulation (TMS) method has been used to study the excitability of the corticospinal system, as well as the intracortical inhibitory and facilitatory processes. In brief, the TMS delivers pulses to the motor cortex through a double cone coil that is centred over the scalp. It has been reported that motor-evoked potentials of TMS were enhanced when direct vibration ($f$=80 Hz) was applied to extensor carpi radialis muscle (Kossev et al., 2001). It has been suggested, without verification, that vibration exercise may influence the motor cortex to increase muscular performance (Cardinale & Bosco 2003). Recently, Mileva, Bowtell, and Kossev (2009) investigated the effects of vibration exercise ($f$=30 Hz, $A$ = 1.5mm, VV [FitVibe]) on corticospinal excitability and intracortical processes by studying motor-evoked potentials in the tibialis anterior and soleus muscles in response to TMS of the contralateral motor cortical leg area. They found that vibration exercise increased tibialis anterior corticospinal excitability pathway, but the intracortical facilitatory processing for tibialis anterior was reduced. However, no significant changes were evident in the corticospinal excitability or processing intracortical facilitatory of the soleus. These findings suggest that vibration exercise may stimulate the corticospinal pathway; however, no performance measures were included with TMS, and further work is required in other lower limb muscles, such as the quadriceps and hamstrings, before it can be confirmed that vibration exercise causes an excitatory response of the motor cortex.

In conclusion, it is possible that spinal reflexes, muscle tuning and neural mechanisms enhance muscular performance, but not by working in isolation. Other body systems may also play a role, such as hormone secretion, blood flow, muscle temperature, skin, joint receptors and central motor command.

### *2.6.7.4 Tensegrity*

Tensional integrity or tensegrity is when biological structures such as muscles and bones, or rigid and elastic cell membranes, are made strong by the unison of tension and compression (Ingber, 1997). Tension is continuous and compression discontinuous, where continuous pulling is balanced by the equivalent discontinuous pushing force (Albasini et al., 2010). In the human body, tension is provided by the connective tissue and the bones and fluid provide the compression. When mechanical stresses or physical forces are applied it influences the growth and remodelling of tissues at the molecular level, where molecular components of the extracellular membrane and cytoskeletal properties undergo change (Ingber, 2003). Figure 19 provides an illustration of the complementary force balance between tensed microfilaments, compressed microtubules and transmembrane integrin receptors and living cells, where 1→2 refers to the chemical conversion of substrate 1 to substrate 2, which represents a change in kinetics. (Ingber, 2003).

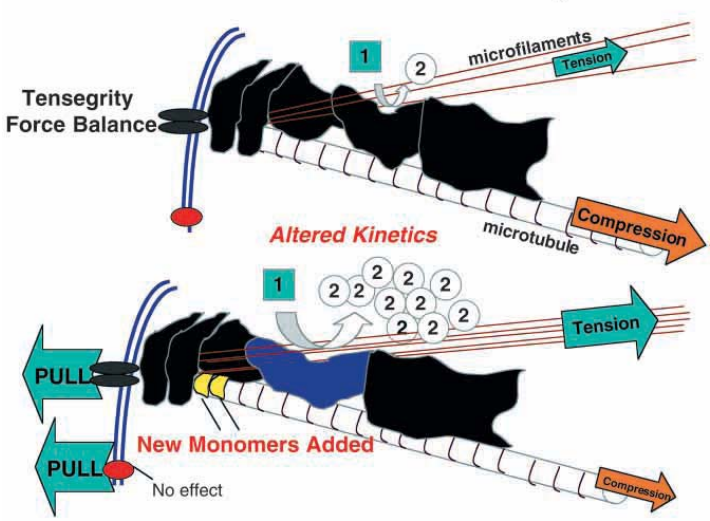



**Figure 19** Contribution of cellular tensegrity to mechanochemical transduction (Ingber, 2003)

According to Ingber (1997) living cells and nuclei are quick to respond when mechanical stresses that are transmitted over the cell receptors that physically combine the cytoskeleton to extracellular matrix or to other cells. Although the mechanism of tensegrity remains unclear, this microcellular theory suggests that the sinusoidal oscillating stimulus of vibration exercise has the potential to induce mechanochemical conversion (Albasini et al., 2010), where heat stress proteins and cytoskeletal filaments may help explain the positive effect that vibration has on tissues and blood vessels. Further, the tensegrity theory proposes that direct mechanotransduction of vibration to cell-cell adhesion molecules at a cellular cytoskeletal level are also likely to have a significant impact on pre-stress and health of muscular tissue through

the additional influx of mechanical kinetic and potential energy, which vibration exercise imparts (Albasini et al., 2010). However, if this mechanism is to be realised, advanced vibration research at the molecular level needs to be conducted.

## 2.7. The Effects of Vibration Exercise

*Introduction*

The previous sections have centred on the mechanics of vibration exercise (the types, parameters and proposed mechanisms), which have provided an understanding of vibration exercise. The focus will now turn to exploring and critiquing the literature on how vibration exercise affects reflex and muscle activity, the various body systems, and muscular performance. Although, this is a large and diverse area to review, it is important to appraise the literature in order to explore and uncover the potential effects of vibration exercise.

### 2.7.1 The Effects of Vibration Exercise on Reflex Activity and Jendrassik Manoeuvre

#### *2.7.1.1 Tendon Reflex (Stretch Reflex)*

Typically the tendon reflex (also known as the stretch reflex) is monosynaptic. An external stimulus causes the muscle to lengthen and the muscle spindle is stretched, which sends an impulse via the sensory afferent that causes an excitatory response of the efferent α motoneuron to produce muscle contraction (shortening) (Kandel, Schwartz, & Jesse, 2000).

Rittweger *et al*. (2003) reported an enhancement of the patellar tendon stretch reflex following continuous vibration exercise (*f*=26 Hz, *A*=6mm, SV, [Galileo]) with dynamic squatting to exhaustion (349s), and suggested that α motoneurons were augmented by the vibration, which recruited high-threshold units and muscle fibres. However, Hopkins *et al*. (2008) found no effect on patellar tendon reflex after intermittent vibration exposure (*f*=26 Hz, *A*=4mm, SV, [Galileo 2000]). In another study, Melnyk, Kofler, Faist, Hodapp, and Gollhofer (2008) elicited the stretch reflex in the hamstrings by inducing an anterior tibial translation during standing, and compared the reflex response between a control and eight minutes after intermittent vibration group (*f*=30 Hz, *A*= 4mm, VV, [Power Plate]). The researchers found post-vibration, anterior tibial translation displacement decreased with a corresponding increase in EMG of the hamstring short latency response. The authors concluded that the effect of increased knee stability was caused by reflex excitability. However, from this study it is not conclusive whether the stretch reflex was potentiated from vibration exercise, and it remains speculative whether spinal reflexes are the main mechanism. Therefore, future research should examine whether an acute continuous exposure of vibration exercise augments the stretch reflex by using the patellar tendon and/or Achilles tendon.

### *2.7.1.2 Hoffmann Reflex (H-Reflex)*

The Hoffmann reflex (H-reflex) is a measure of assessing monosynaptic activity of the spinal cord (Palmieri, Ingersoll, & Hoffman, 2004). The H-reflex differs to the tendon reflex because it is activated by electrical rather than mechanical stimulation, which bypasses the muscle spindle by acting directly on the afferent fibre. The electrical stimulus causes a corresponding H-reflex, which is the result of increased excitability of the Ia afferent and α motoneurons. As the strength of the electrical stimulus increases, the efferent fibres become excited and induce a direct muscle contraction, known as the *M-response*. Further increase in the stimulus strength results in suppression of the H-reflex but M-response becomes augmented.

When an external stimulus excites the axon of an α motoneuron, it generates an action potential that travels in both directions – to the muscle (*orthodromic conduction*) and to the spinal cord (*antidromic conduction*). The orthodromic conduction elicits the M-response, and the antidromic conduction either induces an action potential or it dissipates (Figure 20). If the strength of the electrical stimulus is further increased, the axon of the α motoneurons will conduct action potentials both ortho- and anti-dromically. This results in more and more motoneurons unable to be conducted by Ia afferents and the H-reflex begins to disappear, while the M-response commences its peak amplitude. The diminishing of the H-reflex is due to the orthodromic action potential travelling towards the motoneuron and colliding with the antidromic action potential, which is travelling in the motoneuron toward the spinal cord. Several parameters can be measured from the H-reflex, such as H max, which represents the maximal reflex activation (Palmieri et al., 2004). M max refers to the activation of the entire motoneuron pool (Pierrot-Deseilligny & Mazevet, 2000) and its maximum muscle activation Zehr (2002), and H max/M max ratio provides a measure of the entire motoneuron pool being recruited (Palmieri et al., 2004), which is influenced by pre-synaptic inhibition (Zehr, 2002).

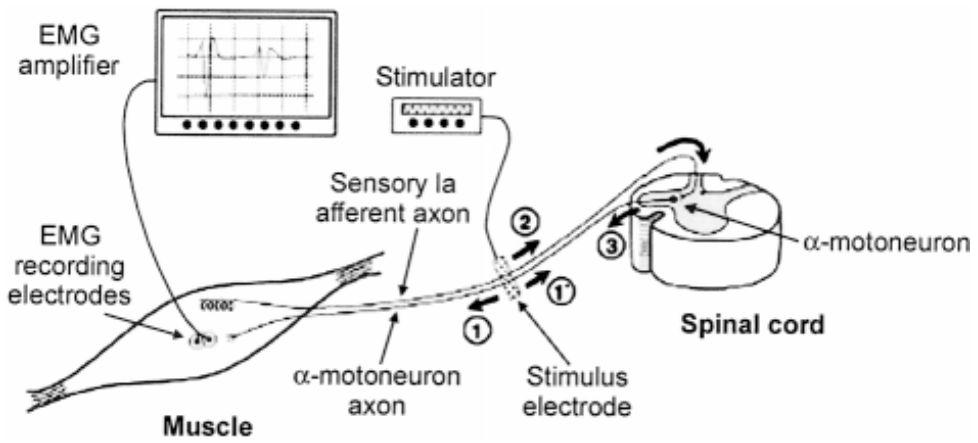



**Figure 20** Illustration of the Hoffmann reflex (Aagaard et al., 2002b)

Thompson and Belanger (2002) reported that self-paced inline skating for 35 minutes elicited a mean vibration frequency of 141 Hz from the skate chassis and 34 Hz from the middle portion of the tibia, which resulted in the H-reflex being suppressed by 35% compared to resting conditions. The authors concluded that pre-synaptic inhibition was the main factor for suppressing the reflex response. Similarly, Armstrong *et al*. (2008) found the H-reflex was suppressed in the first minute post-vibration after a single minute bout of VV ($f$=40 Hz, $A$= 2-4 mm). But earlier work from Nishihira and associates (2002) found that post-vibration the H-reflex and H max/M max ratio was enhanced, suggesting that motoneuron excitability was heightened. The dissonance between Nishihira and associates (2002), and Armstrong *et al*. (2008) findings is probably due to the different protocols. Nishihira *et al*. (2002) used a three minute exposure performed on SV platform ($f$=25 Hz) where the H-reflex was elicited from a seated position, which differs to Armstrong's *et al*. (2008) one minute duration performed on VV ($f$= 40 Hz) with the H-reflex elicited from a supine position. More disconcerting was that both studies lacked a control and the authors failed to fully disclose the exact testing protocol of the H-reflex.

#### *2.7.1.3 Jendrassik Manoeuvre*

The Jendrassik manoeuvre involves contracting remote muscles, normally of the upper-body (particularly the forearm and jaw muscles) to induce a reflex response (Jendrassik, 1885). A common method for eliciting a reflex is to grasp the hands and pull them apart – this potentiates the stretch reflex and H-reflex (Delwaide & Toulouse, 1980; Dowman & Wolpaw 1988). In the clinical setting, the Jendrassik manoeuvre has been used to induce a full-sized reflex in neurologically impaired patients. In young (25yrs) and older (75yrs) healthy people when vibration ($f$=100 Hz, $A$=2mm) was directly applied to the quadriceps it produced a post-vibration decrease in patella tendon reflex force, but the younger group showed a greater reflex inhibition (Burke, Schutten, Koceja, & Kamen, 1996). Moreover, the effect of the Jendrassik manoeuvre facilitated the patella tendon reflex more in young people than in the older group (97% vs. 64%). The authors concluded that the age-related changes were due to changes in pre-synaptic inhibition pathways and motoneuron input resistance, confirming that the integrity of the spinal interneuronal pathways deteriorate with ageing (Burke et al. 1996). The reduction in muscle vibration inhibitory effect of older adults suggests that the pre-synaptic inhibition of Ia afferents may deteriorate with an increase in age, or the aged muscle spindle may reduce the number of Ia afferents activated by the vibratory stimulus. However, the mechanism of the Jendrassik manoeuvre remains unclear because the fusimotor system and the pre-synaptic disinhibitory effect on motoneurons can influence the manoeuvre (Gregory, McIntyre, & Proske, 1989).

## 2.7.2 The Effects of Vibration Exercise on Muscle Activity

### *2.7.2.1 Lower Body*

It is possible to measure electromyography (EMG) activity without any artefacts in response to vibrating muscle. Seroussi, Wilder, and Pope (1989) evoked vertical sinusoidal vibrations (*f*=3 to 10 Hz; *A*=0.4-13mm) from a servohydraulic shaker, and were successful in removing motion artefacts by passing the raw EMG through a phaseless digital six pole Butterworth high pass filter with a cut-off frequency of 30 Hz. They found that when EMG of the erector spinae was adjusted for torque, a significant increase (19%) in mean torque was observed. Likewise, Bongiovanni and Hagbarth (1990) reported that tibialis anterior EMG activity and single motor unit discharge were augmented from a pneumatic vibratory unit (*f*=150 Hz, *A*=1.5mm).

Using a similar device, Warman *et al*. (2002) applied an actuator (*f*=50 Hz, *A*=5mm) to the quadriceps and reported an increase in $EMG_{rms}$ of the rectus femoris during isometric (30%), isokinetic (43%), and concentric (107%) contractions. In a subsequent study, Humphries, Warman, Purton, Doyle, and Dugan, (2004) used the same vibrating actuator and reported no significant differences in peak normalised $EMG_{rms}$ between the vibrating and resting conditions. Additionally, there were no corresponding changes in the rate of force development or peak force. However, the vibration exposure duration was not documented, therefore it may have been too short or too long to elicit the desired responses. Similarly, Torvinen, *et al*. (2002c) found no significant changes in soleus mean power frequency and $EMG_{rms}$ from four minutes of vibration (*f*=25-40 Hz, *A*=2mm, VV), but reported a decrease in mean power frequency and $EMG_{rms}$ for the vastus lateralis and gluteus medius muscles, which was accompanied by no changes in muscle function.

In a subsequent study, Tovinen *et al*. (2002a) used the same exercise routine and time constructs as the previous study, but in the latter study the participants performed the exercise routine on a SV plate and the vibration frequency was incrementally increased from 15-30 Hz (*A*=10mm). During vibration $EMG_{rms}$ was significantly augmented in the soleus and gastrocnemius but there was no change in vastus lateralis $EMG_{rms}$. However, an increase in isometric leg force and vertical jump height was reported. The authors provided no discussion on the dissonance between the two studies, consequently the reader can only surmise, that either the type of vibration plate (VV vs. SV) or vibration parameters (*f*=15-30 vs. 25-45 Hz, *A*=2mm vs. 10mm) may explain the variation in results. However, it is feasible that the action of the side alternating plate with a larger amplitude may have contributed to the increase in muscle performance. Recent evidence has reported that using a fixed frequency (30 Hz) and amplitude (4mm), a SV platform generates greater muscle activation of lower limb muscles compared to a VV platform (Abercromby et al., 2007a).

### 2.7.2.2 Upper Body

The effect of upper-body vibration exercise on EMG activity has produced similar results to that of lower-body vibration. Using a vibrating dumbbell, Bosco *et al*. (1999a) observed that during intermittent vibration ($f$=30 Hz) biceps brachii $EMG_{rms}$ increased two-fold compared to baseline measurements, and during post-vibration bicep power was augmented but no corresponding increase in EMG activity was found. Further evidence of increased $EMG_{rms}$ has been observed from vibrating isometric elbow pull and push actions, with increases in co-contraction at loads of 20% and 40% of maximum force (Mischi & Cardinale, 2009). The authors speculate that the mechanism for increased $EMG_{rms}$ cannot be entirely accounted for by spinal reflexes but by increased motor unit synchronisation and firing frequency.Although Mischi and Cardinale (2009) study provides some new insights to the response of superimposing vibration on agonist, antagonist and co-contraction of muscles, caution is required because only one vibration frequency (28 Hz) was tested with brief exposures of vibration and the recorded absence of amplitude restricts the findings of the study. Conversely, Moran *et al*. (2007) reported that when a custom built vibrating unit was directly placed on the bicep brachii tendon ($f$=65 Hz, $A$=1.2mm) it did not elicit an increase in $EMG_{rms}$ during the lifting phase of bicep curls at 70% 1RM. Similarly, post-vibration showed no enhancement in $EMG_{mpf}$ (mean power frequency) and peak force or power.

Finally, a novel study was conducted to investigate muscle activity on a VV platform on both the lower (vastus lateralis, and biceps femoris) and upper limb muscles (biceps brachii and triceps brachii; (Hazell, Jakobi, & Kenno, 2007). The investigators used a range of frequencies ($f$=25, 30, 35, 40, 45 Hz), amplitudes ($A$=2 and 4mm), and body positions of static squat, dynamic squat, static bicep curl and dynamic bicep curl. They reported that a static squat $EMG_{rms}$ of vastus lateralis and biceps femoris was augmented when vibration frequency and amplitude were increased, but no increase in muscle activity was evident in static and dynamic bicep curls, probably because the transmission of vibration was damped by the lower extremity. It is important to note that the raw EMG signal was passed through a sixth order Butterworth filter between 100 Hz and 450 Hz, which may have removed important muscle activity signals.

### 2.7.2.3 Posture

Roelants *et al*. (2006) has recently determined whether posture affects muscle activation by investigating three different isometric squat positions of a two-leg, high squat (knee angle 125°, hip angle 140°); two-leg, low squat (knee and hip angle 90°); and one-leg, high squat (knee angle 125°, hip angle 140°). The investigators reported that vibration increased $EMG_{rms}$ activity more in a one-leg high squat, compared to two-leg high and low squats. However, further testing is required to determine if any EMG differences exist in squatting positions that occur between 90° and 125° knee flexion.

Abercromby *et al*. (2007a) investigated muscle activity of different lower limb postures of static squat (18.5°) and dynamic squat (eccentric and concentric 10-35°knee angle) performed on both SV and VV plates (*f*=30 Hz, *A*=4mm). For isometric, eccentric and concentric muscle action, $EMG_{rms}$ increased significantly in all four lower limb muscles for both SV and VV. In support of this, Delecluse *et al*. (2003) reported that standing in a static half-squat position on a VV platform (*f*=35 Hz, *A*= 5mm, 3.9*g*) increased gastrocnemius and rectus femoris $EMG_{rms}$ activity, compared to placebo (0.4*g*) control (no vibration); however, the sampling period was only conducted over 20s. Conversely, Cormie *et al*. (2006) observed that in a half-squat position (knee angle 100°) there were no changes in average iEMG (integrated) activity of the vastus lateralis, vastus medialis, bicep femoris during vibration treatment (*f*=30 Hz, *A*=2.5mm, VV [Power Plate]).

#### *2.7.2.4 Prolonged Vibration*

Unlike acute exposure of vibration, prolonged vibration exercise causes a decrease in muscle function. Herda *et al*. (2009) reported that prolonged 20 minute of direct Achilles tendon vibration (*f*=70 Hz, *A*=not given) decreased EMG amplitude of the gastrocnemius and soleus by 10% and 25%, with a corresponding decrease in ankle plantar flexion isometric peak torque of 5%. These findings are consistent with previous reports (Jackson & Turner, 2003; Richardson et al., 2006) that prolonged vibration has a deleterious effect on EMG activity and force parameters. It has been proposed that prolonged vibration may cause a decrease in γ loop excitability that causes a decrease in the force-generating capacity (Bongiovanni & Hagbarth 1990; Kouzaki et al., 2000). Contrarily, Rittweger *et al*. (2003) reported EMG median frequency of the vastus lateralis increased significantly after exhaustive squatting exercise (349s) with vibration (*f*=26 Hz, *A*=12mm) and extra load (40% of body mass), suggesting that type I motor units were recruited during the fatigue.

In summary, during vibration exercise EMG activity can be augmented from 34 to 361% (Cardinale & Lim, 2003; Bosco et al., 1999a; Roelants et al., 2006). This large range is most likely due different EMG capture times with various vibration machines, frequencies, amplitudes and postures. However, it is unclear whether an increase in muscle activity during vibration will enhance post-vibration muscle performance. It is also unknown whether vibration exercise causes muscle length changes that are associated with temporal EMG activity.

### 2.7.3 The Effects of Vibration Exercise on Metabolism and Body Composition

From EMG vibration exercise studies there is strong evidence to suggest that muscle activation is elicited; however, the energy demand in response to vibration exercise is quite low. Rittweger *et al*. (2000) compared the fatigue effects of exhaustive vibration with dynamic squatting (3s up, 3s down) plus additional load (40% of body mass) to that of progressive exhaustive cycling. It was found that vibration ($f$=26 Hz, $A$=10.5mm, SV) could only elicit a metabolic demand of 23 ml/kg/min compared to 44 ml/kg/min of the cycle test. In a follow up study, Rittweger *et al*. (2001) found that standing on a vibrating plate ($f$=26 Hz, $A$= 6mm, SV) elicited 10.2 ml/kg/min compared to no vibration of 4.8 ml/kg/min, while dynamically squatting at a tempo of 3s up and 3s down (to 90° knee flexion) for three minutes recorded 14 ml/kg/min with vibration and 10.7 ml/kg/min without vibration. The authors propose that the increase in oxygen uptake was indirectly related to muscle activity and vibration at 26 Hz ($A$=6mm) was equivalent to moderate walking at 4.5 km/hr (1.25m/s).

In a further study Rittweger *et al*. (2002a) quantified the effect of metabolic demand by examining a range of vibration frequencies ($f$=18, 26, 34 Hz) and amplitudes ($A$=2.5, 5, 7.5mm). They reported that oxygen uptake increased proportionally to an increase in vibration frequency, with external load showing a similar trend. However, oxygen uptake increased more than proportionally at higher amplitudes of 5mm and 7.5mm. The researchers concluded that the increase in oxygen demand may be parametrically controlled by frequency, amplitude, and external load when applying vibration. The aforementioned studies (Rittweger et al., 2000, 2001, 2002a) suggest that vibration may cause a small increase in metabolic rate that results from muscular work and reflects a reflex response, for which TVR may be responsible. However, the above studies (Rittweger et al., 2000, 2001, 2002a) have failed to directly measure muscle activity, thus, to date, there is no direct evidence to substantiate that during vibration muscle length changes as a result of mechanical work.

Using indirect calorimetry procedures, Da Silva *et al*. (2007) reported that vibration exercise ($f$ =30 Hz, $A$=4mm, VV) with 10RM half-squats produced a significantly higher energy expenditure (18.8 kJ/min) compared to without vibration (15.9 kJ/min). This is equivalent to Rittweger's *et al*. (2001) finding of ~19.3 kJ/min of squatting with vibration and confirms that energy expenditure is enhanced when vibration is combined with resistance exercise. Using $\dot{V}O_2$ and respiratory exchange ratio to calculate total energy expenditure, carbohydrate and fat oxidation rate, Garatachea *et al*. (2007) found that a fast cadence 2s (1s up, 1s down) of dynamic squatting with vibration ($f$ =30 Hz, $A$=4mm, VV [Nemes]) produced a greater oxygen cost and higher total energy expenditure compared to slower squatting cadences of 4s (2s up, 2s down) and 6s (3s up, 3s down). However, the highest fat oxidation proportion and the lowest carbohydrate oxidation proportion was evident in the squatting cadence of 6s, suggesting that

slower squatting may be more effective for fat burning, but the total energy expenditure was higher in the faster squats. An estimation of the energy cost of vibration (*f*=26 Hz, *A*=6mm) can be indirectly calculated. Based on Rittweger *et al*. (2001) findings that three minutes of vibration exercise (*f*=26 Hz, *A*=6mm, SV) for a 70 kg person increases oxygen uptake by 4.5 ml/kg/min would equate to an expenditure of 18.9 L of $O_2$/hr. Assuming an energy equivalent of 20.9 kJ/L of $O_2$ and a caloric equivalent of 37 kJ/g of body fat, this would accrue a loss of ~ 10.7g fat/hr ([18.9x20.9]/37). Therefore, it is unlikely that vibration exercise can be exclusively used as a form of aerobic exercise to reduce body fat. This has been be supported by Roelants *et al*. (2004a), who observed no changes in body weight, percentage body fat or skinfold thickness in young untrained females following 24 weeks (3x/week) of vibration training. Further there were no reported differences in body composition between the combined cardiovascular/resistance training and control groups. Although these young female participants were classified as untrained, no specific data was given on their baseline fitness levels; however, the vibration group were non-significantly heavier (3.8kg) and had a higher percentage body fat (2.5%) compared to cardiovascular/resistance group. A further limitation of this study was that the vibration and the cardiovascular/resistance training groups were not identically matched for volume or training intensity. This is difficult to achieve, however, because there are currently no prescription guidelines to increase the vibration parameters in a progressive and systematic fashion.

Given the limitations of Roelants *et al*. (2004a) the study still indicates that vibration exercise cannot reduce body fat, which requires a combined strategy of conventional aerobic exercise with dietary changes (Hammer, Barrier, Roundy, Bradford, & Fisher, 1989). However, Roelants *et al*. (2004a) did report an increase in muscle strength and fat free mass from the vibration group; therefore, changes in body composition may have gone undetected by the small increase in lean muscle tissue. Conversely, Fjeldstad *et al*. (2009) reported that percentage body fat decreased by 3.2% in sedentary postmenopausal women who performed vibration exercise (*f* =30 & 40 Hz, *A*=3mm, VV, [Power Plate]) for eight months (3x/week) in comparison to resistance and control groups. However, increases in lean tissue mass for the total body, arm and trunk were similar between vibration and resistance training groups after eight months. The difference between the findings of Fjeldstad *et al*. (2009) and Roelants *et al*. (2004a) is that Roelants *et al*. (2004a) participants were younger and more active than Fjeldstad *et al*. (2009) postmenopausal women. Moreover, the resistance training group of Fjeldstad *et al*. (2009) performed resistance exercises only; in comparison Roelants *et al*. (2004a) performed both resistance and aerobic exercises which would have increased the metabolic rate. Finally, the method of measuring body composition differed between the two studies. Fjeldstad *et al*. (2009) used DEXA (dual energy X-ray absorptiometry) and Roelants *et al*. (2004a) used under-water weighing, and skinfolds.

Recently, a 12 month study was conducted on older men and women (60-80yrs) to investigate the effect of intermittent vibration (*f*=35 & 40 Hz, *A*= 2.5 & 5mm, VV [Power Plate]) and conventional exercise (aerobic, resistance, balance & flexibility) on cardio-respiratory fitness (Bogaerts et al., 2009). The researchers observed that post-training $\dot{V}O_2$ peak was significantly higher in both vibration exercise (18%) and conventional exercise groups (21%) compared to control (8%). However, the time to reach peak $\dot{V}O_2$ was significantly higher (14%) in conventional exercise compared to vibration exercise (9%) and control (6%). Caution is required when interpreting the results as the vibration and conventional groups were not matched for intensity, metabolism, or load. Further, no measurement of the metabolic cost was taken during the vibration or conventional exercise sessions, and the study lacked a placebo intervention.

A few studies have examined local muscle metabolism using near-infrared spectroscopy (NIRS) technology with varying results. Yamada *et al*. (2005) found that fast dynamic squatting for three minutes with vibration (*f*=15 Hz, *A*=2.5mm, SV [Galileo]) increased muscle oxygenation of the vastus lateralis compared to squatting with no vibration, which the authors attributed to an increase in oxygen uptake. Further confirmation that vibration increases oxygen consumption through muscle activation has also been made by Mileva, Naleem, Biswas, Marwood, and Bowtell (2006). They observed that the rate of vastus lateralis deoxygenation (as estimated from the slope of HHb) was significantly faster (32%) after 35% 1RM knee extension exercise with vibration, compared to non-vibration. On the contrary, Cardinale *et al*. (2007) found no significant differences between muscle oxygenation levels of vastus lateralis and gluteus maximus performed at 30, 40, 50 Hz (*A*=4mm, VV [Fitwave]). The difference between these findings may be explained by the different protocols of the exposure time, vibration platform type (VV vs. SV), vibration frequency, and body positions.

Recently, there has been some support for the idea that muscle perfusion may play an important role in vibration exercise. Using $^{31}P$ magnetic resonance spectroscopy ($^{31}P$-MRS), Zange, Haller, Muller, Liphardt, and Mester (2009) reported an increase in ATP consumption from a custom built vibration pedal (*f*=20 Hz, *A*=2mm) but it was not significantly different to no vibration. However, when arterial occlusion was applied to the ipsilateral thigh, ATP consumption increased by 60%, PCr increased by 35% and pH by 0.16 units compared to no vibration. This confirms the findings of Rittweger *et al*. (2001) that vibration exercise elevates metabolism, but it cannot elevate energy turnover by large amounts.

In summary, the evidence firmly suggests that vibration exercise can increase whole and local oxygen uptake; however, even with additional load, or high vibration frequency and/or amplitude it cannot match the demands of conventional aerobic exercise. Therefore, caution is required when a vibration exercise programme is solely used for the purpose of reducing body fat without considering dietary and aerobic conditioning guidelines. To date, only one study has

reported that vibration exercise is able to burn extra calories but it cannot reduce overall body fat (Fjeldstad et al., 2009). Akin to Rittweger's *et al*. (2000, 2001) studies have conclusively reported that vibration is not a suitable option to reduce body weight, as the metabolic cost of a 'standard' vibration session is equivalent to burning only 10g/hr. Finally, it is equivocal whether the increase in energy turnover can be accounted for by an increase in muscle activation caused by neural potentiation, which has been based on spinal reflexes. However, to date, no direct measures of muscle have been made to verify that vibration exercise acts solely through a reflex potentiation causing a change in muscle length that increases oxygen uptake. It has been suggested by Cardinale and Bosco (2003) that vibration causes small and rapid changes in muscle length by eliciting reflex muscle activity in an attempt to damp the mechanical vibration, where various researchers have speculated that the muscle activation is similar to that of TVR (Bosco et al., 1998; Bosco et al., 1999a; Rittweger et al., 2000; Cardinale & Lim 2003; Cardinale & Bosco 2003; Cardinale & Rittweger 2006).

## 2.7.4 The Effects of Vibration Exercise on Cardiovascular System

Previous sections have stated that vibration exercise is a rhythmic activity that evokes a small increase in oxygen uptake, which indicates muscle energy turnover exists where vibration may have a beneficial effect on the cardiovascular system especially blood flow, heart rate, blood pressure and arterial stiffness.

### *2.7.4.1 Blood Flow*

Hand-held tools are normally operated at high vibration (80-100 Hz), which has been shown to decrease blood flow to the digits of the hand, resulting in 'white finger vibration' (Lundstrom & Burstom, 1984; Noel, 2000). However, Kerschan-Schindl *et al*. (2001) were the first to report that vibration exercise (*f*=26 Hz, *A*=3mm, SV [Galileo]) increases blood flow of the popliteal artery (100%) and causes erythema in the foot and calf. Furthermore, Lythgo *et al*. (2009) found that when an intermittent vibration protocol was performed (*f*=10 Hz to 30 Hz, SV [Galileo]), an increase in mean blood cell velocity of the femoral artery was evident, with 30 Hz providing the greatest increase in blood flow compared to resting levels. In contrast, Hazell *et al*. (2008) found that an intermittent vibration protocol (*f*=45 Hz, *A*=2mm, VV [Wave]) reported no increases in femoral artery blood flow after three minutes. Likewise, Button, Anderson, Bradford, Cotter, and Ainslie (2007) reported no significant differences in leg blood flow of a vibrating cushion (*f*=60 Hz, *A*=not given, VV [ATL]) placed under the gluteal muscles, and a hand unit placed under the right foot while in the seated position. In this particular study, no rationale was given for using a hand-held device applied to the foot, or why the vibrating cushions were applied to the gluteals rather than directly to the legs. Additionally, the participants were seated with knees and ankles at 90°, which suggests that vibration transmission may have been damped, attenuating blood flow.

When both calf muscles were rested on a VV platform (f=30 Hz, A=5-6mm, [Power Plate]) for 3 minutes the gastrocnemius skin blood flow increased by 250% compared to baseline level and remained elevated (200%) for 10 minutes (Lohman, Scott, Maloney-Hinds, Betts-Schwab, & Thorpe, 2007). However, using the same vibration parameters but performing an isometric squat and calve raises with and without vibration, did little to increase skin blood flow. Maloney-Hinds, Petrofsky, and Zimmerman (2008) found that when the forearm was placed on a VV platform [Power Plate] at 30 & 50 Hz (*A*=5-6mm) for 10 minutes it significantly increased skin blood flow within five minutes and remained elevated for nine minutes post-vibration. However, the study did not include a control (0 Hz), and no documentation was given as to how much force was applied to the forearm during the vibration, which may have affected blood flow.

### *2.7.4.2 Heart Rate, Blood Pressure, and Arterial Stiffness*

Vibration exercise has been reported to have little effect on heart rate (HR) and blood pressure (Button et al., 2007; Hazell et al., 2008; Otsuki et al., 2008). Kerschan-Schindl *et al*. (2001) observed no change in HR, systolic and diastolic blood pressure values after vibration (*f*=26 Hz, *A*=3mm, SV [Galileo]). However, exhaustive vibration exercise did increase heart rate and systolic pressures by 30% and 15% (Rittweger et al., 2000). To date, only one study has investigated the effects of vibration on arterial stiffness by measuring brachial-ankle pulse velocity (Otsuki et al., 2008). Following intermittent vibration (*f*=26 Hz, *A*=2-4mm, VV [Power Plate]) arterial stiffness was significantly reduced (3%) at 20 and 40 minute post-vibration compared to the same time series of no vibration.

In conclusion, vibration exercise has little effect on HR and blood pressure, unless extra load is used with dynamic exercise; however, vibration exercise cannot increase HR to the same extent as conventional aerobic exercise. Conversely, there is reasonable evidence to suggest that vibration exercise increases skin and muscle blood flow and decreases arterial stiffness. As yet no study has investigated the short and long-term effects of vibration exercise on blood flow and arterial stiffness, which may have clinical applications in the health area to reduce the healing time of skin ulcers or when microcirculation is compromised in patients, such as diabetics (Maloney-Hinds et al., 2008).

### 2.7.5 The Effects of Vibration Exercise on Hormonal Levels

It has been well documented that resistance training of large muscles performed at moderate to high intensity with high volume is capable of elevating hormonal levels, such as testosterone and growth hormones, which are essential to muscular power and force production (Kraemer & Ratamess, 2005). Similarly, vibration exercise is capable of using high loads to activate large muscle masses; the neuromuscular system may be potentiated by the spinal reflex activity and certain parts of the brain may be centrally activated to stimulate hormonal secretion (Cardinale & Bosco 2003).

Bosco's *et al*. (1999b) study was one of the first to investigate the acute effect of vibration exercise on hormonal response. The researchers exposed eight healthy active handball players to a single session of intermittent vibration (*f*=26 Hz, *A*=10mm, SV [Galileo]) and found that serum testosterone and cortisol levels significantly decreased compared to baseline levels. The authors concluded that vibration exercise acted in a similar manner to heavy resistance training, where at the start of heavy resistance training testosterone levels in the blood decrease. However, this study lacked a control condition to compare the effects of vibration with that of no vibration. In a follow up study, Bosco and associates (2000) used a similar vibration protocol without a control condition and observed that physically active males produced a significant increase in testosterone (7%) and growth hormone (360%) with a significant decline in cortisol (32%). Using Bosco's *et al*. (2000) intermittent vibration protocol, albeit at a slightly higher frequency (30 Hz) Di Loreto *et al*. (2004) reported a significant decrease in plasma glucose concentration (3%, p=0.049) five minutes post-vibration compared to control, with an increase in norepinephrine (28% p=0.038) after 25 minutes post-vibration. Additionally, no significant changes in plasma concentrations of glucagon or serum concentrations of insulin, cortisol, growth hormone, insulin growth factor-1 (IGF-1) and total testosterone were reported.

In a similar intermittent vibration protocol (*f* = 26 Hz, *A* = 2.5mm, SV [Galileo]) Goto and Takamtsu (2003) reported a significant increase in plasma epinephrine and norepinephrine (42%, 58% respectively) that returned to baseline levels post-20 minutes, but detected no significant differences in serum growth hormone. They also reported that free fatty acids (FFA) were elevated post-vibration but no corresponding increases in serum glycerol were evident. It remains unclear what role vibration exercise may have in lipolysis.

Further support from Cardinale *et al*. (2006), Erskine *et al*. (2007) and Cardinale, Soiza, Leiper, Gibson, and Primrose (2008) has confirmed that vibration exercise has little effect on increasing hormonal responses. Cardinale *et al*. (2006) reported no significant increases immediately after and 24 hours post-vibration for serum testosterone or IGF-1. Using salivary sampling, Erskine *et al*. (2007) observed that an intermittent vibration protocol (*f*=30 Hz, *A*=4mm) had no effect on testosterone or cortisol concentration at any time points post-immediately, 1 hour, 2 hours, and 24 hours. Likewise, Cardinale *et al*. (2008) reported that in

elderly participants (70 yrs), plasma hormone concentrations of testosterone and growth hormone did not change significantly over time from using a five minute intermittent protocol (*f*=30 Hz, *A*=4mm, VV [FitVibe]). However, there was a significant elevation in IGF-1 (28%) and cortisol (46%) immediately post-vibration.

To date, most of the research has focussed on acute intermittent vibration exposures, and has been based on Bosco's *et al*. (2000) protocol; however, little research has been conducted on the effects of continuous acute vibration exercise, or the response of dynamic squatting and extra load. Recently, Kvorning , Bagger, Caserotti, and Madsen (2006) conducted a nine week study where young male participants were exposed to SV while dynamic squatting at a vibration of 20-25 Hz (*A*=4mm), 2-3x/week with a load equalling 10RM, and observed no significant increases in testosterone, growth hormone following completion of the programme.

In conclusion, there is little consensus on whether hormonal secretion is augmented by acute vibration exercise. This is due to early studies having no control condition (Bosco et al., 1999b, 2000), while later studies used a similar protocol to that of Bosco *et al*. (2000) and reported contrasting results, which can be attributed to the various methods used to sample the hormonal secretion, the small sample sizes and varying fitness levels of participants. Future research should investigate whether acute continuous vibration exercise can change hormonal levels in different sub-populations, such as post-menopausal women, and whether external loading of the body at different amplitudes and frequencies can enhance hormonal secretion.

### 2.7.6 The Effects of Vibration Exercise on Body Temperature and Warm-Up

It has been reported that vibration exercise produces erythema of the lower limbs (Broadbent et al., 2008; Hazell et al., 2008; Kerschan-Schindl et al., 2001; Rittweger et al., 2000; Roelants et al., 2004b; Russo & Lauretani, 2004), with anecdotal reports from participants that vibration exercise causes a hot sensation in the legs and acute itchiness that subsides within minutes of the vibration. It has been speculated that post-vibration, flexibility increases (Issurin et al., 1994), as does explosive power, due to increased muscle temperature (Issurin & Tenenbaum, 1999).

De Ruiter *et al*. (2003) have also suggested that muscle temperature plays a part in countering a possible decline in maximal rate of force development post-vibration exercise, and Warman *et al*. (2002) have proposed that localised vibration induces vasodilation, which raises muscle temperature. Conversely, Cronin *et al*. (2004) have speculated that vibration exercise may enhance muscle performance by increasing muscle temperature, which masks the so-called neurogenic potentiation. To date, only a few studies have examined the effects of vibration on skin temperature. Using a vibratory device ($f$=100 Hz, $A$= not given) secured to the forearm for 15 minutes, Oliveri, Lynn, and Hong (1989) observed skin erythema with an increase in skin temperature that remained elevated for 10 minutes post-vibration. Hazell *et al*. (2008) also reported an increase in thigh skin temperature following 16 minutes of intermittent vibration ($f$=45 Hz, $A$=2mm, VV [Wave]). The authors proposed that the increase in skin temperature was due to the reflexive muscle contractions causing changes in muscle fibre length, which could increase muscle temperature and augment skin blood flow to dissipate heat.

Some authors have advocated that vibration exercise may be used as a warm-up procedure, but this has been without any scientific rationale and based on observations that vibration exercise increases VJ height (Cormie et al., 2006; Bazett-Jones et al., 2008). Recently, Bullock *et al*. (2009) investigated the effect of vibration exercise as a warm-up procedure. They reported that when five female elite skeleton athletes performed a specific and extensive warm-up in addition to three bouts of 60s vibration exercise ($f$=45 Hz, $A$=4mm, VV [Nemes]), it produced no effect on 30m sprint time. The authors conceded that the potential effects of vibration may have been masked by the intense specific warm-up performed prior to the vibration protocol. However, Bullock *et al*. (2009) did not take any direct measurements of core or muscle temperature to test their claim. Previous research has used muscle performance tests to speculate that vibration exercise can be used as an effective warm-up modality, but this has been misleading because it is unknown if core and muscle temperature actually increase from vibration exercise, and if they do, whether this concurrently potentiates performance more than conventional warm-up procedures. In summary, the effect of vibration exercise on muscle, skin and core temperature remains speculative, untested and warrants further investigation.

### 2.7.7 The Effects of Vibration Exercise on Balance and Proprioception

The balance system of the human body relies, in part, on the somatosensory feedback, which diminishes in elderly persons and is associated with increased chances of falling (Judge, King, Whipple, Clive, & Wolfson, 1995). Likewise, balance can be problematic in patients suffering from Multiple Sclerosis, Parkinson's disease, and Stroke. Therefore, the oscillating nature of vibration exercise may have a beneficial effect on balance and proprioception in the ageing and compromised health groups. Research from Torvinen *et al*. (2002a) found that in healthy young participants (19-38yrs) static balance parameters improved by 15.7% after four minutes of vibration (*f*=15-30 Hz, *A*=4mm, SV [Galileo]). Using vibration exercise to increase balance performed over a short-term period has produced little effect in healthy people (Torvinen et al., 2002b). Likewise, Mahieu *et al*. (2006) found no significant differences in postural control of very young competitive skiers (mean age 12.4 yrs) that underwent six weeks of vibration exercise training (*f*= 24-28 Hz, *A*=2-4mm, VV [FitVibe]). However, these young skiers may not have fully developed their musculoskeletal or proprioceptive system to have benefited from the vibration training.

In the elderly, the effect of vibration has been beneficial. Priplata Niemi, Harry, Lipsitz, and Collins (2003) found that vibrating insoles (*f*= 100 Hz, *A*=was set individually) improved the lateral postural instability of older people (73 yrs) compared to young (23yrs), therefore vibration could overcome postural instability caused by age-related sensory loss in the elderly. In longer-term vibration studies there have been positive effects from using vibration exercise to improve balance in older people. Cheung *et al*. (2007) found that in elderly females (72yrs), exposure to vibration exercise (*f*= 20 Hz, *A*= not given, SV [Galileo]) for 3x/week (3 months) significantly improved balance, while Bautman, Van Hees, Lemper, and Mets (2005) reported that nursing home residents (76.6yrs) improved their balance after six weeks of vibration exercise (*f*= 35-40 Hz, *A*= 2mm, VV [Power Plate]) compared to a control group that performed the same static exercise regime without the vibration, but with a tape recording mimicking the sound of the vibration machine. Bruyere *et al*. (2005) also reported that institutionalised elderly people improved their body balance score after six weeks of combined vibration exercise and physical therapy training, and Runge, Rehfeld, and Resnicek (2000) observed that after two months of vibration exercise (*f* = 27 Hz, *A*=7-14mm, SV [Galileo]), chair-rising time improved by 18% in institutionalised elderly participants.

