Acute and chronic neuromuscular adaptations to local vibration training

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INVITED REVIEW

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Abstract Vibratory stimuli are thought to have the potential to promote neural and/or muscular (re)conditioning. This has been well described for whole-body vibration (WBV), which is commonly used as a training method to improve strength and/or functional abilities. Yet, this technique may present some limitations, especially in clinical settings where patients are unable to maintain an active position during the vibration exposure. Thus, a local vibration (LV) technique, which consists of applying portable vibrators directly over the tendon or muscle belly without active contribution from the participant, may present an alternative to WBV. The purpose of this narrative review is (1) to provide a comprehensive overview of the literature related to the acute and chronic neuromuscular changes associated with LV, and (2) to show that LV training may be an innovative and efficient alternative method to the 'classic' training programs, including in the context of muscle deconditioning prevention or rehabilitation. An acute LV application (one bout of 20–60 min) may be considered as a significant neuromuscular workload, as demonstrated by an impairment of force generating capacity and LV-induced neural changes. Accordingly, it has been reported that a training period of LV is efficient in improving muscular performance over a wide range of training (duration, number of session) and vibration (frequency, amplitude, site of application) parameters. The functional improvements are principally triggered by adaptations within the central nervous system. A model illustrating the current research on LV-induced adaptations is provided.

Keywords Local vibration · Training · Neuromuscular fatigue · Neuromuscular adaptations · Rehabilitation

Abbreviations

ACL	Anterior cruciate ligament
CSP	Cortical silent period
EMG	Electromyography
ICF	Intracortical facilitation
LICI	Long-interval cortical inhibition
LV	Local vibration
LVT	Local vibration training
MEP	Motor evoked potential
MRI	Magnetic resonance imaging
MVC	Maximal voluntary contraction
PET	Positron emission tomography
rMV	Repeated muscle vibration
SICI	Short-interval cortical inhibition
TMS	Transcranial magnetic stimulation
TVR	Tonic vibration reflex
WBV	Whole body vibration

Introduction

Since pioneering work that aimed to develop athlete's maximal strength (Nazarov and Spivak 1987), mechanical vibratory stimuli have been considered as a training or rehabilitation modality. Whole-body vibration (WBV) training, where the participant must stand or squat on a vibrating platform while vibration is transmitted through the feet to the targeted pre-contracted muscles,

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is particularly well documented (for review, see Cochrane 2011; Rittweger 2010). For instance, WBV has been shown to increase maximal strength over 4- to 24-week training programs in healthy participants (Roelants et al. 2004; Schlumberger et al. 2001; Verschueren et al. 2004), although other studies failed to demonstrate similar findings (de Ruiter et al. 2003; Delecluse et al. 2005). Despite its advantages and promising perspectives in the clinical and rehabilitation fields (Aaboe et al. 2009; Moezy et al. 2008; Pamukoff et al. 2016; Pedro et al. 2014), WBV programs may not be suitable to everyone. Indeed, some participants are unable to bear weight due to (1) long-term immobilization (lower limb trauma), (2) immobilization by a cast, or (3) being in the early rehabilitation period after surgery (anterior cruciate ligament reconstruction). These individuals may not be able to benefit from WBV because they cannot squat or stand on the vibrating platform.

For those individuals, local vibration (LV) could be an alternative form of vibration training. LV requires the use of a small and portable device that applies vibration directly over the muscle or the tendon (Lapole and Pérot 2010). The main interest of LV compared to WBV is that it does not necessarily require any active contribution from the participant (i.e. it can be used while the person is seated or lying down). LV is not an entirely novel concept and in fact, it has actually been used for a long time in the field of neuroscience. Indeed, LV applied to the tendon can induce a number of physiological effects such as a tonic vibration reflex or muscle movement illusion depending on the experimental conditions (see "Neurophysiological responses associated with local vibration"). These are not the only physiological changes associated with LV and instead, several other neuromuscular adaptations occur at spinal and supra-spinal levels during and after LV. The purpose of this narrative review is to provide a comprehensive overview of the literature related to the acute and chronic neuromuscular changes associated with LV. We will review evidence that shows that acute LV application may be considered as a significant neuromuscular workload, as indicated by LV-induced fatigue (reduction of maximal voluntary strength) (see "Local vibration as a stimulus for neuromuscular adaptation"). Next, we will outline the immediate and long-lasting LV-induced alterations in neuromuscular properties that are dependent on the LV characteristics (e.g. the duration of application, LV frequency, contracted vs. relaxed muscle). In line with the acute neuromuscular adaptations, we will provide evidence that chronic use of LV may improve strength by inducing several neuromuscular adaptations at different levels of the corticospinal pathway, suggesting that it could be used as an innovative and efficient alternative method to other forms of training (resistance training, WBV training) (see "Chronic effects of local vibration training"). Finally, we will discuss the perspectives of using LV in the context of muscle deconditioning prevention or rehabilitation (see "Local vibration training and clinical applications").

Articles were searched through the PubMed database (up to March 2017) using the keywords "muscle vibration", "tendon vibration" or "local vibration". Articles were then checked for pertinent content and included in the current review when investigating acute (single session) or chronic (repeated sessions) effects of local vibration on neuromuscular function and functional capacities of healthy or clinical participants. Further articles were considered after scanning the reference lists of the included articles. Only articles published in English were included.

Neurophysiological responses associated with local vibration

What receptors are sensitive to local vibration?

Several receptors are known to be sensitive to vibratory stimuli. First, cutaneous receptors such as Pacini, Meissner, Ruffini or Merkel receptors are all responsive to LV at varying frequencies (Hunt 1961; Ribot-Ciscar et al. 1996; Roll and Vedel 1982). Golgi tendon organs are also responsive to LV through Ib afferents (Burke 1980; Burke et al. 1976a, b; Hayward et al. 1986). Yet, it has been well reported that muscle spindles, which encode information about the dynamic characteristics of movement (Cordo et al. 1993), are the most responsive receptors to LV. Hunt (1961) was the first to report the sensitivity of muscle spindles to LV in the cat, and follow-up research has confirmed that these findings are applicable to other animal species (Bianconi and Van der Meulen 1963; Matthews 1966). Muscle spindles are innervated by primary (i.e. Ia) and secondary (i.e. II) afferents with the Ia afferents being the most responsive, as shown in animal (Matthews 1964) and human experiments (Burke 1980; Burke et al. 1976a, b; Roll et al. 1989). The level of Ia afferent response to LV is mainly dependent on the LV amplitude and frequency. Indeed, it has been reported that small LV amplitudes (i.e. 0.2–0.5 mm) preferentially recruit Ia afferents (Roll and Vedel 1982), whereas Ib and II afferents predominantly respond to larger amplitudes (Roll et al. 1989). While group II afferents can be recruited with input frequencies between 20 and 60 Hz, the rate of discharge of Ia afferents is proportional to LV frequency in a one-to-one manner for all frequencies up to 120 Hz (Burke et al. 1976a; Roll et al. 1989). For higher frequencies (>150 Hz), the discharge of Ia afferents has been shown to be disharmonious or even unpredictable (i.e. the discharge rate loses one-to-one synchrony with LV frequency) (Burke et al. 1976a; Martin and Park 1997; Roll and Vedel 1982; Roll et al. 1989).

The response of Ia afferents is not rigid and instead is dependent on the state of the muscle, i.e. stretched, relaxed,

or contracted (Brown et al. 1967; Burke 1980; Burke et al. 1976b). Ia afferents are more responsive to LV when the muscle is stretched and during voluntary isometric contractions (Burke 1980; Burke et al. 1976a), probably due to the presence of alpha–gamma co-activation (Vallbo 1974) and/or superior transmission through the musculo-tendinous structures (Burke et al. 1976a), respectively. It should be noted, however, that during WBV where vibration is transmitted through the feet via a platform, vibration transmission can be conversely impaired due to the dampening of mechanical energy by soft tissues (Abercromby et al. 2007; Wakeling et al. 2002).

The tonic vibration reflex

As the discharge of Ia afferents elicits an excitatory input on alpha motoneurons, a reflex contraction of the vibrated muscle has been widely described as the tonic vibration reflex (TVR), which has been observed during LV (De Gail et al. 1966; Eklund and Hagbarth 1965; Hagbarth and Eklund 1966; Nakajima et al. 2009; Park and Martin 1993) and WBV (Zaidell et al. 2013). The TVR reflects an excitatory state mediated by polysynaptic pathways converging onto monosynaptic excitatory inputs (Matthews 1966; Romaiguere et al. 1991). The TVR is also known to be associated with relaxation of the antagonist due to reciprocal inhibition (Hagbarth and Eklund 1966). Theoretically, TVR occurs in almost all muscles for frequencies in the range of 20–200 Hz (Eklund and Hagbarth 1966), but has not systematically been observed. For instance, several studies employing LV frequencies in the 20–200 Hz range were subthreshold for the evocation of a TVR when a low LV amplitude (≤1.5 mm) was used (Lapole et al. 2015b; Rosenkranz et al. 2003; Rosenkranz and Rothwell 2003; Siggelkow et al. 1999; Ushiyama et al. 2005). This contrasts the early work of Eklund and Hagbarth (1966) who demonstrated that TVR was present for amplitudes of 0.6 mm, but was greater using a 1.8-mm LV amplitude. More recent studies have confirmed the work of Eklund and Hagbarth (1966) and reported that TVR can be obtained with a low LV amplitude (≤1 mm) (Martin and Park 1997; Nakajima et al. 2009). Hence, the TVR appears to be a complex response depending not only on the combination of optimal LV frequency and amplitude but also on other experimental parameters such as the muscle, the vibration site (muscle vs. tendon), the initial muscle length, the state of contraction (relaxed vs. contracted) or the visual control (i.e. closed vs. open eyes) (Eklund and Hagbarth 1966; Roll et al. 1980). Moreover, it has been reported that the TVR can be voluntary suppressed under the influence of descending pathways altering the transmission in the polysynaptic component of the reflex rather than the monosynaptic one (Burke et al. 1976a; Lance 1973). Thus, the optimal experimental parameters to induce TVR are still unclear. Furthermore, it may be difficult to determine the occurrence of the TVR due to common presence of artefacts in electromyography (EMG) data resulting from electrode/cable motion and nearby electrical noise that may influence the measurements (Abercromby et al. 2007; Fratini et al. 2009). Therefore, WBV studies that investigated EMG amplitude during vibration exposure have attenuated artefacts in raw EMG amplitude with spectral smoothing procedures (Lienhard et al. 2015; Zaidell et al. 2013). While such a methodological approach has, to our knowledge, not been used in the context of LV, this could be utilized to isolate LV effects from movement artefact.

