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ORIGINAL ARTICLE

Reactive knee stiffening strategies between various conditioning histories

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Abstract
Optimizing joint stiffness through appropriate muscular activation is crucial for maintaining stability and preventing injury. Conditioning techniques may affect joint stability by increasing joint stiffness and altering neuromuscular control; however no studies have assessed this in a controlled setting. Fifteen endurance athletes, 12 power athletes, and 15 control subjects sat on a stiffness device that generated a rapid knee flexion perturbation and were instructed to react to the perturbation. Main outcome measures included short-range (0–4°) and long range (0–40°) stiffness and muscle activation from quadriceps and hamstring muscles. Stiffness results revealed greater short-range stiffness in endurance athletes (0.057 ± 0.012 Nm/deg/kg) than controls (0.047 ± 0.008 Nm/deg/kg, $p = 0.021$); while passive long-range stiffness was greater in power (0.0020 ± 0.001 nm/deg/kg) than endurance athletes (0.0016 ± 0.001 nm/deg/kg, $p = 0.016$). Endurance athletes had greater reactive stiffness (0.051 ± 0.017 nm/deg/kg) than control (0.033 ± 0.011 nm/deg/kg, $p = 0.001$) and power (0.037 ± 0.015 nm/deg/kg, $p = 0.044$) groups. Endurance athletes also displayed greater quadriceps activity during passive and reactive conditions ($p < 0.050$) compared to power athletes and controls. These findings suggest that power-based training history may be associated with greater passive joint stiffness across the full range of motion, while endurance-based training could positively influence reactive muscular characteristics, as well as resting muscle tone. These unique variations in stiffness regulation could be beneficial to programmes for prevention and rehabilitation of joint injury.

Keywords: Endurance training, Power training, Joint Stiffness, Neuromuscular control

Highlights
- The manner in which athletes with different conditioning histories (i.e. endurance, power) activate muscles has potentially important implications for preventing and treating injuries and improving performance.
- Endurance-trained athletes demonstrated greater short-range stiffness and reactive stiffness, linked to quadriceps activation that may suggest increased muscle tone in this population.
- Power-trained athletes had greater passive stiffness that could indicate changes to the elastic components of the musculotendinous unit.

Introduction

Coordinated movement is dependent on an individual's ability to detect joint loads, muscle lengths, and movements, while generating the appropriate level of muscle activation such that injury may be avoided (through stress-shielding) and maximizing performance. This sensorimotor loop integrates afferent feedback from peripheral mechanoreceptors (*proprioception*) into an efferent muscular response capable of modifying joint stiffness (Needle et al., 2014; Santello, 2005). Multiple factors can impact this stiffness regulation, including training and rehabilitation protocols, and physiologic differences across individuals (Needle et al., 2014). While higher stiffness was originally thought to contribute to greater joint stability, recent evidence suggests compliance may assist in load absorption and subsequent prevention of injury (Boden, Torg,

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Knowles, & Hewett, 2009; Wilson, Wood, & Elliott, 1991). This study attempts to understand how athletes with varying conditioning histories, emphasizing fast- or slow- twitch fibre dominance, differ in their ability to sense joint loads & changes in muscle length and react to sudden joint perturbations with stiffness regulation strategies. The knowledge from this study may set the framework by which future research can assess a myriad of exercise techniques to optimize stiffness regulation for individual athletes/sports, targeting their specific needs for injury prevention, rehabilitation or performance.

Joint stiffness, the resistance of a joint to changes in displacement, is a key factor in optimizing performance and preventing injury (Latash & Zatsiorsky, 1993; Needle et al., 2014). Static stabilizers formed by capsuloligamentous structures provide a degree of innate joint stiffness, however, these structures alone are inadequate to absorb higher levels of force associated with vigorous physical activity (Colby, Hintermeister, Torry, & Steadman, 1999; Johansson, 1991). Therefore, the musculotendinous unit will provide stability through reflexive muscular responses mediated by the fusimotor system and volitional contraction. The musculotendinous unit will continuously integrate sensory information and develop motor responses in preparation for, and in response to potentially injurious loads (Dunn, Gillig, Ponsor, Weil, & Utz, 1986; Needle et al., 2014). Neuromuscular control thereby represents cyclic activity where current levels of muscular output are sensed and modified based on previous and future events. Disruptions in this neuromuscular control that affect joint stiffness have been observed among subsets of injured and functionally unstable joints (Johansson, 1991; Wilson et al., 1991); but limited research exists regarding training and conditioning techniques that may enhance stiffness regulation and contribute to injury prevention, such as hamstring strains or anterior cruciate ligament ruptures. While alterations to the motor response to sensory stimuli have been observed in individuals with a history of injury (Needle, Kaminski, et al., 2017; Needle, Lepley, & Grooms, 2017; Swanik, Lephart, Swanik, Stone, & Fu, 2004) and in response to external stimuli (DeAngelis et al., 2015) limited research exists regarding the role of training and conditioning techniques.