In post-menopausal women (65yrs), Verschueren *et al*. (2004) found a significant improvement in postural sway after six months of vibration. Likewise, Gusi, Raimundo, and Leal (2006) reported that in post-menopausal women (66yrs) postural balance as assessed by the blind flamingo test improved significantly (28%) after eight months (3x/week) of vibration exercise (*f*=12.6 Hz, *A*= 3mm, SV [Galileo 2000]). Moreover, Bogaerts, Verschueren, Delecluse, Claessens, and Boonen (2007b) found that healthy elderly people (67 yrs) showed dramatic

improvement in sway energy scores after six months of vibration (*f*=35-40 Hz *A*=2.5-5mm (VV [Power Plate]).

In people with compromised health, such as patients suffering from Multiple Sclerosis and Parkinson's disease, acute vibration exercise protocols have reported improvements in balance (Schuhfried, Mittermaier, Jovanovic, Pieber, & Paternostro-Sluga, 2005; Turbanski, Haas, Schmidtbleicher, Friedrich, & Duisberg, 2005). Ebersbach *et al*. (2008) reported that Parkinson's disease patients improved their balance by 38% from three weeks of vibration exercise. For stroke patients, improved balance and somatosensory measures were noted after six weeks of vibration exercise (*f*= 30 Hz *A*= 3mm, SV [Galileo 900]), but van Nes *et al*. (2006) reported no significant differences were found in balance tasks of stroke patients that received vibration or exercise therapy to music.

Depending on the type of injury that has been sustained, proprioception training will be required during the rehabilitation phase. For instance, following an ankle injury, balance boards with weight-bearing exercises are often used to retrain proprioception (Sheth, Yu, Laskowski, & An, 1997). It has been reported that in healthy young adults acute low frequency vibration (*f*=18 Hz, *A*=10mm, SV [Galileo 2000]) can improve lumbopelvic proprioceptive by 53% compared to those that received no vibration (Fontana, Richardson, & Stanton, 2005). However, Haas, Turbanski, Kaiser, and Schmidtbleicher (2004) found no improvement in proprioceptive performance in Parkinson's disease patients from acute vibration exercise. Trans *et al*. (2009) reported patients with knee osteoarthritis that performed eight weeks of vibration training (*f*=25-30 Hz, *A*= not given, [VibM]) showed a non-significant (p=0.051) improvement in proprioception with increases in knee strength.

In summary, there is some consensus that vibration exercise can improve static balance in the elderly and compromised health groups. Additionally, there are promising signs that proprioception is enhanced by vibration exercise which acts on muscle to induce changes in balance and proprioception, but extensive research is needed to confirm this. Furthermore, it would be of interest if future studies focussed on whether vibration exercise was able to improve dynamic balance and whether this could increase fall prevention in the elderly.

## 2.7.8 The Effects of Vibration Exercise on Muscular Performance

Various studies have been conducted on the effects of vibration exercise on power and force; therefore, this section will be subdivided into acute, short and long-term effects of lower-body and upper-body vibration exercise on power and force.

### *2.7.8.1 Power*

*Acute Vibration – Lower Body Power*

Vertical countermovement jump (CMJ) has been used by many investigators to assess muscle power and its acute effects on vibration exercise. The enhancement in VJ height has varied across the different research protocols. For instance, using a SV (Galileo) platform, Cochrane and Stannard (2005) reported an 8.2% increase in VJ height from five minutes of acute vibration ($f$=26 Hz, $A$=6mm), and Torvinen *et al*. (2002a) observed a 2.5% enhancement following four minutes of vibration ($f$=15-30 Hz, $A$=10mm). Studies that have used VV platforms (Power Plate, Nemes) have shown similar findings. Bosco *et al*. (2000) reported an increase in VJ height of 4% from 10 minutes of intermittent vibration ($f$ =26 Hz, $A$=4mm, VV [Nemes]). Brief, single vibration ($f$=30 & 40 Hz, $A$=2-4mm) exposures of 30s and 45s performed on a VV platform have respectively recorded a 0.6% and 9% improvement in VJ height (Cromie et al., 2006; Bazett-Jones et al., 2008). Conversely, Torvinen *et al*. (2002c) found no significant increases in VJ height when dynamic exercise was combined with four minutes of vibration exercise ($f$=25-40 Hz, $A$=2mm, VV [Kuntatory]). It is difficult to explain why Torvinen's *et al*. (2002a) earlier study reported a 4.4% increase, as both studies used similar protocols and participants. However, a VV platform was used in the latter study where the amplitude was smaller (2mm) than the previous study (10mm), which could have negated the desired responses.

Measuring muscle power has not only been confined to vertical jump. Leg press, weighted squats, and knee extension have also been used to assess muscle power from an acute bout of vibration exercise. Bosco *et al*. (1999c) found a 6-8% increase in single leg press power across loads of 70, 90, 100, 139 kg in elite volleyball players (n=6) from intermittent vibration exercise ($f$=26 Hz, $A$=10mm, SV [Galileo 2000]). Similarly, Rhea and Kenn (2009) observed a 5.2% increase in squat power (3 reps, 75% 1RM) of male college athletes that received vibration ($f$=35 Hz, $A$=4mm, VV [iTonic]) while dynamic squatting their body weight. However, the depth and cadence of the squat was not accounted for during the vibration or strength testing, which may have affected the results, especially when the athletes were instructed to complete each concentric phase as fast as possible. Therefore, the depth of the squat may not have consistently been attained in determining power output.

*Acute Vibration – Upper Body Power*

The effects of acute vibration on upper body power have been explored less so, and with varying results. Bosco *et al*. (1999a) found a post-vibration 8% increase in average power of bicep brachii from twelve international boxers that isometrically gripped a hand-held vibrating device (*f*=30 Hz, *A*=6mm). In a similar study, Issurin and Tenenbaum (1999) used an isotonic vibrating cable (*f*=44 Hz, *A*=3mm) where non-elite and elite athlete performed 3 sets of 3 reps (65-70% 1RM) at a tempo of 2s per rep. They reported that both non-elite and elite athlete groups produced a 10.2% and 10.7% increase in mean power and 10.4% and 7.9% increase in peak power during vibration compared to no vibration.

Further support of vibration enhancing upper-body power has been reported by Poston, Holcomb, Guadagnoli, and Linn (2007). Applying a vibrating electric motor to an Olympic barbell, experienced weight lifters performed 3 sets of 3 reps (70% 1RM) bench press without or with vibration (*f*=30 Hz, *A*=1.1mm). Although the average power of the bench press was higher with vibration than without, there were discrepancies in baseline power outputs, suggesting that either the athletes should have been blinded before receiving vibration or a greater familiarisation period with the vibration apparatus should have been included. Additionally, a placebo or sham vibration condition could have been included to overcome this problem.

Some researchers have shown little or no effect of vibration on upper-body power. Moran *et al*. (2007) observed no significant pre-post changes in power, moment, and angular velocity when a custom built vibratory unit (*f*=65 Hz, *A*=1.2mm) was directly applied to the tendon of bicep brachii while resistance trained males performed 3 sets x 5 reps of bicep curls (70% 1RM). The same group of researchers (Luo, Clarke, McNamara, & Moran, 2009) repeated a similar experiment and used the same vibratory unit (*f*=65 Hz, *A*=10mm) that was directly applied to the distal tendon of quadriceps, and confirmed their previous finding that vibration produced no changes in power variables. From these results the researchers proposed that when performing dynamic exercise, such as bicep curls, it relies on the stretch-shortening cycle where smaller vibration amplitude may be required to activate and optimise muscle spindle sensitivity. However, Bosco's *et al*. (1999a) findings contradict this because they reported an 8% increase in bicep brachii peak power from five minutes of intermittent dumbbell vibration (*f*=30Hz, *A*=6mm). Therefore, the direct application of the vibratory unit and its frequency (65 Hz) may have caused the insignificant findings from Moran *et al*. (2007) and Luo *et al*. (2009). Finally, Cochrane and Hawke (2007) have also reported no significant increases in upper-body power of climbers that were exposed to an electric powered vibrating dumbbell (3kg) (*f*=26 Hz, *A*=3mm, [TOP Galileo]).

### *Short-Term Vibration (<2 months) – Lower Body Power*

Repeated single bouts of vibration performed over days or weeks have been investigated. Bosco *et al*. (1998) reported an increase of 1.6% in CMJ height and a 11.9% increase in VJ height from continuous jumping protocol in handball and waterpolo players after receiving 10 days of intermittent vibration exercise (*f*=26 Hz, *A*=10mm, SV, [Galileo]). However, there are a few inconsistencies in this study; the control group did not perform the same exercise routine without vibration, and the continuous jumping protocol was not controlled for the amount of work completed, which could have influenced the VJ height between the control and vibration groups.

Further support for short-term vibration potentiating CMJ has been reported by Fagnani, Giombini, Di Cesare, Pigozzi, and Di Salvo *et al*. (2006) where competitive female athletes increased their CMJ by 8.7% from eight weeks of vibration training (*f*=35 Hz, *A*=4mm, VV [Nemes]). Annino *et al*. (2007) observed an increased in vertical jump height (6.3%) in well-trained ballerinas after performing two months (3x/week) of vibration exercise (*f*=30 Hz, *A*=5mm, VV [Nemes]) additionally, the average power, force and velocity of leg press increased significantly at loads of 50, 70, and 100kg. In a recent study where vibration frequency was set individually for each participant, the authors reported that after eight weeks (3x/week) of vibration training, squat jump height increased by 11%, continuous rebound jump height was enhanced by 22% and mean power significantly increased by 18% respectively compared to a fixed (30 Hz), or no vibration (Di Giminiani et al., 2009). But no significant increases in CMJ height were found after eight weeks in all three groups.

### *Long-Term Vibration (>2 months) – Lower Body Power*

Several studies have investigated the long-term effects of vibration on muscle power using VJ performance. The majority of these studies have found increases in VJ jump performance. Delecluse *et al*. (2003) randomised 37 untrained females (21 yrs) into groups of vibration exercise (*f*=35-40 Hz, *A*=2.5-5mm, VV [Power Plate]) or resistance training (8-20RM of knee extension and leg press) and found that after 12 weeks (3x/week) of training, VJ height increased by 7.6% from vibration exercise. Well-trained strength males (21-40yrs) who underwent five weeks (3x/week) of vibration (*f*=40 Hz, *A*=not given, VV [Nemes]) combined with 6RM squats reported an increase in VJ by 8.8%, but there was no significant difference between vibration and reistance training groups in relative jump height increase (Ronnestad, 2004). Furthermore, healthy non-athletic males and females increased VJ height by 9.0% and 7.7% following four and eight months of vibration exercise (*f*=25-40 Hz, *A*=2mm, VV [Kuntotary]) (Torvinen et al., 2002b; Torvinen et al., 2003). In addition, post-menopausal women have shown increases in VJ height of 19.4% and 4.7% from 24 weeks (3x/week) and six months (2x/week) of vibration training (Roelants et al., 2004a; Russo et al., 2003).

However, no significant improvements in VJ height have been reported from various vibration training protocols conducted over different time periods such as, 11 weeks (3x/week), (de Ruiter, van Raak, Schilperoort, Hollander, & de Haan, 2003b), nine days, (Cochrane, Legg, & Hooker, 2004) and five weeks (3x/week) (Delecluse et al., 2005).

In summary, there is strong evidence that acute vibration exercise can enhance upper and lower-body muscle power, and there is some indication that vibration exercise can enhance lower and upper-body muscle power over a longer-term, although this is less convincing. Future studies need to be conducted to determine the optimal duration of the rest between repetitions, the optimal frequency and duration necessary to maximise power in both short and long term studies.

#### *2.7.8.2 Force*

*Acute Vibration – Upper Body Force*

There are many methods of applying vibration to the upper-body; some researchers have used custom-built vibratory units applied directly to the muscle or attached to resistance training cables, while in recent times commercially manufactured vibrating dumbbells have also become available. In a study conducted by Kin-Isler and colleagues (2006), an electromotor was used to transmit vibrations through a cable attached to a leather belt that was placed over the belly of the biceps brachii. Using a range of vibration frequencies ($f$=6, 12, 24 Hz, $A$=4mm,) and joint angles (90°, 120°, 150°) the researchers reported that during a 10s vibration exposure, a 6.4% increase in MVC elbow flexors was observed. However, the length of muscle (conducted at various angles) did not affect isometric MVC. Finally, vibration has no effect on grip force when using an electric powered vibrating dumbbell (Cochrane & Hawke 2007) or when arms are exposed less proportionally to the legs on a vibrating platform (Cochrane & Stannard 2005).

*Acute Vibration – Lower Body Force*

There is little consensus on whether acute vibration increases lower-body force of isometric, isokinetic and isoinertial muscle actions. De Ruiter *et al*. (2003a) reported that when the knee extensors were electrically stimulated, the maximum force-generating capacity and isometric contraction significantly declined after an intermittent acute bout of vibration ($f$=30 Hz, $A$=8mm, SV, [Galileo 2000]). However, no changes in maximal isometric leg extensor strength were found following four minutes of vibration ($f$=25-40 Hz, $A$=2mm, VV [Kuntotary]; Torvinen et al., 2002c). Conversely, in a follow up experiment using the same design and experimental protocol, the researchers reported an increase of 3.2% leg in extensor strength (Torvinen et al., 2002a). The likely explanation for this finding is the type of vibration machine used and its parameters. It's possible that the SV elicited a greater response in leg extensor

strength compared to the VV machine; this has recently been confirmed by Abercromby *et al.* (2007b). However, other studies from Humphries *et al*. (2004), Kemertzis, Lythgo, Morgan, and Galea (2008), and Erskine *et al.* (2007) have indicated that acute vibration does not enhance isometric or isokinetic force and claim that inhibition rather than facilitation occurs. On the contrary, there have been reports that vibration does increase force attributes with Warman *et al*. (2002) reporting that direct vibration increased isotonic knee extensor force, but no changes were seen in isometric or isokinetic force. However, using a vibration platform of two and six minute exposure ($f$=26 Hz), Stewart *et al*. (2009), and Jacobs and Burns (2008) have reported increases in knee isokinetic peak torque of 3.8% and 7.7% respectively and Mileva *et al*. (2006) reported an increase in isoinertial peak torque by 25% and 12% using loads of 30% and 70% 1RM knee extension with a vibrating system ($f$=10 Hz, $A$=not given).

The discrepancy of the above findings could be due to the various protocols used for testing vibration. These include different methods of vibration, types and parameters of muscle contraction, and duration and frequency of vibration, as well as the muscle contraction velocities and variables.

### *Short-Term Vibration (<2 months) – Upper Body Force*

Using an electric motor to transmit eccentric oscillations ($f$=44 Hz, $A$=3mm) to a cable system a series of seated bench-pull repetitions were performed by male physical education students at 80-100% 1RM for three weeks (3x/week) while control groups performed the same resistance routine without vibration or performed calisthenics. The researchers found that by combining vibration with force the 1RM bench-pull significantly increased by 50% compared to a 16% improvement by the conventional resistance group, with no change being reported in the calisthenics group (Issurin & Tenenbaum 1994). Conversely, acute vibration has had no impact on potentiating grip force (Torvinen et al., 2002a; Torvinen et al., 2002c). This is not an unusual finding, as most of the vibration is indirectly applied through the feet on a vibration platform where the vibration may be attenuated by soft tissues, fluids, joints, muscles and bones that are capable of absorbing and damping vibration before reaching the upper extremities.

In a recent upper-body study by Silva, Couto, and Szmuchrowski *et al*. (2008), untrained participants (24yrs) were either assigned to isometric bicep training (12 MVC's, 6s in duration) without or with vibration ($f$= 8 Hz, $A$=6mm). The participants were seated with elbow flexed at 90°, and vibrations were produced by an amplifier connected to a steel cable with a hand grip that was applied in the opposite direction of muscle shortening. After four weeks of training (3 days/week) there was a significant increase of 26% bicep MVC from the group that received isometric and vibration compared to isometric alone (10% increase bicep MVC).

### *Short-Term Vibration (<2 months) – Lower Body Force*

Short-term vibration on lower-body force has produced mixed results with de Ruiter *et al*. (2003a) and Delecluse *et al*. (2005) reporting no increase in muscle force. De Ruiter *et al*. (2003a) reported that MVC and maximal force-generating capacity with and without muscle stimulation of knee extensors were not enhanced after 11 weeks (3x/week) of intermittent vibration (*f*= 30 Hz, *A*=8mm, SV [Galileo]). Delecluse *et al*. (2005) found that five weeks of vibration (*f*= 35-40 Hz, *A*=1.7-2.5mm, VV [Power Plate]) failed to increase isometric and dynamic knee extensor and flexor strength in well-trained sprinters. A closer examination of the de Ruiter *et al*. (2003b) study shows that the authors failed to periodise the 11 week vibration protocol by systematically and progressively increasing vibration frequency, amplitude, and load. Additionally, a two week break was enforced without explanation between week four and five.

Increases in force from vibration exercise have been reported from Mahieu *et al*. (2006) and Fagnani *et al*. (2006); both studies observed an increase in torque post-vibration. Mahieu *et al*. (2006) noted an increase in isokinetic torque of ankle plantar flexors of young skiers after six weeks (3x/week) of vibration training (*f*= 24-28 Hz, *A*=2-4mm, VV [FitVibe]). Fagnani *et al*. (2006) reported a 11.2% in isokinetic knee extensor in trained female athletes after eight weeks (3x/week) intermittent vibration protocol (*f*=35 Hz, *A*=4mm, VV [Nemes]). Both studies failed to compare vibration with the appropriate controls of performing the same activity with and without vibration. However, Ronnestad *et al*. (2004) compared 5 weeks (2-3x/week) of weighted squats (6-10RM) with vibration (*f*=40 Hz, *A*=not given) and without vibration. They reported a 32% increase in 1RM squat from vibration, but it wasn't significantly different from the 24% increase in 1RM squat without vibration. However, the small sample size (n=7) of each group, and the lack of description about the vibration amplitude and the cadence and depth of squatting performed on the vibrating plate may explain why no statistical differences were reported.

### *Long-Term Vibration (>2 months) – Lower Body Force*

Tovinen *et al*. (2002b; 2003) conducted two separate studies on the long-term effects of vibration (*f*=25-35 Hz, *A*=2mm, VV [Kuntotary]) performed over four and eight months in healthy young participants (19-38yrs). In the four month study the authors found that isometric knee extensor strength improved by 3.7% at two months compared to the control condition, but no further improvements were evident at four months. Likewise, after eight months vibration had produced no significant differences in isometric knee extensor strength. Therefore, the vibration stimulus of frequency, amplitude, and duration may have not been effective in eliciting the desired neuromuscular responses. Ideally, the additional load should have been included in the latter stages of an eight month programme, which could have been achieved by

progressively increasing body mass with external loads of a weighted vest or belt. Additionally, all of Torvenien's studies (2002a, 2002b, 2002c, 2003) have included an exercise routine of light squatting, light jumping and standing performed in addition, to the vibration stimulus; however, this routine has never been quantified in terms of load or cadence. Similarly, the dynamic nature of the protocol could have inhibited the vibratory stimulus to realise its full potential.

To overcome the shortcomings of Torvenien's studies (2002b; 2003), Delecluse *et al*. (2003) devised a 12 week (3x/week) periodised training plan, where vibration frequency, amplitude, exercise duration, load, sets and reps were progressively and systemically overloaded. Seventy-four untrained females were randomly allocated to: 1) vibration ($f$=35-40 Hz, $A$=2.5-5mm, $a$=2.3$g$-5.1$g$ VV [Power Plate]); 2) cardio-resistance training; 3) placebo ($f$=35-40 Hz, $A$=2.5mm, $a$=0.4$g$ VV, Power Plate); and 4) control. A 16% and 9% increase of isometric and dynamic knee force were observed in the vibration group, which was similar to the resistance training group (14% and 7% respectively), but significantly different to placebo and control groups.

Roelants *et al*. (2004b) reported that in older post-menopausal women (64yrs) isometric knee extensor strength increased by 15% and isokinetic strength by 16% from 24 weeks of vibration training ($f$=35-40 Hz, $A$=2.5-5mm, VV [Power Plate]), however, there was no significant difference in the respective increase found in the resistance training group. In a follow up study using untrained females, this research group (Roelants et al.,2004a) confirmed their earlier findings of isometric and isokinetic knee extensor force, but they also noted that the increases were not significantly different from those who did a combined cardio and leg-strength programme.

The same research group (Bogaerts et al., 2007a) conducted a year-long study on older men (67yrs) and repeated the same protocol, and observed that vibration increased muscle mass and isometric force but it was not significantly different from performing a combined cardio, strength, balance, and flexibility programme. Finally, Kvorning *et al*. (2006) reported that after nine weeks (2-3x/week) of weighted squats with vibration ($f$=20-25 Hz, $A$=4mm, SV [Galileo 2000]) isometric leg press strength produced an increase of 9.3% which was comparable to the 12% increase in leg strength from weighted squats without vibration. Therefore, combining vibration with squats was no more beneficial than weighted squatting alone.

In summary, it is not conclusive whether acute vibration increases force attributes. This has been fraught by the type and parameters used for various muscle contractions, and the different sample populations that have varied in chronological age, experience and training status. Furthermore, the debate surrounding the length-tension proposal that muscle must be lengthened in order to benefit from vibration has caused confusion. However, recent evidence

suggests that when vibration was applied at 120° knee flexion, which maximises the limb's greatest mechanical advantage, no increases in force were evident. Moreover, vibration applied to concentrically active muscle has shown to improve muscle force, which cannot explain the length-tension relationship. There may also be an optimal contraction velocity where vibration is most effective, and testing the effect of vibration on self-selected isokinetic contraction velocities warrants further investigation. Moreover, the impact of vibration on the different types of muscle contraction modes of isometric, isokinetic, and isoinertial remains unclear in both the upper and lower-body.

Long-term studies show that vibration exercise improves force parameters, but provides no further augmentation than conventional resistance and cardiovascular programmes; therefore, it may lend itself to initially helping people that cannot undertake conventional resistance and aerobic programmes.

#### *2.7.8.3 Sprinting Speed and Agility*

##### *Sprinting Speed*

Evidence suggests that power and force variables are potentiated from acute vibration, where the proposed mechanisms are most likely neural, as vibration exercise is thought to produce an effect similar to that of explosive power training (Bosco et al., 1999a; Bosco et al., 1998; Bosco et al., 1999c; Bosco et al., 2000). Furthermore, increased motoneuron excitability is regarded as integral to enhancing sprint performance (Ross, Leveritt, & Riek, 2001) and is common to both vibration exercise and power/strength training. Sprinting requires a large force production in a short period of time (Mero, Komi, & Gregor, 1992) and it is likely that power and resistance training may contribute to developing sprint performance (Delecluse, 1997). Therefore, it is reasonable to suggest that vibration exercise may be a favourable method for enhancing sprint performance.

*Acute Vibration*

Recently Bullock *et al*. (2008; 2009) investigated the effect of vibration exercise on 30m sprint performance in elite skeleton athletes. However, a higher frequency (*f*=45Hz vs. *f*=30Hz), shorter recovery (180s vs. 60s) and a greater rest period (5min vs. 10min) were implemented in the later study. Despite these differences, neither study reported an improvement in 30m sprint performance compared to vibration or control (no vibration). Both studies employed an extensive 30 minute specific warm-up, which may have masked possible potentiation effects. Moreover, the studies contained small sample sizes of five and seven athletes respectively, which may have increased the likelihood of a type II error. Furthermore, because these elite athletes were highly trained, the dose-response of the vibration parameters of frequency, amplitude and duration may not have been at an appropriate level to

elicit the desired responses of the neuromuscular system. Finally, these athletes had well developed musculotendinous apparatus that can resist high impact loads (Mero et al., 1992), which can damp vibration and minimise changes in muscle length (Mester et al., 1999).

*Short Term (<2 months) Vibration*

Cochrane *et al*. (2004) investigated the effect of nine days of vibration exercise (*f*= 26 Hz, *A*= 11mm, SV [Galileo 2000]) on sprint (5, 10, 20m) performance in healthy young (23.9 yrs) participants, and found no significant changes. Likewise, Delecluse *et al*. (2005) found no significant differences in 30 m sprint performance between conventional training (strength, speed plyometric sessions) and vibration training (*f*=35-40 Hz, *A*=1.7-2.5mm, VV [Power Plate]).

To the contrary, Paradisis and Zacharogiannis (2007) reported improvements in sprint times at 10, 20, 40, 50, and 60m (2.1% to 4.3%) with significant increases in step rate (5.1%) and step length (3.4%) after six weeks (3x/week) of vibration exercise (*f*=30 Hz, *A*=2.5mm, VV [Power Plate]). The discrepancy between this study and the findings of Cochrane *et al*. (2004) and Delecluse *et al*., (2005) is probably due to the different protocols used for exposure time, vibration frequency and training volume. However, Paradisis and Zacharogiannis (2007) failed to implement a reasonable control. In comparison Cochrane *et al*. (2004) and Delecluse *et al*. (2005) had control groups that performed exactly the same exercise routines as the vibration group but with no vibration.

### *Agility*

Agility is an important component of many sporting activities, especially in team sports where changes in direction with abrupt deceleration and cutting are important in making attacking and defensive plays. Agility, as measured by the 505 up and back test, showed no statistical improvements after nine vibration training sessions in healthy active participants (Cochrane et al., 2004). Likewise, no improvement in agility performance has been documented when performing a shuttle run from four minutes (Torvinen et al., 2002a) or four months of vibration (Torvinen et al., 2002b). Many factors influence agility performance, such as limb length, flexibility, stride length and limb strength. Therefore, the interaction of all the perceptual and technical agility factors makes it difficult to identify the components that influence agility performance.

In summary, vibration exercise has little impact on sprint performance. The likely explanation for this finding is that sprinting generates high forces of repeated movement, which may be more closely related to the velocity component of the force-velocity relationship (Bullock et al., 2009). In contrast, power and force tasks rely on a one-off maximum effort; therefore, vibration exercise may have a greater effect on the force parameter of the force-velocity relationship.

There is also the issue of the muscle-tendon complex; well-trained athletes have the ability to produce a stiffer muscle-tendon complex (Kubo, Kawakami, Kanehisa, & Fukunaga, 2002) that is related to sprinting speed (Ross et al., 2001) where a higher rate of force development of stiffer muscle-tendon complex increases the ability to transmit force quickly (Wilson, Murphy, & Pryor, 1994). However, in elite sprint athletes the increase of stiffness is predominantly due to pre-contracted muscle. Also, there are reports that well-trained athletes have compliant tendons that may act to damp the vibration waves, which protects the muscle from further stimulus.

#### *2.7.8.4 Flexibility*

Flexibility is defined as the range of motion (ROM) about a joint or related series of joints (Alter, 1996) and has importance for sports that require large ranges of motion such as gymnastics, diving, synchronised swimming and martial arts. Various manual stretching methods such as static, ballistic and proprioceptive neuromuscular facilitation (PNF) have been used to maintain or increase ROM (Bandy & Irion, 1994; Ross, 1999; Shellock & Prentice, 1985), which probably involves neurophysical mechanisms and stretch reflexes. One proposed mechanism of vibration exercise is based on spinal reflexes; however, changes in blood flow could create heat, which may, in turn, enhance the extensibility of the muscle and enhance ROM.

##### *Acute Vibration*

Various vibration machines have been used to study the effects on flexibility. Atha and Wheatley (1976) used mains powered vibration cushions ($f$=44 Hz, $A$=0.1mm) placed on the hamstrings and lower back. The authors observed that vibration increased hip flexion mobility (~8.5%) by the same amount as stretching alone. Custom built vibratory units have been used to elicit specific flexibility positions. Sands *et al*. (2008b; 2006) found significant increases in forward split position by combining vibration ($f$=30 Hz, $A$=2mm) and passive stretching in young male gymnasts (10yrs) and elite adult female synchronised swimmers (21yrs). However, the gymnastics study had only five males in each group, which could have increased the possibility of a type I error. Likewise, Cronin *et al*. (2007) used a specifically designed vibratory unit and observed that 30s of 44 Hz ($A$=5mm) significantly increased dynamic hip ROM (2.1%) in competitive club athletes but no significant increases in ROM measurements for 24 Hz and 34 Hz ($A$=3mm) were noted. It is unclear from the methods what specific instructions were given on positioning the leg on the vibrating apparatus, as this may have influenced how much force and stretching was applied by the leg during the different vibration frequencies.

It has been documented that static stretching alone may have a deleterious effect on muscle strength and power attributes (Avela, Kyrolainen, & Komi, 1999; Church, Wiggins, Moode, & Crist, 2001; Stone et al., 2006; Young & Behm, 2003). Conversely, flexibility and explosive power have been shown to be enhanced by vibration (Sands et al., 2006). In sports where flexibility and power are equally important, stretching may cause decrements in power however, combining vibration and stretching may provide a method for increasing flexibility without sacrificing power. Kinser *et al*. (2008) tested this claim and found that forward split flexibility in very young (11.3yrs) competitive female gymnasts was significantly enhanced by combining stretching with vibration ($f$=30 Hz, $A$=2mm) that did not alter explosive power variables. The authors postulated that the increase in flexibility from vibration and stretching was, in part, due to a decrease in musculotendinous stiffness, muscle antagonist inhibition and increased pain threshold. Because there were no changes to explosive power the researchers suggested that the addition of vibration enhances a neurogenic potentiation, but does not alter explosive power, which normally declines with static stretching alone.

Contrary to Kinser *et al*. (2008) Cronin and associates (2008) found that passive hamstring stretching with vibration ($f$=34 Hz, $A$=3mm) did not improve hip flexion ROM, but stretching alone increased hip flexion ROM by a small amount (2.1%). The dissonance is likely due to the duration of the vibration exposure, the participants (healthy males vs. young female competitive gymnasts) and different methods used to assess flexibility (passive vs. dynamic). Additionally, the vibratory units were of a different make and the stretch was held to a 'comfortable sensation' (Cronin's et al., 2008), compared to an intensity of 'discomfort' (Kinser et al., 2008).

To date, only a few studies have explored the effects of vibration plates on flexibility. Using a series of static exercises (no stretching) performed on a SV platform (Galileo) elite female field hockey players significantly increased their sit and reach flexibility score by 8.2% when vibrated for five minutes ($f$=26 Hz, $A$=6mm) compared to a 5.3% increase from no vibration and seated cycling at 50W (Cochrane & Stannard, 2005). In a similar experimental design, 20 recreationally active male and females showed a significant increase 16.2% from six minutes continuous vibration ($f$=26 Hz, $A$=not given, SV [Galileo 2000]) compared to a 2.6% increase in 50W of upright stationary cycling (Jacobs & Burns 2009). The difference in sit and reach results between the two studies is likely due to the recreation participants making greater gains compared to elite athletes who frequently use stretching routines. Likewise, Sands *et al.* (2008a) reported a significant increase (27.5%) in flexibility of the split position compared to 13.7% increase of no vibration in young male (10.7yrs) gymnasts that underwent 45s of vibration ($f$=30 Hz, $A$=2mm, VV [Power Plate]). Using a SV platform Kemertzis *et al*. (2008) reported a longer muscle length when the plantar flexors were passively stretched with vibration ($f$=26 Hz, $A$=4-4.5mm) compared to no vibration. Again, the differences in vibration methodology may explain the discrepancies in the increases in flexibility gains.

### *Short-Term (< 2 months) Vibration*

Issurin *et al*. (1994) was one of the first to explore the effects of vibration on flexibility over a short term period. Using a custom built vibrating cable system young male (19-25yrs) physical education students stood on one leg while the other leg was placed in a hanging ring. Whilst holding onto an upright bar, each student performed a stretching routine of 3-4 sets of static stretching (6-7s) and one set of ballistic stretching (10-30s) with vibration ($f$=44 Hz, $A$=3mm) for three weeks (3x/week). Findings showed a significant increase in leg splits (8.7%), and trunk flexion (43.6%) compared to conventional flexibility training (2.4%, 19.2%) and control (no flexibility, 1.2% and 5.8%) respectively. The researchers suggested that the increases in flexibility from vibration may be due to several mechanisms, such as an increase in pain threshold, increase in blood flow and muscle temperature.

Similarly, van den Tillaar *et al*. (2006) found that after three weeks of vibration ($f$=28 Hz, $A$=10mm, VV [Nemes]) hamstring flexibility ROM was enhanced by 30% compared to the control group that only performed a stretching routine (14%). The authors postulated that the superior result of vibration training over the conventional flexibility training was a result of: 1) increased blood flow, which generated heat to enhance muscle elasticity; 2) antagonist inhibition that increased activation of quadriceps and relaxed the hamstrings; and 3) an increase in proprioceptive feedback that inhibited pain. In a longer study conducted over eight weeks (3x/week), vibration exercise ($f$=35 Hz, $A$=4mm VV [Nemes]) has shown to increase sit and reach flexibility by 13% compared to a control group (Fagnani et al., 2006)

In summary, acute or short-term vibration exercise improves flexibility measures in young, sporting and healthy people. This suggests that muscle is influenced by vibration. Whether it is an increase in blood flow, muscle temperature or pain threshold, the mechanism of explaining flexibility enhancement requires further investigation, as does the effect of vibration exercise on flexibility in the elderly.

## 3. Summary

There is strong evidence that vibration exercise acts on muscle to enhance force, power, and flexibility, and improves balance and proprioception; however, its effect on the cardiovascular system is small. A number of studies have outlined various protocols for testing vibration exercise. These include different methods of application, vibration parameters, training duration and different populations, as well as various exercises performed with vibration. Further, different types of commercially manufactured platforms have been used, such as side-alternating vibration (SV), where unilateral vibration is applied alternately to the left and right foot, compared to vertical vibration (VV) platform, which produces simultaneous and symmetrical vibration to both sides of the body.

Recent evidence suggests that during dynamic and static squatting SV produces greater activation of the lower limb muscles than VV (Abercromby et al., 2007a), with a greater amount of vibration (71 to 189%) being transmitted to the upper-body and head during VV compared to SV (Abercromby et al., 2007b). Additionally, hand-held powered vibrating dumbbells have been commercially manufactured for exercising the upper-body, and portable vibratory units have been attached to barbells and cables of resistance training machines, both of which have made it difficult to compare studies. Confounding this problem is the lack of prescriptive guidelines for vibration. The optimal vibration frequency, amplitude and the type of exercises performed on the vibrating platforms have not been sufficiently validated. Furthermore, there is a lack of evidence on the appropriate way to progressively increase the vibration parameters and to match the vibration volume and intensity to other modalities, such as resistance and power training. Additionally, sample size, statistical power, and blinding of participants and investigators have been poorly documented. The comparison of protocols and study designs leaves the magnitude of adaptations and the mechanism of improvements in these studies uncertain.

It appears that the research of vibration exercise is still in its infancy and requires further investigation. For instance, no research has been conducted on the effect of vibration exercise on muscle or core temperature, and whether vibration is an appropriate modality for warming-up. Moreover, the mechanism of vibration exercise has been based on spinal reflexes and neural aspects; however, there has been little supporting evidence to substantiate these claims because testing H-reflex and stretch-reflex with vibration exercise have produced equivocal results. Further, it is unknown whether vibration exercise causes muscle to ‘wobble’ or to contract by causing changes in muscle length. In the muscle performance area, several studies have reported vibration exercise augments CMJ, which is an explosive eccentric-concentric muscle action. But little research has been conducted on the effect of vibration exercise on explosive concentric muscle performance and how it compares other concentric modalities.

It has been documented that vibration exercise causes mild cardiovascular changes in young people, but, it is unknown what effect vibration has on cardiovascular function of the older population.

From the current research presented in this review, there is a lack of uniformity in vibration protocol, training dose and reported effects of vibration on muscle. Therefore, more robust studies are required to obtain conclusive evidence of the beneficial effects that vibration exercise has on muscle physiology and performance.

## 4. Hypotheses

The hypotheses are postulated from a series of studies; it is anticipated that vibration exercise will enhance muscle function through raising muscle temperature, which will potentiate muscle twitch characteristics to a greater extent that conventional modalities. During vibration it is envisaged that muscle will lengthen, with the muscle-tendon complex increasing, and a temporal relationship will coexist between muscle activity and muscle lengthening. Specifically it was hypothesised that:

***Hypothesis 1*** Post-vibration exercise peak power and EMG activity will be significantly potentiated, compared to either arm cranking or control (no vibration exercise) interventions (**Chapter 2**).

***Hypothesis 2*** The purported increase in muscular power is likely to be partly muscle temperature dependent, but acute vibration exercise will elicit a greater increase in peak power output compared to stationary cycling and passive warm-up techniques that is consequent upon neurogenic or other potentiation (**Chapter 3**).

***Hypothesis 3*** When shallow dynamic and static squatting are matched for metabolic rate, there will be no significant difference in raising muscle temperature between the two types of squats during acute vibration exercise. Secondly, when dynamic squat is superimposed upon vibration exercise, muscle temperature will exceed both dynamic squat alone and static squat with vibration (**Chapter 4**).

***Hypothesis 4*** There is reason to assume that the stimulus for muscular work of vibration exercise, which occurs either mechanically direct, or via the stretch-reflex, will be reduced, and the metabolic requirement will be decreased in older individuals. Therefore, it was hypothesised that the increase in oxygen uptake evoked by vibration exercise will be lower in older people than in the young. Secondly the metabolic rate in young and older participants will increase when the Jendrassik manoeuvre is superimposed with vibration exercise and load (**Chapter 5**).

***Hypothesis 5*** Acute vibration exercise will significantly increase EMG activity concurrently with measurable changes in muscle contractile length and muscle-tendon complex compared to resting levels (**Chapter 6**).

***Hypothesis 6*** Acute vibration exercise will result in a greater muscle twitch potentiation compared to the vibration induced patellar stretch reflex (**Chapter 7**).

## 5. Aims

Broadly, the aim of this thesis is to investigate the effect of acute vibration exercise for enhancing human performance and to examine the physiological aspects. More precisely, to test the hypotheses indicated, the aims of this study are:

***Aim 1*** Compare acute vibration exercise with a concentric exercise on upper-body power output and muscle activity (**Chapter 2**).

***Aim 2*** Compare *vastus lateralis* muscle temperature change in acute vibration exercise concentric exercise, and passive warm-up (**Chapter 3**).

***Aim 3*** Compare *vastus lateralis* muscle temperature change of shallow dynamic squat and static squat with concurrent acute vibration exercise (**Chapter 4**).

***Aim 4*** Examine the acute physiological effects of acute vibration exercise in young and older people (**Chapter 5**).

***Aim 5*** Qualitatively investigate whether acute vibration exercise causes changes in muscle lengthening and muscle-tendon complex (**Chapter 6**).

***Aim 6*** Examine whether a temporal relationship exists between muscle activity and muscle lengthening from acute vibration exercise (**Chapter 6**).

***Aim 7*** Examine whether muscle twitch properties and patellar reflex aspects are simultaneously potentiated from acute vibration exercise compared to concentric exercise (**Chapter 7**).

## 6. Prelude

The above aims are addressed by conducting a series of studies. Firstly, it has been reported that lower limb acute vibration exercise increases vertical jump performance. As vertical jumping relies on the stretch-shortening cycle, it has been postulated that an increase in vertical jump height may be due to the eccentric stimuli provided by the vibration exercise. However, it is unknown what effect acute vibration exercise will have on muscle action that is solely concentric. Thus, **Chapter 2** investigates to what extent acute vibration exercise affects concentric contractility and neuromuscular activity when it is compared to other concentric modalities.

Based on the observations that vertical jump, flexibility, and blood flow is augmented after acute vibration, some authors have advocated that vibration exercise may be used as a warm-up modality, where muscle temperature may have an effect on producing post-activation potentiation which is part of the warm-up effect. However, muscle temperature has never been directly measured during vibration, nor has it been confirmed if vibration potentiates muscle power more than conventional warm-up procedures. Therefore, **Chapter 3** compares muscle temperature changes and muscle power output in acute vibration exercise, stationary cycling, and passive warm-up (hot-water tub). Further to this, dynamic and static squatting are exercises commonly performed on vibrating platforms; but the effect of shallow dynamic versus static squatting with concurrent vibration exercise on muscle temperature and cardiovascular indices are unknown, which is addressed in **Chapter 4**.

Exercise is generally advocated as a countermeasure to offset age-related frailty and to enhance mobility and well-being. Whilst the fact of physiologic decline may not be avoided, it can be mitigated by exercise; however, the physiological effects of acute vibration on older adults are unknown. Therefore, **Chapter 5** investigates cardiovascular and metabolic aspects of young and older adults exposed to different loads of acute vibration exercise, and to provided some insight to whether vibration exercise elicits muscle contractions via the stretch reflex the Jendrassik manoeuvre is assessed to determine age-related differences from vibration exercise-related oxygen uptake.

The last chapters (6-7) continue the work of Chapter 5, which investigates the possible mechanism(s) of vibration exercise. It has been purported that the physiological mechanism of vibration occurs via spinal reflexes, which elicits small changes in muscle length; however, there is no direct evidence that this actually occurs during vibration exercise. Therefore, **Chapter 6** examines whether acute vibration exercise results in muscle lengthening and coincides with a temporal change in muscle activity. However, to directly test for the presence of spinal and muscle potentiation, patellar tendon reflex and muscle twitch properties are simultaneously assessed in **Chapter 7**, which may provide additional information about its

effect to elicit post-activation potentiation in either or both neural or myogenic aspects. Finally, **Chapter 8** discusses and concludes the most important findings of each chapter, and their relationship to each other. The overview of the thesis chapters and their relationship are shown in figure 21.

As previously stated this is a European style thesis where a cohort of studies were undertaken following a review of the literature which identified biological knowledge on which proposed hypotheses were to be tested and culminates in a collection of published papers. Based on this format and given that the vibration equipment was central to the publications (chapters 2-7) there is some repetition and duplication in the methodology sections of the various chapters but this minimal as different machines of vibration exercise were used in the thesis. Moreover, the sample populations recruited for this thesis were either, young adults, active older adults or national competitive athletes, therefore differences exist in chronological age, training experience and fitness status for the various studies conducted. The measurement of muscle temperature was confined to the *vastus lateralis* and the analysis of kinematic data, contractile tissue displacement, muscle activation, and spinal reflexes were restricted to the lower-body, which was proximal to the source of vibration. Finally, some peer-reviewed journals prefer to use the term whole-body vibration rather than vibration exercise therefore, the published papers from this thesis feature both terms (Appendix 9) however, for consistency and to avoid confusion the term vibration exercise has been adopted because it provides a better description of the modality (see the section on the Definition of Vibration Exercise).

**Figure 21** Overview of the thesis chapters and their relationship

# Chapter 2 – The Effect of Acute Upper-Body Vibration Exercise on Concentric Muscular Performance

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## 2.1 Abstract

This study was designed to compare the acute effect of vibration exercise with a concentric-only activity (arm cranking) on concentric-only muscle action using an upper body isoinertial exercise. Twelve healthy, physically active men, 30.0 ± 6.1 years (mean ± SD); height 1.81 ± 0.06 m; and weight 83.4 ± 9.7 kg performed four maximal prone bench pull (PBP) efforts before and after a 5 minute period of three different interventions: 1) with vibration exercise (VBX+); 2) arm cranking (AC); and 3) without vibration exercise (VBX−). Electromyography (EMG) activity was assessed from the middle trapezius muscle during PBP. VBX+ was induced with an electric-powered dumbbell (DB) ($f$= 26 Hz, $A$= 3 mm), with 30-sec exposures at five different shoulder positions. VXB was performed with the participants holding the DB with the machine turned off, and AC was performed at 25W. There was a significant (intervention x pre-post) interaction such that acute VBX and AC enhanced peak power by 4.8% ($p<0.001$) and 3.0% ($p<0.001$) respectively compared to VBX− (-2.7%). However, there was no effect of any treatments on EMG activity compared to the control. In conclusion, acute VBX+ provides an acute ergogenic effect which potentiates concentric-only muscle performance, though not to a greater extent than concentric (arm-cranking) exercise.

*Keywords* Muscle, strength, warm-up, peak power, EMG, shoulder rehabilitation

## 2.2 Introduction

Extensive or constant exposure to high frequency vibration is classified as a hazardous activity that can be detrimental to health (Griffin, 2004). However, acute low frequency vibration exercise (VBX) is commanding attention in a number of disciplines in assisting physical rehabilitation, sports conditioning, and compromised health, although its efficacy remains unclear. Nevertheless, acute VBX has been shown to be effective for improving muscle strength (Roelants et al., 2004), muscle power (Cochrane & Stannard, 2005), and balance (Runge et al. 2000).