Local vibration-induced cortical activation and proprioceptive illusions

Due to cortical projections of muscle afferents (Mima et al. 1999), LV may induce proprioceptive illusions (Goodwin et al. 1972; Naito and Ehrsson 2001; Roll and Vedel 1982; Roll et al. 2012). For instance, Goodwin et al. (1972) reported that LV applied to either biceps or triceps tendons (100 Hz; 0.5 mm) induced a systematic misjudgment of the elbow angle. These proprioceptive illusions were mainly attributed to the activation of Ia afferents (Goodwin et al. 1972; Roll and Vedel 1982), but the role of other receptors cannot be totally ruled out (Pacinian corpuscules, Golgi tendon organs). Indeed, higher neural centres at the cortical level may have interpreted the Ia afferent discharge as if they were due to excitation of the spindles by actual muscle stretching (Goodwin et al. 1972). These proprioceptive illusions have been related to the presence of evoked potentials (i.e. cortical activation) on the primary somatosensory cortex of the contralateral vibrated arm, with greater LV-induced evoked potentials observed when the LV frequency was 80 Hz compared to 40 and 160 Hz (Munte et al. 1996). Positron emission tomography (PET) studies further demonstrated that LV-induced proprioceptive illusions activate the same motor cortical areas as the ones activated during voluntary contraction, i.e. contralateral sensorimotor, premotor, and parietal cortices, as well as bilateral supplementary and cingulate motor areas (Naito and Ehrsson 2001; Naito et al. 1999; Radovanovic et al. 2002). Using magnetic resonance imaging (MRI), Romaiguère et al. (2003) reported similar findings when movement illusions were induced by LV. These authors also observed no or minimal activation of motor-related areas when no illusory movement was perceived.

Local vibration as a stimulus for neuromuscular adaptation

While transient muscle or tendon vibration strongly activates Ia afferents that project their excitatory synaptic inputs to the

spinal cord and cortical areas (see above), it may be questioned whether prolonged (i.e. 20–60 min) LV exposure may be sufficient to induce neuromuscular fatigue and thus, be a potential stimulus for neuromuscular adaptations. Accordingly, many LV studies have investigated acute changes in force output during or following a single LV exposure. It is worth noting that few studies have also considered the potential effect of acute LV proposed for shorter periods as a warm-up intervention (Cochrane 2016b; Pamukoff et al. 2014), in a similar way as it is classically performed for WBV (for a review, see Cochrane 2013). Despite some positive results, likely explained by peripheral ergogenic effects (increased muscle temperature, post-activation potentiation) (Cochrane 2016a), others failed to show any improvement in muscular performance (Cochrane 2016b; Cochrane and Hawke 2007; Pamukoff et al. 2016). Given the lack of consensus in this scientific framework, it is difficult to draw conclusions about the effects of LV as a warm-up modality, and further studies are needed on this topic.

Acute effects of local vibration on neuromuscular output

Local vibration-induced motor performance impairment

Studies that investigated acute changes in neuromuscular output following a single session of prolonged (20–30 min) LV exposure have been summarized in Table 1, which provides detailed information on experimental (participants characteristics, tested muscles) and LV (frequency, amplitude, vibration site) characteristics. 10 out of 16 reviewed LV studies demonstrated a short-term decrease in force-generation capacity of the vibrated muscle group. Indeed, force production decrement (mean 10.2%; range 4.3–19%) was reported following prolonged LV for knee extensor (Jackson and Turner 2003; Konishi et al. 2002, 2009; Kouzaki et al. 2000; Richardson et al. 2006; Saito et al. 2016a), plantar flexor (Herda et al. 2009; Ushiyama et al. 2005; Yoshitake et al. 2004) and first dorsal interosseus muscles (Shinohara et al. 2005). A force decrement has also been demonstrated on the contralateral non-vibrated limb (Jackson and Turner 2003). Conversely, other studies failed to demonstrate maximal force production impairment on plantar flexor (Cattagni et al. 2016; Ekblom and Thorstensson 2011), dorsiflexor (Farabet et al. 2016) and knee extensor muscles (Fry and Folland 2014; Saito et al. 2016b), while one study reported a 8.1% increase in knee extensors maximal voluntary contraction (MVC) after prolonged LV (Iodice et al. 2011). The LV parameters in the latter study, however, were not clear enough to make comparison with the other reviewed studies. Although it is difficult to identify which differences in study design cause the discrepancies in the main findings, we suggest that differences in the combination of vibratory parameters (i.e. time of application, frequency, amplitude, site of application, muscle investigated) offer the most likely explanation (see Table 1). Although there are inconsistencies in the effects of LV, there is evidence that LV-induced neuromuscular alterations may result in fatigue in contracting muscles exposed to LV. For instance, LV has been demonstrated to decrease the endurance time of sustained submaximal voluntary contractions (Rothmuller and Cafarelli 1995) and to induce a faster force decline during sustained MVC (Bongiovanni et al. 1990; Färkkilä et al. 1980; Samuelson et al. 1989). In the following section, we will discuss the possible causes of LV-induced fatigue.

Aetiology of local vibration-induced neuromuscular fatigue

As for neuromuscular fatigue induced by ‘classic’ exercise, LV-induced fatigue may in theory arise from central and/or peripheral changes. Since the TVR described above is a tonic contraction of small amplitude, a deficit occurring downstream of the neuromuscular junction (i.e. peripheral fatigue) is unlikely. Moreover, LV-induced fatigue may be observed without evocation of a TVR (Ushiyama et al. 2005). Unfortunately, there is a lack of information about the presence of TVR during the vibratory exposure, aside from the study of Kouzaki et al. (2000) where the presence of rectus femoris muscle electromyographic (EMG) responses to LV is reported (although not synchronous with LV frequency; see Fig. 2). Although studies that addressed the question of muscular effects of LV are very scarce, it has been consistently reported that electrically elicited twitch amplitude (Fry and Folland 2014; Herda et al. 2009; Saito et al. 2016a, b; Ushiyama et al. 2005) and twitch characteristics (i.e. contraction time and half relaxation time) (Ushiyama et al. 2005) are unchanged after a 20-30-min prolonged LV period, using LV frequencies in the range 70-100 Hz and amplitudes in the range 1-1.5 mm (Table 1). This finding suggests that LV-induced decreased MVC are not due to alterations in muscle properties. Maximal muscle response waves (M-wave) were not affected after LV either, suggesting the absence of neuromuscular propagation failure (Cattagni et al. 2016; Ekblom and Thorstensson 2011; Farabet et al. 2016; Fry and Folland 2014; Saito et al. 2016a, b; Ushiyama et al. 2005). Hence, a reduction in the neural command probably fully explains the decrement in force-generation capacity after prolonged LV. This is supported by a decrease in maximal EMG amplitude on soleus (Herda et al. 2009), gastrocnemii (Ushiyama et al. 2005; Yoshitake et al. 2004), knee extensor (Jackson and Turner 2003; Konishi et al. 2002, 2009; Kouzaki et al. 2000; Richardson et al. 2006) and first dorsal interosseous (Shinohara et al. 2005) muscles, after 20–30 min of LV with frequencies between 30-100 Hz and amplitudes between 1.5-3 mm. Conversely, other studies failed to demonstrate any variation in EMG

Table 1 Acute effects of prolonged local vibration on force-generation capacities

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Duration (min)	Fr (Hz)	A (mm)	Main outcomes
Kouzaki et al. (2000)	8 HE (7 M, 1F)	25 ± 2	COD	RF (proximal portion)	30	30	2–3	MVC: ↓ ≈10% RFD: ↓ ≈25% EMG: RF ↓ ≈15%, VL and VM →
Konishi et al. (2002)	10 ACL (5 M, 5 F)	22 ± 5	No	Infrapatellar tendon	20	50	1.5	MVC: → (+0.4%) EMG: VL → (−4.1%), VM → (−3.1%), RF → (−2.2%)
	12 HE (8 M, 4 F)	25 ± 4						MVC: ↓ 9.0% EMG: VL ↓ 18.1%, VM ↓ 14.3%, RF ↓ 15.8%
Jackson and Turner (2003)	10 HE M	26 ± 2	COD	RF	30	30	1.5–2.0	MVC: ↓ 6.9% RFD: ↓ 33.3% EMG: RF ↓ 30%, VL → (+15%) Similar contralateral effect for MVC and RFD
						120		MVC: ↓ 4.3% RFD: ↓ 20.9% EMG: RF and VL → Similar contralateral effect for MVC and RFD
Yoshitake et al. (2004)	8 HE M	27 ± 4	CG	Achilles tendon	30	100	1.5	MVC: ↓ 19% EMG: GM ↓ 32%, GL ↓ 12%, SOL → (−12%)
Ushiyama et al. (2005)	13 HE M	22–49	COD	Achilles tendon	30	100	1.5	MVC: ↓ 16.6% EMG: GM ↓ 12.7%, GL ↓ 11.4%, SOL → (− 3.4%)
Shinohara et al. (2005)	20 HE	22 ± 3	CG	FDI muscle belly	30	75	NR	MVC: ↓ 15% EMG: FDI ↓ 14%
Richardson et al. (2006)	14 HE	23 ± 1	No	Infrapatellar tendon	20	50	1.5	MVC: ↓ 7.2% EMG: VL ↓ 11.1%, VM ↓ 15.2%, RF ↓ 8.9%
	14 ACL	22 ± 1						MVC: → (+4.7%) EMG: VL → , VM → (+5.0%), RF → (+5.4%)
	7 EL HE	66 ± 1						MVC: → (−0.3%) EMG: VL → (+9.1%), VM and RF →
Herda et al. (2009)	15 HE M	24 ± 3	COD	Achilles tendon	20	70	NR	MVC: ↓ ≈5% EMG: SOL ↓ ≈22%, MG ↓ ≈7% VA_{PNS}: →

Table 1 (continued)

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Duration (min)	Fr (Hz)	A (mm)	Main outcomes
Konishi et al. (2009)	8 HE M	18 ± 0	No	Infrapatellar tendon	20	50	1.5	CON and ECC: ↓ 15.5% and ↓ 17.4% EMG CONC: VL ↓ 12.6%, VM ↓ 5.4%, RF → (−5.6%) EMG ECC: VL ↓ 16.6%, VM ↓ 19.2%, RF → (−17.0%)
Ekblom and Thorstensson (2011)	8 HE F	23 ± 2	COD	Achilles tendon	30	100	NR	CON: → (− ≈8.5%) ECC: → (− ≈7.0%) EMG: SOL and GM →
Iodice et al. (2011)	18 HE	21 ± 4	CG	VI, RF, VL, VM, GMA, BF, TA, G	30	300	2	Bilateral knee extension MVC: ↑ 8.1%
Fry and Folland (2014)	18 HE M	20 ± 2	COD	Infrapatellar tendon	30	80	1.5	MVC: → EMG VL, VM and RF →
Cattagni et al. (2016)	10 HE M	22 ± 4	COD	Achilles tendon	30	40	0.2	MVC: → (−2.5%) VA_{PNS}: → (+1.3%) EMG: SOL and GM →
						100		MVC: → (−8.4%) VA_{PNS}: → (−3.3%) EMG: SOL and GM →
Farabet et al. (2016)	13 HE (9 M, 4 F)	23 ± 4	COD	TA muscle belly	30	100	1	MVC: → (+1.7%) EMG: TA → (+3.8%) VA_{TMS}: TA → (+0.6%)
Saito et al. (2016a)	9 HE M	25 ± 4	COD	Patellar tendon (mid-portion)	30	80	1	MVC: ↓ 7.2% EMG: VM ↓ 11.0%, VI → (−6.0%), VL → (−0.4%), RF → (+1.0%)
Saito et al. (2016b)	9 HE M	25 ± 4	COD	VI (superficial region)	30	80	1	MVC: → (−3.6%) EMG: VI → (−1.7%), VL → (−3.3%), VM → (+4.8%), RF → (−0.8%)