An individual's training background may predict whether adaptation occurs to favour fast- or slow-twitch muscle fibres, and may have important implications in joint injury prevention and treatment (Hobara et al., 2008; Hobara et al., 2010). Slow-twitch (Type I) muscle fibres' growth is facilitated mainly through implementation of high repetitions and low loads commonly seen in endurance athletes, while fast-twitch (Type II) muscle fibre adaptations are promoted through training with lower repetitions and higher loads, commonly seen in power athletes (Ricoy, Encinas, Cabello, Madero, & Arenas, 1998). Structural and performance differences exist in fibre type, with Type II fibres showing a higher intrinsic speed of contraction, larger peak power, and quicker fatigue than Type I fibres (Burke, Levine, Tsairis, & Zajac, 1973; Tihanyi, Apor, & Fekete, 1982). Previous research examining stiffness differences in populations with traditionally greater Type I and Type II fibre distribution, such as endurance and power athletes, respectively, has suggested that power athletes appear to have increased stiffness compared to endurance athletes; however, the source of these changes is unclear (Arampatzis, Karamanidis, Morey-Klapsing, De Monte, & Stafilidis, 2007; Harrison, Keane, & Coglan, 2004; Hobara et al., 2008; Laffaye, Bardy, & Durey, 2005). Continued ambiguity related to the source of stiffness changes is largely due to methodological variations, which are often functional and indirect measurements that do not isolate potential contributions of static stabilizers, reflexive contraction, and volitional activation towards joint stability in these individuals. The application of precise joint perturbations could potentially solve this limitation, as studying the stiffness and muscular responses with a high degree of temporal resolution and manipulating participant instructions could allow investigators to determine the relative contributions of static stabilizers, reflexive responses, and volitional activation throughout a specific range-of-motion. Specifically, passive responses to perturbations can highlight changes to the elastic component of muscle & tonal regulation, while reactive conditions provide information regarding the individual's ability to regulate reverse cross-bridge formation through appropriate muscle activation (Needle et al., 2014; Rack & Westbury, 1974; Sinkjaer, Toft, Andreassen, & Hornemann, 1988). Modifying levels of pre-activation further highlight the role of descending drive and regulation of linked cross-bridges in maintaining stiffness (Needle, Kaminski, et al., 2017). Data extracted by modifying instruction amongst various training populations may highlight athletes' needs and identify exercise techniques to optimize joint stiffness.

Altered joint stiffness and sensorimotor function has been associated with an increased risk of injury, but little is known regarding how conditioning may affect these factors. While limited research has examined differences in joint stiffness between power and endurance athletes (Hobara et al., 2008; Hobara et al., 2010), these studies lacked experimental control to understand the mechanisms behind dissimilarities between these groups. Using methodology that

provides precise joint perturbations while manipulating preparatory and reactive conditions may offer new insights, potentially highlighting the role of conditioning history on muscle stiffness properties, reflexive activation, and volitional activation in athletes. Understanding joint responses to load and/or muscle responses in specific subsets of athletes can provide knowledge that can be used by practitioners throughout training, rehabilitation, or injury prevention; and provide the foundation for further interventions capable of manipulating these responses. Therefore, the purpose of this study was to determine how conditioning history affects knee joint stiffness and neuromuscular control across different perturbation conditions.

Methods

Experimental approach to the problem

The present study utilized a case control design. Independent variables included conditioning history (power athletes, endurance athletes, and controls), reaction condition (relaxed or pre-contracted; reactive or non-reactive; *see procedures*), and muscle (vastus medialis, VM; vastus lateralis, VL; biceps femoris, LH; semitendinosis, MH). Dependent variables included short-range and long-range joint stiffness, and muscle activation patterns including peak and time-to-peak (TTP) activation, as well as muscle activation quantified by area under-the-curve (AUC) before and after the perturbation.

Subjects

Forty-two healthy male subjects were recruited for this study from a university population. Participants were stratified into groups based on conditioning history: 12 power athletes were recruited from sprinters on a collegiate track team; 15 endurance athletes were recruited from a collegiate cross-country team; and 15 control subjects were recruited as physically active volunteers from a university population, defined as exercising at least 20 min per day 3 days per week. All power athletes competed in races of 400 m or less, all endurance athletes participated in races of 3000 m or more, and all control subjects did not compete in competitive sports, nor did they have a history of sport specialization prior to testing. Participants were excluded from the study if they reported fractures or surgery to the knee or leg; current injury to the hip, knee or ankle; or any cardiovascular or metabolic problems that would limit physical activity. All subjects provided University-approved informed consent [156420-5], and the Physical Activity Readiness Questionnaire determined participant eligibility for the study (Thomas, Reading, & Shephard, 1992). Specific means & effect sizes related to differences in stiffness between athletes with differing training backgrounds were unavailable from previous research (Hobara et al., 2008; Hobara et al., 2010); however, our pilot data and previous studies utilizing consistent methodology supported a minimum of 12 per group, (DeAngelis et al., 2015; Kim et al., 2016).