Until recently the production of acute vibration was confined to custom made electromotor devices that were directly applied to the muscle (Warman et al., 2002). However, commercial companies are now manufacturing machines that vibrate the whole body via an oscillating platform (Galileo Sport®, PowerPlate®, VibroGym®) or by a perturbating dumbbell (Galileo TOP®). The oscillatory movements of these devices are thought to cause rapidly repeating eccentric-concentric muscle work that elevate metabolic rate of the muscles that are activated by the vibration (Rittweger et al., 2000). Although as yet there is no consensus on the ergogenic mechanism of VBX, it is thought that the vibration causes a neurogenic and muscle response known as tonic vibration reflex, which activates the muscle spindles thereby enhancing the excitatory drive reflex of the alpha motoneurons (Cardinale & Bosco, 2003; Rittweger et al., 2001). Additionally it is hypothesised that the joint and skin sensory receptors and secondary endings of the muscle are heightened from the sensitivity of the primary afferent endings by the gamma motoneuron system (Cardinale & Bosco, 2003) , which further promotes the sensitivity of the stretch reflex, thereby generating a more forceful muscular contraction (Cardinale & Bosco, 2003; Roelants et al., 2004). Moreover, the peripheral system may also play a role in acute VBX with erythema and increase blood flow being reported post-vibration (Kerschan-Schindl et al., 2001; Rittweger et al., 2000).

To date, lower limb acute VBX studies have reported increases in vertical jump performance (Bosco et al., 2000; Cochrane & Stannard, 2005) with equivocal findings from isometric leg force (de Ruiter et al., 2003; Humphries et al., 2004; Rittweger et al., 2003; Torvinen et al., 2002a). As vertical jumping relies on the stretch shortening cycle it has previously been postulated that the increase in vertical jump height may be due to the eccentric stimuli provided by the VBX (Cochrane & Stannard, 2005). However, it is unknown what effect acute VBX will have on muscle action that is solely concentric.

There are few studies on the acute effects of VBX on upper limb concentric muscle contractile properties. The work of Issurin *et al.* (1999) and Bosco *et al.* (1999a) have shown that acute VBX significantly enhances concentric strength and power attributes. However, neither study directly compared the effects of vibration on concentric activity with other concentric

modalities. Thus, the effect of VBX on concentric movement remains largely untested. Nor is it known to what extent acute VBX affects concentric contractility and neuromuscular activity. Therefore, if the proposed mechanism of neuromuscular excitability does in fact exist, electromyography (EMG) should increase as concentric peak power increases.

The aim of this study was to compare the acute effect of VBX with arm cranking (a solely concentric activity) on an upper body (prone bench pull) isoinertial (concentric only) exercise. To better understand the mechanism by which VBX may exert a neural effect, EMG activity during the prone bench pull was analysed. As VBX is suggested to potentiate the neuromuscular response, it was hypothesised that post-VBX peak power and EMG activity would be potentiated, compared to either arm cranking or control (without VBX) interventions.

## 2.3 Methods

### *Participants*

Twelve healthy, active males aged 30.0 ± 6.1 years (mean ± standard deviation); height 1.81± 0.06 m; and weight 83.4 ± 9.7kg, who had at least 2 years resistance training, trained 1-2x/week using weights and were familiar with the prone bench pull (PBP) volunteered to participate in the study. The protocol was approved by the University Ethics Committee and written consent was obtained from each participant.

### *Study Design*

A prone bench pull (PBP) at a load of 25% of body weight (BW) was performed before a five minute period of one of three different interventions.

- With Vibration Exercise (VBX+)
- Without Vibration Exercise (VBX−)
- Arm Cranking (AC)

Within 15s of completing the 5 minute intervention, the participants were re-tested on the PBP. Warm-up was prohibited prior to the interventions, to reduce the possibility of influencing the outcome of the study.

The order in which each participant undertook the three interventions was allocated in a randomised, balanced design, with at least 24 hours recovery between each intervention session. Prior to the study, each participant was familiarised with all the equipment and the PBP test. During the course of the study, participants were not permitted to undertake any power and strength training. The participants were instructed to strictly refrain from undertaking any vigorous activity 24 hours prior to the interventions. To prevent variations of daily biorhythm every participant performed all three interventions at the same time of day.

## Testing procedures

### *Prone Bench Pull (PBP)*

The PBP was conducted on a Smith machine (Fitness Works, Auckland, New Zealand) that consisted of an integrated barbell (BB) connected to two steel rods, which moved the BB upwards and downwards in the vertical plane by a chain, counter-weight and dual cog system (Figure 1). For this study, a 10-bit potentiometer (Model 533, Vishay, Malvern, Pennsylvania) was fixed to the upper cog of the Smith machine to measure the displacement of the BB to an accuracy of 1mm (Figure 2). This was interfaced with a computer (Apple Macintosh G4) and acquisition system (Powerlab, 8/30, and Chart v4, ADInstruments, Australia). By moving the BB through its full vertical range of 1.63 m, the voltage signal from the potentiometer was converted into a unit of displacement by the acquisition system. A low pass filter at a cut-off frequency of 5 Hz was used for the displacement-time data. The velocity of the bar was calculated by differentiating the displacement data. A second order derivative of displacement data was used to calculate acceleration of the system. The net force was calculated by the product of [mass of bar + load (25% BW)] multiplied by (bar acceleration + gravitational acceleration) subtracted by (mass of counter-weight x gravitational acceleration). Power was calculated from the product of net force and velocity obtained from the previous calculations and the peak power was determined as the highest power output over the pull phase.

A high bench (WP 130, Fitness Works, Auckland, New Zealand) was placed over the BB and bolted to the floor to prevent movement. The participants were instructed to lie prone, and place their chin on the padded edge of the bench (Figure 1). The pulling phase began with both elbows being in full extension while the BB was grasped with hands shoulder width apart. The participants were instructed to pull the BB with maximum effort until it struck the underside of the bench, after which the BB was lowered to the starting position. Every participant performed four maximal lifts, both pre- and post-intervention, with each lift separated by 15s of rest. The peak power (PP) of the four lifts were averaged for both pre- and post-intervention and used for statistical analysis.


**Figure 1** Prone bench pull machine

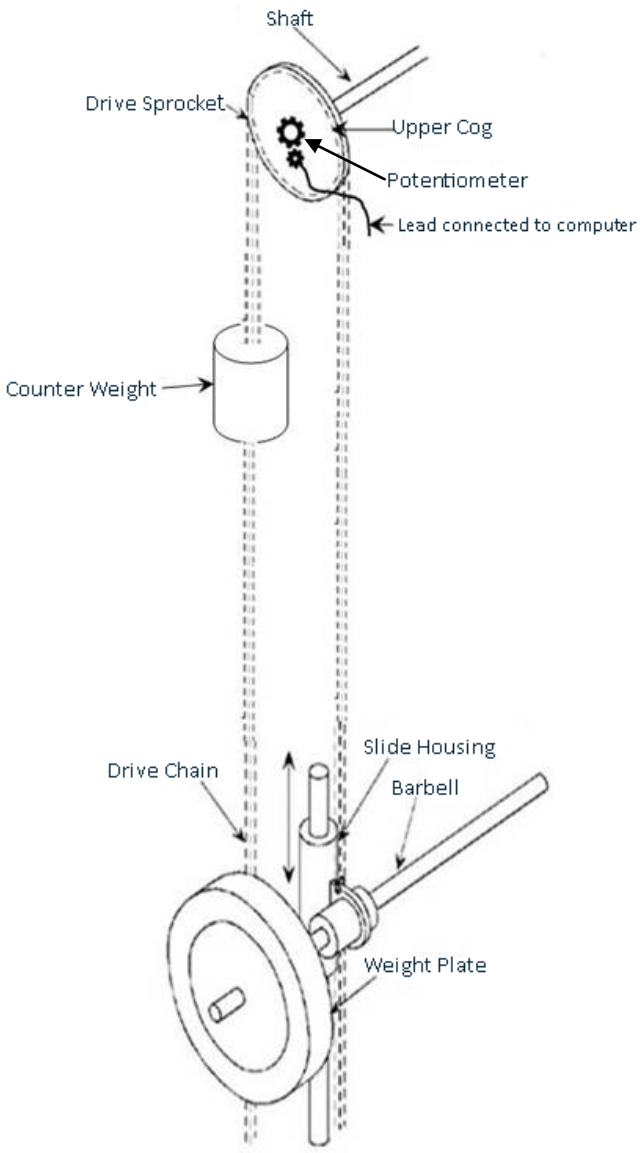



**Figure 2** Illustration of the potentiometer fixed to the upper cog of the Smith machine

*Electromyograph (EMG) data analysis*

Electromyography (EMG) and BB displacement data were analysed by an acquisition and software system (Powerlab, 8/30, and Chart v4, ADInstruments, Australia) that sampled at 1000 Hz. The PBP is a common multi-joint upper body exercise that involves numerous muscles, such as the middle trapezius, latissimus dorsi, and biceps brachii. However, the middle trapezius is the main muscle activated with other muscles assisting the movement therefore EMG activity was assessed from the right middle trapezius muscle. Prior to electrode placement the skin was shaved, gently abraded using a gel-based product (Nuprep, D.O. Weaver, USA), and cleansed with an alcohol tissue pad. The site of the electrode location was marked with a permanent pen to ensure identical placement for subsequent testing. Two surface pre-gelled Ag-AgCl electrodes, 10 mm diameter (Medicostest, Rugmarken, Denmark) were placed over the muscle belly at an inter-electrode distance of 20mm with a reference electrode located on the acromion process. The EMG signal was filtered using band-pass (10-200 Hz) and notch (60 Hz) filters performed in double-pass fashion. To prevent artefact, the EMG cables were secured by surgical tape to the torso of the participant. The EMG root-mean-square (rms) was analysed over a 300 ms period, with the $EMG_{rms}$ of the four lifts being averaged for both pre- and post-intervention and used for statistical analysis.

## Interventions

***1. VBX+***: This was performed by a Galileo TOP® (Novotec, Pforzheim, Germany) device, which produced vibration to shoulders and arms from an electric-powered dumbbell (DB), weighing 2.5kg. The central handle piece rotates and produces oscillatory movements to the body of varying frequencies (0 to 30 Hz) with amplitude of 3mm around a horizontal axis (Figure 3). A vibration frequency of 26 Hz was selected for the current study as previous research has found that low vibration frequency elicits the greatest EMG response from a given muscle group (Cardinale & Lim, 2003). Lying prone on the bench, each participant held the DB for 60s exposures at five different shoulder positions (Figure 4)**,** with neutral (0º) position defined when the hand and forearm were alongside the ipsilateral leg.


**Figure 3** Vibrating dumbbell

The five shoulder positions were selected to ensure that the back musculature was adequately exposed to the vibration, the five positions were;

(i) Shoulder abducted to 45º

(ii) Shoulder abducted to 90º

(iii) Shoulder abducted to 135º

(iv) Shoulder abducted to 180º

(v) Shoulder forward flexion to 90º (sagittal plane)

For each shoulder position the DB was alternatively held in the right and left hand for 30s with an interval of < ls separating the changing of the DB. The alternating of the DB provided a 30s rest period for the contralateral hand prior to the start of a new shoulder position. Moreover, the duration of the vibration exposure used in the current study is also in agreement with other vibration protocols that have found a post-vibration neuromuscular enhancement (Cochrane & Stannard, 2005).

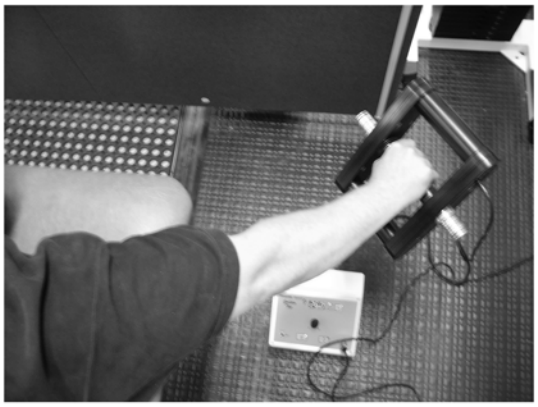
***2. VBX–***: The control intervention was performed on the Galileo TOP® machine (*f*=0 Hz, *A*= 0mm) in the exact five shoulder positions and time constructs as performed in VBX+, but with the machine switched off.

***3. AC***: Using a fractioned-braked arm-crank ergometer (Monark 881e, Varberg, Sweden) each participant cranked for 5 mins lying in the prone position on the bench. The crank was set at a 40 deg angle and a cadence of 25 revs, which equated to a workload of 25W.

### *Statistical Analysis*

All statistical analyses were performed using a specialized statistical software package (SPSS for Windows Version 14). Dependant variables were compared using a three-way (intervention x pre-post x lifts), repeated-measures analysis of variance (ANOVA). Each test data measure (4 lifts for PP and $EMG_{rms}$) was included as part of the ANOVA analysis. Pair-wise comparison between means was performed using post-hoc contrasts to identify intervention difference. Intraclass correlation coefficients (ICC) assessed the intra-day reliability of the four pre-intervention PBP's of PP and EMG rms. Significance was set at the 95% level of confidence ($p < 0.05$).

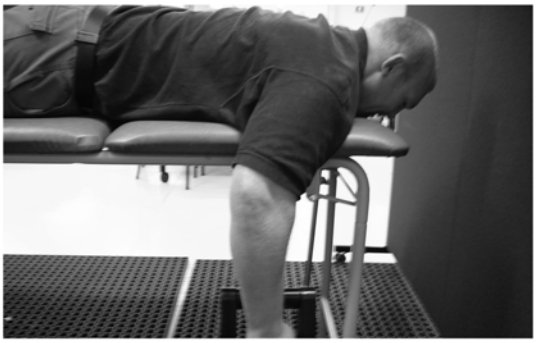
(i) Shoulder flexed to 180 degrees



(ii) Shoulder abducted to 135 degrees

(iii) Shoulder abducted to 90 degrees

(iv) Shoulder abducted to 45 degrees



(v) Shoulder horizontally adducted

**Figure 4** The five shoulder positions used with vibrating dumbbell

## 2.4 Results

There was a significant ($p<0.001$) (intervention x pre-post) interaction (Figure 5) such that VBX+ and AC enhanced PP by 4.8% and 3.0% respectively compared to a non-significant reduction (-2.7%) after VBX−. There was no difference between the increase in peak power after VBX+ and AC. There was no significant effect of VBX+, VBX− and AC on $EMG_{rms}$ (Table 1). The ICC test-retest reliability of PP ($r=0.950$) and $EMG_{rms}$ ($r=0.830$) was highly significant ($p<0.001$), indicating little variability and thus a high degree of consistency attained between testing sessions.

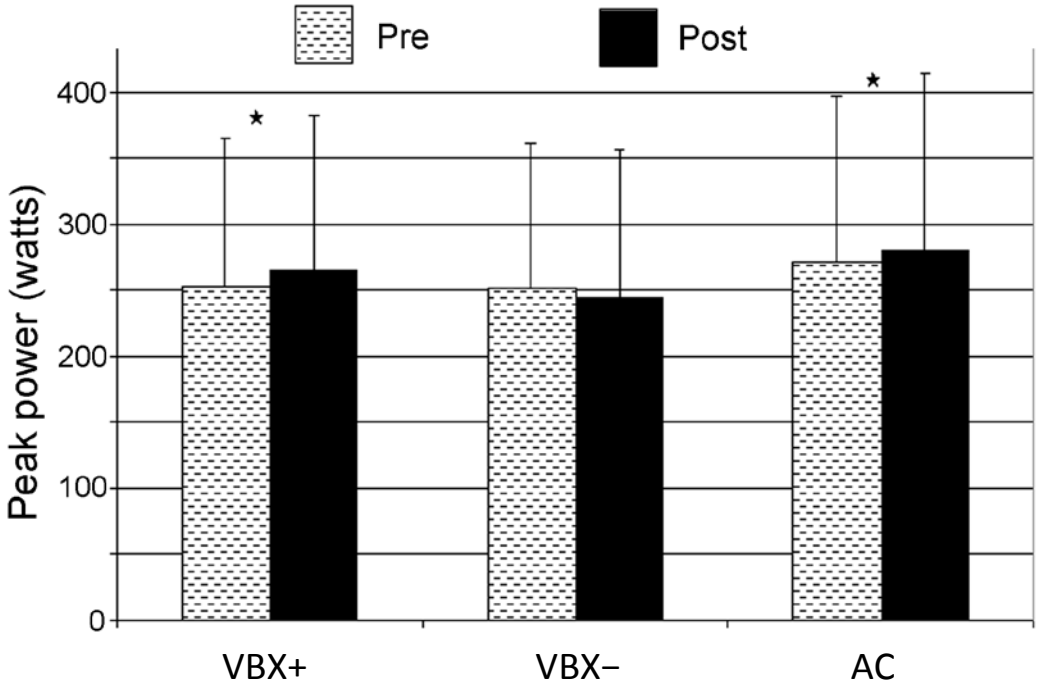



**Figure 5** Mean (± SD) PBP peak power for pre- and post-vibration exercise (VBX+), without vibration exercise (VBX−) and arm crank (AC).

★A significant interaction effect ($p<0.001$) for intervention x pre-post.

| Intervention | Pre | Post |
|---|---|---|
| VBX+ (mV) | 0.64 (0.26) | 0.66 (0.28) |
| VBX− (mV) | 0.62 (0.27) | 0.60 (0.27) |
| AC (mV) | 0.65 (0.29) | 0.64 (0.30) |

**Table 1** Mean (± SD) $EMG_{rms}$ middle trapezius for pre- and post-vibration exercise (VBX+), without vibration exercise (VBX−) and arm crank (AC).

p value interaction effect (intervention x pre-post); VBX+ pre-post vs VBX− pre-post = 0.319; VBX+ pre-post vs AC pre-post = 0.469; VBX− pre-post vs AC pre-post = 0.833.

## 2.5 Discussion

The aim of this study was to compare the effect that VBX+, VBX− and AC had on concentric muscle maximal performance. The results support the hypothesis that acute VBX+ would be associated with enhanced peak power, compared to control (VBX−). However, AC also increased PP, and EMG activity remained unchanged after the three interventions.

The augmented peak power of this study is in agreement with results of Issurin *et al.* (1994) who found that acute superimposed vibration bilateral bicep curl increased peak power by 8% in male amateur athletes. Furthermore, Bosco *et al.* (1999a) reported that acute vibration increased unilateral bicep curl power output by 12% in national representative boxers. However, no previous studies have collected EMG data on concentric peak power.

The exact mechanism through which acute vibration causes strength and power increases is yet to be fully elucidated. The current theory is that vibration provides mechanical stimuli that cause the muscle fibres to stretch thereby evoking the stretch reflex, which enhances the neuromuscular function through neurogenic excitability and recruitment (Cardinale & Bosco, 2003; Rittweger et al., 2000). Evidence for that theory is supported by the potentiation of EMG activity during vibration (Bosco et al. 1999a; Delecluse et al. 2003; Torvinen et al. 2002b), enhanced tendon reflex amplitude (Rittweger et al., 2003) and an enhancement of the H-wave to M-wave ratio of the Hoffmann reflex (Nishihira et al., 2002). However, little consideration has been given to possible peripheral influences. These remain elusive, given that the observed increases in power and strength from acute vibration exercise were compared to the acute changes seen in different studies in the resistance training literature, which are founded on neural adaptations (Bosco et al. 1999b; Torvinen et al. 2002a).

In the present study, the increase in peak power from acute VBX+ did not occur in conjunction with a corresponding potentiation in the EMG activity of the middle trapezius muscle. To date, the response of EMG activity after vibration remains equivocal. For example, Bosco *et al*. (1999a) reported a concomitant increase in mean power and reduced EMG activity of the biceps brachii after 5 x 60s vibration exposure ($f$=30 Hz, $A$=6mm) from a custom built hand-held vibratory machine. In contrast, Cormie *et al*. (2006) found that after a 30s vibration exposure ($f$=30 Hz, $A$=2.5mm) from a commercially manufactured standing vibration plate, countermovement jump height increased with no change to peak power. Additionally, there was no post-vibration change in EMG activity of the quadricep and hamstring muscles. The differences in vibration duration, amplitude, muscle groups, and vibration machines used in the aforementioned studies, may account for the discrepancy in results. On the basis of the results, it was postulated that acute vibration exercise may provide a peripheral (ergogenic) effect that potentiates muscular performance. However, in the future it would be of interest if the EMG of

the latissimus dorsi and bicep brachii were assessed to determine if the vibration stimulus altered their activity.

The increase in power output from acute VBX may be explained by the mechanism of the vibration causing activation of the gamma fusimotor input which enhances the discharge of primary afferents to increase motoneuron activation, thereby causing more powerful muscle contractions (Bosco et al. 1999b). Furthermore, it is possible that the initial adaptations seen in power-strength training, which are neural in origin, may also be applicable to acute VBX (Delecluse et al. 2003; Roelants et al. 2004). For example, the increase in neural activation may increase motor unit firing rates, enhance motor unit synchronisation, recruit a greater number of motor units and increase the coordination of agonist muscle activity (Behm, 1995; Hakkinen & Komi, 1985; Issurin et al., 1994).

Previously, it has been shown that vertical jump performance was enhanced by acute VBX compared to control (no VBX) and cycling interventions (Cochrane & Stannard 2005). Therefore, the increase in peak power of the current study illustrates that acute VBX potentiates not only the neurogenic shortening after lengthening activity but also concentric action. However, the increase in peak power did not parallel a rise in EMG activity and was no different from arm cranking. This may indicate that the peripheral system is providing an ergogenic effect to the stretch reflex response. Moreover, evidence of this peripheral effect is supported by acute erythema after exhaustive VBX (Rittweger et al., 2000) and an increase in mean blood flow of the popliteal artery after acute VBX (Kerschan-Schindl et al., 2001). In contrast to a previous study which showed an increase in muscle power of acute VBX compared to cycling (Cochrane & Stannard, 2005), the present study found no difference in peak power between acute VBX+ and AC, most probably because the previous study employed a performance test that required the use of the stretch reflex.

Given that the vibration amplitude may play an important role for inducing the muscle stretching response, it is probable that the vibration amplitude of the dumbbell in the current study may have been too small to produce the required neural potentiation (Cardinale et al., 2006), which relies on a greater magnitude and number of primary afferents being activated (Matthews & Muir, 1979). Indeed, other studies have either used a greater amplitude of 6mm (Bosco et al., 1999a) or have utilised a similar amplitude to the present study (3mm), with a greater vibration frequency (Issurin & Tenenbaum, 1999). Additionally, vibration amplitude was probably slightly reduced towards the proximal (shoulder) end of the limb due to the damping effects of the soft tissues in the arm (Crewther et al., 2004).

As there are no prescribed guidelines for acute VBX use by the public at the time of writing, different investigators have used vastly differing protocols with few comparisons with other controls (Bosco et al. 1999b; Issurin and Tenenbaum 1999; Torvinen et al. 2002b). In this study

acute VBX+ and AC elicited an increase in peak power compared to VBX− which suggests that, like AC, acute VBX may be an effective intervention for warming-up. Numerous warm-up mechanisms have been described to increase performance (Bishop, 2003). However, one such mechanism known as post-activation potentiation has been proposed to enhance muscle contractility and performance from previous muscular activity (Gourgoulis et al., 2003; Young et al., 1998). The mechanism currently being proposed for this potentiation is the phosphorylation of myosin regulatory light chains and the increased sensitivity of calcium released from the sarcoplasmic recticulum (Sale, 2002).

In the current study there was an increase in peak power from acute VBX+ and AC, but no change in EMG activity, and hence it is feasible to speculate that a post-activation potentiation may have been evident. Therefore, acute VBX may be an alternative method to other conventional warm-up procedures and could also have a place in enhancing performance of sporting activities, for both of which acute VBX requires little effort and time. However, further research should focus on the postulated peripheral mechanism and investigate the effects of comparing passive and dynamic warm-up with VBX. Furthermore, the appropriate VBX guidelines of duration, amplitude and frequency are required before VBX can be used as a potential warm-up intervention.

In conclusion, the results of the present study indicate that both VBX+ and AC enhanced peak power compared to without vibration (control), with no change in EMG activity. There was no significant difference in peak power between VBX+ and AC which may be accounted by the smaller vibration amplitude used in the current VBX protocol. Additionally, the performance test (prone bench pull) was a concentric-only contraction, which may have not maximised the stretch reflex response. It is proposed that the heightened response of VBX+ and AC represents an ergogenic phenomenon known as a post-activation potentiation, which is part of the warm-up effect. However, before vibration exercise may be used as a potential warm-up intervention further research should focus on comparing muscle performance effects of increased muscle temperature of acute vibration exercise and other traditional warm-up modalities.

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# Chapter 3 – The Effect of Acute Lower-Body Vibration Exercise on the Rate of Muscle Temperature Increase

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## 3.1 Abstract

This study compared the rate of muscle temperature ($T_m$) increase during acute vibration exercise (VBX), to that of stationary cycling and passive warm-up. Additionally, this study set out to determine if the purported increase in counter-movement jump and peak power cycling from acute VBX could be explained by changes in muscle temperature.

Eight active participants volunteered for the study, which involved a rest period of 30 minutes to collect baseline measures of muscle, core, skin temperature, heart rate, and thermal leg sensation, which was followed by three vertical jumps and 5s maximal cycle performance test. A second rest period of 40 minutes was enforced followed by the intervention and performance tests. The change in $T_m$ elicited during cycling was matched in the hot bath and VBX interventions. Therefore, cycling was performed first, proceeded by, in a random order of hot bath and acute VBX.

The rate of $T_m$ was significantly greater ($p<0.001$) during acute VBX (0.30ºC/min) compared to cycle (0.15ºC/min) and hot bath (0.09ºC/min) however there was no difference between the cycle and hot bath, and the metabolic rate was the same in cycling and VBX (19 ml/kg/min). All three interventions showed a significant ($p<0.001$) increase in countermovement jump peak power and height. For the 5s maximal cycle test there were no significant differences in peak power between the three interventions.

In conclusion, acute VBX elevates $T_m$ more quickly than traditional forms of cycling and passive warm-up. Given that all three warm-up methods yielded the same increase in peak power output, it was proposed that the main effect would be caused by an increase in $T_m$.

*Keywords* Oxygen uptake, passive heating, concentric-eccentric oxygen cost, hot water immersion.

## 3.2 Introduction

As shown by Asmussen and Boje (1945) raising body temperature prior to exercise has the potential to enhance short-term performance (for a recent review see Bishop 2003). Active and passive modalities are frequently used to elevate muscle and/or core temperature. Passive modalities normally involve using an approach such as hot water baths and saunas whereby externally applied heat is absorbed by the body's tissues. Active modalities require dynamic and rhythmic muscular work such as running, cycling, and skipping which metabolic heat production leads to an increase in muscle temperature.

Acute vibration exercise (VBX), a currently popular exercise modality, is receiving a lot of interest as a warm-up tool. It initiates a rapidly and repeating eccentric-concentric action which evokes muscular work and an elevation in metabolic rate (Rittweger et al., 2000). This is achieved by standing on a commercially manufactured machine with an oscillating platform, which moves in the vertical plane or tilts up and down about a central axis. When standing on the platform with the centre of gravity over the axis and feet either side, the machine produces vertical sinusoidal vibrations indirectly to the body. Using low vibration frequency (10-45 Hz), acute vibration has been reported to increase peripheral circulation, muscle blood flow, skin temperature (Kerschan-Schindl et al., 2001; Lohman et al., 2007; Oliveri et al., 1989) and increases lower limb erythema (Oliveri et al., 1989; Rittweger et al., 2000). Typically, acute VBX seems to elicit mild cardiovascular changes similar to that of moderate walking (Rittweger et al., 2000). These effects are all indicative of an increase in muscle temperature, although the effects of acute VBX on muscle temperature have not been investigated.

Acute VBX has been shown to enhance muscle power (Bosco et al., 1998; Cochrane & Stannard, 2005). It has been suggested that acute VBX may improve muscular performance via neurogenic potentiation involving the spinal reflexes and muscle activation (Cardinale & Bosco, 2003; Rittweger et al., 2003). The main evidence in support of neurogenic potentiation is based on measurement of the tonic vibration reflex (TVR). Previous studies have reported that a vibration stimulus is capable of augmenting muscle spindle activity which causes an excitatory response in the primary endings of non contracting muscle (Burke et al., 1976; Ribot-Ciscar, Rossi-Durand, & Roll, 1998).

It is well known that by increasing tissue temperature, muscular performance can be enhanced (Asmussen & Boje, 1945; Bennett, 1984; Bishop, 2003; Davies & Young, 1983; Dolan, Greig, & Sargeant, 1985; O'Brien et al., 1997; Sargeant, 1987). Likewise, raising muscle temperature (cycling 98 W for 5 minutes) by 1.4°C is associated with improvement in short-term cycling performance by 4.2% (Asmussen & Boje, 1945), with further evidence suggesting that re-warming the muscle by 1.4°C during the intermission of a soccer game does not deteriorate 30m sprint performance compared to no re-warming (Mohr, Krustrup, Nybo, Nielsen, &

Bangsbo, 2004). Given that 70 W of cycling for 10 minutes is commonly used as a laboratory based warm up (Gavin et al., 2007; Paulsen, Myklestad, & Raastad, 2003; Ronnestad, 2004), it is probable that it will elicit a similar muscle temperature change to that of Asmussen and Boje (1945) and Mohr *et al*. (2004), and therefore one might expect an increase in short-term performance.

However, it is unclear whether an increase of muscular power from acute VBX can be explained by an increase in muscle temperature alone. Knowledge of this is crucial to a elucidating the physiological effects of acute VBX. Therefore, comparing the performance effects of increases in muscle temperature elicited by acute VBX and other traditional warm-up modalities will provide additional information about the role of muscle temperature and the suitability of acute VBX as a warm-up intervention.

Thus the aim of this study was to compare the muscle temperature changes elicited by acute VBX, stationary cycling, and passive warm-up and relate these changes to the increase in muscle power. Acute VBX has been reported to increase muscular power (Bosco et al., 1998; Cochrane & Stannard, 2005) although the precise mechanism is unclear. Therefore, it was hypothesised that the purported increase in muscular power was likely to be partly muscle temperature dependent but that acute VBX would elicit a greater increase in peak power output compared to stationary cycling and passive warm-up techniques consequent upon neurogenic or other potentiation.

## 3.3 Methods

### *Participants*

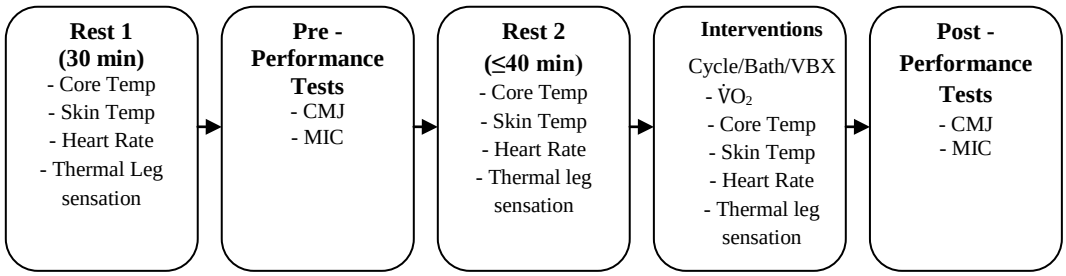
Six males and two females with a mean age (± SD) of 28 ± 7 yr; body mass 72 ± 7 kg, height 1.76 ± 0.03 m, and having a moderate active lifestyle and were not active in cycling volunteered to participate in the study. Informed written consent was obtained from the participants and ethical approval was granted by the Manchester Metropolitan University Ethics Committee.

### *Study Design*

The study consisted of three interventions: 1) control warm-up (stationary cycling); 2) passive warm-up (hot water bath immersion); and 3) active warm-up (VBX). All participants completed the control (cycling) condition followed in a random order by the other two interventions. To minimise the circadian influence the participants performed all three interventions at the same time of day. Given that stationary cycling for ~10 minutes at ~70 W is commonly used for laboratory based warm ups (Gavin et al., 2007; Paulsen et al., 2003; Ronnestad, 2004), it was intended that for each individual the change in muscle temperature elicited by the cycling intervention would be matched in the subsequent hot bath and VBX conditions. Moreover, from

pilot testing it was found that squatting on the vibration plate at tempo of 3s up and 3s down at approximately 90° of knee flexion matched the metabolic rate of cycling at 70 W.

Each participant visited the laboratory on three separate occasions, with at least 3 days separating each testing session. Following instrumentation of rectal and intra-muscular thermocouples each participant rested on a reclined examination table for 30 minutes during which time muscle ($T_m$), core ($T_c$) and skin temperature ($T_{sk}$), heart rate (HR), and thermal leg sensation (TLS) were recorded (Figure 1). Immediately following the rest period, performance tests were conducted which included three vertical countermovement jumps (CMJ) and a five second maximal isokinetic cycle test (MIC). Following this, a second rest period on the reclined examination table was administered for 40 minutes or until muscle temperature reached the resting 1 30-minute value. This was followed by the interventions (stationary cycling, hot water bath, or VBX) and post performance tests of CMJ and MIC that were performed within 90s of completing the interventions. All participants were familiarised with equipment, tests and procedures before the commencement of the study. Additionally the laboratory conditions for the study were maintained at an ambient temperature ($T_{amb}$) of 22 ± 0.4°C and relative humidity (RH) 37 ± 0.7%.




CMJ = countermovement jumps; MIC = five second maximal isokinetic cycle test; $\dot{V}O_2$ = rate of oxygen uptake

**Figure 1** Schematic of the test protocol featuring the five phases, the physiological and performance measures.

## *Physiological Measures*

The procedure for muscle temperature ($T_m$) involved sterilising the insertion area of vastus lateralis muscle with betadine antiseptic solution (Medlock, Oldham, UK). An 18-guage, 1.2 x 45mm cannula (BD, Venflon, Sweden) was inserted perpendicular to the vastus lateralis at two thirds of the line joining the anterior superioriliac spine to the base of the patella (Kenny et al., 2003). To ensure that subsequent thermocouple placement was consistent between interventions the insertion site was marked with a permanent pen, measured and recorded.

The needle of the cannula stylet was then withdrawn and a flexible muscle thermocouple (MAA, Ellab, Denmark) was inserted into the cannula to a depth of 40mm leaving an exposed

tip of approximately 12mm. To prevent any dislodgement, a water-proof plaster (Opsite, Smith & Nephew, Hull, UK) and surgical tape (Transpore, 3M, Neuss, Germany) was used to secure the catheter shaft and the thermocouple to the leg. To ensure that the thermocouple remained at the same depth during each session a permanent pen was used to mark the entry of the thermistor into the cannula, which was frequently checked by the researcher for any displacement. The thermocouple was connected to a display unit (Ellab, Denmark) and recorded $T_m$ every 5 minutes for the rest phases, every 2 minutes for the cycling and hot bath interventions and every minute for VBX.

Skin temperature ($T_{sk}$) was measured from the right side of the body by an infrared thermometer (First Temp Genius, Sherwood, Davis & Geck, St Louis, USA) at the shin, anterior thigh, shoulder and chest. Mean skin temperature was calculated using the four site weighted method of Ramanathan (1964). $T_{sk}$ was recorded at the commencement and completion of the rest phases and interventions. Core temperature ($T_c$) was measured by inserting a thermistor probe (Grant Instruments, Cambridge, UK) 100mm beyond the anal sphincter that was connected to a 1250 series Squirrel data logger (Grant Instruments, Cambridge, UK). $T_c$ was recorded every 5 minutes for the rest phases, every 2 minutes for the cycling and hot bath interventions and every minute for acute VBX.

Thermal leg sensation (TLS) was recorded from an 11-point chart rating adapted from Parsons (2003), where 0 = extremely cold, 5 = neutral, 10 = extremely hot. TLS was recorded every 5 minutes for the rest phases, 2 minutes for the cycling and hot bath interventions and every minute for the acute VBX. Heart rate (HR) was recorded every 5 minutes for the rest phases and the final 2 minutes of each intervention from a watch and telemetric belt (Polar Electro Oy, Kempele, Finland). Rate of oxygen uptake ($\dot{V}O_2$) was analysed during the three interventions by a portable breath by breath gas exchange system (K4b$^2$, Cosmed, Rome, Italy). The K4b$^2$ was calibrated with ambient air ($O_2$ 20.93%, $CO_2$ 0.03%) and a gas concentration mixture ($O_2$ 16.00%, $CO_2$ 5.00%) and the turbine was calibrated using a known 3 litre volume syringe (Cosmed, Rome, Italy). The mean value of $\dot{V}O_2$ was calculated in the final 2 minutes of each intervention and expressed in ml/kg/min.

### *Performance Tests*

Three vertical countermovement jumps (CMJ), separated by 10s of rest were performed on a strain gauge ground reaction force platform linked to a desktop computer using an integrated analog-digital board and software system (Leonardo version 4.1.19, Novotec, Pforzheim, Germany). The participants were instructed to remove their shoes, stand with feet shoulder width apart and position hands on hips to negate any influence of the upper body. Additionally the participants were informed to keep their head still during the flight of the jump. The maximum jump height (CMJ-Ht) was calculated as the highest displacement of the centre of mass and the relative peak power (CMJ-PP) was calculated from the product of force and velocity. The three CMJ's were averaged to give mean height and peak power which were used for further analysis.

The 5s maximal cycle test (MIC) was performed at a cadence of 110 rpm on an isokinetic cycle ergometer which has been described in detail elsewhere (Beelen, Sargeant, & Wijkhuizen, 1994). In brief, this is a custom built cycle that has a 2.2 kW electric motor and a variable gear box that permits a constant braking system over a wide range of pre-set pedal cadences (60-180 rpm). Strain gauges mounted in the pedals measured the horizontal and vertical forces. Incremental encoders were used to record the angle between crank and pedal surface, and the angle of the crank with respect to the vertical (Beelen et al., 1994). The forces, pedal and crank angles were captured at 140 Hz and stored on a desktop computer for subsequent off-line analysis. Custom-built computer software (Igor, version 1.0b, Alsager, Cheshire, UK) was used to calculate peak power, which was derived from the mean of three consecutive values in which the highest peak value occurred.

For every test, the participant was seated on the cycle with feet secured to the pedals by toe clips. A large belt was placed around the anterior pelvis that was secured to an adjustable pulley to the rear of the ergometer to prevent the participants rising from the seat to ensure the power output was generated by the legs. From a stationary start the participants cycled maximally at a cadence of 110 rpm for five seconds. It has been previously reported that the greatest mechanical power output is generated at a pedal cadence of approximately 110–130 rpm (Beelen & Sargeant, 1991; Sargeant, Hoinville, & Young, 1981).

### *Interventions*

The cycling intervention involved pedalling for 10 minutes at 70 W (pre-set at 50 revs/min) on an electronically braked cycle ergometer (Ergometrics 800; Ergoline, Bitz, Germany). Given that stationary cycling at ~70W is a frequently used work-load for warming-up in the laboratory (Gavin et al., 2007; Paulsen et al., 2003; Ronnestad, 2004), the present study was designed so that the change in muscle temperature elicited during the stationary cycle warm-up was matched to the hot water bath and acute VBX interventions. Therefore, the intervention

sequence was cycling followed by, randomly allocating the order of the hot water bath and acute VBX.

For the water bath intervention the participants stood with both legs immersed in a large plastic drum of hot water (41°C). The water temperature was kept constant by regularly monitored by a thermometer and when required, hot water was added to the drum. The participants then adopted a semi-crouch position of approximately 20° hip and knee flexion to ensure adequate water coverage up to the gluteal fold. For support, the participants were able to rest their back against the inside of the drum. $T_{sk}$, $T_c$, and leg thermal sensation (TLS) were recorded every 2 minutes with $\dot{V}O_2$ and heart rate being recorded by the portable breath by breath gas exchange system.

VBX was performed on a commercial machine (Galileo, Novotec, Pforzheim, Germany), which has a teetering board that produces vertical sinusoidal vibration to the body. The participants wore socks and placed their feet on either side of the central axis which corresponded to a vibration amplitude (peak-to-peak) of 6mm, with the vibration frequency set to 26 Hz, which concurs with previous reports that 26 Hz augments jump height from a possible warming-up effect (Bosco et al., 1998; Cochrane & Stannard, 2005). The participants were exposed to vibration in the squatting position at a tempo of 3s down and 3s up at an approximate depth of 90° knee flexion. It is unlikely that the current VBX protocol would have induced fatigue, as previous research has reported that squatting to exhaustion takes approximately 6 minutes and requires an additional load of 40% of body mass at a depth, tempo and vibration frequency of the current protocol to elicit a post-exercise blood lactate of 5.5 mmol/L (Rittweger et al., 2003).

### *Statistical Analyses*

The values of $T_c$, $T_m$, rate $\dot{T}_m$, final and start $T_m$, $T_{sk}$, HR, TLS, $\dot{V}O_2$, CMJ-Ht, CMJ-PP and MIC were averaged and were used for further analyses and compared with pre- intervention values taken from the second rest period.

All observed rates of increase in $T_m$ were linear and for every participant $T_m$ was analysed by linear regression to determine the rise of rate of muscle temperature ($\dot{T}_m$), which was analysed by a univariate ($\dot{T}_m$, intervention, participant) ANOVA. A repeated measures 2 (pre-post) x 3 (cycle, bath, VBX intervention) ANOVA were performed to examine $T_c$, mean $T_{sk}$, start and final $T_m$, CMJ-Ht, CMJ-PP and MIC. Additionally, a one factor repeated measure (cycle, hot bath, VBX intervention) ANOVA was performed to examine the duration to reach $\dot{T}_m$, $\dot{V}O_2$, HR, thermal leg sensation and $\dot{T}_m/\dot{V}O_2$. For multiple comparisons significance, post-hoc pairwise comparisons were performed and adjusted to Bonferroni's rule with a post-hoc simple

contrast performed for $\dot{T}_m/\dot{V}O_2$. Intratrial reliability of CMJ-Ht, CMJ-PP and MIC was calculated using a coefficient of variation (CV = SD/mean × 100). All statistical analyses were performed using statistical software SPSS for Windows Version 14 (Chicago, IL, USA) and significance was set at the 95% level of confidence ($p < 0.05$).

## 3.4 Results

For all the interventions final $T_m$ was significantly higher (37.1 ± 0.9ºC) compared to the start $T_m$ (35.6 ± 0.7ºC), however there was no significant difference of the start $T_m$ or final $T_m$ for the three interventions (Table 1). Moreover, the mean rate of increase in muscle temperature $\dot{T}_m$ (Table 1) was significantly greater ($p<0.001$) during acute VBX with dynamic squatting (0.30ºC/min) compared to cycle (0.15ºC/min) and hot bath with static squatting (0.09ºC/min) but there was no significant difference between cycle and hot bath.

The metabolic rate of the bath intervention (5 ml/kg/min) in raising mean muscle temperature 1.5°C was significantly less ($p<0.001$) than that of either cycling (19 ml/kg/min) or acute VBX with dynamic squatting (19 ml/kg/min). Post-hoc contrast revealed that acute VBX with dynamic squatting had a significantly higher ($p<0.05$) $\dot{T}_m$ increase per oxygen cost (0.29ºC/L) compared to cycling (0.11ºC/L). Furthermore, there was no significant difference in pre- and post- $T_c$ and mean $T_{sk}$ between the three interventions (Table 1).

HR exhibited a significantly ($p<0.05$) higher response to acute VBX (105 ± 16 bpm) and cycle exercise (105 ± 15 bpm) compared to hot bath intervention (85 ± 16 bpm) (Table 1) and all interventions revealed that post-thermal leg sensation (TLS) was significantly higher ($p<0.05$) compared to the pre-values. For the TLS the hot bath exhibited a greater pre-post difference (2.9) compared to the cycle (1.7), however no other significances were found (Table 1).

All three interventions reported a significant ($p<0.001$) post increase in CMJ-Ht (2.3 cm cycle; 2.8cm hot bath; 3.2 cm VBX) and CMJ-PP (2.5 W/kg cycle; 2.8 W/kg hot bath; 2.5 W/kg VBX), however there was no significant difference between comparing the pre-post jump height of the other interventions (Table 2). There was no significant increase or difference between the interventions for the 5 s maximal isokinetic cycle test (MIC).

The coefficient of variation (CV) of the three interventions for CMJ-Ht (Cycle 3.0%; Hot bath 3.6%; VBX 3.5%) and CMJ-PP (Cycle 2.2%; Hoth bath 2.0%; VBX 2.4%) suggests a high degree of stability was achieved. Likewise, the MIC-PP between the interventions reported good reliability (CV = 4.0%).