A vibration amplitude, *ACL* subjects who had undergone anterior cruciate ligament surgery in the last 6 months, *BF* biceps femoris, *COD* cross-over design, *CON* maximal voluntary concentric contraction, *CG* control group, *ECC* maximal voluntary eccentric contraction, *EMG* electromyography, *F* female, *FDI* first dorsal interosseus, *Fr* vibration frequency, *G* gastrocnemius, *GL* gastrocnemius lateralis, *GM* gastrocnemius medialis, *GMA* gluteus maximus, *HE* healthy population, *M* male, *MVC* isometric maximal voluntary contraction, *NR* not reported, *RF* rectus femoris, *RFD* rate of force development, *SOL* soleus, *TA* tibialis anterior, VA_{PNS} voluntary activation assessed via peripheral nerve stimulation, VA_{TMS} voluntary activation assessed via transcranial magnetic stimulation, *VI* vastus intermedius, *VL* vastus lateralis, *VM* vastus medialis, ≈ mean changes estimated from values displayed on a graph, → not statistically significant, ↑ statistically significant increase, ↓ statistically significant decrease

amplitude on soleus (Cattagni et al. 2016), knee extensor (Fry and Folland 2014) or tibialis anterior (Farabet et al. 2016) muscles in line with unchanged muscular performance (Table 1). Since LV-induced excitatory Ia afferents project to both the spinal cord and higher cortical structures, the decreased MVC observed after LV exposure may be related to an alteration in neural drive occurring at a spinal and/or supra-spinal level.

Acute effects of local vibration on spinal cord excitability

The role of Ia afferents on force production capacities

It is well known that Ia afferents facilitate neural activation during voluntary contraction (Gandevia 1998). Indeed, when LV is superimposed during brief submaximal contractions, an enhancement in contraction force level and EMG amplitude activity is reported due to the supplemental Ia excitatory input onto alpha motoneurons (Kihlberg et al. 1995; McCloskey et al. 1974; Mosier et al. 2016). Similarly, increased MVC (Curry and Clelland 1981; Humphries et al. 2004; Samuelson et al. 1989) and power (Issurin and Tenenbaum 1999; Liebermann and Issurin 1997) has been reported during the first seconds of superimposed LV using 20 (Samuelson et al. 1989), 44 (Issurin and Tenenbaum 1999), 50- (Humphries et al. 2004) and 120-Hz LV frequencies (Curry and Clelland 1981). Such LV-induced increases of muscle performance may only be observed when central activation is incomplete. Accordingly, similar findings have been reported when the contracted muscles were in a fatigued state, or after peroneal nerve block by partial anaesthetic, which induces reduced activity of gamma motoneurons (Bongiovanni and Hagbarth 1990). Yet, when LV exposure is prolonged, there is a LV-induced central drive impairment due to decreased alpha motoneuronal activity through attenuation of Ia afferent inputs onto alpha motoneurons (Bongiovanni and Hagbarth 1990; Bongiovanni et al. 1990) (see below). Indeed, most studies that reported a force decrement after prolonged LV suggested an altered spinal loop excitability as the proposed mechanism (Herda et al. 2009; Jackson and Turner 2003; Konishi et al. 2009; Kouzaki et al. 2000; Richardson et al. 2006; Ushiyama et al. 2005). This is confirmed by the studies of Konishi et al. (2002) and Richardson et al. (2006) showing force decrement on knee extensor muscles in healthy young participants after prolonged LV (−9.0 and −7.2%, respectively). Conversely, the same vibratory stimulus failed to elicit similar results in young participants who had undergone ACL reconstruction, as well as in healthy elderly participants (Richardson et al. 2006), i.e. two populations who are known to present with impairments in gamma-loop function.

Local vibration-induced decreased spinal loop excitability

Spinal loop excitability is commonly assessed through the Hoffmann reflex (H-reflex), considered to be the electrical analogue of the stretch reflex but bypassing muscle spindles (McNeil et al. 2013; Schieppati 1987; Zehr 2002). H-reflex amplitude is known to depend not only on motoneuronal excitability (Schieppati 1987), but also on the synaptic efficiency of the Ia–alpha pathway, which depends on the level of presynaptic inhibition exerted on Ia afferents (Mazevet et al. 2003; Misiaszek 2003). Following prolonged LV, H-reflex amplitude was reported to decrease by 10-15 (Lapole et al. 2012a), 11 (Lapole et al. 2012b), 33 (Ekblom and Thorstensson 2011), 36 (Ushiyama et al. 2005) and 45% (Heckman et al. 1984) for the soleus. H-reflex reductions were also reported for gastrocnemius medialis (−34%) and lateralis (−39%) (Ushiyama et al. 2005), while this was depressed by 60% for the vastus medialis (Fry and Folland 2014) and 21% for the tibialis anterior (Farabet et al. 2016) (Table 2). Similar results were reported after LV applied during short periods on the soleus muscle (i.e. between 30 s and 2 min) (Abbruzzese et al. 2001; Van Boxtel 1986). A reduction in H-reflex amplitude also occurred after a single session of LV, suggesting similar vibration-induced effects as those observed with WBV (Armstrong et al. 2008; Krause et al. 2016). Based on unchanged F-waves that reflect backfiring of a small number of motoneurons which are reactivated by antidromic impulses following supramaximal stimulation of a peripheral nerve (McNeil et al. 2013), previous studies have suggested an unchanged motoneuronal excitability following prolonged LV (Christova et al. 2011; Lapole et al. 2012b). As a result, attenuation of Ia afferent inputs onto alpha motoneurons probably results from presynaptic inhibition of Ia afferents (Hultborn et al. 1987a, b) (see below), an increased firing threshold of Ia afferents (Hayward et al. 1986), or neurotransmitter depletion at the Ia afferent terminal level (Curtis and Eccles 1960). This latter mechanism has been suggested to mainly impair force production capacity but not H-reflex amplitude (Bongiovanni et al. 1990). Reduced Ia afferent excitability has been recently rejected as a contributor to the observed LV-induced vastus medialis H-reflex decrease, as indicated by an unchanged current required to elicit maximal H-reflex amplitude (Fry and Folland 2014). As a result, and although transmitter depletion cannot be ruled out, it is likely that the post-LV decrease in H-reflex amplitude is due to increased presynaptic inhibition of Ia afferents (Lapole et al. 2012b). Local vibration-induced increases in presynaptic inhibition have been extensively described in the early works regarding neuromuscular responses to LV. Indeed, while increased muscle spindle discharge during LV leads to a reflex activation of alpha motoneurons, it is associated with a depression of the H-reflex during the LV application (Abbruzzese et al. 1997; Ashby and Verrier 1975; Brooke and McIlroy 1990; De Gail et al. 1966; Hagbarth 1973; Hagbarth and Eklund 1966; Lance et al. 1966; Van Boxtel 1986), a phenomenon called the vibration paradox (Desmedt 1983; Desmedt and Godaux 1978). This phenomenon was mainly attributed to an increased presynaptic inhibition of the discharging Ia afferents through GABAergic interneurons under supraspinal control (Delwaide 1973; Gillies et al. 1969; Hultborn et al. 1987a). Thus, in response to the high activation of Ia

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Table 2 Acute effects of prolonged local vibration on spinal and corticospinal excitability

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Duration (min)	Fr (Hz)	A (mm)	Main outcomes
Heckman et al. (1984)	4 HE	NR	No	Achilles tendon	20	100	<3	H-reflex: SOL ↓ ≈45%
Hayward et al. (1986)	5 HE	21–35	No	Triceps surae	20	100	2	H-reflex: SOL ↓ ≈52–93%
Steyvers et al. (2003a)	16 HE (10 M, 6F)	20–27	COD	Distal tendons of the wrist flexor muscles	30 (25 s on/5 s off)	80	0.5	MEP: FCR → (− ≈35%) POST, ↑ ≈40% POST-30 min MEP: ECR ↑ from POST-10 min to POST-30 min
Rosenkranz and Rothwell (2004)	6 HE (5 M, 1 F)	26–32	No	FDI and APB muscle bellies	15 (2 s on/2 s off)	80	0.2–0.5	MEP: APB, FDI and ADM → SICI: APB, FDI and ADM → LICI: APB, FDI and ADM → Changes in sensorimotor organization
Smith and Brouwer (2005)	10 HE (6 M, 4F)	26 ± 3	No	ECR muscle belly	15 (30 s on/15 s off)	100	0.5	MEP: ECR ↑ 33%, FCR →
	10 HE (4 M, 6F)	28 ± 6			30 (30 s on/15 s off)			MEP: ECR and FCR →
Ushiyama et al. (2005)	13 HE M	22–49	COD	Achilles tendon	30	100	1.5	H-reflex: GM ↓ 34.0%, GL ↓ 38.6%, SOL ↓ 36.1%
Rosenkranz and Rothwell (2006a)	6 HE (4 M, 2F)	30–48	COD	FDI and APB muscle bellies	15 (2 s on/2 s off)	80	0.2–0.5	MEP: APB, FDI and ADM → SICI: APB, FDI and ADM → Changes in sensorimotor organization
Christova et al. (2011)	12 HE (3 M, 9 F)	26 ± 4	COD	Whole hand	20	10	±2	MEP: FDI and APB → SICI: FDI and APB → ICF: FDI and APB →
						25		MEP: FDI and APB ↑ up to POST-2 h SICI: FDI and APB ↓ up to POST-2 h ICF: FDI and APB ↑ up to POST-2 h
Ekblom and Thorstensson (2011)	8 HE F	23 ± 2	COD	Achilles tendon	30	100	NR	H-reflex: SOL ↓ 32.6%
Lapole et al. (2012a)	11 HE	27 ± 3	CG	Achilles tendon	60	50	1	H-reflex: SOL ↓ ≈10% (sitting), SOL ↓ ≈15% (standing)

Table 2 (continued)