Instrumentation

Testing was performed using a custom-built stiffness and proprioception assessment device (SPAD) (DeAngelis et al., 2015; Huxel et al., 2008). The SPAD is a modified isokinetic dynamometer with a motor capable of providing rapid and controlled joint perturbations at precise velocities and accelerations while transmitting analog signals of joint position and torque (DeAngelis et al., 2015; Hamstra-Wright, Swanik, Ennis, & Swanik, 2005; Huxel et al., 2008; Kim et al., 2016).

Muscle recruitment strategies were determined using surface electromyography (EMG), collected from the vastus medialis (VM), vastus lateralis (VL), medial hamstrings (MH, semitendinosis), and lateral hamstrings (LH, biceps femoris). Self-adhesive Ag/AgCl bipolar surface electrodes (Phillips Medical Systems, Andover, MA) were used with a wired telemetered EMG amplifier (Bortec AMT-8, Bortec Biomedical, Calgary, Alberta, Canada) to record data and transmit to a computer synchronized with position and torque. Electrode placement was identified by bony landmarks and through palpation of the mid-belly muscle contractile component during isometric contraction (Delagi, Iazetti, Perotto, & Morrison, 2011). The reference electrode was placed on the patella. The electrode placement site was shaven, abraded, and cleansed with an alcohol swab (70% ethanol solution) to decrease impedance from skin. Data were collected and synchronized in custom LabVIEW software (National Instruments, Austin, TX) at 2400 Hz.

Procedures

Participants reported to a biomechanics laboratory for a single testing session, where they first rode a stationary bike for 5-minutes followed by 5-minutes of static quadriceps and hamstring stretching. Participants were then seated in the SPAD with the hip flexed 90°, test knee flexed 30°, and the ankle immobilized at 90°. Prior to stiffness trials, maximal voluntary isometric contractions (MVIC's) were

tested by instructing individuals to increase and hold maximum effort knee extension and flexion for the quadriceps and hamstrings, respectively. Three trials were performed in each direction with 60 s between efforts.

Knee stiffness was assessed across multiple conditions using a 40-degree knee flexion perturbation (30–70°) at 100° per second (Supplementary Material 1). The testing conditions were defined based on the amount of torque production prior to the perturbation, and the instructions on how to react to the perturbation. The conditions included relaxed non-reactive (passive, PS), relaxed reactive (PRS), 85% quadriceps MVIC non-reactive (AS), and 85% quadriceps MVIC reactive (ARS) (Needle, Kaminski, et al., 2017). Briefly, during PS participants were asked to remain relaxed throughout the perturbation; during PRS participants were asked to relax prior to the perturbation and maximally resist the perturbation once it started; during AS participants were asked to extend the knee at 85% of MVIC and maintain that contraction through the perturbation; and during ARS participants were asked to extend the knee at 85% of MVIC and react with a maximal contraction to resist motion once the perturbation was sensed. Participants were provided real-time visual feedback of their knee extension torque to ensure correct levels of pre-activation prior to all trials, and this level was monitored by the primary investigator, with the perturbation only initiated when participants were within 2.5% of the target torque. Although a lower level of muscular pre-contraction has been utilized with this method previously (DeAngelis et al., 2015; Kim et al., 2016), we elected to increase the level of pre-activation in this study due to the highly athletic nature of the participants to establish stiffness regulation values at a greater range, and better approximate muscle activation occurring during functional activities (e.g. sprinting or fast running, jumping) (Carrier, Schilling, & Anders, 2015; Howard, Conway, & Harrison, 2018). Participants were instructed that the perturbation would begin within 10 s of a warning, and the quick perturbation was applied at a random interval in that 10-second window to minimize anticipation. Three trials were collected for each condition, with rest periods of 60 s between repetitions to offset the effects of fatigue.

Data reduction

Data were analyzed in custom LabVIEW software. All trials were visually inspected for artifacts and marked for start and the end of the perturbation. Knee extension torque and position were low-pass filtered (10 Hz) to remove electrical noise. Knee joint quasi-stiffness was calculated as (ΔTorque/ΔRotation) across 2 ranges: 0–4° (short-range) and 0–40° (long-range) (DeAngelis et al., 2015; Latash & Zatsiorsky, 1993). These ranges were selected to isolate the elastic components and reverse cross-pivoting of actin–myosin heads in the short-range, and the effects of muscular activation over the long-range (Rack & Westbury, 1974; Sinkjaer et al., 1988). Stiffness values were normalized to body mass (kg) to allow for between-group comparisons and control for potential hypertrophy-related differences.