**Table 1** Mean (± SD) of pre- and post-exercise time, core temperature, mean skin temperature, heart rate, thermal leg perception oxygen uptake of cycle, hot bath and VBX.

| Variable | Cycle | Hot bath | VBX |
|---|---|---|---|
| Start $T_m$ (ºC) | 35.7 ± 0.8 | 35.6 ± 0.7 | 35.6 ± 0.7 |
| Final $T_m$ (ºC) | 37.2 ± 0.9** | 37.1 ± 0.9** | 37.1 ± 0.9** |
| Pre $T_c$ (ºC) | 37.2 ± 0.3 | 37.0 ± 0.4 | 37.1 ± 0.3 |
| Post $T_c$ (ºC) | 37.3 ± 0.3 | 37.2 ± 0.5 | 37.1 ± 0.4 |
| $\dot{V}O_2$ (ml/kg/min) | 19 ± 2$^{+}$ | 5.1 ± 1.5 | 19 ± 2$^{+}$ |
| Rate of $\dot{T}_m$ (ºC/min) | 0.15* | 0.09* | 0.30 |
| Duration to reach $\dot{T}_m$ (min:s) | 10:00 ± 0* | 17:18 ± 1.5* | 5:15 ± 0.5 |
| Pre Mean $T_{sk}$ (ºC) | 32.6 ± 0.4 | 32.2 ± 0.8 | 32.1 ± 0.7 |
| Post Mean $T_{sk}$ (ºC) | 32.3 ± 1.0 | 33.1 ± 2.1 | 32.0 ± 0.5 |
| Pre HR (bpm) | 69.7 ± 5.4 | 65.9 ± 8.0 | 68.6 ± 8.3 |
| Exercise HR (bpm) | 105.1 ± 14.6$^{\#}$ ‡ | 85.0 ± 16.3$^{\#}$ | 105.0 ± 16.3$^{\#}$ ‡ |
| Pre TLS | 4.9 ± 0.6 | 4.4 ± 1.3 | 4.8 ± 0.7 |
| Post TLS | 6.6 ± 0.6** ‡ | 7.3 ± 0.9** | 6.9 ± 0.8** |
| $\dot{T}_m/\dot{V}O_2$ (ºC/L) | 0.11 ± 0.03 | 0.22 ± 0.11$^{++}$ | 0.29 ± 0.14$^{++}$ |

$T_m$ = muscle temperature; $T_c$ = core temperature; $\dot{T}_m$ = muscle temperature change; $T_{sk}$ = skin temperature; HR= Heart Rate; TLS = Thermal leg sensation

* $p<0.01$ compared to VBX; $^{+}$ $p<0.001$ compared to Hot bath; ** $p<0.001$ compared to Start; $^{\#}$ $p<0.001$ compared to Pre; $^{‡}$ $p<0.05$ compared to pre-post difference of Hot bath; $^{++}$ $p<0.05$ compared to Cycle

**Table 2** Mean (± SD) of pre- and post-isokinetic cycle, CMJ peak power of cycle, hot bath and VBX

| Variable | Cycle | Hot bath | VBX |
|---|---|---|---|
| Pre CMJ - Ht (cm) | 40 ± 7 | 39 ± 7 | 39 ± 8 |
| Post CMJ - Ht (cm) | 43 ± 8* | 42 ± 7* | 43 ± 8* |
| Pre CMJ - PP (W/kg) | 45 ± 9 | 46 ± 7 | 45 ± 9 |
| Post CMJ - PP (W/kg) | 47 ± 10* | 49 ± 8* | 47 ± 10* |
| Pre MIC - PP (W) | 1622 ± 284 | 1659 ± 265 | 1693 ± 274 |
| Post MIC - PP (W) | 1609 ± 287 | 1695 ± 322 | 1768 ± 323 |

CMJ-Ht = Counter movement jump height; CMJ-PP = Counter movement jump peak power; MIC = 5 sec maximal isokinetic cycle test

* $p \leq 0.001$ compared to Pre

## 3.5 Discussion

Warming-up prior to physical activity is a procedure aimed at optimising performance. Active and passive modalities are frequently used to elevate $T_m$ which is a major determinant for enhancing short-tem explosive events (for review see Bishop 2003). The aim of this study was to compare the effect of acute VBX with dynamic squatting and hot water immersion with static squatting to stationary cycling on the elicited change in $T_m$ and associated increases in muscle power. The major finding of the current study was that the rate of $T_m$ during acute VBX with dynamic squatting was around twice that achieved by cycle and hot water immersion interventions.

The question arises as to which physiological mechanisms are responsible for this difference. Similar to cycling, acute VBX is a rhythmical activity that increases muscle temperature via active movement and increased metabolism of the major muscle groups. However, unlike the concentric-only action of cycling, acute VBX is able to elicit rapidly repeating concentric-eccentric muscle actions. One could argue that the total number of muscle contractions, and thus total work differs for acute VBX with dynamic squatting, compared to cycling and hot bath interventions. However, the whole-body metabolic cost and heart rate was identical between cycling and acute VBX ($\dot{V}O_2$ 19 ml/kg/min; HR 105 bpm), and not surprisingly active warm-up induces a greater metabolic and cardiovascular change compared to passive methods (Dolan et al., 1985; O'Brien et al., 1997). Therefore, the rate of muscle temperature increase can be quite different even when the energy turnover is matched muscle temperature must be measured in order to compare different warm-up strategies.

In all three interventions there was a small significant increase in post CMJ-PP and CMJ-Ht but there were no differences between the interventions, this is not surprising given that the mean change in $T_m$ of 1.5°C was identical in all three interventions. Moreover, the rate of improvement (VBX 5.4%/°C; cycling by 3.8%/°C; hot bath 4.7%/°C) for the CMJ-Ht was comparable to other findings (Asmussen & Boje, 1945; Bergh & Ekblom, 1979). However, there were no comparable post increase in MIC. The likely explanation is that the MIC involved an all out 5s sprint which is temperature dependent (Mohr et al., 2004), and relies on augmenting the neural transmission rate (Bishop, 2003). Given that $T_m$ was only elevated 1.5°C, this may not have been sufficient to facilitate the nerve conduction rate during the high-velocity muscular contractions of isokinetic cycling. Furthermore, the peak power of MIC was slightly higher than those reported by Sargeant (1987), this can be accounted for by the higher cadence (110 rpm) used in the current study.

It was hypothesised that acute VBX with dynamic squatting would augment peak power cycling (MIC-PP) and counter-movement jump (CMJ-PP) more than conventional active and passive warm-up techniques. Although there is no current consensus on the mechanism of acute

VBX, it has been proposed that an increase in muscular power from acute VBX is primarily due to a neurogenic potentiation involving the spinal reflexes and muscle activation (Cardinale & Bosco, 2003; Rittweger et al., 2003). Therefore, acute VBX may provide an additional neurogenic effect to $T_m$. However, the CMJ-PP and CMJ-Ht performance improvement was similar between all three conditions suggesting that for acute VBX a non-temperature effect in the form of an excitatory neuromuscular response was not additional to the $T_m$ change. Therefore, short-term performance, such as CMJ-PP, CMJ-Ht and MIC-PP can be explained by the increase in $T_m$ alone (Sargeant, 1987). However, one cannot discount the possibility that other non-muscle temperature related factors may have enhanced short-term performance such as neuromuscular activation (Gourgoulis et al., 2003) and increases in muscle blood flow (Lohman et al., 2007).

According to Saltin *et al.* (1968) $T_m$ is directly proportional to the exercise intensity due to the heat generation of the working muscle. Stationary cycling at ~70W is a frequently used work-load for warming-up in the laboratory (Gavin et al., 2007; Paulsen et al., 2003; Ronnestad, 2004) hence the mean change of 1.5°C in $T_m$ reflects the low level of exercise intensity sustained in the current interventions. Therefore, no significant difference in $T_c$ was reported between the three interventions, this is not unexpected given that a higher metabolic rate would be required to generate enough heat to raise $T_m$ beyond that of $T_c$ (Sargeant, 1987). Likewise, there was no significant change from baseline in mean $T_{sk}$ for the three interventions. Moreover, for each intervention the post-thermal leg sensation value was significantly higher ($p<0.001$) compared to pre-values, and suggests that the participants were able to detect a thermal change however there was no significant difference between the interventions. Again, this is not unexpected given that $T_m$ of 1.5°C was identical to all three interventions.

Acute VBX is thought to cause rapidly repeating eccentric-concentric muscular work that elevates metabolic rate (Rittweger et al., 2001), conversely, cycling is dominated by concentric muscle action. Therefore, it is plausible that the eccentric component of acute VBX is more effective in warming the muscle as shown by the significantly higher ($p<0.05$) rate of $\dot{T}_m$ per L $O_2$ in acute VBX (0.22ºC /L $O_2$) compared to cycling (0.11ºC/L $O_2$). This is in agreement with previous research that has shown the oxygen cost is much lower in eccentric compared to concentric work (Abbott et al., 1952; Knuttgen, Patton, & Vogel, 1982).

Although the energy turnover was similar in the cycling and VBX interventions it is probable that in VBX this reflected an increase in muscle activity of many more muscles than simply the leg extensors. Thus the concentric component may be lower in the leg extensor muscles during VBX, while the eccentric component, which requires energy to be absorbed by the muscle in the form of heat without any metabolically related increase in muscle blood flow would be higher than in cycling (Sargeant & Dolan, 1987b). The net result is that the rate of muscle temperature increase in VBX was twice that of cycling warm-up. This has implications for

warming-up prior to physical activity. Currently, it is common practice for team sports such as professional rugby to have portable stationary cycle ergometers on the sidelines during competition to keep substitute players warm throughout the course of the game. However, it may be of greater benefit if vibration platforms or active whole body movements were utilised given that $T_m$ per L $O_2$ is higher in acute VBX compared to cycling. Additionally, a hot bath requires less energy turnover, however this may not be a practical option to have on the sidelines but it may be relevant for sports such as track and field to keep $T_m$ elevated between efforts which are separated by several rest periods or when it is vital for conserving valuable energy (Bishop, 2003).

In conclusion, acute VBX with dynamic squatting elevates $T_m$ more quickly than traditional forms of active and passive warm-up. However, acute VBX with dynamic squatting was not able to significantly augment short-term performance more effectively than cycle and hot bath interventions when the same muscle temperature was achieved. The observed increase in short-term performance following the interventions seems to be muscle temperature dependent (Sargeant & Dolan, 1987a). Therefore, squatting with acute VBX may be an alternative and faster method of increasing muscle temperature compared to other conventional warm-up techniques and may have a place in enhancing performance of sporting activities. To gain a better understanding of the physiological responses involved in the application of VBX, the effect of vibration and different types of squatting (dynamic and static) on muscle temperature require further investigation.

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# Chapter 4 – The Comparison of Muscle Temperatures during Static and Dynamic Squatting With and Without Acute Vibration Exercise

In Press - Clinical Physiology and Functional Imaging

## 4.1 Abstract

The aim of this study was to investigate the influence of shallow dynamic squatting (DS) versus static squatting (SS) with or without concurrent side-alternating vibration exercise (VBX) on vastus lateralis temperature and cardiovascular response as indicated by heart rate (HR). Ten participants (5 male, 5 female) participated in four interventions [DS with VBX (DS+), DS without VBX (DS–), SS with VBX (SS+), SS without VBX (SS–)] 48 hours apart, in a randomised order. The interventions were preceded by a ~ 20 minute rest period, consisted of 10 minutes with or without VBX (26 Hz or 0 Hz) with SS (40º of knee flexion) or DS (55º of knee flexion, at a cadence of 50 bpm) where SS+ and DS– were metabolically matched. Muscle ($T_m$), core ($T_c$), skin temperature ($T_{sk}$), HR and $\dot{V}O_2$ were recorded during each intervention. For $T_m$ there was a time ($p<0.01$) and VBX ($p<0.01$) effect but no squat effect was evident, and there was time x VBX interaction effect ($p<0.01$). In all four interventions the work load was too low to cause cardiovascular changes. Instead normal, moderate physiological effects of exercise on autonomic control were observed as indicated by HR; there were no significant increases in $T_{sk}$ or $T_c$. There appears to be no benefit in performing a shallow DS+ at a tempo of 50 beats per minute (bpm) as $T_m$, HR, $\dot{V}O_2$ are likely to be increased by the same amount and rate without VBX. However, combining SS with VBX could be advantageous to rapidly increasing soft tissue temperature prior to performing rehabilitation exercises when dynamic exercise cannot be performed.

*Keywords* Warm-up, skin temperature, metabolic rate, energy dissipation

## 4.2 Introduction

Acute vibration exercise (VBX) has been reported to increase muscular power (Cochrane & Stannard, 2005), strength (Torvinen et al., 2002), peripheral circulation (Lohman et al., 2007), and muscle blood flow (Kerschan-Schindl et al., 2001). The rapid and repetitive eccentric-concentric action of VBX evokes muscular work (Rittweger et al., 2001), elevates metabolic rate (Rittweger et al., 2000), and elicits mild cardiovascular changes similar to those of moderate walking (Rittweger et al., 2001). These physiological responses are normally associated with an increase in muscle temperature, which is central to optimal muscle function (Bennett, 1984), injury prevention (Bixler & Jones, 1992), and exercise-associated rehabilitation (Rimington, Draper, Durrant, & Fellingham, 1994).

The oscillating action of the VBX platform is thought to elicit rapid, reflex-mediated stretch-shortening of the distal supporting (calf and thigh) muscles (Cardinale & Bosco, 2003; Rittweger et al., 2003; Rittweger et al., 2001). It is well known that eccentric muscle work acts like a brake by absorbing external mechanical energy and results in local heat production (Constable, Barclay, & Gibbs, 1997). On the other hand, concentric muscle contraction acts like a motor to convert chemical energy into mechanical work and local heat production ensues primarily as a by-product of metabolism, although to a lesser extent during concentric than eccentric work (Curtin & Woledge, 1978). Previously, it has been shown that concentric exercise (cycling) produces less heat than VBX for the same metabolic rate ($\dot{V}O_2$) (Cochrane, Stannard, Sargeant, & Rittweger, 2008 [Chapter 3]), probably because little external work is absorbed during concentric exercise.

Enjoying popularity as an alternative exercise modality, VBX is currently being prescribed by trainers, coaches, exercise therapists on the basis that it may provide a nuerogenic potentiation to the muscle which other traditional exercises are not capable of. Previous work has found that matched for metabolic rate, the rate of muscle temperature rise doubled when acute VBX was superimposed upon gentle, slow dynamic squatting compared to stationary cycling (Cochrane et al., 2008 [Chapter 3]). However, the aforementioned study did not distinguish between the effects of the vibration and squatting on the increase in muscle temperature. Static (SS) and dynamic (DS) squats are common exercises utilised in VBX training (Abercromby et al., 2007a; Cardinale & Lim, 2003; Cochrane et al., 2008 [chapter 3]; Rittweger et al., 2001; Roelants, Verschueren, Delecluse, Levin, & Stijnen, 2006; Ronnestad, 2004), but it is unknown if SS or DS is superior in increasing muscle temperature ($T_m$), heart rate (HR) and $\dot{V}O_2$. Shallow squatting of body weight up and down is similar to cyclic vibration-induced concentric/eccentric muscle action, albeit at a much larger amplitude yet lower frequency. Therefore, the effect of vibration and different types of squatting on muscle temperature is crucial to understanding the physiological responses involved in the application of VBX. It may

assist to provide guidance to optimising warm-up strategies and for soft tissue rehabilitation; two reasons for which VBX is commonly employed. It is reasonable, therefore, to propose that the responses of $T_m$ and HR would be very similar between shallow squatting and VBX when matched for metabolic rate.

The primary aim of this study was to investigate the effect of shallow DS versus SS with concurrent VBX on *vastus lateralis* temperature and cardiovascular change as indicated by HR. It was hypothesised that, the increase in $T_m$ and HR would not be significantly different when matched for metabolic rate. It was further hypothesised that when DS was superimposed upon VBX, *vastus lateralis* temperature, HR, and $\dot{V}O_2$ would exceed both DS alone and SS with VBX.

## 4.3 Methods

### *Participants*

Five males ([mean ± SD] age 31.4 ± 5.2 yr; body mass 79.9 ± 10.9 kg, height 1.76 ± 0.1 m) and five females (28.1 ± 5.8 yr; body mass 63.7 ± 5.8 kg, height 1.67 ± 0.1 m), with an active lifestyle of undertaking routine physical activity at least three times a week volunteered to participate in the study. Written informed consent was obtained from the participants and ethical approval was granted by the local University Human Ethics Committee.

### *Study Design*

Every participant performed four interventions, in a randomised order with 48 hours separating each testing session. The interventions were preceded by a ~ 20 minute rest period followed by 10 minutes with a SS (40° knee angle) or DS (55° knee angle, cadence 50 bpm) with or without VBX (*f*=26 Hz or 0 Hz), with the four interventions being DS with VBX (DS+), DS without VBX ( DS–), SS with VBX (SS+), SS without VBX (SS–). For the rest phase the participants sat quietly on an adjustable reclined examination table. Forty degree flexion was selected for SS and 55º knee flexion for DS, the angle being in the lower half of those used in past studies in which SS and DS with VBX were typically performed at knee flexion angles of 20-70º (Abercromby et al., 2007a; Cardinale & Lim, 2003; Cormie et al., 2006; Roelants et al., 2006) (erect stance = 0º knee flexion), and between 35-90º (Abercromby et al., 2007a; Cochrane et al., 2008 [chapter 3]; Rittweger et al., 2001). Additionally, the metabolic rate needed to be matched and from the pilot testing DS– at 50 bpm and 55º of knee flexion was identical to the metabolic rate of a SS+ held at 40º of knee flexion when performed at 26 Hz and 40º of knee flexion. Hence for this study 40º knee flexion for SS and 55º knee flexion for DS were selected.

Participants were fully familiarised with equipment and protocols before undertaking their first session. Lastly, all interventions were performed at a constant ambient temperature (20.3 ± 0.6 ºC [SE]) and relative humidity (47.5 ± 2 % [SE])

*Physiological Measures*

Measuring muscle temperature ($T_m$) involved sterilising the insertion area of vastus lateralis muscle with betadine antiseptic solution (Faulding HealthCare, QLD, Australia). The muscle was then anesthetized to a maximum depth of ~38mm by infiltrating 5 ml of 2% xylocaine (Astra Zeneca, Australia), using a 25 gauge, 0.5mm x 38mm, needle (BD, Singapore). A 16-gauge, 1.2 x 45mm cannula (BD, Venflon, Sweden) was inserted at an inclination of ~45° into the vastus lateralis at a site two thirds the way along a line joining the anterior superioriliac spine and the proximal aspect of the patella with the leg extended (femoro-tibial angle = 0º). To ensure that subsequent thermocouple placement was consistent between interventions the insertion site was marked with a permanent pen, measured and recorded.

The needle of the cannula stylet was then withdrawn and a flexible sensor muscle thermocouple (Physitemp Instruments Inc, Clifton, NJ, USA, Model IT-17:3) was inserted into the cannula to a depth of 45mm. To prevent any dislodgement, a sterile dressing (Tegaderm I.V., 3M Health Care, Neuss, Germany) and surgical tape (Transpore, 3M, Neuss, Germany) were used to secure the catheter shaft and the thermocouple to the leg. To ensure that the thermocouple remained at the same depth during each session a permanent pen was used to mark the entry of the thermistor into the cannula, which was constantly monitored by the researcher for any displacement. The thermocouple was connected to an electronic display unit (TH-8 Thermalert, Physitemp Instruments Inc, Clifton, NJ, USA). $T_m$ was monitored every 5 minutes during the rest phase, and during the interventions $T_m$ was recorded every minute from 0 min ($T_0$) to 10 min ($T_{10}$).

Using an infrared thermometer (First Temp Genius, Sherwood, Davis & Geck, St Louis, USA), skin temperature ($T_{sk}$) was measured at four sites: the right shin, anterior thigh, shoulder and chest and mean $T_{sk}$ was calculated using the four site weighted method of Ramanathan (1964). The $T_{sk}$ was recorded at the commencement and completion of the rest and intervention phases. Core temperature ($T_c$) at 100mm proximal to the anal sphincter was measured with a thermistor probe (Grant Instruments, Cambridge, UK) connected to a USB temperature measurement module (Measurement Computing Corporation, Middleboro, Massachusettes, USA) which was interfaced with a PC notebook computer. $T_c$ was recorded every 5 minutes during the rest phase and every minute during the interventions.

HR was recorded from a watch and telemetric belt (Polar Electro Oy, Kempele, Finland) every 5 minutes during the rest phase and the final 2 minutes of each intervention. Rate of oxygen uptake ($\dot{V}O_2$) was analysed during the four interventions by open circuit breath-by-breath gas exchange system (Turbofit Vacumed, Ventura, California, USA). The metabolic cart was calibrated with ambient air ($O_2$ 20.93%, $CO_2$ 0.03%) and a known gas concentration mixture ($O_2$ 15.7%, $CO_2$ 4.9%) and calibrated using a known 3 litre volume syringe (Vacumed, Ventura,

California, USA). The mean value of $\dot{V}O_2$ was calculated in the final 2 minutes of each intervention and expressed in ml/kg/min.

*VBX and Squatting Interventions*

VBX was performed on a commercial machine (Galileo Sport, Novotec, Pforzheim, Germany), which has a teeter board that produces side-alternating vertical sinusoidal vibration to the body. To negate the possibility of discomfort to the sole of the foot and to standardise the vibration damping caused by footwear, participants wore the same sport shoes for all interventions and placed their feet at a fixed distance marked on the plate either side of the central axis which corresponded to a vibration amplitude (peak-to-peak) of 6mm, magnitude (peak-to-peak acceleration) of 9*g*. Vibration frequency was either 26 Hz or 0 Hz (peak-to-peak amplitude= 0 mm) for both SS (knee angle 40˚) or DS (knee angle 55˚). For SS and DS a manual goniometer was used to set the knee angle and an adjustable hurdle was placed under the gluteal fold. For temporal control during DS a metronome was set at 50 bpm and the participants were instructed to maintain a continuous and smooth squatting motion.

*Statistical Analyses*

The change in $T_m$ was calculated from each interval between $T_0$ and $T_{10}$ and averaged to obtain the intervention mean for the 10 participants. $T_c$ and mean $T_{sk}$ were averaged at 0 and 10 minutes and for HR and $\dot{V}O_2$ the final 2 minutes (8-10 minutes) were averaged. A three factor repeated measures [time x squat (dynamic and static) x VBX (with VBX and without VBX)] ANOVA was performed to test variation in $T_m$, $T_c$, mean $T_{sk}$, and HR. A two factor repeated measures (squat x VBX) ANOVA was used to analyse $\dot{V}O_2$. For multiple comparisons significance, post-hoc pairwise comparisons were performed and adjusted to Bonferroni's rule. All statistical analyses were performed using statistical software SPSS for Windows Version 16 (Chicago, IL, USA) and level of significance was set at $p < 0.05$.

## 4.4 Results

There was no difference in $\dot{V}O_2$ between SS+ and DS– nor was there a difference in $\dot{V}O_2$ between DS+, DS–, and SS+ (Table 1). DS was associated with higher ($p<0.01$) $\dot{V}O_2$ than was SS, $\dot{V}O_2$ was higher ($p<0.01$) during VBX than without VBX, but there was no significant squat x VBX interaction effect on $\dot{V}O_2$.

Following a rest period of 20 minutes there was no significant difference in starting (0 min) $T_m$ ($p=0.77$) between the four interventions (Table 1). $T_m$ increased with time ($p<0.01$) and with VBX ($p<0.01$) but there was no difference in elevated $T_m$ between the two types of squat ($p=0.06$). There was a time x squat interaction effect where DS and SS were associated with increased ($p<0.01$) $T_m$ with time (Figure 1a). Likewise, there was time x VBX interaction effect

($p<0.01$), showing that with VBX $T_m$ increased with time to a greater extent than without VBX (Figure 1b). Also there was a VBX x squat x time interaction effect ($p<0.01$) and Figure 1c shows the time course of the change in $T_m$ for DS+, DS–, SS+ and SS–. Finally, for $\Delta T_m$ there was a squat and VBX effect but no significance was detected between the interventions (Table 1).

There was no significant difference in $HR_0$ ($p=0.43$) between the interventions (Table 1). A time x VBX interaction effect was found where HR was higher at 10 minutes with VBX compared to without VBX. For mean $T_{sk\,0}$ there was no significant difference ($p=0.62$) between the interventions and there were no significant time interaction effect. The change in thigh $T_{sk}$ was significantly higher in SS+ compared to SS–, DS+, and DS– but there were no other differences (Table 1). There were no significant differences in core temperature between the interventions (Table 1) and no time interaction effects.

**Table 1** Group mean (±SE) oxygen uptake, mean skin temperature, core temperature, and heart rate associated with combinations of dynamic and static squatting with and without vibration exercise.

| | **Intervention** | | | |
|---|---|---|---|---|
| **Variable** | **DS+** | **DS–** | **SS+** | **SS–** |
| $\dot{V}O_2$ (ml/kg/min) | 13 ± 1.2$^{\dagger}$ | 10 ± 0.8$^{\dagger}$ | 11 ± 1.2$^{\dagger}$ | 7 ± 1.1 |
| $T_{m\ 0}$ (ºC) | 35.6 ± 0.3 | 35.9 ± 0.3 | 35.8 ± 0.2 | 35.5 ± 0.3 |
| $T_{m\ 10}$ (ºC) | 37.2 ± 0.3*$^{\dagger}$ | 37.0 ± 0.3*$^{\dagger}$ | 37.2 ± 0.2*$^{\dagger}$ | 36.3 ± 0.3* |
| Δ $T_m$ (ºC) | 1.6 ± 0.2 | 1.1 ± 0.1 | 1.4 ± 0.1 | 0.7 ± 0.1 |
| $HR_{0}$ (bpm) | 77.4 ± 4.8 | 72.1 ± 3.0 | 77.2 ± 2.7 | 69.9 ± 5.8 |
| $HR_{10}$ (bpm) | 109.6 ± 6.5$^{\ddagger}$ | 89.0 ± 5.4$^{\ddagger}$ | 113.5 ± 4.1$^{\ddagger}$ | 91.1 ± 5.8$^{\ddagger}$ |
| Δ HR (bpm) | 32.2 ± 9.9 | 13.9 ± 5.5 | 36.3 ± 11.0 | 21.2 ± 6.9 |
| $T_{c\ 0}$ (ºC) | 37.2 ± 0.1 | 37.2 ± 0.1 | 37.2 ± 0.1 | 37.2 ± 0.1 |
| $T_{c\ 10}$ (ºC) | 37.3 ± 0.1 | 37.3 ± 0.1 | 37.2 ± 0.1 | 37.3 ± 0.1 |
| Δ $T_c$ (ºC) | 0.1 ± 0.0 | 0.1 ± 0.0 | 0.0 ± 0.0 | 0.1 ± 0.0 |
| Mean $T_{sk\ 0}$ (ºC) | 31.4 ± 0.4 | 31.5 ± 0.3 | 30.9 ± 0.4 | 31.1 ± 0.4 |
| Mean $T_{sk\ 10}$ (ºC) | 31.6 ± 0.5 | 31.7 ± 0.4 | 31.7 ± 0.5 | 31.0 ± 0.4 |
| Δ Mean $T_{sk}$ (ºC) | 0.2 ± 0.2 | 0.2 ± 0.2 | 0.8 ± 0.3 | -0.1 ± 0.2 |
| $T_{sk\text{-}th\ 0}$ (ºC) | 29.1 ± 0.5 | 29.5 ± 0.5 | 28.7 ± 0.6 | 28.6 ± 0.8 |
| $T_{sk\text{-}th\ 10}$ (ºC) | 29.9 ± 0.8 | 29.7 ± 0.5 | 31.0 ± 0.8$^{\#}$ | 29.4 ± 0.4 |
| Δ $T_{sk\text{-}th}$ (ºC) | 0.8 ± 0.4 | 0.2 ± 0.2 | 2.3 ± 0.2# | 0.8 ± 0.2 |

DS+ = Dynamic Squat with VBX; DS– = Dynamic Squat without VBX; SS+ = Static Squat with VBX; SS– = Static Squat without VBX

$\dot{V}O_2$ = Rate of oxygen uptake values averaged over 8-10min;

$T_{m\ 0}$ = muscle temperature at 0 min; $T_{m\ 10}$ = muscle temperature at 10 min; Δ $T_m$ = change in muscle temperature

$HR_{0}$ = heart rate at 0 min; $HR_{10}$ = mean heart rate between 8 and 10 min; Δ HR = change in heart rate

$T_{c\ 0}$ = core temperature at 0 min; $T_{c\ 10}$ = core temperature at 10 min; Δ $T_c$ = change in core temp

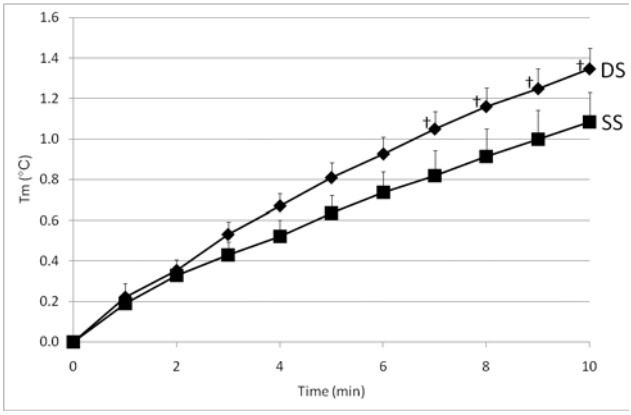
Mean $T_{sk\ 0}$ = mean skin temperature at 0 min; Mean $T_{sk\ 10}$ = mean skin temperature at 10 min; Δ Mean$T_{sk}$ = change in mean skin temperature

$T_{sk\text{-}th\ 0}$ = skin thigh temperature at 0 min; $T_{sk\text{-}th\ 10}$ = skin thigh temperature at 10 min; Δ $T_{sk\text{-}th}$ = change in skin thigh temperature

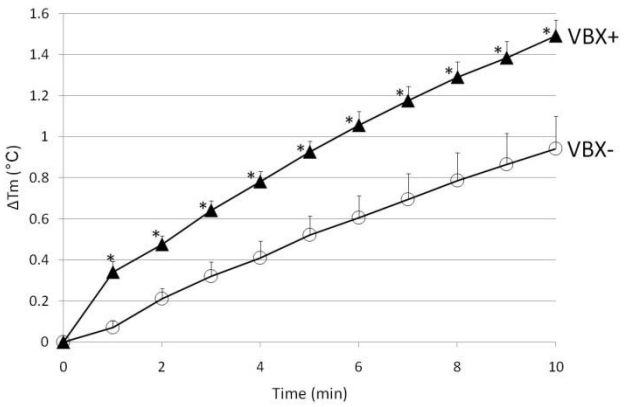
* $p<0.01$ compared to $T_{m\ 0}$; $^{\dagger}$ $p<0.05$ compared to SS–; $^{\ddagger}$ $p<0.01$ compared to $HR_{0}$; $^{\#}$ $p<0.05$ compared to DS+, DS–, SS–

**Figure 1** Group mean (±SE) interaction effects of time, squat and VBX for $T_m$

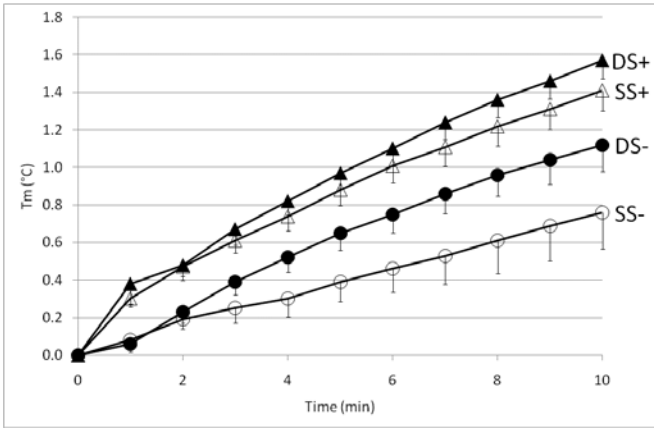
(a) Time x Squat interaction effect




(b) Time x VBX interaction effect




(c) $T_m$ over time during the four interventions




DS = Dynamic Squat, SS = Static Squat; VBX+ = with vibration exercise, VBX− = without vibration exercise; DS+ = Dynamic Squat with VBX, SS+ = Static Squat with VBX, DS – = Dynamic Squat without VBX, SS– = Static Squat without VBX

† $p<0.05$ compared to SS; * $p<0.01$ compared to VBX−

## 4.5 Discussion

The primary aim of this study was to compare the $T_m$ and HR responses to SS+ and DS– when matched for metabolic rate. There were no significant differences in $T_m$, HR and $\dot{V}O_2$ thereby confirming the hypothesis that when matched for metabolic rate $T_m$ and HR responses are similar when comparing shallow DS and SS with concurrent VBX. This suggests that the type of squat performed (DS or SS), with or without VBX will independently increase $T_m$.

For the second hypothesis, that DS+ at 50 bpm (55º knee flexion) would increase $T_m$ significantly more than DS– and SS+, was not sustained, as the addition of VBX to DS+ did not significantly elevate $T_m$ (Figure 1c). The expectation has been that when muscles are stretched more, the vibration effect on muscle activation will be greater, due to an increased sensitivity of the muscle spindles during the stretch (Kemertzis et al., 2008; Roelants et al., 2006). Furthermore, during a DS the lowering phase should elicit a larger knee angle and produce a greater muscle stretch and increase Ia-afferent stimulation and augment muscle activation (Abercromby et al., 2007a). With an increase in muscle activation from VBX, a greater energy expenditure and greater rise of $T_m$ would be expected, but this did not occur in the current study as DS+ did little to increase muscle activation and increase $T_m$ over and above that obtained with DS–. However, eliciting a larger knee angle may not increase muscle activation as other researchers have reported that when the knee angle is decreased during DS with VBX muscle activity of the leg extensors actually increased (Abercromby et al., 2007a) further, when a deeper SS (75° to 90°) was used with VBX it did not change muscle activity (Roelants et al., 2006). Therefore, it is difficult to postulate the physiological mechanism which prevented VBX from elevating $T_m$ to a greater extent than without VBX. However, DS+ may be influenced by a postural control mechanism and/or a damping response (Abercromby et al., 2007a) that prevented or blunted a rise in $T_m$.

The current proposed mechanism of VBX is based around a neurogenic potentiation involving spinal reflexes and muscle activation (Cardinale & Bosco, 2003; Rittweger et al., 2003). Given that DS is a multi-joint technique that involves a complex movement, motor and sensory patterns, DS may have altered the magnitude of the reflex response and/or the intrafusal fibre tension and Ia sensitivity during the concentric and eccentric phases of DS (Abercromby et al., 2007a), which in turn would evoke less muscular work and a lower metabolic rate. Damping of vibration results in mechanical energy and muscle activation (Wakeling et al., 2002) which is dependent on joint angle and muscle force and tension (Wakeling & Nigg, 2001). It has been shown that during VBX, head acceleration decreases when DS occurs from 10-30° of knee flexion but increases when performed at 31-35°, suggesting that the ability of the lower limbs to damp vibration is less effective as flexion angle increases (Abercromby et al., 2007b). It is plausible that during DS+ there is an optimal knee angle range which damps the vibration to

enhance muscle activation. In the current study DS was performed at 55°, thus for 45% of the duration of each DS cycle the knee flexion angle was greater than 30°, which may in part have suppressed the damping response compared to that associated with SS+. Therefore, it may be more favourable if static or single joint exercises are performed with vibration, because the full potential of the neurogenic response during VBX is more likely to be realised.

There was an interesting observation that after the first minute of DS+ and SS+, $T_m$ increased by 0.4ºC and 0.3 ºC respectively compared to 0.1ºC for DS– and SS– (Figure 1b). It is possible that the rapid and significant rise in $T_m$ might be due to vibration causing mechanical energy to be absorbed by the muscle (Ettema & Huijing, 1994), especially as there may be a short interval before the muscles accommodate or adapt to the acute imposition of vibration. Another possible explanation for this rapid rise in $T_m$ is that VBX may have caused venous dilatation of the thigh muscle, as it has been previously reported that strenuous dynamic knee-exercise increases thigh $T_m$, venous blood temperature, and venous blood flow (Gonzalez-Alonso, Quistorff, Krustrup, Bangsbo, & Saltin, 2000). Therefore, the rapid increase in $T_m$ may be due to some effect of VBX exercise or because the dynamic exercise was less strenuous (as shown by the slow $T_m$ rise in DS–).

The level of cardiovascular change in DS+ was not greater than that in DS– and SS+. However, HR increased significantly with time, with HR higher at 10 minutes with VBX than without VBX. This indicates that muscular work above isometric took place and supports earlier studies indicating that acute VBX does elicit very mild cardiovascular changes (Lythgo et al., 2009; Rittweger et al., 2000). $T_m$ never increased above $T_c$ therefore various sources of heat could have increased $T_m$, such as, an increase in muscle perfusion causing muscle to heat with warmer blood from the core; the conversion of mechanical energy to heat; or an increase in muscle ATP turnover. The increases in HR of the four interventions do not sufficiently indicate an increase in thigh muscle perfusion. However, $T_{sk}$ at the thigh may provide some indication about the heat transport in thigh muscle was altered by perfusion. In the present study $T_{sk\text{-}th}$ was greater in SS compared to DS where SS+ exhibited a greater increase compared to SS–. Therefore, the static posture with vibration may have changed muscle perfusion by increasing heat transport. Given the specific heat of muscle at 37.5°C is 3590 J/kg/°C (Gonzalez-Alonso et al., 2000) and skeletal muscle mass of the lower limb is approximately 16 kg (Shih, Wang, Heo, Wang, & Heymsfield, 2000), then an estimated 80kJ would have been required to raise $T_m$ by 1.4°C during SS+. However, Zange *et al.* (2009) found that during 3 minutes of isometric ankle plantarflexion ATP consumption did not change with either vibration ($f$=20 Hz, $A$=2mm) or without vibration. But when both arterial occlusion and vibration were applied ATP consumption increased significantly, signifying the importance of muscle perfusion in vibration exercise.

During VBX in the current study, there were several observations of skin erythema, with some participants also experiencing temporary itching, which is an innocuous side effect that normally coincides with a rise in $T_{sk}$ (Oliveri et al., 1989) and subsides after some minutes of VBX (Hazell et al., 2008; Rittweger et al., 2000). However, in the current study there were no significant changes in mean $T_{sk}$ between the four interventions, which differs from previous reports that vibration increases $T_{sk}$ (Hazell et al., 2008; Oliveri et al., 1989). The discrepancy is due to the differences in vibration regimen. For instance, Oliveri *et al*. (1989) administered localised vibration (50 Hz) whilst Hazell *et al*. (2008) utilised an intermittent protocol of 15 minutes at 45 Hz, which differs greatly from the current experimental protocol of 10 minutes continuous at 26 Hz. The moderate but significant change in $T_m$ reflected the low level of exercise intensity sustained in the current interventions, and explains why there were no significant effects on $T_c$. This was not unexpected given that a higher metabolic rate would be required to generate enough heat to raise $T_m$ beyond that of $T_c$ (Sargeant, 1987).

From the results of this study there appears to be no benefit in performing an unloaded, shallow (55º) DS+ at a tempo of 50 bpm, as $T_m$, HR, and $\dot{V}O_2$ are likely to be increased by the same amount and rate without VBX. However, the application of SS+ has advantages in sport performance when re-warming athletes after interval breaks, as it would incur a low metabolic cost and be time efficient. Likewise, SS+ may increase soft tissue temperature rapidly for exercise-associated rehabilitation purposes where dynamic exercise cannot be performed. Lastly, future research should focus on the damping response of VBX by investigating different cadences and depths of DS.

In summary, $T_m$ increased in all four interventions after 10 minutes, verifying that the exercise mode, of, DS+, DS–, SS+, SS– causes an increase in muscle heat production. When $\dot{V}O_2$ was matched between DS– and SS+, $T_m$ increase and HR response were comparable. However, for DS+ $T_m$, HR, $\dot{V}O_2$ were not significantly greater compared to DS– and SS+ which may be influenced by a postural control mechanism and/or a damping response (Abercromby et al., 2007a).

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# Chapter 5 – Comparing the Physiologic Effects of Acute Lower-Body Vibration Exercise in Young and Older People

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## 5.1 Abstract

This study examined the acute physiological effects of acute vibration exercise (VBX) exercise in young and older people. Every participant performed nine conditions, consisting of: without vibration (VBX−) and VBX+ at 30 Hz, and three loads corresponding to, 1) no load (0% Body mass [BM]), 2) load of 20% BM and 3) load of 40% BM. The Jendrassik voluntary contraction was also performed without VBX and VBX at 30 Hz with no load and 20% BM. Twelve (6 men and 6 women) healthy young people (mean age 21.5 yrs) and twelve (6 men and 6 women healthy older people) (mean age 69.2 yrs) participated in the study. Physical Activity Questionnaire, anthropometric measures, counter movement jump, and isometric maximal voluntary contraction with Jendrassik manoeuvre, were assessed in both groups. Oxygen uptake, blood pressure (BP), heart rate (HR), and rating of perceived exertion (RPE) were recorded during VBX and load conditions. Both vibration and load were associated with an increase ($p< 0.001$) in oxygen uptake for older and young groups. VBX+ elicited an equivalent of a 0.35 MET increase in $\dot{V}O_2$, with additional loads of 20% and 40% BM increased $\dot{V}O_2$ by 0.8 and 1.2 METS respectively. Additionally, there was an interaction effect of vibration and group where the VBX-related $\dot{V}O_2$ increase was less in the old compared to the young. Both vibration and load caused an increase in HR, BP, and RPE ($p< 0.001$ for all); however, there were no significant group differences between young and older groups. The Jendrassik manoeuvre elicited an increase in oxygen uptake by 27.6% for the old and 33% for the young group ($p<0.001$); however, there was no significant difference between groups.

Vibration and additional load significantly increased oxygen uptake for old and young people in a similar way (1.2 ml/kg/min) but the elderly responded to an increasing load with a lesser augmentation in oxygen uptake than the young. Moreover, the oxygen uptake in both the older and young also increased when the Jendrassik manoeuvre was superimposed with vibration and load. Therefore, VBX is probably an insufficient stimulus to improve cardiovascular fitness and it should not replace more traditional forms of aerobic exercise training for young and old.

*Keywords* Jendrassik manoeuvre; metabolic rate; oxygen uptake; ageing, frailty

## 5.2 Introduction

Exercise is generally advocated as a countermeasure to offset age-related frailty and to enhance mobility and well-being. It has been widely documented that the natural ageing process is associated with reduced muscular and cardiovascular function, bone loss and increased body fat storage (Daley & Spinks, 2000), all of which contribute to a decline in functional performance (Margaria, Aghemo, & Rovelli, 1966). Over time, in combination with a sedentary lifestyle, further deterioration may lead to a greater reduction in mobility, impaired balance and a higher incidence of falls (Skelton, 2001). Furthermore, the decline in muscle function not only involves a loss of muscle strength but also of muscle power. Margaria *et al*. (1966) were the first to report a decline in maximum muscular power with age, and found from 20 to 70 years of age, muscle power is reduced by about a half, with poor muscle power being a predictor of hospitalisation, falls, and fracture (Guralnik, Ferrucci, Simonsick, Salive, & Wallace, 1995). Whilst the fact of physiologic decline may not be avoided, it can be mitigated by training, even at very old age (Fiatarone et al., 1990). However, factors such as time, convenience, poor compliance through dementia, and poor postural control after a stroke often preclude older people from engaging in physical activity (Hannan et al., 2004). An exercise modality that is convenient, time efficient, and has the benefits of conventional weight bearing exercise would be appealing to this group.

One such modality, known as vibration exercise (VBX), has recently received some attention as a regime to overcome these obstacles whilst producing favourable outcomes (Ahlborg, Andersson, & Julin, 2006; Cochrane & Stannard, 2005; Roelants et al., 2004). VBX requires specialised equipment; however, it can be performed in the convenience of the home and is readily available from commercial companies. Manufactured devices such as a hand-held dumbbell, standing and seated oscillating platforms, produce the vibration. These devices deliver sinusoidal vibrations to the body at a frequency of 5-45 Hz. As little as 6-10 minutes of VBX per day, three times a week for 6-8 weeks has shown to increase balance and gait (Bautmans et al., 2005; Bruyere et al., 2005; Runge et al., 2000) improve quality of life (Bruyere et al., 2005), and improve exercise compliance (Hannan et al., 2004). Additionally, longer duration (6-12 months) VBX studies in postmenopausal women have documented increases in bone mineral density of the hip (Verschueren et al., 2004) and spine (Rubin et al., 2004). Currently there are several large studies being conducted to substantiate the clinical benefits on a large scale.