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Duration (min)	Fr (Hz)	A (mm)	Main outcomes
Lapole et al. (2012b)	12 HE	21 ± 3	No	Achilles tendon	60	50	0.2	H-reflex: SOL ↓ ≈11% POST MEP: SOL ↑ ≈30% POST-1 h, TA ↑ ≈46% POST-1 h
Fry and Folland (2014)	18 HE M	20 ± 2	COD	Infrapatellar tendon	30	80	1.5	H-reflex: VM ↓ 60.2%
Lapole and Tindel (2015)	10 HE (8 M, 2F)	27 ± 9	No	APB muscle belly	15	80	0.8–1	MEP: APB → (+22.3%) ↑ sensorimotor integration for four subjects identified as responders
Farabet et al. (2016)	13 HE (9 M, 4 F)	23 ± 4	COD	TA muscle belly	30	100	1	H-reflex: TA ↓ 21.0% MEP_{MVC}: TA → (+19.7%) CSP_{MVC}: TA → (+7.3%)

A vibration amplitude, *APB* abductor pollicis brevis, *COD* cross-over design, *CG* control group, CSP_{MVC} cortical silent period during maximal voluntary contraction, *ECR* extensor carpi radialis, *F* female, *FCR* flexor carpi radialis, *FDI* first dorsal interosseus, *Fr* vibration frequency, *GL* gastrocnemius lateralis, *GM* gastrocnemius medialis, *HE* healthy population, *ICF* intracortical facilitation, *LICI* long-interval cortical inhibition, *M* male, *MEP* motor evoked potential, MEP_{MVC} motor evoked potential during maximal voluntary contraction, *NR* not reported, *SICI* short-interval cortical inhibition, *SOL* soleus, *TA* tibialis anterior, *VM* vastus medialis, ≈ mean changes estimated from values displayed on a graph, → not statistically significant, ↑ statistically significant increase, ↓ statistically significant decrease

afferents by the vibratory stimuli (Burke et al. 1976a, b; Roll et al. 1989), it was hypothesized that H-reflex depression observed following prolonged LV may result from similar mechanisms as those reported during the vibration paradox (Farabet et al. 2016; Lapole et al. 2012b). Such LV-induced depression of the H-reflex may persist for up to 35 min (Hayward et al. 1986; Heckman et al. 1984; Ushiyama et al. 2005), and return to baseline values 1-h after the end of exposure (Lapole et al. 2012b).

Motor unit recruitment failure in response to decreased Ia afferent efficacy

Using recordings of motor unit firing rates, the decrease in synaptic input from Ia afferents to alpha motoneurons has been suggested to predominantly affect the recruitment of high-threshold motoneurons that supply fast-twitch muscle fibres (Bongiovanni et al. 1990). This can explain why some studies (Ushiyama et al. 2005; Yoshitake et al. 2004) reported a decreased EMG amplitude in medial (−32 and −13%, respectively) and lateral (−12 and −11%, respectively) heads of the gastrocnemius after 30 min of 100-Hz Achilles tendon vibration, with no concomitant change in the soleus muscle (which is known to have more slow-twitch fibres compared to the gastrocnemii). Similarly, LV-induced decreased EMG amplitude was reported to be more pronounced in the rectus femoris, i.e. the muscle with the higher percentage of fast-twitch fibres among quadriceps muscles (Johnson et al. 1973), than vastus lateralis (Jackson and Turner 2003) and vastus medialis muscles (Kouzaki et al. 2000) after a 30-min prolonged LV period at 30 Hz. Furthermore, a decreased rate of force development in the knee extensors after prolonged LV has been suggested to result from a failure in the capacity to completely recruit high-threshold motoneurons at the onset of the contraction (Jackson and Turner 2003; Kouzaki et al. 2000).

Local vibration-induced decreased spinal loop excitability is well acknowledged and suggested to explain the MVC decrease observed after LV, but only one study has reported decreased H-reflex accompanied by an impairment in force following a 30-min prolonged LV period (frequency 100 Hz; amplitude 1.5 mm) (Ushiyama et al. 2005) while others studies have reported decreased H-reflex without impaired MVC (Ekblom and Thorstensson 2011; Farabet et al. 2016). The reasons why force production capacities are unchanged while the spinal excitability is reduced remains unclear, but differences in investigated muscles (dorsiflexor and knee extensor muscles for Farabet et al. 2016 and Fry and Folland 2014, respectively), and type of contractions (dynamic contractions for Ekblom and Thorstensson 2011) (see Table 2) may

have caused these conflicting results. Alternatively, Ekblom and Thorstensson (2011) hypothesized that decreased activity of Ia afferents could have been counteracted by supraspinal inputs (i.e. greater descending neural drive through increased motor cortex excitability). This hypothesis will be examined in the next section addressing the acute effects of LV on corticospinal excitability through the use of transcranial magnetic stimulation (TMS).

Acute effects of local vibration on corticospinal excitability

While proprioceptive inputs are essential at the spinal level (Gandevia 1998), they also play a major role in motor control at the cortical level (Wiesendanger and Miles 1982) and it is thought that afferent inputs can modulate motor cortex excitability. The hypothesis that vibratory stimuli may influence the cortical level is supported by studies showing that LV can induce cortical activation of both sensory and motor areas (see above).

Corticospinal excitability modulation during the application of local vibration

Numerous upper limb studies (i.e. various hand and wrist muscles) have demonstrated an increase in corticospinal excitability within the first seconds of LV application. This has been evidenced by (1) an increased motor evoked potential (MEP) peak-to-peak amplitude or area and (2) a decreased resting motor threshold (i.e. the lowest TMS intensity at which a MEP can be elicited in a relaxed muscle (i.e. no background muscle activity) (Claus et al. 1988; Kossev et al. 1999, 2001; Rosenkranz and Rothwell 2003, 2006a; Siggelkow et al. 1999; Steyvers et al. 2003b). This occurs despite the vibration paradox, i.e. a decreased spinal loop excitability during LV (Desmedt 1983; Desmedt and Godaux 1978). More recently, our research group extended these findings to lower limb muscles and demonstrated both decreased resting motor threshold and increased MEP amplitude in the plantar flexors during brief low-amplitude (1 mm) LV exposure (Lapole et al. 2015b). Interestingly, the magnitude of LV-induced MEP amplitude increase was much greater for the soleus (increased by ≈275% with 50-Hz LV frequency and ≈200% with 80 and 110-Hz frequencies) than gastrocnemius medialis muscle (increased by ≈150% at all 50, 80 and 110-Hz frequencies), possibly due to the higher density of muscle spindles in the soleus (Proske 1997), making this muscle group more sensitive to LV. The only study that investigated corticospinal excitability during WBV demonstrated a 56% increase in tibialis anterior MEP amplitude (Mileva et al. 2009). The LV-induced increase in MEP amplitude was reported to be frequency dependent for upper limb muscles (Siggelkow et al. 1999; Steyvers et al. 2003b). Significant increases in extensor carpi radialis MEP amplitude were reported during 80-Hz LV (increased by 100%) and 120-Hz LV exposure (increased by 50%) while no significant changes were observed with 160 Hz (Siggelkow et al. 1999). Similarly, it has been shown that 75-Hz LV resulted in a more pronounced increase in flexor carpi radialis MEP amplitude compared to 120-Hz LV (50 and 30%, respectively), while a 20-Hz frequency, however, had no effect on MEP amplitude (Steyvers et al. 2003b). The fact that the increase in MEP amplitude depends on LV frequency has mainly been attributed to the one-to-one response of Ia afferents with LV frequencies up to 120 Hz (Burke et al. 1976a; Roll et al. 1989). Since 20-Hz LV is known to induce a suboptimal Ia afferent firing rate (Roll et al. 1989), insufficient central temporal summation of Ia afferent discharge has been suggested to account for the lack of MEP amplitude change (Steyvers et al. 2003b). Similarly, a high LV frequency (i.e. 160 Hz) was ineffective in facilitating MEPs (Siggelkow et al. 1999). This latter finding was hypothesized to result from (1) an overstimulation of Ia afferents (Siggelkow et al. 1999), leading to a possible decrease in their firing rate (Roll et al. 1989) or (2) inhibitory polysynaptic mechanisms mediated by dynamic mechanoreceptors (Siggelkow et al. 1999). In plantar flexor muscles, MEP amplitude was not dependant on the LV frequency as the MEP amplitude increase was the same for input frequencies of 50, 80 and 110 Hz (Lapole et al. 2015b), perhaps because all of these tested frequencies are in the range of optimal firing rates of Ia afferents in response to LV (Burke et al. 1976a; Roll et al. 1989). Hence, the LV-induced increase in MEP amplitude is thought to be directly mediated by the induced discharge of muscle spindle Ia afferents.

Increased corticospinal excitability in the vibrated muscle has concomitantly been reported with modulations for antagonist and contralateral non-vibrated muscles. For instance, while corticospinal excitability in vibrated (upper body) muscles was increased, a reduced excitability was found in the antagonist muscles when LV at 80 (Kossev et al. 2001; Rosenkranz et al. 2003; Rosenkranz and Rothwell 2003, 2006b; Siggelkow et al. 1999), 120 (Siggelkow et al. 1999) and 160 Hz (Siggelkow et al. 1999) were used. This effect is further supported by the increased cortical silent period in the antagonist muscle (Binder et al. 2009), suggesting an enhanced intracortical inhibition via activation of GABAergic inhibitory cortical neurons projecting onto the pyramidal cells in the motor cortex (Inghilleri et al. 1993; Werhahn et al. 1999). Altogether, this highlights the well-known reciprocal inhibitory influences of Ia afferents, mediated by both spinal (Day et al. 1984; Gillies et al. 1969) and supra-spinal inputs (Bertolasi et al. 1998). The role of inhibitory mechanisms originating in vibrotactile afferents or other dynamic mechanoreceptors (cutaneous or joint receptors) has also been suggested (Siggelkow et al. 1999).

This decrease in corticospinal excitability in antagonist muscles was, however, not found in plantar flexor muscles and discrepancies between upper and lower limb muscles remain to be elucidated (Lapole et al. 2015b).

An increase in contralateral agonist MEP area and a decrease in contralateral antagonist MEP area has also been found during 80-Hz LV (Kossev et al. 2001). It was suggested that the facilitatory action of LV may be mediated via transcallosal pathways that in turn cause inhibition of cortical outputs to the contralateral antagonist (Kossev et al. 2001). Conversely, Swayne et al. (2006) reported a decrease in MEP amplitude in the contralateral homologous muscle during 80-Hz LV, suggesting effects occurring in the cortex ipsilateral to the vibrated muscle and mediated via transcallosal fibres. We recently found no effects of LV in contralateral muscles, specifically the plantar flexors muscle (Lapole et al. 2015b). As for antagonist inhibition, the discrepancy between upper and lower limb studies is still unclear.