EMG data were partitioned to isolate a window 150-ms prior to the perturbation and 500-ms after the initiation of the perturbation. Data were band-pass filtered (20–400 Hz), rectified, and smoothed with a 5 Hz low-pass filter to create a linear envelope. EMG was normalized to the average of 3 maximum voluntary isometric contractions (MVICs) for each respective muscle. Peak muscle activation and AUC during 3 windows: 150 ms prior to perturbation (PRE), 0–250 ms following start of perturbation (POST-1), and 250–500 ms following start of the perturbation (POST-2) were extracted for analysis. These windows were selected to isolate the anticipatory effect related to muscle pre-activation, the immediate reflexive responses, and the volitional muscle responses for PRE, POST-1, and POST-2 respectively. Although the perturbation only lasted approximately 400-ms, the instructions provided to the individuals were to maintain contraction or relaxation throughout the perturbation and would often last for 1–2 s.

Statistical analyses

Differences in knee joint stiffness were assessed using a 3-way factorial analysis of variance (ANOVA) with 1 between-group factor (Group, 3 levels) and 2 within-subjects factors (Condition, 4 levels; Range, 2 levels). Changes in peak and TTP activity were assessed using three-way ANOVAs with 1 between-group factor (Group, 3 levels); and 2 within-subjects factors (Condition, 4 levels; Muscle, 4 levels). EMG

Table I. Subject demographics for endurance athletes (END), power athletes (PWR), and controls (CON).

	END	PWR	CON
N	15	12	15
Age (yrs)	19.8 ± 1.1	20.3 ± 1.3	20.1 ± 1.6
Height (cm)	176.6 ± 8.6	175.5 ± 4.9	177.0 ± 4.3
Mass (kg)[a]	65.9 ± 8.8	74.5 ± 10.3	79.0 ± 12.8

[a]Body mass between END and CON significantly different ($p < 0.050$).

Table II. Short-range (0–4°) and Long-Range (0–40°) stiffness values for endurance athletes (END), power athletes (PWR), and controls (CON) across all conditions.

	Group	Short-Range (0–4°) [Nm/deg/kg]	Long-Range (0–40°) [Nm/deg/kg]
PS	END	0.057 ± 0.012[a]	0.0016 ± 0.0009
	PWR	0.052 ± 0.013	0.0020 ± 0.0010[c]
	CON	0.047 ± 0.008	0.0015 ± 0.0009
AS	END	0.085 ± 0.011	0.025 ± 0.022
	PWR	0.087 ± 0.016	0.025 ± 0.021
	CON	0.081 ± 0.013	0.022 ± 0.013
PRS	END	0.055 ± 0.010[a]	0.052 ± 0.013
	PWR	0.055 ± 0.010[d]	0.061 ± 0.022
	CON	0.046 ± 0.011	0.054 ± 0.014
ARS	END	0.084 ± 0.014	0.051 ± 0.017[b]
	PWR	0.088 ± 0.015	0.037 ± 0.015
	CON	0.080 ± 0.020	0.033 ± 0.011

[a]END significantly different than CON.
[b]END significantly different than PWR & CON.
[c]PWR significantly different than END.
[d]PWR significantly different from CON.

AUC values for each condition were assessed with three-way ANOVA with 1 between-group factor (Group, 3 levels); and 2 within-subject factors (Muscle, 4 levels; Time, 3 levels). In the case of significant interaction effects, Fisher's LSD were used for post-hoc analysis. A level of significance was set *a priori* at 0.05.

Results

Participant demographics are presented in Table I. For stiffness testing, a significant three-way interaction effect was observed ($F_{8,156} = 2.52$, $p = 0.013$, Table II). Short-range stiffness was higher than long-range stiffness across all conditions ($p \leq 0.010$). Additionally, all groups had greater short-range stiffness in the pre-activated conditions (AS & ARS) compared to PS & PRS ($p \leq 0.050$); while long-range stiffness was highest in the PRS condition and lowest in the PS condition ($p \leq 0.050$). Endurance athletes displayed higher short-range stiffness in PS compared to the control group ($p = 0.021$); and higher long-range stiffness in the ARS condition compared to control ($p = 0.001$) and power ($p = 0.044$) groups. Power athletes demonstrated higher long-range stiffness in PS compared to endurance athletes ($p = 0.016$). Control subjects had lowest stiffness in the PRS condition compared to endurance ($p = 0.020$) and power ($p = 0.041$) athletes.

For peak EMG a significant three-way interaction effect was observed ($F_{24,\ 468} = 2.219$, $p = 0.001$,

Table III. Peak EMG (% MVIC) for Endurance (END), Power (PWR), and Control (CON) groups across conditions for quadriceps and hamstrings.