The mechanism of VBX has yet to be elucidated; however, it has been proposed that VBX involves monosynaptic reflexes which are induced by the stretch-shortening action in the muscles which act over the joints in which the vibration is being absorbed (Rittweger et al., 2003). Electromyographic activity has been shown to increase during VBX (Bosco et al., 1999;

Cardinale & Lim, 2003), and it has been purported that VBX elicits muscular activity through evoking sufficient muscular work to raise whole-body oxygen uptake (Rittweger et al., 2001), which is supported by a linear increase in oxygen uptake with an increasing vibration frequency (Rittweger et al., 2002).

Importantly, the acute physiological responses to VBX have been investigated only in the young, but not in older people. One might speculate that the responses to VBX are mitigated in older people. Research suggests that ageing and disuse attenuates motoneuron excitability and causes structural changes to the muscle spindle (Jozsa, Kvist, Kannus, & Jarvinen, 1988; Scaglioni et al., 2002); therefore, the muscle spindle in older individuals may be less sensitive to the vibration due to the fibre composition and reflex deterioration that occurs with natural ageing (Lexell, 1997; Liu, Eriksson, Thornell, & Pedrosa-Domellof, 2005). Thus, there is reason to assume that the stimulus for muscular work, either mechanically direct, or via the stretch-reflex, will be reduced and the metabolic requirement will be decreased in older individuals. Therefore, it was hypothesised that the increase in oxygen uptake, evoked by VBX, will be lower in older people than in the young.

As an attempt to shed some light upon the age-related differences in VBX-related oxygen uptake, the effect of Jendrassik manoeuvre was assessed. This established test requires the person to clasp both hands and pull them apart, and it is thought to facilitate the stretch reflex by pre-synaptic activation. Doing so typically enhances the reflex amplitude by a factor of three after brief sustained contraction (Gregory, Wood, & Proske, 2001). As explained above, it is generally assumed that VBX elicits muscle contractions via the stretch reflex. Therefore, as a secondary hypothesis to be tested in this study, it was expected the metabolic rate in both the older and young participants would increase when the Jendrassik manoeuvre was superimposed with VBX and load.

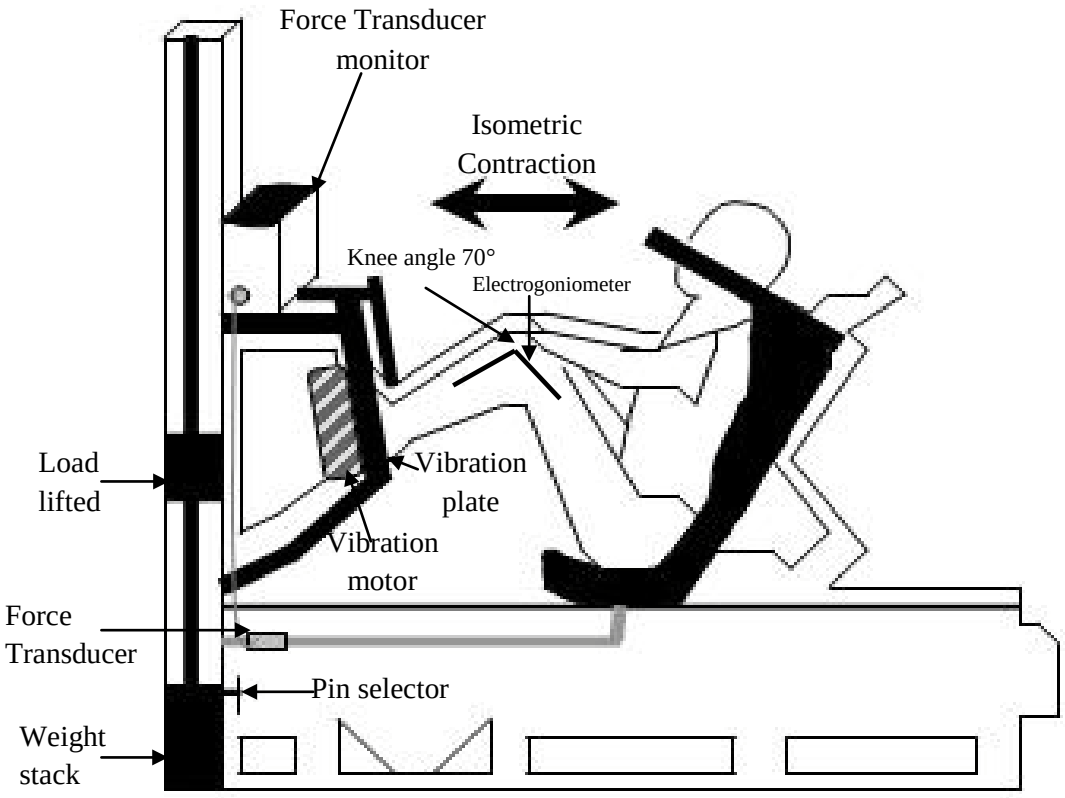
## 5.3 Methods

### *Vibration and Load*

VBX in standing position involves the musculature from the whole body including the musculature of the trunk and of the shoulder girdle. Therefore, to avoid any involvement of the arm and shoulder musculature in the vibration stimulus, which would influence the Jendrassik manoeuvre, a seated version for VBX was constructed. To this purpose, a prototype vibration machine consisting of a motorised horizontal leg press with a pin-weighted plate stack (Technogym, Gambettola, Italy), was fitted with two electrically powered 0.15 KW Motovibratori (COMB, Corlo, Italy) to the rear of the foot plate of the leg press machine (Figure 1). Both units had the capacity to operate at 0-3600rpm or 0-60 Hz at an amplitude of 0.5-1.0 mm of vibration in the Z-plane.

Pilot work with this equipment found that the optimal frequency and amplitude to elicit maximal increases in oxygen uptake was a frequency of 30 Hz and 1.0mm amplitude (peak acceleration of 35.2 m/s$^2$ or 3.6*g*). This concurs with a previous report that squatting on a vibration platform at 30 Hz elicited the highest EMG response in the vastus lateralis muscle (Cardinale & Lim, 2003), and thus 1.0mm amplitude at 30 Hz was selected for this study. The foot plate was fixed at an 80° incline and the participants were instructed to sit in the leg press machine with the seat adjusted to a 60° decline, with both socked feet placed at shoulder width apart placed on a small rubberised foot rest of the vibration plate. Participants adjusted their legs to a knee angle of 70° (full extension = 0°) which was verified with an electrogoniometer (Figure 1).

**Figure 1** Schematic diagram of the prototype vibration leg press machine. The foot plate angle was fixed at an 80° incline and the seat was adjusted to a 60° decline.



The loading regime involved the above foot placement and knee angle for no load (0% body mass [BM], 20% and 40% of BM. For no load (0% BM) the participants were required to place their legs on the foot-rest without any load or additional leg force. For loads of 20% and 40% BM a pin selector was inserted into the appropriate weight stack plate of the leg press machine (Figure 1), which was equivalent to 20% and 40% of the participant's body mass. To ensure that no additional leg force was contributing to the various loads a force transducer was attached to the undercarriage of the leg press seat (Figure 1) and connected to a display unit via an oscilloscope (Hameg, 20MHz, HM 203.7, Frankfurt, Germany). This provided visual feedback to participants to prevent unwarranted increases in isometric leg force which was continually monitored by the experimenter.

### *Participants*

Twelve healthy young (21.5 ± 2.8 years, mean ± standard deviation) and twelve healthy older (69.2 ± 7.2 years) people matched for sex (6 men and 6 women), weight, and physical activity provided written consent to volunteer for the study. The study protocol was approved by the University Ethics Committee. Each participant underwent verbal health and medical screening prior to the study to exclude VBX contraindications of non-consolidated fractures, bone tumours, herniated disks, deep vein thrombosis, aortic aneurysm, metal implants (leg or vertebral column), diabetes with polyneuropathy and pregnancy (young women group only). None of the studied participants reported any signs or symptoms which warranted exclusion from participation in the present study.

### *Study Design*

Every participant performed the nine conditions in a randomised order. The protocol consisted without vibration (VBX−) and with vibration (VBX+) at 30 Hz, and three loads corresponding to, 1) no load, 0% body mass (BM), where the participant placed their feet on a small rubberised foot-rest that was attached to the vibration plate 2) load of 20% of the participants BM, and 3) load of 40% of the participants BM (Table 1). The rationale for selecting a load of 20% and 40% BM, was based from earlier pilot work that fatigue was minimised as illustrated by low blood lactate levels in comparison to larger loads. The Jendrassik voluntary contraction (JVC, see below) was performed without vibration and with vibration at 30 Hz and loads consisting of no load (0% BM) and a mass of 20% BM (Table 1).

**Table 1** Variable settings of the nine experimental conditions

| Condition | Load | Jendrassik |
|---|---|---|
| VBX− | 0% BM | |
| VBX− | 0% BM | JVC |
| VBX− | 20% BM | |
| VBX− | 40% BM | |
| VBX+ | 0% BM | |
| VBX+ | 0% BM | JVC |
| VBX+ | 20% BM | |
| VBX+ | 20% BM | JVC |
| VBX+ | 40% BM | |

VBX− = Without Vibration; VBX+ = With Vibration exercise

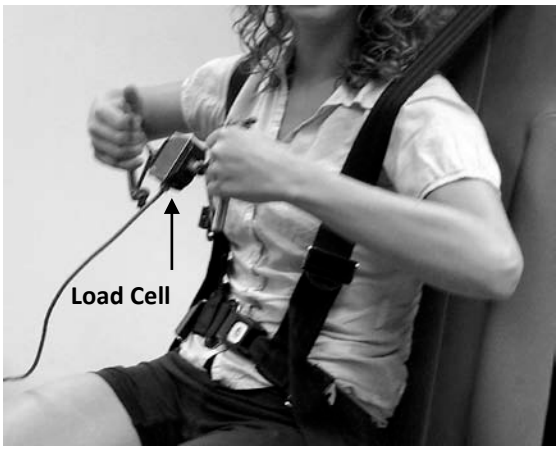
BM = Body Mass; 0% BM = No Load; 20% BM = load 20% of BM; 40% BM = load 40% of BM;

JVC= 10% of Jendrassik maximum voluntary contraction

Each condition was 4 minutes in duration and separated by 30s of rest. The rationale for the 4 minute duration was selected on the basis from earlier pilot work that during VBX oxygen uptake produced a steady state during this time. To prevent unwanted leg movement the participant's knees were restrained by an adjustable elastic band. The participants were asked to place both hands on their knees, and to breathe normally during the course of each 4 minute period. A warm-up was prohibited prior to the start of each testing day to reduce the possibility of influencing the outcome of the study.

The Jendrassik contraction (Figure 2) involved pulling with both arms a dual handle load cell (FN 3030, FGP Instrumentation, Italy) that was connected to a computer (Macintosh powerbook, G3) and acquisition system (Acknowledge v 3.7, Biopac Systems, Santa Barbara, California, USA). First, the maximal voluntary isometric force was identified by two maximal contractions separated by 15s of rest. The greatest maximal contraction was recorded (Table 2), and 10% of this value were then used for the JVC condition. From earlier pilot work levels larger than 10% JVC did not result in a noticeable further reflex augmentation. Therefore, in order to avoid any possible fatigue effect, it was decided to use 10%.

**Figure 2** The Jendrassik contraction involved pulling a load cell at 10% MVC.




Each participant visited the laboratory on two separate occasions with a maximum of five conditions being performed with at least 24 hours rest separating testing days. The participants were asked to refrain from undertaking any vigorous activity 24 hours prior to the testing; participants performed the protocol at the same time of day. The anthropometric measurements, counter-movement jump and Jendrassik's maximal voluntary contraction were conducted prior to the commencement of the main study.

*Physical Activity Questionnaire and Anthropometric measures*

All participants completed a written Physical Activity Questionnaire (PAQ) (Frey, Berg, Grathwohl, & Keul, 1999) to ascertain the metabolic units spent per week undertaking sport and physical activity (Table 2). Circumference measurements, skinfold thickness and limb lengths were used to calculate the thigh (TMV), calf (CMV) and total (ΣMV) muscle volume (Table 2) from the volumetric method devised by Jones and Pearson (1969). The anatomical landmarks of the ankle, knee and hip were identified and marked. These locations provided reference points for the circumferences, skinfold thickness and limb lengths.

Girth measurements were taken with a non-stretch anthropometric tape and limb lengths were measured by a Harpenden anthropometer (Crosswell, Crymych, Wales, UK). Both girth and limb lengths were taken at the subgluteal fold, thigh, knee, calf and ankle locations. Skinfold thickness procedures were conducted in accordance to International Society for the Advancement of Kinanthropometry (ISAK) protocols. A calibrated skinfold calliper (Harpenden, Baty International, England) was used to measure subcutaneous adipose tissue at the medial and lateral calf, anterior and posterior thigh.

### *Counter Movement Vertical Jump*

Each participant performed five vertical counter movement jumps performed on a Kistler force plate (9281B) at a sampling rate of 500Hz, with each jump separated by 60s rest. The participants were instructed to remove their shoes and place their feet in the middle of the plate with both hands on their hips to avoid any unnecessary movement of the arms. From the ground reaction force recordings, jump height and peak power (Table 2) were assessed by the method devised by Davies & Rennie (1968). The highest jump height and peak power for each participant was recorded for statistical analysis.

### *Oxygen Uptake, Heart Rate, Blood Pressure & RPE*

Oxygen uptake was continually assessed using the portable K4b$^2$ breath by breath pulmonary gas exchange system (Cosmed, Rome, Italy). Before each test the K4b$^2$ was calibrated with ambient air ($O_2$ 20.93%, $CO_2$ 0.03%) and a known gas concentration mixture ($O_2$ 16.00%, $CO_2$ 5.00%). The turbine was calibrated using a known 3 litre volume syringe (Cosmed, Rome, Italy).

A heart rate strap (Polar Electro Oy, Kempele, Finland) was attached to the participant's chest and telemetrically interfaced with the K4b$^2$ system. $\dot{V}O_2$ steady state was attained in all conditions between 2-4 minutes with the mean value of $\dot{V}O_2$ in ml/kg/min being used for further analyses. Heart Rate (HR) was recorded in conjunction with every breath by breath analysis of the K4b$^2$ with the mean HR being analysed during the 2-4 minute interval. Blood pressure was assessed at 2 and 4 minutes by an electronic blood pressure monitor (Ohio 2100, Madison, Wisconsin, USA), and ratings of perceived exertion (RPE) (Borg, 1998) being assessed at the 2 and 4 minute interval.

**Table 2** Mean (±SD) physical characteristics of men and women participants

| Physical Characteristics | Age (yrs) | Height (m) | Weight (kg) | PAQ Sport Score | PAQ Total Score | TMV (l) | CMV (l) | ΣMV (l) | JH (cm) | PJP (W/kg) | JMVC (kg) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Older – Men (n=6) | 70.5‡ (3.6) | 1.73* (0.05) | 77.8* (11.7) | 4.0 (3.5) | 12.9 (5.6) † | 4.0 (0.8) | 1.5* (0.3) | 5.5* (1.0) | 25.7* (1.5) † | 30.9* (3.8) † | 21.8* (8.2) † |
| Older – Women (n=6) | 67.8‡ (9.4) | 1.63 (0.09) | 60.8 (5.0) | 3.8 (3.1) | 14.7 (2.3) | 3.6 (1.0) | 1.1 (0.4) | 4.7 (1.4) | 19.4 (7.3) | 26.0 (7.1) | 17.3 (6.0) |
| Young – Men (n=6) | 21.3 (0.5) | 1.78* (0.06) | 76.7* (15.2) | 11.8 (9.4) | 19.3 (10.0) | 4.3 (1.2) | 1.6* (0.5) | 5.9* (1.5) | 37.6* (6.4) | 43.1* (7.3) | 41.4* (11.2) |
| Young – Women (n=6) | 21.5 (3.5) | 1.69 (0.07) | 63.3 (9.0) | 8.7 (6.2) | 17.1 (6.2) | 3.5 (1.0) | 1.0 (0.4) | 4.5 (0.9) | 33.2 (4.8) | 37.8 (4.8) | 33.3 (5.4) |

PAQ – Physical Activity Questionnaire; TMV – Thigh Muscle Volume; CMV – Calf Muscle Volume; ΣMV- Total Muscle Volume; JH -Jump Height; PJP - Peak Jump Power; JMVC – Jendrassik Maximum Voluntary Contraction

* Statistically significant ($p < 0.05$) compared to women

† Statistically significant ($p < 0.05$) group interaction effect of older (men & women) vs young (men & women)

‡ Statistically significant ($p < 0.05$) compared to young

*Statistical Analyses*

All analyses were performed using SPSS for Windows Version 14 (Chicago, IL, USA). The dependent variables of the physical characteristics were analysed by ANOVA. A repeated measures 2 (VBX− & VBX+) x 3 (0% BM, 20% BW, 40% BM) mixed (group, old vs young) ANOVA with post-hoc simple contrasts were performed to examine the effects of vibration, load, HR, systolic, diastolic pressure, RPE, and Jendrassik. For multiple comparisons significance, levels were adjusted using Bonferroni's rule. The level of significance was set at $p<0.05$.

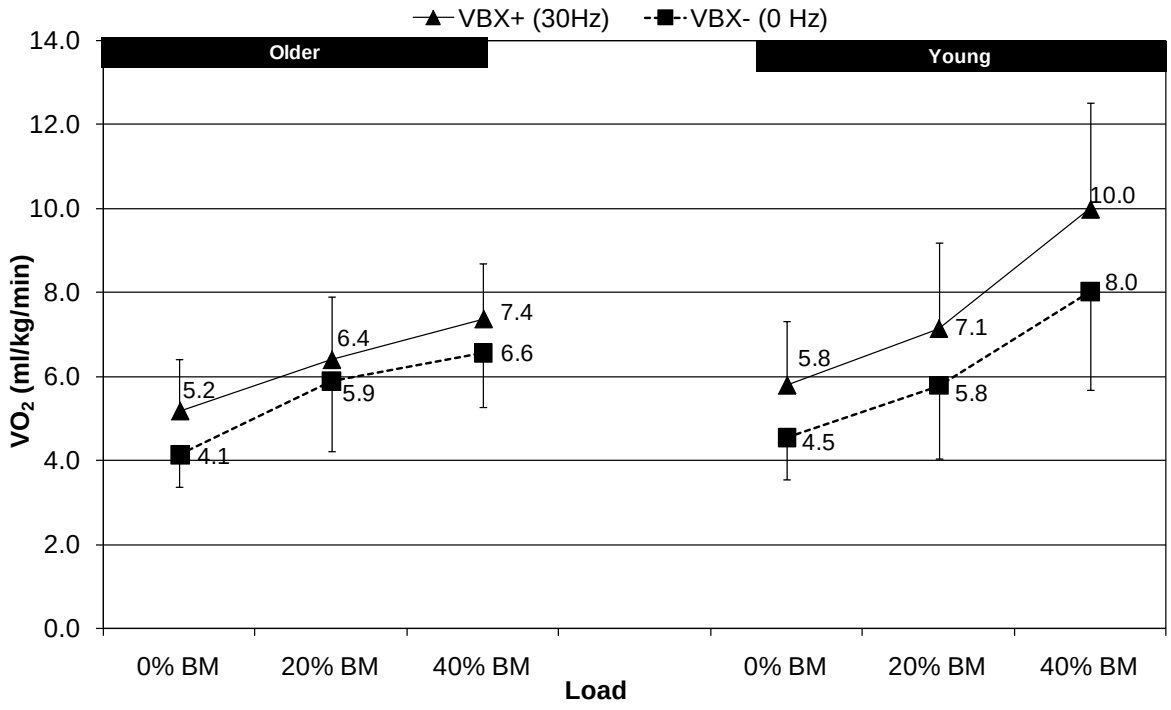
## 5.4 Results

Both vibration and load enhanced ($p<0.001$) oxygen uptake for older and young groups (Figure 3), such that VBX+ increased $\dot{V}O_2$ by 19.7% compared to no vibration. Furthermore, loads of 20% and 40% BM increased $\dot{V}O_2$ by 26.7% and 62.9% in comparison to no load (0% BM). There was a significant but small interaction effect of vibration and group ($P=0.045$), where the VBX-related $\dot{V}O_2$ increase was lower in the old compared to the young. There was a significant load and group interaction ($p<0.01$) such that the younger group increased oxygen uptake to a greater extent with additional load. This difference became significantly detectable at 20% and 40% BM loads. Both VBX and load produced a significant ($p<0.001$) increase in HR, with an interaction between load and group ($p< 0.05$), implying that the young group exhibited a greater increase in HR in response to load compared to the older group. Systolic pressure increased significantly ($p<0.001$) with the corresponding increase in loads (0% BM; 20% BM; 40% BM), however there was no significant effect of vibration influencing systolic pressure (Figure 4).

Both VBX+ and load produced a significant increase ($p<0.001$) in diastolic pressure, with a significant interaction between vibration and time ($p< 0.05$) of vibration (30 Hz) producing a higher diastolic pressure at 4 minutes compared to 2 minutes (Figure 3). RPE increased significantly ($p<0.001$) with VBX and load, with higher RPE's being recorded at 4 minutes compared to 2 minutes (Figure 4). However, no significant effects were found between young and older groups for systolic, diastolic pressure, and RPE.

During VBX, JVC produced a significant increase in oxygen uptake of 25.3% ($p<0.001$) compared without JVC (Table 3), however there was no statistical difference between groups. RPE, HR, systolic and diastolic pressure continued to rise with time during the vibration with JVC compared to without JVC. RPE and systolic pressure were significantly higher ($p<0.001$) at 4 minutes compared to 2 minutes. However, no statistical differences were found between young and older groups. Moreover, there were no significant differences between gender for $\dot{V}O_2$, cardiovascular and JVC.

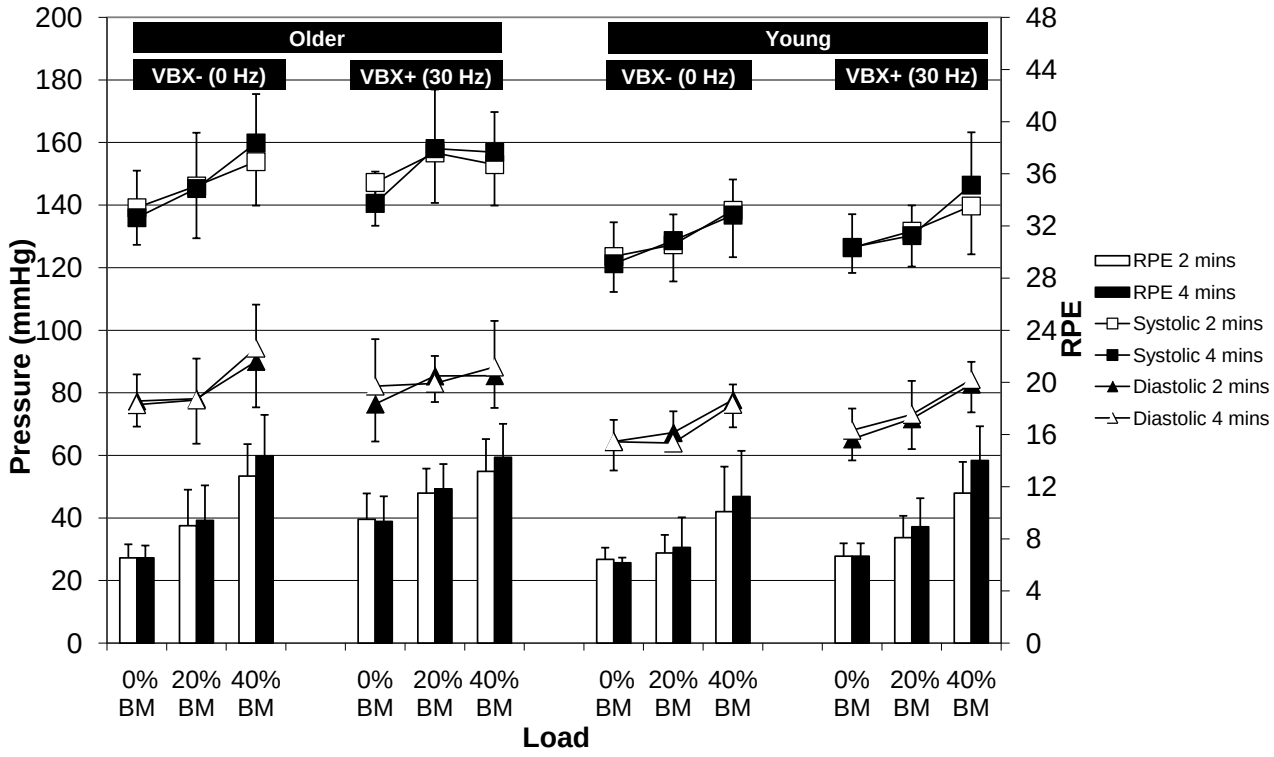
**Figure 3** Mean (± SD) oxygen uptake (ml/kg/min) of vibration frequency and loads of older and young groups




VBX− = Without vibration exercise; VBX+ = With vibration exercise BM = Body Mass; 0% BM = No Load; 20% BM = load 20% of BM; 40% BM = load 40% of BM

Vibration and load significantly (P<0.001) increased oxygen uptake. There was a significant load x group interaction effect (P<0.001) between the additional loads of 20% and 40% BM of increased oxygen uptake and there was a small significant interaction of vibration and group (P=0.045).

**Figure 4** Mean (± SD) of systolic, diastolic pressure, and RPE at 2 and 4 mins during the various vibration frequency and loads for the older and young groups.




BM = Body Mass; VBX− = Without vibration exercise; VBX+ = With vibration exercise; 0% BM = No Load; 20% BM = load 20% of BM; 40% BM = load 40% of BM

Increased loads produced a significant rise ($p<0.001$) in systolic pressure.

Diastolic pressure increased significantly ($p<0.001$) with vibration, and a larger load (40% BM). Vibration and load produced a significant increase in RPE.

**Table 3** Mean (± SD) oxygen uptake (ml/kg/min) of older and young participants during the Jendrassik conditions of vibration and loads

| Group | Jendrassik | Vibration & Load | | |
|---|---|---|---|---|
| | | VBX− 0% BM | VBX+ 0% BM | VBX+ 20% BM |
| Older | - | 4.1 ±0.8 | 5.2 ±1.2 | 6.4 ±1.5 |
| | + | 5.5 ±1.1* | 6.8 ±1.6* | 7.8 ±1.3* |
| Young | - | 4.5 ±1.0 | 5.8 ±1.5 | 7.1 ±2.0 |
| | + | 6.4 ±2.4* | 7.5 ±2.4* | 9.3 ±2.5* |

VBX− = Without vibration exercise; VBX+ = With vibration exercise; BM = Body Mass; 0% BM = no load; 20% BM = load 20% of BM; - = Without Jendrassik contraction; + = With Jendrassik contraction

* The Jendrassik produced a significant ($P < 0.001$) increase in oxygen uptake compared to without the Jendrassik, however there was no difference between groups.

## 5.5 Discussion

The main aim of this study was to investigate whether the aerobic metabolism responses to VBX and additional load of the older population were comparable to those obtained in the young. The findings suggest that both in young and older people oxygen uptake were significantly enhanced with vibration and additional load. In qualitative terms, VBX was found to affect oxygen uptake in older and younger people in a very similar way. There was only one exception to that rule, in that the increase in oxygen uptake per unit of increment in load was lower in the older than the young. In quantitative terms, however, the metabolic response to vibration appeared to be slightly lower than in the young. One might therefore argue that older individuals are less responsive to VBX in terms of aerobic metabolism.

To quantify the increased VBX-related $\dot{V}O_2$ of the older group and its capability to increase aerobic capacity, an estimation of $\dot{V}O_2$ max percentage can be calculated. Given that 5.2 ml/kg/min was elicited by VBX and that maximum oxygen uptakes of 21 ml/kg/min for 68-70 year olds are commonly reported (Johnson et al., 2000), the estimate of VBX-related $\dot{V}O_2$ would be equivalent to 24% of $\dot{V}O_2$ max. However, this would be insufficient to increase aerobic capacity with at least 40% $\dot{V}O_2$ max being required to elicit the appropriate physiological changes (Evans, 1999) and confirms that the $\dot{V}O_2$ elicited by VBX is small, nevertheless only one specific amplitude and frequency was used in the current study.

The candidate mechanism of VBX is thought to activate the stretch reflex response that causes muscle to contract (Issurin & Tenenbaum, 1999; Rittweger et al., 2001). This would imply that every single vibration wave elicits a muscle contraction that involves mechanical power converted metabolically. The results of the present study seemingly contradict this idea. Whilst Jendrassik contraction did elicit an increase in oxygen uptake in all conditions, there was no interaction observed between Jendrassik contraction and vibration. In fact, Jendrassik contraction increased oxygen uptake quite uniformly, namely by approximately 1.5 ml/kg/min and 2 ml/kg/min in the older and younger participants, respectively, and thus enhancing oxygen uptake quite independently of the vibration modality.

From the current study, it is inconclusive as to whether the observed response of older versus younger people to vibration is due to differences in reflex activity. However, it is known that the H-reflex is reduced in older individuals due to a decrease in alpha motoneuron excitability (Scaglioni et al., 2002). Furthermore, the muscle spindle which is central to the vibration mechanism undergoes age related changes in myosin heavy chain expression making it less sensitive to changes in muscle length and affects its ability to fully activate the motoneurons (Liu et al., 2005). Likewise, morphological changes of thickening spindle capsule and diminished periaxial space occurs from disuse and atrophy (Jozsa et al., 1988). Therefore, it is plausible that

an ageing muscle spindle does not have the capacity to activate Ia afferents, which inadvertently affects the excitatory response of the alpha motorneurons (Swash & Fox, 1972). Moreover, the lack of Ia afferent or spinal pre-synaptic inhibitory response from the ageing central nervous system may have caused a less effective excitatory response on the alpha motoneuron, which was not capable of eliciting a greater level of oxygen uptake.

The body posture (seated versus upright) and the type of body movement (static versus dynamic) may influence oxygen uptake. For example, Rittweger *et al*. (2001) has reported that standing on a side-alternating vibration plate elicits an oxygen uptake of 10.2 ml/kg/min compared to 5.8 ml/kg/min in a seated position of the present study. However, squatting at a tempo of 3s up and 3s down on an oscillating plate further increases oxygen uptake to 14 ml/kg/min (Rittweger et al., 2001). For the current study the seated position was preferred, so the loading of 20% and 40% BM of the lower limb was able to be conducted in a safe and appropriate manner. Furthermore, a seated position prevented the arm and shoulder muscles from being vibrated which could have influenced Jendrassik's manoeuvre. Moreover, in the current study the older participants lead a very active lifestyle which may not been a true representation of this population.

In the present study, the young adults showed a 1.3 ml/kg/min increase in oxygen uptake at 30 Hz of VBX, which at a similar frequency, contrasts to the 3.5 ml/kg/min increase finding by Rittweger *et al*. (2002). The discrepancy can be explained by the different vibration machines used. In the present study the vibration was administered by a plate moving uniformly up and down in the Z plane, compared to Rittweger's *et al*. (2002) study where the vibration was elicited by an oscillating teeterboard that moved about a central axis. Additionally, in the present study the participants were seated for vibration compared to standing (Rittweger et al., 2002). The reason for the different approach in this study was that, as explained above, it was important to clearly separate upper and lower body muscle contractions. However, even though the paradigm applied in the present was different from the machines that are commercially available for vibration exercise, there is little reason to question the principle finding of this study, namely that elderly people respond to the vibration in similar way as younger people.

When contemplating the application of vibration exercise in older people, it is also important to consider the effects upon the cardiovascular system. In that sense, the other physiological variables of HR, blood pressure, RPE that were investigated in this study are of interest. As expected, they showed increases with vibration and also with additional loads. The findings are in line with the view that cardiovascular changes by vibration are comparatively small and in relation to muscle activity (Rittweger et al., 2000). The risk of VBX being a cardiovascular hazard thus appears to be very small in an elderly population. Therefore, VBX exercise for older people may provide some advantages; given the low level of cardiovascular changes, VBX could be performed by those suffering from heart conditions. Additionally, VBX may

improve proprioception and can be performed at home to increase exercise compliance (Fontana et al., 2005; Hannan et al., 2004).

In conclusion, vibration and additional load significantly increased oxygen uptake for old and young people in a similar way but the elderly responded to an increasing load with a lesser augmentation in oxygen uptake than the young. Likewise, the metabolic rate in both the older and young increased when the Jendrassik manoeuvre was superimposed with vibration and load. But there was no interaction effect observed between Jendrassik contraction and vibration, which is unlikely to potentiate VBX-induced stretch reflex. However, the physiological mechanisms behind the role of the spinal reflex in VBX warrants further research.

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# Chapter 6 – The Effect of Acute Lower-Body Vibration Exercise on Muscle-Tendon Complex Length, Muscle Contractile Tissue Displacement, and EMG Activity

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## 6.1 Abstract

It has been suggested that vibration exercise (VBX) causes small changes in muscle length, but it has never been proven that it actually occurs. This was an observational study to determine whether acute VBX would result in muscle lengthening. It was hypothesised that acute VBX would increase EMG activity concurrently with measurable changes in muscle contractile length.

Nine healthy males performed two conditions on a side-alternating vibration machine for 15s at 0 Hz (resting) and 6 Hz at a set knee angle of 18º. Muscle tendon complex (MTC) length, contractile tissue displacement (CD) of the medial gastrocnemius (MG) muscle and EMG of soleus, tibialis anterior and vastus lateralis muscles were measured. At 6 Hz the MG MTC amplitude (375 μm) was significantly greater ($p<0.05$) compared to 0 Hz (35μm). The MG CD amplitude at 6 Hz (176μm) was significantly greater ($p<0.01$) compared to 0 Hz (4μm). Significant increases ($p<0.05$) in EMG modulation were found for all muscles during the 6 Hz compared to 0 Hz condition. The major finding was that approximately 50% of the elongation occurred within the muscle itself and was associated with preceding changes in EMG which indicates muscle lengthening may be a prerequisite for eliciting probable stretch reflexes.

In conclusion, there is a temporal association between EMG activity and muscle contractile tissue displacement where low frequency VBX results in small muscle length changes and increases muscle activation.

*Keywords* Muscle length, contractile element, isometric contraction

## 6.2 Introduction

Vibration exercise (VBX) is becoming very popular in the health, rehabilitation and exercise sectors (Cheung et al., 2007; Cochrane & Stannard, 2005; Rittweger et al., 2002; Schuhfried et al., 2005; van Nes et al., 2006), but the scientific enquiry of VBX lags behind its application. Acute and chronic VBX studies where vibration is applied to the lower limbs have been shown to increase vertical jump height (Cochrane & Stannard, 2005) and measures of lower body strength (Delecluse et al., 2003; Mileva et al., 2006). A range of VBX frequencies have been successful in eliciting various responses. For example, VBX frequencies of greater than 25 Hz have shown to increase muscular performance (Cochrane & Stannard, 2005; Delecluse et al., 2003; van Nes et al., 2006) while low frequency (6 Hz) VBX have been observed to increase cerebral oxygenation (Maikala & Bhambhani, 2008); a change which is strongly linked to greater neuronal activation (Villringer & Chance, 1997).

The augmented responses of VBX have been attributed to the 'tonic vibration reflex' (TVR) (De Gail, Lance, & Neilson, 1966); an effect based upon the stretch reflex response (Burke et al., 1976; Roll et al., 1989) where vibration applied to the tendon activates primary muscle spindle afferents, stimulating the alpha motoneurons and eventually contraction of the extrafusal muscle fibres (Liddell & Sherrington, 1924). Support for involvement of the stretch reflex is provided by electromyography (EMG) studies of augmented neuromuscular activity during vibration (Abercromby et al., 2007; Bosco et al., 1999a; Cardinale & Lim, 2003; Roelants et al., 2006).

It seems sensible to assume involvement of stretch reflexes in VBX, but direct evidence for this is not available. Indeed, whereas TVR requires high frequency (80-100 Hz) stimulation applied direct to the tendon (Eklund & Hagbarth, 1966), VBX is delivered through the soles of the feet, at a lower frequency (25-40 Hz). Secondly, recent evidence shows that superimposing the Jendrassik manoeuvre upon VBX enhanced metabolic rate (Cochrane et al., 2008 [Chapter 5]), but there were no significant difference between VBX and no VBX, and is thus unlikely to potentiate VBX-induced stretch reflex. Clearly, the physiological mechanisms behind the role of the stretch reflex in VBX require further investigation.

Activation of the stretch reflex requires that small changes in muscle length occur (Bosco et al., 1999b), however, to date there is no evidence of this during VBX. If VBX does involve the stretch reflex, changes in muscle length must occur in a fixed temporal relationship with EMG activity. Yet, this has been difficult to show because measuring muscle length changes has been challenging. Recently however, an ultrasound tracking technique has been devised to assessing changes in muscle contractile tissue displacement to a high degree of reliability and validity (Loram, Maganaris, & Lakie, 2006).

Therefore, the main aim of this study was to qualitatively investigate whether low frequency VBX would result in muscle lengthening by employing the ultrasound tracking technique. It was hypothesised that acute VBX at 6 Hz would increase EMG activity concurrently with measurable changes in muscle contractile length compared to resting levels.

## 6.3 Methods

### *Participants*

Nine healthy males with a mean age (± SD) of 33.4 ± 7.5 yr; body mass 77.4 ± 13.9 kg, height 1.80 ± 0.1 m, and having an active lifestyle with a non-specific training background volunteered to participate in the study. Written consent was obtained and ethical approval was granted by the local University Ethics Committee.

### *Rationale*

Exercise and health VBX protocols have often prescribed frequencies between 20-45 Hz (Cheung et al., 2007; Cochrane & Stannard, 2005; van Nes et al., 2006), however, in everyday life people are exposed to low level VBX frequencies of 3 to 6 Hz (Wilder, Woodworth, Frymoyer, & Pope, 1982). Furthermore, recent evidence has reported that 6 Hz VBX increases cerebral oxygenation and cardiovascular responses compared to without VBX (Maikala & Bhambhani, 2008) implying there is a greater neuronal activation (Villringer & Chance, 1997) but it is unknown weather low VBX elicits muscle elongation. Moreover, in extensive pilot work VBX frequencies of 8, 10 and 12 Hz were trialled where the ultrasound tracking methodology was unsuccessful. This may have been due to a limitation in the tracking algorithm or some other unknown reason. From the pilot work it was found that 15s of data collection was convenient and provided sufficient data to describe the extent of the muscle shortening and lengthening. A longer duration would not have altered the extent of shortening/lengthening and would have placed a very high demand on the tracking software.

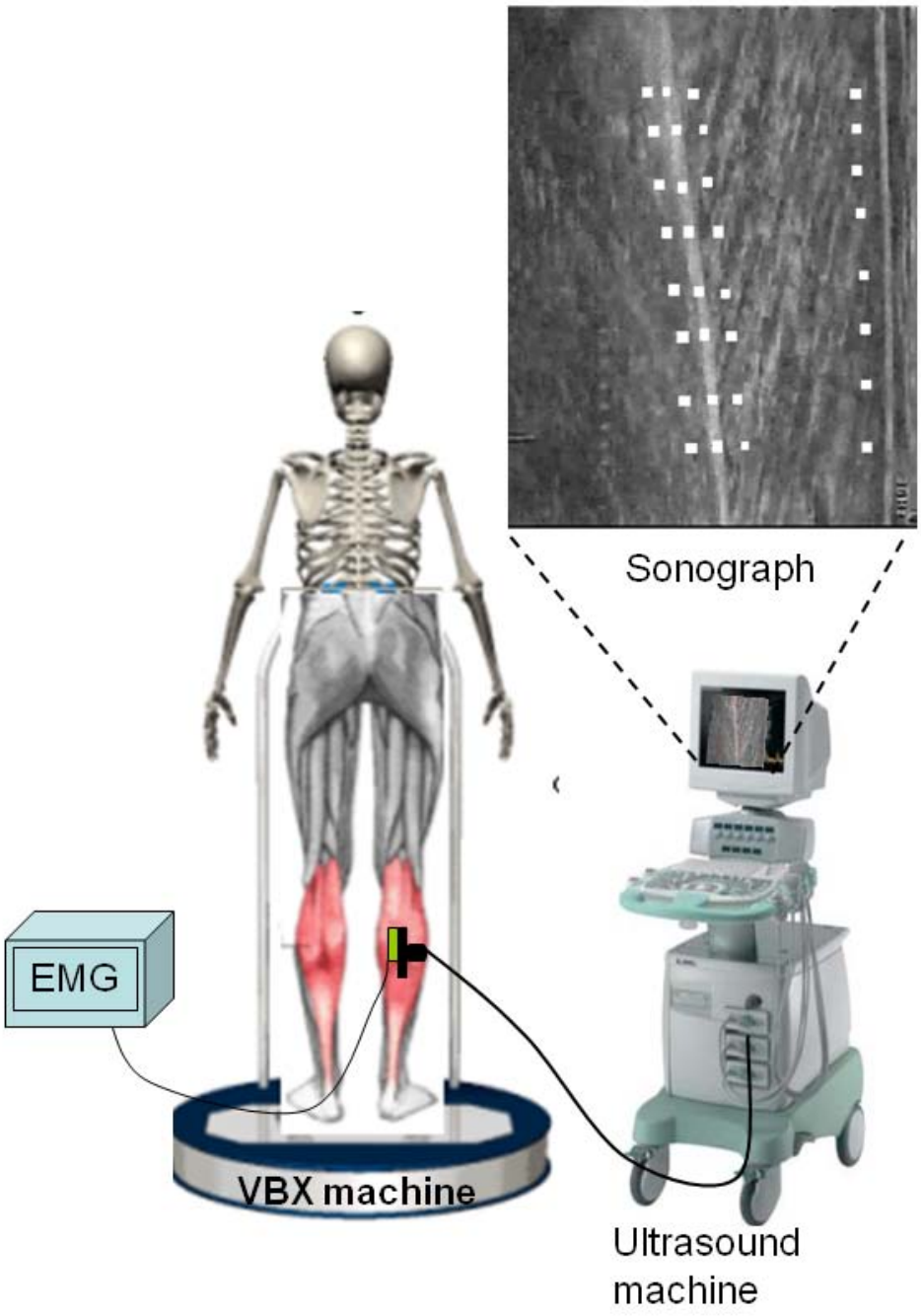
### *Study Design*

To assess the change in contractile tissue displacement from acute VBX, every participant undertook 15s of VBX (6 Hz) at a pre-determined vibration amplitude (3.1mm) and body position (see below), and 15s in the same position but without vibration (0 Hz).

*Vibration Exercise*

This was performed on a commercial machine (Galileo Fitness, Novotec, Pforzheim, Germany), which has a teetering board that produces vertical sinusoidal vibration to the body (Figure 1). The participants placed their bare feet 5cm either side of the central axis which equated to a vibration amplitude (peak-to-peak) of 3.1mm and a peak acceleration of 0.6*g*. This was measured by an accelerometer (ADXL05EM-1 Analog Devices, Norwood, MA United States) that was fixed to the outer edge of the vibration platform in line with the right large toe. The participants maintained an erect body stance with 18º knee flexion (knee fully extended = 0º) at 0 Hz (resting) and 6 Hz, and to ensure the starting position was the same between the two conditions a manual goniometer was used to standardise the knee angle. From earlier pilot work a range of squat depths were kinematically analysed with EMG to ascertain the optimal knee angle. It was found that 18º of knee flexion elicited the greatest EMG response during 6 Hz VBX.