Since MEP amplitude depends on the synaptic relays of the corticospinal projections at both the cortical and spinal levels (Devanne et al. 1997), the interpretation of LV-induced increased MEP amplitude should be made with caution. The contribution of cortical, spinal or peripheral mechanisms to the increased MEP amplitude is difficult to identify. M- and F-waves, which assess sarcolemmal and motoneuronal excitability, respectively, remain unchanged during 120-Hz LV (Rollnik et al. 2001). Further, MEP amplitude during LV is increased in response to TMS but not transcranial electrical stimulation, suggesting a cortical contribution to this increase in MEP amplitude (Kossev et al. 1999). Overall, the literature suggests that the mechanisms are cortical in origin. The exclusion of spinal contributions to increases in MEP amplitude may be further supported by the unaffected postsynaptic excitability of soleus motoneurons reported during 25-Hz LV (Abbruzzese et al. 1997).

Changes in intracortical excitability and inhibition during local vibration

Paired-pulse TMS (Kujirai et al. 1993) has been used to further understand the effects of LV on cortical excitability. By conditioning a supra-threshold TMS test pulse by a preceding subthreshold pulse, the excitability of intracortical circuits can be assessed. Time-dependent modulation of the test MEP by the conditioning stimulus can be observed when using short (≈1 to 6 ms) and long inter-stimulus intervals (≈50 to 150 ms) to elicit short-interval cortical inhibition (SICI) and long-interval cortical inhibition (LICI), respectively. If an inter-stimulus interval of 10–15 ms is used, intracortical facilitation (ICF) can be assessed (Benwell et al. 2007; Kujirai et al. 1993). SICI and LICI are known to reflect activity in cortical GABAergic circuits ($GABA_A$ and $GABA_B$ receptors for SICI and LICI, respectively), which indirectly affect the excitability of pyramidal neurons (Hanajima et al. 1998; Rosenkranz and Rothwell 2003; Ziemann et al. 1996). Conversely, ICF may reflect the activation of excitatory interneurons (Ziemann et al. 1996). In upper limb studies, LV-induced increased MEP amplitude could be explained by a lower inhibition as demonstrated by decreased SICI (Rosenkranz et al. 2003; Rosenkranz and Rothwell 2003, 2006b). Furthermore, an augmentation of LICI occurs during 80-Hz LV application (Rosenkranz and Rothwell 2003). Even if some spinal mechanisms have been proposed to explain the difference between SICI (i.e. decrease) and LICI (i.e. increase), it is likely that LV differently impacts these populations of cortical inhibitory interneurons projecting to cortical zones (Rosenkranz and Rothwell 2003). The results concerning ICF are equivocal with either a small increase or no change observed with 80-Hz LV (Rosenkranz et al. 2003; Rosenkranz and Rothwell 2003). On the soleus muscle, our research group observed unchanged SICI and decreased ICF with LV (frequency 50 Hz; amplitude 1 mm) (Lapole et al. 2015a), a finding in agreement with the study of Mileva et al. (2009) during WBV, and highlighting, once again, the potential differences in LV-induced neural modulations between upper and lower limb muscles. Future studies should consider the use of corticospinal tract electrical stimulation (McNeil et al. 2013) in conjunction with TMS to enable the determination of differential effects of LV on cortical vs. motoneuronal excitability.

The aftereffects of prolonged local vibration on corticospinal excitability

The studies that investigated acute changes in corticospinal excitability after prolonged exposure to LV are summarized in Table 2, which provides detailed information on experimental (subjects characteristics, tested muscles) and LV (frequency, amplitude, vibration site) characteristics. While there is a consensus for increased corticospinal excitability during the application of LV (see above), the findings are equivocal with regard to acute changes after a single session of prolonged LV. While the only WBV study that addressed this question reported increased MEP amplitude after a single session (Krause et al. 2016), resting corticospinal excitability was found to increase (Christova et al. 2011; Lapole et al. 2012b; Smith and Brouwer 2005; Steyvers et al. 2003a), decrease (Farabet et al. 2016; Steyvers et al. 2003a), or even remain unchanged (Lapole et al. 2012b; Lapole and Tindel 2015; Rosenkranz and Rothwell 2004, 2006a; Smith and Brouwer 2005) after prolonged LV. Differences in the investigated muscles, LV parameters and experimental design may all account for these discrepancies (see Table 2 for detailed results). As an example, an increase in MEP amplitude was found after 15 but not 30 min of 100-Hz LV

(Smith and Brouwer 2005). Interestingly, three studies that addressed LV aftereffects on antagonist muscles all demonstrated increased corticospinal excitability (Forner-Cordero et al. 2008; Lapole et al. 2012b; Steyvers et al. 2003a). This may result from reciprocal inhibitory influences of Ia afferents on corticospinal output of the antagonist muscle (Bertolasi et al. 1998). Some studies have addressed whether intracortical circuits are modulated after LV using paired-pulse protocols. Christova et al. (2011) reported decreased SICI and increased ICF for up to 2 h after 20 min of LV. Conversely, it has been reported that 15 min of LV had no effect on SICI (Rosenkranz and Rothwell 2004, 2006a). Differences in vibration frequencies (25 vs. 80 Hz, respectively), vibration amplitudes (2 vs. 0.2–0.5 mm, respectively) and type of vibration (whole hand vs. muscle vibration, respectively) may all account for these discrepancies.

Overall, it remains difficult to interpret this part of the scientific literature because both spinal and supra-spinal levels may be involved in the observed MEP changes (Devanne et al. 1997). As discussed above, cervicomedullary and/or thoracic electrical stimulations (McNeil et al. 2013) used in conjunction with TMS will enable the determination of differential effects of LV on both cortical and motoneuronal excitability in the future. Nonetheless, the existing body of evidence supports the notion that the sensorimotor cortex has a dynamic functional organization and that changes in afferent input may modulate corticospinal excitability. This would rely on topographically and functionally specific reciprocal connections between primary sensorimotor cortex and primary motor cortex (Rocco and Brumberg 2007). These sensorimotor pathways have been investigated by conditioning a TMS test pulse with electrical stimulation of a peripheral nerve (Devanne et al. 2009; Tokimura et al. 2000). Using this technique, changes in sensorimotor integration (i.e. changes in excitability of cortical circuits interposed between the ascending afferent volley and descending corticospinal pathways) after LV occurred for half of the participants while no changes were observed for the others (Lapole and Tindel 2015). The explanation for such dichotomy has been hypothesized to result from the influence of the level of attention that participants gave to the sensory stimulations. For instance, the reorganization of the somatotopic representation of the body within the somatosensory cortex following prolonged sensory stimulation may be achieved only if attention is paid to the changed inputs (Mogilner et al. 1993; Ziemus et al. 2000), even when LV is used (Rosenkranz and Rothwell 2004). Moreover, the reorganization of the somatotopic representation after LV exposure was even greater when attention was paid to the exact site of stimulation (Rosenkranz and Rothwell 2006b).

In summary, prolonged LV exposure may induce decrement in motor performance (i.e. fatigue) and depression of spinal loop excitability. When repeated, this may trigger training adaptations such as increases in strength (Kraemer and Ratamess 2004). Long-term supra-spinal adaptations to LV may also become apparent with repeated exposures, given the acute modulation of corticospinal excitability with a single exposure.

Chronic effects of local vibration training

While current research on the chronic effects of WBV is well studied (Cochrane 2011; Rittweger 2010), the concept of local vibration training (LVT), i.e. the repetition of LV sessions across multiple days with the aim to improve neuromuscular function, is relatively under-investigated. Another limitation of this body of literature is that studies that have addressed this question used a variety of training protocols (i.e. different training duration, LV frequency, number of sessions, etc.) with minimal consistency across studies. Further, these LV sessions have been delivered either on relaxed or contracted muscles, further limiting the comparison between studies (see Table 3 for detailed information on training (duration, number of sessions) and LV (frequency, amplitude, vibration site) characteristics).

Effects of local vibration training on force-generation capacities

As illustrated by Table 3, LVT performed on relaxed muscles has been reported to increase isometric MVC on plantar flexors by 6.9 (Lapole and Pérot 2010) and 9.7% (Lapole et al. 2013), dorsiflexors by 12.0% (Souron et al. 2017) and knee extensors by 13.8 (Tankisheva et al. 2015) and 32.5% (Iodice et al. 2011). Increased performance during dynamic MVC (i.e. +41.0%) was also reported after LVT for knee extensors by Iodice et al. (2011), but not Tankisheva et al. (2015). In the studies presented above, strength gains were achieved with training duration of 2 (Lapole et al. 2013; Lapole and Pérot 2010), 4 (Iodice et al. 2011), 8 (Souron et al. 2017) and 26 weeks (Tankisheva et al. 2015). Souron et al. (2017) further reported that LVT-induced strength gains were not significantly greater after 8 weeks (+12.0%) than 4 weeks (+7.5%), and that gains persisted 2 weeks after the end of LVT (+10.8%). Although most studies have investigated the effect of several weeks of LVT applied on relaxed muscles, other researchers have investigated LVT with shorter training duration [i.e. 3 consecutive days, the so-called 'repeated muscle vibration' (rMV)], with LV being applied either on relaxed or contracted muscles (Aprile et al. 2016; Brunetti et al. 2006, 2014; Caliandro et al. 2012; Fattorini et al. 2006; Filippi et al. 2009; Marconi et al. 2008, 2011; Rabini et al. 2015). However, the feasibility of such rMV designs, when performed during voluntary contractions, is questionable. Indeed, the authors described a single

Table 3 Chronic effect of local vibration training

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Training design	Fr (Hz)	A (mm)	Main outcomes
Young population								
Fattorini et al. (2006)	7 HE (5 M, 2 F)	31 ± 2	CG	Distal part of the quadriceps (UL)	3 consecutive days (30 min/day)	100	0.005–015	MVC: → (+≈10%) RFD: → Fatigue resistance: →
Marconi et al. (2008)	5 HE	35 ± 10	CG	FCR muscle belly (UL)	3 consecutive days (30 min/day)	100	0.05–0.1	H-reflex: FCR → MEP: FCR and EDC→ SICI: FCR and EDC → ICF: FCR and EDC →
Lapole and Pérot (2010)	29 HE	21 ± 2	COD	Achilles tendon (UL)	14 consecutive days (1 h/day)	50	0.2	MVC: ↑ 6.9% EMG: TS ↑ 9.3% Activation deficit: ↓ 44.4% No changes in twitch parameters
Iodice et al. (2011)	18 HE	21 ± 4	No	VI, RF, VL, VM, GMA, BF, TA, G (UL)	4 weeks (3 × 30 min/week)	300	2	Bilateral knee extension MVC: ↑ 32.5% Isokinetic MVC: ↑ 41.0%
Lapole and Pérot (2012)	16 HE	21 ± 2	CG	Achilles tendon (UL)	14 consecutive days (1 h/day)	50	0.2	H-reflex: SOL ↑ 34.2%, GM → (+1.6%), GL → (−1.8%)
Lapole et al. (2013)	11 HE	22 ± 3	COD	Achilles tendon (UL)	14 consecutive days (1 h/day)	50	1	MVC: ↑ 9.7% H-reflex: SOL ↑ 30.1% V-wave: SOL ↑ 43.3% Cross-education effect for MVC (↑ 10.4%) and V-wave (↑ 41.6%)
Aprile et al. (2016)	48 HE (12 M, 36 F)	25–50	CG	Deltoid, BB and pectoralis muscles (UL)	3 consecutive days (30 min/day)	100	±150 mbar	No changes in number and quality of contractions performed in 30 s on a visually guided reaching task
						200		Increased number and quality of contractions performed in 30 s on a visually guided reaching task