	Group	VM	VL	MH	LH
PS	END	13.2 ± 11.9	19.0 ± 15.5[a,b]	11.9 ± 10.3	15.8 ± 12.4
	PWR	8.51 ± 7.29	8.25 ± 7.87	11.6 ± 8.79	10.0 ± 10.9
	CON	8.31 ± 6.22	9.56 ± 10.2	12.8 ± 15.3	17.1 ± 17.9
AS	END	82.1 ± 32.1	81.5 ± 27.6	13.3 ± 8.35	24.3 ± 13.1
	PWR	82.9 ± 27.2	96.4 ± 82.9	14.4 ± 7.28	23.4 ± 16.0
	CON	77.4 ± 16.7	75.8 ± 16.7	17.3 ± 14.0	48.0 ± 45.1[a,c]
PRS	END	79.8 ± 43.2	64.6 ± 23.8	15.7 ± 10.9	21.3 ± 10.2
	PWR	59.7 ± 25.1	71.8 ± 48.3	14.6 ± 8.45	18.2 ± 10.7
	CON	57.6 ± 17.3	56.3 ± 16.5	17.5 ± 14.4	33.6 ± 29.6[c]
ARS	END	121.3 ± 55.2[b]	102.6 ± 44.8	14.2 ± 7.43	29.8 ± 15.7
	PWR	111.1 ± 36.8	118.0 ± 65.6	19.0 ± 7.80	26.2 ± 13.9
	CON	82.1 ± 15.5	87.2 ± 21.8	19.1 ± 14.3	48.3 ± 41.5[c]

[a]END significantly different than PWR.
[b]END significantly different than CON.
[c]PWR significantly different than CON.

Table IV. Area under-the-curve values (% MVIC*ms^2) for endurance (END), power (PWR), and control (CON) groups across conditions for the quadriceps (VM, VL) and hamstring (MH, LH) muscles.

		VM			VL			MH			LH		
		PRE	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*	*PRE*	*POST-1*	*POST-2*
PS	*END*	1.22 ± 1.19	2.90 ± 2.74	2.57 ± 2.33	1.68 ± 1.35	4.15 ± 3.39	3.69 ± 3.01	1.04 ± 0.884	2.56 ± 2.32	2.33 ± 1.99	1.36 ± 0.99	3.42 ± 2.63	3.04 ± 2.28
	PWR	0.750 ± 0.614	1.87 ± 1.60	1.70 ± 1.49	0.749 ± 0.679	1.81 ± 1.73	1.59 ± 1.57	0.996 ± 0.728	2.56 ± 1.95	2.30 ± 1.77	0.877 ± 0.91	2.19 ± 2.43	1.95 ± 2.17
	CON	0.719 ± 0.531	1.85 ± 1.39	1.62 ± 1.23	0.806 ± 0.837	2.11 ± 2.26	1.86 ± 2.02	1.07 ± 1.19	2.81 ± 3.41	2.50 ± 3.03	1.48 ± 1.45	3.84 ± 3.95	3.40 ± 3.55
AS	*END*	8.21 ± 1.62	15.4 ± 4.32	14.7 ± 6.81	8.21 ± 2.11	15.8 ± 4.61	15.6 ± 6.79	1.12 ± 0.808	2.91 ± 1.70	2.41 ± 1.66	2.41 ± 1.55	5.06 ± 2.85	4.43 ± 2.70
	PWR	8.34 ± 2.66	15.7 ± 4.20	12.5 ± 8.33	8.60 ± 3.89	18.5 ± 14.9	15.8 ± 19.9	1.39 ± 0.881	3.20 ± 1.56	2.68 ± 1.44	2.35 ± 1.92	4.94 ± 3.54	3.84 ± 2.37
	CON	7.84 ± 1.62	13.9 ± 2.72	12.9 ± 4.14	8.45 ± 2.47	14.9 ± 3.98	12.4 ± 3.36	1.50 ± 1.28	3.49 ± 2.97	3.34 ± 3.03	5.70 ± 6.29	9.78 ± 9.23	7.60 ± 6.15
PRS	*END*	1.25 ± 1.16	7.49 ± 4.91[b]	13.5 ± 7.05	1.73 ± 1.17[a,b]	7.02 ± 2.84	12.4 ± 5.74	1.04 ± 0.896	2.98 ± 2.40	2.55 ± 1.78	1.28 ± 0.94	3.87 ± 2.23	4.10 ± 2.29
	PWR	0.767 ± 0.54	5.37 ± 2.36	11.7 ± 5.40	0.804 ± 0.629	5.51 ± 3.22	14.5 ± 10.6	1.05 ± 0.733	2.77 ± 1.93	2.73 ± 1.63	0.89 ± 0.91	2.66 ± 1.90	3.51 ± 2.10[c]
	CON	0.719 ± 0.49	4.77 ± 2.18	10.5 ± 3.54	0.943 ± 0.784	5.27 ± 2.78	10.2 ± 3.47	1.08 ± 1.25	3.35 ± 3.38	3.37 ± 2.66	1.45 ± 1.44	4.82 ± 4.05	6.43 ± 5.47
ARS	*END*	9.36 ± 3.16	22.0 ± 9.37[b]	23.9 ± 11.4[b,c]	8.38 ± 3.13	18.8 ± 7.35	21.7 ± 10.5	1.14 ± 0.71	2.83 ± 1.63	2.70 ± 1.66	2.57 ± 1.69[b]	5.71 ± 3.05[b]	5.91 ± 3.16[b]
	PWR	9.26 ± 3.78	20.2 ± 6.19[c]	19.2 ± 8.70	9.07 ± 3.61	20.4 ± 9.62	22.8 ± 14.1	1.55 ± 1.18	3.62 ± 1.65	3.37 ± 1.75	2.71 ± 1.94	5.41 ± 3.04[c]	4.64 ± 2.18[c]
	CON	8.40 ± 1.63	14.5 ± 3.32	16.1 ± 4.34	8.97 ± 2.34	15.8 ± 5.54	16.3 ± 5.30	1.50 ± 1.25	3.80 ± 3.16	3.85 ± 3.22	5.12 ± 5.03	10.1 ± 8.94	9.07 ± 8.20

[a]END significantly different than PWR.
[b]END significantly different than CON.
[c]PWR significantly different than CON.