**Figure 1** Experimental setup of EMG, vibration machine and sonograph of the right medial gastrocnemius muscle with marker placements that were used to calculate contractile tissue displacement



## *Data Acquisition*

### *Kinematic data*

The kinematic data were acquired by a 10 camera infra-red three-dimensional optical motion capture system (VICON 612, VICON motion systems, Oxford, UK) that sampled at 250 Hz. Seventeen fluorescent spherical (14mm diameter) markers were placed directly on the skin at right and left side body locations and secured with double-sided tape on the following bony landmarks: large toe over the second metatarsal head, mid-foot side of the equinus break between fore-foot and mid-foot, calcaneus at the same height above the plantar surface of the foot as the toe marker, medial and lateral malleolus of the ankle, along an imaginary line through the transmalleolar axis. For the knee, hip and anterior superior iliac, markers were placed on the medial and lateral epicondyles of the tibia, on the head of the greater trochanter, and on the spines of the anterior superior iliac (pelvis). For the sacrum, a wand marker was placed on the skin mid-way between the posterior superior iliac spines. The above locations were used according to the standard "plug-in-gait" model of the VICON system and Bodybuilder software (v 3.6, VICON motion systems, Oxford, UK) for segment modelling and calculation of lower limb kinematics. Joint angle and acceleration of the right knee and ankle angles were calculated from the Body Builder model, as was the distance from the right ankle to the knee.

### *Muscle Tendon Complex (MTC) Length*

From the ankle and knee joint angles of the kinematic data, the medial gastrocnemius (MG) MTC length change (muscle plus free tendon and aponeurosis at both distal and proximal ends) was calculated from the equation of Menegaldo *et al*. (2004). In brief, MTC = $a_1$+ $a_2$ KA + $a_3$AA + $a_4$ SA + $a_5$ $KA^2$ + $a_6$ $AA^2$ + $a_7$ $SA^2$+ $a_8$ $KA^3$ + $a_9$ $AA^3$ + $a_{10}$ $SA^3$, where $a_1$ $a_2$ $a_3$ $a_4$ $a_5$ $a_6$ $a_7$ $a_8$ $a_9$ $a_{10}$ are constants, KA = knee angle, AA = Ankle angle, SA = subtalar angle (this was constant at 0˚).

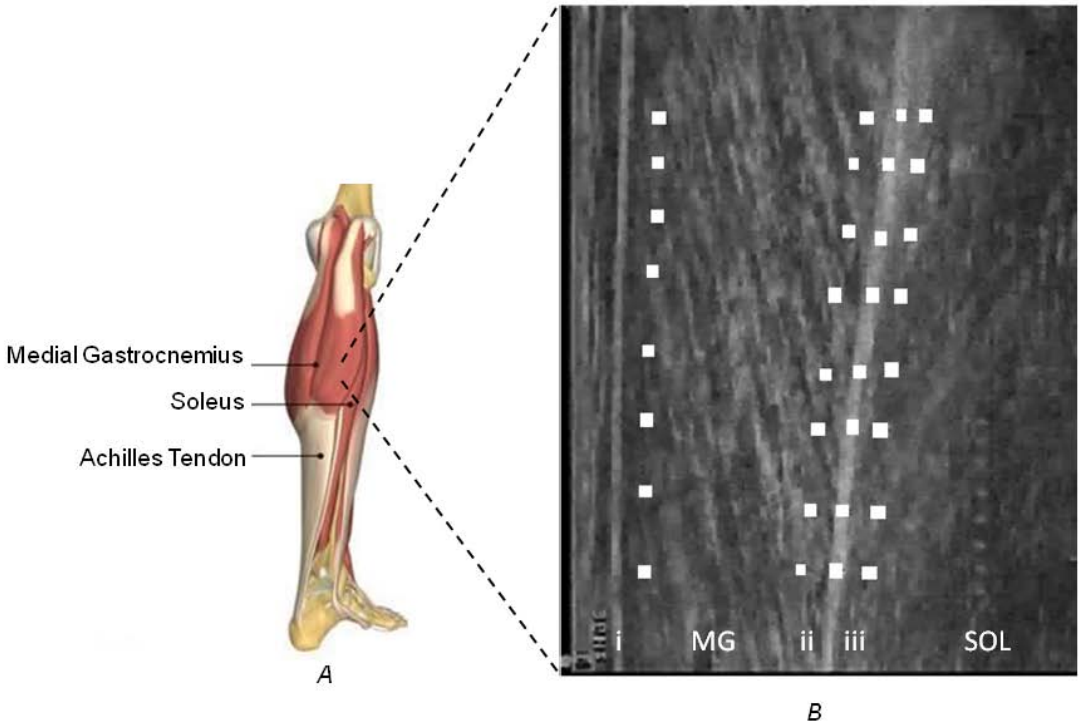
### *Contractile tissue displacement (CD)*

The CD refers to the series contractile portion of the muscle-tendon unit, by measuring the longitudinal changes in contractile tissue length along the line of the muscle-tendon unit (Loram et al., 2006). Note, this quantity is not the same as fascicle length since the displacement is measured longitudinally along the muscle rather than in the direction of the fascicles. CD was assessed using a linear 60mm, 7.5 MH ultrasound probe (Aloka SSD-5000, Tokyo, Japan) which was attached to a custom-built polystyrene mould and secured by velcro straps in the midsagittal plane of the right medial gastrocnemius (Figure 1). The probe was set for multi-beam, 30 Hz, at a depth of 58mm. The ultrasound images were recorded to a DVD

and converted into an audio video interleave (avi) codec file by a computer editing software (Adobe Premiere Pro, San Jose, California, USA). Each condition contained 15s of imaging frames (30 frames/sec, pixel dimension 121 x 133μm) that were saved for subsequent analysis. Changes in muscle contractile tissue displacement were computed offline by the procedure previously described by Loram *et al*. (2006). In brief, on each avi file eight markers were placed on the distal and proximal aponeurosis of the medial gastrocnemius image (Figure 2). These markers were square sided (length of 30–50 pixels, 6–10mm) and a tracking algorithm was used to measure the change in position of each marker for the duration of the avi file. In essence the algorithm measured the position of each marker relative to its position from the first frame of the avi file and used a spatial, 2D cross correlation to calculate the movement of the markers.

This 2D cross correlation was performed using MATLAB (MathWorks, Massachusetts, USA) software which calculated integer shifts in pixel position and used 2D quadratic interpolation to provide sub-pixel estimation of the position of peak correlation (Loram et al., 2006). For the contractile tissue, changes in length were calculated relative to the initial value. The distal and proximal aponeurosis MG were visually identified as landmarks to standardise the marker placement between the vibration and resting conditions.

**Figure 2** (A) Calf muscles of the lower leg; (B) Sonograph of the MG and SOL; i, proximal aponeurosis of MG; ii, distal aponeurosis of MG; distal aponeurosis of SOL. ii and iii are morphologically distinct and can move relative to each other. Hence, muscle markers were placed either side of the central aponeurosis



### *Electromyography*

The electromyographic (EMG) activity was recorded by the Bagnoli EMG system (Delsys, Boston, MA, USA) from the right leg of the medial gastrocnemius (MG), soleus (SOL), tibialis anterior (TA), and vastus lateralis (VL) muscles. Each electrode was housed in a waterproof polyurethane plastic case and contained two pure silver contacts measuring 10mm in length, 1 mm in diameter, and spaced 10mm apart. Each electrode was connected by an electrical lead to the Bagnoli-16 amplifier which had a pre-determined bandwidth filter of 20-450 Hz with a gain of 1000. The skin of the electrode site was prepared by shaving, gentle abrasion using a gel-based product (Nuprep, D.O. Weaver, USA), and cleansed with an alcohol tissue pad. Specially designed doubled-sided tape (Delsys, Boston, MA, USA) was applied to the electrode and attached to the muscle site. Each interface electrode was oriented in the direction of the muscle fibre, but the MG electrode was placed proximal to the ultrasound probe with a reference electrode being attached to skin over the patella. The EMG signal from the Bagnoli amplifier was captured by Notebook computer, PowerLab 8/30 and chart software (version 4) (ADInstruments GmbH, Germany) at a sampling rate of 2000 Hz for 15s. The raw EMG signal was analysed off-line.

### *Data Analysis*

An external trigger was used to synchronize the VICON, EMG and ultrasound machines which embedded an electronic marker into the data files that were subsequently stored off-line on desktop computers and analysed using a custom-built computer software system (CaptainC v 1.0, Rittweger, Manchester Metropolitan University, 2007). This software was designed to compute peri-event histograms (PEH) between the triggered accelerometer, EMG and other analogous signals. First, the accelerometer signal was low pass filtered (cut-off 20 Hz) and trigger-events were generated upon every upstroke through 0 (from negative to positive). This trigger event corresponded to the lowest position of the vibration platform. For the 0 Hz (control) condition, a synthetic 6 Hz sinusoidal oscillation was generated as a sham event. Following this, sweeps of the analogous signals were averaged to yield the mean response in relation to the trigger event. During the testing, and also before analysis, raw EMG signals were carefully checked for vibration-related artefacts. Only recordings without any such artefacts (see Figure 3 & 4) were considered for analysis. However, in order to preclude any bias of results, EMG data were fully rectified and high pass filtered before computation of PEHs (cut-off 20 Hz). Finally, all PEHs computed from EMG signals (but not the other signals) were low pass filtered at 20 Hz in order to provide robust results.

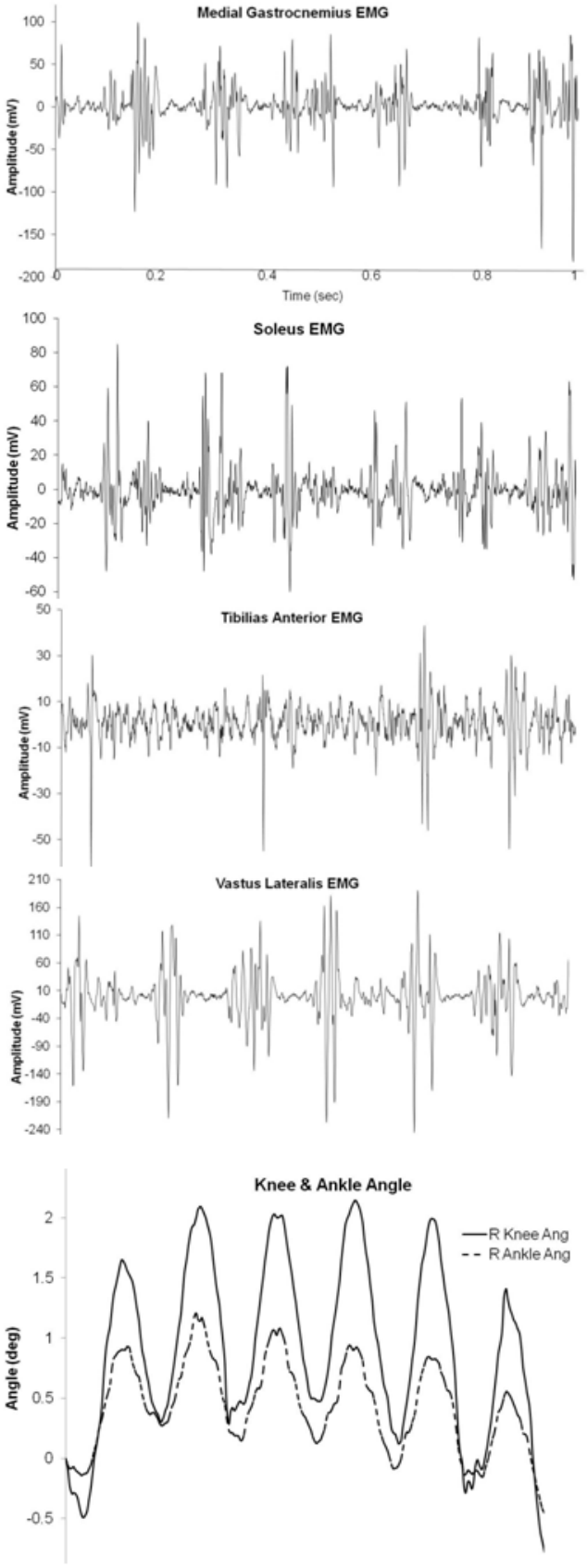



**Figure 3** A typical response of raw EMG, (MG, SOL, TA, VL) knee and ankle angle during 6 Hz VBX.

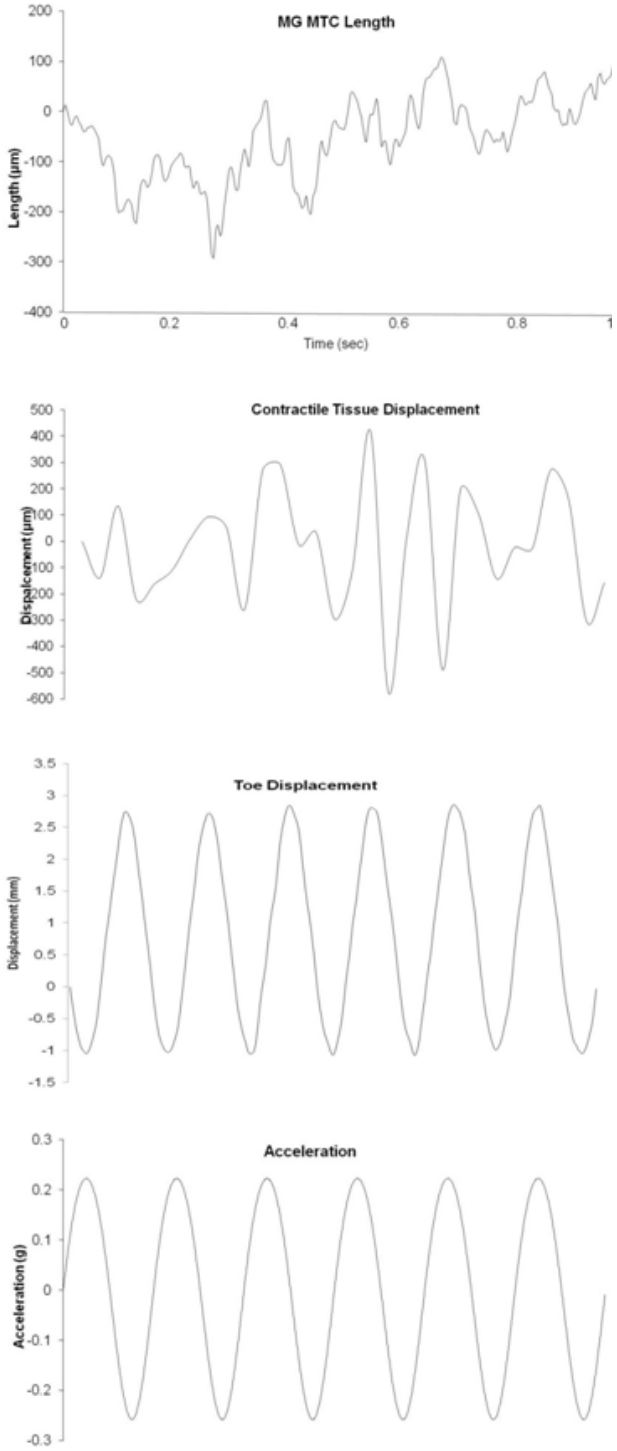



**Figure 4** A typical response of raw MG MTC, contractile length, toe displacement, and acceleration during 6 Hz VBX.

From each EMG PEH, the following information was extracted: mean value and percent modulation, (both explained in Figure 5). In brief, figure 5 provides an overview of information extracted from peri event histograms (PEH) of signal y. The percent modulation was computed as 100 * A / (A+B), where A was the area enclosed by the PEH curve and the horizontal line through the PEH's minimum, and A+B is the total area under the PEH curve. For PEHs from the other signals, only the modulation amplitude was assessed. As mentioned previously CaptainC software was designed to compute peri-event histograms a typical (n=1) analysis of 0 Hz & 6 Hz VBX for rectified EMG, (SOL, MG TA, VL) knee ankle angle is shown in Figure 6 with MG MTC, CD, toe displacement, and acceleration shown in Figure 7.

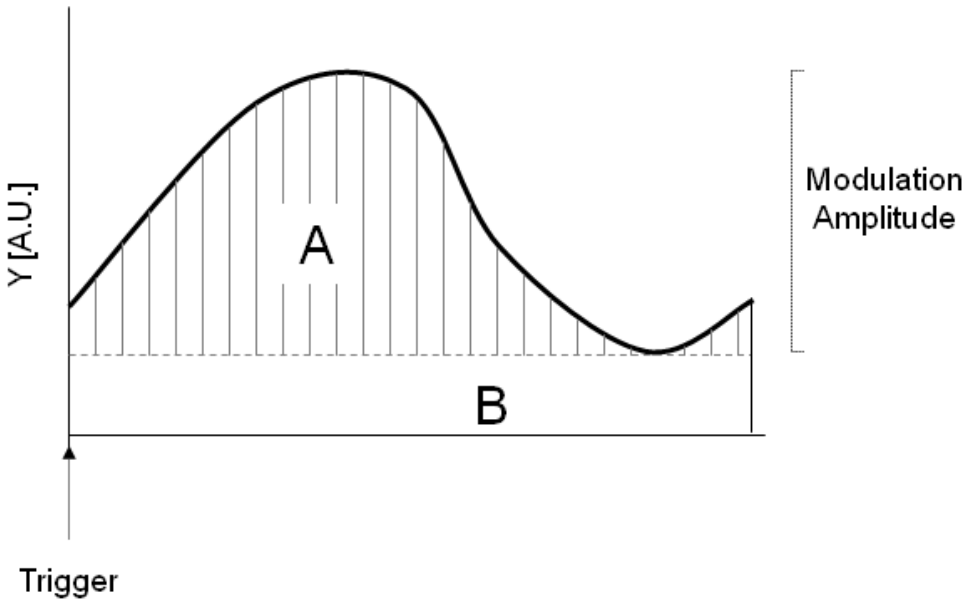



**Figure 5** Overview of information extracted from peri event histograms (PEH) of signal y.

Percent modulation was computed as 100 * A / (A+B), where A is the area enclosed by the PEH curve and the horizontal line through the PEH's minimum, and A+B is the total area under the PEH curve.

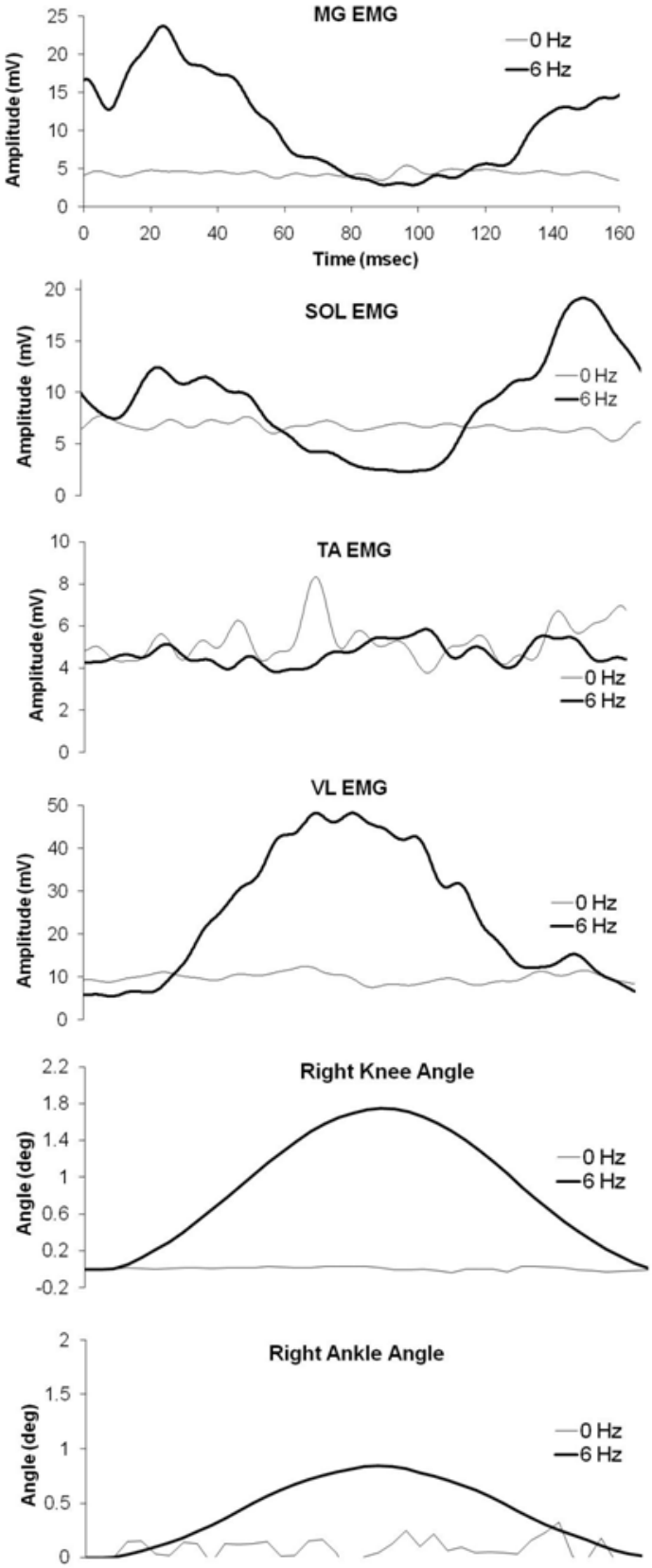



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**Figure 6** A typical (n=1) analysis of 0 Hz and 6 Hz VBX from CaptainC software for rectified EMG, (SOL, MG TA, VL) knee and ankle angle.

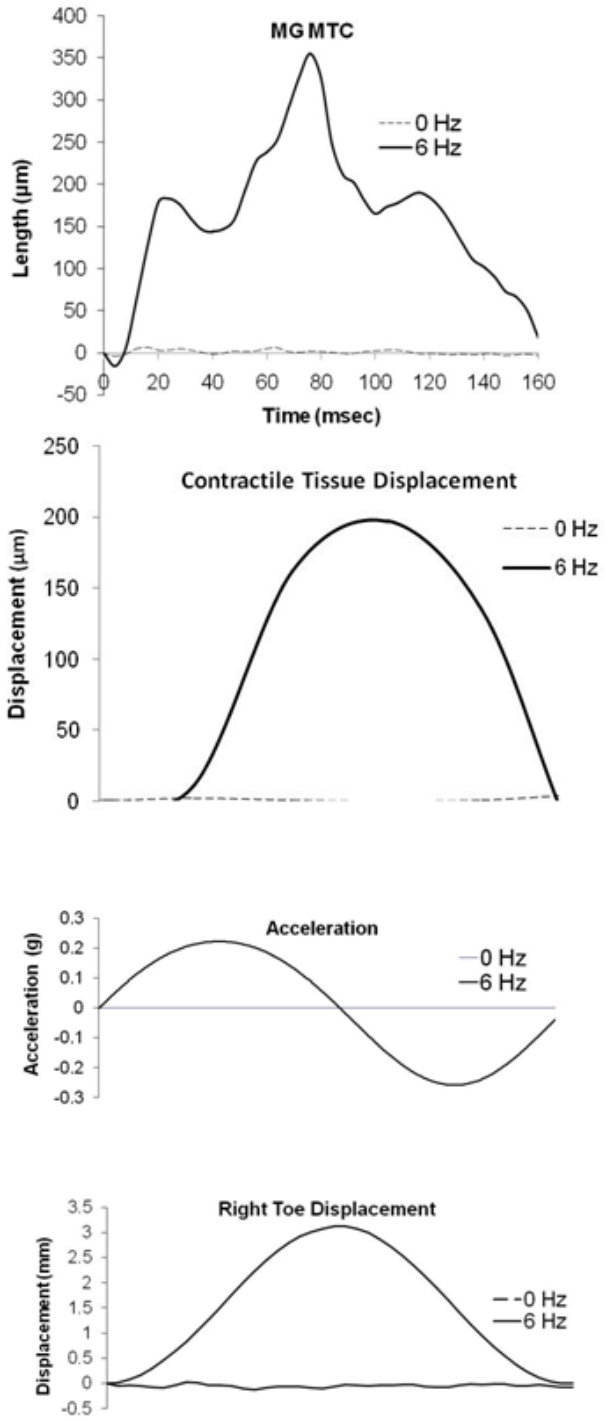


**Figure 7** A typical (n=1) analysis of 0 Hz and 6 Hz VBX from CaptainC software for MG MTC, contractile displacement, toe displacement, and acceleration

*Statistical Analyses*

Student's paired *t*-test was used to analyse the dependent variables (mean right knee and ankle angle, right knee and ankle amplitude, MTC and CD amplitude, mean length of MTC and CD, percent modulation of MG, SOL, TA, VL) between 0 Hz and 6 Hz. All statistical analyses were performed using a specialized statistical software package (SPSS for Windows Version 14, Chicago, IL, USA) and the level of significance was set at $p<0.05$.

## 6.4 Results

The peak-to-peak acceleration of the vibration plate at 6 Hz was 0.6 ± 0.01*g* and the right toe was displaced 3.1 ± 0.5mm. Mean values for knee and ankle angles were comparable between 0 vs 6 Hz, demonstrating that body position was the same during the vibration and control conditions. Conversely, PEH amplitude was significantly larger ($p<0.05$) for knee and ankle angle during the 6 Hz condition compared to 0 Hz (Table 1). Accordingly, the mean medial gastrocnemius (MG) MTC length was the same between the two conditions (472.9 ± 2.4 mm for 0 Hz, 471.5 ± 1.8 mm for 6 Hz) but the MG MTC amplitude was significantly ($p<0.05$) greater during 6 Hz (Table 1).

The MG CD PEH amplitude (Table 1) during 6 Hz (176μm) was significantly greater ($p<0.01$) compared to 0 Hz (4.2μm), confirming that vibration was providing the stimulus for the changes in contractile length. At 6 Hz the amplitude of MG MTC was 375μm and at 0 Hz the amplitude of MG MTC was 35μm.

The mean rectified EMG amplitude of the VL was significantly ($p<0.05$) larger at 6 Hz (12.8 mV ± 7.6) compared to 0 Hz (8.3 mV ± 2.6, see Table 2). Likewise, the MG showed a larger ($p=0.053$) change in EMG amplitude at 6 Hz (8.4 mV ± 6.0) compared to 0 Hz (4.1 mV ± 2.9). In contrast, there was no significant difference in EMG mean rectified amplitude between 0 and 6 Hz for the TA and SOL. However, significant increases ($p<0.05$) in EMG modulation were found for all muscles during the 6 Hz compared to 0 Hz condition (Table 2). For instance the percentage modulation for the 0 Hz MG condition was 10.5% compared to 41.9% for the 6 Hz condition. Likewise, the soleus EMG modulation was 11.5% (0 Hz) compared to 43% (6 Hz).

**Table 1** Mean (± SD) of knee and ankle angle, MTC, CD of medial gastrocnemius for 0 and 6 Hz.

| Vibration Frequency | R Knee Angle (deg) | R Knee Amplitude (deg) | R Ankle Angle (deg) | R Ankle Amplitude (deg) | MG MTC Amplitude (μm) | MG CD Amplitude (μm) |
|---|---|---|---|---|---|---|
| 0 Hz | 15.0 (6.0) | 0.1 (0.1) | 86.1 (3.8) | 0.1 (0.2) | 35.4 (49) | 4.2 (4.4) |
| 6 Hz | 18.4 (6.5) | 1.3† (0.6) | 86.8 (1.1) | 0.6† (0.3) | 374.7* (283) | 175.6† (64.4) |

* $p \leq 0.05$; † $p<0.01$ compared to 0 Hz

R – Right; MG – Medial Gastrocnemius; MTC – Muscle Tendon Complex;
CD – Contractile Length

**Table 2** Mean (± SD) of EMG amplitude and percent modulation of MG, SOL, TA, and VL muscles

| | **MG** | | **SOL** | | **TA** | | **VL** | |
|---|---|---|---|---|---|---|---|---|
| Vibration Frequency | 0 Hz | 6 Hz | 0 Hz | 6 Hz | 0 Hz | 6 Hz | 0 Hz | 6 Hz |
| EMG Mean Amplitude (mV) | 4.1 (2.9) | 8.4# (6.0) | 9.0 (4.5) | 10.0 (4.5) | 4.6 (3.4) | 8.9 (7.8) | 8.3 (2.6) | 12.8* (7.0) |
| EMG Modulation (%) | 10.5 (4.6) | 41.9† (23.2) | 11.5 (4.4) | 43.0† (18.2) | 8.9 (8.2) | 21.0* (17.0) | 11.2 (5.5) | 31.2† (17.2) |

# $p= 0.053$; * $p \leq 0.05$; † $p<0.01$ compared to 0 Hz

MG - medial gastrocnemius; SOL - soleus; TA - tibialis anterior; VL - vastus lateralis;

## 6.5 Discussion

Whilst former studies have reported enhanced neuromuscular activation during or immediately after VBX (Abercromby et al., 2007; Bosco et al., 2000; Cardinale & Lim, 2003; Roelants et al., 2006), this is the first study to establish the temporal association of EMG activity and muscle contractile tissue displacement during a vibration stimulus. Changes in joint angles, muscle tendon complex length, and muscle contractile tissue displacement all increase as an effect of acute 6 Hz VBX. In line with these changes, EMG activity of the VL and MG muscles increased, as reported by others (Abercromby et al., 2007; Roelants et al., 2006). In the current study the EMG mean amplitude of MG increased 105% and VL increased 54% over resting values, which differs to previous reports (Abercromby et al., 2007; Bosco et al., 1999a; Cardinale & Lim, 2003). The likely explanation for this is the different protocols used, including the vibration frequency, body stance, and the type of EMG analysis performed.

Additionally, in the present study the SOL and TA were not consistently activated during VBX. Contrary to this, Abercromby *et al*. (2007) reported that at 18º knee flexion TA was stimulated during 30 Hz VBX. This dissonance may be due to the different peak acceleration experienced; a very low peak acceleration (0.6*g*) was used as opposed to 15*g* utilized by Abercromby *et al*. (2007). Additionally, there may be an optimal ankle angle whereby vibration activates TA and SOL. Previously it has been reported that the SOL EMG response from an Achilles tendon tap was maximised between 10-15º plantar flexion(Lin, Brown, & Walsh, 1997), which differs to 4º plantar flexion used in the current study. Moreover, the sensitivity of the TA and SOL to vibration may be dependent on the amount of pre-stretch and pre-activation that the muscle undertakes (Roelants et al., 2006). However, it is unclear what influence muscle pre-activation and pre-stretching has on VBX performance as it remains equivocal (Bosco et al., 1999a; Cardinale & Lim, 2003; Roelants et al., 2006).

In the present study EMG modulation increased significantly at 6 Hz for MG, SOL, TA, VL compared to the control (0 Hz). EMG modulation was defined as the area under the EMG curve in relation to the power of the signal. This supports the hypothesis that EMG modulation does occur at low frequency VBX and confirms that, as a result of vibration, changes in MTC length also occur, which may indicate that the stretch reflex pathway has a role in facilitating this response. However, if the assumption is true that VBX stimulates MG or SOL via the stretch reflex then TA would have been suppressed via reciprocal inhibition. On the contrary, TA was activated and not inhibited. A likely explanation is that TA EMG and SOL EMG were 180º phase reversed and therefore antagonists, hence the antagonist inhibition from SOL and GM alpha motoneurons was not strong or long enough to suppress the TA stretch reflexes.

The vibration plate produced a peak-to-peak acceleration of 0.6*g* which was enough to elicit changes in MTC, CD and EMG. The SOL and MG EMG preceded MTC and CD by approximately 110ms which is consistent with the interpretation that some muscle shortening and lengthening occurred as a result of modulations in muscle activation. In quantitative terms during vibration the MG MTC elongated to 375μm while CD lengthened by 176μm (1.45 pixel * 121μm = 176μm), indicating that nearly 50% of elongation occurred in the muscle itself. This confirms the hypothesis that acute VBX at 6 Hz increases EMG activity coincident with temporal change in muscle contractile length.

Although no direct measurement of the reflex response was taken, the contractile tissue displacement is thought to be associated with changes in neural activation which is influenced by stretch reflexes and responds with the contractile portion shortening to neural stimulation (Loram et al., 2006). Indirectly, this may support the idea that a neurogenic response is present in low vibration frequency. However, there is a possibility that vibration of the muscle may initiate orthodromic stimulation to the axonal branches where stimulation of one branch antidromically causes a depolarisation down another branch (Stalberg & Trontelj, 1970). However, further more specific work is needed to investigate whether this occurs, and if so in isolation or in conjunction with the proposed stretch reflex mechanism.

A limitation of the current study was the selection of 6 Hz vibration frequency this was due to the capacity of the ultrasound machine which had a maximum capability of capturing at 30 frames per second. Further research is required to investigate if similar findings can be attained at the higher vibration frequencies and amplitudes most commonly employed in VBX prescription. Also, it is essential to confirm the presumption that VBX causes an excitatory neuromuscular response by directly examining the stretch reflex pathways and the contractile properties of muscle.

In conclusion, acute 6 Hz VBX produced a temporal association between EMG activity and muscle contractile tissue displacement with 50% of the elongation occurring within the muscle itself. This indicates that muscle lengthening may be a prerequisite to eliciting probable stretch reflexes and provides evidence that muscles damp vibration (Wakeling et al., 2002). However, the measurement of stretch reflex and muscle twitch potentiation need to be elucidated before it confirmed that VBX operates via a spinal reflex mechanism.

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# Chapter 7 – Acute Lower-Body Vibration Exercise Elicits Post-Activation Potentiation

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## 7.1 Abstract

Vibration exercise (VBX) leads to a rapid increase in intra-muscular temperature and enhances muscle power. The power-enhancing effects by VBX can, at least in part, be explained by intra-muscular temperature. However, this does not exclude possible neural effects of VBX occurring at the spinal level. The aim of this study was to examine if muscle twitch and patellar reflex properties were simultaneously potentiated from an acute bout of VBX in a static squat position.

Six male and six female athletes performed three interventions for 5 minutes, static squat with VBX (VBX+, 26 Hz), static squat without VBX (VBX−) and stationary cycling (CYCL, 70W). Transcutaneous muscle stimulation consisting of a single 200μs pulse and three patellar tendon taps were administered prior to and then 90s, 5, 10 minutes post-intervention. Ninety seconds after VBX+ muscle twitch peak force (PF) and rate of force development (RFD) were significantly higher ($p<0.01$) compared to VBX− and CYCL. However the patellar tendon reflex was not potentiated. An acute continuous bout of VBX caused a post-activation potentiation (PAP) of muscle twitch potentiation (TP) compared to VBX− and CYCL indicating that a greater myogenic response was evident compared to a neural-mediated effect of a reflex potentiation (RP).

*Keywords* Twitch, muscle contractile properties, stretch reflex, electrical stimulation, rate of force development

## 7.2 Introduction

Acute vertical sinusoidal vibration exercise (VBX), eliciting rapid eccentric/concentric of the leg extensors (Cardinale & Bosco, 2003; Rittweger et al., 2003; Rittweger et al., 2001), improves performance of these muscles in the short-term (Cochrane & Stannard, 2005; Cochrane, Stannard, Sargeant, & Rittweger 2008b [chapter 3] Cochrane, Stannard, Walmsley, & Firth, 2008c [chapter 2]; Torvinen et al., 2002). This transient effect is thought to be mediated by a rapid reflex-mediated stretch-shortening (Cardinale & Bosco, 2003; Rittweger et al., 2003; Rittweger et al., 2001) likely to involve the tonic vibration reflex (TVR), which stimulates the muscle spindles (Cardinale & Bosco, 2003; Rittweger et al., 2001). Practically, VBX application leads to enhanced anaerobic power (Cochrane & Stannard, 2005; Cochrane et al., 2008c [chapter 2]). Previous work has also demonstrated that VBX leads to a rapid increase in intra-muscular temperature (Cochrane et al., 2008b [chapter 3]). Intra-muscular temperature in itself enhances muscle power (de Ruiter & de Haan, 2000). Therefore, it seems that the power-enhancing effects by VBX can, at least in part, be explained by intra-muscular temperature. However, this does not exclude a possible neurogenic potentiation from VBX, but limited research has been performed on assessing such spinal effects, with varying results and a lack of comparison to other modalities (Hopkins et al., 2008; Melnyk et al., 2008; Rittweger et al., 2003). For example, Rittweger *et al*. (2003) reported an enhancement of the patellar tendon stretch reflex following VBX in combination with squatting exercise to exhaustion, whereas Hopkins et al., (2008) found no such effect after 5 x 1 minute intermittent vibration exposure of VBX (26 Hz). The results that VBX does not enhance spinal reflex excitability is supported by the finding that superimposing the Jendrassik manoeuvre upon VBX failed to enhance metabolic rate (Cochrane et al., 2008a [chapter 5]). Nevertheless, stretch reflexes seem to be active during VBX, as previous research has reported a temporal association between EMG activity and muscle contractility; however ,VBX was only performed at a very low frequency (6 Hz) (Cochrane, Loram, Stannard, & Rittweger, 2009 [chapter 6]). It is possible that the acute effects of VBX are related to its influence on non-neurally mediated events during the contractile process, such as those which occur distal to the neuromuscular junction.

Depending on the extent of prior work done, the muscles' performance can either be impaired through fatigue or enhanced, most likely through a phenomenon known as post-activation potentiation (PAP) (Sale, 2002). PAP has been measured by either muscle twitch response or twitch potentiation (TP) and H-reflex or reflex potentiation (RP) (Grange, Cory, Vandenboom, & Houston, 1995; Guillich & Schmidtbleicher, 1996; Sweeney et al., 1993; Trimble & Harp, 1998). Enhanced TP has been reported following contractile activity such as a series of evoked twitches or sustained maximal voluntary contraction (MVC) (Sale, 2002), or heavy resistance exercise prior to completing an explosive movement (Hodgson et al., 2005). The proposed mechanism to account for TP is believed to involve phosphorylation of myosin regulatory light

chains making actin and myosin more sensitive to the intracellular $Ca^{2+}$ signal (Moore & Stull, 1984; Sweeney et al., 1993; Zhi et al., 2005). This results in a greater rate of cross-bridge attachment for the same $Ca^{2+}$ concentration, which in turn increases twitch tension (Metzger et al., 1989). Conversely, RP is thought to elicit reflex activity in the spinal cord, by increasing the activation of the α motoneurons (Hodgson et al., 2005; Trimble & Harp, 1998).

It is therefore possible that acute VBX enhances muscular performance, in part, through PAP, by TP and/or RP; however this theory remains untested. None of the aforementioned studies (Cochrane & Stannard, 2005; Cochrane et al., 2008b [chapter 3]; Cochrane et al., 2008c [chapter 2]) assessed the presence of PAP by recording muscle twitch or reflex activity. Therefore, it is unclear whether PAP exists and if muscle twitch and patellar reflex properties are simultaneously potentiated by an acute bout of VBX.

Therefore, the aim of this study was to compare the acute effect of VBX with another modality such as stationary cycling and control (no VBX) on PAP by assessing TP and patellar RP. Given that VBX seems to induce a high rate of α motoneuron discharge, it was postulated that 5 minutes continuous acute VBX (26 Hz) would result in a greater TP compared to centrally mediated effect of a RP from VBX-induced stretch reflex.

## 7.3 Methods

### *Participants*

Six males (mean age (± SE) 22.8 ± 0.7 yr; body mass 83.4 ± 1.7 kg, height 1.78 ± 0.2 m) and six females 22.8 ± 1.5 yr; body mass 64.4 ± 1.4 kg, height 1.71 ± 0.2 m) competing in national level sport (field hockey n=9; cycling n=1; soccer n=2) volunteered to participate in the study. Inclusion criteria required no history of neurological or circulatory disorders, no lower-limb or back injury in the previous 6 months, and no lower-limb or back surgery in the previous 2 years. Participants were tested to determine if they had a measurable patellar tendon reflex response, and those with a measurable reflex were then asked to take part in the study. Written informed consent was obtained from the participants and ethical approval was granted by the University Human Ethics Committee.

### *Study Design*

Every participant performed three interventions, namely static squat with VBX (VBX+), the same static squat regimen without VBX (VBX−), and stationary cycling (CYCL), in a randomised balanced order with 24 hours separating each intervention.

The VBX+ and CYCL were assumed to be matched for heat production. From previous research it has been reported that 70W of stationary cycling for 5 minutes elicits a change of 0.85ºC in muscle temperature ($T_m$) (Cochrane et al., 2008b [chapter 3]), which is comparable to

previous research that $T_m$ increases 0.90ºC from 5 minutes of VBX (26 Hz) with static squat (SS) (40º knee flexion) (Cochrane, Stannard, Firth, & Rittweger, 2010 [chapter 6]). Therefore, the interventions were based on a continuous 5 minute protocol of VBX+ SS (26 Hz) and 70W CYCL.

Participants completed the performance tests at the same time of day and were instructed to strictly refrain from undertaking any vigorous activity 24 hours prior to the interventions. All participants were familiarised with equipment and protocols the day before undertaking their first session. All interventions were performed at a constant ambient temperature (20.7 ± 0.8 ºC) and barometric pressure (755 ± 3.5 mmHg).

Prior to each intervention, ~90s, 5, and 10 minutes post-intervention, each participant underwent transcutaneous muscle stimulation consisting of a single twitch 200μs pulse and three patellar tendon taps.

### *Muscle and Patellar Tendon Stimulation*

#### *1. Muscle Twitch*

Participants were seated in a straight-backed chair apparatus with hips and knees flexed at 90°. To prevent unwanted movement the participant's pelvic region was secured to the chair by an adjustable belt, and arms were crossed with hands placed on the opposite shoulder. The right ankle was secured to a custom-built ankle holder by a Velcro® strap. The ankle holder was connected to a load cell (6000 ICI, Sensortronics, Covina, CA, USA) and DC amplifier (Figure 2). The load cell was fixed firmly to the undercarriage frame work of the chair apparatus. The evoked force was detected by the load cell, amplified and recorded to an acquisition system (Powerlab, 8/30, and Chart v6.1.1, ADInstruments, Australia) and desktop computer. Prior to the participant's arrival the load cell was zeroed and calibrated.

Two 5 x 9 cm self-adhesive surface electrodes were tranversely placed on the distal (8cm from the inguinal crease) and proximal areas (5cm from the proximal border of patella) of the quadriceps muscle group (Figure 2). Skin preparation for electrode placement included shaving, gentle abrasion using a gel-based product (Nuprep, D.O. Weaver, USA), and cleansing with an alcohol tissue pad. To ensure that the electrode placement was the same between the interventions a permanent marker pen was used to trace the position of the electrodes. During the familiarisation the maximum twitch response of each participant was determined by a single 200μs pulse generated by a high-voltage stimulator (model DS 7A, Digitimer Ltd, Welwyn, Garden City, England). This involved increasing the twitch voltage in 50-100 mA steps until a plateau in twitch force was reached. This value was noted as the maximal twitch response and used in the measurements at the time of the three subsequent interventions. A typical twitch stimulus for VBX+ before, ~90s, 5 and 10 minutes after the intervention is shown in Figure 1.