Table 3 (continued)

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Training design	Fr (Hz)	A (mm)	Main outcomes
Souron et al. 2017	22 HE (8 M, 14 F)	20 ± 1	CG	TA muscle belly (UL)	8 weeks (3 × 1 h/week)	100	1	MVC: ↑ 12.0% VA_{TMS}: ↑ 4.9% MEP_{MVC}: TA → (−19.8%) CSP_{MVC}: TA → (−7.2%) Cross-education effect for MVC (↑10.1%) and VA_{TMS} (↑6.2%)
Elderly population								
Filippi et al. (2009)	20 HE F	64 ± 3	CG	Distal part of the quadriceps (BL)	3 consecutive days (30 min/day)	100	0.2–0.5	Jump height: → Leg power: → Balance: →
Tankisheva et al. (2015)	17 HE F	66–84	CG	Quadriceps (middle portion) and around the hip (UL)	26 weeks (5 × 2–8 min/week)	30–45	NR	MVC: ↑ 13.8% No changes in isokinetic and isotonic maximal voluntary contractions Walk test: → (−1.2%) Muscle mass: → (−0.7%)
Clinical population								
Brunetti et al. (2006)	15 ACL M	25 ± 3	CG	Distal part of the quadriceps during 50% MVC contraction (UL)	3 consecutive days (30 min/day)	100	<20 µm	MVC: ↑ Balance: ↑
Pietrangelo et al. (2009)	9 sarcopenia patients (4 M, 5 F)	73 ± 6	No	Distal part of the quadriceps (BL)	12 weeks (1 × 15 min/week for weeks 1–8; 3 × 15 min/week for weeks 9–12)	300	NR	Bilateral MVC: ↑ ≈63% Myofibre transition from IIx to I Muscle mass: → CSA: → (+15.5%)
Marconi et al. (2011)	15 stroke patients (9 M, 6 F)	63 ± 7	CG	FCR and BB muscle bellies during 10–20% MVC voluntary contraction (UL)	3 consecutive days (30 min/day)	100	0.2–0.5	MEP: FCR, BB and EDC ↑ SICI: FCR and BB ↑ and EDC↓ ICF: → Spasticity (MAS scale): ↓
Zhao et al. (2011)	30 HE IMM (17 M, 13 F)	18–46	CG	SOL muscle belly (UL)	12 consecutive days (48 min daily)	100	0.3	Prevention of the immobilization-induced reduced EMG and V-wave No changes in H-reflex and F-wave
Roll et al. (2012)	16 HE IMM	22–35	CG	Tendons of the wrist extensor and flexor muscles (UL)	5 days (illusory movement, 2 × 30 min/day)	100	NR	Prevention of immobilization-induced reduced cortical activation (S1M1, PMd, SMA) during voluntary movement

Table 3 (continued)

Study	Tested subjects	Mean ± SD age (years)	Control condition	Vibration site	Training design	Fr (Hz)	A (mm)	Main outcomes
Caliandro et al. (2012)	28 stroke patients (20 M, 8 F)	57 ± 12	CG	Pectoralis minor and BB during voluntary contraction (UL)	3 consecutive days (30 min/day)	100	0.2–0.5	Functional ability (WMFT FAS): ↑ Spasticity (MAS scale): →
Brunetti et al. (2014)	20 postmenopausal osteoporotic F		CG	Quadriceps during voluntary contraction (UL)	3 consecutive days (30 min/day)	100	0.3–0.5	Leg power: ↑ Balance: ↑ Counteract the progressive bone demineralization induced by osteoporosis
Rabini et al. (2015)	25 M and F with knee osteoarthritis	60 or older	CG	Quadriceps during voluntary contraction (BL)	3 consecutive days (30 min/day)	100	0.2–0.5	Significant improvement in functional capacity (WOMAC, SPPB and POMA)

A vibration amplitude, *BL* bilateral local vibration application, *COD* cross-over design, *CG* control group, *CSA* cross-sectional area, *CSP* cortical silent period, CSP_{MVC} cortical silent period during voluntary contraction, *EDC* extensor digitorum communis, *EMG* electromyography, *F* female, *FCR* flexor carpi radialis, *Fr* vibration frequency, *GL* gastrocnemius lateralis, *GM* gastrocnemius medialis, *HE* healthy population, *ICF* intracortical facilitation, *IMM* subjects who undergone an immobilization period, *M* male, *MAS* Modified Ashworth Scale, *MEP* motor evoked potential, MEP_{MVC} motor evoked potential during voluntary contraction, *MVC* isometric maximal voluntary contraction, *NR* not reported, *PMd* dorsal premotor cortex, *POMA* Performance-Oriented Mobility Assessment, *RFD* rate of force development, *S1M1* primary sensorimotor area, *SICI* short-interval cortical inhibition, *SMA* supplementary motor area, *SOL* soleus, *SPPB* short physical performance battery, *TA* tibialis anterior, *TB* triceps brachii, *TS* triceps surae, *UL* unilateral local vibration application, VA_{TMS} cortical voluntary activation assessed via transcranial magnetic stimulation, *WOMAC* Western Ontario and McMaster Universities Osteoarthritis Index, *WMFT FAS* Functional Ability Scale of the Wolf Motor Function Test, → not statistically significant, ↑ statistically significant increase, ↓ statistically significant decrease

session as 3 bouts of 10 min (with 1-min rest between bouts) with the participants maintaining around 10–20% MVC submaximal contractions (Brunetti et al. 2012; Fattorini et al. 2006; Marconi et al. 2008; Filippi et al. 2009). From our experience, this is likely to be extremely difficult to perform for most participants, even at 10% MVC (Arnal et al. 2016). Moreover, it is not clear from these studies how force contraction was monitored during the intervention. After rMV performed on relaxed upper limb muscles, Aprile et al. (2016) recently demonstrated increased motor performance in a robot-based evaluation consisting of visually guided reaching task (increased number of repetition in 30 s), with rMV-induced improvements still present in follow-up measurements performed 10 days later. Conversely, Fattorini et al. (2006) failed to demonstrate an increased MVC 2 weeks after rMV performed either on relaxed or contracted quadriceps muscle. However, this particular study did not assess muscle performance immediately after the end of rMV and had a small sample size ($n = 7$ per group), which may have caused the small effect of a +5 or +10% increased MVC to be significant when LV was performed under voluntary contraction and under relaxed conditions, respectively. In contrast, there was an increased rate of force development (up to 36% at 30 ms after the onset of the contraction) of the vibrated leg and increased resistance to fatigue during a bilateral task, as evidenced by an increased number of repetitions of isotonic contractions (+40.3%) after rMV performed in combination with voluntary contraction, but not when rMV was performed in the relaxed state (Fattorini et al. 2006). Similarly, increased vertical jump performance in elderly participants was reported 1 (+55%), 30 (+70%) and 90 (+90%) days after rMV combined with voluntary contraction of knee extensor muscles, but not when performed on relaxed muscles (Filippi et al. 2009). Recently, this has been confirmed in female volleyball players, with increased jump heights reported 1 (+16 and +9% for squat and counter movement jump, respectively), 30 (+19 and +11% for squat and counter movement jump, respectively) and 240 (+26 and +13% for squat and counter movement jump, respectively) days after rMV combined with voluntary contractions of knee extensors (Brunetti et al. 2012), although only six participants were included in each group in this study.

It appears that regardless of the LVT design, it may induce persistent and long-lasting effects on force production capacities. With short training duration (i.e. rMV), with the exception of the study of Aprile et al. (2016), improvements were only observed when LV was applied on contracted muscles since LV alone or contraction alone did not induce the same improvements (Brunetti et al. 2012; Fattorini et al. 2006; Filippi et al. 2009). In contrast, others demonstrated that superimposing LV during dynamic strength training did not increase the chronic effects of strength training alone on elbow flexors MVC (Drummond et al. 2014) or vertical jump and running speed (Couto et al. 2012). It is interesting to note that in these last two studies, unilateral LVT induced similar strength gains on the vibrated (+9.7 and +12.0%, respectively) and non-vibrated sides (+10.4 and +10.0%, respectively). With regard to strength training studies, this agrees with the findings of Lagerquist et al. (2006), although it is important to emphasize that most studies reported higher strength increase in the trained but not the untrained leg after voluntary strength training (Munn et al. 2004). This may be explained by the fact that 'classic' strength training triggers a combination of hypertrophy and neural adaptations for the trained leg while only neural adaptations are present in the contralateral side (Carroll et al. 2006). It is likely that only neural adaptations account for the increase in muscle strength after LVT for both the non-vibrated limbs (see below).

Is local vibration applicable for all population?

As discussed above, it is worth noting that LVT has been shown to induce strength gains in the vibrated muscles for healthy young males and females, as well as elderly females (see Table 3). Clinical populations may also benefit from LVT (see below). This suggests that LVT may be feasible and tolerable in many different populations. There seems to be a high inter-individual variability, however, in the LVT-induced strength gains. Indeed, our team reported coefficient of variations for the LVT-induced pre–post-changes from 78 to 182% (Lapole et al. 2013; Souron et al. 2017; Lapole and Pérot 2010). Figure 1 presents the percentage of responders and non-responders to LVT that we extracted from our studies. It shows that 20–40% of the participants may not benefit from LVT. If the reasons for such disparity appear unclear and require further investigations, it may be linked to the characteristics of the tested populations (initial training level and/or initial strength values). This is in agreement with the concept of trainability known with strength training (Hubal et al. 2005) suggesting that everyone will be impacted differently by a given training stimulus.