Table III). The endurance group had greater peak EMG in the VM during ARS when compared to the control group ($p = 0.010$), and also displayed greater peak EMG in the VL compared to power and control groups during PS ($p < 0.050$). The control group had greater peak LH activity than the power group in the AS, PRS, and ARS conditions ($p < 0.050$), while also exhibiting greater peak LH than the endurance group during AS ($p = 0.033$). The control group also demonstrated greater LH than MH peak activity across all conditions ($p < 0.05$).

No group differences were observed for PS AUC values ($F_{2,39} = 1.566$, $p = 0.222$), but a time effect ($F_{2,78} = 93.045$, $p < 0.001$) indicated greater activity at POST-1 than PRE or POST-2 (Table IV). For AS, a significant time-by-muscle interaction was observed ($F_{6,234} = 12.281$, $p < 0.001$) revealing greatest activity in all muscles at POST-1, and POST-2 activation greater than PRE ($p < 0.05$). Additionally, quadriceps activation was greater than hamstring activation ($p < 0.001$). For the PRS condition, there was a significant interaction effect of muscle-by-group-by-time ($F_{12,234} = 1.902$, $p = 0.035$). Similar to peak activity, endurance athletes displayed highest VL activity at PRE and VM activity at POST-1 compared to the other groups ($p < 0.05$). Also, the control group had greater LH activity than power athletes in POST-2. Additionally, while quadriceps activity increased significantly from PRE to POST-1 and POST-2 ($p < 0.01$); LH activity only increased sequentially in the control group at POST-2 ($p < 0.05$), reaching a magnitude significantly greater than the VM ($p = 0.045$). For the ARS condition, there was a significant time-by-muscle-by-group interaction effect ($F_{12,234} = 3.275$, $p < 0.001$). For all muscles, activity was greater in POST-2 and POST-1 than PRE ($p < 0.001$), although endurance athletes increased VL activity from POST-1 to POST-2 ($p = 0.038$), while power and control groups decreased LH activity from POST-1 to POST-2 ($p < 0.050$). Again, control subjects' LH activity exceeded VM activity ($p < 0.001$) and was not significantly different from VL activity ($p > 0.05$) at POST-2. LH activity in the control group also exceeded that of endurance athletes at PRE ($p = 0.043$) and both endurance and power athletes at POST-1 ($p \leq 0.050$) and POST-2 ($p \leq 0.010$). Also, VM activation in endurance athletes was higher in POST-2 than power and control groups ($p < 0.01$).

Discussion

The present study aimed to determine if conditioning history is associated with changes in joint stiffness and

muscle activation strategies during a knee perturbation. The ability to properly regulate joint stiffness through appropriate activation of stabilizing muscles is crucial for functional movements and injury prevention (Needle et al., 2014). Our primary findings indicated that a power-based training history was associated with higher long-range joint stiffness in the passive condition, while endurance-based training was associated with higher long-range stiffness and muscle activation in reactive conditions, and greater short-range passive stiffness. Additionally, we found that long-range joint stiffness increased more than 30-fold with muscle activation (Table II).

Stiffness regulation

Similar to previous studies, short-range stiffness was consistently higher than long-range stiffness; and test conditions requiring muscular pre-activation (AS, ARS) displayed greater stiffness compared to passive conditions (Rack & Westbury, 1974; Sinkjaer et al., 1988). Short-range stiffness is determined by the properties of capsuloligamentous structures, elastic components of muscle, and notably the reverse-pivot of bound actin–myosin cross-bridges; while long-range stiffness largely quantifies the elastic component of the muscle and regulation of cross-bridge cycling through eccentric movement (Nielsen, Sinkjaer, Toft, & Kagamihara, 1994; Rack & Westbury, 1974). Accordingly, heightened muscular activation prior to movement would increase the number of bound actin–myosin cross-bridges, thereby increasing short-range stiffness. Interestingly, long-range stiffness was highest in the passive-reactive condition where subjects were asked to react maximally from a relaxed state. Greatest stiffness has previously been observed in the active-reactive condition where 30 percent quadriceps activation was utilized (Kim et al., 2016; Needle, Kaminski, et al., 2017), suggesting that the higher level of pre-contraction in this study may have diminished the ability to achieve a maximal torque level following the perturbation.