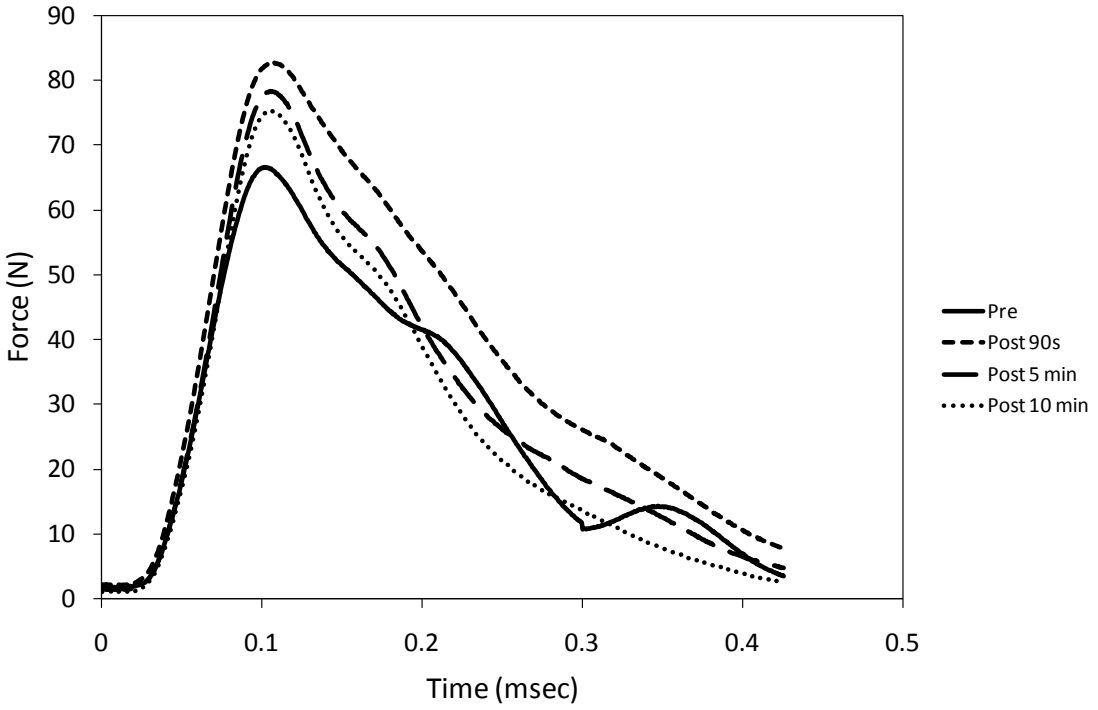
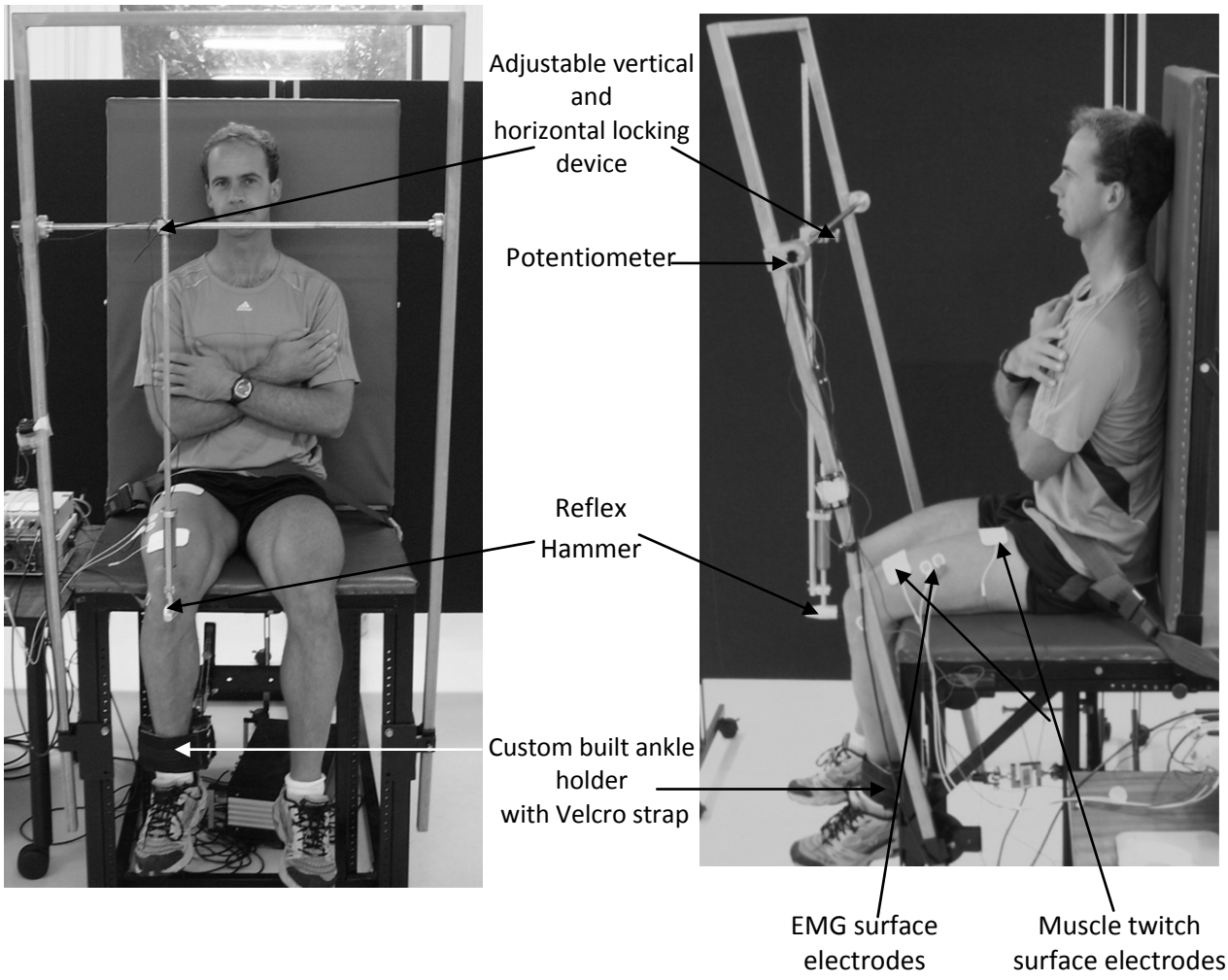



**Figure 1** A typical VBX+ response of transcutaneous muscle twitch for pre-, post- 90s, 5 and 10 minutes

*2. Patellar tendon reflex*

A reflex hammer (MLA93 ADInstruments, USA) was extended onto an alloy rod that rotated about a horizontal axis mounted to a steel frame and bolted to the chair apparatus (Figure 2). A 10-bit potentiometer (Model 533, Vishay, Malvern, PA) was fixed to the bearing of the rotating axis which measured the angle of release recorded by a data acquisition system (Powerlab, 8/30, and Chart v6.1.1, ADInstruments, Australia) (Figure 2). A piezo-electric sensor was located in the head of the hammer that gave an output signal which was indicative of the hammer strike force. The reflex hammer had an adjustable vertical and horizontal locking device that positioned the head of the hammer to strike the centre third of the patellar tendon. The exact contact point of the tendon strike on the skin was then marked with a permanent marker which was used for subsequent trials. For every strike the reflex hammer was raised to 60° from the vertical before release to give consistent and repeatable tendon reflex response. The angle of the reflex hammer was calibrated prior to every testing session. Three patellar tendon strikes were administered with 15s rest separating each strike.

**Figure 2** Participant set up for evoked muscle twitch and patellar tendon reflex



### *3. Electromyography (EMG)*

EMG activity was recorded from the right leg of the vastus lateralis (VL) muscle. Surface pre-gelled Ag-AgCl electrodes, 10mm diameter (Medicostest, Rugmarken, Denmark) were placed over the muscle belly at an inter-electrode distance of 20mm with a reference electrode placed over the head of the fibula. The EMG signal was amplified at a pre-determined bandwidth filter of 10-500 Hz with a gain of 1000. The skin of the electrode site was prepared by shaving, gentle abrasion using a gel-based product (Nuprep, D.O. Weaver, USA), and cleansed with an alcohol swab.

### *Interventions*

#### *1. VBX+*

Acute VBX was performed on a commercial machine (Galileo Sport, Novotec, Pforzheim, Germany), which has a motorised teeterboard that produces side-alternating vertical sinusoidal vibrations to the body. To negate the possibility of discomfort to the sole of the foot participants wore sport shoes and placed their feet on either side of the central axis which corresponded to vibration amplitude (peak-to-peak) of 6mm, magnitude of 9*g*, and the *v*ibration frequency was set to 26 Hz for 5 minutes. This concurred with previous reports that 26 Hz augments jump height from a possible warm-up effect (Bosco et al., 1998; Cochrane & Stannard, 2005; Cochrane et al., 2008b [chapter 3]). During exposure to VBX the participants were in a static squat position of 40°knee flexion. A manual goniometer was used to set the knee angle, and to ensure that the position was maintained through the intervention an adjustable hurdle was placed under the participant's gluteal fold.

#### *2. VBX−*

The vibration machine was set to 0 Hz (amplitude = 0mm), with the same static squat position and time parameters used for VBX+.

#### *3. CYCL*

This involved pedalling at a cadence of 50 revolutions per minute on a stationary friction-braked cycle ergometer (Monark 874 E, Varberg, Sweden) for 5 minutes at 70 W. As mentioned previously, the workload of 70 W was selected from a previous study that showed at 5 minutes $T_m$ increases to 0.85°C (Cochrane et al., 2008b [chapter 3]), which is comparable to work conducted in our laboratory of 0.9°C increase in $T_m$ from VBX+ with static squat performed at 40º knee flexion (Cochrane et al., 2010 [chapter 6]).

*Data Analysis*

All data were recorded and sampled at 2000 Hz using a data acquisition system (PowerLab 8/30 ADInstruments, Australia) and was stored off-line on a desktop computer and analysed using chart software (version 6.1.1, ADInstruments, Australia). Prior to analysis all recorded signals were visually checked for recording-related artefacts. The onset of muscle activation (EMG) and force was defined as the change in three standard deviations from resting values of muscle EMG and force. Each muscle twitch at pre-, post-90s, 5 and 10 minutes was analysed for the following characteristics (Figure 3); (1) muscle twitch peak force (PF) was determined at the highest amplitude; (2) time to peak force (TPF) was defined as the time from force onset to PF; (3) rate of force development (RFD) was defined as the average slope of the twitch force-time curve (ΔPF / TPF). For reflex data, the onset of EMG and onset of force was defined as the change in three standard deviations during baseline values conducted over 0.03s and analysed by the chart software. Individual markers were then placed on the appropriate time interval of EMG, force onset and hammer contact to calculate reflex latency and electromechanical delay.

The mean of three hammer strikes at pre-, post-90s, 5 and 10 minutes were analysed for (1) electromechanical delay (EMD), defined as the time from the onset of muscle activation to force onset (Figure 4); (2) patellar reflex latency (RL), defined as the time interval from the reflex hammer impact to onset of muscle activation (Figure 4); (3) patellar reflex peak force (RPF), calculated as the maximum amplitude and normalised to the amplitude of the reflex hammer impact (Figure 4). RL and RPF were chosen because they are able to assess changes in muscle spindle sensitivity or motoneuron excitability (Hopkins et al., 2008; Rossi-Durand, 2002). Likewise, EMD was selected because it is an indirect measure of musculo-tendinous stiffness (Isabelle et al., 2003) and provides information of spindle sensitivity via alpha-gamma co-activation (Hopkins et al., 2008).

**Figure 3** A typical trace of an evoked muscle twitch response pre-intervention illustrating muscle twitch peak force, time to peak force, and rate of force development

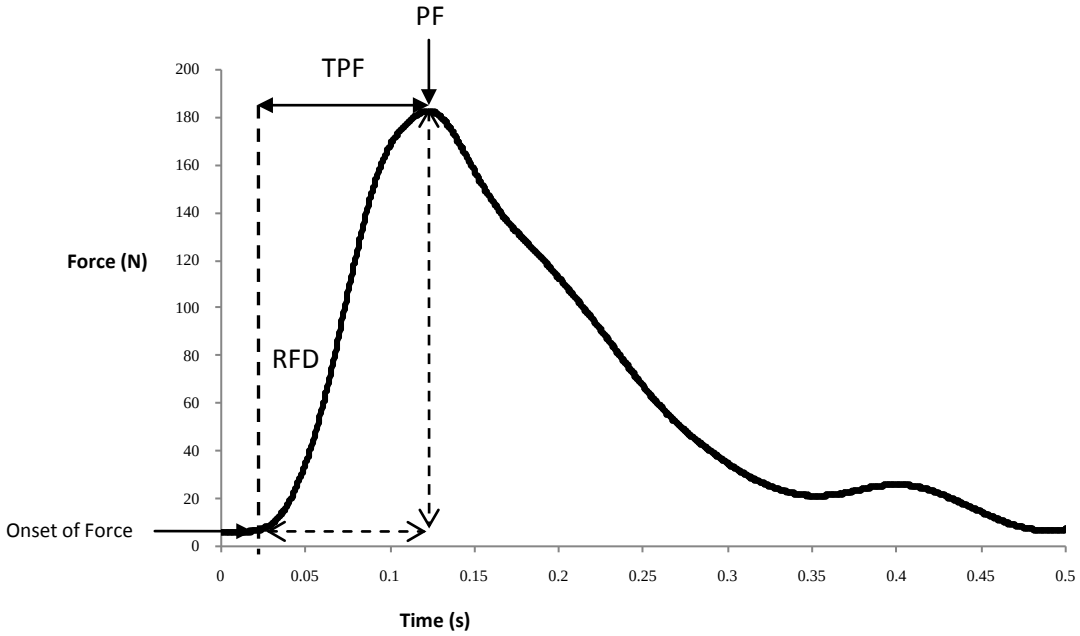
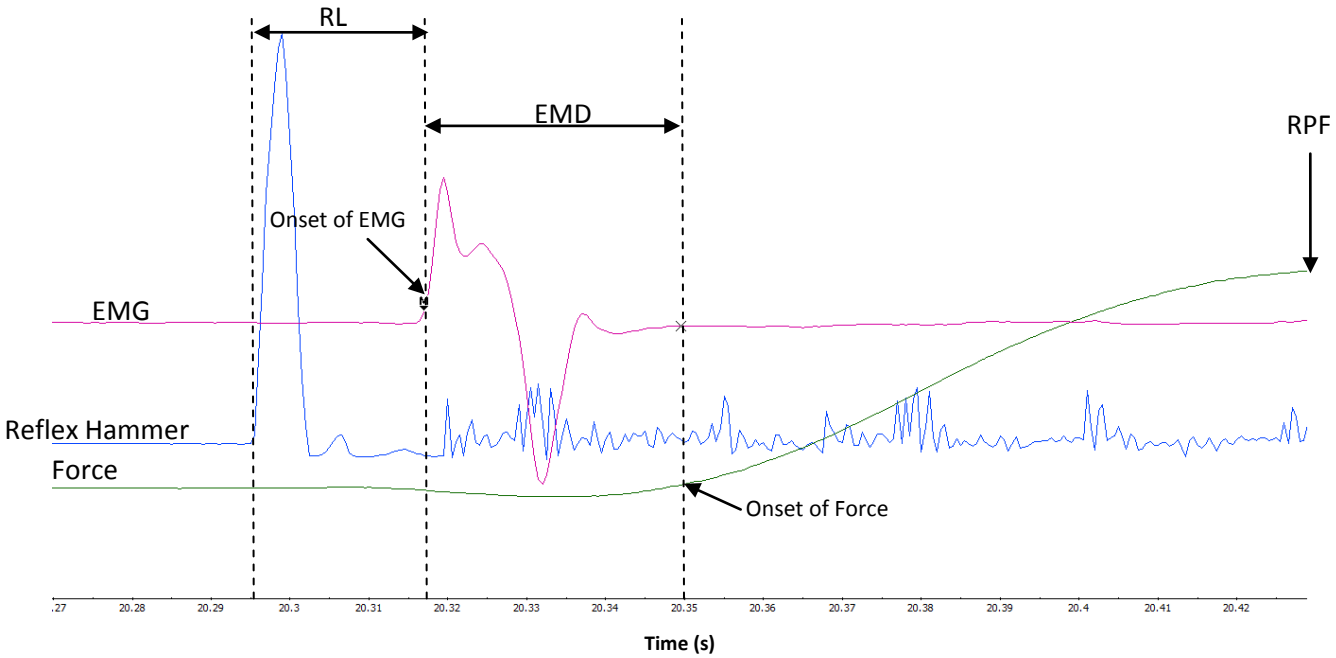



**Figure 4** A typical trace of a patellar tendon reflex pre-intervention showing electromechanical delay, patellar reflex latency, and patellar reflex peak force



*Statistical Analyses*

For all the interventions the mean relative changes were calculated as the difference between post- (90s, 5 and 10 minutes) and pre-values. For EMD and RL absolute changes were calculated between post- and pre-values. A 2 factor [time (Pre-, Post- 90s, 5, 10 minutes) x intervention (VBX+, VBX−, CYCL)] repeated measures ANOVA was performed to examine the magnitude and significance of these on PF, TPF, RFD, EMD, RL, RPF. For multiple comparisons significance, post-hoc pairwise comparisons were performed and adjusted to Bonferroni's rule. Using pre-values intraclass correlation coefficient (ICC) assessed the day-to-day reproducibility of PF muscle twitch and patellar tendon impact strikes. All statistical analyses were performed using statistical software SPSS for Windows Version 16 (Chicago, IL, USA) and level of significance was set at $p < 0.05$.

## 7.4 Results

### *Muscle twitch peak force (PF)*

There was a significant treatment, time, and treatment x time interaction effect for PF. Post-hoc analyses showed that 90s after VBX+ PF was significantly higher ($p<0.01$) compared to VBX− and CYCL (Figure 5), which equated to a 12.4% increase for VBX+, compared to 0.5% VBX−, and -1.7% CYCL. However, the potentiation was not evident at post-5 and 10 minutes VBX+, indicating the transient nature of VBX on muscle twitch potentiation. No other significant differences over time (post-5 and 10 min) were found between the interventions (Figure 5).

### *Time to peak force (TPF)*

There was no treatment, time, or time x treatment interaction effect for TPF (Table 1).

### *Rate of force development (RFD)*

There was a significant treatment, time and treatment x time interaction effect for RFD (Figure 6). Post-hoc analyses revealed RFD being significantly ($p<0.01$) higher post-90s for VBX+ compared to VBX− and CYCL (Figure 6), which equated to a 11.2% increase for VBX+, compared to 1.2% VBX−, and -0.7% CYCL. RFD was significantly higher ($p<0.01$) at post-90s and post-5 min compared to pre but returned to baseline levels at post-10 minutes (Figure 6). There were no significant increases in RFD any time point for either VBX− or CYCL (Figure 6).

### *Patellar Tendon Reflex*

There was no treatment, time or time x treatment interaction effect for electromechanical delay (EMD), patellar reflex latency (RL) (Table 2) and patellar reflex peak force (RPF) (Table 1). There were no significant differences between gender for any muscle twitch or reflex activity parameters.

### *Reproducibility*

The ICC day-to-day reliability of PF muscle twitch ($r=0.986$) and patellar tendon impact strike ($r=0.931$) were highly significant ($p<0.001$), indicating that a high degree of repeatability and consistency were attained between interventions.

**Table 1** Mean (± SE) changes in time to peak force and rate of force development for VBX+, VBX− and CYCL

| | TPF (msec) | | | RPF | | |
|---|---|---|---|---|---|---|
| | VBX+ | VBX− | CYCL | VBX+ | VBX− | CYCL |
| Pre | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 | 0.0 |
| Post-90s | -0.8 (2.0) | 1.5 (2.3) | 1.5 (2.3) | 0.1 (0.9) | 0.4 (1.3) | -1.3 (0.7) |
| Post-5 min | 2.1 (1.9) | -5.8 (2.7) | -5.8 (2.7) | -2.8 (0.9) | -1.0 (1.2) | -2.8 (0.3) |
| Post-10 min | 0.0 (1.9) | -7.4 (2.0) | -7.4 (2.0) | 1.2 (2.6) | 1.2 (1.7) | -2.8 (2.2) |

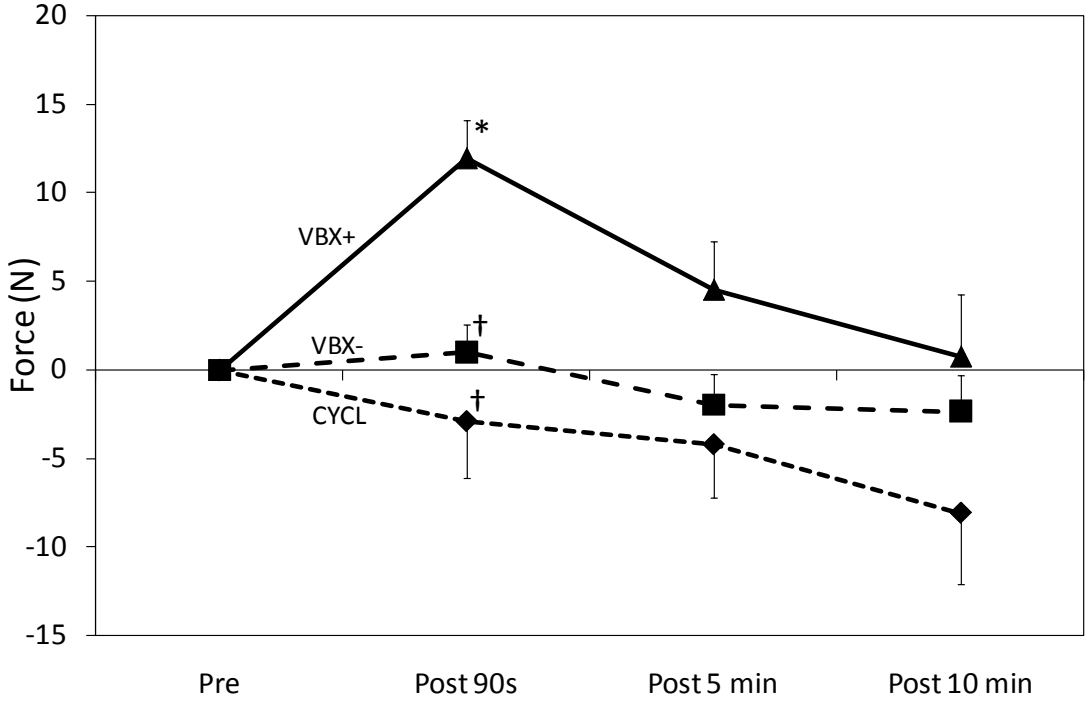
TPT = Time to peak muscle force; RPF = Patellar reflex peak force

**Table 2** Mean (± SE) absolute electromechanical delay and patellar reflex latency for VBX+, VBX− and CYCL

| | EMD (msec) | | | RL (msec) | | |
|---|---|---|---|---|---|---|
| | VBX+ | VBX− | CYCL | VBX+ | VBX− | CYCL |
| Pre | 26.4 (3.4) | 25.9 (2.9) | 24.0 (1.6) | 32.2 (1.8) | 32.8 (1.8) | 33.6 (2.0) |
| Post-90s | 27.0 (2.0) | 25.9 (2.9) | 23.4 (2.6) | 30.0 (2.2) | 33.2 (2.0) | 33.3 (2.2) |
| Post-5 min | 26.8 (2.8) | 24.7 (2.9) | 23.3 (2.5) | 29.6 (2.2) | 32.1 (2.0) | 33.3 (2.2) |
| Post-10 min | 26.5 (2.1) | 26.1 (3.3) | 27.2 (2.5) | 30.3 (2.2) | 32.6 (2.1) | 33.7 (2.3) |

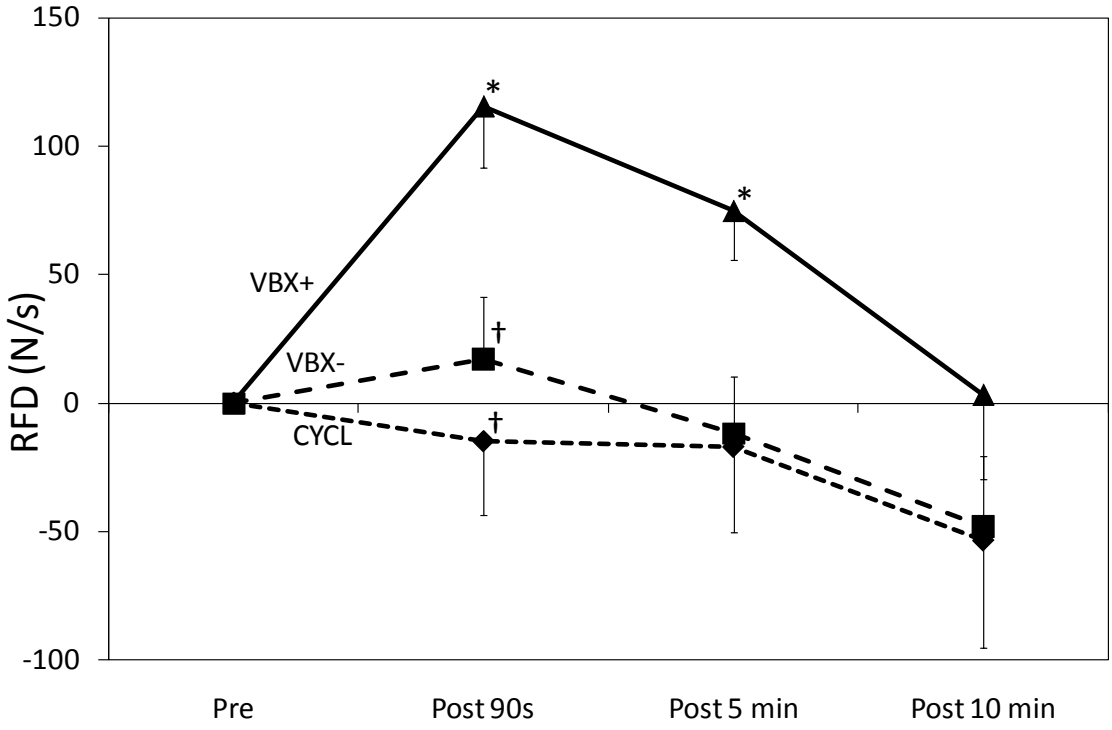
EMD = Electromechanical delay; RL = Patellar reflex latency

**Figure 5** Mean (± SE) change in muscle twitch peak force for VBX+, VBX− and CYCL




* p< 0.01 compared to pre-value, † p< 0.01 compared to VBX+

**Figure 6** Mean (± SE) change in rate of force development of muscle twitch for VBX+, VBX– and CYCL




* p< 0.01 compared to pre-value, † p< 0.01 compared to VBX+

## 7.5 Discussion

The aim of this study was to compare the effect of acute VBX with static squat to that of stationary cycling and control (VBX−) on PAP from muscle twitch and patellar reflex activity. It was hypothesised that 5 minutes of continuous acute VBX would potentiate the muscle twitch response compared to a centrally mediated effect that would potentiate VBX-induced patellar tendon reflex. The results support the hypothesis that an acute continuous bout of VBX at 26 Hz induces a PAP of twitch potentiation (TP) compared to VBX− and CYCL interventions.

It has reported that acute VBX improves short-term muscular performance above cycling-only exercise (Cochrane & Stannard, 2005) which may indicate that VBX has the potential to be considered as a warm-up modality prior to explosive activities such as strength, speed and power (Bazett-Jones et al., 2008; Cochrane & Stannard, 2005; Cormie et al., 2006). Previously it has been shown that VBX rapidly increases intramuscular temperature, and this contributes to an augmented effect on subsequent muscular activity (Cochrane et al., 2008b [chapter 3]). However, it must be assumed that intra-muscular temperature was similar among the different conditions in this study (Cochrane et al., 2008b [chapter 3]), and therefore seems likely that mechanisms other than temperature caused the observed effects. One such additional phenomenon is PAP, which is referred to as an increase in muscle performance preceded by a muscle contractile activity (Sale, 2002) and is assessed by twitch potentiation (TP), or reflex potentiation (RP) (Hodgson et al., 2005). To the author's knowledge, this is the first time following VBX that TP and RP were simultaneously measured. The results showed that VBX+ increased PF, RFD with no change in TPF compared to VBX− and CYCL. This concurs with previous research showing that short duration (5-10s) isometric maximum voluntary contractions enhance PF and RFD (Sale, 2002; Vandenboom, Grange, & Houston, 1993) whereas TPF remains unchanged (Petrella, Cunningham, Vandervoort, & Paterson, 1989). The time course of TP reached a maximal level 90s after VBX+ and returned to baseline levels after 10 minutes, which is in agreement with other studies (Baudry & Duchateau, 2004; O'Leary et al., 1997).

Likewise, the increase in RFD from acute VBX is supported by other studies that have reported increases in RFD following high stimulation frequencies (>100Hz) (Abbate, Sargeant, Verdijk, & De Haan, 2000; O'Leary et al., 1997; Vandenboom et al., 1993). Hence, as a result of enhancing RFD, explosive activities such as jumping, kicking and throwing may be improved if TP is heightened (Hodgson et al., 2005; Sale, 2002). By contrast, one study investigated the effect of acute VBX on muscle activation properties and found no differences in voluntary or simulated maximal rates of force rise between baseline and post-measures of the leg extensor muscle group (de Ruiter et al., 2003). However this study differs from the present one in several ways, as the authors did not include a control group and used an intermittent protocol of 5 x 1

minute VBX exposures with 2 minutes rest between treatments. Moreover, electrical muscle stimulation in that study was in combination with voluntary contractions, whilst the twitch characteristics have been investigated without such interference in the present study.

The explanation for PAP has centred around twitch potentiation (TP) and reflex potentiation (RP) (Folland, Wakamatsu, & Fimland, 2008; Grange et al., 1995; Hodgson et al., 2005; Sweeney et al., 1993; Trimble & Harp, 1998). TP is considered to involve the phosphorylation of myosin regulatory light chains making actin and myosin more sensitive to the intracellular $Ca^{2+}$ signal (Moore & Stull, 1984; Sweeney et al., 1993; Zhi et al., 2005). This would result in greater cross-bridge interaction for the same intracellular $Ca^{2+}$concentration, which in turn increases the muscle tension for the same absolute level of neural stimulus (Metzger et al., 1989). However, if calcium homeostasis was involved, then one would expect a disproportionate change in RFD and PF, yet the increase in RDF and PF 90s post-VBX+ were similar (11.2% RFD, 12.4% PF) suggesting that PAP may be prevalent according to the cross-bridge cycle model (Brenner & Eisenberg, 1986).

Conversely, RP is thought to elicit reflex activity in the spinal cord, by increasing synaptic efficacy between Ia afferent terminals and α motoneurons of the muscle (Hodgson et al., 2005; Trimble & Harp, 1998). VBX is thought to elicit muscle contractions through spinal reflexes (Cardinale & Bosco, 2003; Cochrane et al., 2009 [chapter 6]; Rittweger et al., 2003). However, and in contrast to other exercise modalities, stretch and H-reflexes are typically suppressed during isolated muscle vibration (Arcangel et al., 1971; De Gail et al., 1966). It should be considered that VBX is a combination of muscle vibration and voluntary exercise contractions, and the ambiguous findings of either increased (Melnyk et al., 2008; Rittweger et al., 2003), or unchanged (Hopkins et al., 2008) reflex activity in response to VBX may be due to the complexity of this intervention. Importantly, and in agreement with the findings of Hopkins *et al.* (2008), there were no changes in reflex magnitude or latency. Therefore, the findings suggest that TP from VBX may be more important than RP. Further, it should be noted that using a tendon tap to elicit a stretch reflex response does not necessarily provide a comprehensive measure of spindle sensitivity or its contribution, as there are several factors from which the stretch reflex can be modified either by the excitatory or inhibitory elements of the motoneuron pool and/or the mechanical sensitivity of the muscle spindle itself (Hopkins et al., 2008). Given these reservations, the results indicate that an acute bout of continuous VBX does not elicit a RP, which is in disagreement with the speculated proposal that changes in muscle performance from VBX are in part caused by neurogenic factors from possible changes in muscle spindle sensitivity (Cardinale & Bosco, 2003; Cardinale & Rittweger, 2006).

There were no TP or RP effects observed in VBX− and CYCL even though muscle temperature was indirectly matched between the interventions. An explanation for this finding may be due

to the force levels being too small to elicit increases in TFP and RFD. It is well documented that heavy pre-loading exercise regimes such as 3-5 repetition maximum (RM) or MVC's are often implemented prior to short-term activities to induce PAP (Clark, Bryant, & Reaburn, 2006; Gourgoulis et al., 2003; Young et al., 1998). Therefore, it is plausible that the pre-loading activity of VBX− and CYCL needed to be of higher magnitude to elicit TP and RP, compared to VBX+.

This study has two limitations. Firstly, there is a possibility that the series- or parallel-elastic elements were affected in different ways by the three interventions. However, it is unlikely, given that the forces required to reduce musculo-tendinous compliance, such as in stretching, are normally much greater than those used in this study (Morse, Degens, Seynnes, Maganaris, & Jones, 2008). Moreover, in the hypothetical case of increased musculo-tendinous compliance one would have expected an increase in EMD, which was not observed in this study (see Table 2). Secondly, the time frame to detect post-reflex responses also needs to be considered (Hopkins et al., 2008). In the current study it took approximately 90s to initiate the first patellar reflex measurement, due to the time it took to re-connect leads and re-position the participant in the chair apparatus. But this time frame is in agreement with other studies that have shown increases in muscular performance (Bosco et al., 1999; Cochrane & Stannard, 2005; Cochrane et al., 2008b [chapter3]; Torvinen et al., 2002). It has been shown that a potentiated patellar reflex dissipates after 10-20s following exhaustive VBX (Rittweger et al., 2003) so it is possible that any RP may have been undetected.

In conclusion, the neuromuscular response of muscle twitch and patellar reflex characteristics, and the results suggest that acute VBX causes PAP mainly downstream from the neuromuscular junction. Muscular performance studies have shown that PAP is induced by heavy pre-load activity which enhances muscular activity (Gourgoulis et al., 2003; Young et al., 1998). Likewise, VBX is also capable of inducing high gravitational loads through vibration frequency and amplitude that act as a pre-loading movement to activate PAP. The augmented TP of the current study may have resulted from an increase in muscle activity due to the vibrations being damped by the muscles (Ettema & Huijing, 1994; Wakeling et al., 2002), which may have caused cross-bridge cycling to increase (Cardinale & Wakeling, 2005). However, it is untested whether phosphorlylation of myosin regulatory light chains and cross-bridge attachment are mediated by VBX and requires confirmation before any conclusion can be drawn on whether VBX elicits TP via this mechanism.

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# Chapter 8 – General Discussion and Conclusion

## 8.1 General Discussion

VBX is currently enjoying popularity as an alternative exercise modality for enhancing muscle activity, force and power. VBX has been suggested as an attractive and efficient complement to traditional forms of exercise and therapeutic techniques. The muscle performance benefit supposedly occurs via neurogenic potentiation involving spinal reflexes and muscle activation, which is based on the tonic vibration reflex (TVR) (Cardinale & Bosco, 2003; Rittweger et al., 2003). Previous studies have reported that vibration is capable of augmenting muscle spindle activity which causes an excitatory response in the primary endings of non-contracting muscle (Burke et al., 1976a; Ribot-Ciscar et al., 1998). However, most studies have been conducted on concentric-eccentric muscle action where the effect of VBX on concentric movement remains largely untested. Therefore, **Chapter 2** examined the concentric phase of a prone bench pull following interventions of VBX, no VBX, and arm cranking. VBX increased upper-body concentric-only peak power, but the increase was similar to that of performing concentric-only exercise (arm cranking). The explanation for this is that the concentric-only (prone bench pull) exercise may not have optimised the stretch reflex response or that VBX may have represented an ergogenic phenomenon known as a post-activation potentiation, where muscle temperature ($T_m$) may have an effect on producing a warm-up effect. Because it was unclear whether enhanced muscle power from acute VBX can be explained by an increase in muscle temperature, **Chapter 3** investigated the effect of $T_m$ and muscle performance following VBX. The results showed that VBX elevated $T_m$ more rapidly than an active (stationary cycling) and passive warm-up (hot water bath). But when the same muscle temperature was achieved VBX did not augment short-term performance more effectively than cycling or hot bath interventions. Given that all three warm-up methods yielded the same increase in peak power output, it was concluded that the main effect was temperature dependent.

**Chapter 3** had not distinguished between the effects of vibration and squatting on the increase in $T_m$. Static (SS) and dynamic (DS) squats are common exercises used in VBX training, but the effect of shallow DS versus SS with concurrent VBX on vastus lateralis temperature and cardiovascular response was unknown, which lead to the study described in **Chapter 4**. When $\dot{V}O_2$ was matched between DS without vibration and SS with vibration, $T_m$ increase and HR response were comparable. However, for DS with vibration $T_m$, HR, and $\dot{V}O_2$ were not significantly greater than for DS without vibration and SS with vibration, which may be influenced by a postural control mechanism and/or a damping response (Abercromby et al.,

2007a). Therefore, there appears to be no benefit in performing a shallow DS with vibration at a tempo of 50 bpm, because $T_m$, HR, and $\dot{V}O_2$ are likely to be increased by the same amount and rate without VBX.

**Chapter 4** showed that the workload of squatting with and without vibration was too low to increase indices of cardiovascular function in young and older people. **Chapter 5** thus compared the acute physiological responses of VBX in young and older people and assessed whether age-related differences in VBX-related oxygen uptake existed when the Jendrassik manoeuvre was superimposed with vibration and load. In both groups of people, oxygen uptake was significantly enhanced with vibration and additional load, or when the Jendrassik manoeuvre was superimposed with vibration and load causing the metabolic rate to be increased. But the elicited increase in $\dot{V}O_2$ from vibration was an insufficient stimulus to enhance cardiovascular indices. Since a main objective of a balanced exercise programme is to increase aerobic capacity it would be imprudent to completely substitute conventional aerobic exercise with vibration. However, when conventional aerobic exercise is not possible, for example, in aged, cardiovascular compromised persons, VBX could be implemented at an early stage because it could provide a safe induction of a slight elevation of cardiovascular function indices while providing neural and myogenic benefits that could enhance postural and locomotion ability, which are central to cardiovascular health. It should be noted that in Chapter 5 the prototype leg press vibration machine was limited to a maximum amplitude of 1mm, which differed from the 2-12mm amplitude range of commercial vibration machines used in other chapters (2, 3, 4, 6, and 7). Thus, vibration amplitude may have an effect, which is supported by the observation of Rittweger *et al*. (2002a) that an increase in amplitude resulted in higher metabolic cost. Further research is required to elucidate the effect of amplitude on muscle activity and performance.

**Chapter 5** showed that superimposition of the Jendrassik manoeuvre upon VBX did enhance metabolic rate, but there were no differences between young and older people which suggests the responses of VBX may been attributed to the TVR (De Gail et al., 1966). This is based upon the stretch reflex response (Burke et al., 1976a; Roll et al., 1989) where vibration applied to the tendon activates primary muscle spindle afferents, stimulating the alpha motoneurons and causing extrafusal muscle fibre contraction (Liddell & Sherrington, 1924). Therefore, **Chapter 6** investigated whether VBX caused changes in muscle length with concurrent increases in EMG activity. VBX was performed at a very low frequency which produced a temporal association between EMG activity and muscle contractile tissue displacement. This result indicates that muscle lengthening may be a prerequisite to eliciting stretch reflexes which might be evidence that muscle activity is damped during vibration. Higher vibration frequencies should be tested to confirm these results because there is strong

evidence from other chapters (3, 4, and 5) that non-neurally mediated events such as muscle temperature may potentiate the contractile process, through a phenomenon known as post-activation potentiation, which is an effect of warming-up. To test whether this was the case, it was of interest to examine if both neural and myogenic aspects could simultaneously be potentiated from VBX.

Both twitch potentiation (TP) and reflex potentiation (RP) were investigated in **Chapter 7**. RP was not potentiated but TP was enhanced with VBX compared to without VBX and stationary cycling, indicating that there was a greater myogenic response compared to a neurally-mediated effect on RP. Therefore, the augmented TP may have resulted from an increase in muscle activity due to the vibrations being damped by the muscles (Ettema & Huijing, 1994; Wakeling et al., 2002), which may have caused cross-bridge interaction to increase (Cardinale & Wakeling, 2005). It is not excluded that RP had occurred but was not detected, because it took approximately 90s to re-connect leads and re-position the participant and initiate the first patellar reflex measurement following vibration.

## 8.2 Conclusion

The primary aim of this thesis was to conduct a cohort of studies to examine muscle physiology aspects and muscle performance of vibration exercise in trained, young and older people. The results from this thesis showed that

- VBX enhanced concentric peak power but the increase was similar to that of concentric (arm-cranking) exercise.

- VBX increased muscular power and when matched for metabolic rate VBX elevated $T_m$ more quickly than traditional forms of cycling and passive warm-up suggesting that that the warm-up interventions were temperature dependent.

- There appears to be no benefit in performing a shallow, fast tempo dynamic squat with vibration as $T_m$, HR, and $\dot{V}O_2$ were increased by the same amount and rate without VBX.

- An increase in vibration frequency and additional load caused an increase in metabolic rate and cardiovascular indices in both young and older people. However, these changes were comparatively small and in relation to conventional aerobic training

- The Jendrassik manoeuvre did not potentiate the metabolic rate in young or older adults when superimposed with VBX and the patellar reflex was not enhanced after VBX, but muscle twitch potentiation was evident. However, at low vibration frequency VBX resulted in small muscle length changes and increased muscle activation. Both neural and myogenic aspects may augment muscle performance in addition to increases in muscle temperature, blood flow, and the activation of central commands.

- No adverse affects of VBX were reported in any of the participants that were involved in the various studies.

## 8.3 Direction for Further Research

This thesis has identified a number of areas for future research. Firstly, Chapter 6, could be replicated by measuring muscle forces at higher vibration frequencies, to provide information on whether muscles are performing or absorbing work. A greater understanding of how vibration affects tendon properties such as tendon stiffness, modulus, and tendon temperature warrants further research. Given that VBX may produce a post-activation potentiation of muscle twitch characteristics, the effect of VBX on nerve conduction velocity should be studied.

The results of this thesis have showed that VBX improves muscle performance in healthy adults. Additionally, VBX might play a role in rehabilitating muscle and ligament injuries, and especially to increase proprioception and postural stability after surgical intervention such as, joint replacement, fracture repair, and ligament reconstruction. It is unclear what regime is required at what stages of disease. This should be a target of research to show if improvements exist in physiological parameters in patients that have Multiple Sclerosis, Parkinson's disease, and diabetes. Finally, extensive research needs to compare the variables of SV and VV platforms and verify the optimal amplitude, frequency and duration for prescription purposes, in the young, the aged, and in persons facing compromised health. It is only with exercise prescription based on scientific foundations that the true potential of this exercise modality can be realised (or refuted).

# Appendices

## Appendix 1 - Information Sheet: The Effect of Acute Upper-Body Vibration Exercise on Concentric Muscular Performance

**Introduction**
Darryl Cochrane (Lecturer in Sport Management & Coaching, Massey University) is the principal researcher investigating a new performance regime known as Upper Body Vibration (UBV). This training is thought to evoke rhythmic muscle contractions similar to that of explosive power and strength training. The UBV is produced by a commercialised system. It uses an oscillating dumbbell to create small changes in muscle length, thus evoking the muscle to stretch and contract. The academic literature has shown that short durations of acute whole body exposures can improve strength and power in sports people. However, it remains to be seen if acute exposure to UBV can also enhance explosive power in sports people.

**Participation Recruitment**
A letter attached with the information sheet was sent to your senior club coach seeking permission to meet with the senior playing members. The sole criterion for this study is that you are an active participant in senior sport at least training and playing 2x week over a season. Every participant will undertake three treatments lasting 3 minutes in duration. These are (1) UBV (2) non-UBV (3) Arm-cranking.The number of participants required for this study is fourteen. We need this number to be able to determine if there are real (i.e. statistically significant) differences between the responses of the treatments (UBV, non-UBV, Arm-cranking).

**Procedures**
You are required to complete a health screening and consent form. This is to identity any risk factors that may prevent you from performing the exercise protocols and providing us with your permission to undertake the study. The gathering of data will allow us to determine if UBV has a physical performance effect on measured outcomes of explosive power and muscle activity. The information gained from this study will be reported at conferences and published in a research journal. Each participant will be given a unique code number, only the researcher and participant shall know the code, this is to protect confidentiality of identity. Additionally, the code will ably assist you in attaining feedback from the physical performance results.All paper data will be stored in a key locked cabinet in the researcher's Massey office. Where paper data is transferred to an electronic spreadsheet, this will be stored on the researcher's office desk top computer which is safeguarded by password entry. All consent forms will be stored in a key locked cabinet ion the principal researcher's Massey office for a period of at least five years. All electronic data will be saved to the H drive, of desktop computer which is password sensitive and only the researcher has access to the password code.
For the disposal of paper data the principal researcher will provide appropriate documentation to the Head of Department to be shredded, whilst electronic files will be deleted from the researcher's Massey University desk top computer. At the completion of the study a report summary of the findings will be named in a sealed envelope for personal collection from the researcher's Massey University office.

**Participant Involvement**
The total duration involved for this study is approximately 1.5 hours. You will perform two familiarisation session of the explosive power test. These will be conducted on a separate day before the commencement of the study. This study will be performed at the Massey University Human Performance Laboratory, Palmerston North. Weight and height will be taken using standard measuring equipment. Pre- and post-physical performance tests will be conducted before and after the three treatments of UBV, Non UBV & Arm-cranking. With any fitness testing there is an element of risk. However, the tests used in this study are the industry standard and approved by the national body, Sport Exercise Science New Zealand. The duration of the explosive power and strength tests range from 0.5 to 4 seconds which is unlikely to initiate a cardiac incident. If for any reason you do sustain any injury the Human Performance Laboratory is well equipped with telephone and first aid for such incidents.