Muscle adaptations due to LVT

Direct and indirect data suggest that hypertrophy does not occur after LVT. For instance, Tankisheva et al. (2015) reported no changes in muscle mass for the trained leg assessed by tomography after 26 weeks (5 × 2–8 min of 30–45-Hz LV) of LVT on resting knee extensor muscles. Lapole and Pérot (2010) reported unchanged twitch responses and neuromuscular efficiency during voluntary (i.e. as assessed through the slope of EMG–force relationship) and evoked contractions after 14 consecutive days of LV (frequency 50 Hz; amplitude 0.2 mm) applied over the Achilles tendon. The lack of hypertrophy after LVT was

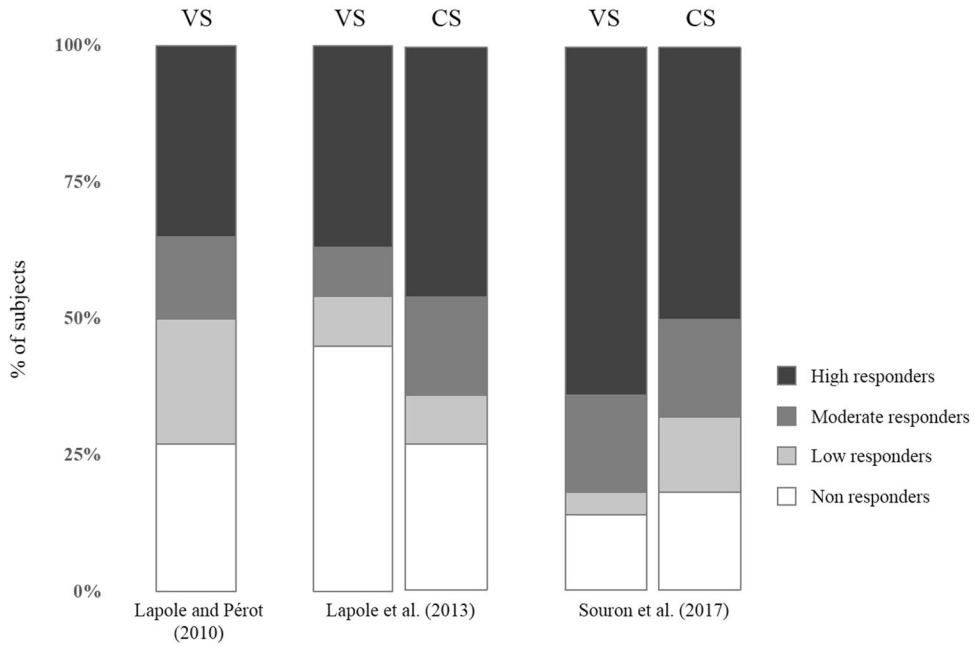

Fig. 1 Percentage of responders and non-responders to local vibration training. Data from our previous studies were re-analysed to characterize the participants as non (less than 2%), low (+2 to +5%), moderate (+5 to +10%) and high (more than +10%) responders based on individual pre–post-changes. *CS* contralateral side, *VS* vibrated side

expected since TVR is not always present, and when it is, its amplitude is moderate. Moreover, LVT periods in the reviewed studies were generally too short to induce an increase in muscle mass, as muscle adaptations reported during strength training periods are typically much longer (Sale 1988). This contrasts with some (Bogaerts et al. 2007; Machado et al. 2010) but not all (Verschueren et al. 2004) WBV training studies that reported increased muscle mass attributed to dynamic contractions performed on the vibrating platform. Nonetheless, an increase in growth hormone and a decrease in cortisol level have been reported after a 30-min bout of 300-Hz LV, suggesting that LVT may potentially have a trophic action in the long term (Iodice et al. 2011). Although this tends to agree with WBV studies (Bosco et al. 2000), this may be questioned with regard to the relatively small muscle mass activated by LV when compared to the much larger muscle mass involved during WBV. Hence, the potential for LV to induce a systemic hormonal response remains to be clarified.

Neural adaptations due to LVT

As for the WBV chronic effects (Goodwill and Kidgell 2012; Ness and Field-Fote 2009), neural adaptations were proposed to mainly account for the strength gains after LVT (Brunetti et al. 2012; Fattorini et al. 2006; Filippi et al. 2009; Iodice et al. 2011; Lapole et al. 2013; Lapole and Pérot 2010; Tankisheva et al. 2015; Aprile et al. 2016), although this has been rarely investigated (Lapole et al. 2013; Lapole and Pérot 2010). Adaptations at the central nervous system level were first shown after 14 consecutive days of LV on the triceps surae by a 9.3% increased normalized EMG (i.e. EMG/M-wave amplitude) during MVC after LVT, while no changes in co-activation were reported (Lapole and Pérot 2010). V-wave, an electrophysiological variant of the H-reflex obtained when a supramaximal electrical stimulus is applied to the nerve during MVC, has also been shown to increase after 14 consecutive days of LV (frequency 50 Hz; amplitude 1 mm) applied over the Achilles tendon in the vibrated (+43.3%) as well as the contralateral non-vibrated leg (+41.6%) (Lapole et al. 2013). However, Souron et al. (2017) reported no parallel changes in normalized EMG amplitude after 8 weeks of LVT (frequency 100 Hz; amplitude 1 mm). Comparison between these last two studies should be made with caution because of the well-known limitations in EMG amplitude to detect small changes in motor unit activity (Farina et al. 2010). Souron et al. (2017), however, reported an increase in dorsiflexor muscles voluntary activation level assessed via TMS in both the vibrated (+4.9%) and non-vibrated (+6.2%) legs after LVT. This is in agreement with previous finding of a 44.4% reduction of activation deficit evaluated by means of the classic (i.e. nerve stimulation) twitch interpolated technique after LVT (Lapole and Pérot 2010). Altogether, these findings suggest an improved spatial and/or temporal motor unit recruitment after LVT.

As for the acute effects of a single session of LV on spinal cord and corticospinal excitabilities (see above), the LVT-induced neural adaptations may come from both an increased spinal reflex excitability and/or an enhanced volitional drive from the primary motor cortex. Since Ia afferent inputs increase force production during isometric contractions (Gandevia 2001; Hagbarth et al. 1986), it has been hypothesized that LVT-induced increased neural drive may

be the result of increased excitatory inputs from the proprioceptive feedback loop during voluntary contractions (Lapole and Pérot 2010). Indeed, an increased soleus H-reflex amplitude has been found after LVT, both in the relaxed state, i.e. +34.2% (Lapole and Pérot 2012) and during MVC, i.e. +30.1% (Lapole et al. 2013); the latter result, however, was not observed in the contralateral non-vibrated leg (Lapole et al. 2013). In contrast, our team recently demonstrated no changes in tibialis anterior H-reflex recorded during submaximal voluntary contraction at 20% MVC after 8 weeks of LVT (Souron et al. 2017). Nonetheless, increased H-reflex amplitude after LVT may be explained by an increase in the amount of Ia afferent input that excites alpha motoneurons (Aagaard et al. 2002; Misiaszek 2003). Since presynaptic inhibitory mechanisms largely contribute to the decrease in H-reflex amplitude reported after an acute LV exposure (Heckman et al. 1984; Lapole et al. 2012b; Ushiyama et al. 2005), it has been speculated that some supra-spinal mechanisms may in turn have modulated the level of presynaptic inhibition exerted at the spinal level after LVT, which may contribute to the increase in muscle activation (Lapole et al. 2013; Lapole and Pérot 2012). Besides the effects of LVT on spinal loop excitability, LVT does not seem to modulate corticospinal excitability in the long term, which contrasts with acute studies (see "Acute effects of local vibration on spinal cord excitability"). Indeed, Souron et al. (2017) reported no changes in tibialis anterior MEP amplitude assessed during MVC after LVT. This is consistent with recent results reporting that superimposed WBV to strength training did not further augment the increase in corticospinal excitability induced by strength training alone (Weier and Kidgell 2012). Similar results were reported for resting MEP amplitude following rMV applied on the relaxed flexor carpi radialis muscle (Marconi et al. 2008). These authors, however, reported decreased MEP amplitude and increased SICI up to 2 but not 3 weeks after the end of rMV when vibration was combined with voluntary contraction (see Table 3). Unfortunately, this last study did not assess motor performance so that the functional significance of such long-lasting neural adaptations remains to be determined.

Can local vibration training become an efficient alternative to whole-body vibration training?

To propose vibration as a training method, its feasibility should be considered. Some studies have reported no adverse effects of LV and the training has been reported to be well tolerated (Aprile et al. 2016; Tankisheva et al. 2015), which is in agreement with our experience (Lapole et al. 2013; Lapole and Pérot 2010, 2012). The smaller vibration amplitude used in LV compared to WBV and, more importantly, the fact that LVT can be used on relaxed muscles may justify the high level of compliance reported in some LVT studies. For instance, a 96.5% compliance was observed by Tankisheva et al. (2015) for a 26-week intervention and 100% for an 8-week program in our study (Souron et al. 2017). Self-reported compliance was also observed to be maximal in our studies when LVT was performed at the participants' home (Lapole et al. 2013; Lapole and Pérot 2010, 2012). Furthermore, LVT has been tested on a variety of designs, populations and muscles (see Table 3). Altogether, these observations suggest that LVT may be considered as a feasible training program.

While a large number of studies have reported improvements in muscle performance after WBV training in healthy participants (Cochrane 2011; Rittweger 2010), similar results have been reported after LVT in young (Fattorini et al. 2006; Lapole et al. 2013; Lapole and Pérot 2010; Iodice et al. 2011; Aprile et al. 2016) and elderly (Filippi et al. 2009; Tankisheva et al. 2015) participants. LVT can improve motor performance over a broad range of muscle groups [knee extensors (Tankisheva et al. 2015), plantar flexors (Lapole et al. 2013; Lapole and Pérot 2010), dorsiflexors (Souron et al. 2017)], by simply positioning the LV device directly on the appropriate area (the muscle belly). It may be hypothesized that muscles that are rich in slow-twitch fibres would be more sensitive to LVT (Lapole and Pérot 2012) due to their greater density in muscle spindles (McCloskey 1978; Proske et al. 2000).

The strength gains reported in the present review after LVT for healthy participants (from 6.9 to 32.5% with a mean +15.2% in the reviewed studies displayed in Table 3) are quite similar to the ones reported after WBV training for the studies reviewed (Rittweger 2010) (from 6 to 24% with a mean +11.3%) despite differences in training characteristics, i.e. training duration between 2–26 and 6–34 weeks, vibration frequencies between 30–300 and 20–45 Hz and vibration amplitudes between 0.2–2 and 1–6 mm for LV and WBV, respectively. Both LV and WBV training improve strength mainly via neural adaptations, although increased muscle mass may play a role in WBV training (Cochrane 2011; Rittweger 2010). Indeed, although an exhaustive review on WBV training was beyond the scope of the present paper, we have reviewed evidence to show that both acute and chronic adaptations are similar between LV and WBV, despite discrepancies between the two methods, amplitude and frequency, duration of the exposure and state of the muscle during vibration. Hence, it can be suggested that LVT may be an efficient alternative to WBV training even if it may be difficult to precisely determine the optimal characteristics for future LVT programs. Indeed, LVT has been reported to be efficient in improving muscular performance when applied on the tendon (Lapole et al. 2013; Lapole and Pérot 2010) or the muscle belly (Iodice et al. 2011; Pietrangelo et al. 2009; Souron et al. 2017; Tankisheva et al. 2015), and with a wide range of vibration parameters,

i.e. frequencies (between 30 and 300 Hz) and amplitudes (between 0.2 and 2 mm) (Table 3). Before considering the implementation of LVT as an ergogenic aid, further studies are needed to confirm the benefits in indices of performance (jump height, sprint performance), as existing literature has mainly focussed on isometric MVC.