The passive, passive-reactive, and active-reactive conditions highlighted several stiffness differences between groups. Endurance athletes displayed the greatest short-range passive stiffness (although not statistically significantly different from power athletes) and long-range active-reactive stiffness, while sprinters demonstrated the greatest long-range passive stiffness. Power-trained athletes have previously displayed higher stiffness at the knee (Harrison et al., 2004; Hobara et al., 2008) and ankle (Arampatzis et al., 2007; Harrison et al., 2004; Hobara et al., 2010) during both perturbations and functional movement. However, previous studies have quantified stiffness indirectly by calculating the dampening effect of muscle from hopping frequencies; or using kinematic and kinetic data to estimate joint load as position changes (Arampatzis et al., 2007; Harrison et al., 2004; Laffaye et al., 2005). While this allows for a functional measure of stiffness, it does not provide the experimental control generated from our procedures (DeAngelis et al., 2015; Hamstra-Wright et al., 2005).

Higher short-range passive stiffness in endurance athletes compared to controls may be attributed to long-term adaptation of capsuloligamentous structures, as well as heightened fusimotor regulation resulting in increased muscle tone, as these would most impact short-range stiffness (Needle et al., 2014). Most likely, heightened muscle tone would increase the amount of linked actin–myosin cross-bridges at rest in passive conditions, supporting the role of low-load, high-repetition exercise for individuals lacking muscle tone. While repeated low level stresses in endurance athletes could potentially increase stiffness of the capsuloligamentous structures, it is not entirely clear why this adaptation would not exist in power athletes, who displayed short-range stiffness not significantly different from controls or endurance athletes in the passive condition, and similar to endurance athletes (but not controls) in the passive-reactive condition. Alternately, the increased long-range passive stiffness in power athletes may be secondary to a heightened reflexive response; however, this explanation seems unlikely, given the paucity of differences in muscle activation measured in this investigation. Alternately, greater passive stiffness characteristics in the series & parallel elastic components of muscle may play a role in this stiffness change (Sinkjaer, 1997), potentially caused by increased concentrations of the protein titin. Athletes, and specifically sprinters, have a higher expression of titin proteins, and this has been hypothesized to contribute to a stiffer elastic component of muscle (Kyrolainen et al., 2003; McBride, Triplett-McBride, Davie, Abernethy, & Newton, 2003; Nishikawa et al., 2012), which stiffens even more as the molecule reaches longer lengths, and once cross-bridge overlap is minimal (Powers et al., 2014). While this may contribute to the observed increases in stiffness, caution should be used as the perturbation took place below the optimal muscle fibre length of the vasti, where greater cross-bridge overlap exists (Bohm, Marzilger, Mersmann, Santuz, & Arampatzis, 2018).

When asking individuals to respond to the perturbation, endurance athletes displayed the greatest long-range stiffness, indicating an improved ability

to regulate cross-bridge cycling through muscle lengthening (Sinkjaer et al., 1988). Although we hypothesized that reactive stiffening strategies would be enhanced in power athletes, we believe that this effect was diminished by the use of 85 percent pre-activation. Although 30 percent of MVIC had been used in similar research testing (DeAngelis et al., 2015; Kim et al., 2016), these were not performed on elite athlete populations and reflected average muscle activation during gait, rather than more strenuous functional activities (Carrier et al., 2015; Sinkjaer et al., 1988).

Muscular activation

Muscle activation might be expected to remain silent in the passive condition; however, the speed and magnitude of the perturbation was sufficient to elicit reflexive contractions. No group differences were observed in AUC over time; however, peak VL activation was greater in endurance-trained subjects. Although this group demonstrated higher short-range stiffness in this condition, muscular activation would not be rapid enough to affect this value (Sinkjaer et al., 1988; Swanik et al., 2004). Rather it would increase long-range stiffness, which was higher in the power group, suggesting the VL response may have been reflexive in nature and not enough to notably impact stiffness. Therefore, we might conclude endurance athletes have greater fusimotor gain and muscle tone. These properties would contribute to a large amount of cross-bridge linkage at rest, facilitating an increased stiffness secondary to reverse cross-bridge pivoting (Needle et al., 2014; Rack & Westbury, 1974). Previous research has examined neuromuscular differences in power and endurance athletes, and several differences that may influence muscle stiffness such as motor neuron recruitment order, presynaptic/postsynaptic control of the motor pool, and motor unit firing frequency, however these variable were not measured in the current protocol (Earles, Dierking, Robertson, & Koceja, 2002; Koceja, Davison, & Robertson, 2004).