**Tests**
*Strength Test*- A four repetition maximum (4RM) prone bench pull will be administered on a weight training machine (known as a smith machine). This test involves a weight training bench being positioned over the weight training bar. You will undergo a warm up lifting a light weight 10 times, with 60 sec rest. Then you will lift a slight heavier weight 6 times. Following a 2 min rest the weight will be further increased you will attempt to successfully lift 4 times. Based on this 4RM load the one repetition maximum can be then be calculated. This IRM will determine your load that you will lift during the explosive power test.

*Explosive power*- A prone bench pull will be administered on a weight training machine. The machine will be modified with a rotatory encoder connected to a PC computer. This will be capable of measuring the displacement of the bar, hence velocity, acceleration and power can be derived. This exercise (prone bench pull) is a common weight training exercise performed in the gym. A weight training bench will be positioned over the weight training bar with a weight of 30% 1RM. You will lie prone (facing down), hold the bar in the bottom position and with an audio call will proceed to bring the bar up to the bench as powerfully as possible. You will perform 3 trials with 15 seconds rest separating each trial.

*EMG(muscle activity)* – Disposable surface electrodes will be placed over the mid point of the biceps brachii of both arms during the prone bench pull. The electrodes will be placed 2 cm apart, in parallel to the muscle fibres. The raw signals from the electrodes will be converted by a machine connected to a desktop computer for further analysis.

**Protocol**
Three treatments will be undertaken in a balanced random order, conducted on separate days with at least 24 hours recovery between them. Pre- and post-physical performance tests will be

conducted before and after the three treatments of Arm-crank (AC), Upper Body Vibration (UBV), and Non Upper Body Vibration (NUBV).

*1. AC* - Using a fractioned-braked arm-crank ergometer (Monark 881e, Varberg, Sweden) each climber cranked for 5 mins in a seated position to eliminate lower body contribution. The crank will be set at 50 W.
*2. UB* - This will be performed by a Galileo TOP® (Novotec, Pforzheim, Germany) device, which produces UBV to shoulders and arms from an electric-powered dumbbell. The central handle piece rotates producing oscillatory movements to the body of varying frequencies (0 to 30 Hz) with amplitude of 3mm around a horizontal axis.
The participants will be instructed to stand upright and grip the vibrating dumbbell handle for 30 second bouts, alternating each hand every 30 seconds. The dumbbell will be placed in the hand in front of the body at 90 degrees. The 2.5kg Galileo TOP® vibration dumbbell will be set at 20 Hz, amplitude 3 mm.
*3. NUBV* - Each athlete will hold the vibrating dumbbell handle (0 Hz, 0 amplitude) and perform the same exercises and time constructs as for UBV.To avoid biorhythmic alterations you will perform the treatments and performance testing at the same time of day.

**Participant's Rights**
You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at the time of physical testing;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Feedback is important for muscular performance testing hence, you will be allowed to approach the researcher and use your personal number to identify your result during any stage of testing. At the end of the research you will be given a research report detailing the outcome of the study, this will be personally named and enveloped for collection at the researcher's university officeThe information gained from this study will be reported at conferences and published in research journals. All data collected including the consent and exercise safety forms will be stored in a locked filing cabinet in the researchers Massey University office.Data will also be stored in electronic form onto the researcher's H drive network (password sensitive) that is only accessible by the researcher. The participant's personal identification number will be used to ensure that your identity is not disclosed. The results will be confidential to the researcher and any publications resulting from it.

After the completion of the study electronic files will be kept on the computer and all raw data will be kept in a lockable filing cabinet and all video tapes will be deleted. This project has been reviewed and approved by the Massey University Human Ethics CommitteeIf you have any concerns about the conduct of this research, please contact Professor Sylvia V Rumball, Chair, Massey University Campus Human Ethics Committee: Palmerston North, telephone 06 350 5249, email humanethicspn@massey.ac.nz.

# Appendix 2 - Information Sheet: The Effect of Acute Lower-Body Vibration Exercise on the Rate of Muscle Temperature Increase

***Supervisor/Principal Investigator:*** Darryl Cochrane, MPhEd

***Investigator/Collaborators***: Joern Rittweger, M.D., PhD.
Marco Narici, PhD, Professor
Olivier Seynnes, PhD

***Ethics Committee Approval Number***: 2006/04/02

***Project Title***: The effects of different warm-up interventions on peak muscle power output

***Purpose of study and brief description of procedures.***

This study is investigating whether vibration exercise may help to warm up your body. This will help us understand if this type of exercise will provide an alternative form of warming-up prior to physical activity.

After answering some questions about your general and medical histories, you will be asked to visit the laboratory on 3 separate days. On each visit to the laboratory you will be asked to self insert a rectal probe (1.5mm diameter) 10 cm beyond the rectum this will stay in this position for duration of each warm-up procedures. There may be some apprehension and anxiety to self inserting a probe into the rectum however you will be assured that the procedure is easy to undertake and relatively painless. The sterilisation of probes will be submerged in **liquid sterilisation** for 48 hours **and** rinsed in running hot water then packed into a labelled plastic bag. A medical professional will then insert a small temperature sensor, which measures how warm your thigh muscle gets. This sensor will be inserted into your thigh muscle via a needle catheter. The needle will then be withdrawn leaving only the sensor in the muscle. The leads from the sensor will be taped down to the skin surface and the entry site treated with antiseptic solution and covered with a sterile bandage. You will then be seated in a room for 30 minutes that has a constant air temperature. Using a non-permanent pen we will mark sites at the shin, thigh, chest and shoulder and using an infra-red thermometer we will take skin temperature at these sites. You will be asked to wear a strap around your chest, this measures your heart rate moreover we will be asking your perception of your thermal leg comfort.

For each warm-up procedure you will be asked to wear a small mask that covers your nose and mouth, this allows us to measure your breathing. One of the three warm-up procedures involves exercising on a specially designed machine that vibrates the whole body and causes muscle contractions. This is not harmful, however in some instances you may experience muscle itchiness in your legs, up to 2-3 minutes after the vibration exercise, this is a normal side effect that is harmless and is due to the temporary changes in leg blood flow. The second procedure requires you to cycle at a gentle pace on an exercise bike for 10 minutes. In the third procedure your legs will be submerged into a hot water tank for 20 minutes. Once the testing is finished the medical professional will remove the muscle sensor, again you may feel some pain on its exit and there is a small possibility of infection when the sensor is removed from your thigh, this may occur in later days, if this does occur you should inform us. The sensor is sterilised according to strict procedures and should minimise the risk of infection. The insertion of the sensor needs to be performed each time for the three warm-up interventions. You will be seated whilst the sensor is inserted to avoid any adverse reactions such as fainting.

It is understood that you can withdraw your consent at any time during the study and discontinue your participation without giving reasons. You may also decide to refuse particular parts of the protocol. No disadvantage to you will arise from any such decision. All data obtained in this study will be treated confidentially.

***Participant Statement***

I fully understand what is involved in taking part in this study. Any questions I have about the study, or my participation in it, have been answered to my satisfaction. I understand that I do not have to take part and that I may decide to withdraw from the study at any point without prejudice. I have had my attention drawn to the document 'Ethical Regulations for the Use of Humans in Research'. My concerns regarding this study have been answered and such further concerns as I have during the time of the study will be responded to. It has been made clear to me that, should I feel that these Regulations are being infringed or that my interests are otherwise being ignored, neglected or denied, I should inform the Chair of the Ethics Committee of the Department of Exercise and Sport Science, Manchester Metropolitan University, Hassall Road, Alsager, Cheshire, ST7 2HL who will undertake to investigate my complaint.

Signed ..... Date .....

# Appendix 3 - Information Sheet: The Comparison of Muscle Temperatures during Static and Dynamic Squatting With and Without Acute Vibration Exercise

**Introduction**
Darryl Cochrane (Lecturer in Sport Management & Coaching, Massey University) is the principal researcher investigating a new performance regime known as vibration exercise (VBX) and kindly invites you to be part of his current study. It has been suggested that VBX evokes rhythmic muscle contractions similar to that of explosive power and strength training. VBX is produced by a commercialised system where an oscillating platform causes muscles to stretch and contract.
Previously it has been shown that an increase in muscle temperature of 1.5 ˚C occurs more rapidly (5 mins) when squatting up and down during VBX, compared to conventional warm-up procedures of cycling (10 mins) and passive heating (17 mins). However it is unknown what effect of squatting with and without VBX for 5 mins has on muscle temperature. Furthermore it is unknown if there are any differences in muscle temperature during a dynamic versus static squat with and without VBX. This may provide additional answers to understanding how VBX works as a warm-up intervention.

**Participation Recruitment**
A flyer (advertisement) was posted on the main noticeboards on the Turitea campus calling for your participation. On making contact with the principal researcher you were provided with this information sheet. The sole criterion for this study is that you are healthy and involved in regular exercise of at least 2x-week. As a participant you will undertake a familiarisation session on the use of the vibration machine followed by four interventions lasting 75 minutes in duration *(30 mins rest, followed by 10 min VBX, followed by a further 10 min rest)*. The four interventions are (1) VBX + Squatting up & down (2) no VBX+ Squatting up & down (3) VBX + Static squat (4) no VBX + Static squat
The number of participants required for this study is six. We need this number to be able to determine if there are real (i.e. statistically significant) differences between the responses of the interventions.You will be required to complete a health questionnaire. This provides a checklist to determine if you have any physical anomalies that may prevent you participating in the study. A consent form will need to be signed by you that endorses your agreement to participate in the study as described in this Information Sheet.

**Procedures**
The total duration involved for this study is approximately 280 minutes. You will need to visit the laboratory on 4 separate occasions each lasting 75 minutes. This study will be performed at the Human Performance Laboratory, Massey University, Palmerston North. Prior to each intervention, a medical professional will sterilise the insertion area of the quadricep muscle by swabbing the area with an iodine antiseptic solution in preparation for the insertion of a small temperature sensor, which measures how warm your thigh muscle gets. This sensor will be inserted into your thigh muscle via an 18-guage, 1.2 x 45-mm cannula. Given that the needle is made from stainless steel and contains nickel and given that the antiseptic solution contains iodine there is small chance that you may be allergic to these products, if so you may be excluded from the study. The needle will then be withdrawn leaving only the sensor in the muscle. The lead from the sensor will be taped down to the skin surface and the entry site treated with antiseptic solution and covered with a sterile bandage. You will then be seated in a room for 30 minutes that has a constant air temperature. You may feel some pain when needle enters into the muscle. There is also a small possibility of infection when the sensor is removed from your thigh, which may occur in later days. If this does occur you should inform us. The sensor is sterilised according to strict procedures and should minimise the risk of infection and following sterilisation it is possible the thermocouple may be used on multiple participants, however the risk is minimal due to the strict autoclaving procedures. Moreover, the insertion of the sensor needs to be performed each time for the four interventions.

**Storing of Results**
The information gained from this study will be reported at conferences and published in a research journal. Each participant will be given a unique code number, only the researcher and participant shall know the code, this is to protect confidentiality of identity. Additionally, the code will assist you in attaining feedback from the physical performance results.All paper data will be stored in a key locked cabinet in the researcher's Massey office. Where paper data is transferred to an electronic spreadsheet, this will be stored on the researcher's office desk top computer which is safeguarded by password entry.

**Participant's Rights**
You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at the time of physical testing;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 07/50. If you have any concerns about the conduct of this research, please contact Professor John O'Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8771, email humanethicsoutha@massey.ac.nz.

# Appendix 4 - Information Sheet: Comparing the Physiologic Effects of Acute Lower-Body Vibration Exercise in Young and Older People

***Name of responsible investigator(s).***

Darryl Cochrane, M.PhEd.
Francesco Sartor
Joern Rittweger, M.D., PhD.
Marco Narici, PhD, Professor
Olivier Seynnes

***Purpose of study and brief description of procedures.***
This study is investigating whether your respiration (amount of oxygen) increases while performing a series of leg exercises whilst your body is being vibrated at different loads. This will help us understand if this type of training will provide benefits as an alternative form of exercise to enhancing oxygen uptake into the muscle. After answering some questions about your general and medical histories, you will then be asked to complete a questionnaire which assesses how fit you are. You will be asked to visit the laboratory on 1 day or 2 separate days according to your choice. Testing will involve bouts of 6 minutes of vibration exercise exercise at varying intensities whilst performing leg extension with different resistance loads. The study will entail nine conditions comprising of three different vibration frequencies (26Hz, 36Hz, 44Hz) and three additional loads of 20%, 50%, 80% body weight. Also you will be asked to exercise by pulling your clasped hands apart. A specially designed machine that causes muscle contractions over the entire body will produce the vibration. This is not harmful, however in some instances the vibration at higher intensities may lead to fatigue and discomfort. If this does arise, you are free to discontinue the test without penalty. The leg exercise involves you being seated and pushing your feet against a resistive load, while being vibrated for 6 minutes.

Additionally your respiration (oxygen uptake) will be analysed, which requires you to place a special 'peg' on your nose and breath into a mouth piece that is connected to a machine. Moreover we will be recording after every minute how fast your heart beats, blood pressure and your perception of how hard (Borg scale) the condition is.All of these measurements are non-invasive and without any known risk, and none of these testing conditions will involve vigorous exercise.It is understood that you can withdraw your consent at any time during the study and discontinue your participation without giving reasons. You may also decide to refuse particular parts of the protocol. No disadvantage to you will arise from any such decision.All data obtained in this study will be treated confidentially.

***Participant Statement***
I fully understand what is involved in taking part in this study. Any questions I have about the study, or my participation in it, have been answered to my satisfaction. I understand that I do not have to take part and that I may decide to withdraw from the study at any point without prejudice. I have had my attention drawn to the document 'Ethical Regulations for the Use of Humans in Research'. My concerns regarding this study have been answered and such further concerns as I have during the time of the study will be responded to. It has been made clear to me that, should I feel that these Regulations are being infringed or that my interests are otherwise being ignored, neglected or denied, I should inform the Chair of the Ethics Committee of the Department of Exercise and Sport Science, Manchester Metropolitan University, Hassall Road, Alsager, Cheshire, ST7 2HL who will undertake to investigate my complaint.
Signed ..... Date .....

I certify that the details of this study have been fully explained and

described in writing to ..... and have been understood by him/her and that I consent to his/her participation in this study.

# Appendix 5 - Information Sheet: The Effect of Acute Lower-Body Vibration Exercise on Muscle-Tendon Complex Lengthand EMG Activity

***Supervisor/Principal Investigator***: Darryl Cochrane, MPhEd
***Investigator/Collaborators***: Prof Joern Rittweger, M.D. PhD;
Ian Loram, PhD

***Project Title***: Assessment of muscle contraction in acute vibration exercise

***Purpose of study and brief description of procedures***.

This study is investigating whether muscle actually contracts during acute vibration exposure. Past research has found that vibration exercise exercise elicits and increase in oxygen uptake which the vibration is thought to generate mechanical work as governed by the increase in oxygen turnover. However, there is no direct evidence for this argument.After answering some questions about your general and medical histories, your height, weight, leg length, knee breadth and ankle breadth will be measured. Following this, four sites of the body will be prepared by shaving the area, applying an electrolyte cream and cleaning the areas with an alcohol swap. The body regions include the calf (x2), shin and thigh. Electrodes with be placed on these sites by self adhesive tape to measure the electrical activity of the muscles, which is not painful. You will be then “marked up” with reflective markers placed on the right and left hip, knee, ankle, toe, and heel. Lastly an ultrasound probe will be attached to your calf muscle by a polystyrene cast. The ultrasound produces an image of your calf muscle by emitting sounds waves that are not harmful to the body.

You will undertake 12 vibration trials lasting 15 seconds each. A specially designed machine that causes muscle contractions over the entire body will produce the vibration. This is not harmful, however in some instances the vibration at higher intensities may lead to fatigue and discomfort. If this does arise, you are free to discontinue the test without penalty. You will stand on the machine with knees slightly bent. It is understood that you can withdraw your consent at any time during the study and discontinue your participation without giving reasons. You may also decide to refuse particular parts of the protocol. No disadvantage to you will arise from any such decision.

All data obtained in this study will be treated confidentially.

***Participant Statement***
I fully understand what is involved in taking part in this study. Any questions I have about the study, or my participation in it, have been answered to my satisfaction. I understand that I do not have to take part and that I may decide to withdraw from the study at any point without prejudice. I have had my attention drawn to the document 'Ethical Regulations for the Use of Humans in Research'. My concerns regarding this study have been answered and such further concerns as I have during the time of the study will be responded to. It has been made clear to me that, should I feel that these Regulations are being infringed or that my interests are otherwise being ignored, neglected or denied, I should inform the Chair of the Ethics Committee of the Department of Exercise and Sport Science, Manchester Metropolitan University, Hassall Road, Alsager, Cheshire, ST7 2HL who will undertake to investigate my complaint.

Signed ..... Date .....

I certify that the details of this study have been fully explained and described in writing to ..... and have been understood by him/her and that I consent to his/her participation in this study.

# Appendix 6 - Information Sheet: Acute Lower-Body Vibration Exercise Elicits Post-Activation Potentiation

**Introduction**

Darryl Cochrane (Lecturer in Sport Management & Coaching, Massey University) is the principal researcher investigating a new performance regime known as vibration exercise (VBX). It has been suggested that VBX evokes rhythmic muscle contractions similar to that of explosive power and strength training. VBX is produced by a commercialised system where an oscillating platform causing muscles to stretch and contract. Previously we have shown that acute VBX increases vertical jump power in elite female hockey players compared to control (no VBX) and cycling conditions. We speculate that the enhanced jump power from VBX may be due to changes occurring within the muscle. To date, there little information on the acute effects of VBX on muscle stimulation and stretch reflex activity, therefore by comparing VBX with other conventional modalities such as cycling it may provide additional answers to understanding how VBX works.

**Participation Recruitment**

As a participant you will undertake three interventions lasting 30 minutes in duration. These are (1) VBX (2) no VBX (3) stationary cycling.The number of participants required for this study is twelve. We need this number to be able to determine if there are real (i.e. statistically significant) differences between the responses of the treatments (VBX, no VBX, Cycling). You will be required to complete a health questionnaire. This provides a checklist to determine if you have any physical anomalies that may prevent you participating in the study. A consent form will need to be signed by you that endorse your agreement to participate in the study as described in this Information Sheet.

**Procedures**

The total duration involved for this study is approximately 120 minutes. You will need to visit the laboratory on 4 separate occasions each lasting 30 minutes. This study will be performed at the Human Performance Laboratory, Massey University, Palmerston North.

The first session is to familiarise you with the VBX equipment and other equipment. We will stimulate your muscles with brief electrical impulses (lasting 0.002 second) delivered to your thigh muscle through some adhesive disposable electrodes. This procedure will be used to identify your ability to activate your thigh muscle voluntarily. The muscle will briefly and suddenly contract and you will experience a brief discomfort. We will increase the stimulus until a plateau is reached in your muscle force production. During your familiarisation we will also record the natural electrical activity of your muscles (EMG) by placing three electrodes on your thigh. This technique is totally painless. We will also measure the reflex of patella tendon. This involves using tendon hammer that has a rubber ending, this will be connected to a custom build rig which will the allow the hammer to be raised to impact onto the patella tendon, this is painless.With any muscle functional testing there is an element of risk. However, the tests used in this study are standard assessments used through out the world. If for any reason you do sustain any injury the Human Performance Laboratory is well equipped with telephone and first aid for such incidents. Some brief physical discomfort that is well tolerated may be experienced during electrical stimulation.

**Interventions**

You will undertake in a balanced random order three treatments lasting 5 mins to be conducted on separate days with at least 24 hrs rest between interventions. Intervention 1 - VBX. Intervention 2 - No VBX; Intervention 3 – Cycling; Intervention The VBX will be performed on a vertical sinusoidal vibration machine (Galileo Sport, Novotec, Pforzheim, Germany). This is performed by standing on vibration platform for a total of 5 minutes of continuous exposure performed in a static squat position. The Galileo Sport vibration machine will be set at 26 Hz (amplitude = 4 mm). For no VBX you will stand on the vibration machine (0 Hz, amplitude = 0mm) and perform the static squat and time onstructs as the VBX intervention. For the cycling treatment you will cycle at a low intensity (70 watts for 5 minutes).

**Storing of Results**

All paper data will be stored in a key locked cabinet in the researcher's Massey office. Where paper data is transferred to an electronic spreadsheet, this will be stored on the researcher's office desk top computer which is safeguarded by password entry.. At the completion of the study a report summary of the findings will be named in a sealed enveloped for personal collection from the researcher's Massey University office.

**Participant's Rights**

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at the time of physical testing;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;

At the end of the research you will be given a report detailing the outcome of the study, this will be personally named and enveloped for collection at the researcher's university office.This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 06/62. If you have any concerns about the conduct of this research, please contact Professor John O'Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8635, email humanethicssoutha@massey.ac.nz.

# Appendix 7 - Pre-Exercise Health Screening Form

Please read the following questions carefully. If you have any difficulty, please advise the researchers who are conducting the muscular performance tests.

Please answer all of the following questions by ticking only one box for each question:
This questionnaire has been designed to identify the small number of persons (15-69 years of age) for whom physical activity might be inappropriate. The questions are based upon the Physical Activity Readiness Questionnaire (PAR-Q), originally devised by the British Columbia Dept of Health (Canada), as revised by [1]Thomas *et al.* (1992) and [2]Cardinal *et al.* (1996), and with added requirements of the Massey University Human Ethics Committee. The information provided by you on this form will be treated with the strictest confidentiality.

PERSONAL INFORMATION

Name:_____

Age:_____ Birth Date:__/_____/_____

Address:_____

Telephone:_____(hm)_____(wk)

Emergency: Contact Name:_____Telephone_____

**Q 1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?**

Yes ☐ No ☐

**Q 2. Do you feel a pain in your chest when you do physical activity?**

Yes ☐ No ☐

**Q 3. In the past month have you had chest pain when you were not doing physical activity?**

Yes ☐ No ☐

**Q 4. Do you lose your balance because of dizziness or do you ever lose consciousness?**

Yes ☐ No ☐

**Q 5. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?**

Yes ☐ No ☐

**Q 6. Do you have a bone or joint problem that could be made worse by vigorous exercise?**

Yes ☐ No ☐

**Q 7. Do you know of any other reason why you should not do physical activity?**

Yes ☐ No ☐

**Q 8. Have any immediate family had heart problems prior to the age of 60?**

Yes ☐ No ☐

**Q 9. Have you been hospitalised recently?**

Yes ☐ No ☐

**Q 10. Do you have any infectious disease that may be transmitted in blood?**

Yes ☐ No ☐

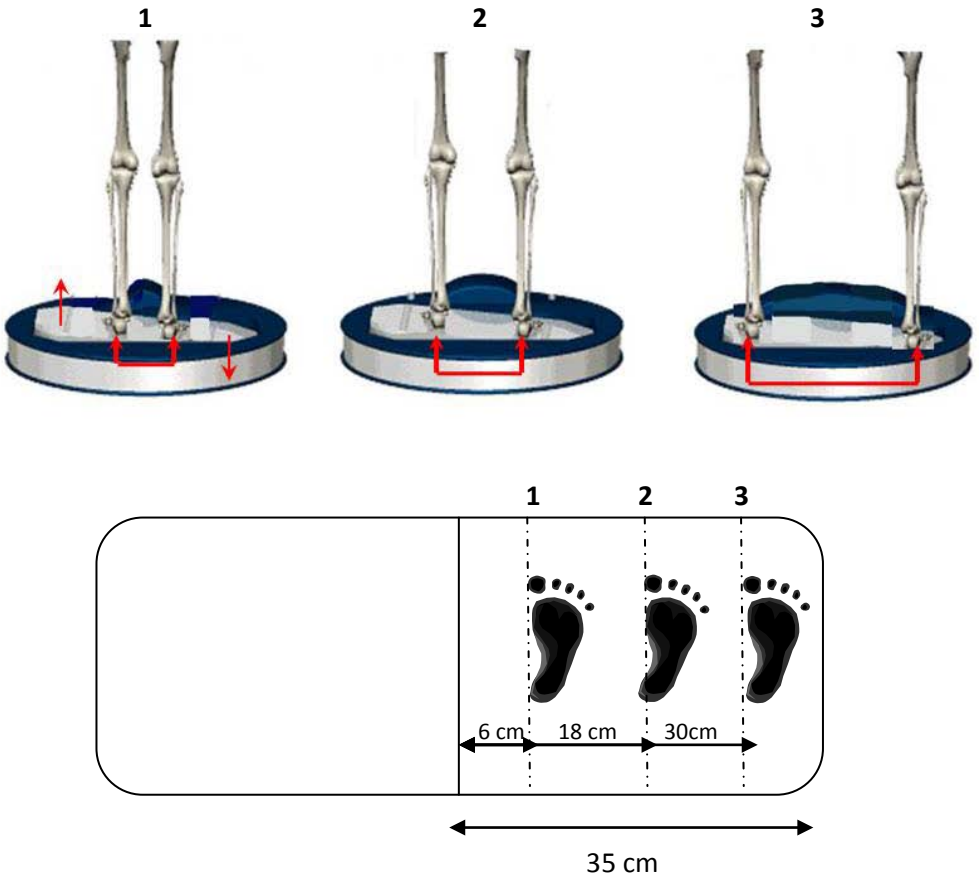
You should be aware that even amongst healthy persons who undertake regular physical activity there is a risk of sudden death during exercise. Though extremely rare, such cases can occur in people with an undiagnosed heart condition. If you have any reason to suspect that you may have a heart condition that will put you at risk during exercise, you should seek advice from a medical practitioner before undertaking the following tests.
I have read, understood and completed this questionnaire.

Signature: _____ Date: _____

**References**

1. Thomas S, Reading J and Shephard RJ. Revision of the Physical Activity Readiness Questionnaire (PAR-Q). *Can J Sport Sci* 17(4): 338-345.
2. Cardinal BJ, Esters J and Cardinal MK. Evaluation of the revised physical activity readiness questionnaire in older adults. *Med Sci Sports Exerc* 28(4): 468-472

## Appendix 8 - Calculation of Amplitude and Acceleration of SV (Galileo Sport)




**Method**

Three accelerometers (Imems ®, ADXL250, Analog Devices, Norwood, MA, USA) with a scaling factor of 76mV/g were bolted to the edge of the vibrating plate (Galileo Sport, Novotec, Germany) in the above three positions. Each accelerometer was connected to a Powerlab (8/30, and Chart v4, ADInstruments, Australia). Each accelerometer was calibrated prior to testing by holding the accelerometer level (with large holes facing upwards) which is equivalent to -1*g* then rotating half a turn (with small holes facing upwards) which is +1*g* and using the two point channel in the power lab. The acceleration channel was set for testing.

**Results**

Performed at 26 Hz

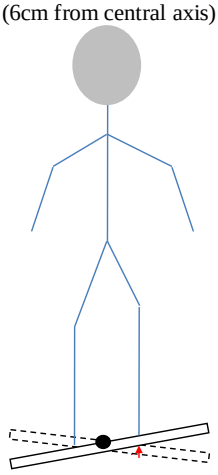
| Foot Position | 1 | 2 | 3 |
|---|---|---|---|
| **Distance from central axis** | 6cm | 18cm | 30cm |
| **Un weighted mass (0kg)** | 4.4-4.5*g* | 9.8-10*g* | 16.0-16.1*g* |
| **Weighted body mass (76kg)** | 4.4*g* | 9.6*g* | 15.9*g* |

**Calculating Peak-to-Peak Amplitude**

To calculate the peak-to-peak amplitude of the foot position. The average maximum and minimum acceleration over a given time period was computed into this integral http://integrals.wolfram.com/index.jsp

| **26 Hz (6cm from the central axis)** | | | |
|---|---|---|---|
| **Max Accel Ave** | Min Accel Ave | Peak-to-peak Accel *g* | Peak-to-peak Accel $m/s^2$ |
| 4.68*g* | -2.93*g* | -7.62*g* | -74.7 $m/s^2$ |

Small foot displacement
(6cm from central axis)



Small peak-to-peak amplitude
(2.8mm)

-74.7 sin[163.7*$x$]
0.4563cos[163.7*$x$]
0.002787m
**2.8mm** (peak-to-peak amplitude)

$a_{peak} = (2\pi f)^2 A$
$A = a_{peak} / (2\pi f)^2$
$74.7/(2 \times 3.141 \times 26)^2$
74.7 / 26687.41
0.002799m
**2.8mm** (peak-to-peak amplitude)

| **26 Hz (18cm from the central axis)** | | | |
|---|---|---|---|
| **Max Accel Ave** | Min Accel Ave | Peak-to-peak Accel *g* | Peak-to-peak Accel $m/s^2$ |
| 9.71*g* | -8.74*g* | -18.46*g* | -181.09 $m/s^2$ |

-181.09sin[163.7*$x$]
1.0623cos[163.7*$x$]
0.00675m
**6.8mm** (peak-to-peak amplitude)

| **26Hz (30cm from the central axis)** | | | |
|---|---|---|---|
| **Max Accel Ave** | Min Accel Ave | Peak-to-peak Accel *g* | Peak-to-peak Accel $m/s^2$ |
| 16.43*g* | -15.29*g* | -31.73*g* | 311.27 $m/s^2$ |

-311.27sin[163.7*$x$]
1.90147cos[163.7*$x$]
0.01161m
**11.6mm** (peak-to-peak amplitude)

# Appendix 9 - Published Papers

Journal of Science and Medicine in Sport (2008) 11, 527–534

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Journal of Science and Medicine in Sport

www.elsevier.com/locate/jsams

ORIGINAL PAPER

# The acute effect of vibration exercise on concentric muscular characteristics

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**KEYWORDS**
Muscle;
Strength;
Warm-up;
Peak power;
EMG;
Shoulder rehabilitation

**Summary** This study was designed to compare the acute effect of vibration exercise with a concentric-only activity (arm cranking) on concentric-only muscle action using an upper body isoinertial exercise. Twelve healthy, physically active men, 30.0 y ± 6.1 (mean ± S.D.); height 1.81 m ± 0.06; and weight 83.4 kg ± 9.7, performed four maximal prone bench pull (PBP) efforts before and after a 5-min period of three different interventions: (1) acute vibration exercise (VBX); (2) arm cranking (AC); and (3) control (no exercise) (NVBX). Electromyography (EMG) activity was assessed from the middle trapezius muscle during PBP. Acute VBX was induced with an electric-powered dumbbell (DB) (frequency 26 Hz, amplitude 3 mm), with 30-s exposures at five different shoulder positions. NVXB was performed with the participants holding the DB with the machine turned off, and AC was performed at 25 W. There was a significant (intervention × pre–post) interaction such that acute VBX and AC enhanced peak power by 4.8% ($p < 0.001$) and 3.0% ($p < 0.001$), respectively, compared to NVBX (−2.7%). However, there was no effect of any treatments on EMG activity compared to the control. In conclusion, acute VBX provides an acute ergogenic effect which potentiates concentric-only muscle performance, though not to a significantly greater extent than concentric (arm cranking) exercise.


## Introduction

Extensive or constant exposure to high frequency vibration is classified as a hazardous activity that can be detrimental to health.[1] However, acute low frequency vibration exercise (VBX) is commanding attention in a number of disciplines in assisting physical rehabilitation, sports conditioning and compromised health, although its efficacy remains unclear. Nevertheless, acute VBX has been shown to be effective for improving muscle strength,[2] muscle power[3] and balance.[4]

Until recently the production of acute vibration was confined to custom-made electromotor devices that were directly applied to the muscle.[5] However, commercial companies are now manufacturing

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ORIGINAL ARTICLE

# The rate of muscle temperature increase during acute whole-body vibration exercise

**D. J. Cochrane · S. R. Stannard · A. J. Sargeant · J. Rittweger**

Accepted: 24 March 2008


**Abstract** This study compared the rate of muscle temperature ($T_m$) increase during acute whole-body vibration (WBV), to that of stationary cycling and passive warm-up. Additionally we wanted to determine if the purported increase in counter-movement jump and peak power cycling from acute WBV could be explained by changes in muscle temperature. Eight active participants volunteered for the study, which involved a rest period of 30 min to collect baseline measures of muscle, core, skin temperature, heart rate (HR), and thermal leg sensation (TLS), which was followed by three vertical jumps and 5 s maximal cycle performance test. A second rest period of 40 min was enforced followed by the intervention and performance tests. The change in $T_m$ elicited during cycling was matched in the hot bath and WBV interventions. Therefore cycling was performed first, proceeded by, in a random order of hot bath and acute WBV. The rate of $T_m$ was significantly greater ($P < 0.001$) during acute WBV (0.30°C $min^{-1}$) compared to cycle (0.15°C $min^{-1}$) and hot bath (0.09°C $min^{-1}$) however there was no difference between the cycle and hot bath, and the metabolic rate was the same in cycling and WBV (19 mL $kg^{-1}$ $min^{-1}$). All three interventions showed a significant ($P < 0.001$) increase in countermovement jump peak power and height. For the 5 s maximal cycle test (MIC) there were no significant differences in peak power between the three interventions. In conclusion, acute WBV elevates $T_m$ more quickly than traditional forms of cycling and passive warm-up. Given that all three warm-up methods yielded the same increase in peak power output, we propose that the main effect is caused by the increase in $T_m$.

**Keywords** Oxygen uptake · Passive heating · Concentric-eccentric oxygen cost · Hot water immersion

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## Introduction

As shown by Asmussen and Boje (1945) raising body temperature prior to exercise has the potential to enhance short-term performance (for a recent review see Bishop 2003). Active and passive modalities are frequently used to elevate muscle and/or core temperature. Passive modalities normally involve using an approach such as hot water baths and saunas whereby externally applied heat is absorbed by the body's tissues. Active modalities require dynamic and rhythmic muscular work such as running, cycling, and skipping which metabolic heat production leads to an increase in muscle temperature.

Acute whole body vibration (WBV), a currently popular exercise modality, is receiving a lot of interest as a warm-up tool. It initiates a rapidly and repeating eccentric-concentric action which evokes muscular work and an elevation in metabolic rate (Rittweger et al. 2000). This is achieved by standing on a commercially manufactured machine with an oscillating platform, which moves in the vertical plane or tilts up and down about a central axis.

ABSTRACT: It has been suggested that vibration causes small changes in muscle length, but to the best of our knowledge, these have yet to be demonstrated during whole-body vibration (WBV). This was an observational study to determine whether acute WBV would result in muscle lengthening. We hypothesized that acute WBV would increase electromyography (EMG) activity concurrently with measurable changes in muscle contractile length. Nine healthy males performed two conditions on a Galileo vibration machine for 15 s at 0 Hz (resting) and 6 Hz at a set knee angle of 18°. Muscle tendon complex length, contractile tissue displacement of the medial gastrocnemius muscle, and EMG of soleus, tibialis anterior, and vastus lateralis muscles were measured. At 6 Hz the medial gastrocnemius (MG) muscle tendon complex (MTC) amplitude (375 μm) was significantly greater ($P < 0.05$) compared to 0 Hz (35 μm). The MG contractile length (CD) amplitude at 6 Hz (176 μm) was significantly greater ($P < 0.01$) compared to 0 Hz (4 μm). Significant increases ($P < 0.05$) in EMG modulation were found for all muscles during the 6 Hz compared to the 0 Hz condition. The major finding was that ≈50% of the elongation occurred within the muscle itself and was associated with preceding changes in EMG. This indicates muscle lengthening may be a prerequisite for eliciting stretch reflexes. In conclusion, there is a temporal association between EMG activity and muscle contractile tissue displacement where low-frequency WBV results in small muscle length changes and increases muscle activation.

*Muscle Nerve* **40:** 420–429, 2009

# CHANGES IN JOINT ANGLE, MUSCLE-TENDON COMPLEX LENGTH, MUSCLE CONTRACTILE TISSUE DISPLACEMENT, AND MODULATION OF EMG ACTIVITY DURING ACUTE WHOLE-BODY VIBRATION

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*Accepted 27 January 2009*

Whole-body vibration (WBV) is becoming very popular in the health, rehabilitation, and exercise sectors,[7,8,19,22,24] but the scientific investigation of WBV lags behind its application. Acute and chronic WBV studies where vibration is applied to the lower limbs have been shown to increase vertical jump height[8] and measures of lower body strength.[12,18] A range of WBV frequencies have been successful in eliciting various responses. For example, WBV frequencies greater than 25 Hz have been shown to increase muscular performance[8,12,24] while low-frequency (6 Hz) WBV has been observed to increase cerebral oxygenation,[16] a change which is strongly linked to greater neuronal activation.[25]

The augmented responses of WBV have been attributed to the "tonic vibration reflex" (TVR).[11] This effect is based on the stretch reflex response[5,21] where vibration applied to the tendon activates primary muscle spindle afferents, stimulating the alpha

**Abbreviations:** CD, contractile length; EMG, electromyography; MG, medial gastrocnemius; MTC, muscle tendon complex; PEH, perievent histogram; SOL, soleus; TA, tibialis anterior; TVR, tonic vibration reflex; VL, vastus lateralis; WBV, whole body vibration

**Key words:** muscle length; contractile element; isometric contraction

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ORIGINAL ARTICLE

# A Comparison of the Physiologic Effects of Acute Whole-Body Vibration Exercise in Young and Older People

*Darryl J. Cochrane, MPhEd, Francesco Sartor, MSc, Keith Winwood, PhD, Stephen R. Stannard, PhD, Marco V. Narici, PhD, Jörn Rittweger, MD, PhD*

ABSTRACT. Cochrane DJ, Sartor F, Winwood K, Stannard SR, Narici MV, Rittweger J. A comparison of the physiologic effects of acute whole-body vibration exercise in young and older people. Arch Phys Med Rehabil 2008;89:815-21.

**Objective:** To examine the acute physiologic effects of acute whole-body vibration (WBV) exercise in young and older people.

**Design:** Every participant performed 9 conditions in a static squat position, consisting of no vibration and WBV at 30Hz and 3 loads corresponding to (1) no load (0% body mass), (2) load of 20% body mass, and (3) load of 40% body mass. A Jendrassik voluntary contraction was also performed with no vibration and WBV at 30Hz with no load and 20% body mass.

**Setting:** Laboratory facilities at a university in the United Kingdom.

**Participants:** Healthy young people (n=12; 6 men, 6 women; mean age, 21.5y) and 12 healthy older people (6 men, 6 women; mean age, 69.2y) from the local community.

**Interventions:** Not applicable.

**Main Outcome Measures:** The Physical Activity Questionnaire, anthropometric measures, counter-movement jump, and isometric maximal voluntary contraction with the Jendrassik maneuver were assessed in both groups. Oxygen uptake ($\dot{V}o_2$), blood pressure, heart rate, and rating of perceived exertion (RPE) were recorded during WBV and load conditions as the outcome of the study.

**Results:** Both vibration and load were associated with an increase ($P<.001$) in $\dot{V}o_2$ for older and young groups. WBV elicited the equivalent of a .35 metabolic equivalent (MET) increase in $\dot{V}o_2$, with additional loads of 20% and 40% body mass increasing $\dot{V}o_2$ by 0.8 and 1.2 METs, respectively. Additionally, there was an interaction effect of vibration and group in which the WBV-related $\dot{V}o_2$ increase was less in the old compared with the young. Both vibration and load caused an increase in heart rate, blood pressure, and RPE (all $P<.001$); however, there were no significant group differences between young and older groups. The Jendrassik maneuver elicited an increase in $\dot{V}o_2$ by 27.6% for the old and 33% for the young group ($P<.001$); however, there was no significant difference between groups.

**Conclusions:** $\dot{V}o_2$ significantly increased in both the older and young people with vibration and additional load and when the Jendrassik maneuver was superimposed with vibration and load. However, the elicited increase in $\dot{V}o_2$ ($1.2mL \cdot kg^{-1} \cdot min^{-1}$) from WBV may be an insufficient stimulus to improve cardiovascular fitness.

**Key Words:** Elderly, frail; Exercise; Oxygen; Rehabilitation.



EXERCISE IS GENERALLY advocated as a countermeasure to offset age-related frailty and to enhance mobility and well-being. It has been widely documented that the natural aging process is associated with reduced muscular and cardiovascular function, bone loss, and increased body fat storage,[1] all of which contribute to a decline in functional performance.[2] Over time, in combination with a sedentary lifestyle, further deterioration may lead to a greater reduction in mobility, impaired balance, and a higher incidence of falls.[3] Furthermore, the decline in muscle function not only involves a loss of muscle strength but also of muscle power. Margaria et al[2] were the first to report a decline in maximum muscular power with age and found from 20 to 70 years of age muscle power is reduced by about a half, with poor muscle power being a predictor of hospitalization, falls, and fracture.[4] Although the fact of physiologic decline may not be avoided, it can be mitigated by training, even at a very old age.[5] However, factors such as time, convenience, poor compliance through dementia, and poor postural control after a stroke often preclude older people from engaging in physical activity.[6] An exercise modality that is convenient, time efficient, and has the benefits of conventional weight-bearing exercise would be appealing to this group.

One such modality, known as whole-body vibration (WBV) exercise, has recently received some attention as a regimen to overcome these obstacles while producing favorable outcomes.[7-9] WBV requires specialized equipment; however, it can be performed in the convenience of the home and is readily available from commercial companies. Manufactured devices such as a handheld dumbbell and standing and seated oscillating platforms produce the vibration. These devices deliver sinusoidal vibrations to the body at a frequency of 5 to 45Hz. As little as 6 to 10 minutes of WBV a day 3 times a week for 6 to 8 weeks has been shown to increase balance and gait,[10-12] improve quality of life,[12] and improve exercise compliance.[6] Additionally, longer duration (6–12mo) WBV studies in postmenopausal women have documented increases in bone mineral density of the hip[13] and spine.[14]

The mechanism of WBV has yet to be elucidated; however, it has been proposed that WBV involves monosynaptic reflexes that are induced by the stretch-shortening action in the muscles that act over the joints in which the vibration is being absorbed.[15] Electromyographic activity has been shown to increase during WBV,[16,17] and it has been purported that WBV elicits muscular activity through evoking sufficient muscular work

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ORIGINAL ARTICLE

# Acute whole-body vibration elicits post-activation potentiation

**Darryl J. Cochrane · Stephen R. Stannard · Elwyn C. Firth · Jörn Rittweger**

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**Abstract** Whole-body vibration (WBV) leads to a rapid increase in intra-muscular temperature and enhances muscle power. The power-enhancing effects by WBV can, at least in part, be explained by intra-muscular temperature. However, this does not exclude possible neural effects of WBV occurring at the spinal level. The aim of this study was to examine if muscle twitch and patellar reflex properties were simultaneously potentiated from an acute bout of WBV in a static squat position. Six male and six female athletes performed three interventions for 5 min, static squat with WBV (WBV+, 26 Hz), static squat without WBV (WBV−) and stationary cycling (CYCL, 70 W). Transcutaneous muscle stimulation consisting of a single 200 μs pulse and three patellar tendon taps were administered prior to and then 90 s, 5, 10 min post-intervention. Ninety-seconds after WBV+ muscle twitch peak force (PF) and rate of force development (RFD) were significantly higher ($P < 0.01$) compared to WBV− and CYCL. However the patellar tendon reflex was not potentiated. An acute continuous bout of WBV caused a post-activation potentiation (PAP) of muscle twitch potentiation (TP) compared to WBV− and CYCL indicating that a greater myogenic response was evident compared to a neural-mediated effect of a reflex potentiation (RP).

**Keywords** Twitch · Muscle contractile properties · Stretch reflex · Electrical stimulation · Rate of force development

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## Introduction

Acute vertical sinusoidal whole-body vibration (WBV), eliciting rapid eccentric/concentric of the leg extensors (Cardinale and Bosco 2003; Rittweger et al. 2003; Rittweger et al. 2001), improves performance of these muscles in the short-term (Cochrane and Stannard 2005; Cochrane et al. 2008b; Cochrane et al. 2008c; Torvinen et al. 2002). This transient effect is thought to be mediated by a rapid reflex-mediated stretch-shortening (Cardinale and Bosco 2003; Rittweger et al. 2003; Rittweger et al. 2001) likely to involve the tonic vibration reflex (TVR), which stimulates the muscle spindles (Cardinale and Bosco 2003; Rittweger et al. 2001). Practically, WBV application leads to enhanced anaerobic power (Cochrane and Stannard 2005; Cochrane et al. 2008c). Previous work from this laboratory demonstrates that WBV leads also to a rapid increase in intra-muscular temperature (Cochrane et al. 2008b). Intra-muscular temperature in itself enhances muscle power (de Ruiter and de Haan 2000). Therefore, it seems that the power-enhancing effects by WBV can, at least in part, be

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