Local vibration training and clinical applications

Perspectives for using local vibration training in clinical settings

In addition to using LV in neurorehabilitation to decrease spasticity (for a review see Murillo et al. 2014), LVT may be used in clinical settings to improve motor performance, as illustrated in Table 3. The main advantages of LVT are its portability and its simplicity, i.e. critical factors in clinical settings when patients have weight-bearing restrictions. LVT can improve neuromuscular performance without any active contribution from the patient, it requires very light equipment and has few technical constraints so that it can be used while simply seated or lying down on a hospital bed. This makes this technique highly promising in the perspective of preventing or counteracting muscle weaknesses due to hypo-activity. This applies to patients undergoing long-term immobilization (lower limbs trauma) or being immobilized in a cast. Indeed, it is well known that in the early stages of immobilization, the neural drive is reduced and can contribute to motor performance impairment (Duchateau 1995; Gondin et al. 2004; Lundbye-Jensen and Nielsen 2008). LVT may also be used during rehabilitation programs such as in the early period after surgery. For instance, anterior cruciate ligament reconstruction generally induces an arthrogenic neural deficit that highly contributes to the quadriceps muscle weakness observed up to several months after the surgery (Hart et al. 2010; Rice and McNair 2010).

Neuromuscular adaptations following local vibration training in clinical settings

The feasibility and benefits of LVT in clinical settings is supported by the findings of Pietrangelo et al. (2009), demonstrating that a 12-week 300-Hz LVT has the potential to increase knee extension MVC in elderly patients with sarcopenia (+63%). The limitation of this study was, however, that it was performed without any control group, meaning that the results should be interpreted with caution. Follow-up measurements further demonstrated that force measured 16 weeks after the end of LVT were similar to the values recorded immediately post-LVT. Interestingly, the improvements after LVT were much greater compared to healthy elderly (+13.8%) (Tankisheva et al. 2015) and young participants (+12.0%) (Souron et al. 2017). Pietrangelo et al. (2009) reported no changes in muscle mass (assessed by thigh circumferences) with LVT, but there was an increase in the percentage of type IIx muscle fibres and a decrease in type I myosin heavy chains. These changes, however, were not accompanied by an increase in muscle fibre-specific tension or fibre thickness and, therefore, cannot explain the increased isometric force, suggesting neural adaptations. In contrast, 14 consecutive days of LV applied on unloaded hindlimb of rats, prevented the I to IIx myofibre transition induced by immobilization (Falempin and In-Albon 1999). In this study, 14 consecutive days of LV was also effective in preventing soleus muscle atrophy where muscle mass was decreased by 41% vs. only 17% with LVT. Despite non-conclusive results from Pietrangelo et al. (2009) on elderly patients with sarcopenia, the possibility exists that LVT may prevent muscle atrophy during disease or immobilization, or potentiate the recovery of muscle mass after surgery, especially considering the increased sensitivity of diseased or atrophied muscles to training stimulus (Lapole and Pérot 2010).

The use of rMV during voluntary contraction on three consecutive days also demonstrated beneficial effects on motor performance in clinical settings. Indeed, rMV was reported to increase physical function in patients with knee osteoarthritis (Rabini et al. 2015) as well as short- and long-term increase in leg power (+35 and +40%, respectively) during vertical jump in postmenopausal osteoporotic females (Brunetti et al. 2014). While an increased bone density was reported 1 year after the intervention in this latter study, it is not known if bone density increased in the short term, which questions the possibility of fracture through an imbalance between changes in muscle strength vs. bone density. In addition, rMV was reported to induce positive effects on force-generation capacities in patients after anterior cruciate ligament reconstruction with a total recovery in leg extensor muscle torque from 120 days after surgery while it was still significantly reduced for the control group (Brunetti et al. 2006). Recently, positive effects of rMV to decrease spasticity in stroke patients have also been reported (Caliandro et al. 2012; Marconi et al. 2011). rMV performed in addition with voluntary contraction, however, may be questioned, especially since in the latter study it may have been difficult to implement the rMV during 50% MVC contractions (3 × 10 min). Other clinical studies reported beneficial effects of 12 sessions of LVT for the prevention of reduced maximal neural drive induced by 2 weeks of calf muscle plaster-cast immobilization (Zhao et al. 2011). Using functional MRI, it was further demonstrated that a LV program designed to provide illusory movements during a 5-day immobilization period can prevent the reduced cortical activation of several areas (sensorimotor cortex, supplementary motor area) when performing voluntary movement

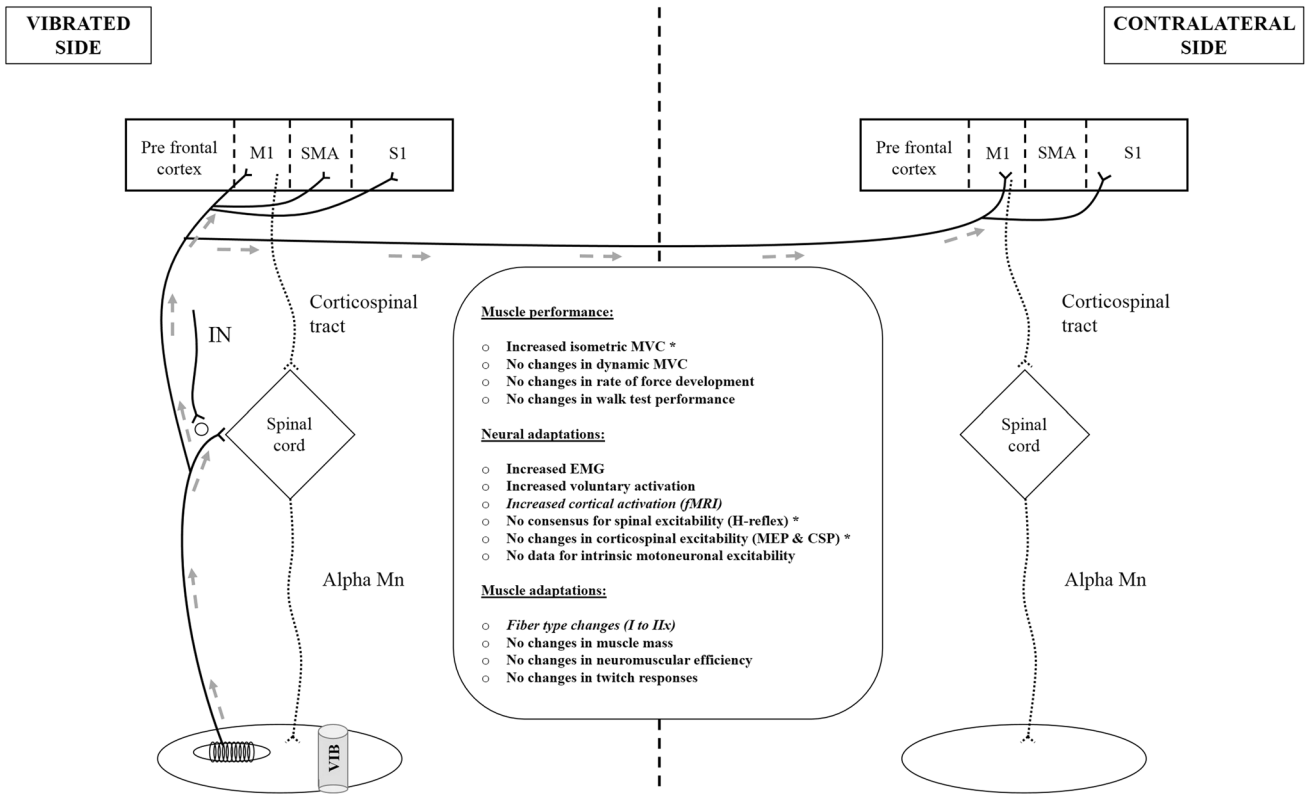

Fig. 2 Summary of the current findings of local vibration training-induced adaptations. The activation and projection of Ia afferents from muscle spindle [spiral elements represented within the muscle (oval element)] toward spinal and cortical levels are represented by *dotted grey arrows*. *Italic writing* in the *box* highlights results obtained for clinical participants. *Asterisk* in the *box* highlights results obtained on both vibrated and contralateral sides. *CSP* cortical silent period, *fMRI* functional magnetic resonance imaging, *IN* interneurons, *M1* primary motor cortex, *MEP* motor evoked potential, *Mn* motoneuron, *MVC* maximal voluntary contraction, *S1* sensorimotor cortex, *SMA* supplementary motor area, *VIB* local vibratory device

(Roll et al. 2012). Whether or not the adaptations reported in these two studies may lead to functional improvement remains to be determined.

While promising results have been reported in WBV (Huang et al. 2017; Pistone et al. 2016; Wang et al. 2016) and neuromuscular electrical stimulation (de Oliveira Melo et al. 2016; Fitzgerald et al. 2003; Glinsky et al. 2007) studies, it has also been suggested that LVT may be beneficial for various clinical populations (participants with knee osteoarthritis, anterior cruciate ligament reconstruction or central nervous system disorders) (see Table 3). However, further studies are needed to confirm the benefits in either muscular or neural components in a variety of clinical populations, especially by further considering muscle performance and functional test measurements.

Conclusion

For more than four decades, vibratory stimuli have been considered as a training or rehabilitation modality. While WBV is particularly well documented, LV techniques have been more recently developed to overcome some limitations related to the use of WBV, i.e. prohibitive cost, non-portability of the system, inability to directly target a specific muscle group or inability for some subjects to maintain an active position during the vibration application on the platform.

There is clear evidence that vibration is a strong stimulus to modulate the central nervous system through the activation of Ia afferents that project on both the spinal cord and supra-spinal structures. When LV is acutely applied (one bout of 20–30 min), a decrease in force-generation capacities has been reported, principally triggered by adaptations within the central nervous system (i.e. spinal loop excitability depression). Thus, LV stimuli have the potential to act as a significant neuromuscular workload by inducing some fatigue which, when repeated, could trigger long-term neural adaptations leading to improved functional performance. Indeed, the present review confirms that LVT can increase motor performance through a wide range of training (duration, number of session) and vibration (frequency, amplitude, site of application) parameters. While there seems to

be a consensus about the neural nature of the induced adaptations, the precise mechanisms of these adaptations at both the spinal and supra-spinal levels remain to be clarified. In an attempt to summarize the current research findings of LVT-induced adaptations, we propose a model in Fig. 2.

LVT has been mainly used with healthy participants, although some preliminary results revealed in clinical populations suggest that its use for prevention or rehabilitation settings may be developed in the future. Further studies are needed to test the effects of LVT in various diseases and ageing populations. It is currently difficult to suggest definitive practical recommendations because of inconsistencies in the experimental settings. Thus, more work is needed to help establish research-based guidelines for LVT programs.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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