Although the passive perturbation provides valuable insight into the role fusimotor regulation plays in knee joint stiffness, rarely during physical activity would muscle activation be absent. Although maintaining 85% of MVIC produced no differences between endurance and power-trained athletes, increased hamstring activation was observed in control subjects, while quadriceps activation and knee stiffness remained equivocal to other groups. This suggests healthy controls require co-contraction of the quadriceps and hamstrings to achieve similar joint stiffness to competitive athletes. The effect of lower hamstring activity in trained athletes has been previously documented, as desensitization of antagonistic Golgi tendon organs is a key adaptation to resistance training and may permit greater stored elastic energy to enhance performance (Hutton & Atwater, 1992; Wilk et al., 1993). Although power training has been described as most effective to achieve this desensitization, we did not detect any differences in this condition between groups. As quadriceps-hamstring co-activation has been described as beneficial for prevention of joint injury, such as rupture of the anterior cruciate ligament, this decreased hamstring activation may appear to be detrimental for the prevention of anterior cruciate ligament injury. However, it may also reflect better antagonist inhibition and neuromuscular control in athletes to minimize the potential for excessive hamstring strain injuries. Further, the increased co-activation in control subjects could indicate a more guarded reaction to the knee joint perturbation to protect from injury (Hirokawa, Solomonow, Luo, Lu, & D'Ambrosia, 1991), whereas athletic groups were more comfortable with high loads transmitted to the knee joint and focused on maximizing torque output.

Passive-reactive trials tested the ability of these individuals to initiate a muscular contraction in response to a sudden perturbation from a resting state. No group differences were detected in peak EMG, but endurance athletes demonstrated greater pre-activation of the VM than power athletes and controls. These findings provide further support towards the hypothesis of increased quadriceps muscle tone among endurance athletes (Dietz, Noth, & Schmidtbleicher, 1981). This supports higher short-range stiffness in endurance athletes compared to controls, but does not explain the lack of differences between endurance athletes and power athletes as prior research has suggested that higher muscle pre-activation exists in endurance athletes compared to power athletes during hopping (Hobara et al., 2008).

Notable group differences were observed when subjects were asked to pre-contract their muscle to 85% of MVIC and then react maximally. While previous studies have utilized a smaller degree of pre-contraction, we opted for a higher degree of muscle pre-activation that more closely mimics activation levels in sport (Carrier et al., 2015; Hamstra-Wright et al., 2005; Needle, Kaminski, et al., 2017). In this condition, endurance athletes displayed greater VM activity than controls at 0–250 ms and 250–500 ms from the start of the perturbation; and power athletes had greater VM activity from 0–250 ms than controls. This finding is consistent with stiffness data where endurance athletes produced the highest long-range

stiffness throughout this condition. Although it was thought that the ability to react would be greatest among power-trained athletes, we cannot discount subtle differences in pre- or post-synaptic inhibition or firing frequencies that may impact muscle recruitment. (Earles et al., 2002; Koceja et al., 2004). It is possible that the high level of pre-contraction caused fatigue; however, rest periods, randomization of test order, and monitoring of force output were implemented to minimize these effects in this study. Both athlete groups were capable of equally increasing their muscular activity compared to controls, suggesting that either training type may be beneficial in improving muscular activation of the knee extensors, and subsequent task-specific stability during the time-range when injury is most likely to occur (within 200 ms) (Swanik et al., 2004).

Another notable difference observed throughout the active-reactive condition was increased LH activation among healthy controls. This supports previous findings where controls appeared to require quadriceps-hamstrings co-activation to attempt to maximize their stiffness. Although not statistically significant, the lowest LH activation values were observed in power trained athletes, suggesting they may be best at inhibiting this antagonistic muscle, consistent with previous literature (Hutton & Atwater, 1992; Nielsen et al., 1994). However, it is unclear whether the co-contraction observed in control subjects is beneficial or detrimental. Prior investigations have indicated that quadriceps-hamstrings co-activation may be ideal for maintaining knee joint stability (Hirokawa et al., 1991); however, certain functional situations may call for optimization of joint stiffness that requires a rapid deactivation of muscles instead of an absolute increase in co-contraction (Boden et al., 2009; Needle et al., 2014).

Practical applications

The results from this study indicate that an individual's conditioning history can play a potential role in stiffness regulation strategies. Both conditioning groups displayed potential alterations in preparatory and reactive muscle activation and stiffness regulation that may benefit dynamic restraint, potentially impacting joint stability or performance. Therefore, either training regimen may be effective in improving knee joint stability. Although higher levels of co-contraction (increased hamstring activity) were observed among control subjects, this could reflect less familiarization to large loads applied to the knee joint. The observational nature of this study leaves us unable to determine how modifiable these properties are given training techniques; however, we hypothesize endurance training may have a beneficial effect on muscle tone and subsequent joint stiffness. Conversely, power training may potentially facilitate the deactivation of certain muscles that can negatively affect performance & stability; although further research would be required to accurately determine these effects.

This study was the first to precisely measure joint stiffness and neuromuscular activation across various reaction conditions in power and endurance trained athletes. Directions for future research might investigate how these factors change across a wider range of athletes including alternate sports, or sex differences. Furthermore, while differences in these factors were observed across groups of trained versus untrained athletes, studies might aim to determine if prospective training incorporating low load, high repetition or high load, low repetition is able to significantly alter joint stiffness and neuromuscular control among untrained controls; as well as following injuries such as rupture of the anterior cruciate ligament.

Disclosure statement

No potential conflict of interest was reported by the authors.

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